UNIT 6 – VIRAL AND PARASITIC DISEASES (VMD)

Index

[Syllabus : Aetiology, epidemiology, clinical manifestations, diagnosis, treatment, prevention and control of viral and parasitic diseases of diseases of cattle, buffalo, sheep, goat, horse, pig, dog, cat and poultry: Foot and mouth disease, rinderpest, bovine viral diarrhoea, malignant catarrhal fever, infectious bovine rhinotracheitis, ephemeral fever, blue tongue, sheep pox, goat pox, PPR, classical swine fever, rabies, equine influenza, equine infectious anemia, equine rhinopneumonitis, canine distemper, infectious canine hepatitis, canine parvoviral disease, corona viral infection, adeno virus infection, feline rhinotracheitis, feline panleucopenia, feline infectious peritonitis, avian influenza, New Castle disease, Marek's disease, avian leucosis, infectious bronchitis, infectious laryngotracheitis, avian encaphalomyelitis, chicken reo virus, fowl pox, infectious bursal disease, chicken infectious anemia, inclusion body hepatitis-hydropericardium syndrome, emerging and exotic viral diseases of global importance. Parasitic diseases: Trematodes, cestodes, nematodes, protozoan infections and external parasites of clinical importance.]

CHAPTER-1: FOOT AND MOUTH DISEASE

Learning objectives

To know in detail about,

- FMD virus
- Animal affected with FMD
- Clinical signs of FMD
- Diagnosis of FMD
- Prevention and control of FMD

INTRODUCTION

Synonym

- Aphthous fever, Epizootic aphthea, Aftosa, Fast Moving Disease

Introduction

- Foot and Mouth Disease (FMD) is an acute, febrile, highly contagious disease of almost all cloven hoofed animals characterized by the formation of vesicles (fluid-filled blisters) and erosions in the mouth, nose, teats and feet with high morbidity and low mortality.
- The causative agent is Apthovirus, is an infectious, positive sense, ss-RNA virus. There are 7 serotypes of the virus: Types O, A, C, South African Territories 1, 2 and 3 (SAT-1, -2, -3) and Asia-1. Large numbers of subtypes were also identified.
- In our country only four major serotypes, O, A, C and Asia 1 are known to occur. Among which A 22, A 5 & A 10 subtypes are more commonly occur.
- Infection with one serotype does not confer immunity against another. This disease occurs naturally in cloven-hoofed animals (Both domestic and wild animals).
- Cattle are most susceptible followed by pigs and the disease is rare in sheep and goats. Several wildlife species including African buffalo, elephants, hedgehogs, deer and antelopes are also susceptible. Man may contact the disease with mild symptoms such as vesicles on the hand.
- Horse is refractory to Foot and Mouth Disease virus (FMDV) infection.
- The healthy animals are infected by contact with the secretions and excretions of the FMD affected animals.
- The main route of transmission is by inhalation and aerosol route. In addition to inhalation, transmission also occurs by direct or indirect contact with infected animals such as through abraded skin, conjunctiva, ingestion of contaminated garbage, inoculation with contaminated

vaccines and insemination. Mechanical transmission may also occur with wild animals, birds and other non-susceptible domestic animals.

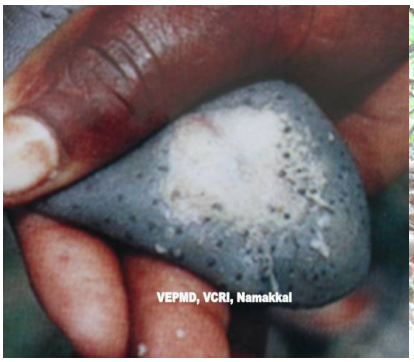
- The virus grows very well in cell cultures.
- The cell cultures that are commonly used are primary bovine tongue epithelium or bovine thyroid cells, primary pig kidney, calf kidney and lamb kidney cells.
- Cell lines such as Baby Hamster Kidney-21 (BHK-21) and IB-RS-2 cells are highly suitable. Most strains of the virus multiply to produce cytopathic effect within 24-48 hrs. This virus is highly cytolytic. Guinea pigs, suckling mice, hamsters and rabbits are used as experimental animals . Some strains grow in embryonated eggs (14 days old). Route is chorioallantoic membrane or intravenous route.

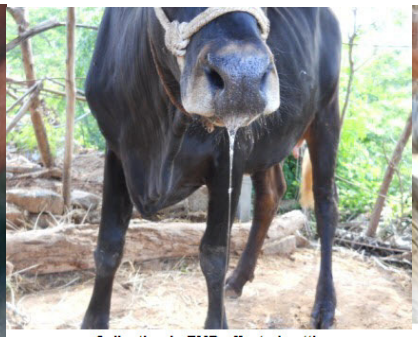
CLINICAL MANIFESTATIONS

- Incubation period is 2 days to 3 weeks. (In pigs, the incubation period is as short as 1-3 days).

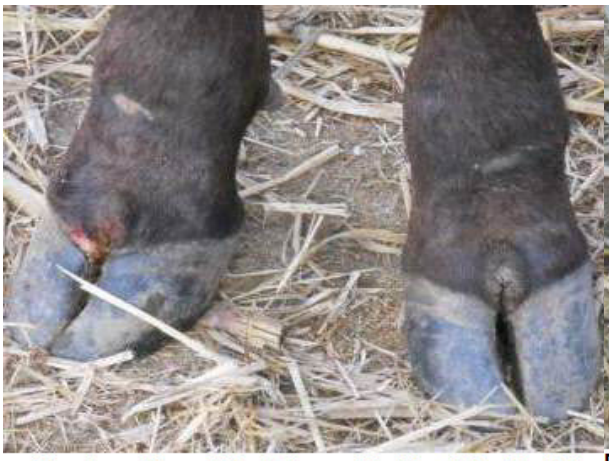
Cattle

- The onset is heralded by a precipitate fall in milk yield and a high fever (40-41^{0}C) accompanied by loss of appetite, depression. This is followed by appearance of painful stomatitis and the temperature subsides.
- There is profuse salivation, the saliva hanging in long, ropy strings, a characteristic smacking of the lips, and the animal chews carefully.
- Vesicles and bullae (1-2 cm in diameter) appear on the buccal mucosa, dental pad, udder and tongue.

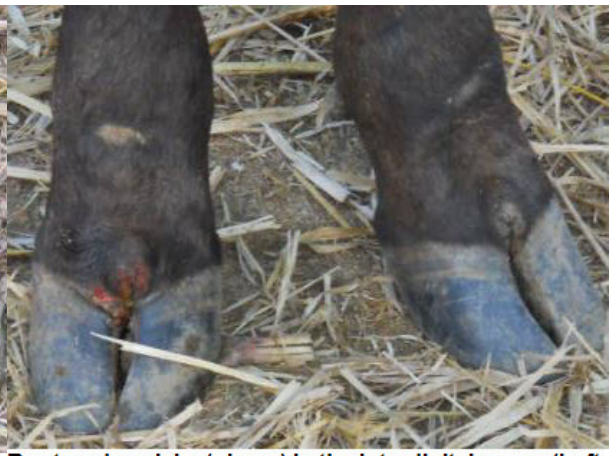



Salivation in FMD affected cattle


Ruptured vesicles in tongue

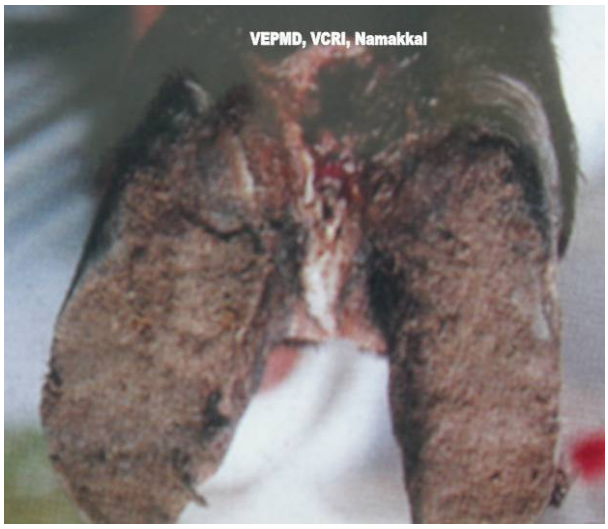


Ruptured vesicles(ulcers) in the interdigital space (Left leg)



Ruptured vesicles(ulcers) in the interdigital space (Left leg)

- The vesicles are thin walled contain a straw coloured fluid. Vesicles rupture within 24hrs, leaving a raw painful surface, which heals in about 1 week.
- Vesicles appear on the feet, particularly in the clefts and on the coronet.
- The lesions on the tongue often heal within a few days but those on the feet and nasal cavities are contaminated with bacteria, maggots, which result in lameness and muco purulent discharge.
- Vesicles may also develop on the teats, which results in severe mastitis. The virus does not cross the placenta but the abortion mostly during second trimester of pregnancy mainly due to fever. Morbidity is high (reaches 100%) and mortality is very low (less than 2%). Due to myocarditis the mortality in young calves up to 6 months of age, is very high.



- A sequel to FMD in cattle due to endocrine damage, is a chronic syndrome of dyspnea, deleterious effects on testes causing production of poor quality of semen, anaemia, over growth of hair and lack of heat tolerance described as ‘panting’.

Sheep and goats

- The pronounced clinical sign is sudden and acute lameness.
- The vesicles develop in the interdigital space of the feet, which rupture in about 2-3 days. Sometimes, the upper layer of the hoof is lost. Oral lesions are rare or they may develop only on the tongue and upper palate but there is no salivation.

Pigs

- Large vesicles and bullae occur in the snout and feet and these may rupture to expose large raw surface.
- Lameness is the first sign. The foot lesions are very painful. Vesicles in the mouth are very less prominent.

DIAGNOSIS

- The highly contagious nature of the disease, profuse salivation and the presence of typical raised vesicles with blanched covering epithelium filled with a clear straw colour fluid are usually pathognomonic.

Samples to be collected

- Vesicular fluids
- Epithelial fragments of recent vesicles especially from lesions of tongue, feet, udder or lips of about 1 gm
- Recently dead animals - pieces of cardiac muscle and pancreas
- Transport medium-The samples should be kept in a solution of phosphate buffer saline at pH 7.6 which contain equal parts of glycerol and 0.00196 phenol red indicator.
- The outside of the container should be disinfected with 4% Na_2 Co_3 or 0.2% citric acid.
- The container is wrapped with cotton wool and kept in a sealed fluid tight container packed in a strong outer box of wood and sent to the lab.

By Serological tests

- Complement fixation test (CFT) is the most commonly used and important test to identify the type of virus. The suspension of the original serum is used as antibody.
- Virus and serum neutralization tests may be used to detect specific antibodies in the serum of recovered animals or for identifying the causative virus.
- The test is done with known hyperimmune sera
- Gel diffusion test, Fluorescent Anitbody Test (FAT), Immunoperoxidase test (IPT) and Iso electric focusing are also useful
- Enzyme Linked Immunosorbent Assay (ELISA) and Polymerase Chain Reaction (PCR) are now standard methods for virus detection and typing in reference laboratories

Cell culture

- Monolayers of bovine thyroid or pig kidney cells are inoculated and CPE develops within 24-48 hours at 37°C.

Mouse inoculation

- The suspected material is inoculated in unweaned mice and the mice die within 1-7 days.

Cross immunity

- For accurate identification inoculation of groups of susceptible and immune cattle are used.

DIFFERENTIAL DIAGNOSIS

Animal species	Route of inoculation	FMD	VS	VE	SVD
Natural infection					
Cattle		+	+	-	-
Pig		+	+	+	+
Sheep and goat		+	+	-	-
Horse		-	+	-	-
Experimental infection					
Horse	Intra dermal in tongue	-	+	+	-
Guinea-pig	Intra dermal in foot pad	+	+	-	-
Suckling mice	Intra dermal in foot pad	+	+	-	+
Adult chicken	Intra dermal in tongue	+	+	-	-

CONTROL

- Spread by air borne route is very difficult to control. Control of movement of livestock is one of the effective measures.
- Where the disease is not endemic the policy of quarantine, slaughter and disinfection of infected premises has proved efficient and economical also.
- Slaughter of infected animal stops the production of virus and slaughter of animals in direct or indirect contact with the source of infection breaks the infective chain.

- This is an economic method only in countries where disease incidence is low. In other countries, it may not be economically or socially acceptable and there vaccination is carried out.
- Where the disease is not endemic the policy of quarantine, slaughter and disinfection of infected premises has proved efficient and economical.
- Inactivated vaccines containing serotypes predominant in the geographical areas are available commercially.
- Vaccines may be monovalent, bivalent, trivalent or polyvalent.
- To establish a satisfactory level of immunity it is usual to give a primary course of two inoculations, 2-4 weeks apart, followed by revaccination every 4-12 months.
- In young animals maternal antibody may last for 3-6 months and can interfere with immunization.
- To avoid gap in protection, countries recommend first vaccination at ages ranging from 2-6 months.
- For calves usually first vaccination is given at the age of 4 months, followed by booster at 2-4 weeks interval, revaccination every 6 months or 4-12 months once.
- Sheep and pigs vaccinated at 6 months of age. The dosage of vaccine in sheep is one-third of that of cattle.
- The first vaccination leads to immunity in ruminants for about 3-6 months. Subsequent vaccinations may give protection for a year in cattle but only about 6 months in sheep.
- In the areas where the greatest risk of infection is likely, ruminants are vaccinated three times a year. With a medium risk, animals are vaccinated twice a year.
- In countries where the infection is low ruminants are vaccinated twice the first year and subsequently annually. The choice of strains of FMDV to use in the vaccine is important.

CHAPTER-2: RINDERPEST

Learning objectives

To know in detail about,

- Rinderpest virus
- Animals affected with rinderpest
- Clinical signs of rinderpest
- Diagnosis, Prevention and Control of rinderpest

SYNONYMS AND INTRODUCTION

Synonyms: Cattle plague

Introduction

- Rinderpest or cattle plague is an acute, highly contagious virus disease, primarily of cattle and to a lesser degree of sheep, goats and wild ruminants.
- In India "Hill Zebu cattle" are more susceptible than "Plain zebu cattle". This disease is characterized by necrosis, and erosions of the mucosa in the respiratory and digestive tracts.
- Early constipation, usually preceded by dehydration and prostration, is followed by diarrhoea.
- Rinderpest is caused by a negative-strand RNA virus of the Morbili virus genus within the family Paramyxoviridae.
- Rinderpest is enzootic in northern equatorial Africa, the Middle East countries. Rinderpest eradicated in India.
- The virus strains are of a single serotype, but represent various pathotypes, i.e. they vary in virulence, pathogenicity.
- The virus is immunologically related to measles, and canine distemper viruses.
- The virus is usually transmitted from sick to susceptible animals in aerosols and normally the contact has to be close because the infectious droplets are large and short-lived.
- Transmission through ingestion of virus contaminated food or water is rare.
- The virus grows in sheep, goats, rabbits, hamster and white mice. It can be grown in chick embryos by intravenous or CAM route.
- Cytopathogenic effect (CPE) is produced by the virus in susceptible cells from cattle, sheep, goats, chick embryos, pigs, hamsters and man.
- Cytopathogenic effect consists of large, well defined multinucleated giant cells known as "syncytia".

- Infected cell cultures have stellate or spindle shaped cells with large fine anastamosing intracellular processes.
- The virus multiplies in the cytoplasm of the host cells like verocells (African green monkey kidney cells).
- Both intra cytoplasmic and intranuclear inclusion bodies are present.

CLINICAL MANIFESTATIONS

- In enzootic areas, where most animals could have been exposed to the virus and develop a certain degree of immunity, it may be longer.
- It is an acute febrile disease with morbidity in susceptible populations reaching 100% and mortality 90-100%.
- The normal route of infection is through nasopharyngeal mucosa.
- The course of the disease comprises of 4 stages.

I stage : Incubation period

- 2-9 days. It depends according to the strain and dose of the virus.
- The virus multiplies rapidly in the lymphoid tissue, lungs, bone marrow and intestines.
- Active proliferation of the virus in the tissue results in fever.

II stage : Prodromal phase

- There is first rise in temperature - 105-107°C (41-42°C) and lasts for about 3-5 days until the appearance of lesions in the mouth.
- Animal shows depression, restlessness and anorexia.
- Muzzle is dry, starry coat and initial constipation noticed, Leucopenia with onset of fever and persists till death.

III stage : Mucosal phase

- Mouth lesions on the inner lips and adjacent gums. Visible mucous membranes are congested.
- The mouth lesions are greyish foci with necrotic centers and shallow erosions with bleeding.
- Ulcers with bran like deposits noticed. Smacking as in FMD is not common. Animal is restless and shows excess thirst.
- Temperature is high and recedes after that diarrhoea begins.
- Rapid dehydration, marked weakness and severe progressive emaciation leads to death.

IV stage: Diarrhoeic phase

- About three days after the appearance of the mucosal ulcers fever regresses and profuse diarrhoea develops.
- The dark fluid faeces often contain mucus, necrotic debris and blood. Dehydration and wasting soon become evident.
- Severely affected animals may collapse and die within 12 days of the onset of clinical signs.
- In surviving animals convalescence lasts several weeks.

V stage: Convalescent phase

- Mouth lesions start healing. Rapid regeneration of the affected epithelium noticed.
- Slow recuperation of general health. Mortality in cattle, sheep and goats and pigs is 90%.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Samples

- Spleen, pre scapular or mesenteric lymph nodes, ocular or nasal secretions of acutely infected animals
- Based on clinical signs
- Isolation and identification of the virus - Isolation of the virus from leukocyte fraction of whole blood that has been collected into heparin or EDTA (ethylene diamine tetra acetic acid), monolayer of primary calf kidney, B95a marmoset lymphoblastoid or African green monkey kidney (Vero) cells.
- Glycerol should not be used as preservative for Rinderpest virus.
- Serological tests – AGID, VNT (virus neutralization test), Competitive ELISA
- RT-PCR

Differential diagnosis

- Haemorrhagic septicemia
- FMD
- Mucosal disease
- Bovine Viral Diarrhoea

PREVENTION AND CONTROL

Vaccination

- *Laprinised vaccine* : Immunity last for 1-7 years
- *Goat tissue vaccine* : Immunity last for 13 years or life long

- In countries where Rinderpest is exotic, confirmed outbreaks are controlled by the slaughter and disposal of all affected and in contact animals, as well as by appropriate quarantine and animal movement controls
- All outbreaks of rinderpest in virgin areas have been due to the importation of live infected animals.
- Prevention in such areas is therefore largely dependent upon vigilant control of the introduction of live animals from potentially infected areas
- Contaminated areas should be physically cleaned of all animal waste and soiled bedding and treated with disinfection solutions of high (>10) or low (<3) pH containing solvents to destroy the virus envelope. Solutions of caustic soda and Lysol have the highest virucidal activity against virus contaminated with organic matter
- Attenuated tissue culture (bovine kidney cells, vero cells) Kabete “O” strain of RPV produced a totally safe and effective attenuated virus which has been the principal vaccine used to combat rinderpest throughout the world during the past 40 years. It induces a lifelong immunity, inexpensive and simple to produce and stable in freeze dried form.
- The only drawback of the vaccine is that after reconstitution in normal saline it has a working life of only a few hours in the hot climates
- Recombinant vaccines in which genes expressing RPV immunizing antigen are incorporated into more thermostable pox viruses have been developed and used for differentiating vaccinated animals with the infected animals and thus helps in eradication of disease
- In endemic areas calves at the age of 6-12 months vaccinated for RP. Annual vaccination of all cattle will produces highest levels of herd immunity.

CHAPTER-3: BOVINE VIRAL DIARRHOEA AND MALIGNANT CATARRHAL FEVER

Learning objectives

To know in detail about

- BVD and MCF virus
- Animal affected with BVD and MCF
- Clinical signs of BVD and MCF
- Diagnosis, prevention and control of BVD and MCF

BOVINE VIRAL DIARRHOEA

INTRODUCTION

- Bovine viral diarrhoea (BVD) and mucosal disease (MD) are clinically dissimilar disease syndromes, and were originally described as separate diseases, but they are now known to have a common viral etiology. The virus can cause both acute disease, bovine viral diarrhoea and a protracted form of illness, mucosal disease, arising from persistent infection.
- BVD may occur at any age. MD is a persistent infection acquired *in utero* and characterized by high mortality and low morbidity.
- The BVD virus can be either non cytopathogenic or cytopathogenic. Isolation and demonstration from both BVD and MD were serologically similar and gave the same experimental disease.
- The clinical evidence that abortions were constant finding of field outbreaks of BVD and also the demonstration of transplacental transfer of the virus to the fetus.
- Early fetal infection with BVD virus may lead to persistent viraemia and a failure to develop antibodies.
- Persistently viraemic animals could subsequently develop MD. Persistently viraemic animals are infected with only non-cytopathogenic virus, whereas those developing MD are infected with both non-cytopathogenic and cytopathogenic forms.
- Mucosal disease is almost invariably fatal disease with low morbidity. It occurs in cattle, which have a persistent BVD infection acquired as fetuses characterized by a specific immune tolerance to the infecting virus strains and consequent lack of antibody to it.
- In MD, if the fetus infected at 110-120 days of gestation, i.e., before the age of immunocompetence, the virus become accepted as self and persist through out the life of the animal. This explains the lack of antibody to persisting virus and the continued state of immuno tolerance.
- Sometimes after birth, when the animal is about 6-18 months of age, super infection of these persistently viraemic animals with the cytopathogenic bio-type may occur causing MD.

- Bovine viral diarrhoea virus (BVDV) is a SS-RNA virus, genus Pestivirus in the family Flaviviridae and is closely related to classical swine fever and Ovine Border disease viruses. This virus affects domestic and wild ruminants and pigs. There are two genotypes of the BVDV recognized.
- Both viral genotypes have been co-circulating in cattle throughout the world. This disease reported from most regions of the world. Natural infections and disease occur not only in cattle but also in sheep, pigs, goats and a wide range of captive, free-living ruminants.
- BVDV also classified into two biotypes-Non cytopathogenic and cytopathogenic biotypes
- The most important sources of BVDV in nature are immuno-tolerant, persistently infected animals.
- High virus levels are usually present in their nasal secretions, saliva, tears, semen, milk, urine and faeces.
- Animals in contact with a persistently infected animal rapidly become infected. Vertical and horizontal transmission may occur. Disease transmits by direct or indirect contact.
- The virus may be isolated in a number of bovine monolayer cell cultures (e.g. kidney, lung, testis or turbinate). Growth of both biotypes is usually satisfactory.

CLINICAL MANIFESTATIONS

The clinical and pathologic manifestation of infection in individual cattle varies with age and pregnancy status. Three situations are considered,

- Postnatal infection in non pregnant cattle
- Infection in pregnant cattle
- Persistent infection in calves and mucosal disease.

Postnatal infection of susceptible non pregnant cattle

- *Acute infection*
    - Infection is most common in animals 8-24 months of age. Although calves may receive antibodies in colostrum, antibody levels decline by 3-8 months of age and animals can then become infected.
    - Incubation period 5-7 days followed by pyrexia (40-41°C), leucopenia, viraemia. In majority it is sub clinical infection.
    - There may be diarrhoea of explosive nature with very high morbidity but no mortality.
    - Drop in milk yield in dairy cows, oculo-nasal discharge and mouth ulcers is referred to as BVD. Animals develop serum neutralizing antibodies which persist life long.
- *Immuno suppression*
    - Virus induces transient but profound immuno suppression.

- The virus suppresses the interferon production, affects lymphocyte function, humoral antibody production and phagocytosis which paves way for many respiratory and other diseases in calves.

- *Venereal infection*
    - Semen from persistently infected bulls by AI or natural service infect cows which results in early embryonic mortality and repeat breeding.

Infection of susceptible pregnant cattle

- *Transplacental infection*
    - The infection to the fetus causes abortion, weak calves, under sized calves and congenitally deformed calves.
    - The virus replicates in almost all the fetal tissues and the extent of damage is more in actively dividing cells.
    - Regarding the nervous system it causes cerebellar hypoplasia, dysplasia, cavitation of the cerebrum and retinal displacement.
- *Immunological competence of the fetus*
    - Infection in early fetal life becomes persistent viral infection in many tissues and organs. After birth, the calf remains infected for life.
    - These calves excrete the virus in large quantity and transmit to all susceptible healthy animals. Therefore, high probability of development of MD is possible.

Persistently infected cattle

- In susceptible healthy herds in which virus was recently introduced, high proportion of calves in the next calving season become persistently infected.
- Mortality rate exceeds 50% in the preslaughter age i.e., 6-18 months age and the most classical clinical manifestations are that of MD. This disease is characterized by pyrexia, anorexia, profuse watery diarrhoea, nasal discharge, buccal ulceration and sometimes lameness. Death is within a few days to 3 weeks.

DIAGNOSIS

- Clinical diagnosis is a complex problem. Signs of the different syndromes may appear alone or in combination.
- Although many of the signs are similar to RP, the mortality in that disease is much higher.
- The oral lesions are very important, but their absence does not exclude BVD.
- This disease must be differentiated from conditions in cattle causing diarrhoea, erosions/ulcerations of the gastrointestinal tract, reproductive failure, teratology, skin disease, laminitis, poor growth and respiratory tract disease such as vesicular diseases, ingestion of caustic substances, mucosal disease complex: Rinderpest, bluetongue, papular stomatitis, malignant catarrhal fever.

Laboratory diagnosis

- Samples to be collected- nasal, ocular discharges, blood, spleen, lymph nodes, lungs, and liver
- Identification of the agent- Persistently viraemic healthy animals resulting from congenital infection can be readily identified by isolation of noncytopathogenic virus in cell cultures from blood or serum.
- It is necessary to use an immune-labelling method to detect the growth of virus in the cultures.
- Alternative methods based on direct detection of viral antigen or viral RNA in leukocytes are also available.
- Persistence of virus should be confirmed by resampling after an interval of at least 3 weeks.
- These animals will usually have no or low levels of antibodies to BVDV.

Serological tests

- Acute infection with BVDV is best confirmed by demonstrating seroconversion using sequential paired samples from several animals in the group.
- The testing of paired sera (acute and convalescent samples) should be done a minimum of 21 days apart and samples should be tested side by side.
- The enzyme-linked immunosorbent assay (ELISA) for antibody and the virus neutralization test are the most widely used. Demonstration of the BVDV by CPE (but many BVDV strains do not produce CPE), FAT, PCR a much more productive method, are necessary for definitive diagnosis.

PREVENTION AND CONTROL

Control

- Primary goal of a control strategy is removal of persistently infected animals from a herd.
- All cattle should be screened for the virus including calves born in the herd for nine months after initiation the programme.
- Newly introduced animals should be tested for the disease before their introduction. Quarantine period for newly purchased animals is 30 days.
- Strict managemental practices.
- Replacement animals especially breeding bulls.
- Cattle should not be allowed to mix with sheep and goats

Vaccination

- Modified Live BVD vaccine - Not recommended for pregnant cattle or in stressed populations
- Killed BVD vaccine, which is safer for administration even to pregnant cattle. A booster dose after 3 to 4 weeks of initial vaccination.

- Calves should be vaccinated at 4-6 months of age and again when they are 8-12 months old.
- Calves less than three months of age generally not vaccinated because of potential vaccination failure associated with the presence of colostral immunity.
- Heifers and cows should be revaccinated 30-60 days before breeding and booster dose one month before calving or in the last trimester.

MALIGNANT CATARRHAL FEVER

SYNONYMS AND INTRODUCTION

Synonyms : Bovine malignant catarrh, Malignant head catarrh, Catarrhal fever, Epitheliosis

Introduction

- Malignant catarrhal fever (MCF) is almost invariably fatal disease of cattle characterized by catarrhal inflammation of the nasal and oral mucosa, keratoconjunctivitis, encephalitis, rapid dehydration and generalized enlargement of lymphnodes.
- The disease is usually sporadic in nature.
- The disease is world-wide distribution and prevalent in both temperate and tropical zones.
- This disease is caused by ovine herpes virus -1 and alcelaphine herpes virus- 2 which are belonging to the family Herpesviridae.
- The disease is primarily a disease of cattle and buffalo. Inapparent infection has been noted in sheep and goat. Now the disease is increasingly encountered in deer.
- This disease is transmitted through wildbeast. The wildbeast develop viraemia which may persist up to 3 months and during that time they remain highly infective for cattle of all ages.
- Cattle acquire the infection through sub clinically affected sheep. The sheep are the indicator host and wild rabbits are the reservoir host for MCF.

CLINICAL SIGNS

- Incubation period of the disease ranges from 2-8 weeks. As a whole the following four clinical findings have been outlined but it is not a rigid one and all the manifestations may follow in a case.

S.No.	Form of the disease	General clinical signs
1	Peracute form	High rise of temperature, dyspnoea and diarrhoea but with out any head and eye lesions
2	Head and eye form	Clinical course of around 9 days characterized by nervous syndrome like paralysis and convulsion prior to death
3	Intestinal form	
4	Inapparent (mild) form	

- The disease as a whole is characterized by
    - Dullness, depression, anorexia and high rise of temperature ranging from 105-108ºF. This is followed by ocular and nasal discharge.
    - Discharges are initially mucoid in nature which soon become mucoprulent, stingy and contain blood flecks. There is acceleration of pulse and respiratory rate. Signs of dysponea may be evident.
    - There is oedema of the eyelids leading to panopthalmitis
    - Changes in the epithelium of mouth cavity comprising of congestive changes, precisely on the gum, beneath the tongue, hard palate and oral papillae. Erosive and ultimate necrotic changes may follow in the oral mucosa giving rise to offensive odour from the mouth.
    - Ocular lesions– Photophobia and meiosis, corneal opacity, blindness
    - Cutaneous lesions (congestion, petechiation, bluish discolouration and thickening) localized or generalized prominent on the muzzle, skin of the hoof, scrotum, base of the horn and teat.

DIAGNOSIS, PREVENTION AND CONTROL

Diagnosis

- Based on clinical signs
- No reliable serological or immunological tests
- Materials to be collected- Lesions on the oral, nasal, ocular lesions, enlarged LN

Differential diagnosis

- Rinderpest
- Mucosal disease
- Infectious bovine rhinotracheitis

Prevention and control

- Inactivated or live vaccines with suitable adjuvants induce antibodies against MCFV.
- In the absence of a vaccine, the only effective strategy is to limit contact between MCF susceptible species and the natural hosts of the virus.
- Sheep has been considered as a spreader of field outbreaks. Therefore, rigid separation of cattle from sheep is suggested.
- The feed used by ewes and lambs should not be provided to cattle.
- Avoid contact between reservoir host (wildebeest and wild rabbits) and cattle

CHAPTER-4: INFECTIOUS BOVINE RHINOTRACHEITIS

Learning objectives

To know in detail about

- Etiological agent of IBRT
- Different clinical forms of IBRT
- Diagnosis, prevention and control of IBRT.

SYNONYMS AND INTRODUCTION

Synonyms : Viral bovine rhinotracheitis, Red nose, Necrotic rhinitis

Introduction

- It is an acute highly contagious viral disease of cattle characterized by high temperature, rhinitis, dyspnoea, abortion, meningoencephalitis, keratoconjunctivitis and pustular vulvovaginitis.
- Dairy and beef cattle are equally susceptible. Besides cattle, the disease also reported in goat, swine and water buffaloes. The disease is widely prevalent in all parts of the world.
- In India the disease has been recorded from various states including Uttar Pradesh, Kerala, Gujarat, Tamil Nadu, Orissa, Andhra Pradesh and Karnataka.
- The disease is caused by a DNA virus called bovine herpes virus-1 belonging to the family herpes viridae, subfamily Alpha herpes virinae and genus Varicello virus.
- Cattle of all ages are affected.
- Dairy and beef cattle are equally susceptible.
- Besides cattle the disease also reported in goat, swine and water buffaloes and in wild ruminants.
- The virus is transmitted through infected feed and water.
- The virus can spread through ocular, nasal and reproductive secretion and excretion of infected cattle.
- Droplet infection is the important way of transmission.

CLINICAL MANIFESTATIONS

- The clinical signs have been grouped as
    - Respiratory form
    - Vulvo-vaginal form
    - Ocular form
    - Encephalomyelitic form
    - Abortive form

Respiratory form

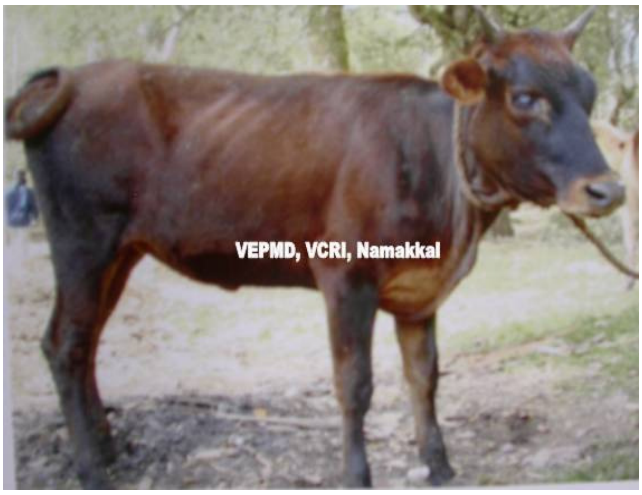
- This form is characterized by mild to severe rise of temperature, depression of appetite, acceleration of respiration and dyspnoea.
- The nasal discharges are initially serous which later turn to mucopurulent. Whole of the upper respiratory tract show hyperemia, edema along with mucopurulent exudation causing dyspnoea.
- Affected cattle may exhibit open mouth breathing in severe cases. Animal may show signs of bronchitis and pneumonitis.
- The nasal mucosa is severely congested hence the disease is named as "Red nose".
- The recovered cattle may remain as carrier and thus shed the virus for a considerable period.

Vulvo-vaginal form

- This form is characterized by sharp fall in milk yield and appearance of erythematous and pustular lesions on the vulvar and vaginal mucosa.
- There is swelling of vulva and frequent urination. There may be mucopurulent discharge from vulva and vagina.
- Animal is unable to put its tail in normal position after urination due to pain. The virus produces pustular balanoposthitis in bull.
- The semen of the affected bull become contaminated and thus pose problem in natural or artificial breeding

Ocular form

- This form may appear along with respiratory form. There is inflammation of the conjunctiva in addition to respiratory changes.
- But in some occasions severe conjunctivitis and ocular discharges may be noted without respiratory involvement.
- The ocular discharges vary from serous to purulent.
- Petechial hemorrhages may be noted on the conjunctiva and sometime corneal opacity may appear as main attribute of the disease.



Encephalomyelitic form

- The virus may produce severe encephalomyelitis syndrome in calf terminating to death.
- The signs of encephalomyelitis comprise of high rise of temperature, incoordination, tremor, circling, falling, coma and death.
- Death ensures within 4 days following appearance of neurological signs.

Abortive form

- The pregnant cattle may abort following infection. The abortion may supervene as “abortion storm”.
- Foetus died at 4 months of gestation and expelled. Foetus is autolysed in most cases.

DIAGNOSIS

Samples to be collected

- Live animals- nasal swabs or genital swabs or conjunctival swabs during the acute phase of infection, aborted materials, tissue samples from the fetal liver, brain, and spleen, semen, placenta, uterine mucus and serum
- Dead animals- Lymph nodes, liver, lungs, brain, aborted foetal liver, brain and spleen

Based on clinical signs

Identification of the agent

- The virus can be isolated from nasal swabs or genital swabs, from animals with vulvovaginitis or balanoposthitis, taken during the acute phase of the infection, and from various organs collected at post-mortem.

- For virus isolation, various cell cultures of bovine origin are used, for example, secondary lung or kidney cells or the Madin–Darby bovine kidney cell line.
- The virus produces a cytopathic effect in 2– 4 days. It is identified by neutralization or antigen detection methods using monospecific antisera or monoclonal antibodies.
- Viral DNA detection methods have been developed, and the polymerase chain reaction technique is increasingly used in routine diagnosis.

Serological tests

- The virus neutralisation test and various enzyme-linked immunosorbent assays (ELISA) are most widely used for antibody detection.
- ELISA antibodies can be detected in serum and with lower sensitivity in milk.
- Fluorescent antibody technique (FAT) and electron microscopy can be made for diagnosis of disease.

DIFFERENTIAL DIAGNOSIS

- This disease should be differentiated from

Pasteurellosis	Respiratory signs alone present and responds to broad spectrum antibiotic therapy
Parainfluenza	Toxaemia, dyspnea, - Antibiotic treatment for secondary bacterial complications and recovery in 3-5 days
Verminous pneumonia	Respiratory signs, esinophilia, responds to broad spectrum antibiotic therapy
CBPP	Fibrinous pneumonia, pleurisy, deep cough, extension of head, abduction of elbow, pleural rub, differentiated with IBR with serological tests

PREVENTION AND CONTROL

- No successful treatment. Antibiotics given to avoid or prevent secondary bacterial complications
- Best way to control this disease is to prevent contact between infected animals and seronegative animals.
- Since the virus is latent in seropositive animals, they should be identified and eliminate them from the herd
- Good hygiene, management and isolation do not seem control the disease adequately
- Most control efforts are based on vaccination. The best practice is to vaccinate females kept for breeding once or twice prior to their first breeding with modified live virus vaccines or inactivated vaccine. Age of vaccination – calves after 5 months of age. Immunity develops within 10-14 days.

- Live vaccines induce a relatively rapid immune response comprising both humoral and cell-mediated responses, including mucosal immunity that resembles a natural infection.
- Live attenuated vaccine administered through intramuscular or intranasal route, but modified live virus (MLV) vaccines contraindicated in pregnant cows because MLV have the property of abortigenicity. In outbreaks- intranasal route selected. Vaccine may be instilled into one or two nostrils.
- Killed vaccines are administered by intramuscular route. Primary and secondary inoculation should be given four weeks apart followed by booster inoculations annually.

CHAPTER-5: ENZOOTIC BOVINE LEUKOSIS AND EPHIMERAL FEVER

ENZOOTIC BOVINE LEUKOSIS

Learning objectives

To know in detail about

- Etiological agent of enzootic bovine leukosis and ephemeral fever
- Different clinical signs, prevention and control of enzootic bovine leukosis and ephemeral fever

SYNONYMS AND INTRODUCTION

Synonyms

- Bovine leukemia, Bovine leukosis, Bovine lymphosarcoma

Introduction

- It is a lymphoproliferative disease of cattle where there is extensive proliferation of leukocyte forming tissues.
- Bovine leukemia virus (BLV) belongs to the family Retroviridae. Only one antigenic type has been found.
- Most infected animals develop a persistent lymphocytosis without any apparent clinical features.
- The virus produces syncytia in cell cultures. This is used as an assay for virus or antibody detection.
- The bovine leukemia virus (BLV) is distributed worldwide and occurs particularly frequently in dairy cattle.
- Cattle and buffaloes are the most susceptible hosts. Although non-immune cattle of all breeds can be infected at any age with bovine leukemia virus (BLV), most infections occur in dairy cattle more than two years of age.
- The fact that disease is seldom seen in younger animals is related to the presence of protective maternal antibody (for 5 - 6 months) and the separation of younger animals from the remaining herd until they reach sexual maturity. Most infections are inapparent and as many as 80% of a dairy herd may be infected.
- Cattle are infected by direct contact with blood from an infected animal during blood sampling, clinical examinations, castration and dehorning.
- Mechanical transmission by biting insects may play a role, particularly in tropical regions.

- This disease also transmitted through vertical transmission routes such as transovarian, transplacental and through colostrum. Approximately 10 to 15% of calves born to infected cows are infected.

CLINICAL MANIFESTATIONS

- Most animals infected with BLV remain clinically normal. Those that develop disease (approximately 2%) ultimately die after a long (weeks to months) clinical course.
- Initial clinical signs are often those of weight loss and reduced milk production, but may be quite varied depending on the site of tumor development.
- The disease is a B-cell lymphoma. The organs most commonly affected are the lymph nodes, heart, abomasum, uterus, and spleen.
- When the superficial lymph nodes are involved they may be swollen and appear as lumps under the skin usually in the neck and rear flank region.

DIAGNOSIS

- *Specimens* - Affected tissues (formalin-fixed) and serum.
- Presumptive diagnosis of BL is often based on the finding of tumors in the locations mentioned above upon clinical and gross necropsy examination.
- Microscopic examination of affected tissues is required to confirm the disease.
- It must be kept in mind that there are sporadic forms of non-viral bovine leukosis (thymic, multicentric); they usually affect younger animals.
- *Identification of the agent* : Virus can be isolated following in-vitro culture of peripheral blood lymphocytes from infected animals by electron microscopy or by BLV antigen detection in the culture supernatant.
- Proviral DNA can be detected in peripheral blood lymphocytes or tumors by the polymerase chain reaction.
- *Serological tests* : The antibody detection methods widely used are the agar gel immunodiffusion (AGID) assay using serum and the enzyme - linked immunosorbent assay (ELISA) using serum or milk.
- These tests have formed the basis for successful eradication policies in many countries. Other tests, such as radio-immunoassay, can also be used.
- A number of AGID and ELISA kits are available commercially.
- The virus can be isolated and cultivated in blood lymphocytes but this is not practicable for diagnosis.

PREVENTION AND CONTROL

- No curative treatment
- Vaccines to prevent BL are not available.
- Eradication is accomplished by testing and removal of serologically positive animals and only admitting to the herd serologically negative cattle.
- Care must be taken to prevent spread of the virus during blood sampling, clinical examinations, castration, dehorning, etc. Insect control may be advisable in affected areas.
- In heavily infected herds eradication may not be feasible. Negative animals should be separated from the serologically positive and efforts directed to preventing spread.
- By taking adequate precautions it is possible to maintain herds with a few seropositive animals, without spread to other animals.

EPHIMERAL FEVER

SYNONYM AND INTRODUCTION

Synonym

Three-day sickness, 3-day fever, Dragon Boat disease

Introduction

- Bovine ephemeral fever (BEF) is a benign acute, non-contagious, arthropod-borne viral disease of cattle and water buffalo characterized by sudden onset, lameness and quick recovery with high morbidity and low mortality.
- This disease occurs in tropical and subtropical regions of Africa, Asia and Australia.
- The BEF virus belongs to the genus Rhabdovirus. This virus is related to dengue fever virus in human.
- All breeds of cattle are susceptible.
- Among the cattle, 6 months to 2 years are more susceptible.
- This disease is prevalent both in indigenous and exotic breeds of cattle as well as water buffalo.
- The disease is not reported in goat, sheep, pig and dog. Virus transmitted mainly by sand flies (Ceratopogonidae family).
- Mosquitoes (Aedes, Anophles and Culex), and biting midges are playing a major role in transmission.
- Strong wind can transport vectors for longer distances.
- Epizootics associated with recent rainfall. The virus does not spread by any other modes.

CLINICAL MANIFESTATIONS

- The incubation period is 2 to 10 days
- Calves are least affected, those less than 6 months of age showing no clinical signs.
- After an incubation period of 2-10 days, there is sudden onset of fever. A biphasic or triphasic fever and sharp fall in milk yield are commonly observed.
- There is severe ruminal stasis followed by constipation.
- Muscular signs become more evident on the second day with severe stiffness.
- Swellings in shoulders neck and back may be seen. A posture similar to that of acute laminitis, with all four feet bunched under the body is characteristic.
- On the third day the animal begins eating and ruminating and the febrile reaction disappears
- Some animals remains standing in acute stage and exhibit sternal recumbency associated with hypocalcaemia (Milk fever posture). Most recovered animals develop a solid immunity.
- The morbidity rate may reach 100% and the fatality rate is 1%. Milk yield returns to normal within 2 to 3 weeks.

POST DISEASE COMPLICATIONS AND LESIONS

Post disease complications

- Fall in milk yield
- Increased susceptibility to mastitis
- Delay in next estrus
- Pneumonia due to prolonged recumbency

Lesions

- Enlargement and edema of the lymph nodes
- Polysinovitis, tendovaginitis and periarthritis
- Pulmonary and pleural congestion
- Edema and haemorrhages of the brain and meninges
- Necrotic changes in the skeletal muscle.

DIAGNOSIS

- Sample of choice - Blood (Buffy coat) and serum is the highly suitable specimen.
- Based on clinical signs - rapid and short course affecting a large number of cattle in an area
- Virus isolation - By intracerebral inoculation of mice or hamster or isolation in cell culture such as Baby hamster kidney cell lines and monkey kidney cell lines.
- Other tests - CFT, AGID, FAT, ELISA

- Based on clinical pathology - Increased level of plasma fibrinogen and decreased serum calcium level, high level of creatinine kinase and marked leukocytosis

Differential diagnosis

- Laminitis
- Milk fever

PREVENTION AND CONTROL

- Treatment revolves around the generalized inflammation and the depression of serum calcium.
- The inflammation can be treated with phenylbutazone given intramuscularly for eight hourly periods of up to 3 days depending on response.
- Treatment with calcium borogluconate is warranted when signs of hypocalcemia are present.
- Supplementary antibiotic treatments to avoid secondary pneumonia or mastitis are warranted.
- Vector control is usually impractical in endemic areas. In endemic areas, it is important to immunize cattle; particularly highly valuable animals.
- Both formalin killed and attenuated vaccines are available. Calves aged more than six months vaccinated.
- Trials have been carried out using a limited vaccine based on the envelope-glycoprotein has been developed.

CHAPTER-6: BLUE TONGUE

Learning objectives

To know in detail about

- Causative agent of blue tongue
- Transmission, clinical signs, diagnosis and prevention of blue tongue

SYNONYMS AND INTRODUCTION

Synonyms : Sore mouth or sore muzzle, ovine catarrhal fever.

Introduction

- Blue tongue (BT) is an infectious, non-contagious acute arthropod borne virus, primarily affecting sheep characterized by catarrhal inflammation of the mucous membrane of buccal mucosa and gastro intestinal tract.
- This disease frequently involves the udder, coronary band of the foot and sensitive laminae of the hoof. There is epithelial desquamation but no vesicle formation occurs.
- Blue tongue virus (BTV) is the double stranded RNA virus, species of the Orbi virus genus, family Reoviridae.
- The disease first recognized in Africa and reported from Canada, Japan, Australia, Brazil, West Indies, USA, Egypt, Israel, Cyprus, and in some other countries.
- This disease first reported in India in 1964.
- The distribution of the disease is dependent on the presences of reservoir and amplifying host such as cattle and on suitable species of *Culicoides* being present in large numbers to effect transmission to sheep.
- It is basically a disease of sheep but amongst sheep susceptibility varies in different age groups.
- Young sheep within the age group of one year are more prone to infection.
- Goats, cattle and wild ruminants exhibit milder symptoms and may act as non-clinical carriers.
- Horses, dogs, cats, ferrets, rabbits and guinea pigs are not susceptible to natural infection.
- The disease spread through blood sucking midges of the genus *Culicoides*.
- Besides mosquitoes and other ectoparasites like sheep ked, *Melophagus ovinus,* may transmit the disease mechanically.
- Wild animal reservoirs play an important role in maintaining the infection during the interepizootic period.

- Stress factors like lower plane of nutrition, worm burden, inclement weather, and fatigue due to transportation are predisposing factors for the disease.
- Blue tongue virus can most readily be identified isolated by inoculating into a 10-12 days old embryonated chicken eggs.
- Primary isolation can only achieved by inoculating the suspected materials into intracerebral inoculation of new-born mice or in monolayer cell cultures such as BHK-21, mouse L cells, vero cells.

CLINICAL MANIFESTATIONS

- Incubation period one week. The disease has been arbitrarily divided into three forms.
- Acute form
    - High rise of temperature, nasal discharge, salivation and lacrymation
    - Hyperemia, Petechiation and swelling of the buccal mucosa, dental pads and tongue. Later hyperaemic regions become "cyanotic" or "purplish blue"
    - Extensive necrosis of the dental pad
    - Muzzle turns dry show burnt appearance

    - Cyanotic and bluish appearance of tongue
    - Blood stained diarrhoea occurs when the intestinal mucosa is involved which is always fatal.
    - There may be involvement of sensitive laminae and breaking of wool fibres
    - Heavy mortality occurs in early stages and chronically affected animals become thin and emaciated. Morbidity is 50%.
- Sub acute form or subclinical form
    - Common in cattle and generally passed unnoticed

- Abortive form
    - Abortion of pregnant ewes, which results in the loss of entire breeding season. Young animals and Merino sheep are particularly susceptible.

Lesions

- *Tongue :* Blue and gangrenous
- *Leg :* Coronary band show congestion and hemorrhage
- *Lungs :* pneumonic changes

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Diagnosis

- Based on history, clinical findings, seasonal occurrence
- Samples to be collected -unclotted blood from febrile animals or fresh spleen and lymph node collected at post mortem
- Virus isolation in unweaned mice or growing in susceptible cell cultures or in embryonated eggs (i/v or yolk sac routes).
- The virus propagated in primary cultures of calf kidney, adrenal and testis, HELA cell cultures and also BHK21. CPE within 24-72 hours, which consists of foci of enlarged and retractile cells
- Other tests – CFT, SNT, Plaque inhibition test, Microgel diffusion test, FAT, AGID, ELISA

Differential diagnosis

- Foot and mouth disease
- Malignant catarrhal fever
- Photosensitization

PREVENTION AND CONTROL

- The recovered sheep are immune for 6 months. Grazing of the animals should be avoided in areas where there is lot of vectors.
- Vector control by applying insecticides and good water management
- Quarantine of the newly purchased animals

Vaccination

- Live, attenuated, avianated vaccines are used as monovalent strain or quadrivalent vaccine.
- Live vaccines should not be used in pregnant sheep.
- Sheep are vaccinated annually, and several weeks before service and before the start of rainy season.
- Lambs vaccinated at 6 months of age and booster vaccination at once in a year

CHAPTER-7: SHEEP POX AND GOAT POX

Learning objectives

To know in detail about

- Sheep pox and goat pox virus
- Pathogenic effect of sheep pox and goat virus
- Diagnosis, prevention and control of sheep and goat pox.

SHEEP POX

SYNONYMS AND INTRODUCTION

Synonyms : Ovine pox, Variola ovina

Introduction

- Sheep pox is a contagious pox disease of sheep caused by sheep pox virus and characterized by febrile condition and generalized development of pock lesions in the areas denied of wool.
- It is considered as one of the most dangerous pox virus infections among animals. This disease also affects the mucous membranes of the respiratory and gastrointestinal tracts.
- Sheep pox virus is a DNA virus belonging to the family Poxviridae, sub family chorodopox virinae, genus Capripox.
- The disease is prevalent in many areas of Middle East countries, Africa, Asia and Mediterranean countries. The disease is widely prevalent in different parts of India.
- The sheep are the naturally susceptible host to this virus. Young sheep are more susceptible than older ones.
- In the young lambs the disease may flare up in an epidemic proportion.
- Transmission takes place due to direct contact of the infected animals with the healthy one.
- Droplet infection through nasal inhalation is an important way of disease transmission.
- Wounds and abrasion of the skin augment spread from one animal to other.
- Dog, cat, bird and animal handlers may mechanically transport the infective materials from one place to other.
- The scabs contain the virus and the virus may remain active on the wool or infected pens for as long as 6 months.
- On inhalation the virus enters the lungs. Transplacental transmission from ewe to the foetus results in the birth of lambs with pox eruptions.

CLINICAL MANIFESTATIONS

- Incubation period is 4-8 days after natural infection.
- The disease may appear in three clinical forms
    - Malignant form
    - Mild (benign)form
    - Abortive form
- *Malignant form*
    - Most common
    - Lacrymation, salivation, serous nasal discharge, swelling of the eye lids, congestion of mucous membranes.
    - High fever with pock lesions on the eye lids, lips, nostrils, ears, cheeks, inner side of the thigh, scrotum, prepuce, vulva, under the tail, chest region and buccal mucosa.
    - Lesions first appear as macules, then turn into papules, which transform into large vesicles followed by necrotic changes in the vesicles results in scab formation.

- If lambs are affected mortality-50 %
- Death due to respiratory embarrassment
- *Mild (benign) form*
    - Common in adult sheep
    - Common form of the disease in indigenous breeds of sheep
- *Abortive form*
    - Pregnant ewes abort, foetus show pock lesions
    - In lactating ewes- mastitis due to lesions in the udder

Lesions

- Characteristic papules, vesicles, pustules and scabs on cutaneous surface
- Lesions in the mucosa of respiratory and alimentary tract especially in the trachea

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Diagnosis

- Samples- vesicles and vesicular fluid, scabs
    - Based on characteristic lesions on the skin and infected cells show intracytoplasmic acidophilic inclusion bodies
    - Animal inoculation test- The lymph or skin materials of the affected sheep inoculated on the scarified skin surface of the healthy sheep.
    - Development of the disease in the inoculated sheep – positive reaction
    - other tests- CFT, AGID, counter immuno electrophoresis

Differential diagnosis

- FMD
- Orf or contagious ecthyma

PREVENTION AND CONTROL

Ovination

- The lymph of the vesicle applied in scarified areas or the lymph is inoculated by intradermal injection on the ventral surface of the tail - oldest method of immunization.

Vaccination

- Inactivated vaccines are commonly used for vaccination.

- First vaccination is given to the sheep at the age of 3 months.
- There after annual boosters are recommended. The vaccine is administered intradermally.
- At the site of inoculation a local reaction should develop.
- All vaccinated animals should be checked after a week for positive reaction.
- If the reaction is not developed the animal should be revaccinated.

GOAT POX

SYNONYMS AND INTRODUCTION

Synonyms : Variola capra

Introduction

- It is a malignant disease of goats characterized by fever and appearance of generalized pock lesions.
- It is caused by a member of the genus Capripox virus, sub family chorodopox virinae, family Pox viridae.
- Goat pox virus, sheep pox virus and virus of bovine lumpy skin disease constitute Capri pox genus of pox viruses and they cannot be distinguished serologically.
- The disease has been reported from various parts of the world.
- The disease has been reported from different states of India.
- All breeds of goats are susceptible.
- The usual mode of transmission of this disease is by contact with the infected animal.
- The viral entry is through wound and abrasions.
- The biting insects, dogs are mechanically transmits the disease.
- Aerosol or droplet infection is quite possible.

CLINICAL MANIFESTATIONS

- Incubation period is 4-8 days after natural infection.
- Fever, formation of skin papules in the hairless regions at 2-5 days following fever.
- Papule later converted into macules.
- After papules formation rhinitis and conjunctivitis.
- Swelling of superficial lymph nodes and eye lids.
- Mucopurulent discharge from nose and eye.
- Macules convert into scabs.
- Death due to labored breathing due to bronchopneumonia.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Diagnosis

- Samples of choice - Scabs
- Based on clinical findings
- Isolation of the virus in cell cultures
- SNT, AGID

Differential diagnosis

- Contagious ecthyma or Orf
- FMD

PREVENTION AND CONTROL

- Strict sanitary measures
    - Disinfection of the goat shed with 1-3% formalin, acids, caustic soda and virucidal disinfectants
- Caprination
    - The lymph from the vesicles is to be withdrawn and to be inoculated in the underside of tail or inner surface of the ear of young goat.
    - Alternatively, the lymph may be rubbed on scarified surface of the skin.
    - This will produce mild form of the disease and will protect against subsequent virulent attack

CHAPTER-8: PESTE DES PETITS RUMINANTS

Learning objectives

To know in detail about,

- PPR virus
- Animals affected with PPR
- Clinical signs of PPR
- Diagnosis, prevention and control of PPR

SYNONYMS AND INTRODUCTION

Synonyms : Goat plague, Kata

Introduction

- Peste des petits ruminants (PPR) is a highly contagious, acute or sub acute viral disease of goats and sheep characterized by fever, erosive stomatitis, conjunctivitis, gastro enteritis and pneumonia.
- This disease was first described in 1942 in West Africa and it is closely related to rinderpest virus, canine distemper virus and human measles virus.
- Peste des petits ruminants virus (PPRV) is a single stranded, non-segmented RNA virus belonging to the genus Morbili virus, subfamily Paramyxovirinae, family Paramyxoviridae.
- It is primarily a disease of goats and sheep. Goats, particularly young ones, (4 months to 1 year of age) are usually more severely affected than sheep.
- The infection also occurs in wild ungulates.
- Cattle, buffaloes, camels and pigs are rarely susceptible. They do not exhibit clinical signs and are unable to transmit the disease to other animals.
- Cell culture systems are ideal for virus cultivation. The PPRV grows well in vero and lamb kidney cells.
- The CPE develops within 5 days and consists of cell rounding, aggregation and syncytia formation in lamb kidney cells. In Vero cells syncytia are rare and if present are very small.
- Intracytoplasmic inclusions and vacuolation of cells are also seen. Affected animal excrete the virus in all secretions and excretions.
- Transmission requires close contact between animals. Infection occurs mainly through inhalation of aerosols and by ingestion. There is no known carrier state. The spread is not dependant on vectors.

PREDISPOSING FACTORS

Predisposing factors for PPR infections are,

- History of recent movement or gathering together of sheep and/or goats of different ages
- Introduction of recently purchased animals
- Change in weather such as the onset of the rainy season or dry, cold periods
- Contact with trade or nomadic animals.

CLINICAL MANIFESTATIONS

- Incubation period is 6 days.

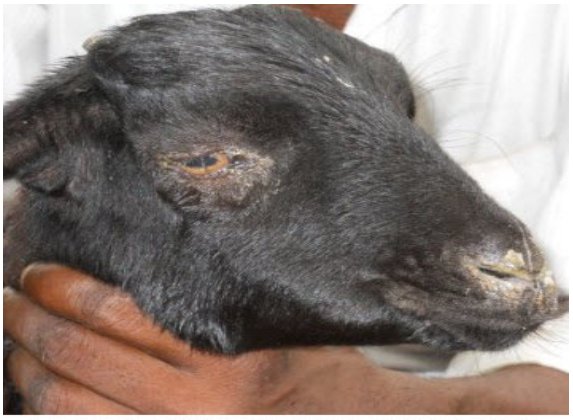
Acute form

- Fever and serous rhinorrhoea - Erosions in the mucous membranes lining the upper alimentary, upper respiratory and urogenital tracts for first 1-2 days followed by fever
- Salivation profuse, tongue is continuously protruded and retracted
- Rhinorrhea becomes mucopurulent often blocks the nostrils

PPR-Mucoprulent ocular and nasal discharge

PPR-Mucoprulent nasal discharge

- Mucosal erosions coalesce, formation of ulcers



PPR-Dried mucoprulent ocular and nasal discharge

Goat flock affected with PPR

- Diarrhoea and pneumonia.

Per acute form

- Follow incubation period that are often as short as 2 days
- Profuse nasal catarrh precedes a sudden high fever with signs of depression, dyspnoea, anorexia and constipation
- Diarrhoea, leucopaenia

DIAGNOSIS

Samples to be collected

- *Live animals:* Buccal and rectal mucosa, tears, whole blood (buffy coat), nasal secretions, faeces and gum debris.
- *Dead animals:* Oral mucosa, tonsils, lungs, small, large intestines and mesenteric lymphnodes.

Diagnosis is based on

- Classical clinical signs
- Virus isolation and identification in cell cultures
- Demonstration of viral antigen in buffy coat, body secretions, feces, lymphnode and tonsils by immunohistochemical methods, dot-ELISA, AGID and CIEP.
    - *Note:* unlike rinderpest, PPR viral antigen is still high in tissues of animals dying from the disease.
- *Serology:* Virus neutralization tests (VNT) and competitive ELISAs recommended serological tests by OIE.
- Complement Fixation Test and AGID are most commonly used. More recently cELISAs have been developed based on monoclonal antibodies specific for the N or H proteins of PPR and rinderpest viruses, and which enable differential diagnosis of the two viruses.

DIFFERENTIAL DIAGNOSIS

- Rinderpest: In Rinderpest both cattle and small ruminants are involved. The symptoms are very severe in cattle than small ruminants.
- Pasteurellosis: The disease is characterized by obvious respiratory signs, infrequent diarrhoea, and a mortality rate rarely exceeding 10 per cent. The bipolar organisms can be readily demonstrated in smears.
- CCPP: There is no digestive system involvement, and the clinical signs and lesions are confined to the respiratory system and pericardium.
- Bluetongue: Sheep are mostly affected. Swelling of the lips, muzzle, oral mucosa and coronitis are more common.

- Contagious ecthyma/orf: Proliferative necrotic lesions are seen in the lips rather than the whole oral cavity. The absence of nasal discharges and diarrhoea also distinguish orf from PPR.

PREVENTION AND CONTROL

- There is no treatment for PPR.
- Oxytetracycline and chlortetracycline are recommended to prevent secondary bacterial infections.
- Hyper immune serum which may be obtained from cattle hyper immunized against rinderpest can be used as therapy.
- Quarantine of the newly purchased animals, isolation of the affected animals, and following strict hygienic measures will helps to control the disease.

Vaccines

- Sheep or goats vaccinated with PPR vaccine at the age of 6 months and booster dose should be given once in a year.
    - The tissue culture rinderpest vaccine at a dose of $10^{2.5}$ TCID50 protects goats for atleast 12 months against PPR.
    - The vaccine is currently used in many African countries for vaccination against PPR. This vaccine is safe in pregnant goats.
    - A homologus PPR tissue culture vaccine produced by attenuation in vero cells is commercially available.
    - In southern India, a homologus PPR vaccine using AR-87 strain is used to control PPR in sheep and goats.
    - This vaccine was developed at the Department of Veterinary Microbiology, Madras Veterinary College.
    - Newly developed recombinant vaccinia or capripox viruses expressing the fusion (F) and Haemagglutinin (H) protein genes of the rinderpest virus are also effective against PPR.

CHAPTER-9: CLASSICAL SWINE FEVER, AFRICAN SWINE FEVER, SWINE VESICULAR DISEASE AND VESICULAR STOMATITIS

Learning objectives

To know in detail about

- Etiological agents of classical swin fever, African swine fever, Swine vesicular disease and Vesicular stomatitis
- Clinical signs, diagnosis, prevention and control of Classical swine fever, African swine fever, Swine vesicular disease and Vesicular stomatitis

CLASSICAL SWINE FEVER

SYNONYMS AND INTRODUCTION

Synonym

- Hog cholera, European swine fever.

Introduction

- Highly contagious viral disease of pigs of all ages, characterized by rapid and sudden onset, high mortality and morbidity with generalized hemorrhages of internal organs.
- This disease is caused by pantropic RNA virus belonging to the genus Pesti virus, family Flaviviridae.
- The virus is closely related to Bovine viral diarrhea virus.
- The disease was widespread in South African countries, Europe, China and Japan and prevalent in moderate to severe proportion in India, Myanmar, Nepal, Pakistan, Bangladesh and Philippines.
- In India this disease was first reported in West bengal in the year 1961.
- Swine are the natural host of the virus. All breeds, sex and ages of pigs are susceptible to this infection.
- Wild pigs often remain as inapparent carrier of the virus.
- Young pigs are more susceptible than the adult pigs. Infection is transmitted readily by direct and indirect contact.
- The virus is usually acquired by ingestion of food and water contaminated with discharges and secretions from infected pig.
- Urine and nasal and ocular discharges are generally regarded as most infective.

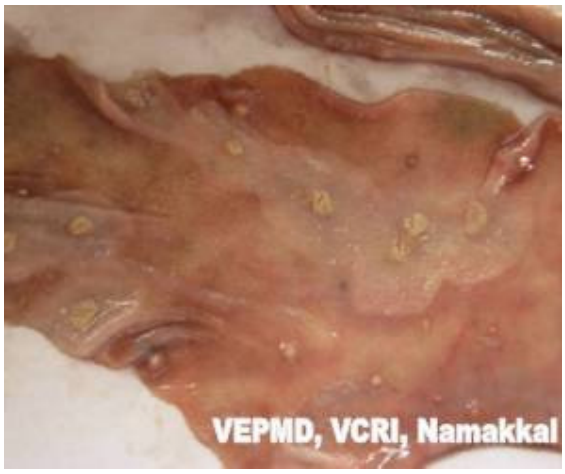
- The isolation of CSFV should be attempted in the pig kidney (PK-15) cell line, or other suitable cell lines.

CLINICAL MANIFESTATIONS

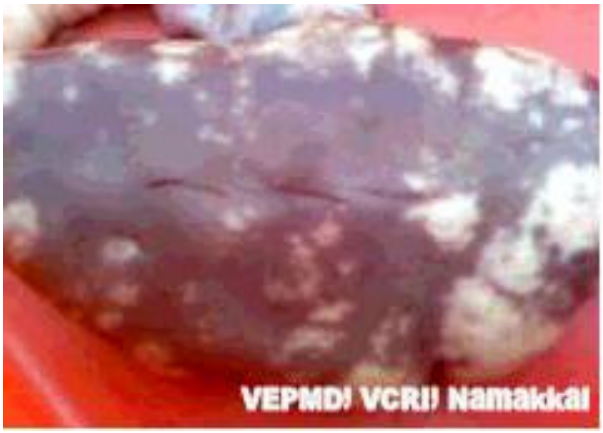
- Incubation period is 3-8 days
- The disease may appear in three clinical forms
- Per acute form
    - Most commonly noticed in young pigs
    - Disease terminates fatally within about 24 hours of developing disease
    - No appreciable clinical manifestations except high raise of temperature and erythematous patches in the non-hairy parts of the skin
- Acute form
    - Sharp rise of body temperature. Temperature reaction may persist up to 8th day or till death
    - Dullness, depression, anorexia, vomition, constipation, severe foul smelling diarrhoea, dehydration and loss of body weight
    - Hyperemia of the skin with purplish discoloration of snout, ears, abdomen, inner side of the legs, a peculiar blotching effect on the ears, small areas vesicular lesions followed by necrotic lesions on the edges of the ears, tail, lips and vulva
    - Mucopurulent to purulent discharges from eyes with signs of conjunctivitis
    - Severely affected pigs may suck urine and water due to obvious temperature reaction and dehydration
    - Central nervous system may be affected. The signs are wobbling gait, ataxia, tremor, convulsion, paralysis, circling, tremor and coma
    - Reproductive disorders in sows due to low virulent strain with signs of fever and birth of mummified, still birth, abnormal piglets
- Chronic form
    - Chronic diarrhea
    - Chronic pneumonia

Gross lesions

- Degeneration of small blood vessels leading to hemorrhages in kidney, bladder, skin and lymph nodes
- Circular or oval raised button ulcers are most prevalent in caecum and proximal portion of the colon



- Turkey egg appearance of kidney is the pathognomonic lesion of the disease.



DIAGNOSIS

Samples to be collected

- Based on characteristic symptom and lesions
- Nasal, ocular discharges, blood, spleen, lymph nodes, lungs, and liver
    - Virus isolation can be carried out in porcine cell lines using homogenates of spleen and tonsil.
    - The cultures are examined for virus growth by immunofluorescence or immunoperoxidase staining; positive isolates are further characterized by the use of monoclonal antibodies and by partial genetic sequencing.
    - Polymerase chain reaction protocols for the identification of CSFV nucleic acid have now gained international acceptance and are being used in several laboratories, both for detection of the agent and differentiation from ruminant pestiviruses.
    - Antigen-capture enzyme-linked immunosorbent assays (ELISAs) are also useful for herd screening, but should not be used on a single animal basis.

- Serological tests - VNT, ELISA

DIFFERENTIAL DIAGNOSIS

Salmonellosis

- common in pigs between 2-4 months of age but swine fever affects pigs of all age groups.
- In salmonellosis diarrhea is the leading feature.
- Bacteriological investigations are able to differentiate the salmonellosis and swine fever.
- Button ulcers of intestine is not present in salmonellosis

Swine erysipelas

- Characterized by diamond markings on the skin.
- In swine fever turkey egg appearance of the kidney is seen, but in swine erysipelas the kidney is congested and dark red in colour.
- Bacteriological examination differentiate the swine erysipelas with swine fever

Colibacillosis

- Enteritis during the first week of age, high fever and death within 48 hours are the common clinical findings in Colibacillosis.
- Smears from faeces and culture will reveal gram negative *E.coli* organism.

Purpura haemorrhagica

- Etiology of the disease is not known. It is a self-limiting disease.
- Postmortem examination will reveal subcutaneous hemorrhage extending to skeletal muscles, heart, lungs, intestine, kidneys and urinary bladder.
- Laboratory tests will rule out the possibility of swine fever.

Mulberry heart diseas

- This disease usually affects the best, healthy pigs exclusively. No fever, lesions confined to heart. Pericardium remains distended with jelly like fluids.
- Increased creatinine phosphokinase level in the serum is noticed.

Necrotic enteritis

- Predilection site caecum and colon. The lesions are superficial in nature and can be removed easily. Button ulcers are not present

Aujeszky's disease

- Widespread nervous manifestations in the young suckling pigs characterized by convulsions and prostration than the nervous manifestations in swine fever

Salt poisoning

- This is an afebrile condition which sets in suddenly and affects large number of piglets in a pen.
- Grinding of teeth, champing of jaws, frothing of mouth, blindness, head pressing, and vomition are seen. Responds to therapy with adlibdum water.

TREATMENT, PREVENTION AND CONTROL

- No specific treatment. Prohibit the feeding of uncooked garbage to pigs
- Hyperimmune serum is the only treatment which may have value in the very early stages of disease if given at the dose level of 50-150 ml per animal.
- If hyperimmune serum is given to the incontact animals it gives better protection
- Simultaneous method of vaccination : simultaneous adminstration of hyperimmune serum along with vaccination with virulent swine fever virus
- Vaccinate the pigs after six months of age with attenuated live virus vaccines or inactivated vaccines then annual revaccination to be done

SWINE VESICULAR DISEASE

INTRODUCTION

- This disease is caused by Swine vesicular disease virus (SVDV) of Entero virus genus, Family Picornaviridae.
- The SVD virus is closely related to the human enterovirus, Coxsackie B-5 virus.
- This disease was first reported in feeder pigs in Italy in 1966. It has since been reported in Great Britain (declared free in 1980), several European countries, and Asia.
- There have been reports of aseptic meningitis in humans caused by SVDV.
- Spread is by direct contact and fomites. Several outbreaks have been traced to uncooked or insufficiently cooked pork in garbage.

CLINICAL MANIFESTATIONS, DIAGNOSIS AND PREVENTION

Clinical manifestations

- Clinically indistinguishable from foot-and-mouth disease (FMD), vesicular stomatitis (VS), and vesicular exanthema (VE).
  Epithelial tissue is initially involved, followed by viraemia with generalized infection of lymphoid tissues

- The first signs - reduced feed intake, lameness and tenderness of the feet, fever up to 106°F, and the formation of vesicles on the feet, snout, tongue, mouth, nostrils, and teats.
- The prognosis is favorable, but in most countries infected animals are slaughtered.

Diagnosis

- Samples to be collected: Vesicular fluid, affected skin and mucous membranes, blood with anticoagulant, and serum.
    - Cell culture : The virus can be propagated in cell cultures of porcine kidney. Cytopathic changes typical of Picornavirus are produced in 2 - 4 days.
    - ELISA to detect viral antigen in vesicular material. Because SVD resembles clinically FMD a correct diagnosis is imperative.

Prevention

- Vaccines are not available to prevent SVD.
- SVD is a reportable disease and, if it is suspected strict measures of control including quarantine and slaughter are implemented.

AFRICAN SWINE FEVER

SYNONYMS AND INTRODUCTION

Synonyms **:** African pig disease, Wart hog disease

Introduction

- African swine fever (ASF) highly contagious, mostly acute, febrile disease of domestic swine with cyanosis of the skin, hemorrhages of the lymph nodes, kidney, and gastrointestinal mucosa.
- African swine fever virus (ASFV) belonging to Iridovirus group.
- The ASFV is immunologically different from swine fever virus.
- The pigs immunized against swine fever are susceptible to ASF.
- Domestic pigs, Wart hogs, forest hogs and bush pigs are reservoirs.
- The virus can be cultivated in developing chicken embryo and tissue culture.
- Domestic pigs get the infection when they have close contact with the wild pigs.
- Virus is transmitted via aerosol.
- The virus is abundantly present in all the secretions and excretions of the affected pigs.
- The virus is also transmitted through ticks and lice.
- Mortality is usually 100% in infected pigs. Recovered pigs are the carriers of disease.

CLINICAL SIGNS AND DIAGNOSIS

Clinical signs and diagnosis

- Incubation period is 5 to 15 days
- Common signs - Fever, depression, ocular discharge, cough, diarrhea and dehydration
- Lesions resemble hog cholera but are more severe.
- Hemorrhages are present frequently on the epicardium and endocardium, and in lymph nodes, less frequently on the kidneys and bladder.
- The spleen is enlarged, but there are much less infarcts than in Hog cholera.

Diagnosis

- Samples to be collected - blood, spleen, lungs and lymph nodes
    - By characteristic clinical signs
    - Isolation of virus in cell culture- bone marrow leucocytes cells
    - Serological tests - AGID, ELISA, Radio immuno assay

Differential diagnosis

- Must be differentiated from
    - Hog cholera
    - Erysipelas
    - Salmonellosis

PREVENTION AND CONTROL

- Prohibit the movements of infected animals
- Contact of vectors, animal attendants, Veterinarians with the diseased pigs should be restricted
- Following strict quarantine measures
- Burial of the dead animals
- Killed virus vaccines, egg adapted vaccine, Lapinized vaccine and tissue culture vaccine used for prevention.

VESICULAR STOMATITIS

SYNONYMS AND INTRODUCTION

Synonyms : Sore mouth, Pseudo foot and mouth disease, Mouth thrush, sore nose

Introduction

- It is an infectious viral disease of cattle, horse, pig characterized by vesicular lesion containing serous fluids in the mouth, foot, interdigital space, udder and teats.
- Vesicular stomatitis virus belongs to number of closely related, antigenically distinct members of the genus *Vesiculovirus,* RNA virus belonging to Rhabdo viridae family.
- Thre are three serotypes of the virus named as Indiana strain, NewJersey strain and Trinidad strain or Eocal strain.
- The strains can be separated from each other by serum neutralization and complement fixation test.
- The indiana serotype is comprised of four sub types. New Jesrsey strain is more virulent over other strains.
- This febrile disease affects mainly horses, donkeys and cattle and pigs. Other susceptible species include camels, several wildlife species and humans.
- Vesicular stomatitis is clinically similar to foot and mouth disease.
- The disease is limited to the western hemisphere and the infection is endemic in Central America and in regions of South America.
- Outbreaks of the disease occur every two to three years in tropical and subtropical regions, with clinical cases most common at the end of the rainy season and early in the dry season.
- The virus can be biologically transmitted by black flies and mechanically by Culicoides, houseflies.
- The saliva and vesicular fluid from clinically affected animals are highly infective but infectivity diminishes rapidly and may be lost within 1 week after the vesicle rupture.
- From this source, virus may be transmitted by fomites, such as contaminated feed, milking machine and restraint devices.
- Recovered cattle act as convalescent carrier for 38 days to 5 months.

CLINICAL FEATURES

- The incubation period is up to five days.
- Subclinical infection is common. Affected animals, which are usually more than one year old, become febrile
- Vesicles develop on the tongue and on oral mucous membranes, often accompanied by profuse salivation

- Secondary lesions may occur on the coronary band and teats. Lameness is often a prominent feature of the disease in pigs
- Mastitis may develop in cows with severe teat lesions. In the absence of secondary infection, lesions generally heal within two weeks.
- Following infection, animals develop high levels of neutralizing antibodies but the duration of protection is variable.

Cattle

- The incubation period is 3-15 days.
- There is a sudden appearance of mild fever and the development of vesicles on the dorsum of the tongue, dental pad, lips and the buccal mucosa.
- Development of vesicles is usually accompanied by ropy salivation. Vesicles ruptured followed by erosive necrotic lesion. Subclinical infection is common.
- Affected animals, which are usually more than one year old, become febrile.
- Secondary lesions may occur on the coronary band and teats. Mastitis may develop in cows with severe teat lesions.
- In the absence of secondary infection, recovery is rapid, affected animals are clinically normal in 3-10 days.
- Cross-protection between vesicular stomatitis Indiana virus and vesicular stomatitis New Jersey virus is limited.

Horses

- There is fever, depression and drooling of saliva.
- Affected horse may rupture their lips on troughs.
- Vesicles coalesce, rupture and formation of shallow ulcers.
- Lesions are also seen in coronary band and teats.

Pigs

- Lameness is more common.

DIAGNOSIS, TREATMENT AND CONTROL

Diagnosis

- Specimens for isolation of virus -epithelium from lesions and vesicular fluid
- Prompt laboratory confirmation is required because of similarities between vesicular stomatitis, foot-and-mouth disease and swine vesicular disease.

- If horses present with vesicular lesions, infection with vesicular stomatitis virus should be considered
- Antigen detection- CFT or ELISA
- Virus isolation in suitable cell lines, in embryonated eggs or in suckling mice by intracerebral inoculation.
- FAT, ELISA, CFT or the virus neutralization tests are suitable for identification of the isolates
- Electron microscopy can be used to identify virus in specimens or tissue culture.
- Serological tests- CFT, the virus neutralization test, competitive ELISA or IgM-specific capture ELISA.
- Because levels of complement fixing and IgM79 antibodies persist for only short periods, assays based on procedures involving these antibodies can be used to confirm recent infections in endemic areas.

Treatment and control

- Specific treatment is not available. Measures aimed at minimizing secondary infections may be beneficial.
- Movement restrictions and a 30-day quarantine period following the last clinical cases are recommended for infected premises.
- Insect-proof buildings and avoidance of habitats associated with insect vectors reduce the likelihood of infection.
- Although both inactivated and attenuated vaccines have been used, they are not commercially available.

CHAPTER-10: RIFT VALLEY FEVER

Learning objectives

To know in detail about

- Causative agent of rift valley fever
- Transmission, clinical signs, diagnosis and prevention of rift valley fever

SYNONYMS AND INTRODUCTION

Synonyms : Enzootic hepatitis

Introduction

- Rift valley fever is an acute arthropod-borne disease of sheep, cattle and goats, causing high mortality among young lambs, calves and kids and abortion in pregnant females.
- Many species can be infected by Rift Valley fever virus (RVFV), including humans. Horses and pigs are resistant to this disease.
- This disease found throughout Africa. Recent outbreaks have occurred in Saudi Arabia and Yemen considerable humans have succumbed.
- This disease is caused by single stranded RNA virus belonging to the genus Phlebovirus, family Bunyaviridae.
- Rift Valley fever occurs in Africa and the Middle East in sheep, goats, cattle, camels, antelopes and humans.
- This disease is transmitted by mosquitoes (*Aedes spp.*), by contact and also by aerosol.

CLINICAL MANIFESTATIONS

- The incubation period is up to 3 days in sheep and goats and in newborn animals, it can be as short as 12 hours
- Infection is most severe in young animals, and is characterized by a high fever, anorexia, weakness, and rapid death.
- Some affected animals may have nasal discharge and hemorrhagic diarrhea
- Hepatic necrosis is the primary lesion, and is especially extensive in younger animals and fetuses
- Adult animals are less severely affected. Abortion is the most prominent sign in pregnant animals (up to 100%).
- In adult animals, hepatic necrosis can be more focal and may only be visible microscopically

- Mortality rate may exceed 70% in young animals but is considerably less in adults
- Humans may become infected by mosquitoes and through contact with diseased tissues.
- Infections are "flu-like", and can infrequently be severe and fatal
- Cattle are less severely affected than sheep

DIAGNOSIS

Specimens: Liver and spleen.

- A presumptive diagnosis is made on the basis of clinical signs and gross and microscopic lesions observed in the liver.
- Confirmation requires isolation and identification of the virus.
- The virus replicates on the chorioallantoic membrane of chicken embryos and in various cell cultures including vero, CER, BHK 21, mosquito line cells, primary calf, lamb and goat kidney or testis cells or in suckling and weaned mice or hamsters inoculated intracerebrally or intraperitoneally.
- Cytopathic effect may be evident one to five days after inoculation of cell cultures and the virus identified by FAT.
- Serological tests- by CFT, ELISA, FAT, HI, RPHA, IPT.

Differential diagnosis

- Agents or poisoning caused by poisonous plants such as S*eneciao sp, Crotalaria sp, Lasiospermum*, pasteurellosis, salmonellosis, anthrax, RP, and PPR.

PREVENTION

- Modified live virus and killed virus vaccines are used in countries where the disease is endemic.
- The modified live vaccine should not be used in pregnant animals.
- Mosquito control reduces the chances of infection.
- In countries where the disease does not occur, outbreaks are dealt with by strict quarantine and slaughter.

CHAPTER-11: AUJESKY'S DISEASE AND RABIES

Learning objectives

To know in detail about

- Causative agent of Pseudorabies and rabies
- Different clinical manifestation of pseudorabies and rabies
- Diagnosis of pseudorabies and rabies
- Prophylactic and postexposure vaccination schedule for rabies
- Control of pseudorabies and rabies.

AUJESKY'S DISEASE

SYNONYMS AND INTRODUCTION

Synonyms : Aujeszky's disease, Mad itch, Infectious bulbar paralysis

Introduction

- This is an acute viral infectious disease that primarily affects pigs but may occur in other species of animals.
- This disease is caused by the DNA virus belongs to the *alpha-herpesvirinae* subfamily, family Herpes viridae and is distributed worldwide.
- It infects a wide range of natural hosts including mammals and birds.
- Rodents and pigs are the primary hosts of the virus.
- This disease is reported world-wide except in few countries including Australia, Canada and Norway.
- The disease is prevalent in cattle, sheep, goat, pig, horses, dog, cat and mink.
- Pigs are considered as reservoir host for this virus. The disease is prevalent in pigs all round the year.
- Pigs may remain as asymptomatic immune carrier and thus may act as a source of infection to other species of animals.
- The natural way of entry of virus is through a breach in the continuity of skin or integument due to injury, bite or abrasions.
- Virus may enter through inhalation as aerosol way of transmission.
- The virus can be grown in susceptible cell line such as porcine kidney (PK-15) or SK6, or primary or secondary kidney cells.

CLINICAL MANIFESTATIONS

Cattle

- Incubation period- 3-6 days.
- Pyrexia along with pruritus starts at the site of entry of virus. Pruritus mostly observed in the nose, eyes, ears, lower jaw, chest or in the udder, limbs, flanks and anal region.
- Involvement of central nervous system- tremor, continuous or intermittent chewing movements of jaws, profuse salivation and sweating, excitement, bellowing, roll up and down, stumping on the ground, aimless staggering, aggressiveness and circling.

Pigs

- All ages may be affected but very young pigs are more susceptible than older ones
- Changes in the respiratory, nervous and reproductive systems
- High rise of temperature, with development of nervous signs
- Incoordination develops in the posterior limbs which force the animal to move sideways
- Muscle tremors and paddling movements, tilting of head, froth at the commissure of mouth, nystagmus, ocular discharge and convulsion
- Vomition, diarrhea, signs of blindness

Sheep, dog, goat, cat and horses

- Similar to that of cattle
- In dogs and cats – fatal disease where death is eminent within 2 days following manifestation of clinical signs

Lesions

- Congestion of meninges, brain, haemorrhage, odema and necrosis of lungs, focal necrosis of liver

DIAGNOSIS AND DIFFERENTIAL DIAGONSIS

Diagnosis

- Samples to be collected- Brain and tonsil, oro-pharyngeal fluid, nasal fluid (swabs)
- Based on clinical signs
- Identification of the agent - Isolation of Aujeszky's disease virus can be made by inoculating a tissue homogenate, for example of brain and tonsil or material collected from the nose/throat, into a susceptible cell line such as porcine kidney (PK-15) or SK6, or primary or secondary kidney cells.

- The specificity of the cytopathic effect is verified by immunofluorescence, immunoperoxidase or neutralisation with specific antiserum.
- The viral DNA can also be identified using PCR; this can be accomplished using the real-time PCR techniques.
- Serological tests- Aujeszky's disease antibodies are demonstrated by virus neutralisation, latex agglutination or enzyme-linked immunosorbent assay (ELISA).

Differential diagnosis

- Rabies- No pruritus, biting tendency, dropped jaw, ascending paralysis, demonstration of negri bodies in the neurons of hippocampus and purkinje cells of the cerebellum
- Listeriosis- History of intake of silage, Facial paralysis
- Polyencephalomalacia- History of carbohydrate engorgement, no pyrexia
- Poisoning- Specific signs depending on the source of poison. Gastric content analysis to pin-point the toxin

PREVENTION AND CONTROL

- No treatment.
- Adminstration of hyperimmune serum @ 5ml per piglet by subcutaneous route.
- Quarantine for a period of three weeks for newly purchased pigs and test the animals before mingling with other pigs and isolation of pigs from the other susceptible animals.
- Disinfectants such as 3 percent formaldehyde or chlorine releasing disinfectants (3 percent chloramine solution).
- Due to low incidence of disease no vaccines are available in India.
- Killed vaccine adjuvanted with aluminium hydroxide passaged in egg yolk administered to reduce the prevalence of disease.

RABIES

SYNONYMS AND INTRODUCTION

Synonyms : Hydrophobia, Lyssa, Mad dog disease

Introduction

- Rabies is acute viral encephalitis of all warm-blooded animals characterized by altered behaviour, aggressiveness, progressive paralysis and in most species by death.
- It is an important disease of man and animals, which is usually transmitted through the bite of rabies animals to other animals or human result in rapid fatal encephalomyelitis after a somewhat lengthy incubation period.
- The disease in man is called hydrophobia because the patient exhibits fear of water, being incapable of drinking though subject to intolerable thirst.

- Rabies in animals is not called hydrophobia because they do not have this peculiar feature.
- Fox, wolf, coyotes and jackal are extremely susceptible. Guinea pig, Hamster, Bat, Mongoose, mice, rabbit, skunk and cattle are highly susceptible. Dog, sheep, goat, horse and human are moderately susceptible. Poultry and opossum are resistant.
- At the present time rabies occurs in most parts of the world except in Japan, UK, New Zealand, Antarctica, Australia, Hawaii islands and Switzerland. In India the incidence is very high.
- Rabies virus belongs to the Rhabdo virus group, one of the RNA helical enveloped virus group. It appears as filamentous, bell shaped or bullet shaped and approximately 60-175 nm in size. The virion is rounded at one end and flat or truncated at the other.
- It is surrounded by a fringe of short pointed projections. Rabies virus can agglutinate RBCs of day old chicks and goose at 0-4^{0}C and pH 6.2.The virus is deficient in neuraminidase activity.
- The nucleic acid consists of single stranded RNA. It is found in nerve tissue, saliva, salivary glands, pancreas, less often in urine and lymph and rarely in blood, milk and other body fluids of infected animals.
- It is readily inactivated by sunlight, drying in air, 40-70% alcohol, quaternary ammonium compounds, tincture-iodine, carbolic acid and any lipid solvents.
- The virus is very resistant to autolysis and putrefaction. The virus will persist for months in infected nervous tissue in 50% glycerol. The virus survives at 4^{0}C for weeks. It can be preserved at –70^{0}C or by lyophilization.
- Rabies virus grows very well in human diploid cell line WI-38 and MRC-5, BHK21, Vero and Chicken embryo fibroblast.
- Cytopathic effects are not apparent. In continuous lines of human diploid cells and BHK21 infected with rabies, almost 100% of the cells are infected and there is a marked CPE. Five or six day's old embryonated hen's eggs are preferred for growth of virus by yolk sac route.
- The virus also grows in CAM route and produce miliary pock lesions. The strains that have been adapted in egg are Flurry strain, Keller strain. Low egg passage (LEP) strains can be obtained by 40 to 50 passages. High egg passage (HEP) strains can be obtained by 180 passages. Duck eggs are commonly used for preparation of vaccine.
- Rabies can be cultivated in a wide range of laboratory animals such as hamsters, mice, guinea pig and rabbits in the order of decreasing susceptibility by intracerebral or intramuscular routes.
- In usual circumstances the only risk of rabies virus transmission is by the bite or scratch of a rabid animal, although in bat caves, where the amount of virus may be very high and the extremely high humidity may stabilize the virus transmitted by aerosol infection.
- In rare circumstances the virus can pass through the intact mucous membrane
- The amount of virus required and to initiate infection varies considerably and depends upon the susceptibility of the patients.
- Susceptible wild living animals particularly foxes, wolf, jackals constitute the most important residual focus of rabies infection the most prevalent and most difficult to control. Because they carry 10^6 infectious units of virus/ml of saliva.

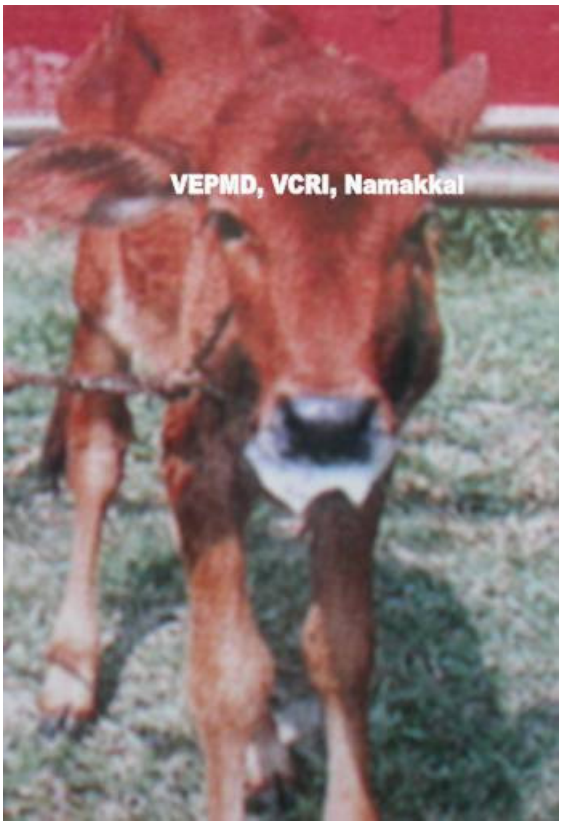
- Domestic dog is still the most important source of infection. Although virus titers in the saliva of confirmed case of rabies in dog varies widely.
- The saliva is believed to be infective in only 50-60% of the rabid dogs at the time of bite and the presence of virus is greatly evident in a few days before death.
- The factors, which influence the course of infection, include the virulence, invasiveness and concentration of inoculated virus, the amount of nervous tissue near the site of wound, the degree of trauma and the type of animal inflicting the bite.
- Blood licking bats in chiroptera family constitute an important reservoir in central and South Africa and they frequently infect cattle, man by biting at nights.
- In a report from Ethiopia in 1964, the isolation of true rabies virus was obtained from the saliva of dog, which remains clinically healthy 4 years after the sample had been taken.

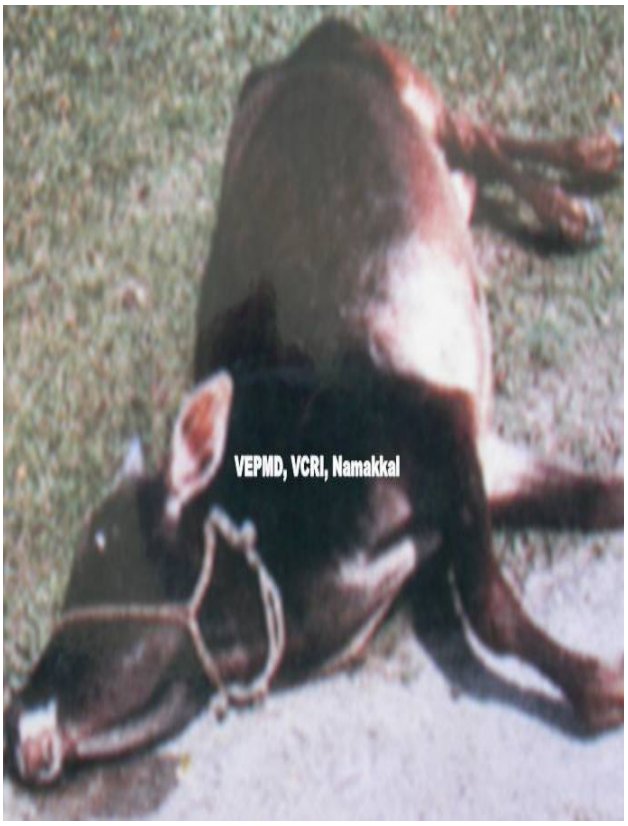
CLINICAL MANIFESTATIONS

- The incubation period in natural outbreak of dog rabies averages from 3-8 weeks. But it may be as short as 10 days to as long as one year.
- The clinical features divided into three phases.
    - *Prodromal form :* During the prodromal period, which lasts 1-3 days, animals show only vague CNS signs, which intensify rapidly. Dog has a change in the temperament aimless snapping and barking at imaginary objects.
    - *Furious form :* By about 3rd day after the onset of illness the dog enters the furious stage which lasts for 3-7 days. During this stage dog becomes irritable, restless, nervous, deprived appetite, aggressive, and often dangerous as it loses all fear of humans and bites at anything that gains its attention. There is usually exaggerated response to light and sound. Dog exhibit characteristic change in its barking and may howl in an unusual tone due to the paralysis of laryngeal muscle. Salivation and frothing at the mouth becomes progressively more profound. Terminally, there are often convulsive seizures, coma and respiratory arrest, with death occurring 2 to 14 days after the onset of clinical signs. If it does not die it passes into paralytic stage.
    - *Dumb or paralytic form :* In cases when the furious phase is extremely short or absent the animal rapidly enters the paralytic or dumb stage; where the dog is rarely irritable and seldom bites. Paralysis of the throat and masseter muscles, dropping of the lower jaw or lower jaw paralysis is the clinical signs common in dogs. In dumb form animals are not vicious and rarely attempt to bite. The paralysis progresses rapidly to all parts of the body, and coma and death follow in a few hours.
- Note : Veterinarians/ Owners frequently examine the mouth of dogs and livestock searching for foreign body or administer medication with their bare hands, thereby exposing themselves to rabies.
- In cats the clinical signs are similar to those of dogs but lost for 2-4 days before death occurs.

Cattle

- Among farm animals, cattle are most commonly affected. In the paralytic form, knuckling of the hind fetlocks, sagging and swaying of the hindquarters while walking, often deviation or flaccidity of the tail to one side, are common signs.
- Decreased sensation over the hindquarters is one of the best criterions for the detection of rabies.
- There is drooling of saliva, tenesmus, pumping of anus and followed by recumbency seen in later stages.
- In furious rabies, the animal alert, hypersensitive, violently attack, loud and coarse bellowing, sexual excitement and collapses suddenly.
- Cattle are very restless, excited and aggressive with salivation, abdominal pain, diarrhoea and rectal straining. Paralysis of hind quarters occurs followed by death in 3-6 days after the first signs of illness.
- In sheep and goat the symptoms are similar to cattle.





Sheep

- Clinically the picture is similar to cattle. Sexual excitement, violent attack, vigorous wool pulling, sudden falling and salivation are characteristic.
- Goats are commonly aggressive, and continuous bleating is common.

Horse

- Muzzle tremors and pharyngeal paresis are common.
- In addition to these abnormal postures, frequent whinnying, kicking, biting, colic, sudden onset of lameness in one limb followed by recumbency, high stepping gait, blindness, recumbency, paddling, convulsions and terminally paralysis.

Pigs

- Tendency to attack, twitching of the nose, rapid chewing movements, excessive salivation, walk backward and terminally paralysis.

SAMPLE TO BE COLLECTED

- Live animals : Saliva, corneal/ Conjunctival smear.
- Dead animals
    - The whole carcass or the severed head of the animal suspected to have died of rabies.
    - Alternatively, the brain may be removed carefully and two portions, one in 50% glycerol saline and the other in Zenker's fixative, sent for biological test and microscopy, respectively.
    - The brain tissue selected should include portions of hippocampus, cerebellum, cerebral cortex, and placed in 50% glycerol saline to preserve the virus.
    - No refrigeration is required.
    - Glycerinated tissues are generally unsuitable for immunofluorescence staining for which smears fixed in acetone free methyl alcohol and dried at room temperature without blotting should be sent.

DEMONSTRATION OF NEGRIBODIES

- Negribodies are abundantly present in pyramidal cells of ammon's horn and purkinje cells of cerebellum.
- They can be demonstrated in impression smears either by Seller's method (for unfixed sections) or Mann's method (fixed sections).
- Seller's method has the advantage that fixation and staining is done simultaneously.
- Negribodies are intracytoplasmic, magenta red or cherry red spherical or oval bodies with characteristic basophilic inner granules.
- They are varying in size from 3-27µ. They are inclusions of aggregates of nucleocapsids and they are specific to rabies infection.
- Note: Animal should not be killed for the demonstration of negri bodies. They can be demonstrated in only 80-85% cases.
- It should be differentiated from intranuclear inclusions produced by Infectious Canine Hepatitis and Canine Distemper.

ISOLATION AND IDENTIFICATION OF VIRUS

- Confirmatory diagnosis must be obtained by animal inoculation or other means.
- Animal inoculation: For isolation of virus, the brain or other tissue specimens are prepared as 10% suspension.
- Suckling mice or hamsters of 3-6 weeks age are generally used for isolation and identification.
- Route of inoculation is intracerebral. Infected mice almost always develop clinical symptoms within 17 days of inoculation.
- The clinical signs to look far in mice are ruffled fur, arching of back, flaccid paralysis of legs.
- To establish a positive diagnosis the mouse brain are removed and examined microscopically for negribodies.
- The confirmatory diagnosis can be obtained by serum neutralization test in mice or by lmmunofluorescence.
- Rabies virus can be cultured in human diploid cells, neuroblastoma and BHK21. A 5 or 6 day old embryonated hen's eggs are preferred for growth of virus by yolk sac route.

DEMONSTRATION OF VIRUS ANTIGEN

- Demonstration of viral antigen in ammon's horn, cerebellum, corneal/conjunctival impression smear, skin biopsy (from facial area) and in sub maxillary salivary glands by fluorescent antibody technique (FAT) is the most widely used, confirmatory test for the diagnosis of rabies since it provides rapid reliable and highly specific means of detecting the viral antigen.
- This test is recommended by both WHO and OIE.
- By serology
    - Immunoperoxidase test
    - ELISA
    - Rapid fluorescence focus inhibition test (RFFIT)
    - Virus neutralization test
    - Rapid rabies enzyme immuno diagnosis test (RREID) based on the use of antinucleocapsid lgG are highly useful
    - RT-PCR amplification technique is 1000 times more sensitive than other tests.

TREATMENT

- There is no specific treatment for rabies.
- Dogs usually die after showing clinical signs.
- The site of bite should be washed with water or soap.
- Alkali prevents multiplication of virus. Sodium bicarbonate or caustic soda may be used.

- Then the wound is treated with 2% quaternary ammonium compound or tincture iodine or 40-70% alcohol.
- Wound may also be cauterized with carbolic acid or nitric acid.
- Antirabies serum may be applied topically or infiltrated around the wound.
- Treatment with antirabies serum is also effective.
- Antirabies serum is prepared by hyperimmunisation of horses.
- It should be given as early as possible after exposure and in any case within five days, after which it may not be beneficial.
- The dose recommended is 40 I.U. per kg body weight.

PROPHYLAXIS

Nervous tissue vaccine

- This is a flurry type vaccine. These are prepared from brain tissues of sheep inactivated through 1% phenol. These are made as *5%* and 20% suspension.
    - *Pasteur's cord vaccine:* this vaccine is prepared by drying over caustic potash for varying period's pieces of infected rabbit spinal cord.
    - *Semple vaccine :* This vaccine was developed by Semple (1911) at the Central Research Institute, Kasauli. It is a 5% or 20% suspension of sheep brain infected with fixed virus and inactivated with phenol at 37^0C.
    - *Beta Propinolactone vaccine :* This is a modification of Semple vaccine in which BPL is used as the inactivating agent instead of phenol.
    - *UV treated vaccines (Webster) :* Inactivated vaccines prepared from brain of infected animals, which were exposed to UV rays.

Non - nervous tissue vaccine

- *Duck egg vaccin :* This is fixed virus adapted for growth in duck eggs and inactivated with BPL. This has been discontinued because of its poor immunogenicity.
- *Live attenuated chick embryo vaccine: Two* types of vaccines are available.
    - *Avianized or chick embryo vaccine*
        - This is made through passaging the virus in chicken embryo. Low egg passage (LEP) (40 to 50 passages) is completely safe for dogs.
        - Avianized live virus vaccine is a single dose vaccine. Age of first vaccination is 3 months, dose 3 ml i/m. Immunity 3 years. Repeat after 3 years.
        - High Egg Passage (HEP) vaccine at 180 passage level used for cattle and cats.
    - *Tissue culture vaccine*
        - The first cell culture vaccine was the human diploid cell strain (HDCS) vaccine developed by Koprowsky, Wiktor and Plotkin.

- It is a purified and concentrated preparation of fixed rabies virus (Pitman – Moore Strain) grown on human diploid cells (WI 38 or MRC 5) and inactivated with betapropinolactone. It is highly antigenic and free from side effects.
- Its only disadvantage is high cost. Nowadays more economical, primary chicken embryo and vero cell culture adapted, vaccines are available.
- The avianized live virus vaccine is a single dose vaccine. Age of first vaccination is 3 months, dose 3 ml i/m. Immunity 3 years. Repeat after 3 years.
- In tissue culture vaccine, age of first vaccination is 3 months.
- Revaccination should be done after 6 months and then at yearly intervals. Dose 5 ml s/c in the neck or flank region.

- *Subunit vaccine*
  - The glycoprotein subunit on the virus surface, which is the protective antigen has been cloned and recombinant vaccines produced. They are still in the experimental stage.
- *Pre exposure vaccination-* Pre exposure vaccination schedule for dogs starts at 12th week age – first dose, 28 days later – second dose, one year later- third dose, three years later- fourth dose.
- *Post exposure immunization:* 0, 3, 7, 28, 60, 90 days after exposure.

Control (as per the WHO recommendations)

- Notification of suspected cases, and destruction of dogs with clinical signs and dogs bitten by a suspected rabid animal.
- Compulsory immunization of dogs
- Sterilization and vaccination of stray dogs by using baits
- Epidemiological Surveillance
- Education of Public
- Development of cost effective vaccine

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CHAPTER-12: AFRICAN HORSE SICKNESS, EQUINE INFLUENZA, EQUINE INFECTIOUS ANAEMIA AND EQUINE RHINOPNEUMONITIS

Learning objectives

To know in detail about

- Causative agent of equine influenza, equine infectious anaemia and african horse sickness
- Transmission, clinical signs, diagnosis and prevention equine influenza, equine infectious anaemia and african horse sickness.

AFRICAN HORSE SICKNESS

INTRODUCTION

- African Horse Sickness (AHS) is an acute or sub acute, febrile, arthropod borne disease of horses characterized by oedema of subcutaneous tissues, lungs, hemorrhages of internal organs and accumulation of serous fluids in the body cavities.
- This disease is caused by orbi virus belonging to the family Reo viridae which is a double stranded RNA virus.
- Horses of all breeds are highly susceptible to natural infection.
- Mules and zebras are less Affected and donkeys are generally resistant. The disease occurs principally in Southern, Eastern and Central Africa.
- From there it spreads to Syria, Lebanon, Israel and other parts of Middle East.
- It has been recorded in Afghanistan, West Pakistan, Turkey and Iraq.
- It is a seasonal disease, common in late summer in warm, humid, low lying marshy districts. This disease is not directly contagious.
- Various nocturnal biting insects including mosquitoes are the most important vectors of infection in Africa.
- Culicoides species of mosquito and insects belonging to Tabanidae family and genus stomoxys have been incriminated in outbreaks in Turkey.
- Many serotypes have been described. In Africa 9 distinct serotypes are recognized and considerable variation in virulence amongst individual strains within each immunological type occurs.
- On the other hand only a single antigenic type involved in the Asian outbreak in 1961. Almost all strains grow in the in the yolk Sac of fertile eggs.
- Virus can be propagated in cell cultures of many different types. Monkey cell lines are most susceptible.

- In Rhesus kidney cultures the characteristic CPE is rounding and shrinkage of infected cells which remain attached to the glass.

CLINICAL MANIFESTATIONS

- Incubation period is 6-9 days. 4 different clinical forms of illness were described.
    - *Very mild form* - only rise in temperature to 41°C.
    - *Acute or pulmonary form (DUNKOP)* in this there is severe dyspnoea, pyrexia and coughing with abundant frothy discharge from the nostrils. This form is common in virulent outbreaks and mostly the affected horses die.
    - *Sub acute form (DIKKOP).* It is characterized by remarkable swelling of head, neck and supraorbital fossa associated with cardiac dyspnoea. This form usually occurs when the immunity has been broken down by a natural infection or in animals inoculated with mild strains of virus. This form is much milder than the acute form and the affected animals recover.
    - *Mixed form :* Combination of pulmonary form and cardiac form. Heart is affected.
- Mortality : In virulent form it is as high as 90% in horses and lower in mules. In milder outbreaks mortality is 25%.

LESIONS AND DIAGNOSIS

Lesions

- Varies depending upon the severity of the case.
- In acute pulmonary form - extensive oedema of lungs and thorax may contain several litres of fluid.
- In more chronic cardiac form there is oedematous infiltration of subcutaneous tissue, sub-serosal tissue and other tissues together with gross accumulation of fluid in the pericardial sac.

Diagnosis

- Samples to be collected
    - *Live animals:* Blood from affected animal should be collected during early febrile phase
    - *Dead animals:* Spleen
- Based on clinical signs and by post mortem findings.
- For virus isolation, the tissues of choice for diagnosis are spleen, lung, and lymph nodes, collected at necropsy.
- Sample preparations can be inoculated in cell cultures, such as baby hamster kidney-21 (BHK-21), monkey stable (MS) or African green monkey kidney (Vero), intravenously in embryonated eggs, and intracerebrally in newborn mice.

- Several enzyme-linked immunosorbent assays (ELISAs) for the rapid detection of African horse sickness virus (AHSV) antigen in spleen tissues and supernatant from infected cells have been developed.
- Identification of AHSV RNA has also been achieved using a reverse-transcription polymerase chain reaction method.
- Virus isolates can be serotyped by a type-specific serological test such as virus neutralisation (VN) and by reverse-transcription polymerase chain reaction and sequencing.
- Serological tests-complement fixation test, ELISA, immunoblotting and VN.

CONTROL

- Vector control
- Quarantine of affected animals
- Vaccination
- Recently freeze dried live attenuated neurotropic mouse brain virus vaccine has been proved useful and polyvalent vaccines from 7 or 8 antigenically distinct serotypes appear to be safe and highly effective against all field strains.
- Regular annual immunization is advocated.
- Tissue culture adapted virus, Hamster or Monkey kidney cell culture vaccines are widely used in Africa and Middle East.

EQUINE INFLUENZA

SYNONYMS AND INTRODUCTION

Synonyms : Equine distemper, Typhoid fever, pink eye

Introduction

- Equine influenza is an acute febrile highly infectious disease of horses characterized by general septicemia, respiratory problem accompanied by severe persistent dry cough.
- This is an economically important disease occurs worldwide except in Australia, New Zealand and Iceland.
- Two immunologically distinct subtypes of influenza “A” virus are described in horses.
- The virus, first isolated from horses in 1956, was designated A/equine/Prague/l/56 (H7N7) or influenza A/equine 1.
- In 1963, a second subtype was isolated in the USA and designated A/equine/Miami/2/63 (H3N8) or influenza A/equine 2.
- Infection or vaccination with one subtype does not induce protection against infection with the other subtype.

- Although the last outbreak of disease attributed to influenza A/equine I occurred in 1979, there is serological evidence that this subtype continues to circulate in the horse population.
- Antigenic drift accounts for several variants of influenza A/equine 2 with two antigenically and genetically distinct lineages identified in Europe and the Americas.
- In contrast, the H3N8 subtype isolated from horses in China was more closely related to avian strains than to the H3N8 subtype circulating in horses elsewhere.
- Outbreaks are associated with movement and assembly of horses for shows, sales, racing or training.
- The initial source of infection is often a partially immune horse shedding virus without showing clinical signs.
- Equine influenza is highly contagious disease and spreads rapidly among susceptible horses.
- Large quantities of virus are shed in aerosols by the frequent coughing of affected animals.
- Infection can be acquired at distances up to 30 metres. Indirect transmission through contamination of clothing, equipment and vehicles can also occur.

CLINICAL MANIFESTATIONS

- The incubation period is up to two days.
- High temperature, nasal discharge and dry cough. Anorexia and depression, although common, can vary in intensity.
- Ocular discharge, limb oedema and stiffness.
- Age and previous exposure or vaccination status may influence the severity of the clinical signs and the likelihood of secondary bacterial infection with the development of respiratory complications.
- Exercise exacerbates the clinical signs. Animals with mild infections usually recover within three weeks. In severe cases, several months may be required for convalescence.

DIAGNOSIS

Samples to be collected

- *Live animals :* Nasopharyngeal swabs collected during the acute phase of the infection are suitable for isolation of the virus in embryonated eggs or in cell culture.

Identification of the agent

- Embryonated hen's eggs and/or cell cultures can be used for virus isolation from nasopharyngeal swabs or nasal and tracheal washes.
- Viral growth is monitored by haemagglutination (HA) or, in cell cultures, by haemadsorption (HAD) using chicken or guinea-pig red blood cells. Isolates can be characterized by haemagglutination inhibition (HI) using strain specific antisera.

- Isolates should always be sent immediately to an International Reference Laboratory (OIE or World Health Organization).
- Samples that yield negative results should be repassaged up to five passages may be necessary to isolate viruses from vaccinated horses.
- Infection may also be demonstrated by detection of viral antigen in respiratory secretions using an enzyme-linked immunosorbent assay.

Serological tests

- Diagnosis of influenza virus infections is usually only accomplished by tests on paired sera; the first sample should be taken as soon as possible after the onset of clinical signs and the second approximately 2 weeks later.
- Antibody levels are determined by HI or single radial haemolysis (SRH).

TREATMENT AND CONTROL

- Supportive therapy and rest is indicated for affected horses. Several inactivated vaccines are commercially available.
- However, immunity is usually short-lived and booster injections are required in accordance with manufacturer's instructions.
- The incorporation of polymer adjuvants or Quil-A based immune-stimulating complexes (ISCOMs) into vaccine preparations extends the duration of protective levels of immunity.
- Vaccinated horses generally exhibit milder clinical signs and shed virus for shorter periods than unvaccinated animals.
- Vaccine manufacturers must update vaccinal strains regularly.
- Vaccines should include antigenic material representative of the influenza “A” virus subtypes prevalent in the horse population.
- In addition to vaccination, control of equine influenza requires isolation of affected horses and cleaning, disinfection and isolation of infected premises.
- Animal movement should cease until contaminated premises have been cleaned and disinfected.

EQUINE INFECTIOUS ANAEMIA

SYNONYMS AND INTRODUCTION

Synonyms : Swamp fever

Introduction

- It is a chronic persisting infectious viral disease of equines characterized by emaciation, anaemia, intermittent fever and generalized lymphoproliferative changes and odema.

- The anaemia may be for few days or may persist for a longer period and this is considered to be immunologically induced problem.
- The etiological agent for this disease is a RNA virus belonging to Lenti virinae, subfamily Retroviridae family.
- The virus can be cultivated in tissue culture and the virus can be killed by exposing them to heat and commonly available detergents.
- The members of the Equidae family are the principle hosts for this disease.
- This disease is transmitted through blood sucking arthropods such as Tabanus, Stomaxys and mosquitoes.
- Fly breeding season actually augment the spread of the disease.
- It is also transmitted by iatrogenic transmission.

CLINICAL MANIFESTATIONS AND LESIONS

Clinical manifestations

- Long incubation period- 1-4 weeks
- Varied clinical manifestation- Anorexia, pyrexia, depression, odema of dependent parts
- Icterus, wasting, anaemia

Lesions

- Entire carcass – subcutaneous odema, submucosal and subserous hemorrhages
- Generalized lymphadenopathy
- Hepatomegaly, spleenomegaly

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Diagnosis

- Based on history and clinical signs
- Laboratory tests- Coggin's agar gel immunodiffusion test, ELISA-Alternative test for AGID

Differential diagnosis

- Should be differentiated with other diseases of horses including Babesisois, trypanosomiasis where anaemia is the important clinical picture.
- The above diseases ruled out with peripheral blood smear examination, AGID and ELISA
- Viral arteritis –have clinical resemblance but can be differentiated be looking at the ocular changes characterized by palpebral odema, photophobia and lacrymation

PREVENTION AND CONTROL

- Limit the movements of carrier animals
- Suspected animals should be isolated and identified by a serological test
- Disease is mostly prevalent in swampy areas where the vectors are abundant, so such areas should be avoided for equine rearing
- Strict vector control measures
- Vaccination with different subtypes of the virus prevalent in the particular area

EQUINE RHINOPNEUMONITIS

INTRODUCTION, ETIOLOGY, DISTRIBUTION

- Equine viral rhinopneumonitis (EVR) produces an acute respiratory catarrh, which is inflammation due to excessive discharge or buildup of mucus in the throat and nose.

Etiology

- Equine viral rhinopneumonitis is caused by Equine Herpes Virus (EHV).
- Most significant and important are EHV-1 and EHV-4, they are 2 antigenically distinct groups of viruses previously known as subtypes 1 and 2 of EHV-1.

Distribution

- Both of these viruses can be found commonly in horse populations worldwide.
- The natural reservoir of both EHV-1 and EHV-4 is the horse, latent infections and carrier status occur with both types of the virus.

HOST RANGE AND TRANSMISSION

Host

- In areas with substantial horse populations there are annual outbreaks amongst foals, the intensity of the infections vary and are dependant on the age, seasonal and geographical distributions as well as the immune status and horse population.
- In individual horses, the outcome of exposure to the virus is determined by viral strain, immune status, pregnancy status and possibly age.

Transmission

- Transmission of the virus occurs by both direct and indirect contact with infectious nasal fluid, aborted fetus, placentas or the fluids thereof.

CLINICAL MANIFESTATIONS

- The incubation period of EHV is 2-10 days with the first classic symptom being a temperature of 38.9-41.7° C
- Clear fluid nasal discharge, a vague feeling of bodily discomfort, pharyngitis, cough, inappetence and/or swelling of the lymph nodes on the lower and back jaw.
- A secondary bacterial infection is common which is characterised by a thick pus-like nasal discharge, this often causes pulmonary disease.
- Mares that, abort with EHV-1, do so with a sudden, unheralded event, the foetus is fresh or slightly decomposed, and the placenta is expelled shortly afterwards. Abortions occur 2 to 12 weeks after EHV-1 infection, during the period 7 to 11 months of pregnancy, the cause of death to the foetal foal is characteristically damage to the liver, lungs and other organs.
- Certain strains of EHV-1 cause neurological disease. Symptoms present from as slight incoordination with a little lameness in the back legs to severe paralysis of the hind limbs with the horse lying down, loss of bladder and tail function with loss of sensation to the skin in area around the anus and genitals.

DIAGNOSIS

- Equine viral rhinopneumonitis cannot be clinically differentiated from equine influenza, equine viral arteritis, or other equine respiratory infections solely on the basis of clinical signs.
- Definitive diagnosis is determined by virus isolation from samples obtained via nasopharyngeal swab and citrated blood sample (buffy coat) early in the course of the infection and by serologic testing of acute and convalescent sera.
- In cases of suspected EHV-1 abortion, a diagnosis is based on characteristic gross and microscopic lesions in the aborted fetus, virus isolation, and demonstration of viral antigen in fetal tissues. Lung, liver, adrenal, and lymphoreticular tissues are productive sources of virus.
- Serologic testing of mares after abortion has little diagnostic value. Diagnosis of herpesvirus myeloencephalopathy depends on demonstration of characteristic vascular lesions in sections of CNS tissue of horses that die or are destroyed.

TREATMENT

- There is no specific treatment for EHV infection.
- Rest and nursing care are indicated to minimize secondary bacterial complications.
- Antipyretics are recommended for horses with a fever >104°F (40°C).
- Intensive nursing care is necessary to avoid pulmonary congestion, pneumonia, ruptured bladder, or bowel atony.
- An excellant herbal remedy consisting of colloidal silver, stabilised oxygen, herbal anti-viral, herbal anti-inflamatory and DMSO is suggested, and has been found to give excellent results.

IMMUNITY

- Immunity after natural infection with either EHV-1 or EHV-4 involves a combination of humoral and cellular immunity.
- Little cross-protection occurs between virus types after primary infection of immunologically naïve foals, significant cross-protection develops in horses after repeated infections with a particular virus type.
- Most horses are latently infected with EHV-1 and EHV-4. The infection remains dormant for most of the horse's life, although stress or immunosuppression may result in recrudescence of disease and shedding of infectious virus.
- Immunity to reinfection of the respiratory tract may persist for up to 3 months, but multiple infections result in a level of immunity that prevents clinical signs of respiratory disease. Diminished resistance in pregnant mares allows cell associated viremia, which may result in transplacental infection of the fetus.

SAMPLE TO BE COLLECTED

- New horses (or those returning from other premises) should be isolated for 3-4 weeks before commingling with resident horses, especially pregnant mares.
- Management-related stress-inducing circumstances should be avoided to prevent recrudescence of latent virus.
- Pregnant mares should be maintained in a group away from the weanlings, yearlings, and horses out of training.
- In an outbreak of respiratory disease or abortion, affected horses should be isolated and appropriate measures taken for disinfection of contaminated premises.
- Vaccine should be administered during 3^{rd}, 5^{th}, 7^{th}, and 9^{th} of pregnancy.

CHAPTER-13: CANINE DISTEMPER, INFECTIOUS CANINE HEPATITIS AND CANINE PARVO VIRAL INFECTION

Learning objectives

To know in detail about

- Causative agent of canine distemper, infectious canine hepatitis and canine parvo viral infection.
- Transmission, clinical signs, diagnosis and prevention of canine distemper, infectious canine hepatitis and canine parvo viral infection.

CANINE DISTEMPER

SYNONYMS AND INTRODUCTION

Synonyms : Carre's disease, Hard-pad disease, Canine influenza

Introduction

- This is an acute febrile systemic, infectious and contagious disease of dogs caused by morbili virus and the disease is characterized by biphasic fever, acute catarrhal inflammation of various mucous membranes, pneumonia and in some cases skin lesions and involvement of nervous system.
- This disease is caused by canine distemper virus (CDV) is an enveloped single stranded RNA virus.
- This virus is the member of genus morbili virus sub family Paramyxo virinae, family Paramyxoviridae.
- This virus is antigenically related to Rinderpest, Peste des petits ruminants and measles viruses. There is only one serotype causing this disease in canines.
- There are three strains of this virus namely A, B and C. The strains A and B were more virulent than C strain. All these strains vary in their pathogenicity and tissue trophism.
- This disease has a worldwide distribution except in hot and arid regions such as parts of Africa.
- All members of *canidae* (dogs, wolves, dingo, fox, coyote, jackal), *Mustelidae* (ferret, skunk, mink, otter, weasel), *Viveridae* (Binturong) and *Procyonidae* (Raccoon, panda, coati) are susceptible to distemper.
- Canine distemper virus causes higher morbidity and mortality than any other virus that infects dogs.
- Incidence is more in foreign breeds than Indigenous breeds.
- The disease is more common in dogs at the age group of 3-6 months.

- The rate of incidence gradually declines with the attainment of age.
- More the age, lesser the rate of infection.

CLINICAL MANIFESTATION

- The disease is most important among dogs.
- The disease may be noted in all age groups, but the young ones between age group of 3-6 months are more susceptible and case fatality rate in this age group are maximum.
- There is high rise of temperature.
- This temperature reaction is characteristic and is known as biphasic reaction.
- The body temperature of the animal raises up to 103˚ to104˚F.
- In this stage the nose of the animal will turn dry and hot and eyes will turn congested.
- The animal is markedly depressed and refuses to take food.
- Within few days yellowish green discharge is voided from the eyes and nose. The temperature is usually for 3-4 days and then drops.
- The temperature is normal till 11^{th} or 12^{th} day which again shows a rise which remains continuous during the course of the systemic infection.
- The second rise of temperature is accompanied by rhinitis, conjunctivitis, gastroenteritis and bronchopneumonia.
- The clinical manifestations vary in respect to severity and involvement of the system concerned.

There are five different forms of manifestation of clinical signs

- Pulmonary form
    - A coryza like syndrome is characterized by oculonasal discharge, pharyngitis, bronchitis are seen.
    - Bronchopneumonia is the common feature.
    - The pulmonary form is more prevalent than digestive form.
    - The pneumonic condition may persist for a long period.
- Gastrointestinal form
    - This form is characterized by loss of appetite, vomition and abdominal pain.
    - The faeces is semisolid or loose; foul smelling.
    - The diarrheic faeces may contain blood.
    - The hemorrhagic enteritis is commonly seen in young pup.
- Ocular form
    - This is manifested as swollen eye lids, conjunctivitis and purulent discharge from the eyes.

- Ulceration of the cornea and transient keratitis may also be located.

Canine distemper-Keratitis

- Nervous form
  - The affection of the nerve cells lead to neurological disorders.
  - These are characterized by restlessness, excitement, chewing movements, excessive salivation and convulsion.
  - The nervous manifestations are noted when the animal can resist the primary infection and suffer from secondary complication.
  - Lymphopaenia is the distinct feature of distemper but this may not occur in dogs with delayed encephalitis.
  - A condition known as “old dog encephalitis” characterized by mental disorders, motor deterioration and death as clinical features.
  - The nervous form may be characterized by jerky movements of group of muscles.
  - The muscular spasms may be observed in the lips, cheeks, jaws, head, neck or limb muscles.
- Cutaneous form
  - There is appearance of rash, vesicles and pustules on the body of the affected dog especially on the ventral aspect of the abdomen and on the inner parts of the thigh.
  - In some cases the skin of the foot pads and nose may become hard due to hyperkeratitis and the condition is ascribed as hardpad disease.

DIAGNOSIS

- Based on clinical signs
- Postmortem lesions
    - Congestion of the brain and meninges. Reduction in the size of thymus, pneumonic changes of the lungs, catarrhal or hemorrhagic enteritis, hyperkeratitis in the foot pad and nose
    - Intracytoplasmic inclusion bodies in the epithelial cells of the respiratory system, intestine, kidney, urinary bladder and renal pelvis
- Animal inoculation
    - Dogs or ferrets are to be inoculated with the suspected materials and observations are to be recorded about clinical signs, lesions and inclusion bodies.
- Detection of inclusion bodies
    - In living clinically affected dogs, intracytoplasmic eosinophilic inclusion bodies are noted in the conjunctival or vaginal epithelial impressions.
    - These inclusions are reliable aid in diagnosis of canine distemper
- Fluorescent antibody technique (FAT)
    - The direct or indirect FAT is employed to detect the presence of viral antigen.
    - This test offers reliable clue for the demonstration of the virus in tissue smears and sections.
    - Impressions of mucous membrane and tissues from foot pad are the samples for this disease diagnosis by FAT.
    - The virus used to persist in foot pad for a longer period of time.
- Isolation of the virus
    - Isolation of the virus from nasal or conjunctival swabs and inoculation in the cell cultures and embryonated chicken eggs

PREVENTION AND CONTROL

- Canine distemper vaccine alone or in combination with other diseases may be used with the first dose of vaccine at 6-8 weeks of age with additional doses at 3-4 weeks intervals until puppies are 14 weeks of age. Annual revaccination may be required at enzootic areas.
- Besides immunization, isolation of CD diseased dogs appears to be the most important measure in controlling the disease.
- As the virus is shed in all body secretions and excretions during the acute systemic disease and direct dog-to-dog contact appears to be the main route of viral spread.
- Disinfection of a CDV contaminated environment can be accomplished with commonly used disinfectants because the enveloped virus destroyed outside the host.

INFECTIOUS CANINE HEPATITIS

SYNONYMS AND INTRODUCTION

Synonyms : Rubarth's disease, contagious hepatitis

Introduction

- It is an acute infectious viral disease of dogs of all ages, characterized by high rise of temperature, vomition, diarrhoea and convulsion.
- This disease is caused by a DNA virus belongs to adenovirus.
- Among the members of the *Canidae* family dogs and foxes are more important species to suffer from this disease.
- Among the dogs younger dogs under one year of age suffer with acute form of the disease after which the dogs are inapparently infected with periodic relapses.
- Excretions and secretions are the important sources of the virus to healthy animals.

CLINICAL MANIFESTATION

- Incubation period for this disease is 6-9 days.

Acute form

- Acute form of the disease starts with apathy, anorexia and high rise of temperature up to 105ºF.
- There is often vomition and diarrhoea.
- The faeces is often blood tinged and abdominal pain also evinced.
- The first rise of temperature usually falls after 24-48 hours without leveling to normal temperature; it rises again to form a saddle curve which will last for 6 days.
- Animal shows intense thirst and the buccal mucous membrane turn fiery red or even hemorrhagic.
- There is swelling of liver capsule which results in pain on palpation of xiphoid region.
- The dogs may show "tucked up" condition of the abdomen.
- After 1-3 weeks following disappearance of clinical signs, a transient corneal opacity may develop.
- This condition is known as "Blue eye". This is due to iridocyclitis and corneal oedema.

DIAGNOSIS, PREVENTION AND CONTROL

Diagnosis

- Based on clinical signs
- Serological tests – AGID, CFT, VNT

Prevention and control

- Combined vaccine with distemper is used to prevent this disease.
- First vaccine administered at 6-8 weeks of age and followed by booster at 3-4 weeks interval and annual revaccination.

CANINE PARVO VIRAL INFECTION

INTRODUCTION

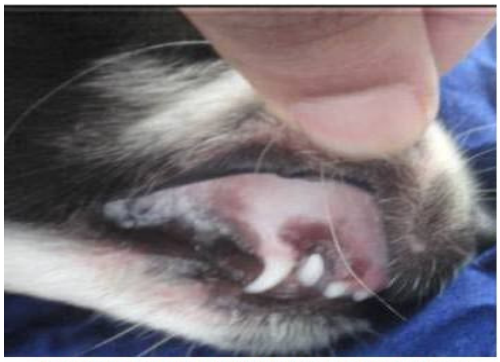
- Canine parvo viral infection is caused by canine parvo virus 1 (CPV), smallest DNA virus belonging to the family Parvo viridae.
- Wide varieties of hosts are affected with Parvo virus including cattle, pigs, dogs, cats.
- Breeds of dogs like Dobermann and Labrador are highly susceptible than other breeds of dogs.
- The virus is transmitted through direct contact with the infected animal or its excretions.
- Virus excreted in large quantities in the faeces of the affected dogs.
- Infection spreads when the dogs are placed in close aggregation in large breeding establishments, pet shops, clinics.

CLINICAL MANIFESTATIONS

- Two forms of manifestations are observed in parvo virus infection

Parvoviral enteritis

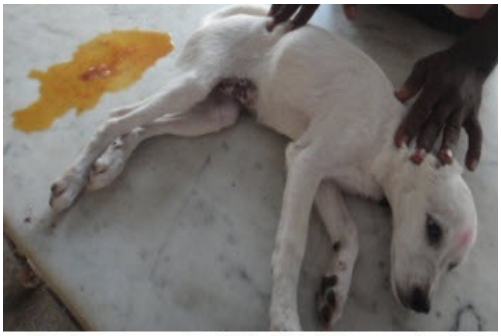
- Hemorrhagic gastroenteritis
- Occurs in dogs of any age but serious proportions in pups
- Slight to high rise of temperature in initial stage of disease but gradually turn to subnormal level with advancement of vomition and diarrhoea
- Inappetance, polydipsia, frothy yellow coloured vomitus, retching
- Dehydration and exhaustion
- Death due to dehydration leading to peripheral circulatory failure



Canine parvo viral infection-pale buccal mucous membrane

Canine parvo viral infection-shunken eyeball

Vomition in Canine parvo viral infection



Canine parvo viral infection-Haemorrhagic enteritis

Canine parvo viral infection-Hameorrhagic enteritis

Parvo virus myocarditis

- Common in pups under 10 weeks of age
- Heart muscles damaged, affected animals show the signs of circulatory failure
- Pulmonary oedema
- Death due to cardiogenic shock
- Death in dogs of 4-8 weeks of age

DIAGNOSIS,PREVENTION AND CONTROL

Diagnosis

- Specimens- Faeces, serum
- Based on clinical signs
- Serological tests- HA, HI, Serum neutralization test, FAT, ELISA,
- Isolation of the virus from faeces

Prevention and control

- Strict hygienic measures adopted in a kennel
- Segregation of infected animal from the normal healthy animals
- Vaccination- Primary vaccination with inactivated vaccines at 6-8 weeks of age, booster at 12th week of age and annual booster vaccination.

CHAPTER-14: HIGHLY PATHOGENIC AVIAN INFLUENZA AND NEWCASTLE DISEASE

Learning objectives

To know in detail about

- HPAI virus and NDV
- Transmission of ND and HPAI
- Various clinical manifestation of HPAI and ND
- Diagnosis, prevention and control of HPAI and ND.

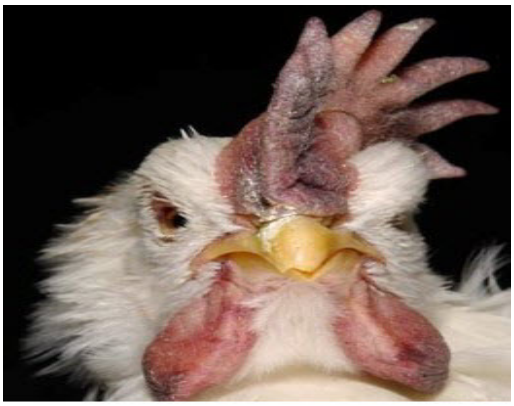
HIGHLY PATHOGENIC AVIAN INFLUENZA

INTRODUCTION

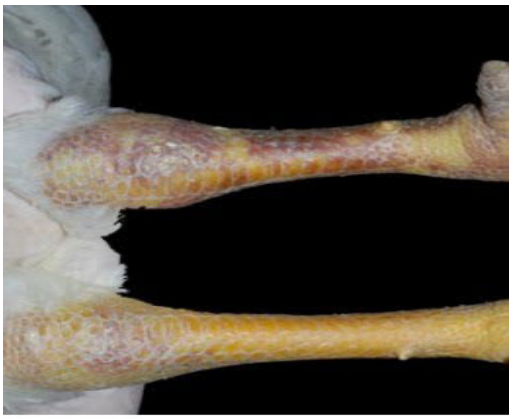
- Influenza is an infection or disease syndrome caused by any type "A" influenza virus, a member of the *Orthomyxoviridae* family.
- Influenza "A" viruses are responsible for major disease problems in birds, as well as in human and lower mammals.
- Avian influenza viruses are distributed throughout the world in many domestic birds including turkeys, chickens, guinea fowl, chukars, quails, pheasants, geese and other waterfowls.
- Domestic turkeys and chickens have experienced the most substantial disease problems due to influenza.
- The disease caused in chickens and turkeys by HPAI viruses has historically been termed "fowl plague".
- The highly pathogenic avian influenza viruses should be used to describe viruses causing high mortality upon experimental infections of chickens.

CLINICAL MANIFESTATION

- The signs of disease are extremely variable and depend on the species affected, age, sex, concurrent infections, virus, environmental factors etc. Signs may reflect abnormalities of the respiratory or enteric or reproductive or nervous systems.
- The signs most commonly reported include pronounced depression and decreased activity, decreased feed consumption and emaciation, increased broodiness of hens and decreased egg production, mild to severe respiratory signs including coughing, sneezing, rales and excessive lacrymation, huddling, ruffled feathers, odema of head and face, cyanosis of unfeathered skin, nervous disorder and diarrhoea.
- Any of these signs may occur singly or in various combination.



AI-Bilateral odema on face

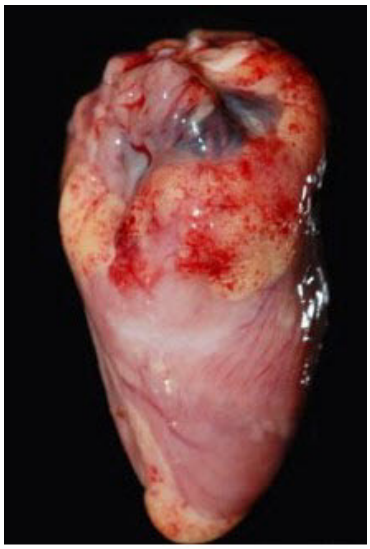


AI-Different degree of haemorrhages on shank

AI-Depression of the bird with ecchymoses on the comb

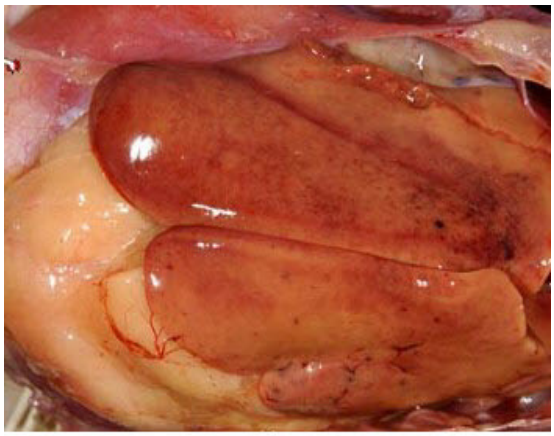
AI-Ecchymotic haemorrhage in the skin

LESIONS

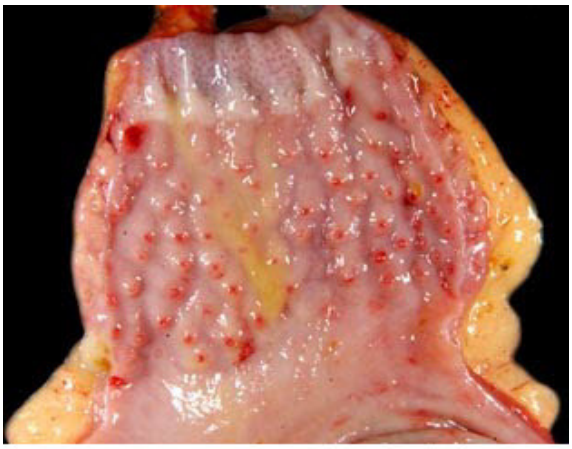


AI-Petechiae in heart

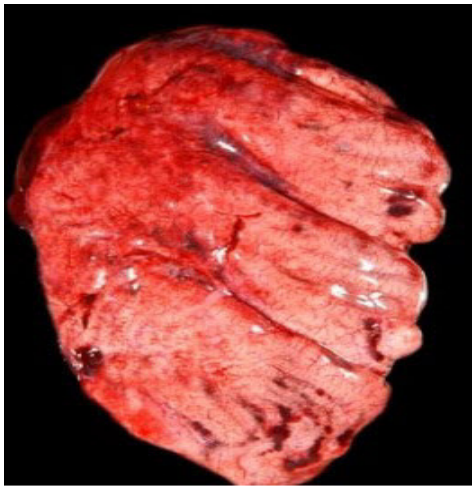
AI-Congestion and haemorrhage in ovarian follicles



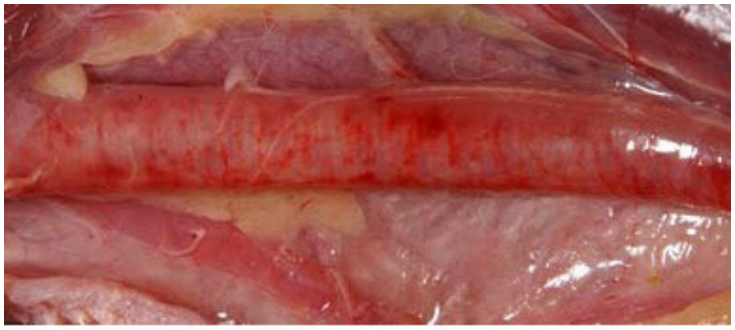
AI-Diffuse haemorrhage on the liver with hepatopathy



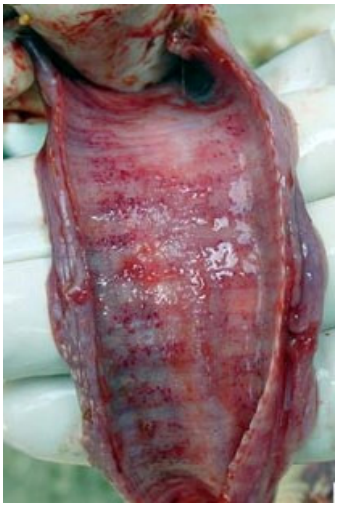
AI-Haemorrhage in proventriculus

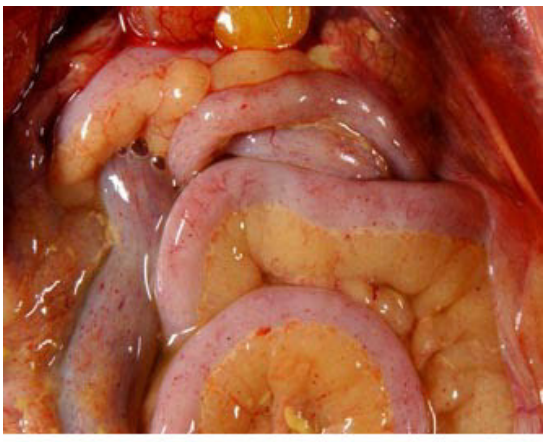


AI-Haemorrhages in lungs

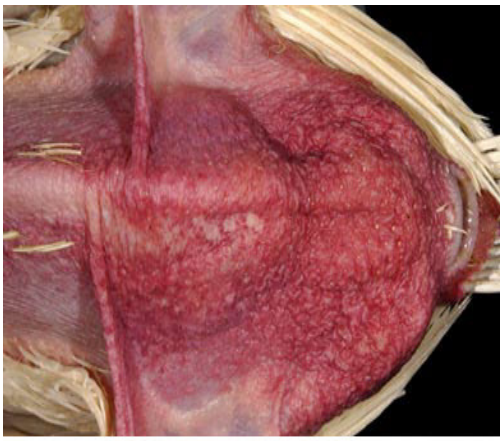


AI-Haemorrhagic tracheitis

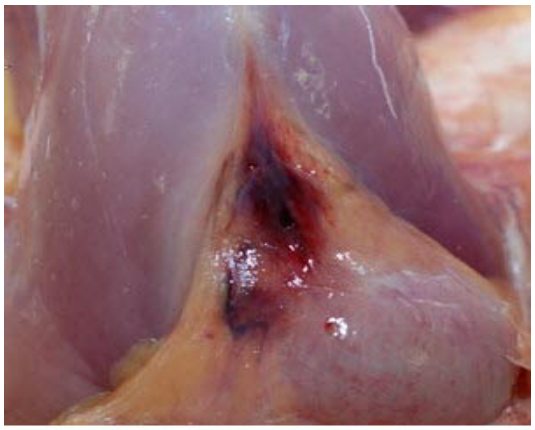




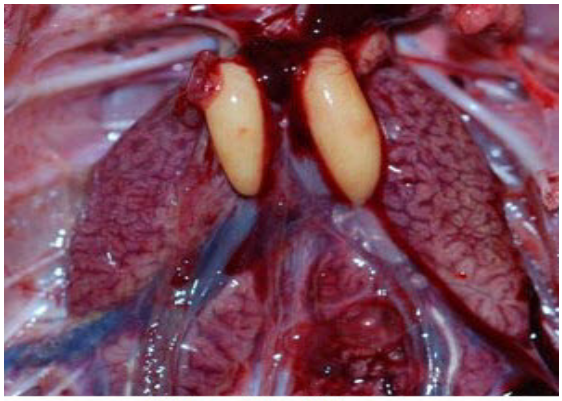
AI-Multifocal haemorrhages in intestine



AI-Subcutaneous haemorrhage in skin



AI-Haemorrhage in fat



AI-Swollen kidneys

HPAI INFECTIONS OF CHICKENS,TURKEYS AND DUCKS

HPAI infections of chickens and turkeys

- None of the clinical signs seen in HPAI infections of turkeys and chickens is pathognomonic.
- Many symptoms and post mortem lesions are similar to avian pasteurellosis.
- In general, more clinical signs are seen the longer the birds survive HPAI infections, and in birds surviving >48 hours post infection, the following series of classical clinical signs can be seen:
    - Sudden onset of high mortality
    - Cessation of egg laying
    - Respiratory signs
    - Excessive lacrymation
    - Sinusitis
    - Odema of the head, face and neck
    - Cyanosis of unfeathered skin, particularly the comb and wattles and diarrhea

- The very virulent viruses cause only sudden death.

HPAI in ducks

- Ducks are refractory to even the most pathogenic influenza viruses for chickens and turkeys, and this may be important in the epizootiology of HPAI.

DIAGNOSIS

Samples to be collected

Dead birds

- Intestinal contents (faeces) or cloacal swabs
- Oropharyngeal swabs
- Samples from trachea, lungs, air sacs, intestine, spleen, kidney, brain, liver and heart should also collected and processed either separately or as a pool

Live birds

- Oropharyngeal and cloacal swabs.
- Swabbing of small delicate birds should be done with the use of especially small swabs that are usually commercially available and intended for use in humans.
- Where these are not available, the collection of fresh faeces may serve as an alternative.

Other specimens

- Drinking water or water from the water sources frequented by the birds.
- All samples should be placed in PBS pH 7.0-7.4 containing antibiotics. Faeces and tissue should be homogenized in PBS to give a suspension of 10-20% w/v.
- The range and concentration of antibiotics are penicillin 2000 units/ml, streptomycin 2mg/ml, gentamycin 50µg/ml and mycostatin 1000 units/ml.
- These levels are suitable for tracheal swabs and tissue suspensions. But 5 fold higher concentrations should be used for cloacal swabs and faeces.
- Samples should be processed as soon as possible, but they may be stored in the antibiotic solution for up to 2 days at 4°C or for longer periods in the freezer at -70°C or on dry ice.
- Samples should be left in antibiotic solution at room temperature for 1-2 hours before inoculation.
- Embryonated fowl eggs are the most commonly used growth system for Influenza “A” viruses.
- Sample volume of 0.2 ml is inoculated into the amnio-allantoic cavity of five 8-10 day old embryonated fowl eggs.

- Tissue and faeces samples may need centrifugation at 1000 X g for 10 minutes before samples are taken for inoculation.
- Inoculated eggs are usually incubated at 37 ◦ C and their viability is monitored twice a day by candling.
- Dying or dead embryos as they arise and all eggs at 5 days post inoculation are chilled to 4◦C and the harvested amnio-allantoic fluids are tested for HA activity.
- Negative fluids should be passaged, undiluted once more.

Serological diagnosis

- Haemagglutination inhibition (HI)
- Agar gel immuno diffusion (AGID) test

Differential diagnosis

- This disease should be differentiated from the other diseases such as
    - Newcastle disease (ND) and other Paramyxo viral infections
    - Chlamydia
    - Mycoplasma infections

PREVENTION AND CONTROL

Prevention

- Separation of susceptible birds from infected birds and their secretions and excretions
- Entry of wild birds and swine into the farm premises should be prevented
- Following strict insecurity measures

Vaccination

- Selection of subtype of the HPAI virus dominant in the particular area and an inactivated vaccine was prepared from the particular strain of the virus and used for vaccination.

Control

- All methods for controlling the spread of influenza are based on preventing the contamination of, and controlling the movement of, people and equipment.
- Persons have direct contact with birds or their manure should not be moved from farm to farm, and it is important to keep the traffic area near the poultry house from becoming not contaminated with manure.
- In high risk areas such as those on migratory waterfowl routes, all domestic poultry will have to be reared in bird-proofed confinement, if freedom from infection is to be maintained.

- Prevention of secondary spread is also important.
- Secondary spread is prevented by
    - Controlled marketing and depopulation of infected flocks after recovery from acute disease
    - Cleaning and disinfection of buildings after depopulation
    - A rest period of 3-4 weeks before repopulation
    - Controlled movement of people and strict sanitary procedures
    - Prevention of access of wild birds to poultry flocks
    - Vaccination
- In HPAI outbreaks governmental eradication procedures (quarantine, slaughter, disposal and cleanup are employed.

NEWCASTLE DISEASE

SYNONYMS AND INTRODUCTION

Synonym : Ranikhet disease, Avian Pneumoencephalitis, Pseudo fowl pest, avian pest, Avian distemper, Vellaikalichal (Vernacular), Doyle's disease

Introduction

- Newcastle disease is a highly contagious acute viral disease of birds characterized by respiratory signs (distress, coughing, sneezing), nervous signs leading to wing paralysis, inco-ordination, torticolis, swelling of head and chalky white diarrhoea.
- It was first observed in 1926, in Java, Indonesia and in Newcastle-upon-Tyne, England.
- The name Newcastle disease was coined by Doyle. In India in July 1927, the virus was recognized in Ranikhet by Edwards. Hence it named as Newcastle disease or Ranikhet disease. Nine serotypes of avian paramyxoviruses recognized but only avian paramyxovirus 1 is associated with clearly defined disease.
- This disease is caused by single stranded RNA virus namely Avian paramyxovirus type -1, Genus: Avula virus subfamily: Paramyxo virinae, family: Paramyxo viridae order: Mononegavirales.
- The genome of NDV codes for six proteins.
- They are NP – Nucleocapsid protein, P – Phosphorylated - Nucleocapsid associated, M – Matrix protein, F – fusion protein, forms the smaller of the surface projections, HN – haemagglutinin and neuraminidase – larger of the surface projections and L - RNA directed RNA polymerase associated with the nucleocapsid.
- The “HN” protein is responsible for attachment and release of virus from the host cell in addition to its serological identification.

- The “F” plays a major role in fusion of the virus and cell membrane and it has a critical role in pathogenesis.
- The Newcastle disease virus (NDV) causes agglutination of chicken and human ‘O’ erythrocytes.
- The agglutination reaction is not permanent and after 30 minutes as a result of loss of receptor the agglutination is lost due to the action of neuraminidase and this phenomenon is referred as elution.
- Only one serotype of NDV recognized, but many strains are there.
- All strains are immunologically identical. Some of the important virus strains are as follows

Pathotype Strains

  - Velogenic Hertz 33, Texas GB
  - Mesogenic K, R 2 B
  - Lentogenic F, LaSota, Ulster 2C
  - Avirulent V4

- The virus is comparatively stable virus and survives for long periods at ambient temperature, especially in faeces and can persist in houses (in faeces, dust etc.) for up to 12 months. However it is sensitive to disinfectants, fumigants and sunlight.
- The virus is inactivated by temperatures of 56^0C for 3 hours or 60^0 C for 30 min, acid pH, formalin and phenol, and is ether sensitive.
- This virus can be readily cultivated in embryonated eggs. The preferred route is allantoic cavity in 9-11 days old embryos.
- Virulent strains cause death of embryos in 24 hours with hemorrhages through out the body particularly in occipital region.
- Lentogenic strains do not cause death. Presence of virus is usually confirmed by HA and HI test using specific serum.
- The virus also grows readily in primary cell cultures like chicken embryo fibroblast and pig kidney cells and in BHK 21 cell line.
- Domestic chicken is the principal host. In addition to chickens, turkeys, pheasants, guinea fowl, ducks, geese, and pigeons, a wide range of captive and free ranging semi domestic and free-living birds, including migratory waterfowl, is susceptible.
- NDV has been reported at least 241 species of birds. In birds that survive, virus is shed in all secretions and excretions for at least 4 weeks.
- Transmission occurs by direct contact between birds by the airborne route via aerosols and dust particles by inhalation, ingestion of faeces or contaminated material, by inanimate objects, by movement of people, contaminated feed and water, by non-avian species like flying insects, fly (*Musca domestica*), ectoparasites (*Argus persicus*), endoparasites (*Ascardia galli, Eimeria tenella* etc.) and by vaccine also (incomplete inactivation and exaltation).

- With lentogenic strains, tvertical transmission is important, and virus –infected chicks may hatch from virus-containing eggs.
- Some psittacine species may become persistently infected with velogenic virus and excrete it intermittently for more than 1 year without showing clinical signs.
- Virus may also be disseminated by frozen chickens, uncooked kitchen waste, foodstuffs, bedding, manure and transport containers.

CLINICAL FEATURES

- There are five forms of disease recognized.

Viscerotropic velogenic (Doyle's form or Asiatic form)

- Very acute form with hemorrhagic lesions of the digestive tract.
- The infection caused by this also called as Asiatic or exotic ND.

Neurotropic velogenic (Beach's form)

- Viruses causing disease characterized by high mortality, which follows neurological and respiratory disease.

Mesogenic (Beaudette's form)

- Acute respiratory and sometimes lethal nervous infections of young chicks.

Lentogenic (Hitchner's form)

- Mild or inapparent respiratory infections of chicks.

Asymptomatic enteric

- Inapparent intestinal infection
- Incubation period is varies from 2 to 15 days (VVND – 2 to 6 days).
- Morbidity is usually high and up to 100%. In chickens respiratory, circulatory, gastro intestinal and nervous signs are seen.
- A combination of dyspnea, cyanosis of comb and wattles, and clonic muscular spasm is indicative
- There is a loss of appetite, listlessness, abnormal thirst, huddling, weakness, and somnolence
- Intestinal symptoms may include crop dilatation, presence of foamy mucus and fibrinous exudates in the pharynx, a similar discharge from the beak, and yellow-green diarrhoea
- Nervous system involvement is indicated by paralysis of wings and /or legs, torticolis, ataxia or circular movements, bobbing-and-weaving movements of the head, and clonic spasms.

- In layers, there is a sudden decrease in egg production together with depigmentation and/or loss of shell and reduction in the albumen quality of eggs
- In VVND, edema of the head, especially around the eyes may become apparent after birds have been sick for 2 or 3 days.
- This odema usually does not involve comb and wattle as in a case of HPAI. A dark ring sometimes forms around the eye, probably due to cyanosis and poor blood circulation in the odematous tissue.
- This 'black eye' appearance is especially visible in white chickens. Bile stained, greenish-dark diarrhoea may be noted 2 to 3 days after onset of illness.

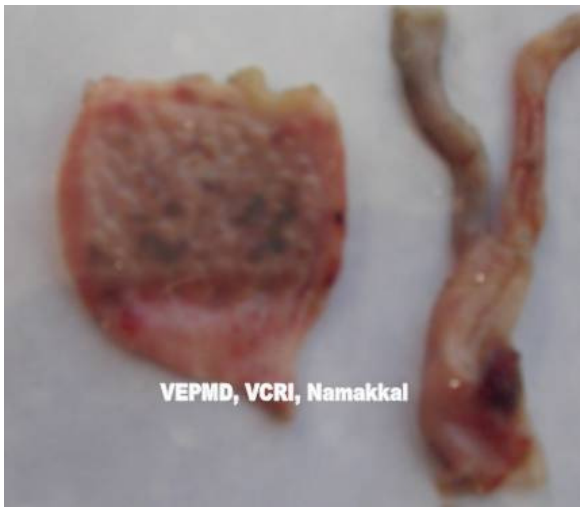
ND-Greenish white diarrhoea

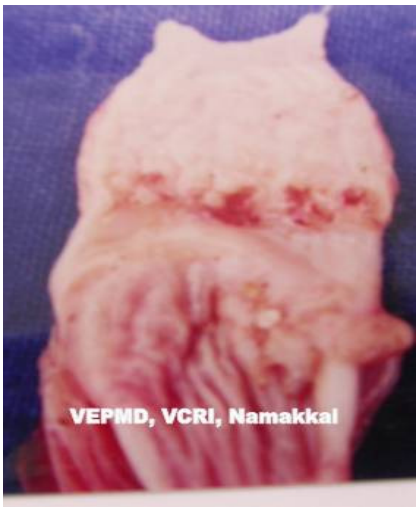
ND-Torticolis

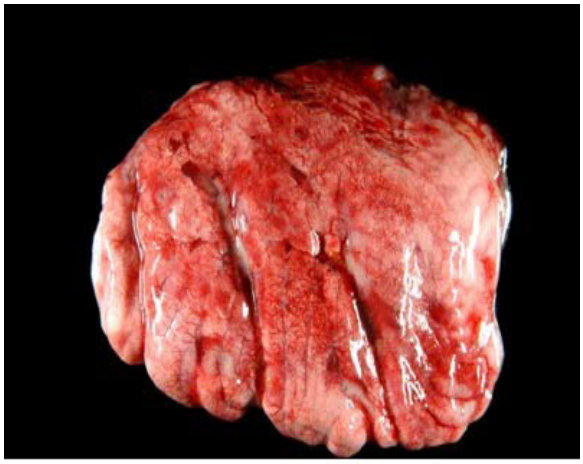
ND-signs of severe depression

LESIONS

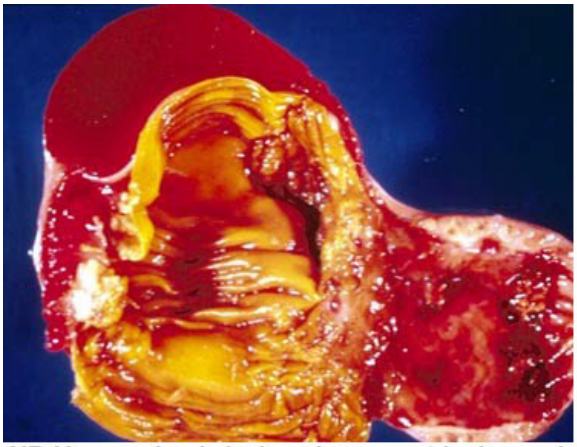
- Ecchymotic hemorrhages in the larynx, trachea, esophagus and throughout the intestine
- The most prominent histological lesions are necrotic foci in the intestinal mucosa and lymphatic tissue and hyperemic changes in most organs, including the brain.
- Non-purulent encephalomyelitis in CNS is also seen
- Virulent velogenic strains cause predominantly hemorrhagic lesions, in particular at the oesophagus/proventriculus and proventriculus/gizzard junctions and in the posterior half of the duodenum, the jejunum, and ileum. These lesions are virtually pathognomic for velogenic strains
- Hemorrhages with necrosis in the intestinal wall. In addition to this egg peritonitis, edematous, hemorrhagic or degenerated ovary is also seen.







ND-Extensive haemorrhages in the lung



ND-Haemorrhagic lesions in proventriculus and gizzard

DIAGNOSIS

Samples to be collected

- Live birds: Cloacal swabs, droppings, oro-nasal swabs
- Dead birds: Brain, bile, caecal tonsils, liver, spleen, lung, kidney and heart tissue or pooled organ samples
- Based on history and clinical signs
- Samples taken from birds are inoculated into the allantoic cavity of 9–11-day-old embryonated fowl eggs. The eggs are incubated at 37°C for 4–7 days. The allantoic fluid of any egg containing dead or dying embryos, as they arise, and all eggs at the end of the incubation period are tested for haemagglutinating activity. Any haemagglutinating agents should be tested for specific inhibition with a monospecific antiserum to NDV. The NDV may show some antigenic cross-relationship with some of the other avian paramyxovirus serotypes, particularly APMV-3 and APMV-7. The pathogenicity of any newly isolated virus can be assessed by determining the intracerebral pathogenicity index (ICPI), Mean death time (MDT) and Intravenous pathogenicity index (IVPI). The most virulent viruses give ICPI values approaching the maximum score of 2.0, while lentogenic viruses give values of, or close to 0.0.
- Serological tests: The haemagglutination inhibition test is used most widely in NDV serology, its usefulness in diagnosis depends on the vaccinal immune status of the birds to be tested and on prevailing disease conditions.
- Demonstration of viral antigen on impression smears by IFT and other serological tests viz., HI, ELISA and molecular techniques like RT-PCR are highly valuable.

PREVENTION AND CONTROL

- Control is mainly by vaccination.
- There are three types of vaccines used for ND
    - Live Lentogenic-F, LaSota
    - Live mesogenic - Komarov, Roakin, Mukhteswar, R_2B
    - Inactivated vaccines
- Live lentogenic vaccines are usually derived from field viruses that have been show to have low pathogenicity for poultry but produce an adequate immune response.
- Typical vaccine strains are HB1, LaSota, F, V4 or Ulster 2C viruses. These vaccines are given to young chicks through eyes or nostrils.
- They are very safe and only LaSota will produce mild post vaccinal reaction in certain flocks. This type immunization is also called as priming.
- Mesogenic strains are used only in areas where ND is epidemic and in village chickens.
- These vaccines are suitable only for secondary vaccination because of their high virulence.
- Normally mesogenic chickens, such as Komarov, Roakin, Mukhteswar and R2B are used as secondary vaccines after a primary vaccination with a lentogenic vaccine.

- Inactivated vaccines are produced by growing a ND virus in eggs, and then treating the infective allantoic fluid with an inactivating agent and adjuvant, such as formalin or betapropinolactone-and mineral oil as adjuvant.
- Inactivated vaccines are usually applied after an initial priming vaccination with a live vaccine.
- Vaccination is usually applied through water, aerosol spray, eye or nasal drops, or by injection.

Ranikhet F strain	4-10 days	Intra ocular or intra nasal
Ranikhet disease F strain (Booster)	5-6 weeks	Intra ocular or drinking water
Ranikhet (R2B) strain	8-9 weeks	Subcutaneous
Ranikhet disease (Killed)	18-19 weeks	Intramuscular or subcutaneous

- Broilers are vaccinated with Ranikhet disease F strain at 7, and 21 days. Layers are also vaccinated at 10 and 22 weeks (point of lay).
- In addition to vaccination usual biosecurity measures should be followed.

Control

- National Level- Quarantine, slaughter and vaccination (Ring vaccination)
- Farm level- Strict hygienic practices and medical measures
- By vaccination
- Import restrictions on chickens and eggs
- Quarantine of psittacine birds in the same air space as non-immune chickens.

CHAPTER-15: MAREK'S DISEASE AND AVIAN LEUKOSIS

Learning objectives

To know in detail about

- MD virus and lymphoid leukosis virus
- Host range of MDV and lymphoid leukosis virus
- Clinical manifestation of MD and lymphoid leucosis
- Diagnosis, prevention and control of MD and lymphoid leukosis.

MAREK'S DISEASE

SYNONYMS AND INTRODUCTION

Synonyms: Range paralysis, skin leucosis, Neural leucosis, Neural lymphomatosis, pearl eye

Introduction

- Marek's disease (MD) is a lymphoproliferative, highly contagious disease of poultry and caused by a highly cell associated gallid herpes virus, a DNA virus belonging to Herpes viridae family.
- There are three serotypes of the virus recognized. Serotypes 1 and 2 are designated as virulent and avirulent chicken isolates respectively.
- Serotype 3 designates the avirulent turkey herpes virus.
- Serotypes 2 and 3 and attenuated serotype-I virus are used for vaccine production. The virus remains stable for about 24 hours at 30˚C.
- The Marek's disease virus (MDV) has been propagated and assayed in newly hatched chicks, tissue cultures (co-cultivation of lymphocytes with chicken kidney cells or duck embryo fibroblasts) and embryonated eggs. This disease is exists in poultry-producing countries throughout the world.
- The infection is transmitted through inhalation of infected material from the environment.
- Virus particle can persist for a considerable period of time in the dandruff of feather follicles, which are released in environment.
- The infective materials are oral, nasal and tracheal secretions and litter materials. The darkling beetle (*Alphitrbius diaperinus*) is acting as mechanical transmitter of the disease.
- The chickens are the most important natural host and MD is very rare and probably of no real importance in other species with the possible exception of quail.

- Chickens of 12-24 weeks of age are mostly susceptible to Marek's disease and generally it does not occur in chickens below 6 weeks of age and older birds above 24 weeks of age.

CLINICAL MANIFESTATIONS

- Incubation period-ranges from 3 weeks to 9 weeks.
- The disease appears in several forms.
- Classical form or neural form
    - Birds of 16-20 weeks age usually suffer.
    - Signs are mostly concerned with the affection of nerves.
    - Paralysis of legs, drooping wings.
    - Nerves like sciatic nerve, brachial nerve, celiac and vagus nerve running through neck, thoracic and abdominal viscera are affected.
    - Birds unable to stand remain in recumbent position, legs and wings may stretched in either direction. The "split leg" stance is the usual feature.
    - Vents remain soiled with green diarrhoea.
    - Mortality rate is comparatively low and mostly noted at the onset of maturity.
- Acute or visceral form
    - Generally birds at the age of 3-4 weeks are affected.
    - Depression, droopiness, unthirfitness, dehydration, emaciation and anaemia.
    - Internal organs of the birds affected.
    - Mortality rate may go as high as 60%.
    - Chicks may die suddenly without showing any clinical manifestation.
    - Ovaries of the affected layers and pullets – looks like a cauliflower and mulberry respectively.
- Transitional paralytic form
    - Occurs in chickens at the age of 5-18 weeks of age.
    - Sudden development of paresis or paralysis of the legs, wings and neck.
    - Signs usually disappear within 24-48 hours.
- Ocular form
    - Blindness in birds due to mononuclear cell infiltration in the iris causing "grey eye" or "pearl eye".
- Skin or cutaneous form
    - Distinct white nodules on the skin and in extreme cases looks like brownish nodules.
- Muscular form
    - Superficial and deep muscles like pectoral muscles affected.

- Muscles look lusterless, whitish grey and there are tiny white streaks to nodular tumours in the muscles.

Torticolis in MD

Lymphoid infiltration in feather follicles-MD

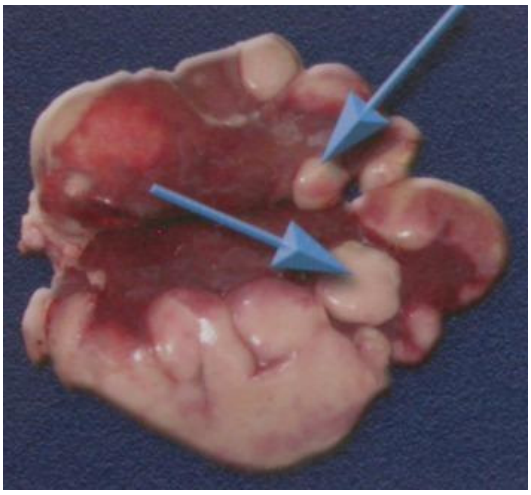
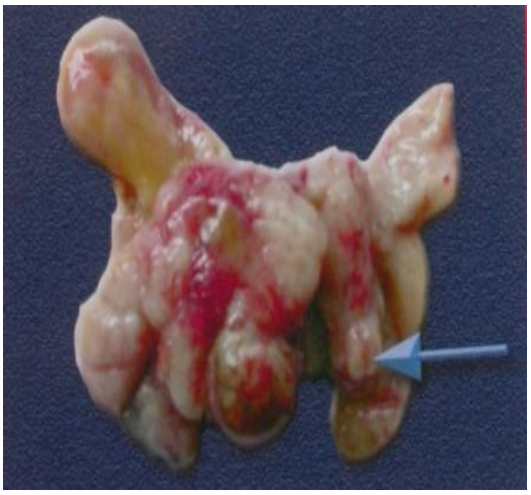
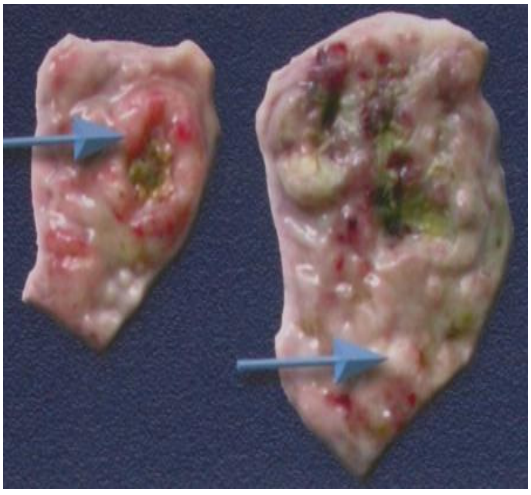
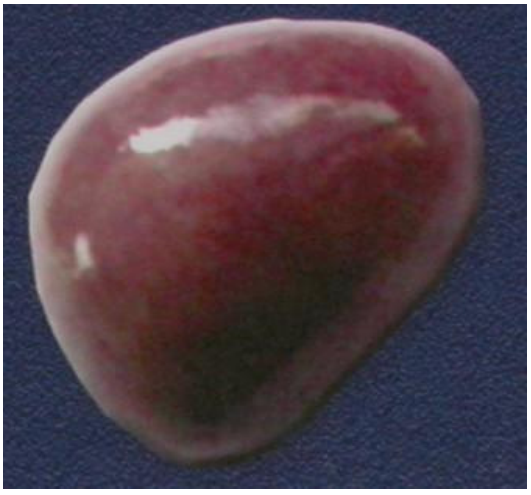
Leg paralysis-MD

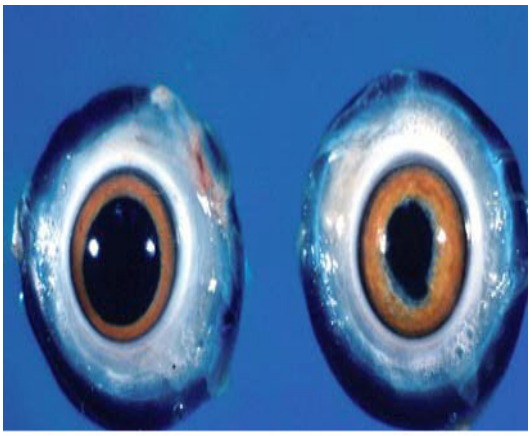
MD-Spastic paralysis

MD-Swelling of wattle and lymphomatosis

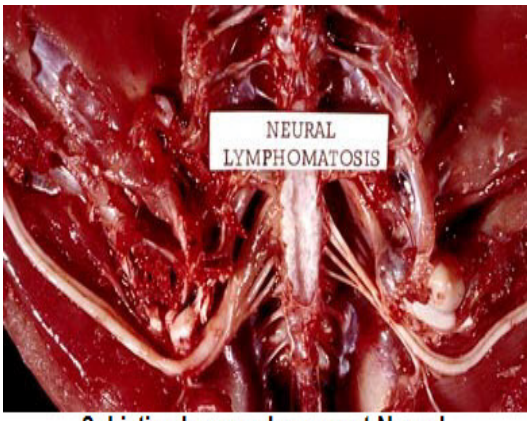
LESIONS

- Affected nerves thickened to more than 2-3 times than normal
- Striation and glistening appearance of nerve is lost and looks oedematous
- Celiac, cranial, mesenteric, brachial and sciatic plexes and greater splanchnic nerves are mostly affected
- Tiny whitish streaks to nodular tumours in muscles
- Atrophy of bursa
- Ovary- cauliflower like appearance
- Pale, single or multiple nodular tumours in myocardium
- Skin- whitish nodule, scab with brownish colour.

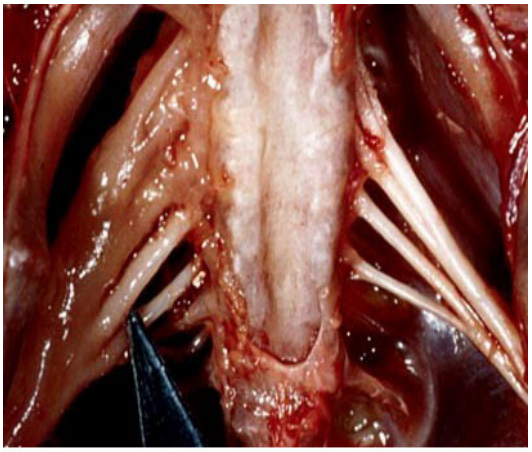







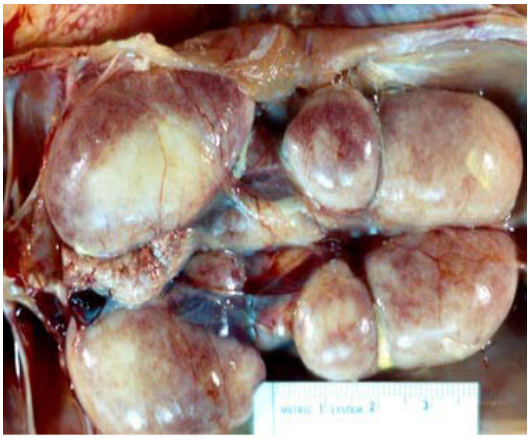
Lymphoid infiltration of iris-MD



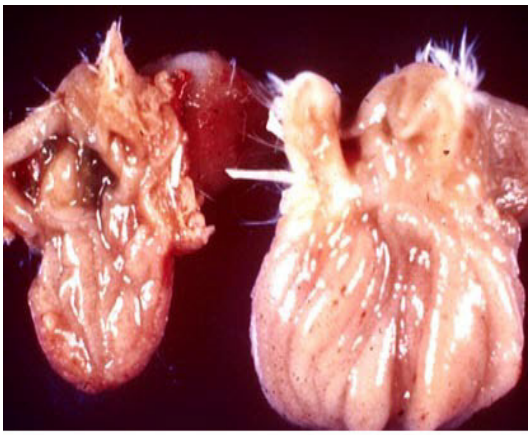
Schiatic plexus enlargement-Neural lymphomatois -MD



Odema, loss of straiation of nerves-MD

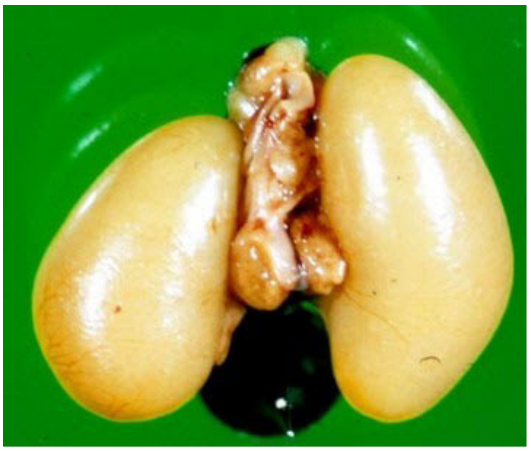


MD-Diffuse enlargement of kidneys

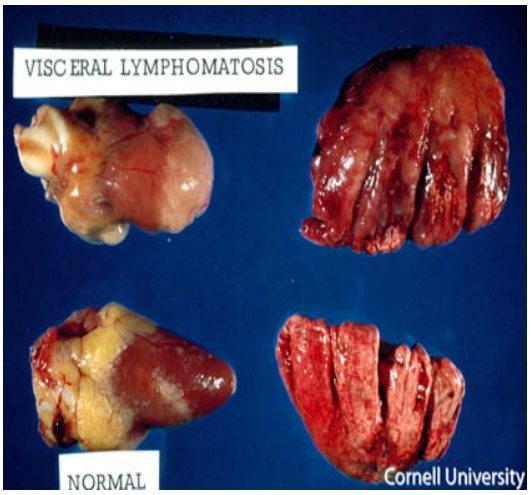


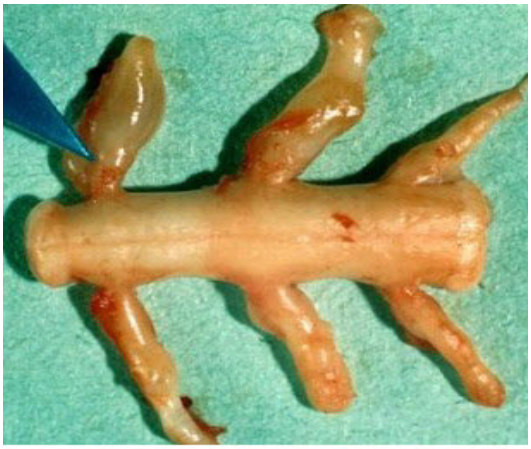
MD-Bursa compared with normal (Left)

MD-Liver

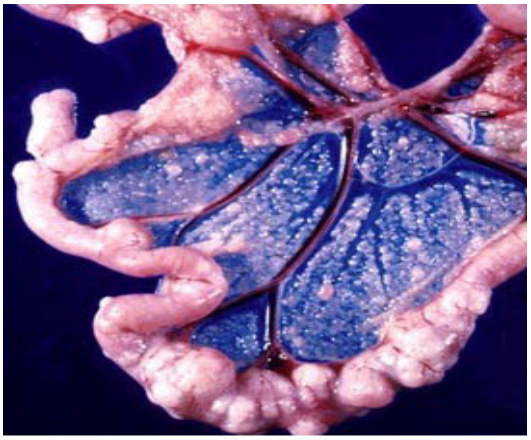


MD-Testes tumour

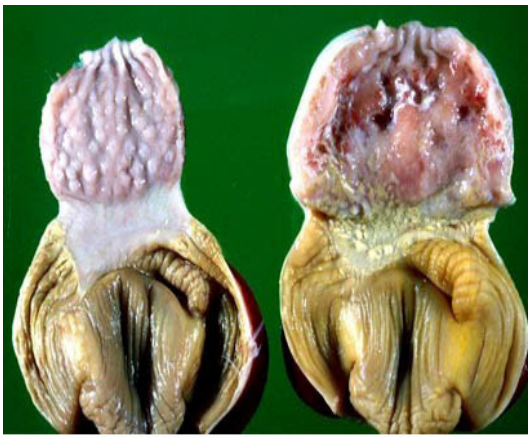




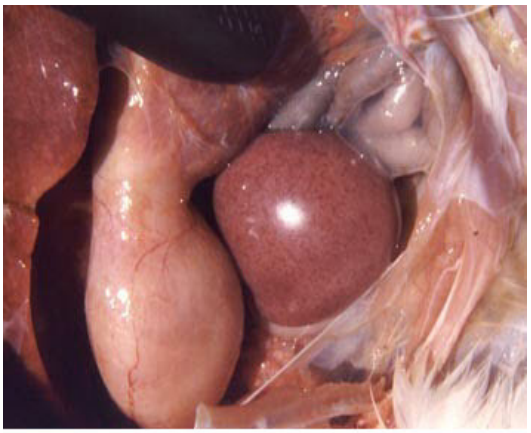
MD-Dorsal root ganglia enlargement



MD-Lymphoid tumours - Intestine



MD-Proventriculus compared with normal (left)



MD-Splenomegaly

DIAGNOSIS

- Specimens to be collected
    - Skin, dander, feather tips of infected chickens, blood
- Based on the clinical signs and postmortem lesions
- Identification of the agent
    - Under field conditions, most chickens become infected with MDV during the first few weeks of life and then carry the infection throughout their lives, often without developing overt disease.
    - The infection is usually detected by inoculating live buffy coat cells on to monolayer cultures of chicken kidney cells or duck embryo fibroblasts, in which characteristic viral plaques develop within a few days.
    - Two serotypes of MDV are recognized – 1 and 2 – and a third serotype is represented by the related herpes virus of turkeys (HVT). Serotype 1 includes the virulent strains and serotype 2 the naturally avirulent strains. MD viral antigen can be detected in the feather tips of infected birds using a radial precipitin test.
- Serological tests
    - Antibodies to MDV develop within 1–2 weeks of infection and are commonly recognized by the agar gel immunodiffusion (AGID) test, the indirect fluorescent antibody test, and by other serological tests such as enzyme-linked immunosorbent assay, virus neutralization tests.

PREVENTION AND CONTROL

- Three classes of vaccines using Attenuated serotype 1 MDV, HVT and natural a virulent isolate of serotype 2.
- Herpes virus turkey (HVT) – most extensively used because it is economical to produce and cell free virus extracted from infected cells.
- The vaccine usually administered at 0 day age by intra nasal or intra ocular route.

AVIAN LEUKOSIS

SYNONYMS AND INTRODUCTION

Synonyms: Avian sarcoma, Big liver disease, visceral lymphoid leukosis, lymphomatosis

Introduction

- Avian leukosis is an infectious cancerous condition of the mature birds involving the haemopoietic and lymphoid tissues like liver, bursa, spleen, gonads, kidneys and bones etc .
- The avian leukosis group of viruses (ALGV) is comprised of 10 subgroups, A to J, based on differences in envelope glycoproteins. Subgroups A to E, and J viruses infect chickens.

- The other subgroup viruses occur in quail, partridges and pheasants. Subgroup 'A' viruses cause most outbreaks of avian leukosis that manifest as a lymphoid leukosis, a B cell lymphoma.
- This is the most important and common neoplasm associated with the disease. Viruses of subgroup J have been associated with myeloid leukosis in broilers.
- Most of the viruses causing avian leukosis are exogenous. Endogenous ALGVs, which mostly belong to subgroup E and occur commonly in chickens and other avian species, are of little or no pathogenicity.
- These proviral DNA sequences are integrated into germ line cells and are thus transmitted vertically.
- Avian leukosis viruses occur commonly in chickens, quail, partridges and pheasants worldwide and the disease is of great economic importance.
- Although transmission may occur by contact, as virus is present in feces and saliva, the principle means of transmission is via the egg.
- Blood-sucking parasites are potential vectors.
- Endogenous viruses, which are common in the chicken but not important as causes of leukosis, are also transmitted genetically in the germ line from parent to offspring

CLINICAL MANIFESTATIONS

- All avian leukemia group viruses are oncogenic, except those belonging to subgroup E. The mechanisms involved in the development of neoplasia by viruses, that is, oncogenesis. Such factors as genetics (autosomally transmitted susceptibility or resistant avian cells) and the age and sex of the host can also be important in the development of tumors.
- The incubation period is variable and may be as long as months or years
- Viruses of subgroups A through D are associated with a variety of neoplastic conditions in chickens, including lymphoid leukosis, erythroblastosis, myeloblastosis, myleocytoblastosis, osteopetrosis, nephroblastomas, hemangiomas, and sarcomas. The more common forms are as follows:
    - *Lymphoid leukosis* is by far the most prevalent form of avian leukosis/sarcoma. The disease is most often seen in chickens at least 16 weeks of age and occurs more commonly in females. Clinical signs may be absent or birds may appear thrifty with pale combs. The abdomen may be enlarged because of massive tumor growth in the liver.
    - *Osteopetrosis* is a sporadic disease that occurs primarily in males. The shafts of the long bones are thickened, often resulting in a stilted gait. Affected birds may be anemic and also have lesions of lymphoid leukosis.
    - *Erythroblastosis* may be manifested in one of two forms, anemic or proliferative, with the latter form being the more common. Clinical signs are similar for both forms. Chickens become depressed, emaciated, and dehydrated. With the anemic form, there are few circulating erythroblasts and chickens appear pale, as opposed to cyanotic with the proliferative form in which circulating erythroblasts are present in large numbers.

- *Myeloblastosis* is similar clinically to erythroblastosis. There is an abnormal proliferation of myeloblasts resulting in severe leukemia.

Lesions

- Diffuse enlargement of liver and spleen and to a lesser extent in kidneys.
- The above organs are cherry red to dark mahogany and are soft and friable
- The bone marrow – hyper plastic, soft, watery, dark blood red or cherry red often with hemorrhages
- Atrophy of the visceral organs and organs of the immune system particularly spleen.

DIAGNOSIS AND PREVENTION

Diagnosis

- *Specimens:* Plasma, serum, tumours, whole bird with extremities, visceral organs (liver, kidney, spleen), oral washings, faeces
- Based on clinical signs and gross and histopathologic lesions, but lymphoid leukosis may be confused with the acute form in older birds.
- Differential features, nodular tumors in the bursa of fabricius as opposed to diffuse enlargement with Marek's disease; intrafollicular cell proliferation in the bursa of fabricius as opposed to interfollicular cell proliferation with Marek's disease; and the cytologic appearance of lymphoid cells that are uniformly "blast" cells with lymphoid leukosis but a mixture of mature and immature pleomorphic cells with Marek's disease.
- Isolation of the virus or the demonstration of antibody is not considered to be of value in diagnosis because avian leukosis group viruses are ubiquitous in chickens.

Prevention

- There is no vaccine available and eradication is the preferred method of control. Since the viruses are primarily transmitted via the egg, virus infected breeders are detected by testing the egg albumin for viral antigen by an ELISA method. Positive birds are eliminated.
- ELISA and Immunofluorescence assays are used to detect antibodies in serum and egg yolk.
- Most commercial flocks of chickens are now free of exogenous ALG viruses.
- Some birds are genetically more resistant to fowl leukosis; they have decreased numbers of specific cell surface viral receptors.
- Incubators should be clean and adequately disinfected.
- Control blood-sucking parasites.

CHAPTER-16: INFECTIOUS BRONCHITIS AND INFECTIOUS LARYNGOTRACHEITIS

Learning objectives

To know in detail about

- IB virus and ILT virus
- Host range of IBV and ILTV
- Clinical manifestations of IB and ILT
- Diagnosis, prevention and control of IB and ILT.

INFECTIOUS BRONCHITIS

SYNONYMS AND INTRODUCTION

Synonym : Gasping disease

Introduction

- An acute, highly contagious respiratory disease of chickens characterized by respiratory signs such as coughing, sneezing, tracheal rales with accumulation of mucus in the bronchi and depression, heavy mortality in young chickens, stunting growth, nephritis, increased feed convertion ratio, long-term reproductive problem, decreased egg production and affect both internal and external quality of egg.
- This disease is distributed worldwide.
- Infectious bronchitis virus (IBV) is a member of the family Coronaviridae, genus Coronavirus positive stranded linear ssRNA virus. Chicken is the only bird that is usually infected by IBV.
- All ages are susceptible, but the disease is most severe in baby chicks. This virus spreads rapidly among chicks by aerosol transmission.

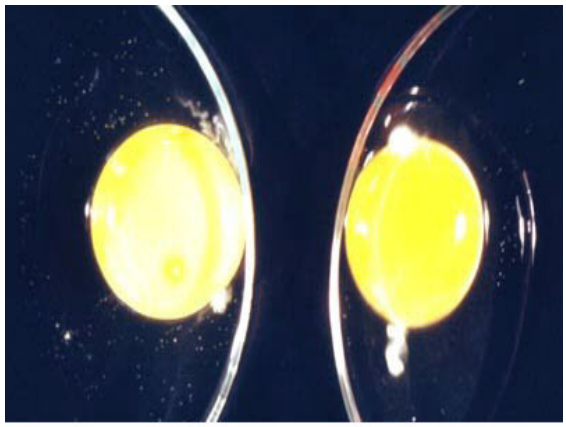
CLINICAL SIGNS AND GROSS LESIONS

Clinical signs

- The incubation period of IB is 18-36 hour depending on the route of inoculation. The characteristic respiratory signs of IB in chicks are gasping, coughing, sneezing, tracheal rales and nasal discharges.
- Wet eyes may be observed and occasional swollen sinuses.
- The chicks appear depressed, may be seen huddled under a heat source and feed consumption and weight gain are significantly reduced.

- In chickens over 6 weeks of age and in adult birds the signs are similar to those in chicks, but nasal discharge does not occur as frequently and the disease may go unnoticed unless the flock is examined carefully by handling the birds or listening to them at night when the birds are normally quiet.
- In laying flocks there is declined egg production and quality are seen in addition to respiratory signs.
- Internal qualities of eggs also affected.
- The albumen of the egg maybe thin and watery without definite demarcation between the thick and thin albumen.

IB - chick with open mouthed breathing



IB- Altered internal quality of egg (left) with normal egg (right)

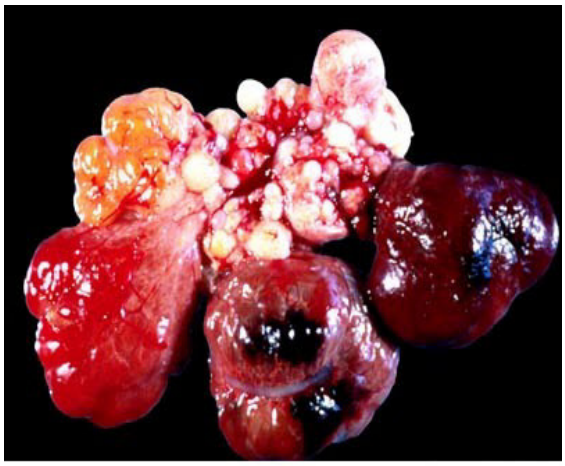
IB-Mucoprulent nasal discharge

Gross lesions

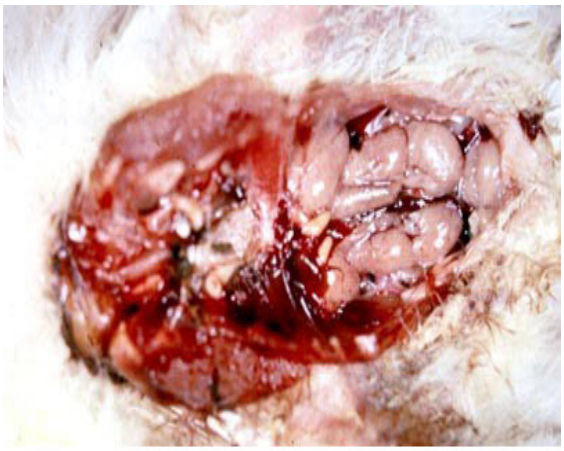
- Infected chickens have serous, catarrhal or caseous exudates in the trachea, nasal passages and sinuses.
- Airsacs may appear cloudy or contain yellow caseous exudates.

- A caseous plug may be found in the lower trachea or bronchi of the chicks die due to the disease.
- In affected layer birds fluid yolk material may be found in the abdominal cavity.

IB-Odema and congestion of trachea



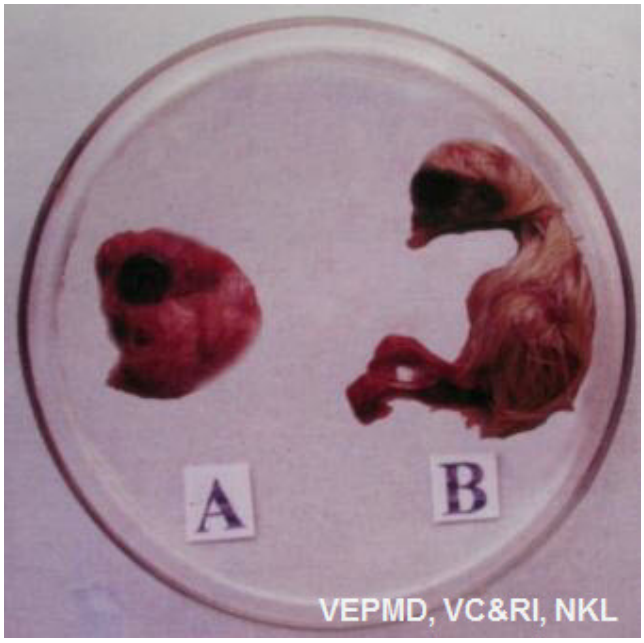
IB-Involution of ovaries



IB-Swollen and pale kidneys

DIAGNOSIS

- Based on clinical history, lesions, seroconversion of affected birds, detection of IBV antigen.
- Isolation and identification of causative agent.
- *Samples to be collected :* Trachea , Caecal tonsils
- *Virus isolation*
    - Samples for virus isolation are inoculated into embryonated chicken eggs or tracheal organ cultures.
    - Fluids should be harvested after 48-72 hours from either culture system for blind passage.



- *Serological tests*
    - ELISA, Immunoflourescence, Immunodiffusion tests, Virus neutralization, HI.

Differential diagnosis

- Newcastle Disease
- Infectious laryngotracheitis
- Infectious coryza.

PREVENTION AND CONTROL

Management procedures

- Ideal managemental procedures include strict isolation and repopulation with only day old chicks, following the cleaning and disinfection of the poultry house.
- Ventilating houses with filtered air under positive pressure

Vaccination

- Both live and inactivated virus vaccines used for IB immunization.
- Live vaccines are used in broilers and for initial vaccination of breeders and layers.
- Inactivated oil emulsion vaccines are used primarily at point of lay in breeders and layers.
- Live vaccine administered individually by eye drop, intratracheal or intranasal route.
- Mass application methods include administration of vaccine as coarse spray, aerosol and drinking water.

- Inactivated vaccines require injection of individual birds.
- These vaccines are usually given after priming with live virus and are administered a few weeks before production commences.

Time of vaccination

- Two weeks of age is frequently used as the time for initial immunization.
- Booster doses administered at 7-12 or 16-18 weeks of age.

INFECTIOUS LARYNGOTRACHEITIS

INTRODUCTION

- Infectious laryngotracheitis (ILT) is a viral respiratory tract infection of chickens that may result in severe production losses due to mortality result and decreased egg production.
- This disease is caused by gallid herpes virus 1, belonging to the sub family Alphaherpesvirinae, family Herpesviridae.
- In areas of intensive production and large concentrations of poultry such as in the United States, Europe, China, Southeast Asia and Australia ILT is usually endemic.
- The chicken is the primary natural host of this virus. Birds of all age groups are affected but this disease is mostly common in adult birds.
- Natural portals of entry for infectious laryngotracheitis virus (ILTV) are through the upper respiratory and ocular routes.
- Ingestion is also a mode of infection. Transmission occurs more readily from acutely infected birds than through contact with clinically recovered carrier birds.

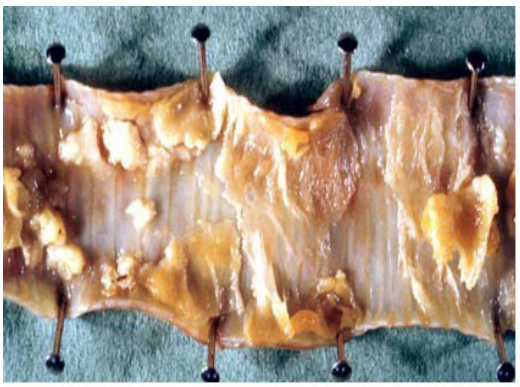
CLINICAL MANIFESTATION AND GROSS LESIONS

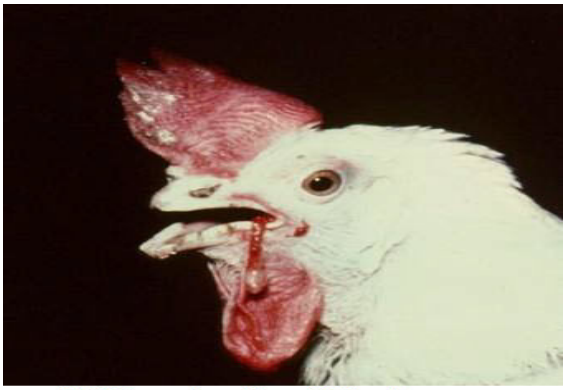
Clinical manifestations

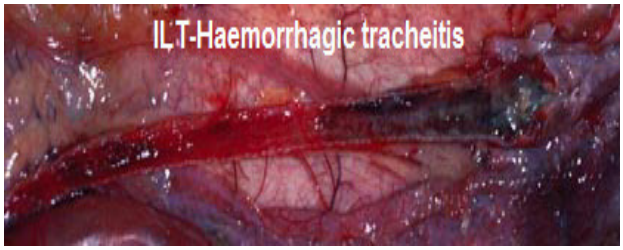
- Incubation period for this disease is 6-12 days.
- This disease causes an acute respiratory disease in chickens.
- Characteristic clinical signs include nasal discharge and moist rales followed by coughing and gasping.
- Marked dyspnea and expectoration of blood stained mucus is characteristic of severe forms of the disease.
- Clinical signs associated with mild enzootic forms include unthirftiness, reduction in egg production, watery eyes, conjunctivitis, swelling of infraorbital sinuses, persistent nasal discharge.

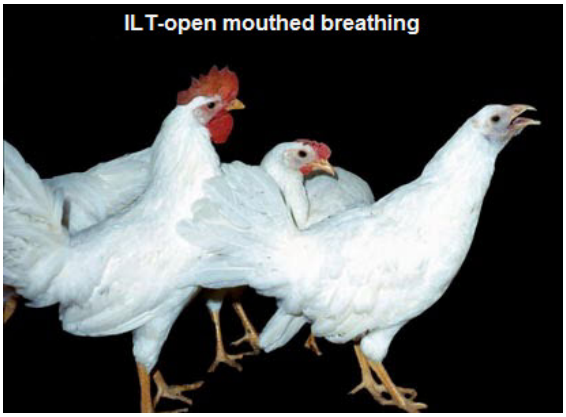
Gross lesions

- Gross lesions found at conjunctiva and throughout respiratory tract of ILTV infected chickens, but most consistently observed in larynx and trachea.
- Tissue changes include excess mucus, hemorrhagic and diphtheritic changes.


ILT-diphtheritic changes in the trachea


ILT-Expectoration of bloody mucus





DIAGNOSIS

Samples for virus isolation

- Respiratory exudates, conjunctival exudates, trachea, larynx, lungs

Method of isolation

- Inoculation of suspensions of samples into 9-12 day old embryonated chicken eggs through CAM route or onto susceptible cell cultures.
- Cell cultures of choice are Chicken embryo liver cells (CEL), and CK cells.
- A maximum of two passages in CEL and CK cell cultures required.

Other tests

- FAT
- ELISA
- IPT
- DNA hybridization techniques and PCR techniques.

CHAPTER-17: AVIAN ENCEPHALOMYELITIS AND FOWL FOX

Learning objectives

To know in detail about

- Etiology of avian encephalomyelitis and fowl pox
- Clinical manifestation of avian encephalomyelitis and fowl pox
- Diagnosis, prevention and control of avian encephalomyelitis and fowl pox

AVIAN ENCEPHALOMYELITIS

SYNONYMS AND INTRODUCTION

Synonym: Epidemic Tremor

Introduction

- Avian encephalomyelitis is an acute viral disease of very young chickens (1-21 days) characterized by nervous signs of ataxia, paralysis, tremors in head and neck.
- Avian encephalomyelitis virus (AEV) is not pathogenic to older chicken even though affected most flocks show no clinical signs of the disease other than a drop in egg production, which is transitory and passed unnoticed.
- This disease was first described in the New England states of the U.S.A. in 1932 and is now recognized worldwide.
- In addition to chicken this disease has also been recorded in pheasants, quail and turkeys.
- Avian encephalomyelitis caused by avian entero virus belongs to the family Picornaviridae. This virus is resistant to lipid solvent, acid pH, trypsin and pepsin.
- The virus is able to survive very well at ambient temperature.
- Formaldehyde fumigation is effectively inactivating AEV with an 18hrs exposure period.
- Only a single antigenic type but strains vary in virulence.
- This virus grows very well in yolk sac route of embryonated chicken eggs, chicken embryo fibroblast and chicken embryo kidney cell culture.
- This disease is an enteric infection and affected chicks shed the virus in the faeces for about 2 weeks.
- The virus is live in litter material for longer time. Horizontal spread by fomites occurs.
- Vertical transmission is a very important mode of transmission. Breeder hens get infected.

- A proportion of the eggs of infected hens are infected. Chicks infected *in ovo* hatch normally but shed virus in their droppings and infect other chicks in the incubator shortly after hatching.

CLINICAL MANIFESTATIONS

- Incubation period is 1-7 days
- Affected chicks exhibit dullness, depression, progressive ataxia, tremors in head & neck and weight loss.
- They fall on one side or sit back on their hocks or refuse to move.
- Blindness, paralysis, prostration and coma also occur.
- Death is mainly due to affected chicks not able to take feed and get trampling by pen mates.

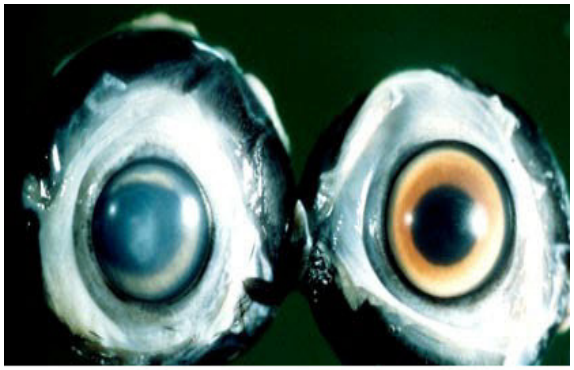
Lesions

- No characteristic lesion
- Nonsuppurative encephalomyelitis, which is, characterized by perivascular cuffing, neuronal degeneration and microgliosis.
- Large amount of lymphocytic accumulations in viscera especially in pancreas, myocardium and proventriculus are characteristic.
- Mononuclear cell infiltration in the iris leads to cataract.

Hock sitting posture-Avian encephalomyelitis

Neurological signs-Avian encephalomyelitis

Paralysis and prostration-Avian encephalomyelitis



Extensive cataract-Avian encephalomyelitis

Extensive ventriculitis-Avian encephalomyelitis

Cataract-Avain encephalomyelitis

DIAGNOSIS AND CONTROL

Diagnosis

- *Samples*: Brain is the best choice, Pancreas
- Based on clinical signs
- Inoculate suspected material into yolk sac route of chicken embryo.
- Allow the egg to hatch out and observe characteristic signs in chick for 7 days
- Serological tests- FAT, AGID, ELISA, IPT and virus neutralization test
- Embryo susceptibility test- Inoculate embryo-adapted strain into yolk sac route in embryonated eggs.
- At the age of 18 day old, all the (100%) embryos exhibit paralysis and severe muscular dystrophy
- Differential diagnosis should be made with New Castle disease

Control

- Depopulation
- Attenuated live-virus vaccine administered in the drinking water to be administered after chickens reach 10 wks of age or 4 weeks before laying-Passive immunization.
- High levels of maternal antibodies protect the chicks up to 21 days.
- Inactivated vaccine may also be used

FOWL FOX

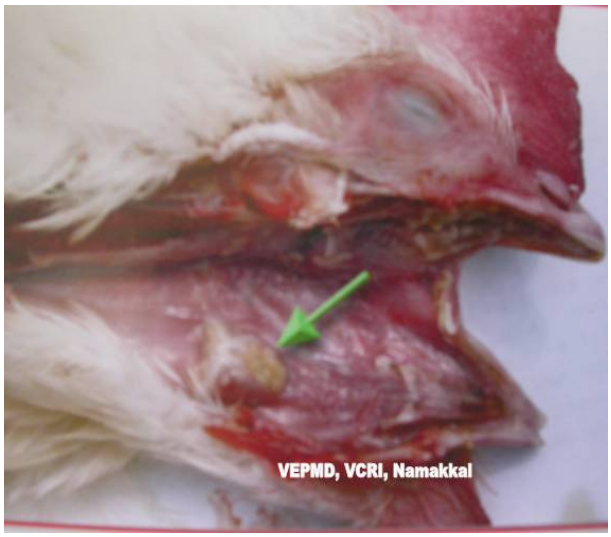
INTRODUCTION

- Fowl pox is a slow spreading disease characterized by the development of discrete, nodular proliferative skin lesions on the unfeathered parts of the body (cutaneous form) or fibrino-necrotic and proliferative lesions in the mucous membrane of the upper respiratory tract, mouth and esophagus (diphtheritic form).
- Fowl pox is a common viral disease of domestic birds and has been reported in more than 60 species of wild birds representing 20 families.
- It affects chicken of all ages, sexes and breeds.
- Fowl pox virus (FPV) belongs to the genus Avi pox, sub family Chordopoxvirinae, family Pox viridae.
- Pox virus infection occurs through mechanical transmission of the virus to the injured or lacerated skin.
- Insects mechanically transmit the disease.
- For isolation and propagation of FPV chicken embryos, and cell culture of chicken origin such as chicken embryo fibroblasts, chicken embryo dermis and kidney cells are used.

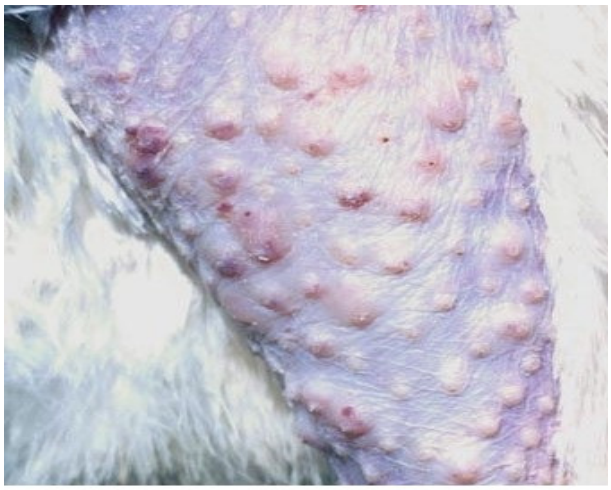
CLINICAL MANIFESTATIONS AND LESIONS

Clinical manifestations

- Incubation period varies from 4-10 days
- The disease may occur in one of the two forms such as diphtheritic or cutaneous or both
- Signs vary depending upon the susceptibility of the host, virulence of the virus, distribution of the lesions and other complicating factors
- Cutaneous form- nodular lesions on the comb, wattle, eyelids and other unfeathered areas of the body
- Diphtheritic form (wet pox)- cankers or diphtheritic yellowish lesions on the mucous membranes of mouth, esophagus or trachea with accompanying coryza like mild or severe respiratory signs when the lesions involve the trachea



Diphtheritic form of fowl pox



Multifocal follicular hyperplasia

Pox lesions on the head

- Mortality in severe cases may be as high as 50%

Lesions

- Cutaneous form- local epithelial hyperplasia involving epidermis and underlying feather follicles with formation of nodules
- Diphtheritic form- Slightly elevated white opaque nodules on the mucous membrane of the mouth, oesophagus, trachea

DIAGNOSIS, PREVENTION AND CONTROL

Diagnosis

- *Specimens:* Scabs
- Based on clinical signs

- *Identification of the agent*
    - Fowl pox should be suspected where skin eruptions occur on exposed areas.
    - Histological examination of cutaneous or diphtheritic lesions reveals epithelial hyperplasia with intracytoplasmic inclusions in affected cells.
    - Elementary bodies may be detected in smears from lesions by the use of the Gimenez method.
    - Electron microscopy of lesions will detect virus particles with the characteristic poxvirus morphology by negative staining or in ultrathin sections of the lesion.
    - The diphtheritic form of fowl pox involving the trachea must be differentiated from infectious laryngotracheitis, which is caused by a herpes virus and is characterized by the presence of intranuclear inclusion bodies.
- Virus isolation is done by inoculation on to chorioallantoic membranes of 9–12-day-old developing chicken embryos or avian cell cultures
- *Serological tests:* Immune responses to fowl pox virus may be demonstrated by the use of virus neutralisation, agar gel immunodiffusion, immunofluorescence, or passive haemagglutination tests, enzyme-linked immunosorbent assay and by immunoblotting.

Prevention and control

- No specific treatment
- Live virus vaccines-contain a minimum concentration of 10^5 EID 50 /ml to establish good immunity
- Given at 4 week old chickens and 1-2 months before egg production by wing web method.

CHAPTER-18: INFECTIOUS BURSAL DISEASE, INCLUSION BODY HEPATITIS / HYDROPERICARDIUM SYNDROME

Learning objectives

To know in detail about

- Eiology of IBD
- Host range of IBDV
- Immunosuppressive effect of IBDV
- Clinical manifestation, diagnosis and control of IBD.

INFECTIOUS BURSAL DISEASE

SYNONYMS AND INTRODUCTION

Synonyms: Gumboro disease, Avian bursitis, Infectious nephritis.

Introduction

- Infectious bursal disease (IBD) is an acute, highly contagious viral infection of young chickens that has lymphoid tissue as its primary target with a special predilection for the bursa of fabricius.
- Gumboro disease was recognized as a disease of chicken in 1957, and a virus reported in Gumboro (USA) in 1962 by Coxgrove.
- In India this disease was first reported by Mohanty *etal* (1971) in Uttar Pradesh. Chicken are the only natural hosts for the virus in which natural infection occurs.
- All breeds were affected, White leghorns more susceptible.
- Infectious bursal disease is most severe in chicks 3-6 weeks old, when the target organ, the bursa of fabricius, reaches its maximal stage of development.
- Naturally virus has special affinity for pre-B lymphocytes of the bursa of fabricius, leading to acquired B-lymphocyte deficiency in affected birds. Infectious bursal disease virus (IBDV) is a double stranded RNA virus and is a member of the Birna viridae family, genus Birna virus.
- It is inhibited by formalin and not by ether, chloroform and phenol etc. It is stable at pH-2, but inactivated at pH 12.
- There are three serotypes of the virus
- Infectious bursal disease virus replicates in both chicken and mammalian cells. Chicken embryo kidney cell culture is good for isolation.

- Cytopathogenic effect is seen in 3-5 days. Chicken embryo fibroblasts are more sensitive than fertile eggs for viral titration.

CLINICAL MANIFESTATIONS AND LESIONS

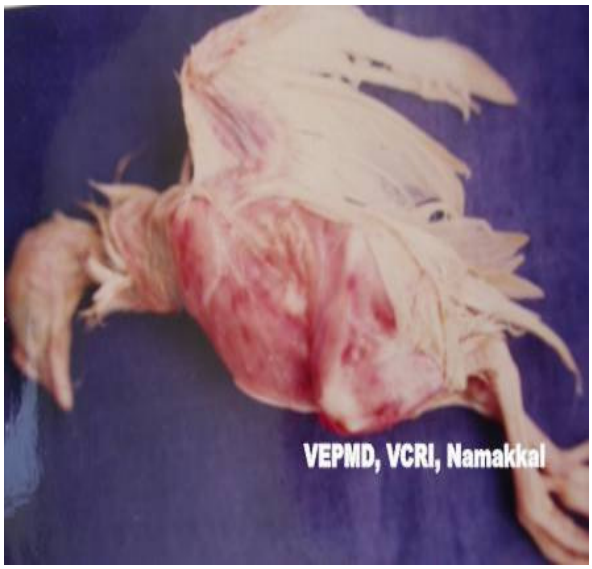
Clinical manifestations

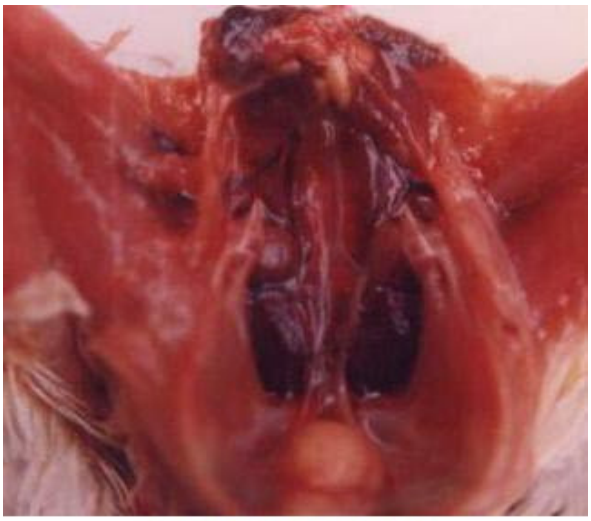
- Incubation period is very short -2-3 days
- Earliest sign – birds picking their own vents
- Soiled vent feathers, whitish or watery diarrhoea, anorexia, depression, ruffled feathers, trembling, severe prostration and death
- Affected birds became dehydrated, subnormal temperature in terminal stages
- Morbidity 100%, mortality 20-30%

Lesions

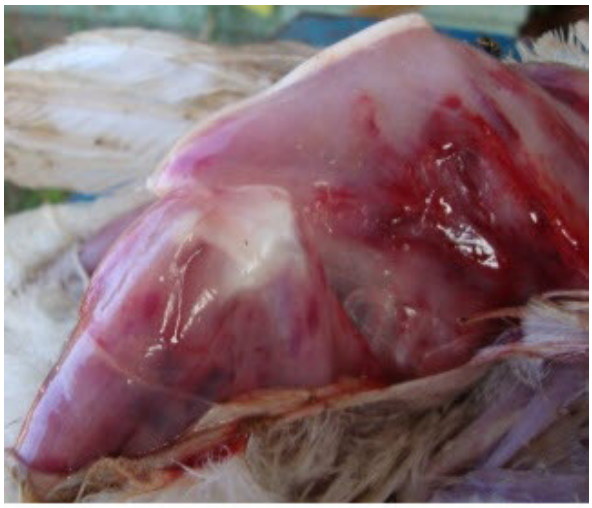
- Dehydration, hemorrhages in the leg and thigh muscles, hepatic infarcts, enlarged kidneys with pronounced tubules

IBD-Haemorrrhage in bursa

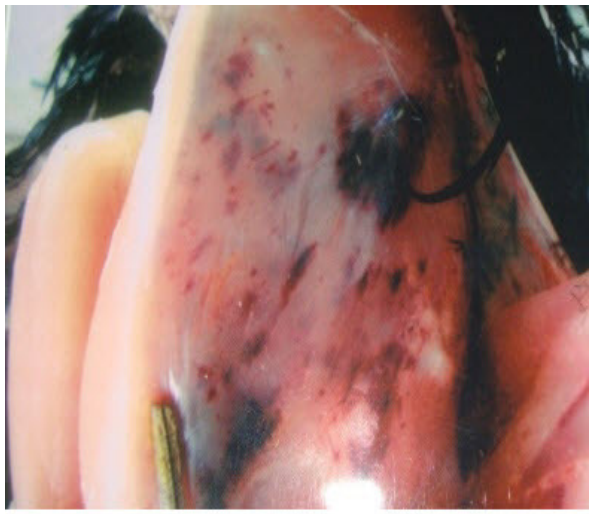




IBD-Enlarged bursa



IBD-Haemorrhage in breast muscles



IBD-Haemorrhage in breast muscle

- Bursa highly enlarged twice or 2-4 times of its normal size-approximately three days after infection and becomes edematous and hemorrhagic, atrophies by 12 days, post infection.
- Spleen slightly enlarged and mottled with deep red spots. Hemorrhagic lesions present in the proventriculus and hypertrophy of the liver is noticed.

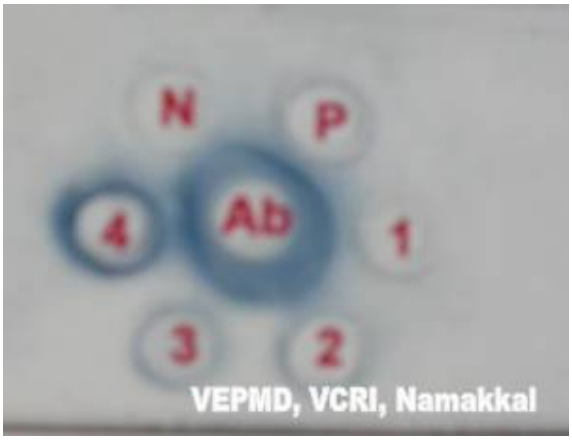
IMMUNOSUPPRESSIVE PROPERTIES

- Infectious bursal disease (IBD) infection reduces the HI antibody titers and lgG levels for Newcastle disease vaccination.
- Further humoral antibody response to various vaccines was also suppressed.
- Immunosuppressive property as been recorded in the cases of Infectious bronchitis (IB) and Infectious laryngeotracheitis (ILT) also.
- Report of suppression of CMI by IBD also has been recorded.

DIAGNOSIS

- *Specimens:* Bursa, spleen, blood
- *Based on history and clinical signs*
- *Identification of the agent*
    - Isolation of IBDV is not usually carried out as a routine diagnostic procedure.
    - Specific antibody-negative chickens may be used for this purpose, as may cell cultures or embryonating eggs from specific antibody-negative sources.
    - However, some difficulty may be experienced if using the latter two systems as the virus does not readily adapt to them.
    - If successful, the identity of the virus can be confirmed by the virus neutralisation (VN) test.

- The agar gel immunodiffusion (AGID) test can be used to detect viral antigen in the bursa of fabricius.



- A portion of the bursa is removed, homogenized, and used as antigen in a test against known positive antiserum.
- This is particularly useful in the early stages of the infection, before the development of an antibody response.
- An lmmunofluorescence test using IBDV-specific chicken antiserum can also be used to detect antigen in bursal tissue.
- Antigen-capture enzyme-linked immunosorbent assays (ELISAs) based on plates coated with IBDV-specific antibodies have also been described for the demonstration of IBDV antigens in bursal homogenates.
- The reverse transcription polymerase chain reaction (RT-PCR) with specific primers may be used to detect viral genomic RNA in the bursa of fabricius.

- *Strain characterization*
  - IBDV strains can be further identified by testing their pathogenicity in specific antibody-negative chickens, by investigating their antigenic reactivity in cross VN assays or in tests based on monoclonal antibodies, by determining the nucleotide sequence of RT-PCR amplification products derived from IBDV genome, or by studying the number and size of the restriction fragments obtained following digestion of such RT-PCR products with restriction endonucleases.
  - Tests should be performed by specialised laboratories and should include a panel of reference strains as controls.
  - Although the molecular basis for antigenic variation is now better understood, no validated virulence marker has been described yet.

PREVENTION AND CONTROL

- Maternal antibodies protect day old chicks against death and clinical IBD.
- By vaccination. Ideal age of vaccination –14 to 16 days of age by using IV95/ Intermediate plus/ MB strain and this vaccine should be repeated after one week.

- Vaccination of breeders to produce high maternal antibody level in the offspring to prevent infection during first week of age.
- Vaccination of the progeny at day old to prevent early infection.
- Long lasting and more uniform levels of maternal antibody was present in, the chicks which were from breeders receiving two live IBD vaccines and are inactivated oil emulsion IBD vaccine during the growing period.

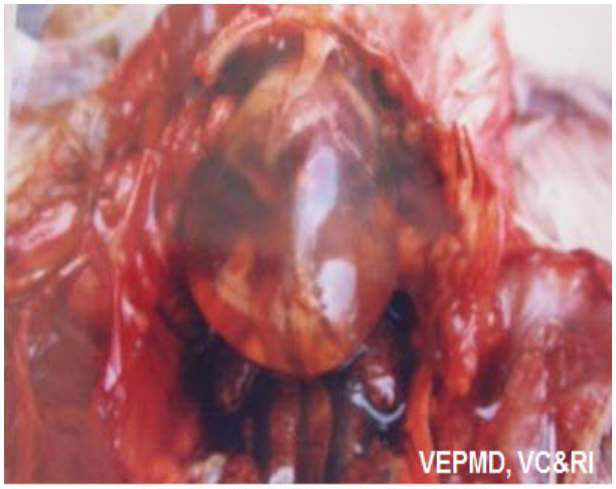
INCLUSION BODY HEPATITIS / HYDROPERICARDIUM SYNDROME

INTRODUCTION

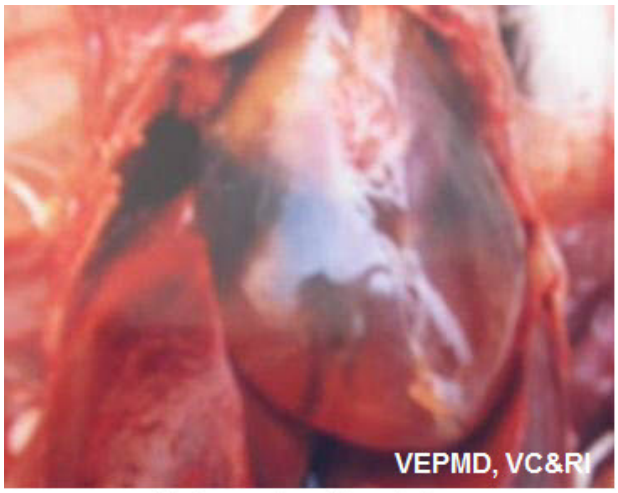
- Inclusion body hepatitis/hydropericardium syndrome is an acute disease of young chickens associated with anemia, hemorrhagic disorders, and hydropericardium.
- It is a common disease in several countries, where broilers are severely affected, resulting in high mortality rates. It is caused by avian adenovirus. Although there are 12 different serotypes of AAV, the most common viruses isolated in cases of IBH/HP belong to serotypes 4 and 8. This disease commonly affects the broiler birds of 3-6 weeks age.
- Sometimes layer and breeder pullets also affected. This disease was first reported in Pakisthan in the year 1987 and first reported in India in the year 1994.
- Horizontal and vertical transmission plays an important role in IBH/HP.
- In the breeder flocks the vertical transmission of IBH/HP reported when the progeny from breeder flocks infected with AAV serotypes 4 and 8.
- Young chicks in contact with infected chicks can die of peracute IBH/HP. Chicks and young chickens are commonly affected.
- Infection with some strains of AAV may result in minimal hepatic disease; however, the disease becomes more severe when there is combination of infection with immunosuppressive diseases such as infectious bursal disease and chicken infectious anaemia

CLINICAL SIGNS AND LESIONS

- Sudden onset of mortality , dullness, huddling together, ruffled feathers and mucoid/ yellowish dropping
- Sudden mortality usually is seen in chickens < 6 wk old and as young as 4 days of age. In chickens of < 3 weeks age mortality normally ranges from 2-40%. However, there have been outbreaks in which mortality has reached 80%. Mortality rates also vary depending on the pathogenicity of the virus and infection with other viral or bacterial agents. Signs associated with diseases caused by other pathogens (eg, bacteria, fungi, or viruses) commonly occur if birds are immunosuppressed.
- Gross lesions include up to 10 mL of a straw-colored transudate in the pericardial sac, generalized congestion, and an enlarged, pale, friable liver



Hydropericardium in broiler bird



Hydropericardium lesion

DIAGNOSIS

Virus isolation

- Virus isolation by inoculation of 10% suspension of the affected organ and faeces suspension into cell cultures such as chick embryo liver or lung cells or chick kidney cell cultures

Serology

- AGID or double immunodiffusion, IFAT, ELISA, SNT

PREVENTION AND CONTROL

- Both live and inactivated vaccines are used to control the syndrome. The AAV serotypes most frequently used to prepare commercial vaccines are serotypes 4 and 8. Sometimes autogenous inactivated vaccines are also used to ensure the transfer of maternal immunity from breeding flocks to their progeny.
- When breeders are properly vaccinated, antibodies generated by the vaccine are transmitted to the progeny, providing protection against field infections and clinical disease.
- Broilers are vaccinated at < 10 days of age when their parents either do not have serotype-specific adenovirus antibodies or maternal antibody transmission is erratic due to improper vaccination procedures that result in a substantial number of unvaccinated birds.

CHAPTER-19: AMPHISTOSOMIASIS

Learning objectives

To know in detail about

- Amphistomes of veterinary importance
- Intermediate hosts of amphistomes
- Lifecycle
- Clinical signs, diagnosis, prevention and control of amphistomiasis.

DIFFERENT SPECIES OF AMPHISTOMES

- Amphistomes are called as Stomach flukes or Conical flukes

Name of trematodes	Host	Location
Paramphistomum gotoi	Cattle	Fore stomach
Paramphisomum cervi	Cattle	Fore stomach
Cotylophoron cotylophorum	Sheep, Goat & Cattle	Rumen & Reticulum
Calicophoron calicphorum	Sheep & Cattle	Rumen & Reticulum
Ceylonocotyle streptocoelium	Cattle, Sheep & Antelope	Rumen & Reticulum
Ceylonocotyle scoliocoelium	Cattle, Sheep & Antelope	Rumen & Reticulum
Gigantocotyle explanatum	Buffalo * & Cattle	Bile duct, Gall bladder & Duodenum
Gastrothylax crumenifer	Sheep, Cattle & Buffalo	Rumen & Reticulum
Fischoederius elongatus	Cattle & other Bovidae	Rumen
Fischoederius cobboldi	Zebu cattle	Rumen
Carmyerius spatiosus	Cattle & Antelope	Rumen
Carmyerius gregarius	Cattle & Buffalo	Rumen
Gastrodiscus aegyptiacus	Equine & Pig	Large & Small intestine
Gastrodiscus secundus	Horse & Elephant	Colon
Gastrodiscoides hominis	Man & Pig	Colon
Pseudodiscus collinsi	Horse	Colon

INTERMEDIATE HOST FOR AMPHISTOMES

- Planorbis, Bulinus
- Pseudosuccinea, Fossaria
- Indoplanorbis, Lymnaea
- Pygmanisas, Glyptanisus & Cleopatra

Snails of Veterinary Importance in India

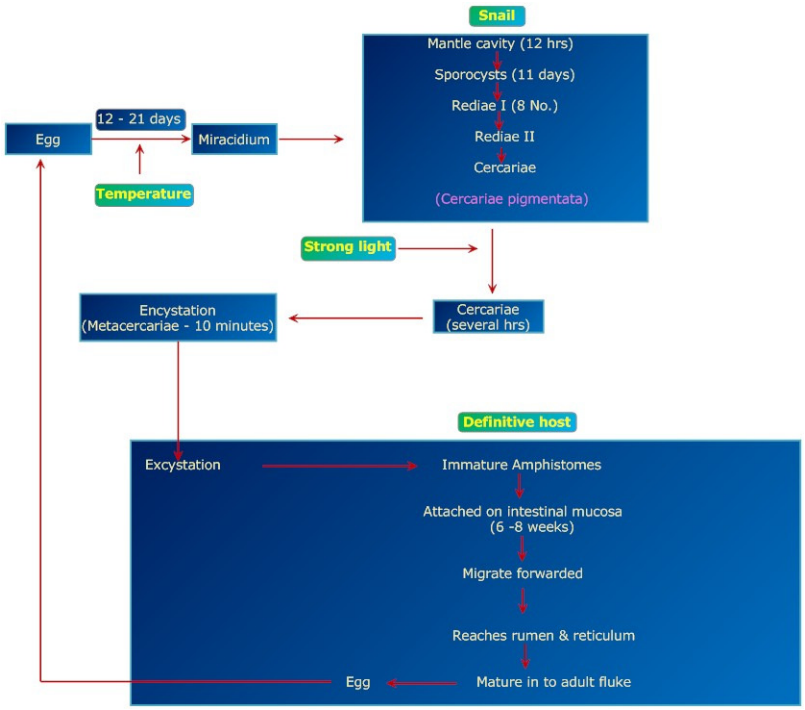
Name of the Snail	Name of trematode
Indoplanorbis exustus	*Gigantocotyle explanatum, Cotylophoron cotylophorum and Gastrodiscus secundus*
Gyraulus convexiculus	*Gastrothylax crumenifer*
Lymnaea luteola	*Fischoederius elongatus and Fischoederius cobboldi*

MORPHOLOGY AND LIFE CYCLE OF AMPHISTOMES

Morphology

- Small, conical in shape & pink in colour

Life cycle



EPIDEMIOLOGY

Factors influencing ourbreak of amphistomiasis

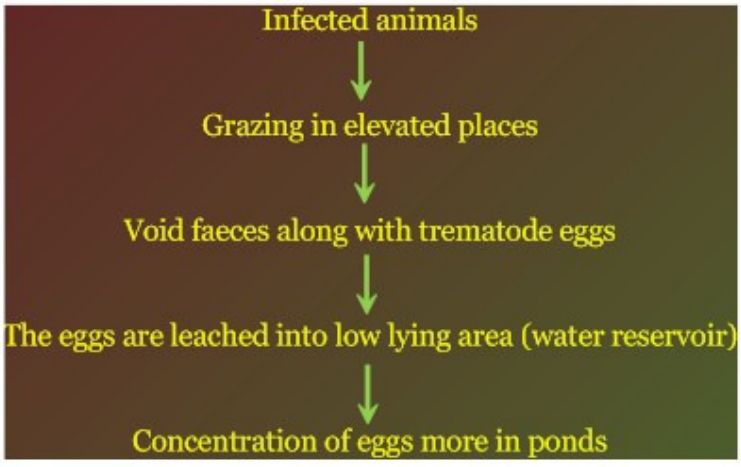
- Management
- Environmental factors
- Intermediate host
- Definitive host
- The egg contain the miracidium when passed in the faeces

Management

- Animals are reared in extensive systems of management
- Grazing near pond area/ other natural resources, especially - drier months
    - Snail population - concentrated more
    - Fresh palatable grasses are more
        - Favour for encystation of metacercariae
        - Attracts animals

Environmental factors

- Season – drier months
- High rain fall



- Temperature – embryonation (miracidium) & maturation of cercariae (27°C)
- Light - Strong light influences release of cercariae from snail

Intermediate host

- Availability of suitable snail
- Young snails are highly susceptible than adult
- Multiplication of snails take places in warm, watery environment

Definitive host

- Young animals are highly susceptible than adult
- Preinfection – give resistant

The egg contain the miracidium when passed in the faeces

- The egg do not hatch

Planorbid snails

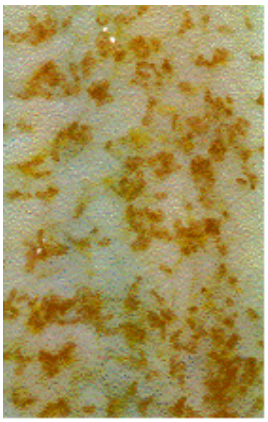
PATHOGENESIS AND CLINICAL SIGNS

Pathogenesis

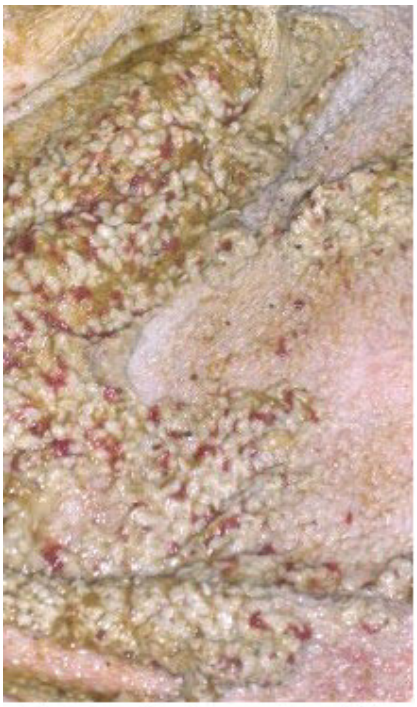
- *Adult flukes*
    - Forestomach–large number of worms–Not much pathogenic changes
    - Bile duct & gall bladder–Superficial Haemorrhage
- *Liver–Fibrosis*
    - Immature flukes (Pitto)
    - Duodenum, upper ileum-plug feeder: Plug intestinal mucosa–Haemorrhages and necrosis (Haemorrhagic duodenitis)
    - Embedded in mucosa/ muscularis

Clinical signs

- Fetid diarrhoea
- Marked weakness
- Thirsty and drink more water



Immature amphistomes in small intestine



Paramphistomes in cattle stomach

DIAGNOSIS, TREATMENT AND CONTROL

Diagnosis

- Based on history and Clinical signs
- Faecal examination
- Gross – presence of immature worms in fluid faeces
- Microscopic - Presence of typical egg

Treatment

- Bithionol sulphoxide – 40 mg/ kg (activity against Immature – 100%)
- Resorantol – 65 mg / kg (Immature – 65% &Mature – 100%)
- Niclosamide - 90 mg / kg (Immature – 99.9% &Mature – 8%)
- Niclofolan - 6 mg / Kg (Immature – 96% & Mature – 43%)
- Oxyclozanide – 18.7 mg / kg for two days (Immature & Mature - 100 %)
- Hexachlorophene – 20 mg/ kg - single dose
    - Resorantol and Oxyclozanide are the drug of choice for both immature and mature Amphistomes

Control

- Avoid grazing near pond area
- Prevent access of natural water resources – fencing of water resources
- Provide wholesome water supply at convenient places
- Drainage of water pools & swampy area
- Snail control
- Prophylactic deworming

CHAPTER-20: FASCIOLIASIS

Learning objectives

To know in detail about,

- Fasciola species of veterinary importance
- Intermediate hosts of *fasciola gigantica* and *fasciola hepatica*
- Life cycle
- Clinical signs, diagnosis, prevention and control of fascioliasis.

INTRODUCTION, MORPHOLOGY, HOST AND LOCATION

Introduction

- Fascioliasis is called as liver fluke disease or liver rot
    - *Fasciola hepatica:* Mostly found in temperate and cooler area of high altitude in tropical and subtropical countries
    - *Fasciola gigantica:* Mostly in tropical countries

Morphology

- Broad-leaf like body

Host affected

- *Natural host:* Cattle, Buffalo, Sheep, Goat and Other ruminants
- *Unusual host:* Horse & Man

Location

- *Immature flukes*: Small intestine, peritoneal cavity and liver parenchyma
- *Mature flukes*: Bile duct

Intermediate host (Amphibious snail)

- *Fasciola hepatica: Lymnaea truncatula*
- *Fasciola gigantica*
    - *L. auricularia*
    - *L. acuminata & L.rufescens* (India & Pakistan)

EPIDEMIOLOGY

Hatching of eggs

- Optimum temperature - 10 – 26°C & Relative humidity – 70%
- Summer – 21 days & Winter – 90 days

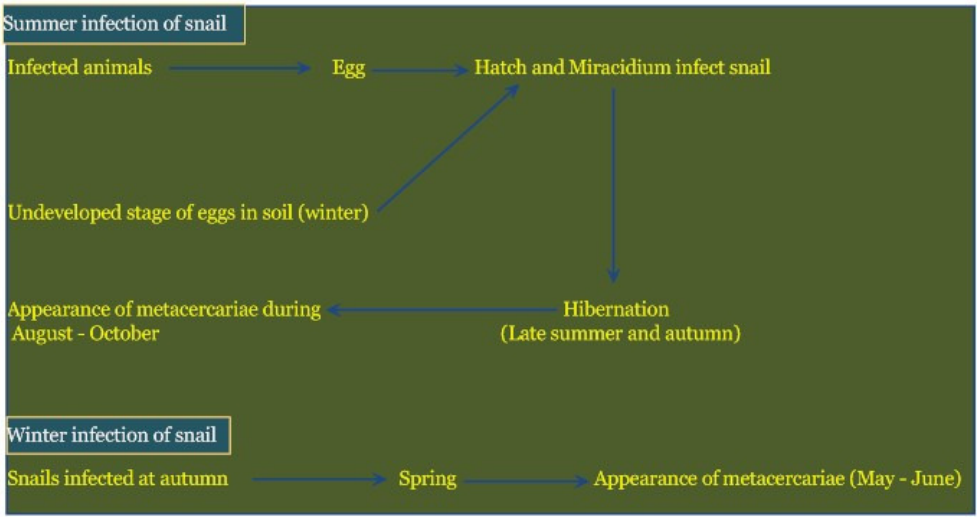
Snail

- Optimum temperature for breeding: 10 – 28°C
- Increasing in environmental temperature: Mortality of snail occurs (Infectivity falls)
- Size of snail: Size directly related with release of Metacercariae
- Moist and warm condition (mud) grow well
- Under unfavorable condition goes to Hibernation

Ecology of Lymnea

- Permanent habitat – poorly drained soils, ditches, area of seepage & spring
- Temporary habitat – Hoof marking of animals (clay soil)
- pH- slight acid pH favour growth

INFECTION OF SNAIL

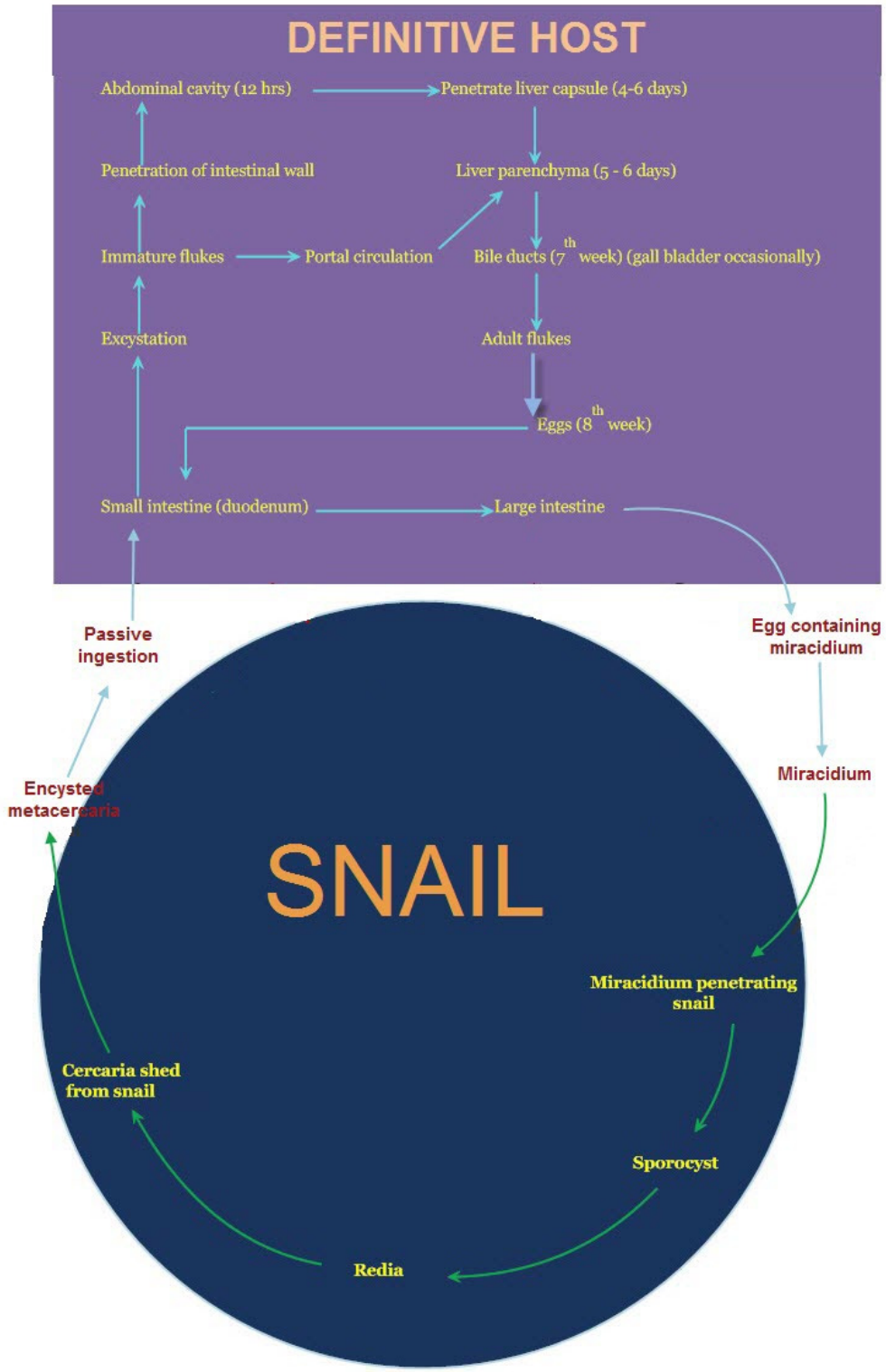


Longevity of Metacercariae

- *Natural condition:* 4-6 weeks
- *Experimental Condition:* 9 – 11 months
- *Relative Humidity:* > 70% - increases the survivability

- *Good hay making:* infectivity retained considerable period
- *Ensilage:* Survive not more than 35 – 57 days

LIFE CYCLE OF FASCIOLA



PATHOGENESIS

- Presence of worms and eggs in the intestine and peritoneal cavity: not much pathogenic changes
- Presence of worms and eggs in the liver and bile ducts: principle lesions encountered

Sheep

Acute fascioliasis

- Morbidity & Mortality High
- 6 – 8 weeks old sheep are highly affected
- Severity of disease is directly related to No. of metacercariae ingested (10,000)
- Traumatic hepatitis – migration of immature fluke aided by suckers
- Extensive destruction of liver parenchyma & marked haemorrhage
- If No. of immature flukes entered in to liver is so high – rupture of liver capsule - haemorrhage falls into peritoneal cavity

Post - mortem Examination

- Liver enlarged in size, pale, friable & fibrinous clot observed on surface
- While incision extensive - haemorrhage tracts with immature flukes
- If liver capsule is ruptured the blood clots observed in the abdominal cavity

Sub-acute fascioliasis

- If No. of metacercariae ingested is less (<10,000)
- Liver is covered by migratory tracts – infiltration of inflammatory cells
- Inflammatory cells mount immune response (not upto protective level)
- Early fibrosis
- Majority of fluke reaches bile ducts and become mature fluke – Cholangitis

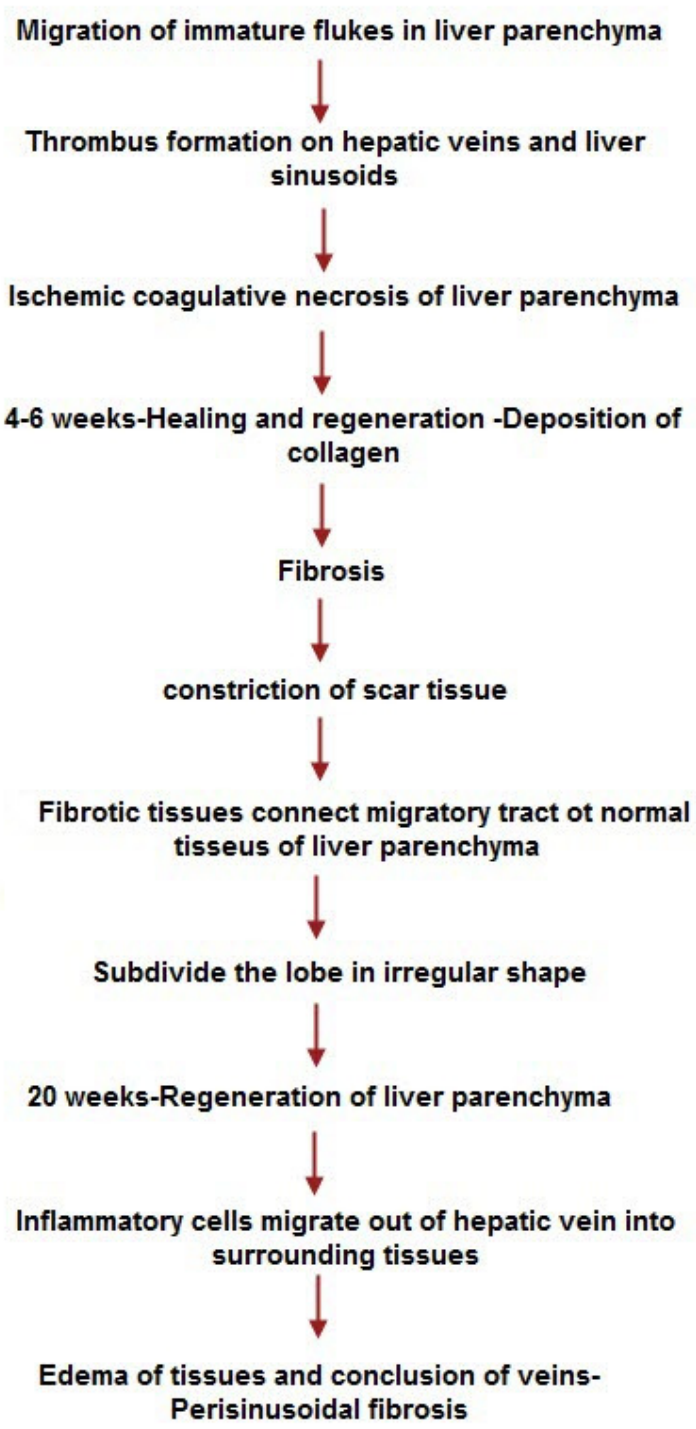
Complication

- Anaerobic condition created by migratory flukes favours multiplication of *Clostridium novyi* – **Black disease**

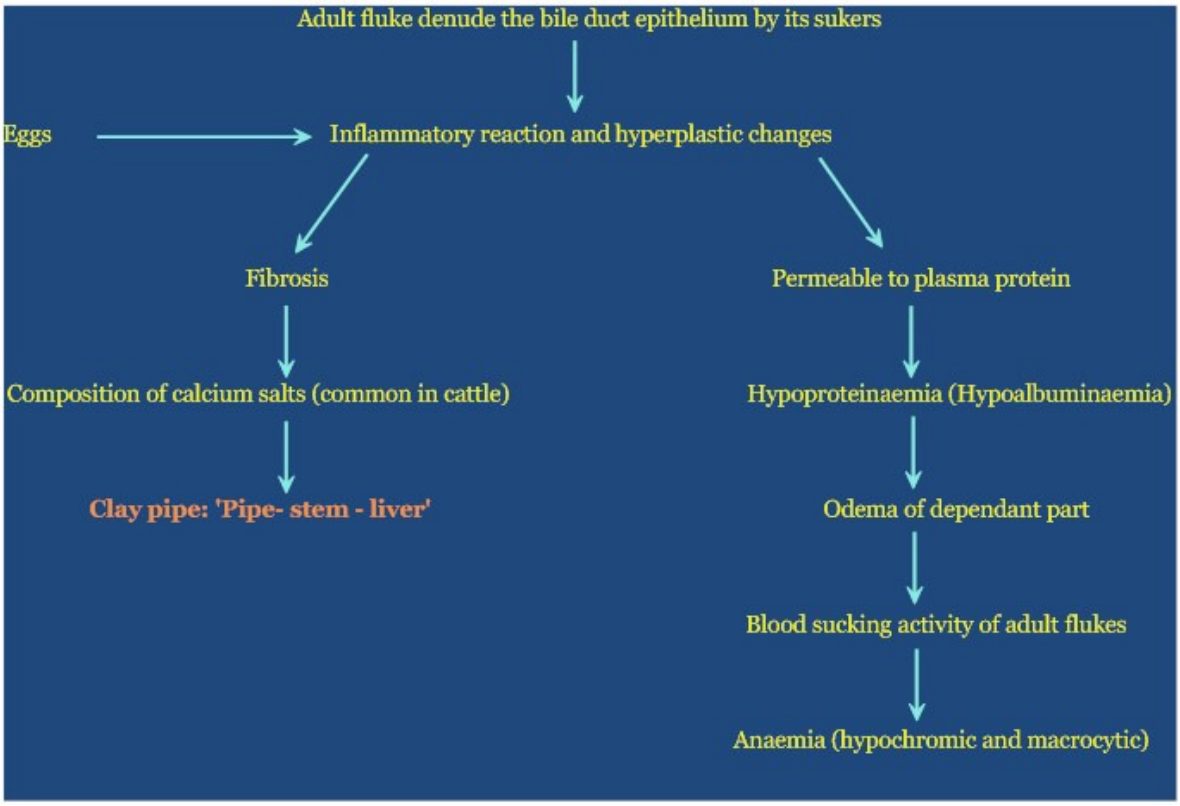
CHRONIC FASCIOLIASIS

- Most common form (ingestion of few metacercariae)
- Morbidity – 30 % and Mortality – 10 %

Hepatic Fibrosis - Immature flukes



Hyperplastic cholangitis - Mature flukes and eggs



CLINICAL SIGNS

Sheep

Acute

- Sudden death without showing clinical signs
- Blood stained frothy exudate from nostrils and anus – Resembles Anthrax

Sub acute / chronic

- Sub-acute severity is more but duration is less
- Chronic – severity is less with long course
- Intermittent soft faeces, occasionally diarrhoea
- Progressive weight loss, Inter-mandibular oedema – Bottle Jaw
- Pale mucous membrane, skin become dry and Brittle , falling out in punches

Cattle

- Similar to sheep
- Reinfection of adult pregnant cow – migration to foetus (prenatal infection)

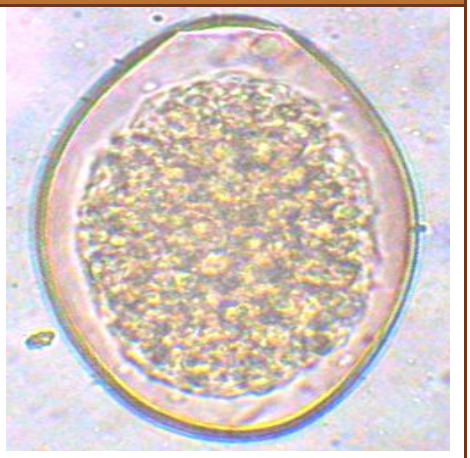
- Experimental infection of fascioliasis increase susceptibility of cattle to *Salmonella dublin* infection

Fascioliasis/ Ostertagiasis complex

- Diarrhoea is not feature of fascioliasis unless it is complicated by the presence of *Ostertagia* larva

DIAGNOSIS

- Based on clinical signs & History
- Identification of characteristic egg in faeces

Amphistome egg (*Paramphistomum cervi*)	Fasciola egg (*Fasciola hepatica*)
 Large size	 Smaller in size
Transparent shell	Yellow coloured shell
Distinct operculum	Indistinct operculum
Embryonic cells are clear	Embryonic cells are not clear
Small knob at posterior end	Knob absent

POSTMORTEM CHANGES

Acute

- Blood stained exudate – stained & rule out for Anthrax
- Liver enlarged in size, pale, friable & fibrinous clot observed on surface
- While incision extensive - haemorrhage tracts with immature flukes
- If liver capsule is ruptured the blood clots observed in the abdominal cavity

Subacute and Chronic

- Calcification of bile ducts & enlargement of gall bladder
- Cattle - Lungs – Hazel-nut sized cysts containing brownish gelatinous material in which a live/dead & calcified flukes are observed.

TREATMENT

Older drug (mostly effective against mature flukes)

- Carbon tetrachloride (oldest – 50 years)
    - Recommended for adult fluke of sheep only (not for Cattle)
- Hexachorethane – 90% Effective against mature flukes
    - Cattle – 220 to 400 mg/kg in divided doses (3-4)
    - Sheep – 20 to 30 grams / animal
- Hexachlorophene - 90% Effective against mature flukes
    - Cattle – 10 to 20 mg / kg
    - Sheep – 15 to 20 mg / kg
- Hetol – against adult flukes
    - Cattle – 125mg / kg
    - Sheep – 150 mg / kg
- Bithionol
    - Cattle – 30 –35 mg / kg (66 to 68%)
    - Sheep - 40 mg / kg (90 to 100% -chronic fasioliasis)

Recent drugs (Active against mature & immature flukes)

- Diamphenethide – 100 to 150 mg / kg (90 to 100 % for Adult)
    - Drug of choice for acute fascioliasis in sheep
- Oxyclozanide

- Cattle – 10 to 15 mg / kg (mature -100%)
- Sheep – mature - 15 to 20 mg / kg & Immature - 45 mg /kg

- Rafoxanide
  - Sheep & Cattle – 7.5 mg / kg
  - Mature - more than 90 % & Immature – 50 to 90%
- Nitroxynil
  - Sheep & Cattle
  - Mature (100%)- 10 mg / kg & Immature (90%) – 15 mg / kg
- Benzimidazole (Albendazole & Oxfendazole)
  - Albendazole – Sheep - 7.5 mg /kg & Cattle – 15 mg / kg

Latest drug (Active against mature and highly effective against immature flukes)

- Triclabendazole – 10 mg/kg body
  - Highly effective against immature flukes
  - Sheep – single dose remove all stages (over one week old)
  - Cattle – two doses remove over 4 weeks aged flukes
  - Movement of stock from the infected pasture
- Closantel - 7.5 mg/ kg

DEWORMING SCHEDULE

Sheep

- Deworming schedule for sheep in endemic areas to reduce pasture contamination
  - late April/May – adult sheep – treated for adult & late immature flukes (Triclabendazole)
  - October – entire flock with triclabendazole
  - January – entire flock drug off mature flukes (Albendazole)
- In areas having high rain fall - Additional dose on
  - June (4-6 weeks after Early April/ May dose)
  - October/ November (4 weeks after early October dose)

Cattle

- Every December and May to prevent pasture contamination on spring and winter

IMMUNITY AND IMMUNIZATION

Natural immunity

- Cattle - Reinfection of *F. hepatica* - moderate to high immunity
- Sheep - fails to develop protective immunity

Active immunity

- Cattle – Three doses of irradiated metacercariae of *F. hepatica*
- Age - 6 – 9 months
- Sheep – Not effective
- Sheep infected with *Taenia hydatigena* give immunity to *F. hepatica* but reverse not possible

CONTROL

- Control similar to Amphistomes

Control of Snail

- Physical – Improving drainages
- Chemical - Applying molluscicides
    - Copper sulphate - 1 in 1,00,000 solution for water reservoir
    - Copper sulphate powder - 10 – 35 kg / hectare
        - Applied along with sand for easy apply and Sheep are not allowed to graze until rainfall occurs
    - N-tritylomorpholine – 0.45 kg/ 680 liters / hectare
        - Time of application - Spring or mid summer
- Biological – rearing of ducks, geese and frog - infected water sources

CHAPTER-21: ASCARIS AND PARASCARIS

Learning objectives

To know in detail about

- Epidemiology, Lifecycle, Clinical signs, Prevention and Control of *Ascaris suum* and *Parascaris equorum.*

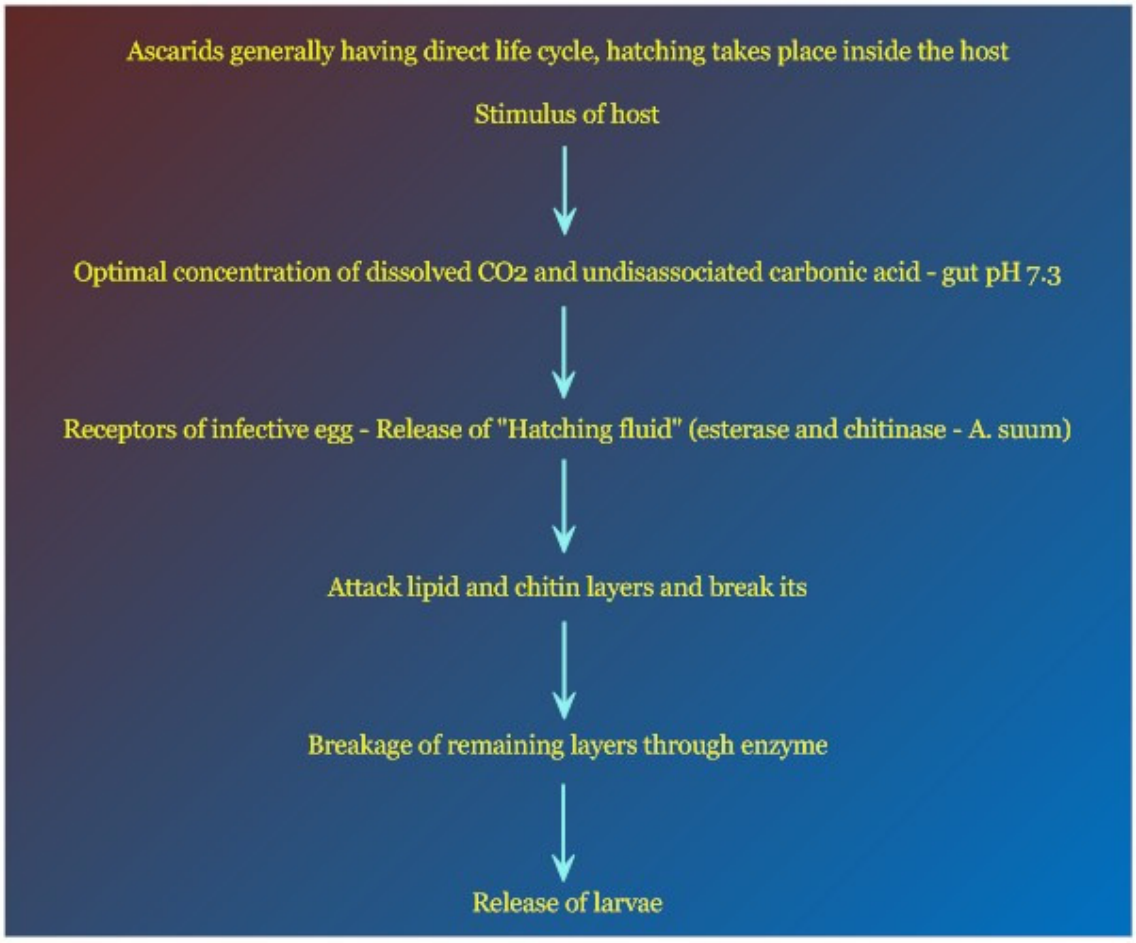
ASCARIS

GASTROINTESTINAL NEMATODIASIS

- *Ascaris: A. suum*
- *Parascaris: P. equorum*
- *Toxocara: T. canis; T. cati and T. vitulorum*
- *Toxascaris: T. leonina*

General Morphology

- *Adult:* Generally larger worms.
- *Females:* Oviparous, produce large No. of eggs.
- *Egg:* Unsegmented when laid, the eggs are oval or subglobular and the shell is in most cases thick.
    - Inner -Thin and lipid layer
    - Middle layer -Tough and chitin layer
    - Outer layer -thick and protein layer
    - Hatching of egg



ASCARIS SUUM

- *Host:* Pig
- *Morphology:* 40 cm long
    - Immature some times called as *A. ovis* occasionally found in sheep, dog and cattle
    - Egg: Albumin layer (outer) bears prominent projections "Knob" which is brownish yellow in colour
- *Life cycle:* Direct
- *Prepatent period:* 2 months
- No prenatal infection

EPIDEMIOLOGY

- Host
    - *Pigs:* Age : Young pigs (<4 months) are highly susceptible

- *Cattle:* Infection occurs due to raising of cattle on piggery shed and grazing of cattle on pasture land fertilized with infected pig manure
- *Sheep:* Especially young lambs grazed on pasture land fertilized with infected pig manure
- *Human beings:* Only few reports
- *Paratenic host:* Earth worm and dung beetle

- Parasite
  - *Infection rate:* Adult worm lay 2,00,000 - 20,00,000 eggs per day
  - Eggs contains infective larvae resist adverse conditions like freezing, disinfectant like 2% formalin - so viable upto 5 years or even more
  - Susceptible to dry hot environmental temperature (>40°C)
- Environment
  - Embryonation - optimal temp - 22 - 26°C , high relative humidity and oxygen tension
  - Cropping & ploughing - no effect on infective egg
  - *Suitable Season*
    - Temperate countries: warmer months - 22- 26°C
    - Tropical countries: warmer months - 30 - 33°C
  - Egg in the environment can be transported through wind, uncooked vegetables, fruits, etc.,

PATHOGENESIS

- Pigs
  - *Migration of larvae* - migratory haemorrhagic tracts
    - *Lungs:* Transient Pneumonia - Ascarids Pneumonia
    - *Liver:* Milk spots (observed in all age groups) - migration with molting - whitish cloudy spots up to 1 cm in diameter. Which represents fibrous repair of inflammatory reactions to the passage of larvae in liver of previously sensitized pigs
  - *Adults*
    - Presence of numerous worms in the intestine may be twisted - become solid mass which cause obstruction - perforation - peritonitis
    - Some worms wander enter into stomach and voided through vomitus
    - If enter liver through bile duct - causing biliary stasis - obstructive jaundice
- Cattle: Acute interstitial pneumonia
- Sheep: Pneumonia as well as milk spots

CLINICAL SIGNS,DIAGNOSIS AND TREATMENT

Clinical signs

In pigs

- *Signs of pneumonia* - cough - retarded growth
- Migration of *A. suum* larvae in the pig may enhances latent infections of Enzootic pneumonia (Viral) and swine influenza
- *Adult worm* - heavy infection - diarrhoea - reduced weight gain
- Breakdown of immunity in hog cholera vaccination with live virus are often attributed to this cause
- *Concurrent infection*: *A. suum* with Salmonellosis

Diagnosis

- *Based on clinical signs* - some time larvae in sputum
- *Characteristic egg* - EPG: >1000 indicative of significant infection

Treatment

- Ascarids pneumonia - levamisole - 7.5 mg / kg and Ivermectin
- Intestinal stage: Benzimidazole and Imidazole compounds are drug of choice
- *Levamisole HCl* : s/c - 7.5 mg /kg; drenching - 8 mg /kg and through feed - 0.72 g /kg of feed
- *Tetramisole Hcl* : - immature and mature, oral - 15 mg / kg and through feed 5 kg/ 500 kg of feed
- *Parbendazole* - 30 mg / kg
- *Cambendazole* - 20 mg / kg
- *Fenbendazole* - 5 mg /kg - ovicidal effect
- *Dichlorvos* - sows - 10 mg /kg & weaned pigs - 40 mg /kg
- *Morantel citrate:* weaner - 5 mg /kg and older - 12.5 mg /kg
- *Piperazine:* 100 - 400 mg /kg.

CONTROL

- *Contaminated pens:* use solutions of hot caustic soda or live steam
- *Pasture management:* rotational cultivation
- *McLean County System*
  It is most important preventive measures are those concentrated with protection of the young pigs immediately after birth or later against Ascarids infection

- Basic principles of McLean County System
  - Clean sow
  - Clean farrowing pen
  - Clean pasture
    - Few hours before furrowing, the pregnant sows are thoroughly washed and scrubbed in order to remove any egg adhering to the body and is then placed in furrowing pen
    - The furrowing pen should made up of concrete floor. The floors and walls should be thoroughly scrubbed with boiling water and caustic soda by hard broom
    - Within 10 days after furrowing the sow and it litters are removed to an Ascarids - free field, planted with a suitable crop
    - After weaning the sow is removed and the young pigs are grow up which are able to resist the major effect of infection

- *Factors impeding control measures*
  - The worms are prolific egg layers
  - The infective eggs are very resistant and long lived
  - Young animals are most susceptible

PARAASCARIS

PARASCARIS EQUORUM (P.EQUORUM)

- *Host:* Equines (Mainly) , Zebra and cattle
- *Location:* Small intestine
- *Morphology*
  - *Adult:* 50 cm long
  - *Egg:* Subglobular with a thick coarsely pitted albuminous layer
- *Life cycle:* Similar to *A. suum,* Prepatent period: 80 days
- *Epidemiology:* Similar to *A. suum*
  - *Host:* Foals: 3- 9 months old are highly susceptible
- *Pathogenesis and clinical signs*
  - *Immature: Heavy infection* - coughing and circulating eosinophilia
  - *Adult*
    - Catarrhal enteritis - diarrhoea - fetid in odour, pale in colour - flatulence
    - General malaise and debility, rough hair coat and pot bellied appearance

        - Summer cold: fever, pneumonia, coughing and whitish nasal discharge
        - Liver damage, bile duct obstruction
- *Complication*
    - Due to migratory adult worms - bile ducts
    - Penetrate bowel wall and cause localized or generalized peritonitis
    - Adults worms twisted and balled up - cause an obstruction
- Diagnosis: Similar to *A. suum*

TREATMENT AND CONTROL

Treatment

- Thiabendazole - 44 mg /kg
- Mebendazole - 10 mg /kg
- Fenbendazole - 7.5 mg / kg
- Cambendazole - 20 mg / kg
- Dichlorvos - 26 -52 mg/ kg
- Haloxon - 50 -70 mg /kg

Prophylactic Schedule

- First dose - first month of age
- Second dose - 4 - 6 weeks after the first dose

Prophylaxis

- Brood mares should be treated for Ascarids before foaling
- Boxes in which mares foal should be thoroughly cleaned before the event
- Stables should be cleaned such a way that contamination is not likely to occur
- Proper manure disposal
- Foal should be run with its mother in a clean paddock

CHAPTER-22: TOXOCARIASIS

Learning objectives

To know in detail about,

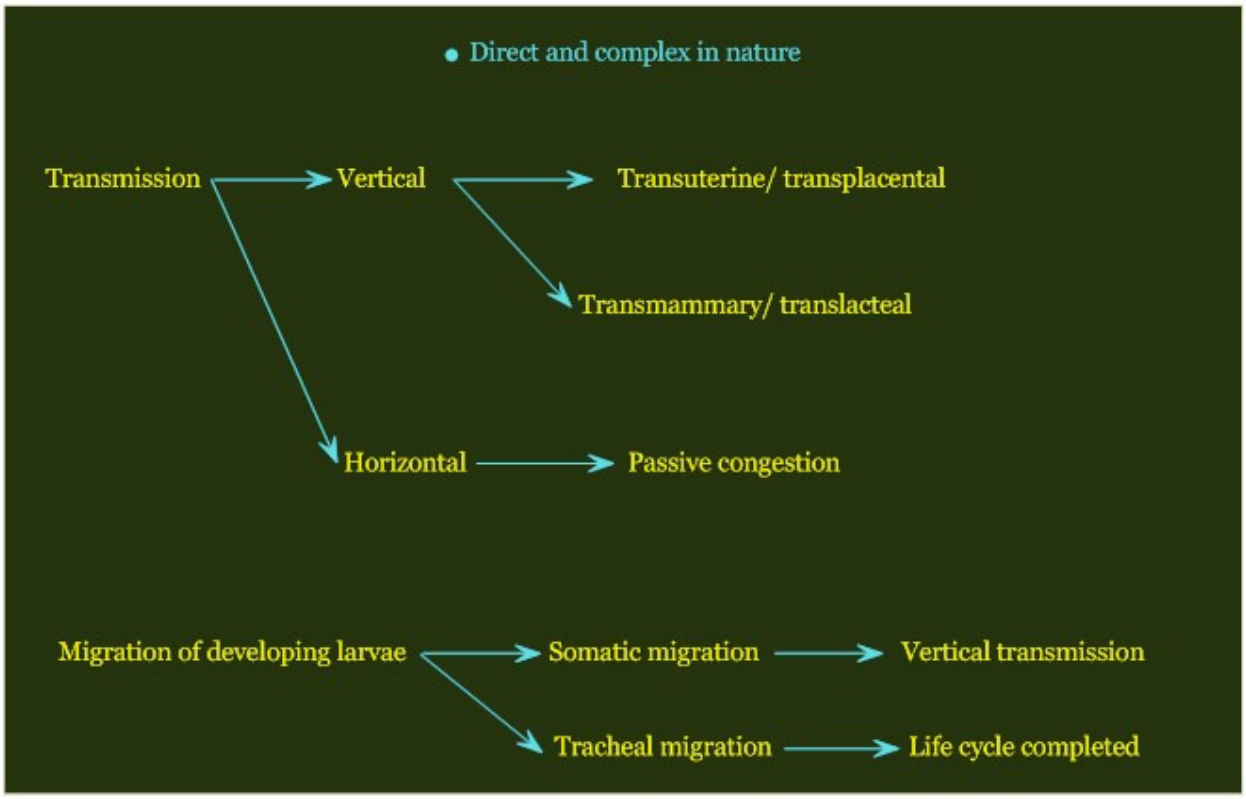
- Epidemiology, lifecycle, clinical signs, prevention and control of Toxacara canis, T.vitulorum, T.cati and T.leonina.

SPECIES, HOST AND LOCATION

Species	*T. canis*	*T. cati*	*T. vitulorum*
Host	Dog and fox	Cat and wild felids	Cattle, Zebu and Indian buffalo
Location	Small intestine	Small intestine	Small intestine

TOXOCARA VITULORUM

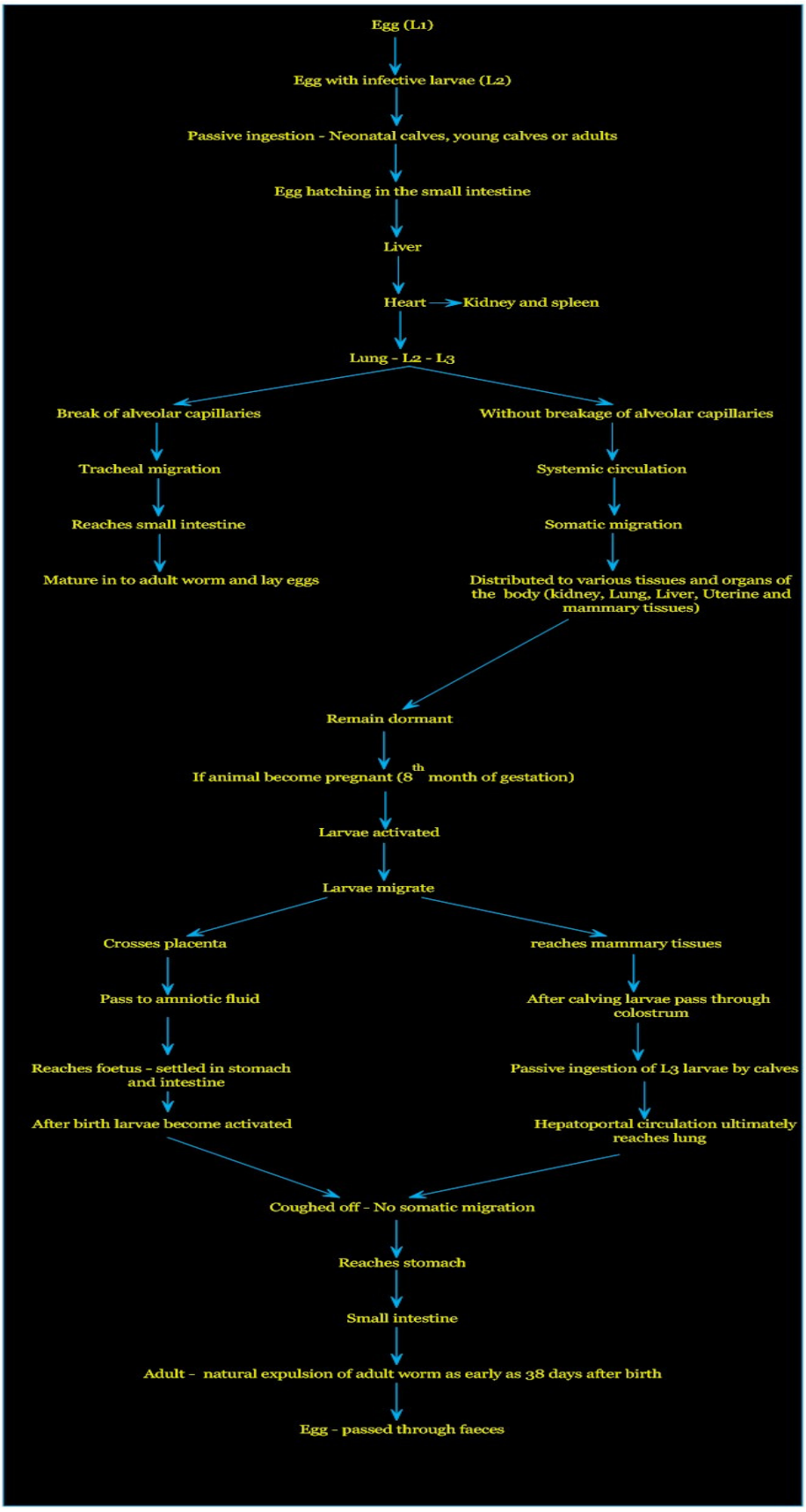
- Morphology
  - *Adult:* 30 cm in length, body does not tapers much towards extremities.
  - *Egg:* Subglobular in shape, finely pitted albuminous layer
- Life cycle and Transmission: Direct and complex in nature



Male	Age less than 4 month old	Horizontal transmission	No vertical transmission
	Age greater than 4 month old	Infection - not completion of life cycle	No vertical transmission
Female	Age less than 4 month old	Horizontal transmission	No vertical transmission
	Age greater than 4 month old	No horizontal transmission	Vertical transmission

LIFE CYCLE OF TOXOCARA VITULORUM

- Direct and complex in nature


Egg (L1)
Egg with infective larvae (L2)
Passive ingestion - Neonatal calves, young calves or adults
Egg hatching in the small intestine
Liver
Heart
Kidney and spleen
Lung - L2 - L3
Break of alveolar capillaries
Without breakage of alveolar capillaries
Tracheal migration
Systemic circulation
Reaches small intestine
Somatic migration
Mature in to adult worm and lay eggs
Distributed to various tissues and organs of the body (kidney, Lung, Liver, Uterine and mammary tissues)
Remain dormant
If animal become pregnant (8th month of gestation)
Larvae activated
Larvae migrate
Crosses placenta
reaches mammary tissues
Pass to amniotic fluid
After calving larvae pass through colostrum
Reaches foetus – settled in stomach and intestine
Passive ingestion of L3 larvae by calves
After birth larvae become activated
Hepatoportal circulation ultimately reaches lung
Coughed off - No somatic migration
Reaches stomach
Small intestine
Adult - natural expulsion of adult worm as early as 38 days after birth
Egg - passed through faeces

EPIDEMIOLOGY

- Host
    - Cattle, Zebu and Indian buffalo, sheep, and goat are also susceptible
    - Buffalo calves are highly susceptible, due to prenatal and neonatal infection
    - Larvae present in dams milk up to 30 days after parturition
    - If no tissue migration - prepatent period: 3 - 4 weeks
    - Pregnant cows body tissues acts as a reservoir of larvae for its offspring (features is the reservoir of larvae in the tissues of cows with subsequent milk borne transmission, ensuring that calves are exposed to infection from the first day of life).
- Agent: Similar *to A. suum*
- Environment: Similar to *A. suum*
    - *Infection rate:* adult worm lay 80,00,000/ day.

PATHOGENESIS, CLINICAL SIGNS, DIAGNOSIS AND TREATMENT

Pathogenesis

- Light infection - less than 70 larvae / calf - infection unnoticed
- Heavy infection - 70 -500 larvae / calf - clinical disease
- Lung: Verminous pneumonia – haemorrhagic & necrotic foci

Clinical signs: Calf hood mortality

- Diarrhoea - steatorrhoea - mud coloured evil smelling faeces
- Colic - signs resembling intestinal obstruction

Diagnosis

- Based on history and clinical signs
- Identification of characteristic eggs

Treatment

- Benzimidazole compounds
- Morantel citrate - 10 mg / kg -mature and immature
- Levamisole - 7.5 mg / kg - mature and immature
- Piperazine - 250 mg / kg

Control

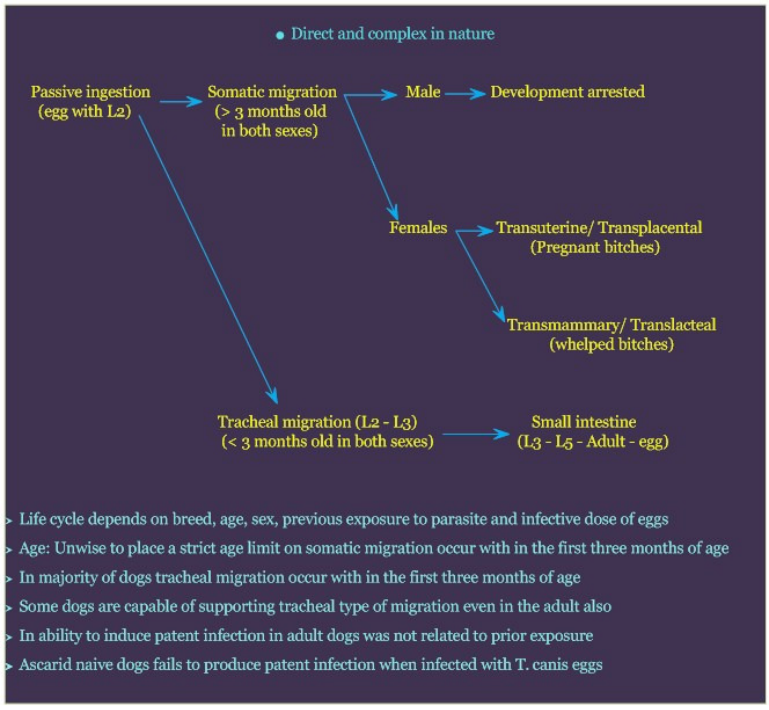
- The prevalence of infection can be drastically reduced by treating of calves at 3 and 6 weeks of age preventing developing worms reaching patency.

Prophylactic Schedule: (Buffalo calf)

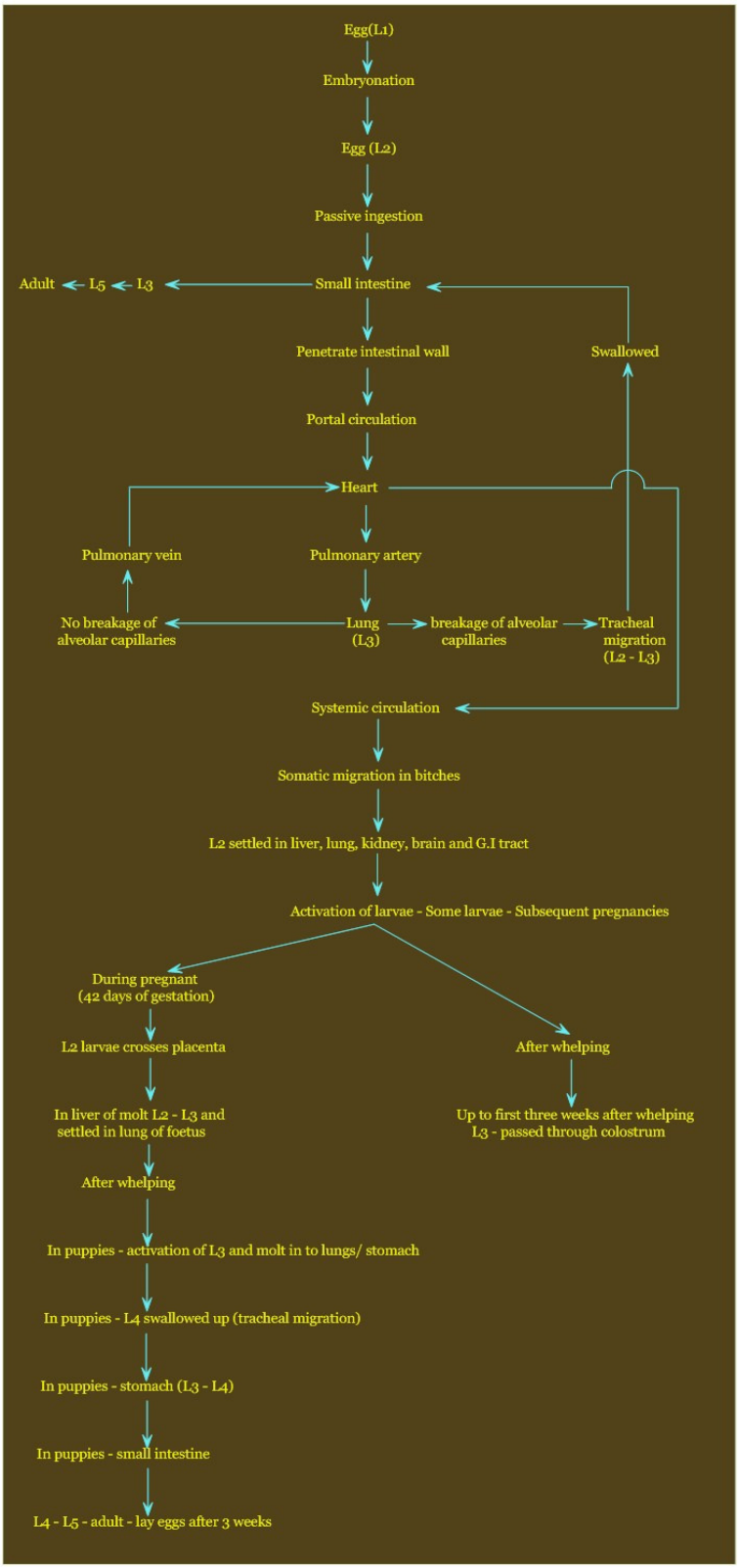
- First dose - 15th days of age
- Second dose - 30th days of age
- Third dose - 3rd month of age
- Fourth dose - 6th month of age
- Fifth dose - one year of age

TOXOCARA CANIS

- Species: Toxocara canis
- Host: Dog and fox
- Location: Small intestine
- Morphology
    - *Adult:* male – 10 cm and female –18 cm
    - *Egg:* Similar to *T. vitulorum* (egg is dark brown and thick pitted in albuminous layer)
- Life cycle: Direct – complex in nature



LIFE CYCLE OF TOXOCARA CANIS



WEAKENING OF IMMUNITY (PPR)

- Occurrence of eggs in faeces of bitches after whelping as a result of weakening of immunity and contaminated environment.
- Lactogenic hormone (prolactin), which permits mobilization of larvae from somatic tissues to small intestine through lung, larvae under go maturation and become adult.
- Habit of whelped bitches to eat faeces of puppies, which may contains immature worms. The immature worms enter small intestine of bitches and become adult.
- Post parturient infection are eliminated within a few weeks of the termination of lactation
- Not all larvae are mobilized during pregnancy and some may remain under in the tissues and eliminated through subsequent pregnancies.
- The duration of larvae in bitches may be long and animals infected for upto 385 days and is being capable of transmitting infection to puppies.
- Factors which induce mobilization and migration are unclear but probably there is a hormonal basis involved.

EPIDEMIOLOGY

- Host
    - Dog and fox
        - Puppies less than six months are highly susceptible
        - Paratenic host: Rodents, birds – ingest egg with L2 larvae, L2 larvae settled in tissues of paratenic host. Definitive host pickup infection while ingestion of paratenic host infected with L2 larvae.
        - Recent evident that bitches may be reinfected during late pregnancy or lactation leading directly to transmammary infection of the suckling pups.
        - Somatic tissues of bitch constant reservoir of infection to puppies as well as adults and unsusceptible to most anthelmintics
- Agent and environment: similar to *A. suum*
    - Females are highly fecund - lay 200 eggs / worm / gram of faeces / day (EPG- 15,000)
- Prepatent period
    - Ingestion of eggs with L2 - without somatic migration: 3- 4 weeks.
    - Ingestion of paratenic host with L2: 4 –5 weeks.
    - In puppies due to prenatal infection: 3 weeks

PATHOGENESIS AND CLINICAL SIGNS

Pathogenesis

- *Puppies*
    - Heavy infection in puppies - death of whole litters
    - Migration of larvae through lung in new born puppies - pneumonia (uncommon) and pulmonary oedema, haemorrhage and eosinophilia
    - In infected puppies fed with milk of food, causes vomition and leads to inhalation pneumonia
    - Death occur in 2- 3 weeks old puppies
    - Mucoid enteritis, intestinal ulcers, perforation of intestine and peritonitis
    - Occlusion of gut - perforation – peritonitis
    - *Aberrant site:*
        - Bile duct – inflammatory changes - occlusion
        - CNS – nervous disorder
- *In Adult*
    - Progressive malaise associated with vomition and diarrhoea as worm mature it
    - stomach and small intestine.

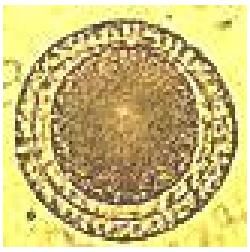
Clinical signs

- Migratory larvae - pneumonic signs - coughing, increased respiratory rate, frothy nasal discharge
- Adult – diarrhoea, pot bellied and tucked up abdomen and rough hair coat.
- Aberrant site CNS - nervous conclusion
- Death due to intestinal obstruction
- Most fatalities from *T. canis* infection occur during pulmonary phase and pups which have been heavily infected transplacentally and may die within a few days of birth.

DIAGNOSIS,TREATMENT AND CONTROL

Diagnosis

- Based on history and clinical signs
- Demonstration of eggs on direct smear method




Treatment

- Piperazine - mature worm - 100 mg/ kg and immature worm - 200 mg /kg
- Diethycarbamazine (DEC) - 50 mg/ kg (contraindicated for dogs with patent infection of *D. immitis*)
- Dichlorvos – 12 – 15 mg/ kg body weight
- Trichlorophan - 75 mg/kg
- Pyrental pamoate – 5 mg/kg
- Nitroscanate - 50 mg/kg
- Mebendazole - 10 mg/ kg (twice a day for 2 days)
- Fenbendazole - 100 mg/ kg (As single dose or divided doses) (useful for pregnant bitches)

Control

- Good hygienic maintenance in kennels.
- More over long term control consist of regular treatments for adult worm to lower or to eliminate environmental contamination.
- Kennels should be made up of impervious surfaces and floor should be thoroughly cleaned.
- To control entry of rodents in kennels.
- Prenatal and neonatal infection of young puppies are controlled by treating of pregnant bitches and her litter with suitable prophylactic schedule as follows.

PROPHYLACTIC SCHEDULE

Puppies		Bitches	
Time schedule	**Purpose**	**Time schedule**	**Purpose**
-	-	Pregnant bitches 3 weeks before whelping to 2 days after	To eliminate transplacental and transmammary infection

		whelping - daily high doses of fenbendazole	
2nd weeks of age (I dose)	To eliminate prenatal infection	Same time when pups are treated	To eliminate transmammary infection
4th to 5th week of age (II dose)	To eliminate prenatal infection	Same time	To eliminate transmammary infection
2nd month of age (III dose)	Neonatal infection through milk or from environment	-	-
Once in every 3 – 6 months	Routine prophylatic	Once in every 3-6 months	Routine prophylatic

TOXOCARA CATI

- Host: cat and wild felids
- Location: small intestine
- Morphology
    - Male - 6 cm and female - 10 cm long
    - Egg: Similar to *T. canis* (Colour less thick pitted albuminous layer)
- Life cycle: Direct
    - Prenatal infection does not occur
    - Paratenic host involves in the life cycle – Earth worm, cockroaches, chicken, sheep and other animals fed with infective eggs
    - Transmammary only occurs
- Paratenic host: Rodents with L2
    - Transmammary: Infection in kittens is derived from the milk of infected queens.
    - Larvae occur in the milk throughout lactation when queens infected with eggs and transported to the mammary gland after on extended period in other tissues before lactation commences.

- Pathogenesis and clinical signs
    - Pot belly, diarrhoea and poor hair coat
- Diagnosis and treatment: Similar to *T. canis*
- Control
    - Removal of kitten from infected dams
    - Similar schedule as that of *T. canis*

TOXASCARIS LEONINA

- Host: Dog and cat
- Location: Small Intestine
- Paratenic host: Mice
- Morphology
    - Male – 7 cm and female – 10 cm
    - Egg: Slightly oval with stomach thick shell
- Life cycle: Direct – but not complex
    - Egg (L2) —> Passive ingestion —> Small intestine —> L2 —> Adult -> eggs
- Pathogenesis and clinical signs
    - Pot bellied, intermittent diarrhoea and possibly anemia
- Diagnosis and treatment: Similar to *T. canis*
- Control
    - Hygienic maintenance
    - Prevent the exposure
    - Prevent capturing of paratenic host.

CHAPTER-23: OXYURIS, HETERAKIS, ASCARDIA AND SUBULURA

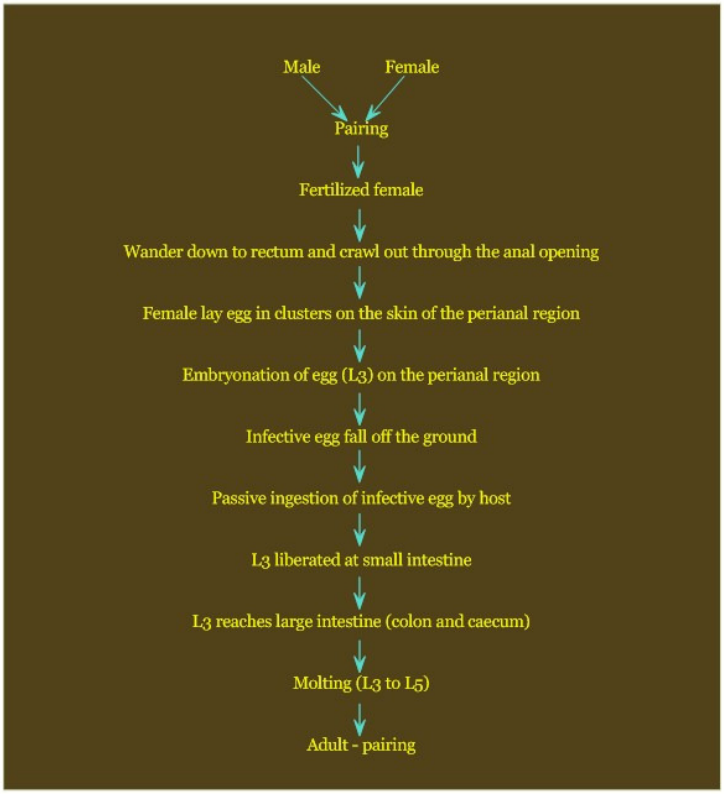
Learning objectives

To know in detail about

- Epidemiology, Lifecycle, Clinical signs, Prevention and Control of *Oxyuris equi*, *Heretakis gallinarum*, *Ascardia galli* and *Subulura brumpti*

OXYURIS EQUI (PIN WORM OF EQUINES)

- *Host:* Horse
- *Location:* Caecum and large colon
- *Morphology*
  - Male – 9 – 12 mm and female – 15 mm long
  - Young females - whitish in colour, slightly curved body with pointed tail
  - Mature females – slightly gray or brownish in colour, narrow tail and tail is more than three times as long as the body
  - *Egg:* Elongated slightly flattened on one side, provided with a plug at one pole
- *Life cycle:* Direct

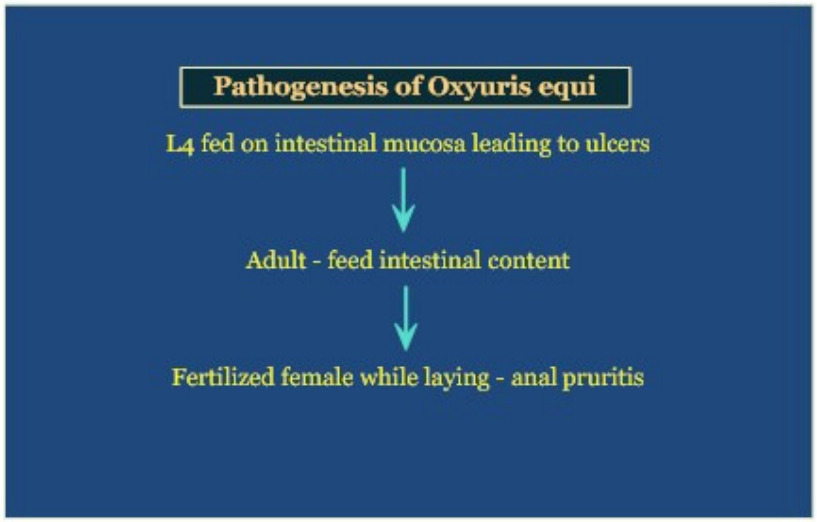


EPIDEMIOLOGY, PATHOGENESIS AND CONTROL

Epidemiology

- *Host*
  - Equines – horse, donkey
  - Young ones are highly susceptible
- *Agent:* Dispersion of egg while rubbing the perianal region by tail
- *Environment*
  - Embryonation takes place on the body surface of the host (perianal region)
  - Embryonated egg fall on the ground and contaminate bedding material and survive for several weeks under moist condition
  - Infection: By ingestion of fodder along with contaminated bedding material

Pathogenesis



Clinical signs

- Restlessness, feeding affected, loss of condition and dull hair coat
- Due to anal pruritis —> Animal rub the base of the tail against hard objects —> hairs to break off tail become ungroomed **"rat tailed"** appearance

Diagnosis

- Based on clinical signs
- Perianal region –creamy colorless mass –exam under microscope

Differential diagnosis

- Mange infection

Treatment

- Mebendazole - 5-10 mg/kg body weight
- Cambendazole – 20 mg/kg body weight
- Dichlorvas - 26- 52 mg/kg body weight

Control

- Good hygiene
- Infected bedding material should be removed
- Clean supply of water

HETERAKIS GALLINARUM

Species

- *H. gallinarum* - Fowl
- *H. isolonche* - Pheasant , fowl
- *H. beramboria* - Chicken
- *H. indica* - Chicken

Heteraks gallinarum

- *Location:* Caeca
- *DH:* Fowl, G. fowl, pea fowl, turkey, geese and other birds
- *Transport host:* Earth worm (egg to L2)
- *Morphology*
    - Female: 10 to 15 mm long
    - Egg: Thick, smooth egg shell covered with unsegmented yolk material, ellipsoidal
- *Life cycle:* Direct
- *Epidemiology:* Egg present in the environment for long period
- *Pathogenesis and clinical* signs
    - Thickening of mucosal epithelium with numerous petechial hemorrhage
    - *H. gallinarum* egg carry protozoa *Histomonus meleagaridis* and cause Black head.

    - Species: *H. isolonche* and *H. beramboria* – nodular typhilitis - diarrhoea, wasting emaciation and death
- *Diagnosis:* Faecal examination
- *Treatment*
    - Phenothiazine - 1 g/ bird – drug of choice
    - Piperazine - less effective
    - Phenothiazine and Piperazine (7:1) – 1 g/bird
    - Hygromycin B – 0.25 % minimum 2.5 kg / tonnes
    - Mebendazole - 2 kg / 28 kg feed
    - Tetramisole - 10 % solution in drinking water
    - Haloxon
- *Control:* Strict sanitation

ASCARIDIA

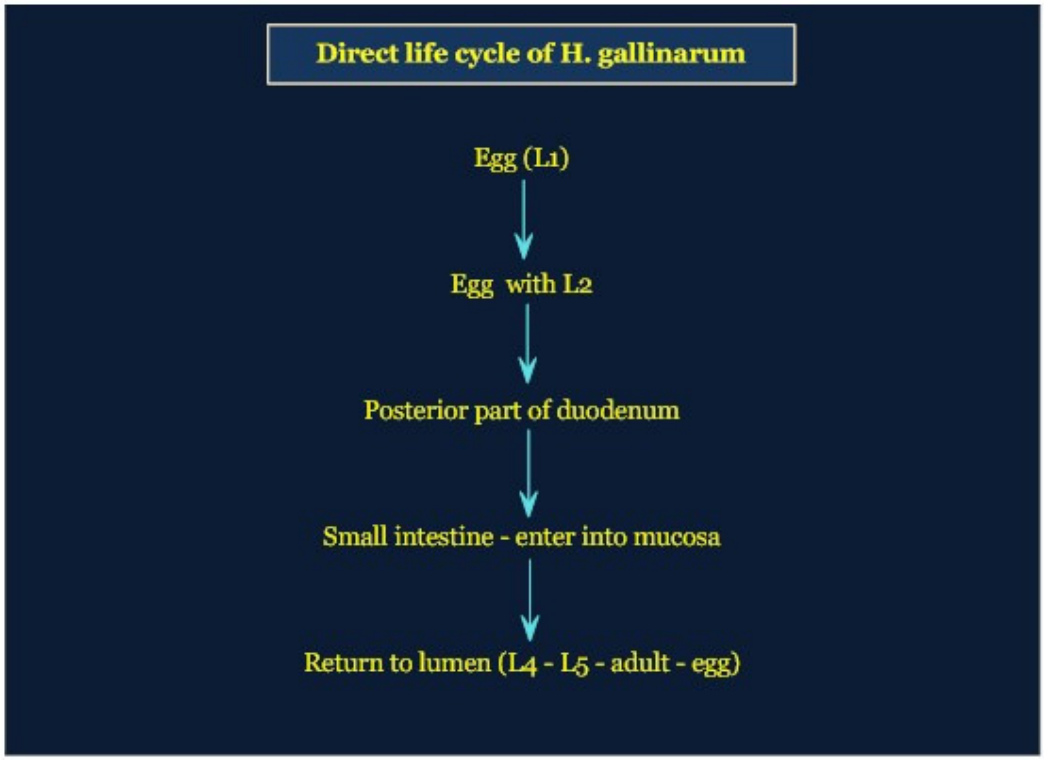
Species

- *A. galli*
    - Host: Fowl, G. fowl, Turkey, geese
    - Location: Esophagus, proventriculus, gizzard, oviduct, small intestine and body cavity
- *A. columbae*
    - Host: Pigeon (Domestic and wild)
    - Location: proventriculus, gizzard, small intestine and liver
- *A. razia*
    - Host: Domestic pigeon
    - Location: Small intestine

Morphology

- Ascardia - Largest nematode of poultry
- Female: 72 – 116 mm & Male: 50 – 76 mm

Life cycle



- Prepatent period: 5 –6 weeks
- Transport host: earth worm and grasshopper

Epidemiology

- *Following environmental factors which favours increases infection rate*
    - Deep litter – excess moisture with shades
    - Feeding and water - uncleanness
    - Moist and cold weather
- *Host*
    - Young birds are most susceptible (less than 3 months old - increased goblet cell)
    - High incidence with previous exposure
    - Dietary deficiences of Vit. A & B, Minerals and protein predisposes to heavy infectiion

PATHOGENESIS, CLINICAL SIGNS, TREATMENT AND CONTROL

Pathogenesis

- Duodenal mucosa – Haemorrhagic enteritis by larvae
- Diarrhoea, anemia and intestinal obstruction by adult leads to perforation and peritonitis

Complications

- *A. galli* + coccidiosis / infectious bronchitis - synergistic effect
- *A. galli* - transmit avian reovirus
- Adult worm present in oviduct – *A. galli* incorporated in the hens egg – it is easily identified while candling of egg - reduces egg value

Clinical signs

- Unthrifty, marked emaciation, general weak and decreased egg production

Diagnosis

- Identification of characteristic egg

Treatment

- Piperazine adipate -300 – 440 mg / kg of feed
- Piperazine citrate – 440 mg / liter of drinking water
- Phenothiazine – 2200 mg / kg of feed
- Mebendazole - 2g /28 kg of feed
- Tetramisole - 10 % solution
- Hygromycin B – 8 g/ ton of feed

Prophylaxis

- Young birds special attention -young and adult birds are separated
- Feeder and waterer should be clean and add dry litter around it
- Proper ventilation - keep the litter dry
- Litter management – heaping for several days - heat generation -detrimental to egg

SUBULURA

Species

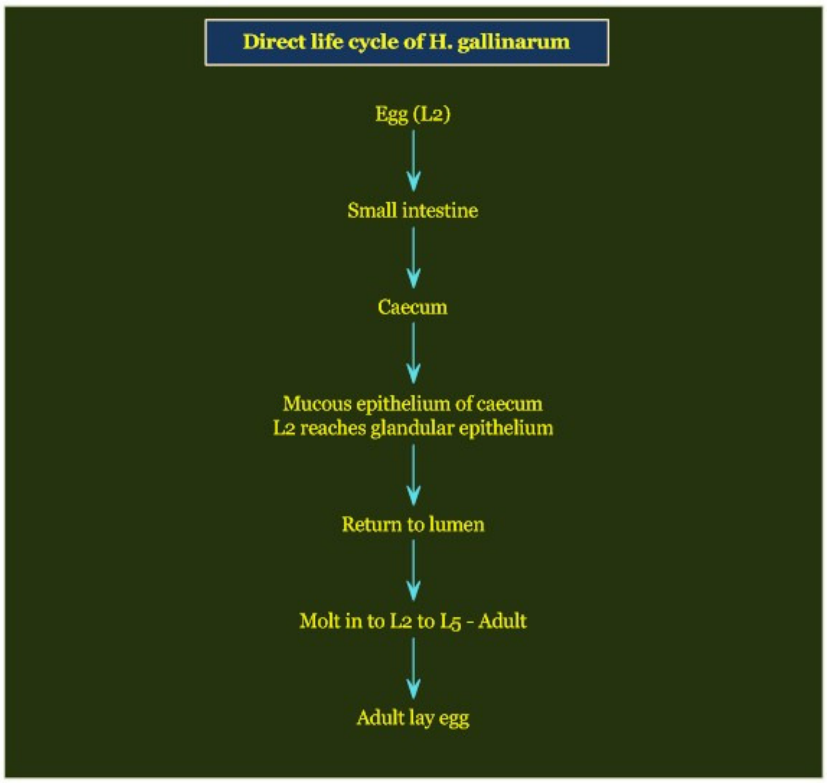
- *S. brumpti* - chicken, turkey, Guinea fowl
    - Location: Caecum
- *S. minetti* – Chicken
    - Location: Caecum

Morphology

- Male - 10 mm and female - 17.5 mm long
- *Egg:* Subglobular, smooth shell with fully developed embryo

Life cycle

- Indirect
- Intermediate host: Beetles and cockroaches



Pathogenesis

- Not important

CHAPTER-24: STRONGYLOIDIDAE AND STRONGYLIDAE

Learning objectives

To know in detail about

- Epidemiology, Lifecycle, Clinical signs, Prevention and Control of *Strongyloididae spp*., S*trongyles spp*. and O*esophagostomum spp.*

STRONGYLOIDIDAE

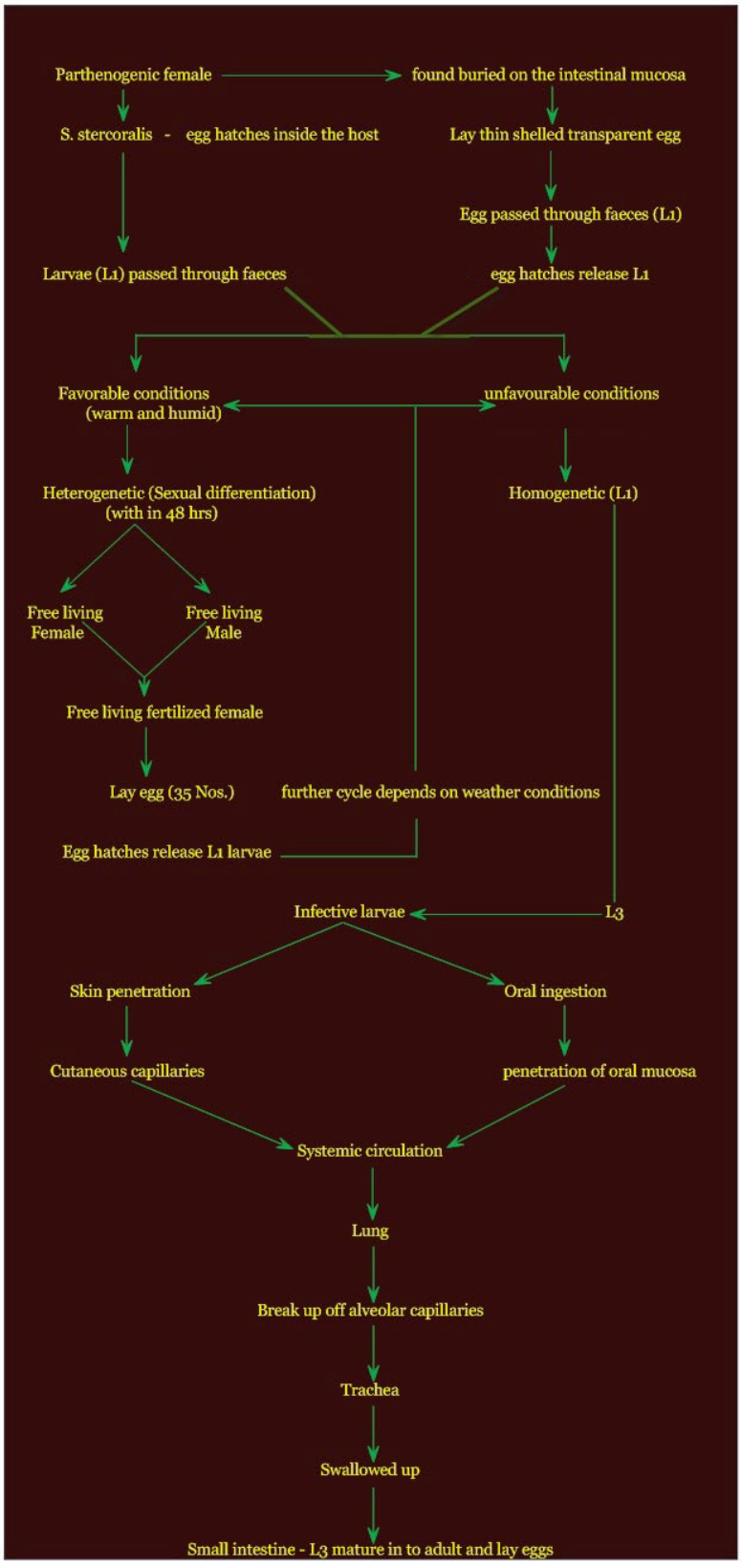
STRONGYLOIDES

- Family Strongyloididae comprises of free living, parasitic and both forms
- Parasitic forms – parthenogenetic in nature
- Species
    - *S. papillosus* - Sheep, goat, cattle, rabbit and wild ruminants
        - Location: small intestine
    - *S. westeri* - Horses, pig and zebra
        - Location: small intestine
    - *S. stercoralis* - dog , cat and man
        - Location: small intestine
    - *S. cati* – cat
        - Location: small intestine
    - *S. ransomi* - pig
        - Location: small intestine
    - *S. avium* - poultry
        - Location: small intestine
- Morphology
    - Egg: Blunt ends and thin shells, contain fully developed embryos when passed in the faeces of host
- Epidemiology
    - *Agent*
        - Parasitic form (Infective larvae) - having Triploid chromosome
        - Free living female - having diploid chromosome

- Free living male - having haploid chromosome
- Parthenogenic female produces following type of eggs
    - Egg with triploid chromosome - give rise to parasitic female
    - Egg with diploid chromosome - infective female larvae and free living female
    - Egg with haploid chromosome - free living male
- Infection rate: In heterogony (under worm and moist condition) – each parasitic female produces 35 egg per copulation (during its life time produce, 180 eggs)

- *Host*
    - Piglets, foals, calves, lambs - neonatal infection (through colostrum) after birth - mobilization of arrested larvae in tissues of ventral abdominal wall of the dam which are subsequently mobilized and excreted through milk.
    - For very young animal reservoir of larvae is tissues of dam - clinical strongyloidosis in foals and pigs - successive progeny from same dam - give heavy infection
    - S.ramsoni – Prenatal infection in pigs
- *Environment*
    - Larvae are not ensheathed and susceptible to adverse environmental condition
    - Warm and moisture - favours accumulation of infective larvae.

LIFE CYCLE OF STRONGYLOIDES



PATHOGENESIS

- *S. papillosus*
    - Catarrhal enteritis - duodenum and proximal jejunum
    - Skin penetration by infective larvae - erythematous reaction – which favours introduction of foot rot organism into skin around feet
- *S. stercoralis*
    - Especially puppies - summer months - catarrhal enteritis of small intestine - necrosis and sloughing of mucosa
- *S. ransomi*
    - Especially suckling piglets
    - Epidemiology
        - oral infection – through infected larvae - adhere on udder and teats
        - from dam through skin penetration - from soil and litter
        - colostral infection - patent infection – very short (4 days)
- Protein losing gastroenteropathy
- Erythematous skin lesion may be seen - pulmonary disorder is not frequent in natural outbreaks – verminous pneumonia in all animals, petechial/ ecchymotic haemorrhagic in lungs

CLINICAL SIGNS, IMMUNITY, DIAGNOSIS AND TREATMENT

Clinical signs

- Skin lesion
- Severe diarrhoea which may be blood stained, dehydration, anorexia, loss of weight and finally death (especially piglets mortality - 50 %)
- Foals - acute diarrhoea

Immunity

- Infection with few infected larvae - in young animals - marked immunity
- Foals - satisfactory immunity -15 to 23 weeks after birth

Diagnosis

- Based on clinical signs
- Demonstration of eggs, larvae in faeces

Treatment

- *In sheep*
  - Thiabendazole - 75 mg/ kg orally
- *In pigs*
  - Thiabendazole - 50 mg/ kg through feed
  - Levamisole – 5 - 10mg / kg
- *In dog*
  - Diethylcarbomazine – 100 mg / kg
  - Dithiazanine – 5 mg /kg (10 daily doses)
  - Pyvinium pamoate – 20 mg /kg (5 days)
  - Thiabendazole - 50 -75 mg/ kg – highly effective
- *In foals*
  - Cambendazole - 20 mg /kg
  - Fenbendazole – 50 mg /kg

Prophylaxis

- Infected larvae susceptible to desiccation - providing clean quarter
- For prenatal and transclostral infection - in anticipated cases and treat before manifestation of clinical signs.
- In pigs
  - Mebendazole @ 72 – 104 mg / kg for daily 12 – 14 days prior to furrowing
  - Levamisole @ 140 mg /kg - total dose
  - Ivermectin -Single dose - 4 –16 days prior to furrowing.

STRONGYLIDAE

ORDER:STRONGYLIDA

- Super family: Strongyloidea
- Family: Strongylidae
  - Genus
    - Strongylus – Strongyles worm
    - Oesophagostomum - Strongyles worm
- Super family: Ancylostomatoidea
- Family: Ancylostomatidae

- Genus
  - Ancylostomum -Hook worms
  - Bunostomum - Hook worms

STRONGYLUS

Species

- *S. equinus* - Caecum and colon of equines, zebra
- *S. vulgaris* – Large intestine – equines
- *S. edentatus* - Large intestine – equines

Morphology

- Adult - Robust, dark red worm, Female - 32 – 40 mm and males – 26 – 35 mm long
- Well developed buccal capsule (*S. edentatus* – buccal capsule – no teeth)
- *Egg:* Oval, thin shelled enclosing segmented yolk when laid
- Coverings
  - Outer chitinous shell
  - Inner delicate vitelline membrane
  - Fluid cavity yolk and inner membrane

Epidemiology

- Host
  - Infection is more severe in foals, adults resistant to reinfection
- Bionomics
  - Embryonation
  - Hatching
  - Survivability of larvae in environment
  - Infection of definite host and exsheathment

Embryonation

- Temperature: optimal temperature 26°C (Embryonation also occur at 7.2°C and arrested at 0°C)
- O_2 tension: Increased O_2 tension favours Embryonation
- Moisture (Relative humidity): 70% (Desiccation unfavourable for embryonation)

Hatching

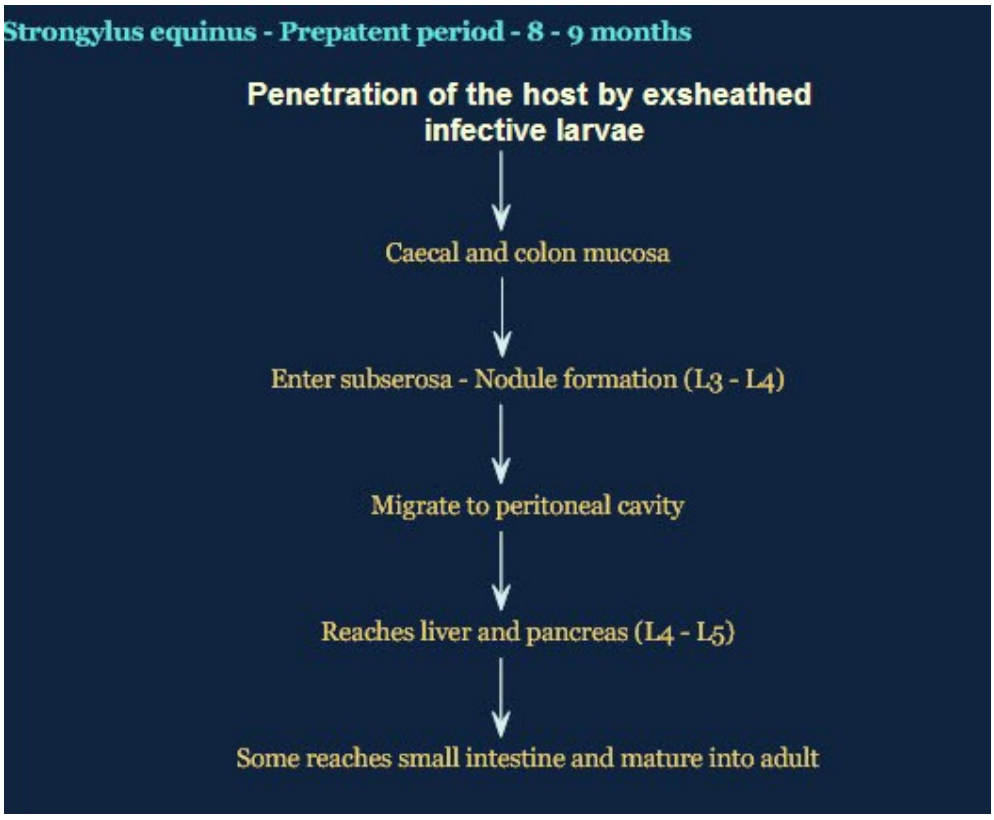
- Optimum temperature - 26°C (below 9°C it is affected)
- Relative humidity: 70%

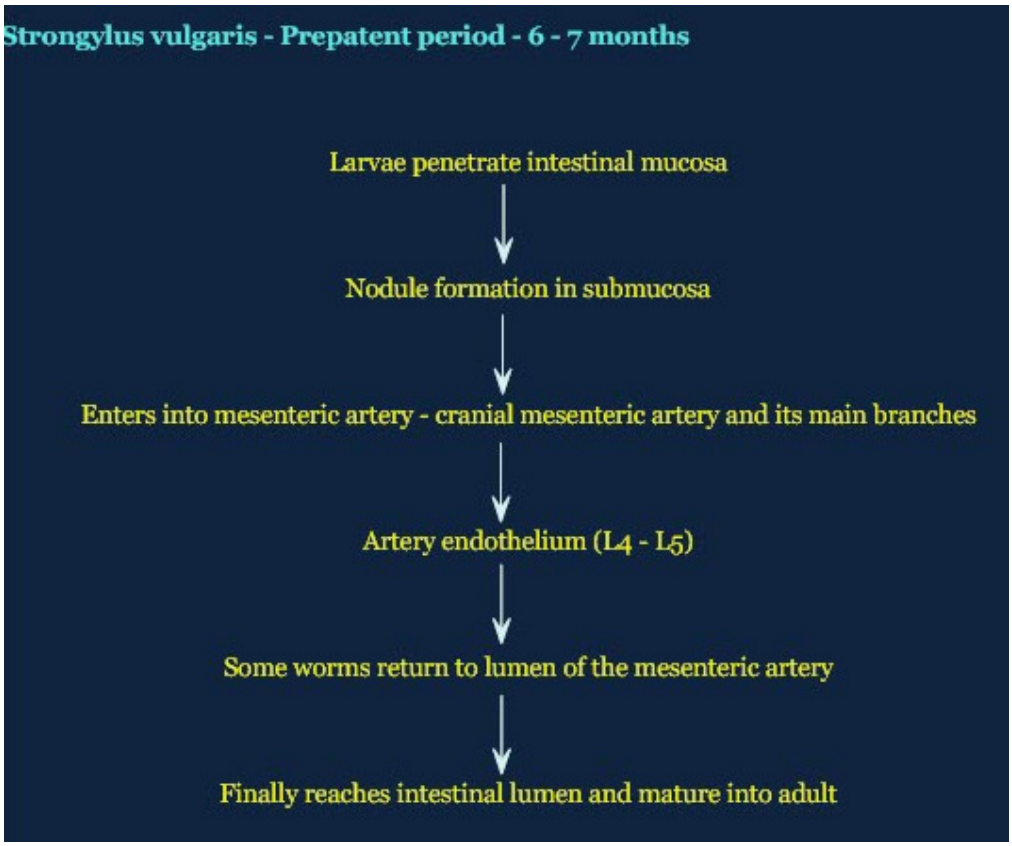
Survivability of larvae in environment

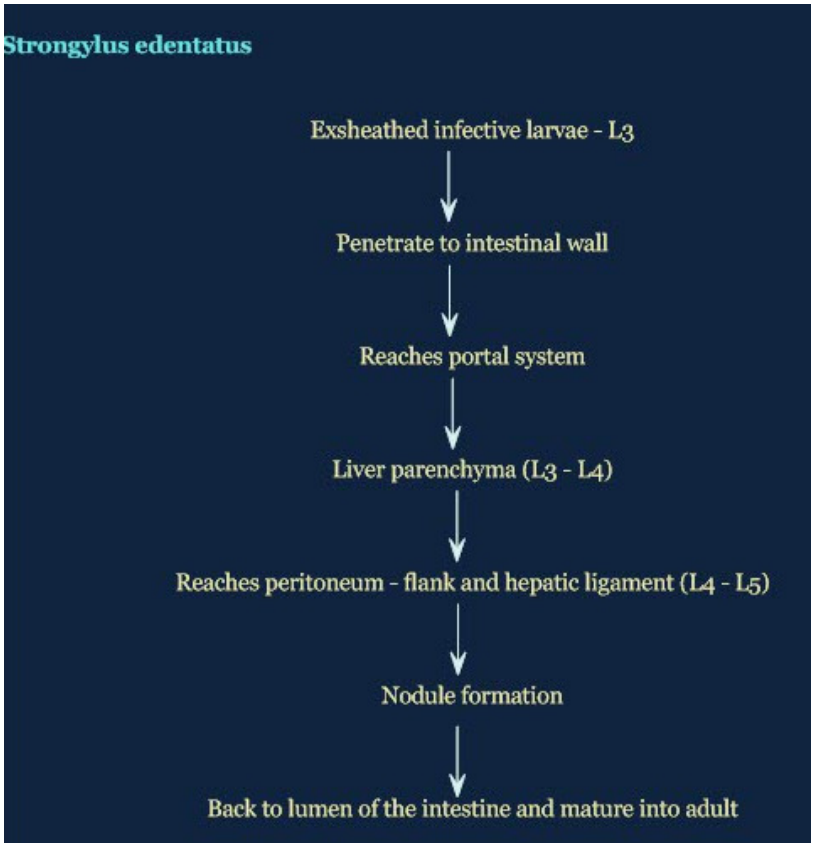
- Ensheathed larvae – protected from adverse weather conditions
- L1 and L2 feed on soil bacteria and L3 doesn't fed and utilise stored nutrient, if L3 not found suitable host leads to exhaustment of stored food reserves – leads to death
- Larvae does not actively enter the host. It is passively swallow along with feed and water
- Environmental stimuli: possibility of finding the host through crawl up grass blades
    - *Soil:* Negatively geotrophic - crawl up blades of grasses or other herbages from soil
    - *Light:*
        - Positively phototrophic to mild light
        - Negatively phototrophic to strong light and darkness
        - Larvae crawl up grass blades only in the early morning, towards evening and other times of the day in dull weather at night and during strong light hours larvae descend to soil.
    - *Moisture:* Flim of water is necessary for crawl up on grass blades
    - *Temperature:* Migration is more and active during warm weather than cold
    - *Survivability of larvae in the environment*
        - *In Soil*
            - Some larvae penetrate into soil where they survive more readily than on the surface
            - Loose sandy soil – penetrate more deeper than clay soil
            - In water - larvae sink on bottom of the pond and survive for a month or more
        - *In pasture grass*
            - Conditions favorable for migration (warm and daily fluctuation of light and loose soil) – Larvae more active and utilize all stored nutrient – death is faster
            - In dry seasons - not more than three months
            - In cooler climate - even year or more

Infection of definite host and exsheathment

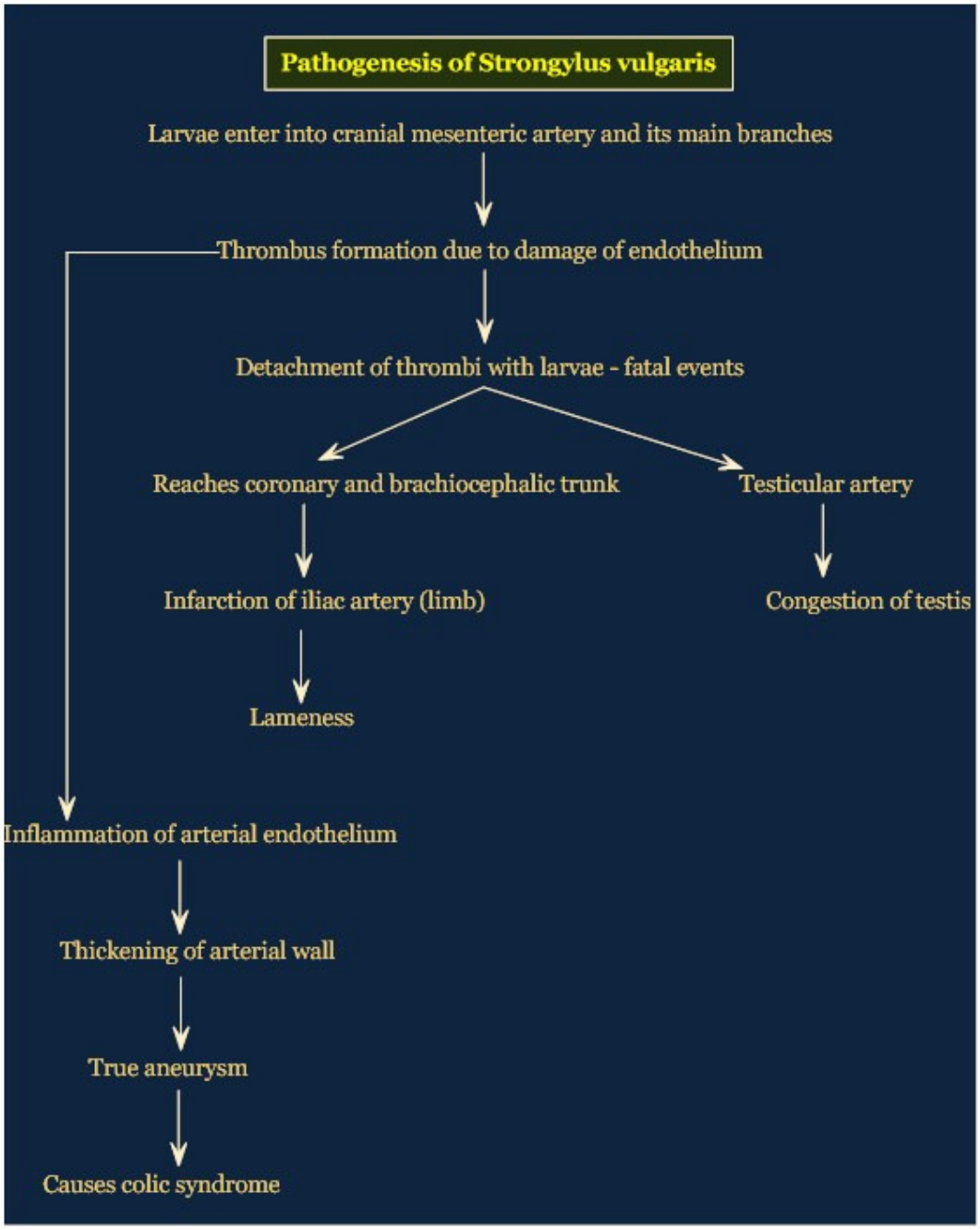
- *Infection of definitive host*
    - Passive ingestion of infective larvae along with grass blades or water
- *Exsheathment in small intestine*
    - Host stimulus are as follows
        - - unionized bicarbonates
        - - undisassociated Co2
        - - Dissolved gas Co2 - 70 %
        - - Intestinal pH – 7.3
        - - Intestinal temperature (37°C)
    - By the host stimuli the larvae secrete "Exsheathing fluid" which contains leucine aminopectidase - LAP. These enzymes breaks sheath along with larval movement.




Strongylus vulgaris - Prepatent period - 6 - 7 months
Larvae penetrate intestinal mucosa
Nodule formation in submucosa
Enters into mesenteric artery - cranial mesenteric artery and its main branches
Artery endothelium (L4 - L5)
Some worms return to lumen of the mesenteric artery
Finally reaches intestinal lumen and mature into adult


Strongylus edentatus
Exsheathed infective larvae - L3
Penetrate to intestinal wall
Reaches portal system
Liver parenchyma (L3 - L4)
Reaches peritoneum - flank and hepatic ligament (L4 - L5)
Nodule formation
Back to lumen of the intestine and mature into adult

PATHOGENESIS OF STRONGYLUS



Strongylus edentatus

- *Larvae migration in liver*
    - More than 4,000 larvae - haemorrhagic and fluid filled nodules
    - Less than 500 larvae - no clinical signs
- *Adult*

- Plug feeder - ingestion of plugs of intestinal mucosa
- Damage to blood vessels
- Blood suckers - Loss of blood - anemia (Normochromic and Normocytic)

CLINICAL FINDINGS, DIAGNOSIS, TREATMENT AND CONTROL

Clinical signs

- Slow development: Diarrhoea - Soft faeces with bad odour
- Reduced appetite, emaciation, rough coat
- Anemia, edematous swelling on abdomen and legs

Diagnosis

- Based on EPG - 1,000
- Fecal culture - Identification of L3 larvae
- Based on clinical signs
  - Aneurysm in cranial mesenteric artery – palpated through rectal examination (Large pulsating bodies - 6 –7 cm)
  - Aneurysm and colic - abdominal auscultation - hyper motility of intestine
  - Vertebral percussion - lumbar and sacral region - pain evinced
  - Paracentesis - peritoneal fluid – Differential count of WBC - neutrophilia and esinophila.

Treatment

- Piperazine at 220 mg / kg - effective various when combined with other compounds
- Thiabendazole at 44 mg / kg - effective against intestinal Strongyles
- Thiabendazole at 440 mg / kg- twice - effective against migratory larvae
- Mebendazole at 10 mg / kg - effective against adult worm
- Fenbendazole at 7.5 mg / kg - effective against adult worm
- Fenbendazole at 60 mg / kg - effective against adult worm & Migratory larvae
- Cambendazole at 20 mg /kg - effective against adult worm
- Oxbendazole at 5- 10 mg /kg
- Pyrental at 90 mg / kg
- Dichlorvas at 26 – 32 mg/ kg through feed
- Holoxon at 50 – 70 mg / kg
- Ivermectin - larvae present in anterior mesenteric artery

Prophylactic Schedule

- High stocking density & favorable environmental condition - once in six weeks
- High stocking density & unfavorable environmental condition (winter) - once in two weeks)
- Low stocking density - once in three months

Treatment for colic

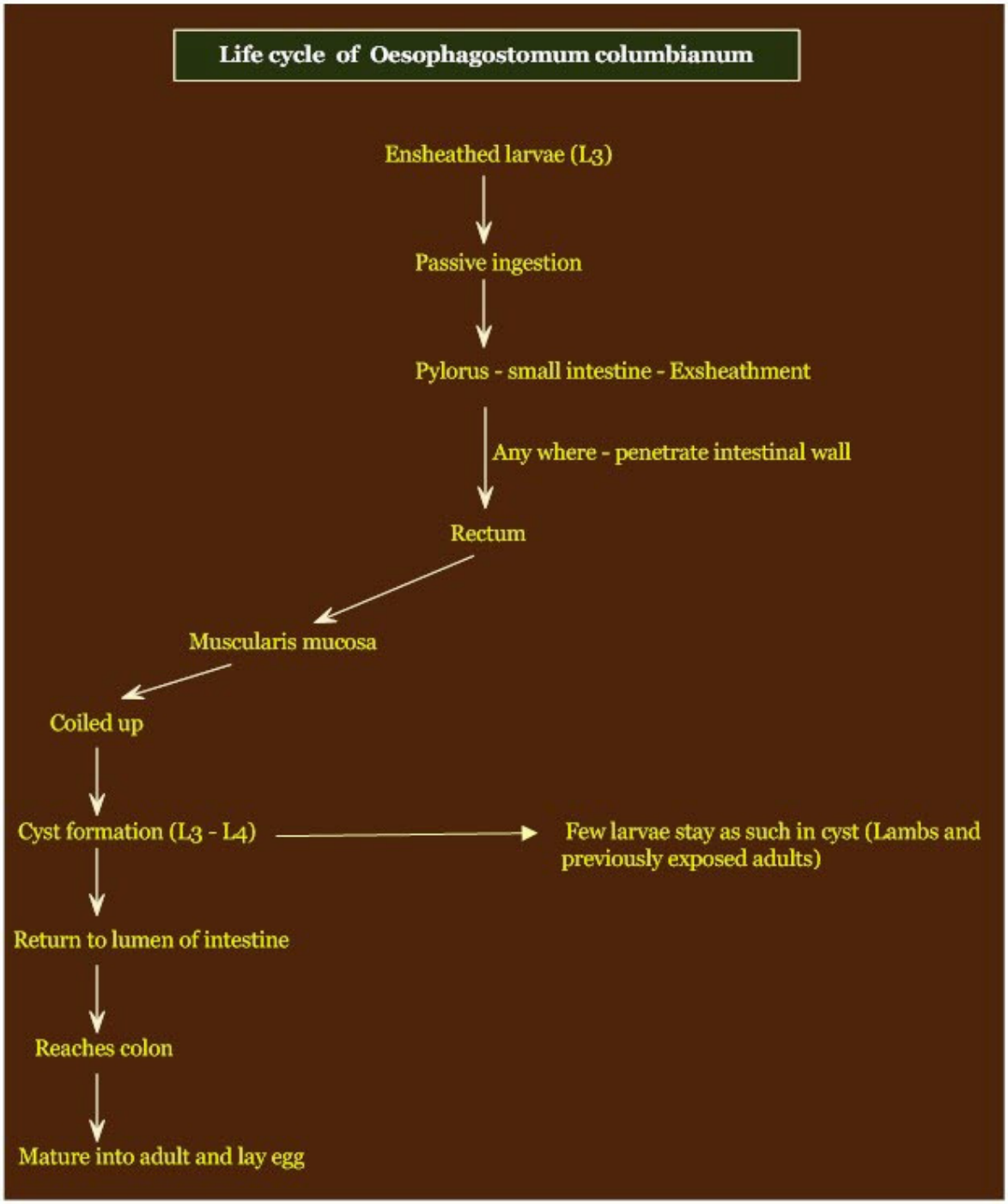
- Verminous aneurysm - 6 % iron dextran & 5% dextrin through I/V – antithrombin activity.

Prophylaxis

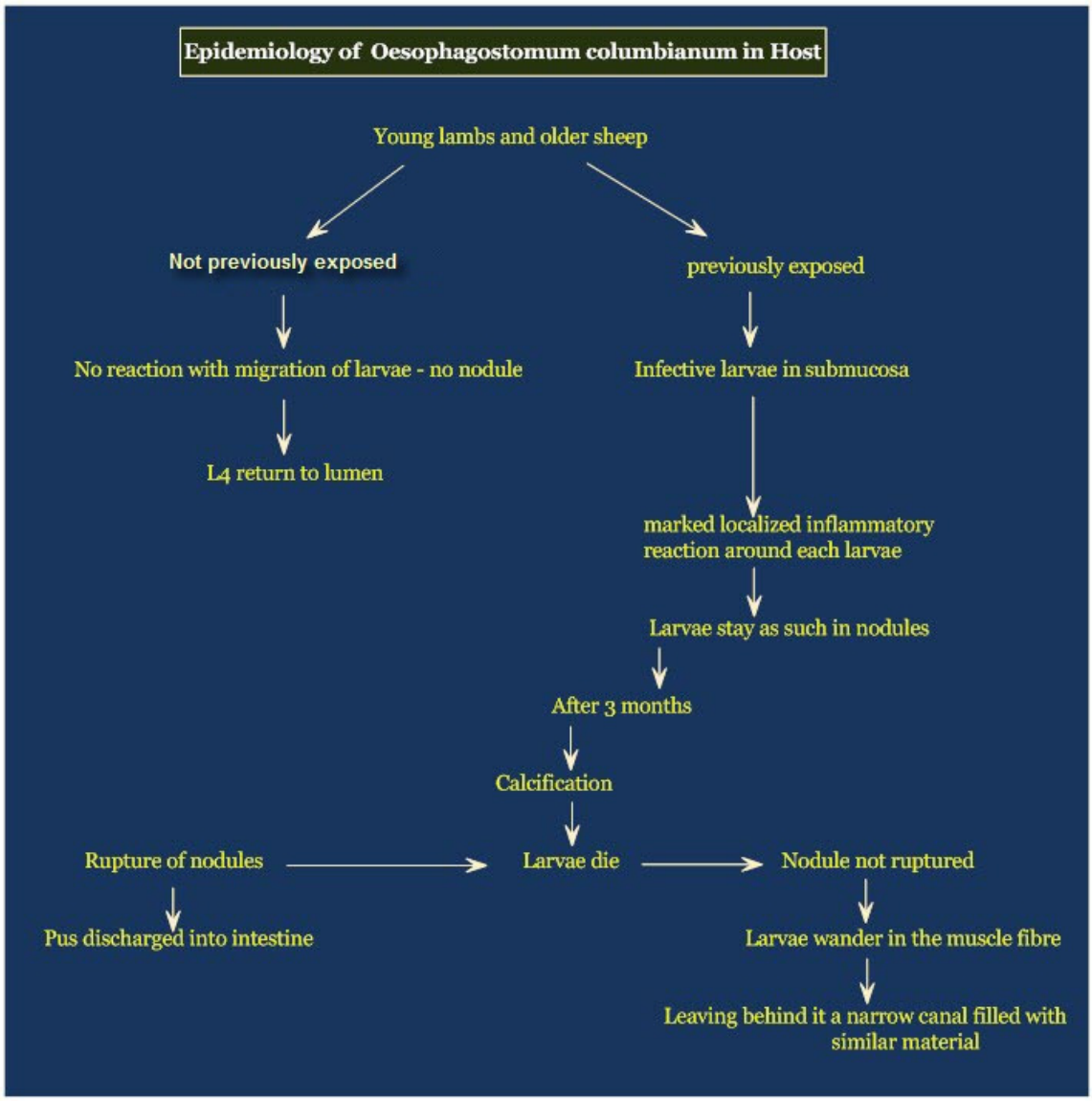
- Routine anthelmintics - all grazing animals over two moths of age treated with broad spectrum anthelmintics (change of drugs once in 6 months)
- Pasture and Grazing management - not over grazed or over stocked
    - Alternative grazing: Horse + Sheep; Horse + Cattle (cattle will assist reduction of larvae burden pasture; horse Strongyles do not infect ruminants)
    - Rotational grazing: nourishing mares and their foals, don't graze the same area for successive year
- Special attention for foals - fed only wholesome fodder
- Proper disposal of manure - heaping - generation of heat - destroy eggs
- Any new animals introduced - treat and quarantine for 48 – 72 hours

OESOPHAGOSTOMUM COLUMBIANUM

- *Common name:* Nodular worm (causes formation nodules on the intestine wall)
- *Host:* Sheep, goat, camel and wild antelopes
- *Location:* Colon
- *Morphology*
    - Adult worm Length: 1 – 2 cm
    - Egg: Thin shelled embryonated eggs (Blastomere 8 – 16 cell stage)
- *Life cycle*


Life cycle of Oesophagostomum columbianum
Ensheathed larvae (L3)
Passive ingestion
Pylorus - small intestine - Exsheathment
Any where - penetrate intestinal wall
Rectum
Muscularis mucosa
Coiled up
Cyst formation (L3 - L4)
Few larvae stay as such in cyst (Lambs and previously exposed adults)
Return to lumen of intestine
Reaches colon
Mature into adult and lay egg

EPIDEMIOLOGY OF OESOPHAGOSTOMUM COLUMBIANUM IN HOST



PATHOGENESIS, CLINICAL SIGNS, TREATMENT AND CONTROL

Pathogenesis

- Young sheep (200 –300 adult worm - severe infection)
    - Extensive nodule formation (2 cm) - Pimply gut
    - Interfere digestion, absorption and bowel movement
    - Superlative nodule - ruptured into peritoneal cavity – peritonitis and multiple adhesions
    - Congestion and thickening of intestinal of wall

Clinical signs (dose dependant)

- 500 larvae – depressed feed intake and utilization
- 500 - 2000 larvae
    - Marked persistent diarrhoea associated with loss of weight
    - Exhaustion and death (if animal is not removed from source)
    - Faeces - dark green in colour, contains mucus (rarely blood)
    - In chronic cases – initially diarrhoea followed by constipation
    - Progressive emaciation and general weakness, skin dry and wool is unthrifty
    - Hypoalbumineamia - submandibular oedema
    - Chronic oesophagostomiasis in sheep – extreme emaciation and cachexia with atrophy of muscles, ending in complete prostration for 1- 3 days and finally death.

Immunity

- Prolonged survival of L4 on nodules in the gut wall and the lack of an effective immunity - control difficult.

Diagnosis

- Examination of faeces – L4 larvae in acute cases & egg in chronic cases
- fecal culture - differentiation from other GI nematode
- Identification of adult worm by autopsy

Treatment

- Benzimidazole compound
- Levamisole and morental

OESOPHAGOSTOMUM SP. OTHER THAN *O.COLOMBIANUM*

Species

- O. venulosum - similar to O. colombianum - harmless – nodule seldom seen
- O. indicum - similar to O. colombianum
- O. radiatum
    - Host: Cattle, zebu, water buffalo
    - Location: Large intestine

Life cycle and epidemiology

- Similar to *O. colombianum*

Pathogenesis

- *Acute:* Infection of small and large intestine - black fetid diarrhoea
- *Chronic:* Young stock – extensive nodule - Pimply gut
    - Intermittent diarrhoea followed by continous purging, resulting in emaciation, prostration and often death. Nodule size - 5 cm

Clinical signs

- Anorexia, Anemia (Normocytic and Normochromic), Hypoproteinemia (protein loosing gastroenteropathy)

Immunity

- Good immunity to *O. radiatum*, partially due to age and partially due to previous exposure (unweaned calves having problem)

Treatment

- Benzimidazole compound
- Levamisole and piperazine

NODULAR WORM OF PIGS

- Species
    - O. dentatum – Large intestine
    - O. brevicaudum - Large intestine
    - O. mapelstonei - Large intestine
- Life cycle, epidemiology, pathogenesis, clinical signs are similar to O. columbianum
- Thin sow syndrome
    - Adult female - chronic infection with periodic acute flare-up
    - Spring farrowing sows - Resumption of development of hypobiotic larvae
    - weight loss after farrowing reduced milk yield and adverse affect of growth of litter.
- Control: Similar to A. suum

CHAPTER-25: ANCYLOSTOMATIDAE (HOOK WORMS)

Learning objectives

To know in detail about,

- Epidemiology, lifecycle, clinical signs, prevention and control of Ancylostoma spp and Bunostomum spp.

INTRODUCTION

- The family Ancylostomatidae (Hook worms) includes the genus
  - Ancylostoma
  - Bunostomum
  - Agriostomum
  - Gaigeria.

ANCYLOSTOMA

Species

- *A. caninum*
- *A. braziliense*
- *A. ceylanicum*
- *A. duodenale*

A. caninum

- *Host:* Dog & other carnivores
- *Location:* Small intestine
- *Distribution:* Cosmopolitan in tropical and subtropical countries
- *Morphology*
  - *Male:* 1 cm & female – 1.5 cm
    - Fairly rigid and grey or reddish in colour (Depending on the presence of blood in the alimentary canal)
  - *Egg:* similar to Oesophagostomum

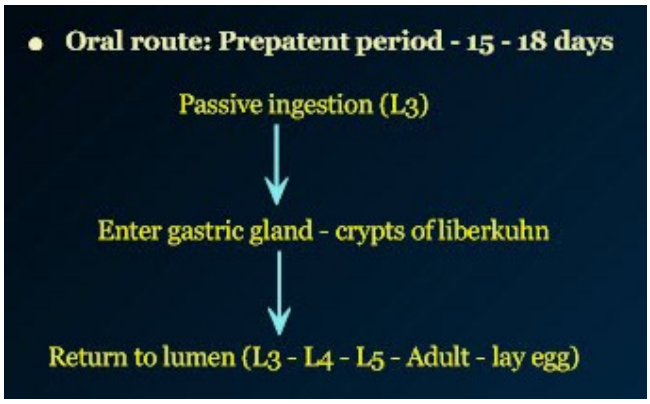
A. braziliense

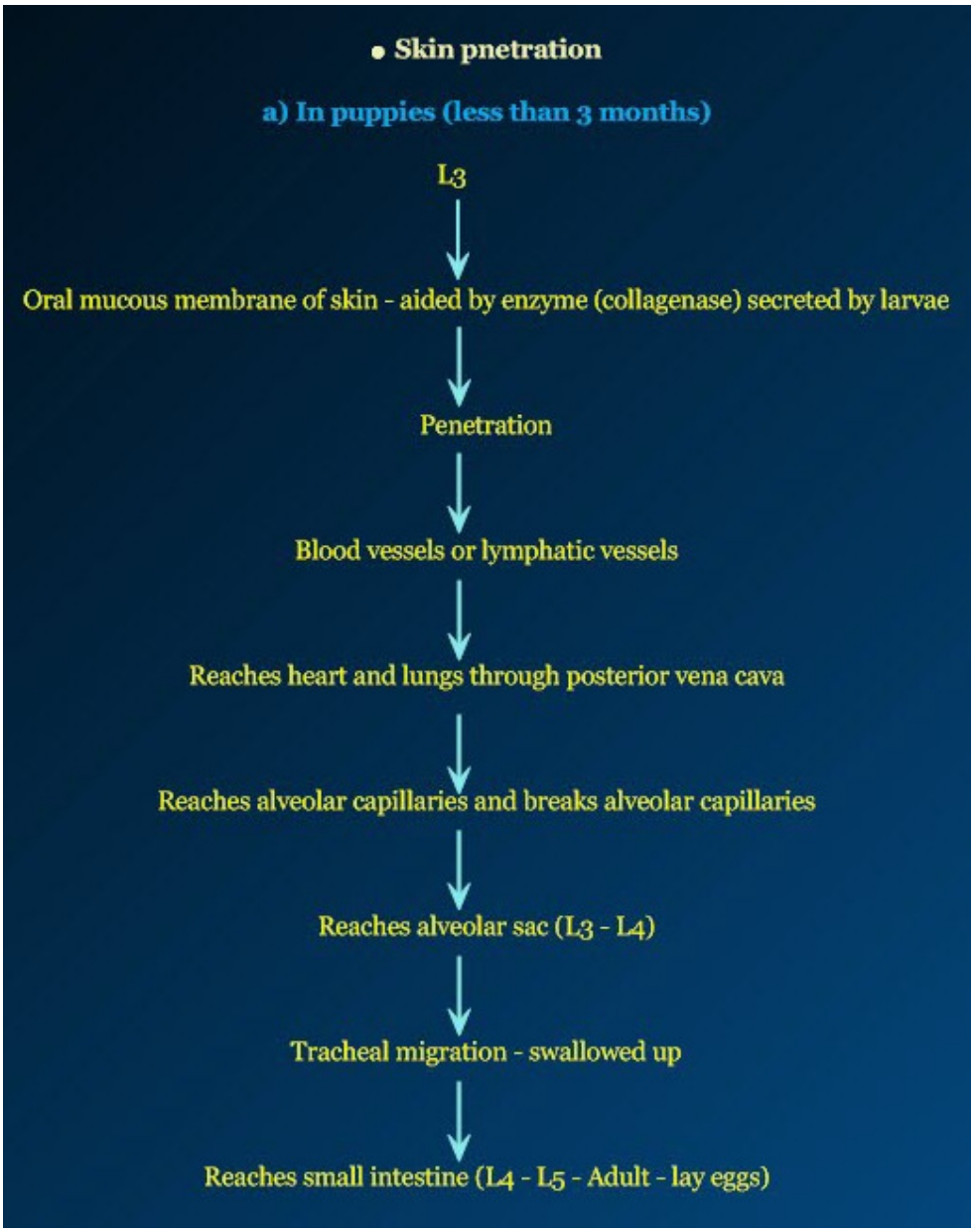
- *Host:* Dog, cat and wild carnivores
- *Location:* Small intestine

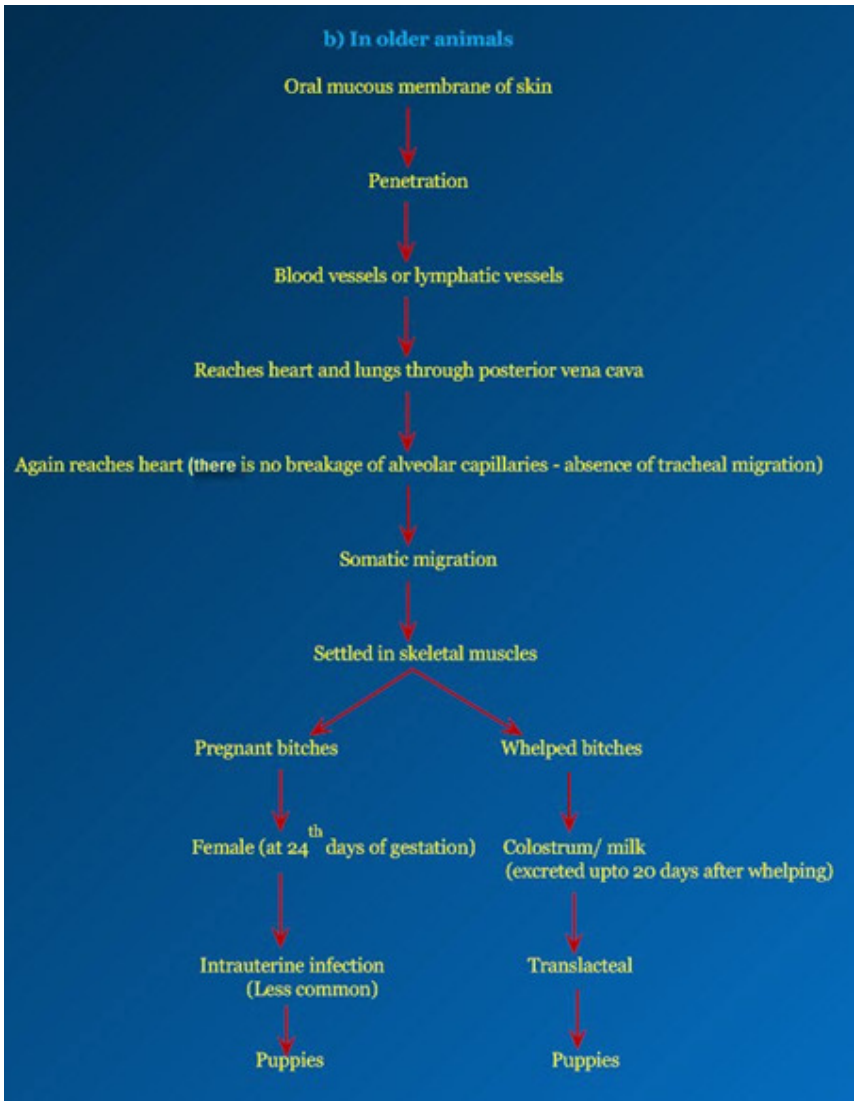
EPIDEMIOLOGY OF ANCYLOSTOMA

- Infection rate
  - Adult female lay - 16,000 egg / day (egg laying inversely proportional to worm load)
- Bionomic
  - Similar to Strongyles
    - Slightly sandy and moist soil suitable for survivability
    - Direct sunlight lethal to larvae
    - Housing: Soiled bedding with damp, porous or cracked floor – leads to massive infection
- Arrested larval development inside the host (in L_3 form)
  - Exposed to sudden chilling
  - Depends upon immunological status
- Host
  - Young puppies are highly susceptible (due to translacteal infection)
  - One year old age groups are highly susceptible
  - Smaller breeds are highly susceptible than heavier breeds
  - Nutritional status - well nourished animal with stand the infection and poor iron reserves in the body leads to severe infection (Milk is the poor source of iron)
  - Stress and corticosteroid therapy precipitate the infection from dormant larvae in muscle
- Infection of definitive host

Infection of definitive host by Ancylostomum sp.


• Oral route: Prepatent period - 15 - 18 days
Passive ingestion (L3)
Enter gastric gland - crypts of liberkuhn
Return to lumen (L3 - L4 - L5 - Adult - lay egg)


• Skin pnetration
a) In puppies (less than 3 months)
L3
Oral mucous membrane of skin - aided by enzyme (collagenase) secreted by larvae
Penetration
Blood vessels or lymphatic vessels
Reaches heart and lungs through posterior vena cava
Reaches alveolar capillaries and breaks alveolar capillaries
Reaches alveolar sac (L3 - L4)
Tracheal migration - swallowed up
Reaches small intestine (L4 - L5 - Adult - lay eggs)



- Paratenic host
    - Rodents (L3) —> passive ingestion —> Definitive host

LIFE CYCLE, PATHOGENESIS AND CLINICAL SIGNS

Life cycle

- *A. braziliense* – failure to induce prenatal infection - Intrauterine infection is absent
- *A. duodenale*
    - Skin penetration is important mode of infection
    - Arrested larval development occurs during unfavorable condition

Pathogenesis: Anemia

- Due to blood sucking activity of worm
- Stages of worm - L4 mainly followed by L5 and adult
- 8 Days of post infection (DPI)– starts to suck blood , 10 –15 DPI peak blood loss and 20 DPI egg output and heavy blood loss
- Death occurs within 10 –24 DPI in untreated cases
- Types of anemia – initially normocytic and normochromic followed by microcytic and hypochromic anemia due to loss of iron reserves

Clinical signs

- *Acute cases*
    - Hydremia, oedema, general weakness, emaciation, stunted growth
    - Diarrhoea – 4 DPI & fresh blood mixed with watery mucus on 8 DPI
    - Skin lesion
        - Associated with percutaneous infection ranges from moist eczema to ulceration
        - Cutaneous larval migration or creeping eruption
        - Damage in feet due to severe infection
        - Lesion aggravated by licking and self biting (after rain when allowed on wet grass or sand in endemic areas)
    - Haemorrhagic pneumonitis: - due to translacteal migration
- *Chronic cases*
    - Reduced appetite, poor growth, poor hair coat
    - A. braziliense – blood loss insignificant (0.001 ml), hypoproteinaemia and protein losing gastroenteropathy

Immunity

- Natural age resistance occurs on 8^{th} month of age in female and 11^{th} months of age in male
- Acquired immunity - Crisis – similar to self cure phenomena occurred in *H. contortus*
    - Graded doses of gamma irradiated L3 larvae when given S/C or orally, egg count increases upto 2 months after challenge there after marked reduction of egg count and large number of adult worm expelled through faeces
    - Dose: 1,000 larvae
    - Age of administration – 72 hours after birth (Presence of maternal antibodies (colostral) will not interferes)
    - Duration of immunity – 7 months in absence of reexposure

DIAGNOSIS, TREATMENT AND CONTROL

Diagnosis

- Based on clinical signs
- Faecal examination: EPG > 1,000 - severe infection

Treatment

- *For anaemia*
    - Blood transfusion
    - Haematinics and iron therapy
    - Protein rich foods
- *Anthelmintics*
    - Dichlorvos - 12 –15 mg/ kg (choline esterase inhibitor)
    - Tetramisole – 7.5 mg/ kg S/C, 20 mg/ kg oral
    - Mebendazole - 40 mg/ kg (total or divided doses)
    - Fenbendazole - 20 mg/ kg (immature and mature)
    - Nitroscanate - 50 mg/ kg oral

Prophylaxis

- Kennel floor should kept as clean and dry. Floor should treated with salt such as sodium borate @ 2 kg / 10 liter -destroys the larvae
- Floor should made up of impervious cement concrete
- Faeces removed and disposed off properly
- Deworming regimen similar to Toxocara
    - In pregnant bitches - 3 weeks before whelping to 2 days after whelping daily with fenbendazole @ 7.5 mg /kg
    - Nourishing litters
        - First dose - 1 – 2 weeks of age
        - Second dose - 3- 4 weeks of age
        - Weaned puppies - once in three months

BUNOSTOMUM (HOOK WORMS OF RUMINANTS)

Bunostomum trigonocephalum

- *Host:* Sheep, goat and cattle (rarely)

- *Location:* ileum and jejunum
- *Life cycle*: Similar to Ancylostoma, prepatent period: 30 – 56 days
- *Epidemiology:* Similar to Ancylostoma
    - Occurrence is more common in warmer climate than cold climate
    - Mixed infection Strongyles and Bunostomum
- *Clinical signs*
    - Anaemia, oedema on intermandibular region – "Bottle jaw"
    - Diarrhoea – not infrequent, faeces dark in colour due to altered blood pigments
    - Prostration and finally death
- *Diagnosis*
    - Based on clinical signs
    - Faecal examination
- *Treatment*
    - Fenbendazole - 5 mg /kg
    - Albendazole –7. 5 mg /kg
    - Parbendazole – 15 5 mg /kg
    - Cambendazole - 20 5 mg /kg
    - Thiabendazole -75 mg /kg
- *Control*
    - Avoid wet floor shelter – larvae highly susceptible to dryness
    - Similar to Ancylostoma

Bunostomum phlebotomum

- *Host:* Young calves, Sheep are rarely affected
- *Location:* Duodenum
- *Clinical signs*
    - Anaemia, diarrhoea, submandibular oedema
    - In stabled cattle itching and stamping of legs

CHAPTER-26: TRICHOSTRONGYLIDAE

Learning objectives

To know in detail about

- Epidemiology, Lifecycle, Clinical signs, Prevention and Control of *Trichostrongylus spp*., *Haemonchus spp., Mecistocirrus spp., Cooperia spp.,* and *Paracooperia spp.*

INTRODUCTION

- The family Trichostrongylidae includes the following genus
  - Trichostrongylus
  - Ostertagia
  - Cooperia
  - Nematodirus
  - Haemonchus
  - Mecistocirrus
  - Paracooperia.

TRICHOSTRONGYLUS (BLACK SCOUR WORMS)

Morphology

- Small, slender pale reddish brown
- Adult – 5 mm , infective larvae ensheathed larvae
- Egg: oval, thin shelled segmented yolk (32 cell stage when laid)

Species

T. colubriformis

- Host: Sheep, goat, cattle, camel and antelopes
- Location: abomasum and anterior part of small intestine

T. axei

- Host
  - Sheep, goat, cattle, deer and antelope Location: Abomasum
  - Pig, horse, donkey and man Location: Stomach

T. tenuis

- Host: Domestic and wild ducks, geese, fowl, G. fowl and turkey
- Location: Caecum and small intestine

Epidemiology

Bionomics

- Embryonation and hatching: similar to Strongyles
- *Development:* (4 –6 days) L1 –L3 (Infective larvae)
    - Temperature- 22 – 26°C (< 9°C no development)
    - RH – 85 - 100%
- *Migration in grass blades*
    - Mild light – 62 foot candles (during early morning and late evening)
    - Moisture - more than 0.12 ml / cm^2
- Survivability of infective larvae in environment
    - *T. axei*
        - Infective larvae more susceptible to cold and high temperature of the soil
        - Relative humidity – low detrimental to larvae
    - *T. colubriformis*
        - L3 resist desiccation, but L2 susceptible desiccation
- *Dissemination of larvae*
    - Larvae itself – migrate upto 5-10 cm
    - Dung beetle and flow of water also play a role
    - Infection rate
        - Adult worm lay – 100 – 200 eggs/ day
        - Infection rate is influenced by
            - No. of adult worms
            - Level of host immunity
            - Age of the host
            - Species of parasite
            - Stage of infection
            - Parturition
            - Consistency of faeces

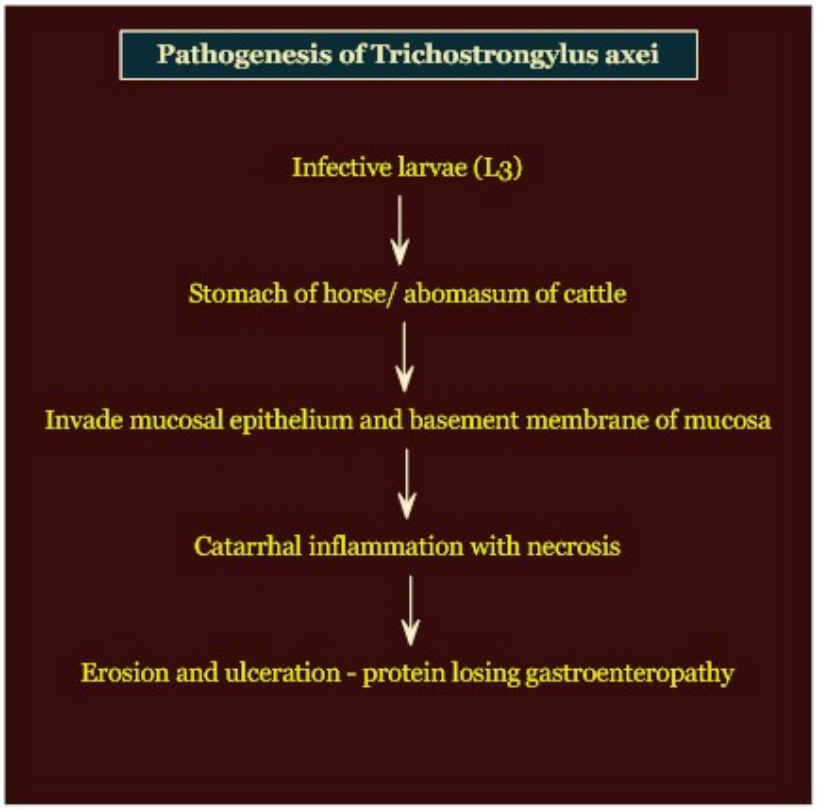
LIFE CYCLE AND PATHOGENESIS OF TRICHOSTRONGYLUS SP.

Life cycle

- Infection of definitive host: Through passive ingestion
- Exsheathment (abomasum or small intestine)
    - Host stimulus
        - Bicarbonate and Co2 buffer
        - Undisassociated Co2 and dissolved Co2
        - Abomosal pH –7.3 and Temperature - 37°C
    - Infective larvae secrete Leucine aminopectidase (LAP) and attack sheath

Pathogenesis

- *T. axei*



- *T. colubriformis: Similar to T. axei*
    - It occurs in anterior part of small intestine
    - In mucosa - formation of tunnels between epithelium and basement membrane
    - Distortion and displacement of mucosal epithelium

- Loss of plasma protein into intestine - hypoalbuminaemia

- *In general Trichostrongylus spp*
  - Loss of serum protein – anorexia - reduced feed intake – reduced food conversion ratio
  - Intestinal parasitism - increased gastric secretion of cholecystokinin – act on appetite centre in brain, leads to decreased appetite
    - Absorption of calcium and phosphorus from intestine reduced – retarded bone growth and osteoporosis.

CLINICAL SIGNS, IMMUNITY AND DIAGNOSIS OF TRICHOSTRONGYLUS SP

Clinical signs: Gastrointestinal disturbances – diarrhoea

- *Acute cases*
  - Sudden death, such animal neither emaciated nor anaemic
  - Leg weakness – unable to stand
- *Chronic cases*
  - variable in appetite emaciation, rough and dry hair coat
  - alternatively constipation and diarrhoea
  - anemia is not well pronounced (mild one)
  - nature of faeces- dark diarrhoea – "Black scour worms"

Immunity

- Infection induces immunity to reinfection (intake of infective larvae or presence of adult worm)
- Cross immunity is observed between *Trichostrongylus* spp, not at generic level

Diagnosis

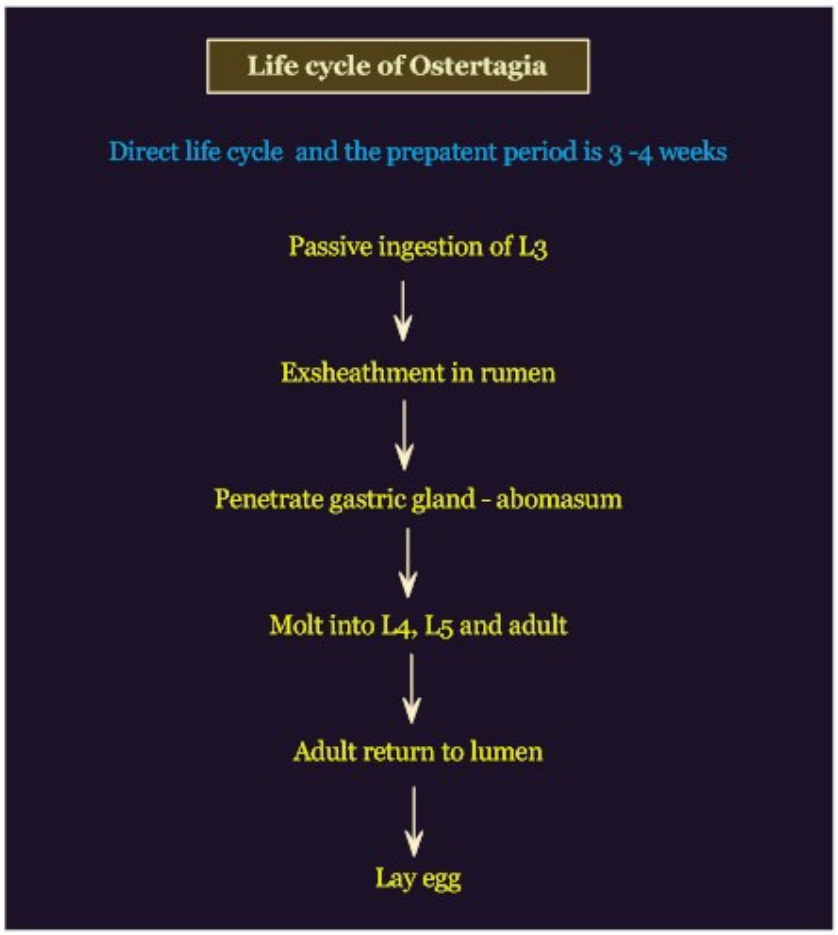
- Demonstration of eggs in faeces
- Faecal culture
- Avian sp: Haemorrhagic typhilitis with diarrhoea – loss of appetite, emaciation and anemia - Grouse disease.

OSTERTAGIA (BROWN STOMACH WORM)

- *Location:* abomasum and rarely in small intestine
- *Morphology:* Adult – 8 – 9 mm long
- *Species*
  - *O. ostertagi* – cattle, sheep and goat – rarely

- *O. circumcincta* - sheep and goat
- *O. trifurcata* – sheep and goat, cattle – rarely
- *O. pinnata* - sheep

- *Life cycle:* Direct – prepatent period – 3 –4 weeks



PATHOGENESIS, CLINICAL SIGNS, DIAGNOSIS AND TREATMENT

Epidemiology

- Similar to Trichostrongylus (air temperature more than 10°C)
  - Infection rate: adult worm lay - 100- 200 eggs /day
  - Survivability of infective larvae in environment
    - Infective larvae resistant to cold and survive prolonged period in soil
    - Infective larvae not markedly resistant to desiccation
  - Hypobiosis
    - Out side the host (during unfavourable environmental conditions)

- Inside the host - depends upon age, previous exposure, reproductive status, proportion of challenge dose

Pathogenesis

- Pathological changes occur in gastric gland and gastric mucosa
  - Hyperplasia and thickening of mucosa and raised nodules
  - Marked increase in abomasal pH – Hcl production decreased
  - Conversion of pepsinogen into pepsin is reduced
  - Leakage of plasma protein into intestine - hypoalbuminaemia

Clinical signs

- Reduction of appetite, profuse watery diarrhoea – bright green in colour
- Emaciation loss of weight

Diagnosis

- EPG – 1,000 severe infection
- Larval count on pasture - 100 / Kg of dried herbage - reduced growth rate - 1000 / kg of dried herbage - leads to clinical diseases
- Clinical pathology – elevation of pepsinogen level
- Response to treatment (indication of disease - Treatment with benzimidazole – animal return to normal).

Control

- Anthelmintics treatment and pasture management
  - Feeding of uncontaminated pasture
  - Alternative grazing and rotational grazing

HAEMONCHUS

- Morphology
  - *Adult worm*
    - Larger in size (1-3 cm) and visible under naked eye
    - Colour: Male: Even reddish in colour
    - Female: Uneven reddish in colour due to whitish ovaries twisted around reddish intestine
    - Having a small buccal capsule with slender tooth or lancet (Blood suckers)

- *Egg:* Similar to *Trichostrongylus* spp (16 –32 cell stage)

- Location: Abomasum
- Species
  - *H. contortus* - Sheep, goat , cattle and other ruminants (stomach worm or wire worm of ruminants)
  - *H. placei* - Cattle, and sheep

EPIDEMIOLOGY

H. contortus

- *Embryonation and hatching:* Similar to Strongyles
- *Development of infective larvae:* (Free living stage (L_1 and L_2) to parasitic stage (L_3)
  - High environmental temperature: 22°C to 27°C , development even occur at 5°C or 30°C but on lower rate.
  - Relative humidity: 85 % to 100 %
  - Rain fall: Precipitation of 5 cm / month (where the annual rainfall is less than 680 mm, development is delayed/arrested)
- *Dissemination of larvae in the environment*
  - By larvae itself - about 5- 10 cm
  - Dung beetle
  - Rainfall – flow of water
- *Migration of larvae in grass blades:* Similar to Strongyles
- *Survivability of egg / larvae in the environment*
  - Egg (Prehatch stage): more resistant to adverse conditions – like freezing and desiccation
  - Infective larvae: resistant to desiccation and very low temperature
- *Arrested larval development (Hypobiosis)*
  - The L_4 larvae in the host undergo hypobiosis. Which is influenced by environmental condition (cold, hot and dry weather) and immunological component ie. At the end of rainy season / beginning of dry season
- *Activation of arrested development):* Influenced by
  - Season: Onset of rainy season
  - Relaxation of immunity
  - Not treated with anthelminthics
- *Infection rate:* Adult worm lay 5,000 to 15,000 eggs / day

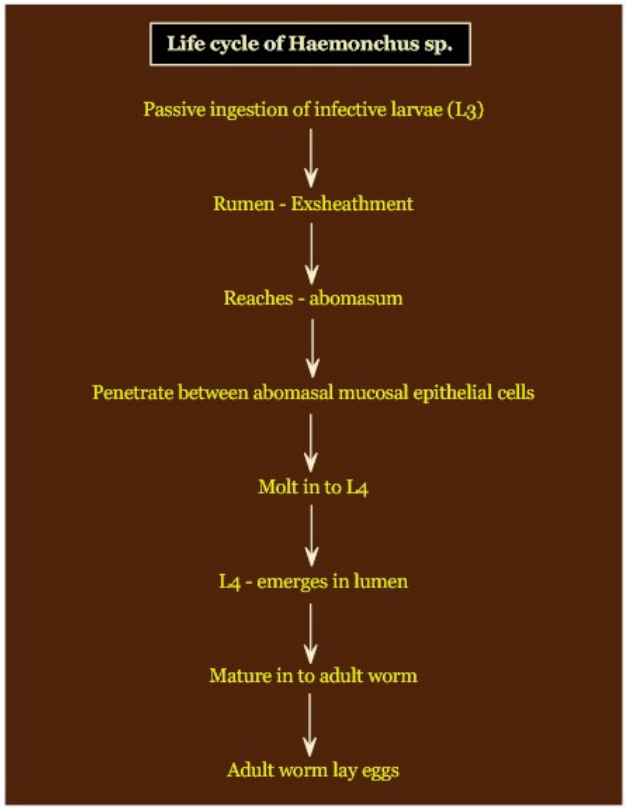
H. placei

- Epidemiology is similar to *H. contortus*.
- In addition to that cattle fecal pad acts as a reservoir of infective larvae.
- In which the larvae survive for several months and ultimately released when fecal pad is sufficiently moistened by rain.

Host

- *H. contortus: Sheep*
    - Age: Weaner age group (3 to 6 months) and lambed ewes are highly susceptible
    - Breed: Native breeds are highly resistant (Presence of anti-larval IgA antibodies) to infection than exotic and crossbred sheep
    - Hemoglobin type: Sheep having homozygous Hb type AA resistant to infection than sheep having heterozygous Hb type AB
- *H. placei:* Cattle - less than 2 years old are highly susceptible.

LIFE CYCLE



Prepatent period

- *H. contortus*: 15 days
- *H. placei:* 30 days

PATHOGENESIS

- Due penetration of infective larvae: Inflammation (haemorrhagic abomasitis) – protein losing gastroenteropathy – hypoproteinaemia (diarrhoea is not a characteristic feature)
- Anaemia: L4, L5 and adult worm suck : 0.05 ml/ day. Anaemia occurs in following ways
    - Blood sucker – blood sucking activity
    - Blood oozes – haemorrhagic – due to anti-coagulant secreted by the worm
    - Hypoproteinaemia - reduced Haemoglobin synthesis
- Anaemia is discussed in three phases based on PCV and serum iron level
    - *Phase I*
        - PCV: Reduced (22- 33%) – Activation of erythropoietic system – compensatory loss
        - Serum iron level: Normal
    - *Phase II*
        - PCV: level maintained, but less than normal level
        - Serum iron: decreased due to following factors
            - Marked loss of iron in the faeces (haemorrhagic)
            - Limited capacity to reabsorp from intestine
            - Low level of bone marrow reserves
    - *Phase III*
        - PCV: rapid drop
        - Serum iron: less than critical level (Dyshaemopoiesis)

CLINICAL SIGNS AND POST MORTEM CHANGES

Clinical signs

- *Sheep:* Ovine haemonchosis
- *Cattle:* Bovine haemonchosis
    - *Hyper acute*
        - Susceptible animal exposed to massive infection (> 10,000 infective larvae)
        - EPG: > 1,00,000

        - Anemia, dark coloured faeces – sudden death due to rapid blood loss
    - *Acute*
        - Young animals exposed to heavy infection (1,000 - 10,000 infective larvae)
        - EPG: 1,00,000
        - Anemia – fairly noticed – due to expansion of erythropoietic system
        - Anemia + Hypoproteinaemia – oedema on dependant part – submandibular region – bottle jaw
    - *Chronic*
        - Susceptible animal exposed to low infection (100 - 1,000 infective larvae)
        - EPG: <2,000
        - Anemia and hypoproteinaemia are fairly noticed
        - Weakness, unthriftyness and emaciation

Postmortem changes

- Hydrothorax, hydropericardium and ascites
- Watery blood
- Abdominal fat – liquefied and become gelatinous in nature
- Liver – light brown in colour – due to fatty changes
- Abomasum: having reddish-brown fluid ingesta; worm present on the mucosal folds and bite marks noticed on the abomasal mucosa.

IMMUNITY: SELFCURE PHENOMENA

- Loss or expulsion of worm burden in the gastro-intestinal tract of the suitably infected and sensitized sheep can be induced by challenge dose of infective larvae.
- Self-cure and protection against infection are not necessarily interrelated - Immunity is not absolute

Parasite factors which induce Selfcure phenomena

- It is well noticed in *H. contortus* - sheep and *H. placei* – cattle
- Among the *H. contortus* sub species – *H. contortus cayugensis* – self-cure was not recorded
- Cross immunity was observed between species of *Haemonchus*
- Cross immunity was observed between genus of *Haemonchus* and *Trichostrongylus*, but it is one way,
- Haemonchus induce immunity against Trichostrongylus and not vice versa.
- Exsheathed larvae induce good immunity than ensheathed larvae.
- Irradiated larvae induce good immunity

- Size of the challenge dose is inversely related to the self-cure

Other factors which induces self-cure

- Feeding of lush pasture, which might due to presence of an allergic / anthelmintic substance in the freshly growing grasses.
- Physiological alteration in the abomasum
- Season: After rainfall it is well pronounced

Host factor which induce Selfcure phenomena

- *Species:* Sheep – it is well pronounced
- *Breed:* Native breed of sheep having good response than exotic and crossbred sheep (due to presence of anti-larval IgA antibodies in the native breed)
- *Age:* Sheep greater than 6 months old having good response
- *Hemoglobin type:* Sheep with homozygous Hb type AA having good response than homozygous Hb type BB
- *Stage of lactation:* Relaxation of immunity – poor response
- *Level of lymphocytes in peripheral blood:* Sheep with low level of lymphocytes having low immune response.

Latent period for induction of Selfcure : 6-7 weeks

Postulated mechanisms of selfcure

- Challenge dose of infective larvae (L_3) molt into L_4 inside the host.
    - Developing larvae releases antigen, which induces immediate type of hypersensitivity reaction
    - Transient raise of blood histamines
    - Increase level of complement fixing antibodies
    - Intense mucosal oedema
    - Expulsion of worms

DIAGNOSIS

- Based on clinical signs
- Identification of eggs in faeces
- Fecal culture
- Autopsy of animal in the suspected flocks

MECISTOCIRRUS, COOPERIA AND PARACOOPERIA

Mecistocirrus

- *Species: M. digitatus*
- *Host and Location*
    - Buffalo, Cattle, Sheep and goat - Abomasum
    - Pig - Stomach
- *Morphology:* similar to Haemonchus, size - 3-4 cm
- Epidemiology, life cycle, pathogenesis, clinical signs and diagnosis - Similar to H. contortus

Cooperia

- *Location:* Small intestine, rarely on abomasum
- *Morphology*
    - Adult - Pale reddish in colour, Size: 5 mm
    - Egg: Similar to *Trichostrongylus* spp
- *Species*
    - *C. curticei* - sheep and goat; cattle –very rare
    - *C. punctata* and *C. pectinata* - Cattle ; Sheep – very rare
- Epidemiology, life cycle, pathogenesis, clinical signs and diagnosis: Similar to *Trichostrongylus* spp
- Difference in Pathogenesis: less pathogenic

Paracooperia

- *Location:* Small intestine, rarely on ceacum and colon
- *Species: P. nodulosa*
- Epidemiology, life cycle, pathogenesis, clinical signs and diagnosis: Similar to *Trichostrongylus* spp
- *Difference in Pathogenesis:* The larvae of this parasite occur in nodules of the intestinal wall.
- *P. nodulosa* may be very pathogenic in young buffalo.

CHAPTER-27: NEMATODIRUS, TRICHINELLA AND TRICHURIS

Learning objectives

To know in detail about

- Epidemiology, lifecycle, clinical signs, prevention and control of Nematodirus spp., Trichinella spp. and Trichuris spp.

NEMATODIRUS

Location: Small intestine

Morphology

- Adult large sized – 10 to 15 mm
- *Egg:* Larger in size (8 cell stage)

Species

- *N. spathiger* – Sheep, goat, cattle and other ruminants
- *N. battus* – Sheep
- *N. abnormarlis* - Sheep, goat and camels

Epidemiology

- Embryonation: similar to Strongyles
- *Hatching:* larvae inside the egg become sensitized to hatch by prolonged exposure to cold conditions (2- 3°C) and the stimulus to hatching is the raise of soil temperature (21°C)
- *Survivability:* only few weeks. Followed by rapid decline in pasture
- Level of infection in lambs depends upon hatching date and lambing date

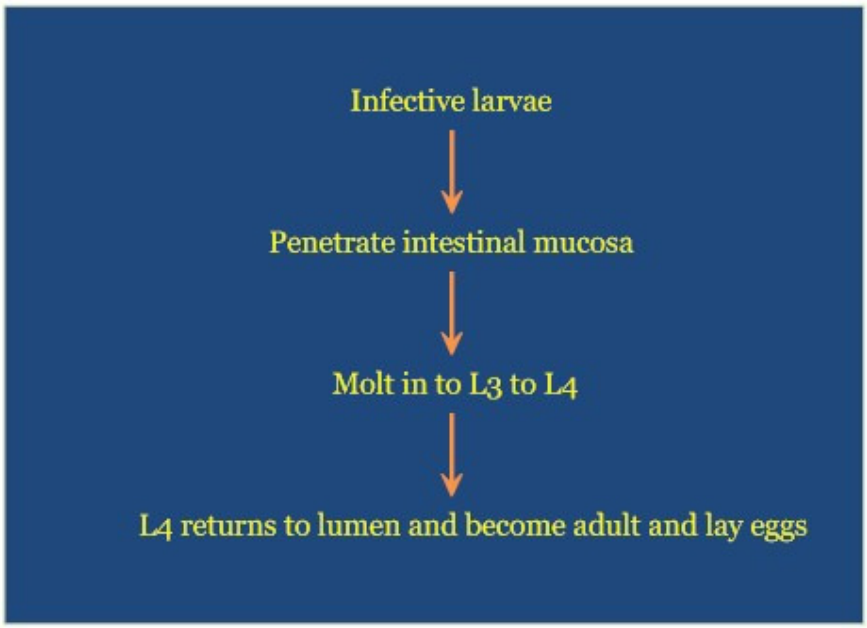
Host

- Young animals (grazing) are highly susceptible
- In older animals and in adults the infection is very milder and become source for pasture contamination.

LIFE CYCLE, PATHOGENESIS, CLINICAL SIGNS AND TREATMENT

Life cycle

- Similar to *Trichostrongylus* spp



Pathogenesis

- L3 penetrate tunneling and extensive destruction of mucosa villous atrophy

Clinical signs

- Acute enteritis: Excessive mucus secretion larvae enclosed by mucus material
- Severe diarrhoea
- Nature of diarrhoea: Blackish- green followed by yellowish in colour
- Anorexia, Inappetance, dehydration, prostration and finally death
- Mortality: 30 % (in 10 weeks old)

Immunity

- Surviving lambs (3 weeks after recovery) – Drop in EPG and become resistant to infection

Diagnosis

- Based on clinical signs
- Fecal examination

Control

- Predict time of occurrence and prevent exposure of lambs to infected pasture grass.
- Prophylatic deworming – two to three time at an interval of 3 weeks.

TRICHINELLA SPIRALIS

- Host: Man, pig, rat and many other mammals
- Location: Small intestine
- Morphology
    - *Egg:* contain fully developed embryos in the uterus of female (larviparous)
- Epidemiology
    - *Independent sylvatic cycle*
        - Wild carnivores (foxes, jackals, wild boars and bush pigs) – these animals maintain the transmission cycle, man become infected following ingestion of meat from wild animals.
    - *Synanthropic – zoonotic cycle*
        - Primarily in swine and rats, occasionally in cats, dogs and man
        - Epidemics of trichinellosis occasionally occur in human beings when a number of people ingest partially cooked trichinous meat of a pig, bear or other host.

TRICHINELLA SPIRALIS

PATHOGENESIS, CLINICAL SIGNS AND DIAGNOSIS

Pathogenesis and Clinical signs

- Intestinal forms – marked irritation and enteritis in heavy infection
- Larvae in muscle - Heavy infection may lead to death, especially paralysis of the respiratory muscle.
- The clinical signs which accompany trichinosis are very variable and may simulate those of a variety of other diseases; they include diarrhoea, fever, retroperitoneal pain, stiffness and pain in the affected muscles, dysponea, hoarseness, some times and oedema of the face and deafness.

Diagnosis

Human

- Provisional diagnosis: Based on the clinical signs of muscular pain, oedema of the eyelid and face etc.
- Presence of adult worm in the faeces (presence only in occasional)

- Identification of larvae in muscle: Biopsy of muscle piece examined either microscopically when pressed between two pieces of glass slide or after digestion in acid pepsin
- Increased eosinophilic count
- Immunodiagnostic methods: CFT, HA, flocculation and intradermal techniques.

Animals

- In addition to that the diagnosis of *T. spiralis* in animals depends mainly on the detection of the infection at meat inspection.
- Trichinoscope – Identification of larvae in muscles
- Mass screening in pigs by automated ELISA and radial immunoassay

TREATMENT, CONTROL AND PREVENTION

Treatment

Human

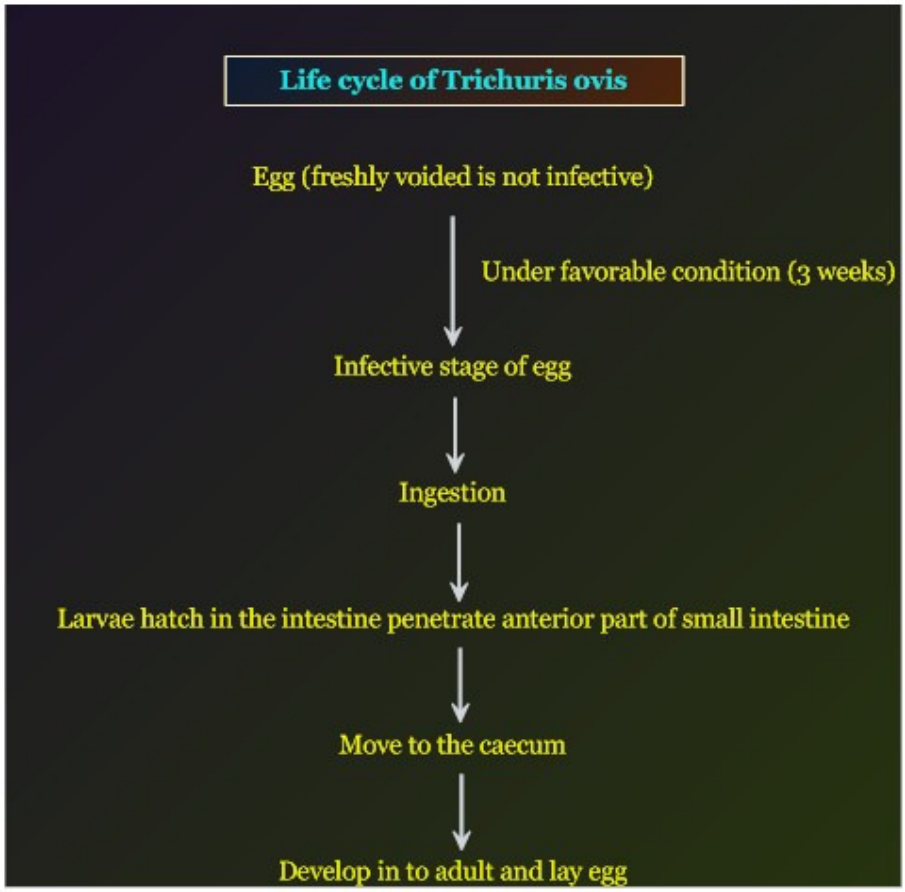
- Thiabendazle – 25 mg/ kg twice daily for five to ten days
- Flubendazole – 200 mg thrice daily for 10 days

Control and prevention

- *Pigs:* Prohibition on the feeding of garbage or garbage to be cooked before being fed to pigs.
- *Man:* Through cooking of pork products (58°C) or freezing at – 25°C for 10 – 20 days.

TRICHURIS OVIS

- Host: Goat , sheep, cattle and other ruminants
- Location: Caecum
- Morphology
    - *Adult:* Anterior part of the body is long and slender while posterior part is much thicker
    - *Egg:* Brown barrel - shaped, with a transparent plug at either pole
- Life cycle



EPIDEMIOLOGY, PATHOGENESIS, DIAGNOSIS, TREATMENT AND CONTROL

Epidemiology

- Development of infective stage depends on
    - Prolonged lower temperature (6 – 20 °C)
    - Soil temperature and moisture
- Infective egg may remain viable for several years
- Sheep over eight months of age show an age resistance to infection and a resistance to reinfection two to three weeks after primary infection

Pathogenesis and clinical signs

- Blood feeder – cause anaemia, haemorrhage and some times cause jaundice and death
- Adult forms a tunnel into the intestinal mucosa
- Cause severe inflammation - diarrhoea

Diagnosis

- Presence of characteristic egg

Treatment

- Methyridine – 200 mg/kg orally subcutaneous - highly effective
- Fenbendazole – 5 – 20 mg/kg
- Oxbendazole - 2.5 mg/kg

Prevention and control

- Improves hygiene
- Heavily infected soil should be avoided for several months to allow the natural agencies of sunlight and dryness to kill the eggs.

CHAPTER-28: SCHISTOSOMIASIS

Learning objectives

To know in detail about

- Epidemiology, Lifecycle, Clinical signs, Pathogenesis, Treatment, Prevention and Control of *schistosoma sp.*

MORPHOLOGY

- Schistosomes are called as blood flukes
- *Unisexual* – dimorphic trematode
- *Female* - long (12 –28 mm) & slender
- *Male* - Comparatively small in length (9-22 mm) & having gutter-like groove (Gynaecophoric canal) in which female lodges at the time of copulation
- *Cercariae* - Tail forked - *Furcocercous cercariae*
- *Egg* - 100 –500 µm long, spindle in shape without operculum. Some species having lateral spines.

LIST OF SCHISTOSOMES

Schistosome sp.	Host	Location
S. bovis	Cattle, sheep and goat	Portal and mesenteric veins
S. spindale	Ruminants, pig and dog	Mesenteric veins
S. indicum	Ruminants, equines and camels	Portal and mesenteric veins
S. haematobium	Pig, monkeys and man	Veins of mesenteric, bladder, urethra and ureter
S. nasalis	Buffalo , cattle, goat, sheep and horse	Nasal vein
S. incognitum	Pig and dog	Mesenteric vein

HUMAN SCHISTOSOMIASIS-BILHARZIASIS

- *S. mattheei, S. japonicum, S. mekongi, S. mansoni and S. haematobium*

Swimmers itch / Clay-diggers itch /Hunters itch / Cercarial dermatitis / Rice-Paddy itch

- Skin penetration of cercariae of non-human Schistosomes – Avian (*Trichobilharzia* spp. and *Austrobilharzia* spp) and animal Schistosomes (*Heterobilharzia* spp.)
- Form of Cutaneous larva migration
- Initial mild erythema, oedema will appear
- *Chronic cases* – severe pruritis and a papular or pustular eruption
- *Treatment* - Application of benzyl benzoate over the body and Antihistaminic therapy
- *Prevention* - Wearing protective clothing while working in the infected water sources.

INTERMEDIATE HOST AND DEVELOPMENTAL STAGES

Intermediate host

Name of the Snail	Name of trematode
Indoplanorbis exustus	*S. nasalis, S. indicum* and *S. spindale*
Lymnaea luteola	*S. nasalis* and *S. incognitum*
Lymnaea acuminate	*S. spindale*

Developmental stages

- Redia and metacercariae stages are absent
- Two generation of sporocysts (I and II)
- Immature schistosomes - **Schistosomula**

EPIDEMILOGY

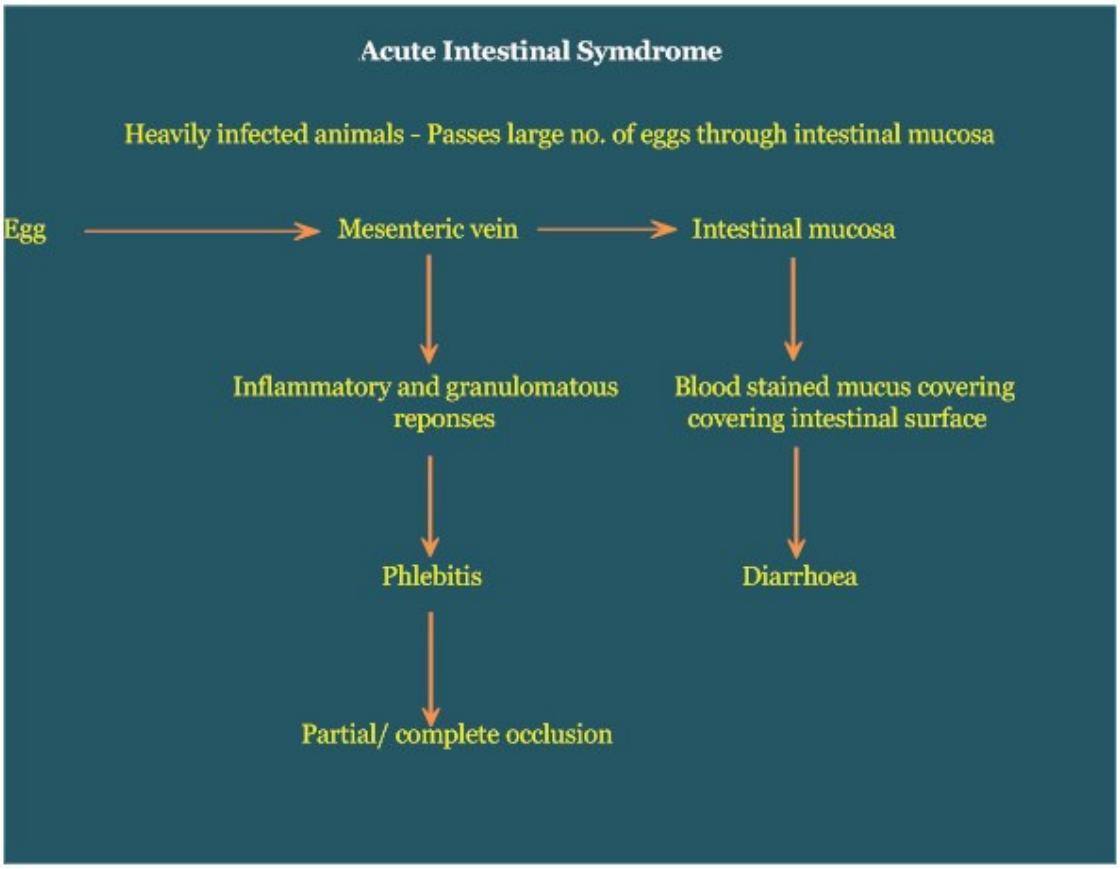
Skin penetration

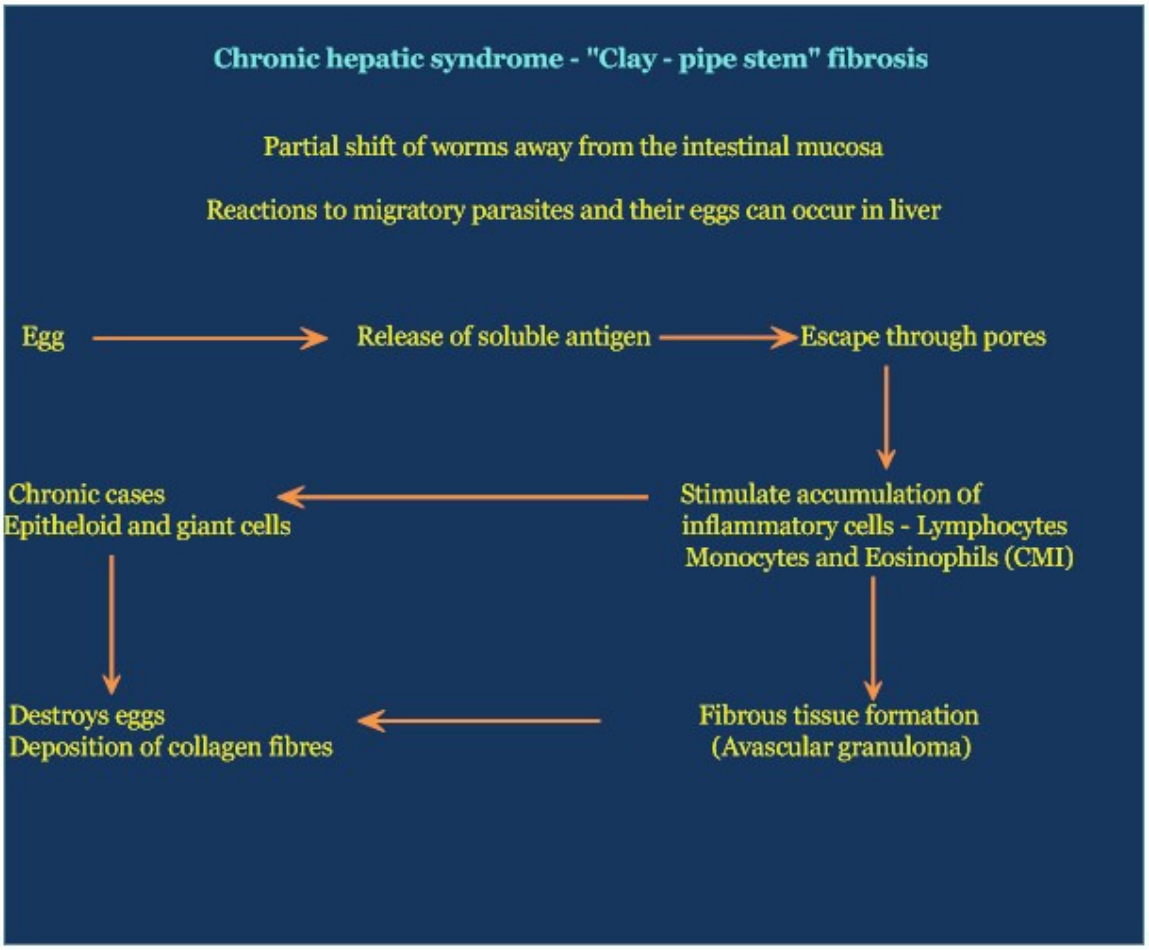
- Horses and cattle may get infection while standing in the infected water sources
- Buffaloes get infection while wallowing in the infected water sources
- Slow moving water favour – entry of cercariae into body of animal
- Consumption of water contaminated with cercariae
- Highest rainfall (Season – Sepember to October)

Species affected

- Sheep and goat
- Cattle >buffalo
- *Age:* Older bovines (>3 years)
- Infection also occurs 5- 6 months old calves
- *Sex:* both sexes are equally affected.

PATHOGENESIS





NASAL SCHISTOSOMIASIS-SNORING DISEASE/PSEUDOTUBERCLE/ACTINOBODY FORMATION

- Cattle and buffalo (infection rate – 40 –50%) and Horses
- *S. nasalis* - nasal mucosal vein – induction of inflammatory and granulomatous response

Clinical signs

Cattle

- Rhinitis – mucoprulent discharge, sneezing and dyspnoea
- Adult parasite – dilation of vein and thrombosis of vein
- Nasal mucosa studded with small granulomas and small abscess containing eggs
- Chronic cases – proliferation of nasal epithelium
- Space occupying lesion - breathing difficulties - snoring

Buffalo

- Pin sized eruption and congestion of nasal mucosa - **Actinobodies**

- Diarrhoea, sometimes blood stained and contains mucus
- Anorexia, thrust and emaciation

Clinical pathology

- Anemia, Eosinophilia, hypoalbuminemia, hypergammaglobulinemia
- Dyshaemopoisis and expansion of plasma volume

Diagnosis

- Based on history, clinical signs and clinical pathology
- Demonstration of characteristic eggs
- Measuring cell mediated immune response

IMMUNITY

- *Natural immunity* – cattle
- *Artificial immunity*
    - Irradiated Schistosomula of *S. mattheei* and *S. bovis*
    - Route - S/C or I/M
    - Cattle and sheep - induce greater than 60% protection

TREATMENT AND CONTROL

Human Schistosomiasis

- Praziquantel – 60 mg/kg single dose or in divided doses (drug of choice)
- Niridazole, Hycanthone, Lucanthone and Furapromidium
    - Stibophen: Cattle - 7.5 mg/kg for 6 days
    - Lucanthone: Cattle – 30 mg/kg; Sheep - 30 –50 mg / kg for 3 days
    - Hycanthone: Sheep – 3 –6 mg/kg
    - Trichlorophon : Cattle – 50 –7 0 mg/kg orally 4 – 6 doses
    - Niridazole: Sheep – 100 mg/kg for 3 days
    - Praziquantel: Sheep and goat – 60 mg/kg

Nasal Schistosomiasis

- Tartar emetic - 2 mg / kg
- Sodium antimony tartrate – 1.5 mg / kg twice a day for 2 days
- Trichlorophon - 30 –40 mg / kg three doses

Control

- Management practices and snail control similar to that of Amphistomiasis and Fascioliasis
- Biological control *Schistosomes* larvae
- Larval stages of *Echinostoma* spp. – used as predatory on schistosome larvae within the snail intermediate host
- Microsporidial protozoa – *Nosema eurytremae*
- Avoid contact of infected water sources

CHAPTER-29: COENEUROSIS AND ECHINOCOCCOSIS

Learning objectives

To know in detail about

- Tapeworms of carnivores
- Epidemiology, clinical signs, diagnosis and treatment of metacestode infections of tapeworms of carnivores in intermediate host
- Hydatidosis-Epidemiology, lifecycle, pathogenesis, diagnosis, treatment and control.

COENEUROSIS

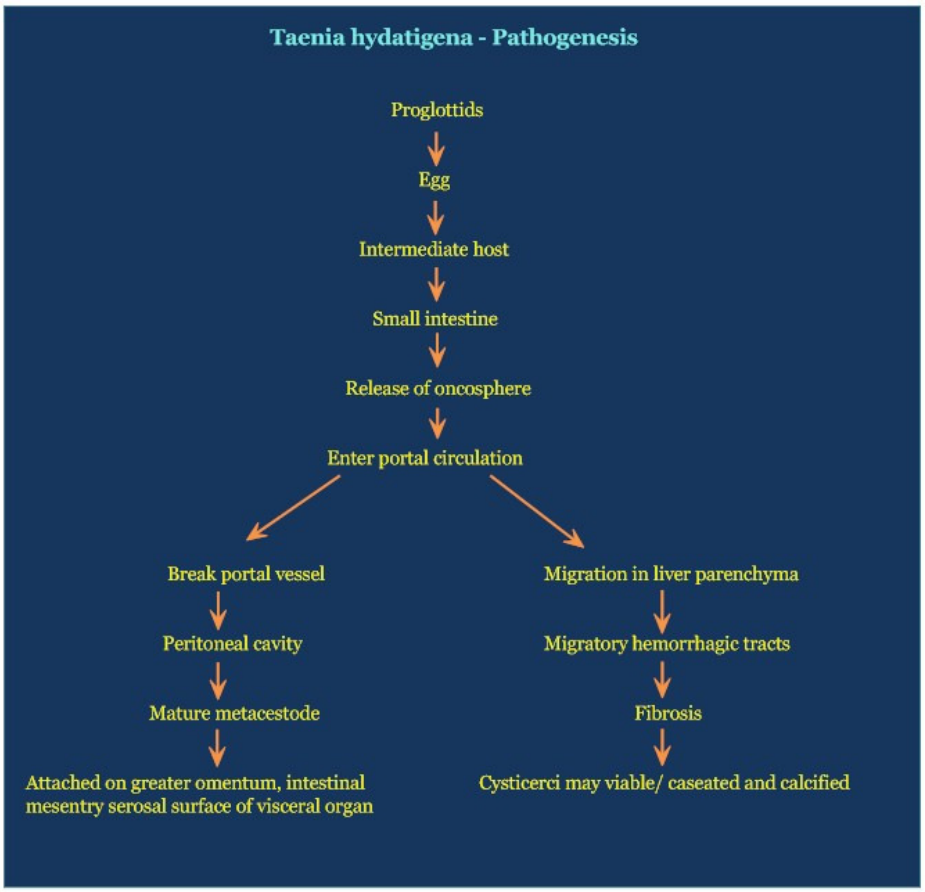
TAENIA OVIS

- *DH:* Dog
- *Location:* Small intestine
- *IH:* Sheep
- *Metacestode stage: Cysticercus ovis*
- *Location:* massetors, heart and diaphragm
- *Life cycle:* similar to *T. saginata*
- *Epidemiology*
    - Infected dogs - passes gravid proglottids through faeces
    - Pasture contamination
        - Entry of stray dogs into pasture land.
        - Using of untreated shepherd dog (that are not dewormed against tape worms) for shepherding of sheep on pasture land.
        - Dissemination of eggs in the pasture land through wind and insects.
    - IH
        - Passive ingestion of egg along with pasture grass.
    - DH - Passive ingestion of meat containing cysticercus possibly by
        - Stray dogs stands around unauthorized slaughter houses on road side, they eat the throwaway inedible portion of meat which containing *cysticercus ovis*
        - Feeding of infected offal to house hold dogs.
- *Pathogenesis:* Similar to *T. saginata*
- *Clinical signs:* IH: No clinical signs, unaesthetic that decrease meat value.

- *Diagnosis*
    - IH: Only by meat inspection & serology no value.
    - DH: Characteristic egg
- *Treatment:* IH: no value
- *Control:* Control of epidemiological factors.

TAENIA HYDATIGENA

- *DH:* Dog, wolves and other carnivores
- *Location:* small intestine
- *IH:* Sheep (Mainly) and pig (Domestic and wild ruminants are also affected)
- *Metacestode stage: Cysticercus tenuicollis* (6cm in size and containing single scolex invaginated into a long neck)
- *Location:* peritoneal cavity
- *Life cycle and epidemiology:* similar to T. ovis
- *Pathogenesis*



- *Clinical signs*
    - In lambs – unthriftiness
    - Immature cysticercus– liver – traumatic hepatitis – death (Hepatic cysticercosis)
    - Mature cysticercus – peritoneal cavity – no harm
- *Diagnosis*
    - IH: similar to *T. ovis*
    - Differential diagnosis – differentiated from acute fascioliasis
- *Treatment:* No valuable
- *Control:* similar to *T. ovis*

TAENIA PISIFORMIS AND TAENIA TAENIAFORMIS

Taenia pisiformis

- *DH:* dog (mainly) and other carnivores
- *Location:* Small Intestine
- *IH:* Lagomorphs, primarly: Rabbit, hares and rodents
- *Metacestode stage: C. pisiformis*
- *Location:* Peritoneal cavity attached to viscera

Taenia taeniaformis

- *DH:* dog (mainly) and other carnivores
- *Location:* Small Intestine
- *IH:* Rodents
- *Metacestode stage: C. fasciolaris*
- *Location:* Liver

COENUROSIS

- *Coenurosis in sheep* – caused by metacestode stage (*Coenurus cerebralis*) of dog tape worm - *Taenia multiceps*
- *Coenurosis in goat* – caused by metacestode stage (*Coenurus cerebralis*) of dog tape worm - *Taenia gaigeri*
- *Coenurosis in rabbits* – caused by metacestode stage (*Coenurus serialis*) of dog tape worm - *Taenia serialis*

TAENIA MULTICEPS

- *DH:* Dog and fox
- *Location:* Small intestine
- *IH:* Sheep
- *Metacestode stage: Coenurus cerebralis*
- *Location:* Brain and spinal cord
- *Coenurus cerebralis* - large fluid filled cyst about 5 cm or more in diameter, the cyst wall containing several hundred scolices in clusters.
- *Life cycle and Epidemiology:* Similar to *T. ovis*
- *In addition:* In IH: older lambs & adults are high risk group
- *Pathogenesis*
    - The developing larval stages of metacestode reaches brain and spinal cord.
    - Large numbers of immature metacestode migrate in the brain and spinal tissues and produces yellowish to reddish migratory tracts.
    - It leads to acute meningioencephalitis especially in adult lambs.
    - The immature metacestodes develop into mature fluid filled cyst.
    - The cyst produces space occupying lesions and exerts pressure on tissues of brain and spinal cord.
    - Some cases it causes softening of skull bone and finally leads to perforation.

CLINICAL SIGNS, DIAGNOSIS AND TREATMENT

Clinical signs

- *Due to Immature*
    - Based on the location the clinical signs may varies
        - *In brain:* **Staggers or Gid or Sturdy**
            - Parietal region - surface of cerebral hemisphere – left side
            - Animals held its head in affected side (left) and moves in circle – left direction and right eye may be blind.
            - Parietal region - surface of cerebral hemisphere – right side
            - Animals held its head in affected side (right) and move in circle – right direction and left eye may be blind.
            - Anterior part of the brain- head lowered and held against chest, walk in straight line until to hit on hard objects and remain motionless for some time.

        - Ventricle of the brain – opposite to above condition
        - Cerebellum – animal become hyperaesthetic – animal walks in jerky or staggering gait.
    - *In spinal cord*
        - Progressive paresis of one or both hind limbs.
- *Due to mature*
    - Ataxia, hyper excitability, varying degree of blindness, muscle tremor, nystagmus and occasionally found dead.

Diagnosis

- Live animals: Through antemortem – difficult to detect
- Opthalmoscopic examination of the eye prior to development of clinical signs.
- Dead animals: postmortem examination

Treatment

- Generally useless, high doses of albendazole may be tried
- Surgical removal of cyst, if it present in the surface of brain.

Control: Control of epidemiological factors.

TAENIA GAIGERI AND TAENIA SERIALIS

Taenia gaigeri

- *DH:* Dog & fox
- *Location:* Small intestine
- *IH:* Goat
- *Metacestode stage: Coenurus cerebralis*
- *Location:* Brain, spinal cord and intermuscular and subcutaneous connective tissue.

Taenia serialis

- *DH:* Dog & fox
- *Location:* Small intestine
- *IH:* lagoamorphs
- *Metacestode stage: Coenurus serialis*
- *Location:* Intermuscular and subcutaneous connective tissue.

ECHINOCOCCOSIS

ECHINOCOCCUS GRANULOSUS

Strain	E. granulosus granulosus	E. granulosus equinus
Distribution	Worldwide	Europe
Definitive host	Dog and carnivores	Dog and carnivores
Intermediate host	Domestic and wild ruminants, pig and man	Horses and donkey

- *Metacestode stage:* Hydatid stage
- *Location:* visceral organs – lung and liver
    - *Sheep:* 70% in lung and 25% in liver
    - *Horse and cattle:* 90% in liver

Morphology

- *Adult worm:* smallest cestode of domestic animals (6mm), consist of 3 or 4 segments
- Gravid segment is half the length of rest of segment.

Hydatid cyst

- *Types of cyst*
    - Sterile cyst: not infective for dog. In horse – 27% of cysts are sterile, in sheep - 51% of cyst are sterile and in cattle & pig most of cysts are sterile.
    - Fertile cyst: infective for dog
- *Size:* 5 – 10 cm in size (maximum of 50cm)
- *Cyst wall*
    - Outer layer: thick and made up of concentrically arranged laminated membrane
    - Inner layer: germinal layer and granular in nature.
- *Cyst fluid* - pale yellow in colour - contains 17 to 200 mg of protein per ml
- *Brood capsule* – protoscolices attached on the inner granular layer or detached and float free in the hydatid fluid – "Hydatid sand"
- *Daughter cyst*
    - Develops within the mother cyst
    - If mother cyst rupture – protoscolices & brood capsules – develops external daughter cyst

Life cycle

- Proglottids/egg —> Intermediate host (6 - 12 months) —> Definitive host (40 - 50 days)

ECHINOCOCCUS MULTILOCULARIS

- *DH:* wild canids, domestic dog and cat
- *Location:* Small intestine
- *IH:* Rodents, Large mammals and man
- *Metacestode stage:* Hydatid cyst – liver – mostly multilocule
- *Pathogenesis*
    - IH
        - Egg —> IMH —> small intestine —> Release of oncosphere —> Development of adult metacestode stage —> Liver and lung —> systemic circulation
- *Clinical signs*
    - IH: Clinical signs based on severity of infection and location of cyst
        - Heavy infection – affected organs function impaired
        - Rupture of cyst – anaphylatic shock.
        - Alveolar hydatidosis: it is due to *E.multilocularis* – malignant tumor and metastasis
- *Diagnosis*
    - IH
        - Animals: rare only by postmortem examination
        - Man: Serology : CFT and CIE
- *Treatment*
    - IH: man
        - Aspiration of cyst fluid and irrigated of cyst cavity with 2.5 % - 10% formalin (not commonly used) or own serum. Care should be taken to avoid spillage of cyst fluid while aspiration.
        - Surgical removal of cyst
        - Higher doses of mebandazloe, albendazole and praziquantel
- *Control:* Similar to *T. ovis*

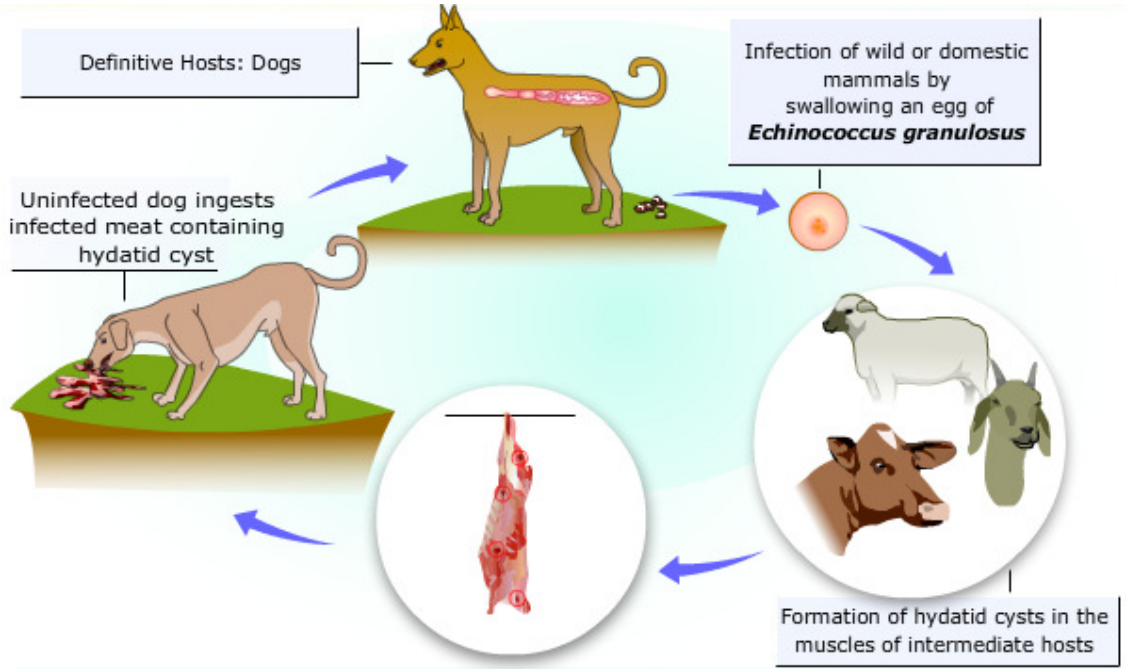
EPIDEMIOLOGY OF METACESTODE INFECTIONS IN THE INTERMEDIATE HOST

Following factors influences incidence and prevalence of infection in IMH

- *Level of environmental contamination*
    - *Infected DH:* Infected DH passes proglottids through faeces which contain numbers of infective eggs, Which acts as source of infection and contaminate feed, water and pasture land.
    - *Pasture contamination*
        - *T. saginata* and *T. solium* – night soiling habit of infected human beings
        - Other Taeniids – entry of stray dogs into pasture land and using of untreated dogs for shepherding of animals in pasture land.
- *Presence of suitable susceptible host*
    - If suitable susceptible IH are exposed to infective egg and pickup the infection harbour the metacestode stage which is infective for DH.
- *Dispersion of egg on pasture land*
    - Taeniid eggs travel upto 80m under suitable environmental conditions.
    - Movement of wind
        - *Invertebrate host:* flies(blow flies), beetles and earth worm
        - *Floating water*
- *Egg survival*
    - Normal condition survive upto 6 months
    - Senescent oncosphere – unable to penetrate the intestinal wall if it is ingested by the suitable intermediate. These type of oncospheres are used for immunization.
- *Age of the IH*
    - *Sheep*
        - Unweaned lambs are under high risk because of infected lambed ewes acts as a potential reservoir of infection for this.
        - Lambs less than 5 weeks old – maternal immunity - protection
        - Lambs greater than 5 weeks old – depletion of maternal immunity and at this time lambs starts nibbling of grasses.
        - Lambs reared on contaminated pasture land from birth onwards - develops strong immunity to reinfection.
    - *Calves*
        - In endemic area – Neonatal infection occur with oncosphere of *T. saginata* – calf not able to develop protective immunity – infection throughout their life.

- *Immune response of host (DH)*
    - Animal develops a marked protective immunity to reinfection with metacestodes. The duration of immunity is about one year in the absence of reinfection.
- *Heterologus infection*
    - Cross protection observed between Taenia species

LIFE CYCLE OF ECHINOCOCCUS GRANULOSUS



PATHOGENESIS OF DOG AND CAT TAPEWORM

General

- Heavy infection – non specific abdominal symptoms
- Diarrhoea of constipation
- Unthriftiness, pot bellied appearance

D. caninum

- Due to irritation of anal spincter, to drag its anus over the ground or other objects
- Gravid proglottids drop off from infected animals and migrate over chair, floor or clothes for some minutes – aesthetically unpleasing to owner

D. latum: Man

- Abdominal discomfort
- Pernicious anaemia – Vit B_{12} - Macrocytic hypochromic anaemia

DIAGNOSIS,TREATMENT AND CONTROL

Diagnosis

- *Characteristic egg*
    - *D. caninum* – egg pockets
    - *D. latum* – operculated egg
- *E. granulosus* – for identification of small gravid segments – dog should be treated with vermifuge to expel whole worm as such and screening for worms in faeces for subsequent two days.

Treatment

- Arecoline hydrobromide – 1 to 2 mg/ kg (contraindicated for cats)
- Bithionol – 200 mg/kg
- Hexachlorophen – 15 mg/ kg
- Niclosamide – 100 to 150 mg/ kg
- Nitroscanate – 50 mg/ kg
- Praziquantel – 5 mg/ kg
- Dichlorphen – Dog: 0.3 mg/ kg and cat: 0.1 to 0.2 mg/ kg
- Micronized mebendazole
    - Body weight less than 2 kg – 100 mg/ kg twice a day for five days
    - Body weight greater than 2 kg – 200 mg/ kg twice a day for five days
- Drug dichlorophen and micronized mebendazole are not effective against Echinococcus infection in dogs.

Control

- Control of IH + regular deworming of dog
- Dogs should not fed raw meat, freezing of fish below – 20°C for 24 hours
- Prevent capturing of small wild animals (rat, mice and hares)which act as a IH
- *D. caninum* – flea control
- *D. latum* – avoid feeding of raw fish

ECHINOCOCCUS GRANULOSUS GRANULOSUS

- *Pastoral cycle*
    - Oncosphere contaminated pasture —> sheep —> feeding of offals with fertile cyst to dog
    - Camel and reindeer act as reservoir
    - Pastoral cycle – primary source of hydatidosis for man.
- *Sylvatic cycle*
    - Wild ruminants —> wild canids
    - Less important source for human beings

Epidemiology

- *Agent*
    - Strain variation occurs (mentioned earlier)
    - Strains are differs in morphologically and chemically
    - Horse strain – host specific – not infective for human.
    - Sheep strain – not host specific – infective for human.
- *Pasture contamination: Similar to T. ovis*
    - Infection rate: less when compare to other Taeniids
        - Infected dog shed – one gravid proglottid per week
        - Egg containing oncosphere – viable in pasture for 2 years.
- *Infection of IH and DH: Similar to T.ovis*
- *Infection of man*
    - Accidental ingestion of egg, when childrens playing with infected dogs.
    - Consumption of infected dog faeces contaminated feed and water.

CHAPTER-30: TAPEWORM INFESTATIONS (CYSTICERCOSIS)

Learning objectives

To know in detail about,

- Epidemiology of cysticercosis
- Epidemiology, pathogenesis, diagnosis, treatment of Taenia saginata and Taenia solium.

CYSTICERCOSIS

- **Muscular cysticercosis**
  - Bovine cysticercosis – caused by metacestode stages (*C. bovis*) of human tape worm – *T. saginata*
  - Porcine cysticercosis - caused by metacestode stages (*C. cellulosae*) of human tape worm – *T. solium*
  - Ovine cysticercosis - caused by metacestode stages (*C. ovis*) of dog tape worm – *T. ovis*
- **Abdominal cysticercosis -** caused by metacestode stages (*C. teunicollis*) of dog tape worm – *T. hydatigena*
- **Other cysticercosis**
  - Cysticercosis of lagomorphs - caused by metacestode stages (*C. pisiformis*) of dog tape worm – *T. pisiformis*
  - Cysticercosis of rodents - caused by metacestode stages (*C. fasciolaris*) of dog and cat tape worm – *T. taeniaformis*

TAENIA SAGINATA

- *Egg* - Oval in shape
- *DH* - Man
- *Location* - Small intestine
- *IH*
  - Cattle, other ruminants
  - Wild animals - Llama and reindeer are also susceptible
  - Wildbeast, Giraffe and antelope – not susceptible
- *Metacestode* - *Cysticercus bovis*
- *Location* - Cardiac, skeletal and masseter muscles (also tongue and diaphragm)
- *Life cycle*

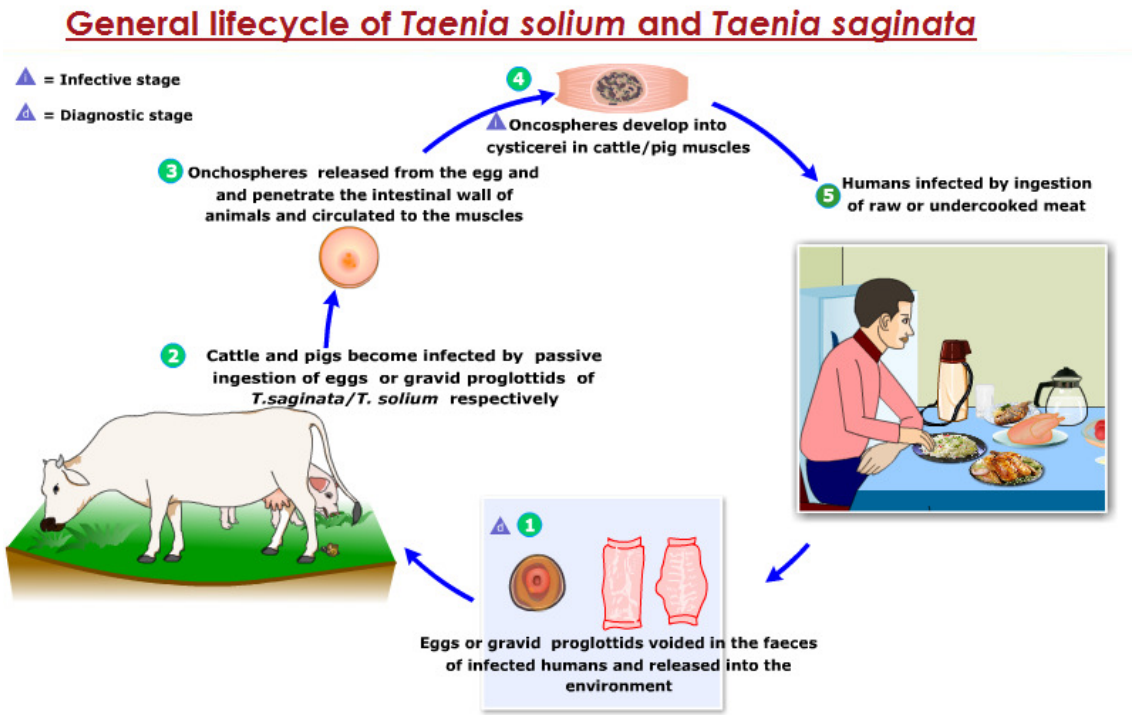
- Infected human beings —> proglottids —> IH (10 weeks) —> DH (100 days).

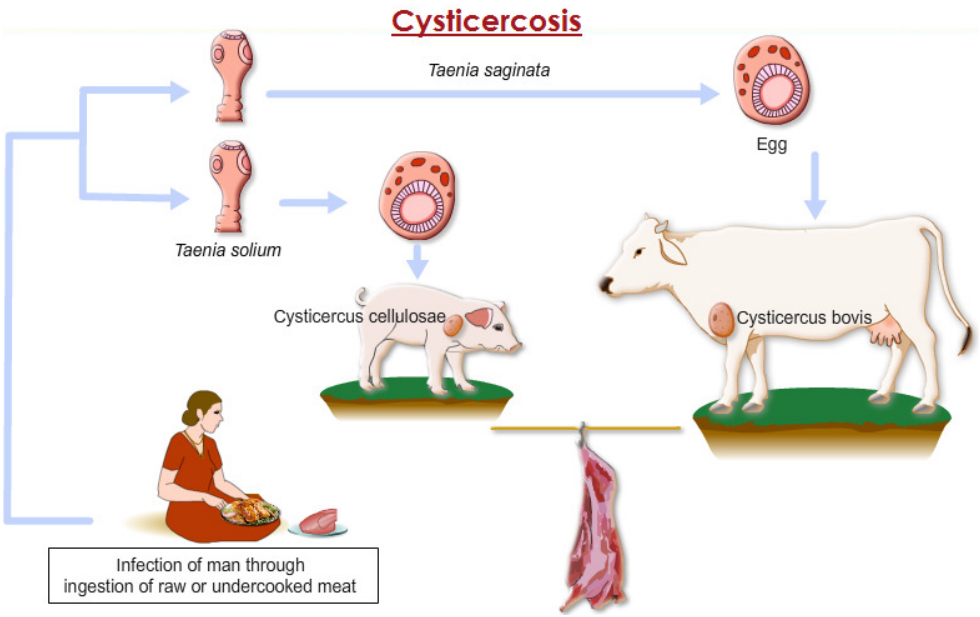
EPIDEMIOLOGY

Infection rate

- Infected person actively sheds about 10 proglottids per day. Rate of egg shedding - 10 X 2,50,000 eggs/ proglottids = 25, 00, 000 eggs per day.
- Proglottids eliminated either passively through faeces or actively migrate out of the anus due to its motility.
- If proglottids migrate out off anus, which contaminate the body surface of infected human beings and his belongings or fall on the ground.
- The survival of the eggs is strongly influenced by climatic conditions. Under wet and moist conditions, eggs may survive over a month, and increases risk to cattle. Eggs are very susceptible to dry conditions and are rapidly destroyed during the drier months. Eggs are released due to disintegration of proglottids, which may remain viable for several weeks or months in environment.
    - Infected liquid manure - 71 days
    - Sewage – 16 days
    - River – 33 days
    - Contaminated pastures – 159 days
    - Sludge – 210 days
- Poor standards of personal hygiene of infected human population is responsible for the spread of cysticercosis. In some societies such as nomadic pastoral people there is a high risk of animals becoming exposed to infected faeces.
- Abnormal eating habits of cattle due to certain mineral deficiencies (pica) may result in cravings that increase the exposure through the ingestion of faeces.
- Sewage water is an important route of dissemination of infection. Sewage water treatment has no effect on infectivity of eggs.
- Birds that consume infected sewage water and disseminate the infection.
- Infection of IH
    - Passive ingestion of contaminated pasture
    - Neonatal calves may be infected when they are handled by infected person (with motile proglottids).
    - Prenatal infection has been recorded.
- Survivability of metacestode in IH
    - Degree of infection & age of animals. Normally 4- 6 months (maximum of 9 months)
    - Neonatal infection – long periods

- Infection of DH
    - Ingestion of raw or under cooked beef (Measely beef)
    - Changing of food habits – fast foods
    - If cattle slaughtered 9 months after infection (without reinfection) - infection rate in human beings is very less.





CYSTICERCOSIS STORM

- In developed countries like Australia and Britain – human sewage is utilized as fertilizer for pasture land in the form of sludge (sedimented or bacterial digested faeces).
- If cattle are exposed to contaminated pasture land immediately after application of sludge.
- The infection rate in exposed groups is high.

TAENIA SOLIUM

- *Egg* - spherical in shape
- *DH* - man
- *Location* - Small intestine
- *IH* - Pigs (including wild boars), dog – rarely susceptible and Human being (acts as both DH and IH)
- *Metacestode stage* - *Cysticercus cellulosae*
- *Location* - Skeletal and cardiac muscles
- *Life cycle* - Similar to *T. saginata*

EPIDEMIOLOGY

- Almost similar to *T. saginata*, differences are
    - Infection rate – 10 proglottids X 40,000 = 4,00,000 eggs per day.
    - Gravid proglottids do not leave spontaneously from the host and voided passively through faeces.

Man acts as an IH

- Autoinfection - by reverse peristalsis is the most important source of infection.
- Accidental ingestion of *T. solium* eggs along with food and water.
- Nail baiting habits – especially with dirty hands.
- If man acts as an IH life cycle is arrested in metacestode stage itself.

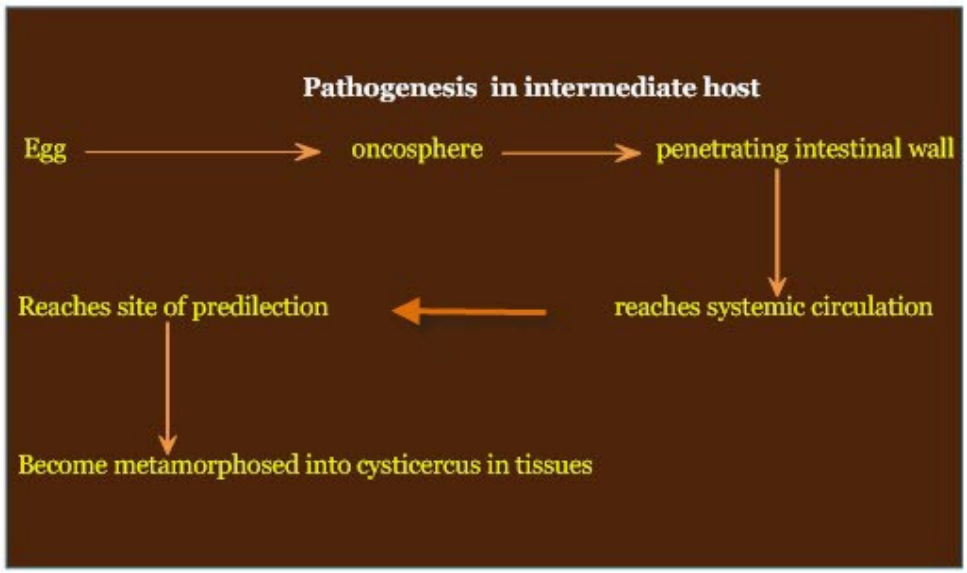
PATHOGENESIS OF T.SAGINATA AND T.SOLIUM

Definitive host

- Cysticercus reaches small intestine and become changed in to adult worm. Adult worm lay the eggs (prepatent period –100 days) – no much pathogenic changes.

Intermediate Host

- Passive ingestion gastric and intestinal juice.



- *Cattle – C. bovis* – masseters, heart, tongue and diaphragm (**measely beef)**
- *Pig – C. cellulosae* - masseters, heart, tongue and skeletal muscles **(measely pork)**
- *Man - C. cellulosae* - primarily on subcutaneous tissues and secondarily on brain and ocular cavity

NEUROCYSTICERCOSIS OR CEREBRALCYSTICERCOSIS

- If human beings infected with metacestode stage of *T. solium*.
- The *C. cellulosae* secondarily present in the ventricle of brain and in affected persons, becomes racemose and proliferate in nature.
- They are clinically characteised by neuralgia, paralysis, epileptic seizures & ends in fatal.

Clinical signs: *T.saginata* and *T. solium*

- *Definitive host*
  - Non- specific abdominal pain (epigastric pain)
  - Diarrhoea or constipation
- *IH*
  - Asymptomatic
  - Heavy infection causes myositis, myocarditis, muscular stiffness and weakness

DIAGNOSIS AND TREATMENT

Diagnosis of *T.saginata* and *T. solium*

DH

- *T. saginata* - perianal swab
- Identification of characteristic egg/ proglottids

IH

- Post mortem examination of carcasses
- Serological test – ELISA, CIEP and Western blotting

Treatment of *T.saginata* and *T. solium*

Human beings

- Praziquantel – 10 mg/kg.
- Niclosamide – 2 g (total dose)
- Paromomycin - 5 mg/kg
- Quinacrine – 7- 10 mg/kg
- Inorganic tin compounds

Animals

- Albendazloe – 50 mg/kg – effective against mature metacestode
- Praziquantel – 50 mg/kg – highly effective against both mature and immature metacestodes.

PREVENTION AND CONTROL

Prevention of *T.saginata* and *T. solium*

- Treating of infected persons (especially animal handlers)
- Public education and hygiene
- Proper meat inspection – made multiple incisions of suspected carcasses
- Freezing of carcasses
    - – 5°C for 15 days
    - – 10°C for 9 days
    - – 15 to – 30°C for 6 days
- Insist through cooking of meat (> 56°C for 5 mts.)

Immunoprophylaxis against *T. saginata*

- *Homologous vaccine* - In-vitro culturing of oncosphere of *T. saginata* releases secretory and excretory antigen
    - *Active immunization in calves*
        - Secretory and excretory antigen administered in calves before infection (after infection there is no useful).
    - *Passive immunization in calves*
        - Secretory and excretory antigen administered to periparturient cows either through intramuscularly or intramammary route.
        - Calves are immunized while consuming colostrum / milk from immunized dam.
    - *Disadvantage of Homologous vaccine*
        - In-vitro culturing of oncosphere – zoonotic significance.
- *Heterologus vaccine*
    - In-vitro culturing of oncospheres of *T. taeniaformis* releases secretory and excretory antigen, which capable of inducing high level protective immunity against heterologus parasite – *T. saginata*
    - Intramuscular injection of eggs of *T. hydatigena* in cattle – induces partial immunity against heterologus parasite.

CHAPTER-31: VERMINOUS BRONCHITIS

Learning objectives

To know in detail about

- Epidemiology, lifecycle, clinical signs, prevention and control of Dictyocaulus spp. and Metastrongylus spp.

INTRODUCTION

Lung worms of domestic animals includes,

- *Dictyocaulus filaria*
  - *Host:* Sheep and goat
  - *Location:* Bronchi
- *Dictyocaulus viviparus*
  - *Host:* Cattle
  - *Location:* Bronchi
- *Metastrongylus apri (M. elongates)*
  - *Host:* Pigs and wild pigs
  - *Location:* Bronchi and bronchioles

DICTYOCAULUS FILARIA

- Morphology
  - *Egg:* Contain fully formed larvae when laid
- Life cycle

EPIDEMIOLOGY, PATHOGENESIS AND CLINICAL SIGNS

Epidemiology

- *Development of larvae*: Suitable moisture and temperature (27°C)
- Young animals are chiefly affected, but all ages are susceptible (Chronic)

Pathogenesis

- Catarrhal parasitic bronchitis – inflammatory process spreads to the surrounding tissues and exudate frequently passes back into the bronchioles and alveoli cause atelectasis and catarrh or pneumonia.
- Secondary bacterial infection leads to extensive areas of pneumonia.

Clinical signs

- Cough and mucus exudes from the nostrils
- Dysponea – abnormal lung sounds.

DIAGNOSIS, TREATMENT AND CONTROL

Diagnosis

- Presence of first stage larvae in fresh faeces
- Eggs may found in the sputum or nasal discharge

Treatment

- Diethylcarbmazine – 22 mg/kg intramusculary for three days or a single dose of 50 mg/kg is primarily effective against the immature parasites
- Tetramisole – 15 mg/kg PO or Parentrally
- Levamisole - 7.5 mg/kg parentrally - effective against immature
- Fenbendazole – 5 mg/kg
- Albendazole - 7.5 mg /kg
- Oxfendazole - 4.5 mg/kg

Prophylaxis

- Animal must be removed from infected ground, placed on dry pastures and supplied with clean drinking water
- Moist pastures must be avoided
- Vaccine - irradiated attenuated infective larvae.

DICTYOCAULUS VIVIPARUS

LIFE CYCLE AND EPIDEMIOLOGY

Life cycle

- Similar to *D. filaria*

Epidemiology

- *Season:* Disease is common in temperate countries due to high rainfall to prevent desications of the larvae
- *Age:* Disease is more common in young calves; older animals generally stronger acquired immunity but this may waned in the absence of reinfection and adult animals may susceptible to massive larval challenge
- Wind borne field-to-field transmission of larvae by Pilobolus sporangiae has been suggested.
- The fungus Pilobolus may accumulate larvae of *D. viviparous* on the upper face of the spornangium, which when it explodes may propel the larvae as far as 3 meters.
- The infective larvae of *D. viviparus* are relatively inactive and are frequently found coiled up, showing very little movement.
- Consequently there is little migration of larvae from the faecal pads on to herbage, except during heavy rainfall and larvae which do reach the herbage are capable of limited vertical migration only
- Persistence of infection in a herd, is the survival of the parasite in an inhibited form in the lungs for several months.

PATHOGENESIS AND CLINICAL SIGNS - HUSK OR HOOSE

Prepatent phase

- It is associated with blockage of many respiratory bronchioles with an eosinophilic exudates and collapse of alveoli-tachypnoea and coughing, emphysema may develop.

Patent phase

- Lasting from days 25 to 55 is associated with adult parasite in the bronchi and trachea – severe damage to the epithelium of these organs, marked exudation in to the bronchi and blockage of air passages.
- Aspiration of eggs and larvae in to the bronchioles and alveoli occurs, leading to consolidation of lobules.

Late prepatent phase

- Late prepatent phase stage of the infection, namely epithelialization of the alveolar epithelium and hyaline membrane formation, remain and may become more marked.
- Animals show dysponea and coughing, with rapid loss of condition.
- Harsh respiratory sounds with bronchi and emphysematous crackling.
- Complication include pulmonary oedema and emphysema

Postpatent phase

- Process of recovery, clinically respiratory rate decreases, coughing is less frequent and weight gain is resumed
- Larvae which reach the lungs are destroyed by the immune response with the formation of lymphoreticular granulomas, nodule and bronchiolar obstruction
- *D. viviparus* infection is acute pulmonary emphysema and that this is related to the condition of fog fever.

DIAGNOSIS, TREATMENT AND CONTROL

Diagnosis

- Based on the clinical signs
- Demonstration of larvae in faeces
- Other pneumonic conditions may be confused with it. eg: Epizootic bronchitis or virus pneumonia, Pasteurella infection and cuffing pneumonia.

Treatment: Similar to *D. filaria*

Prophylaxis

- Grazing management should be improved, especially to provide clean pasture for young calves.
- Animals continuously exposed to infection are at little risk provided the rate of acquisition of the infection is sufficient to stimulate a satisfactory immunity and not enough to cause clinical illness.
- Prophylatic anthelmintic treatment reduces pasture contamination
- Vaccination: Two doses of 1000 irradiated larvae given at a month interval.
- Age of immunization – two months or older and exposure of infection is avoided until two weeks after second dose.

METASTRONGYLUS APRI

LIFE CYCLE OF METASTRONGYLUS APRI

EPIDEMIOLOGY, DIAGNOSIS, TREATMENT AND CONTROL

- Epidemiology
  - *Age:* four to six months pigs are highly susceptible
  - *Verminous bronchitis* and pneumonia are possibly due to secondary bacterial infection
  - Larvae of lung worm carry the swine influenza and swine fever virus
  - Larvae may transmit viruses of Teschen disease
- Diagnosis
  - Based on the demonstration of embryonated eggs in fresh faeces

- Treatment: Similar to *D. filaria*
    - Levamisole, tetramisole and fenbendazole are highly effective
- Control
    - Infected pigs should be kept on dry ground or in sites with concrete floor and their faeces should be disposed of in such a way that they do not spread the infection.
    - Clean and young pigs should be run on clean fields.
    - Infected paddocks and fields may remain infected for a considerable time, since the intermediate stage can be in the earthworm for an unknown period
    - A 3 % solution of carbathion applied to soil kills earthworms.

CHAPTER-32: TRICHOMONOSIS

Learning objectives

To know in detail about

- Epidemiology, lifecycle, intermediate hosts, pathogenesis, clinical signs, prevention and control of Trichomonosis.

INTRODUCTION

Family	Trichomonadidae
Genus	Trichomonas or Tritrichomonas
Species	*Tritrichomonas fetus*

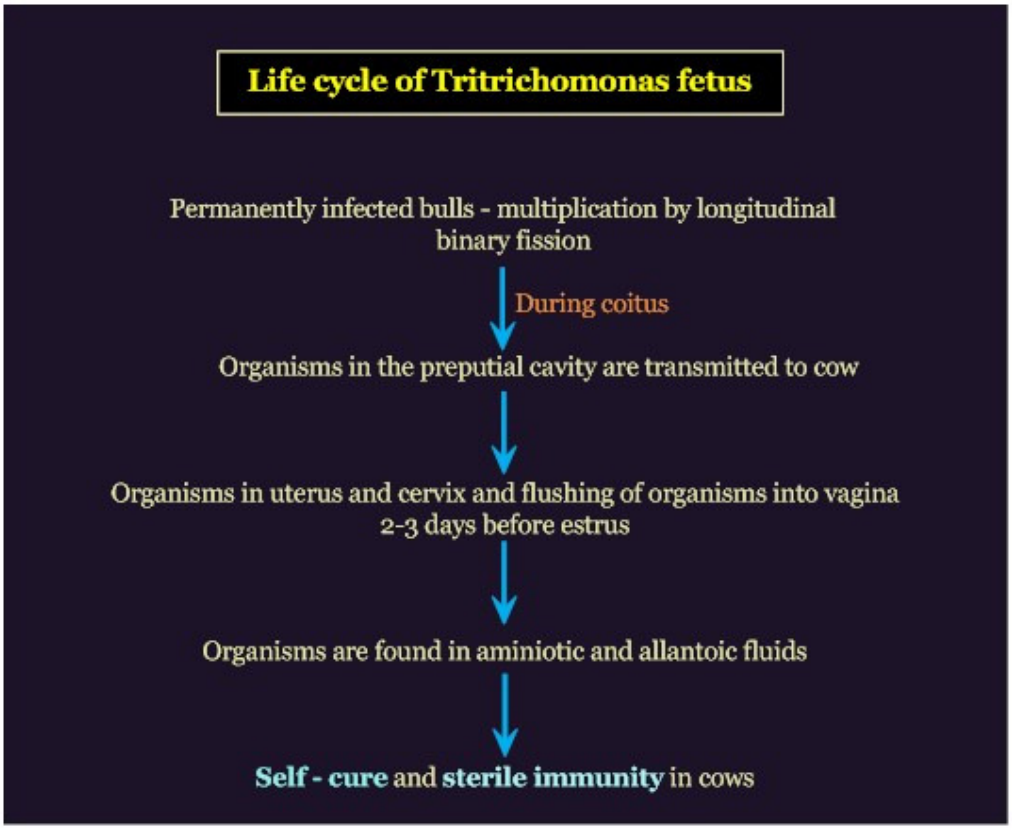
- Members of this family have 3 anterior flagella and no pelta
- They are found in the digestive tract and genital organs as commensals.
- *Tritrichomonas foetus*
    - A sexually transmitted organism causing a specific venereal disease, bovine trichomonosis or bovine trichomonad abortion in cattle and is charecterized by infertility, early abortion, pyometra and inapparent infection in bulls.

TRITRICHOMONAS FETUS

DISTRIBUTION, HOST, LOCATION AND MORPHOLOGY

- Distribution
    - Worldwide
    - India - Reported in West Bengal, Bihar, Rajastan, Jammu and Kashmir and Orissa.
- Host affected
    - Cattle, also Zebu, buffalo, pigs, horses and deer but pathogenic only in cattle
- Location
    - Cows: Uterus and internally the vagina.
    - Bulls: Preputial cavity.
- Morphology
    - Body is spindle to spear shaped (25um to 15 um)
    - It has 3 anterior flagella and one posterior flagella which is free.

LIFE CYCLE



EPIDEMIOLOGY

Strain of the parasite

- Three serological distinct strains: a) Belfast b) Manley c) Brisbane
- Outbreaks due to Manley strains are few

Incidence

- Highest in those countries where bulls are kept for natural service.

Susceptible age group

- All age groups.

Transmission

- By coitus under natural conditions.
- By teaser bulls
- By artificial insemination is the most common of the other methods using infected semen.

- By gynecological examination, i.e., use of speculum.

Survival of Trichomonas

- Does not survive when frozen in presence of glycerol at 37^0C.
- Killed at ordinary refrigeration temperature.
- At lower temperature, organisms remain viable for up to 256 days.

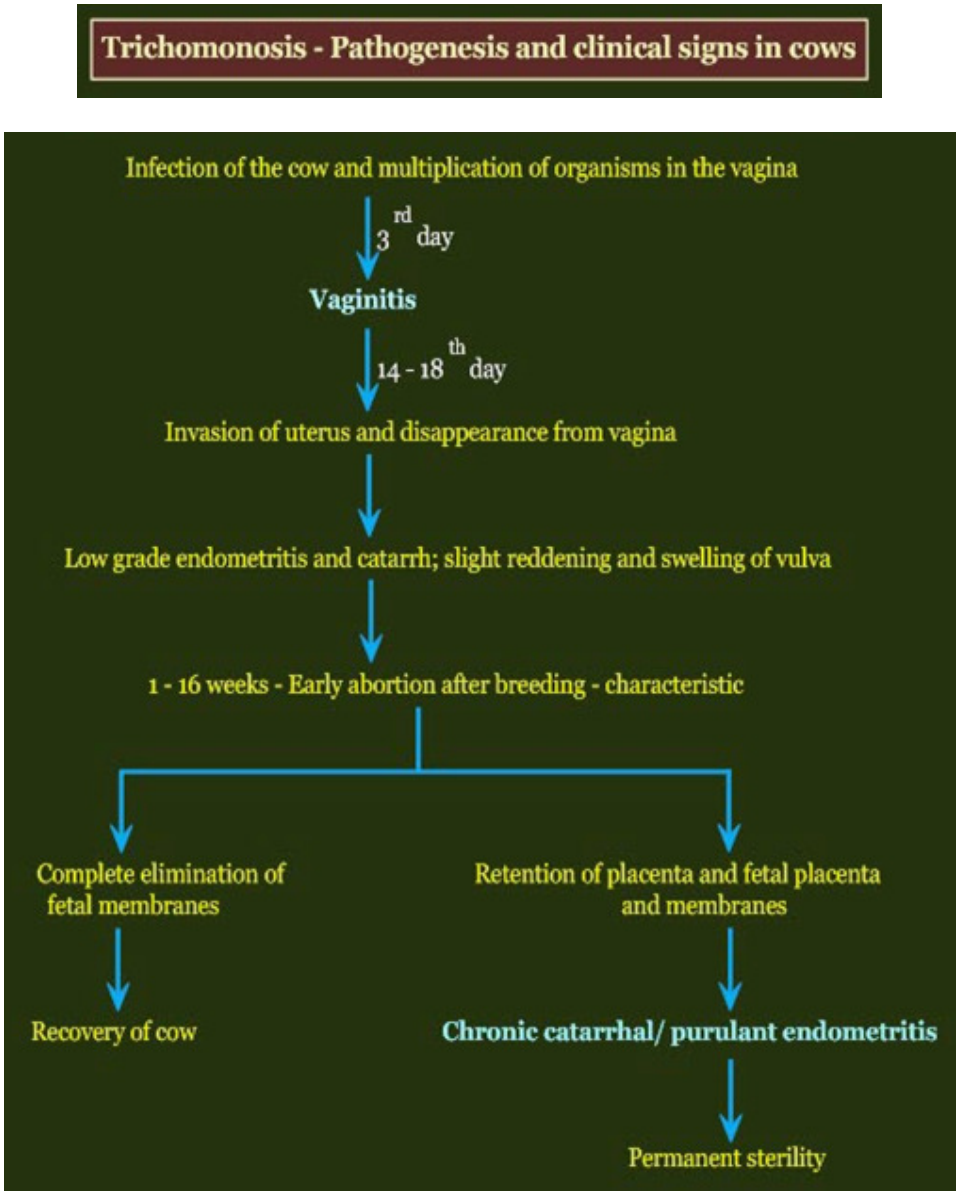
Carrier status

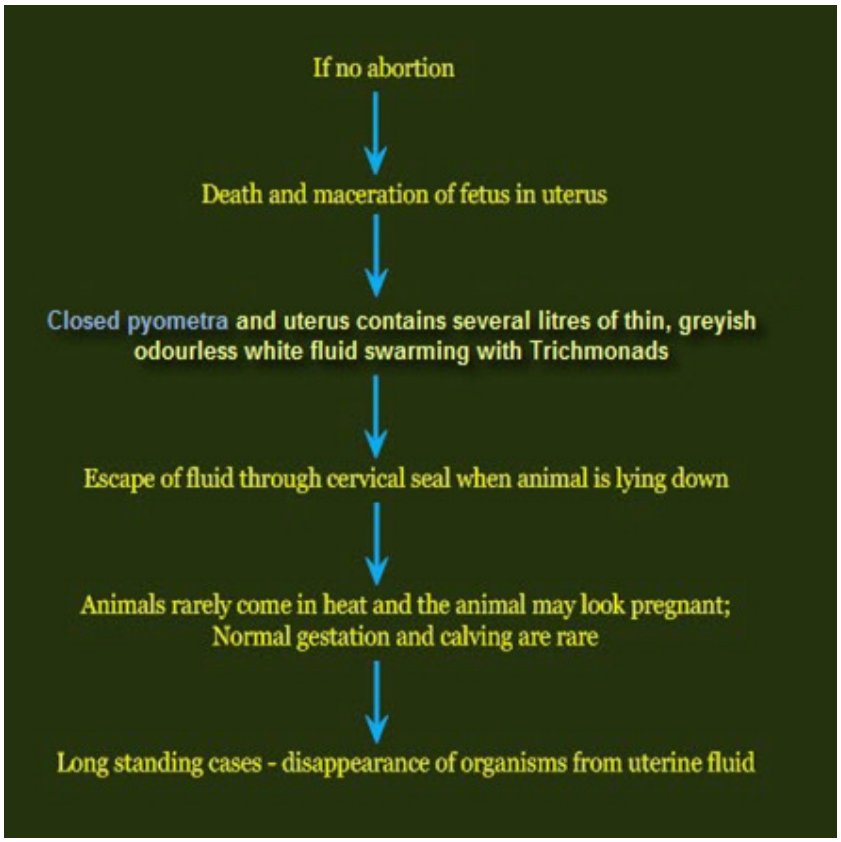
- Bulls are carrier and permanent sources of infection.
- In infected Cows, it is self-limiting, but untreated cows act as carriers for more than 1 year.

Immunity

- Little evidence as bulls becomes immuned to infection.
- Infected non-pregnant animals recover without treatment, but a small proportion of cows act as carriers and permanent sterility may occur despite immunity.
- Antibodies produced are
    - Humoral antibodies reaching systemic circulation from uterus, produced in when there is adequate infection with large number of organisms.
    - Uterine / vaginal antibodies developing locally and appear before humoral antibodies appear; the titre is high when organisms are numerous in uterine discharge and has a local control of parasites in vagina.
- Antibodies persist for 17 - 55 days and acquired through colostrum.

PATHOGENESIS AND CLINICAL SIGNS





Closed pyometra and uterus contains several litres of thin of thin, greyish, odourless white fluid swarming with organisms

Pathogenesis and Clinical signs in Bulls

- Organisms are found in preputial cavity, most commonly; also in testes, epididymis and seminal vesicles.
- Spontaneous recovery is rare.
- Pain on micturition, disinclination to serve cows, mucopurulant discharge.
- Small red nodules in preputial mucus surface
- Signs disappear from1-2 weeks after infection.
- Clinical cases: Pain and swelling disappear.

Mechanism of action

- Cell surface antigens by
    - Hydrolytic enzymes secreted by parasites which are involved in enzymatic alteration/ destruction of host cells
    - Lysosomal extracts containing neuraminidase which is cytotoxic to epithelial monocell layers.

DIAGNOSIS

- Case history
    - Cases of early abortion; increased incidence of cows returning to sevice.
    - Failure of animals to become pregnant except after repeated service.
- Clinical signs: Increase in prevalence of vaginal discharge and pyometra in a herd.
- Confirmation by demonstration of organisms in the vagina / uterine discharges/ fetus/ amniotic fluid/ allantoic fluid /fetal membranes/ placenta/ fetal stomach contents or its mouth fluids.
    - Following abortion, organisms disappear within 48 hours.
    - In bulls: Preputial washings, rarely seminal fluid or semen.
    - The organisms will be observed as jerky movements and rotation of cells on their long axis, lashing flagella when examined in warm saline.
- Examination of the above preparations by Giemsa staining, if the organisms are too low to allow diagnosis.
- Culturing in CPLM (Cysteine-Peptone- Liver extract- Maltose serum) or Diamond's media.
- Serological tests
    - Cervical mucus agglutination tests-most satisfactory
    - Vaginal mucus should be collected and atleast 3 negative examinations and 2 normal estrus periods confirm a cow as free from infection.
- Intradermal test
    - Not satisfactory
    - Inconsistent since animals maybe desensitized during acute uterine infection.

TREATMENT AND CONTROL

Treatment

- *Dimetridazole* - effective systematically for cows and bulls @50 mg/kg, I.V or P.O.
- *Bulls:* Difficult to treat, best to sell it for slaughter.
    - Injection of less than 1% acriflavin solution into the prepuce and retained for 150 minutes with thorough massage and 2 courses with 5 successive treatments.
    - 3% hydrogen peroxide – partially effective.

Control

- Slaughtering/ castration of infected bulls
- Indescriminate breeding of untested bulls should be checked and only test authorized bulls should be permitted for breeding.

- Aborted cows should be given sexual rests for 3 consecutive estrus periods.
- Artificial insemination with proper care will reduce the incidence of disease
- Maintenance of proper records of service date, calving etc.,at the farm helps in early diagnosis and control .
- Examination of breeding bulls for *T. fetus* before purchase.

TRICHOMONOSIS GALLINAE

HOST, LOCATION AND EPIDEMIOLOGY

Host

- Pigeons also turkey, rarely, chicken and other birds.

Location

- Upper digestive tract

Epidemiology

- *Transmission:* Young birds acquire infection from water contaminated with trichomonads by infected birds, through their mouth, not by feces. Pigeons transmit the infection to the squabs through pigeon milk.
- *Immunity:* Varying degres of immunity; relatively harmless strain produces immunity against virulant strains.
- *Age:* Young ones are most affected.

PATHOGENESIS

- Mild to fatal; Morbidity-90 %, but no clinical signs.
- Lesions: Small, yellowish circumscribed areas in mouth cavity after about 2 weeks of infection and soft palate is affected.
- Lesions extend to esophagus, crop and proventriculus increasing in number producing thick, caseous, necrotic nodules occluding the lumen, called "yellow buttons"
- Accumulation of fluid in crop
- Invasion of organisms into pharyngeal glands, underlying tissues, reach liver where abscess formation leads to death.

CLINICAL SIGNS AND TREATMENT

Clinical signs

- Varies with different strains; severe in young ones;
- Affected birds show depression, ruffled feathers, weak and emaciation.
- Accumulation of cheesy material/ greenish fluid in the mouth/ crop
- Chicken or turkey show drowsiness and pendulous crop
- Foul odour from mouth
- P.M lesions: Yellowish and necrotic lesions are seen in the mouth, crop, esophagus, liver and elsewhere.
- Recovered birds are immuned to reinfection but survive as carriers.

Treatment

- Furazolidone-@ 25 –30 mg/kg for 7 days in gelatin capsules
- 2-amino, 5-nitro thiazole -@ 30 –40 mg/kg for 7 days is effective.

CHAPTER-33: TRYPANOSOMA AND LEISHMANIA

Learning objectives

To know in detail about

- Epidemiology, lifecycle, pathogenesis, clinical signs, prevention and control of Trypanosoma and Leishmania spp.

TRYPANOSOMA

CLASSIFICATION, MORPHOLOGY AND DEVELOPMENTAL STAGES

Classification

Phylum	Sarcomastigophora
Sub phylum	Mastigophora
Family	Trypanosomatidae
Genus	Trypanosoma

- Members of the family, the trypanosomes are all parasitic and evolved from alimentary canal of insects.
- Many are found in blood/ tissues of mammals and birds.
- Members of this genus are found in vertebrates, in the blood stream and tissue fluids and, some in tissue cells.
- They are transmitted by blood sucking arthropod vectors with below mentioned developmental stages.
- A few species of this genus are transmitted mechanically by vectors.
- A few species are overwhelming importance as an serious cause of morbidity in man and animals in tropical regions.

Morphology

- They are characteristically leaf like in shape; they have a single flagellum and is attached to the body of the organism by an undulating membrane.

Developmental stages

- *Trypomastigote stage*
  - A blade -like form with the kinetoplast posterior to the nucleus and usually near the posterior extremity
  - An undulating membrane and free flagellum are present
  - It is usually present in vertebrate host and also in arthropods.
- *Epimastigote stage*
  - Kinetoplast and axoneme lie anterior to the nucleus with short undulating membrane
  - In few species, it is seen in vertebrate host, but principally in arthropods.
- *Promastigote stage*
  - Kinetoplast and axoneme are at the anterior tip of body with no undulating membrane
  - Seen in arthropods.
- *Amastigote stage*
  - Rounded body with kinetoplast
  - Absence of flagellum (or with short fibrils)
  - Found in vertebrate arthropods.

SECTIONS / GROUP OF TRYPANOSOMES

- Salivaria (Anterior Station/ Group B)
  - Frequently highly pathogenic.
  - Multiplication in vertebrate host is continuous in trypomastigote stage.
  - In arthropod host, metacyclic trypanosomes (epimastigotes) occur in anterior station (anterior/ midgut).
  - Transmission is by inoculation.
- Stercoraria (Posterior Station/ Group A)
  - Non- pathogenic
  - Multiplication in vertebrate host is discontinuous, typically taking place in trypomastigote, epimastigote/ amastigoteforms.
  - In arthropods, epimastigotes occur in hind gut , accumulate and passed in the faeces, which contaminate skin wounds in vertebrate host thereby transmission of parasite.

TRYPANOSOMES OF DOMESTIC ANIMALS

S.No	Parasite	Vertebrate Host	Arthropod Vector	Transmission	Develop.stage in Vertebrate host
I Salivaria (pathogenic)					
1	*T. vivax*	Cattle, Sheep, Goat & Cattle	Glossina spp (Tse-tse flies)	Cyclical	Trypomastigote
2	*T. congolense* (Nagana)	Cattle, Sheep, Horse & Pig	Glossina spp	Cyclical	Trypomastigote
3	*T. brucei* (Nagana)	Cattle, Sheep, Goat, Horse, Pig, Dog & cat	Glossina spp, Tabanids	Cyclical, Also mechanical	Trypomastigote
4	*T.evansi*	Equines, Dog, Cattle , Sheep, Goat, Cat, camel, Elephant	Tabanids, Stomoxys & other biting flies	Mechanical	Trypomastigote
5	*T. equinum* (Mal-de Caderas)	Equines	Tabanids, Stomoxys & other biting flies	Mechanical	Trypomastigote
6	*T.equiperdum* (Dourine)	Equines	Coitus (rarely tabanids)	Mechanical	Trypomastigote
7	*T. simiae*	Pigs, cattle, horses, Sheep & Goat	Glossina spp	Cyclical	Trypomastigote
8	*T. suis*	Pigs, Sheep, Goat & dogs	Glossina spp	Cyclical	Trypomastigote
9	*T. rhodesiense* (Sleeping sickness)	Man, ruminants (domestic & wild) Zoonotic	Glossina spp	Cyclical	Trypomastigote
10	*T. gambiense* (Sleeping sickness)	Man, also animals	Glossina spp	Cyclical	Trypomastigote
II Stercoraria (Non –pathogenic)					

1	*T. theileri*	Cattle	Tabanids, Hippobosca maculata	Cyclical	Epimastigote
2	*T. melophagium*	Sheep	Melophagus ovinus (sheep ked)	Cyclical	Uncertain
3	*T. theodori*	Goat	Hippoboscid	Cyclical	Uncertain
4	*T. cruzi* (Chaga's disease)	Man, Dog & cat	Reduviid bugs	Cyclical	Amastigote

EPIDEMIOLOGY OF TRYPANOSOMA

Development of trypanosomes in vertebrate host

- All trypanosomes in the final host multiply by binary fission or multiple fission (No gamete formation).
- In Salivaria group, trypanosomes divide chiefly in trypomastigote stage in blood/ lymph glands.Intracellular stages have been found in *T. congolense*, *T. brucei*, *T. evansi*, *T. equinum* and *T. equiperdum*.
- In Stercoraria group, trypanosomes divide in the epimastigote and amastigote stage. In *T. cruzi*, multiplication is intracellular in reticulo-endothelial system (RES) and striated muscles , eg. heart.

Development of trypanosomes in vectors (Transmission)

- *Cyclical transmission*
    - Salivaria group- In arthropods, trypanosomes multiply, undergo morphological transformation in digestive tract and proboscis and upon feeding, the new infection is transmitted by inoculation.Eg. *T. vivax*, *T. brucei*, *T. congolense* by tse-tse flies.
    - Stercoraria group-Multiplication and transformation of trypanosomes occur in gut and migrate to rectum and passed in faeces. Eg. *T. theileri*, *T. melophagium* by Tabanid spp, *Hippobosca* spp, *Melophagum ovi*.
- *Mechanical transmission*
    - Trypanosomes are transferred from one mammalian host to another host by interrupted feeding of biting insects. Eg. Tabanids and Stomoxys.
    - The trypanosomes on the proboscis donot multiply and die quickly so that transmission is possible only for few hours.Eg. *T.evansi* and *T. equinum* by Tabanids/ Stomoxys.
    - Vampire bats can also be the vector in the transmission of the *T. evansi* which multiply and survive for a long period.

- Mechanical transmission can also occur in some salivarian group transmitted cyclically in tse-tse flies. Eg. *T. vivax* and *T. brucei*.

Transmission by oral route

- Dog, cat and wild carniovores may become infected by eating fresh carcasses or organs of animals died of trypanosomiasis, the parasites penetrating oral abrasions.

Immunity

- Trypanosomes are extremely elastic antigenically.
- Members of subgenus Trypanozoon especially change in antigenic constitution between original infection and the first antigenic relapse and successive relapse.
- A succession of variation takes place and the parasite manages to survive. However, it reverts to original antigenic structure upon cyclical transmission.
- As many as 20 or more variants have been reported. The culture forms lack the coating.
- The destruction of the trypanosomes take place through the complement mediated lysis of trypanosomes/phagocytosis of agglutinated and opsonized trypanosomes by macrophages, monocytes, neutrophils and eosinophils.
- The immune response (IgM and IgG) is directed against the surface glycoprotein coat.
- Despite the effective mechanism of host, trypanosomes cause severe and prolonged infection resulting in successive waves of parasitaemia at intervals of few days, each wave representing the multiplication of a new antigenic type. *T. cruzi* evades immunity not by antigenic variation but by intracellular sequestration

Sexuality

- No evidence of sex in trypanosomes.

Cultivation

- Relatively easy to cultivate in any artificial media like NNN media.

TRYPANOSOMA EVANSI

- Disease
  - Surra in all hosts
  - Tebersa in camels
  - Murine in horses
  - 1st trypanosome shown to be pathogenic for mammals and is a intercellular parasite in blood and lymph.

- Host: Camel, Horses, Donkeys, Cattle, Buffalo, Sheep Goat, Dog, Cat, elephant and other animals (tiger, fox, jackal, hyena, orangutan and mongoose), rodents, rabbits and guinea pig.
- Vectors: Tabanids, Stomoxys, Haematopota, Hippobosca, Ornithodorus tick.
- Location: Blood and lymph.
- Morphology
    - *T. evansi* is monomorphic but polymorphism sporadically
    - Slender, thin, indistinguishable from T. brucei and 15-34 µm length.
    - Has sub-terminal kinetoplast, well developed undulating membrane and prominent free flagellum.
- Epidemiology
    - Host susceptibility
        - Increased incidence is in equines and dogs than cattle and buffaloes.
        - Buffaloes greater than cattle; Imported dogs greater than native dogs; pups greater than adults.
    - *Transmission*
        - Mechanically biting flies- Tabanus, Stomoxys, Haematopota, Chrysops, Hippobosca and Lyperosia; Ticks-Ornithodorus spp.
        - Interrupted feeding of flies from one host to other as parasite do not survive in the proboscis of the flies after 4-72 hours.
        - Mechanically by non-blood sucking flies which transmit the diseases by picking the infection from infected meat or open lesions or mucous membrane of susceptible animals.
        - Dogs get infection by ingestion of tissues from infected carcasses.
        - During mass vaccination when sterilization procedures are inadequate.
    - *Carrier status*
        - Several wild and domestic animals carry the latent infection, e.g. cattle and buffaloes.
    - *Stress factors*
        - Strain, malnutrition, vaccination against viral and microbial infections and intercurrent diseases may reduce the vitality of animals and increase the susceptibility to trypanosomes.
    - *Trypanotolerance*
        - Animal hosts are parasitaemic for prolonged periods of time, but generally remain in good health.
        - Half breeds resulting from Jersey bull and N'-Dama female show good tolerance to trypanosomosis
    - *Incidence*

- In India, more common in areas where the environment for breeding of insect vectors being the most suitable for transmission.
- Incidence coincides with rain, flood and inundations.

PATHOGENESIS AND CLINICAL SIGNS

Pathogenesis

- Course of the disease depends on the factors like genetic constitution of the host, previous exposure to disease and the virulence of the infection, strain of the parasite and species of the host.
- Mechanism responsible for anaemia-3 factors
    - Haemolysis of RBCs by trypanosomes.
    - Increased erythrophagocytosis mediated by antibody on antigen with complement
    - Haemodilution due to increased plasma production.
    - Others- reduced erythropoiesis, intravascular coagulation and death.
- *Causes of death*
    - Progressive anaemia
    - Intravascular coagulation
    - Hypoglycemia due to disturbed metabolism by malfunction of adrenals, pancreas and thyroid and due to direct utilization of glucose by the parasite.
- *Biochemical changes*
    - Increase in globulin and decrease in albumin globulin ratio by 14.4%.
    - The infection is severe in dogs and horses than cattle and buffaloes.

Clinical signs

- *Equines*
    - Incubation Period: 4 to 9 days
    - Severe in horses
    - Donkeys are resistant
    - Death within few days to few months depending on the virulence of strain of the organism.
    - Intermittent fever (44°C) and anaemia are main symptoms.
    - Transient local/ urticarial eruptions/ plaques on neck/ flanks; edema of legs and lower parts of the body.

- Hemorrhage at the junction of the skin and mucous membrane at nostrils, eyes and anus and petechial haemorrhage on mucous membrane of eye or vulva in mares; pale and dirty yellowish mucous membrane
- Staggering gait, paraplegia, laboured breathing.
- Diarrhoea ,constipation, urine with albumin and blood and dark yellowish; Increased thirst, signs of catarrh with yellow tinged nasal discharge.
- Less common: Enlargement of lymph nodes, keratitis, petechiae in vaginal mucus membrane.

- *Cattle and Buffaloes*
  - Course varies from a symptomless carrier to per acute infection.
    - Per acute - Death within in 2-3 hour; nervous form and death in convulsion.
    - Acute - Dull, sleepy, staggering gait, encircling movements, nervous excitement, beating head against wall /manger, apparent blindness, stamping of feet, bellowing, groaning, twitching of muscle, shivering of body, coma and death within 6-12 hours.
    - Sub acute/ Chronic -Parasitaemia is usually low and doesn't coincide with temperature and afebrile animal be positive for parasites, edema of legs, diarrhea, intermittent fever, rapid pulse, dullness, sleepy, bilateral lachrymation, progressive emaciation and death; abortion reported in buffaloes.
  - PM findings: Spleenomegaly, hepatomegaly, enlargement of lymph nodes and kidney, petechial haemorrhage at the junction of skin and mucous membrane
- *Dogs*
  - Incubation period: 5 to 6 days; untreated dogs die within 12 months, acute and fatal; more serious in pups and imported dogs.
  - Fever, anorexia, edema of head and throat, corneal opacity or blindness.
  - Laryngeal edema resulting in change of voice simulating rabies.
  - Muscular spasm of limb, staggering gait, excitement like biting kennel bars simulating rabies.
- *Cats:* Chronic cases are reported; at times may be fatal.
- *Sheep and Goats:* Rare; intensity of symptoms are also moderate; emaciation and anaemia may be present.
- *Pigs:* non-pathogenic and low parasitaemia.

DIAGNOSIS

- History of prevalence of *T. evansi* infection and biting flies like tabanids.
- Clinical signs.
- Differential diagnosis

- Anthrax
- Snake bite
- Nervous ketosis,
- Organic and inorganic poisoning,
- Milk fever
- Hypovitaminosis,
- Milk fever,
- Brain tumor/ cyst

- Direct examination
  - In acute cases-demonstration of organisms in blood smears stained freshly with Giemsa stain.
  - Chronic cases- examination of thick and thin blood smear at the height of the temperature will be positive/ lymph node puncture smears.
- Chemical tests
  - To detect the changes in the chemical composition of the blood produced by *T. evansi*, like alteration of proteins
  - Non-specific and less reliable.
    - Mercuric chloride test, Formal gel test, thymol turbidity test are used for camels.
    - Stilbamide test-diagnosis of latent infection in bovines in India.
- Animal inoculation tests
  - For detection of latent infection of bovines and more reliable than microscopic examination. Albino mice/ rat are most suitable for detecting sub patent infection; guinea pigs and rabbits are less susceptible. Injection of 2 ml of blood intraperitoneally into the mice causes organisms appear after 2-3 days in the mice.
- Immunodiagnostic tests
  - IFAT, CFT, ELISA & IHA are most reliable tests.
  - Allergic tests are doubtful- 1ml antigen is injected at the side of the neck with a 2nd dose after 48 hours. Infected cases show hot, edematous, painful swelling and healthy cases show circumscribed, hard, nodular swelling.

TREATMENT

Treatment

- Quinapyramines (Antryside) - Prophylactic (Triquin)- Drug of choice.
  - Antryside methyl sulphate solution

        - 3mg/kg as 10% aqueous solution, S/C; effective against *T. evansi*, *T. congolense*, *T. brucei*, *T. vivax*.
    - Antrycide chloride as antrycide prosalt
        - In combination with antrycide methyl sulphate -3 parts of A. M.S + 2 parts A. chloride
        - A solution of 3.5 gm in 15 ml water is given @ 7.4 mg/kg; effective against *T. evansi* and *T.brucei*, for 3 months.
- Suramins (Sulphonated naphthalamines) - Curative (Naganaol, Antrypol)
    - Effective against *T. evansi*, *T. brucei*, *T. equiperdum*.
        - Cattle: 0.5 g/ 45 kg followed by half dose after 2 weeks, I/V.
        - Horse: 4g/ 45 kg as single dose, I/ V.
        - Dog: 3.5 ml- 10%solution followed by 2nd dose at 3-4 weeks interval, I/V.
- Diamidines: Diminazene aceturate (Berenil) - Curative,@ 3.5 mg/ kg ;
    - cattle - 10 mg /kg; S/C or deep I/M
    - Very effective against *T. congolense* and less effective against *T. evansi*, *T. brucei* , in bovines, ovines and caprines.
    - Dog and camels react to dogs; resistance may occur to the drug.
- Phenanthiridine compounds
    - Homidium bromide and Homidium chloride- 1-2% sol @ 1gm/kg b. wt, I/M against *T. congolense* and *T.vivax* but not against *T. evansi*.
    - Pyrithridium bromide - 4 % sol @ 2mg/ kg b. wt, S/C or I/M against *T.congolense* and *T.vivax*.
    - Isometamidium chloride - 0.5 mg / kg b. wt, against *T. simiae* in pigs.

CONTROL

- *Control is difficult and the factors responsible for the wide spread existence are*
    - Latent cases of cattle and buffalo,
    - Reservoir wild animals
    - Drug resistant strains and
    - Wide spread prevalence of vectors
- *Control program consists of*
    - Treatment of affected animals
    - Chemoprophylaxis
    - Disposal of manures properly
    - Proper drainage

- Regular spraying of insecticides: Sprinkling of kerosene oil over water in ponds, ditches, streams or insecticides to kill adults and aquatic larvae.
- Removal of moist beddings, hay, dung from stable.
- Keeping animals clean by regular grooming.
- Segregation of sick animals from healthy during outbreak in fly -proof shed.
- Identification of endemic areas by regular examination of blood of animals for implementation of preventive measures.

TRYPANOSOMA EQUINUM

- *Disease:* Mal de - caderas or Disease of Hip
- *Distribution:* Central and South America
- *Host:* Horses
- *Vectors:* Tabanid flies and Stomoxys.
- *Epidemiology:* Mechanical transmission by biting flies
- *Pathogenesis and Clinical signs*
  - Usually, chronic, rarely acute and death in 2-6 months after infection.
  - Incubation period is 4 to 10 days.Emaciation in early stage followed by weakening of hind quarters in staggering gait and recumbency.Conjunctivitis, keratitis, edema of the eyelids, cutaneous plaques on neck and flank.
  - Post mortem findings: spleenomegaly, anaemia, ascites, kidney with petechiae.
- *Diagnosis*
  - Demonstration of organisms in peripheral blood smear
  - In chronic cases, animal inoculation test.
- *Treatment and Control:* Similar to Trypanosomiasis in horses

TRYPANOSOMA EQUIPERDUM

- *Disease:* Dourine, Equine Syphilis, Breeding paralysis
- *Distribution:* Africa, America and Russia.
- *Host:* All equines (horses are highly susceptible than mules/ donkeys)
- Epidemiology
  - Mechanically by coitus, rarely by biting flies
  - Foals get infected through contamination of nasal/ conjunctival membrane with vaginal discharge of infected mare.
  - Carriers: Jackals.

- *Pathogenesis*
    - Incubation period - 2 to 12 weeks
    - Phase of edema (4-6 weeks): Mare-edema of genitalia, nymphomania, mucoid vaginal or urethral discharge and ulcers in vaginal mucosa; In severe cases, abortion and frequent micturition. Stallion- Swelling of prepuce and scrotum
    - Urticaria phase: Oval or round plaques (3cm dia) under skin on flank are pathognomonic and are called Dollar spots; Anaemia, emaciation, intermittent fever, lymph node enlargement
    - Paralytic phase: In coordination, unilateral paralysis of hind limbs, nostrils, ears, lips, recumbency and death(50-70%)
- *Diagnosis*
    - Clinical signs.
    - Smears from vaginal mucus membrane and urticarial swellings.
    - Laboratory animal inoculation or into a dog.
- *Treatment:* Suramin
- *Control*
    - Slaughter of infected animals
    - Castration of affected animals
    - Treatment with suramin.

LEISHMANIA

INTRODUCTION

Family	Trypanosomatidae
Genus	Leishmania

- Members of this genus primarily occur in mammals and other hosts like lizards and bats.
- They cause disease in man, dogs and various rodents.
- The parasite, Leishmania is heteroxenous, transmitted by sand flies.
- It is found amastigote stage in the cells of its vertebrate host and in the promastigote stage in the intestine of sand fly and in culture.
- They undergo morphological transformation and multiplication in vectors.
- It is major importance as a disease in man.
- In human, 3 types are reported
    - Visceral

- Cutaneous
- Muco-cutaneous.

LEISHMANIA SP. OF DOMESTIC ANIMALS

S.no.	Leishmania spp	Host	IMH/ Vectors	Location	Disease
1	*L. tropica* *L.major* *L.aethiopica* (India)	Man, Dogs, Gebrils and other wild rodents	Blood sucking Phlebotamine sand flies - *P.argentipes* (Promastigote)	Macrophages and other cells of RES (amastigote)	Cutaneous Leishmaniasis, Oriental Sore, Delhi boil, Muco - Cutaneous Leishmaniasis, Old World Leishmaniasis, Aleppo Button
2	*L .donovani* *L. infantum* (India)	Man, Dogs, and other wild Carnivores	Blood sucking Phlebotamine sand flies - *P.argentipes* (Promastigote)	Macrophages and other cells of RES of Spleen, Liver, Bone marrow and other sites (amastigote)	Visceral Leishmaniasis , Kala- azar , Black Fever, Dum Dum Fever, Asia Fever.
3	*L. braziliense*	Man and Lower animals	Blood sucking Phlebotamine sand flies - *P.argentipes* (Promastigote)	Macrophages and other cells of RES (amastigote)	Muco - Cutaneous Leishmaniasis

LOCATION, MORPHOLOGY AND LIFE CYCLE

Location

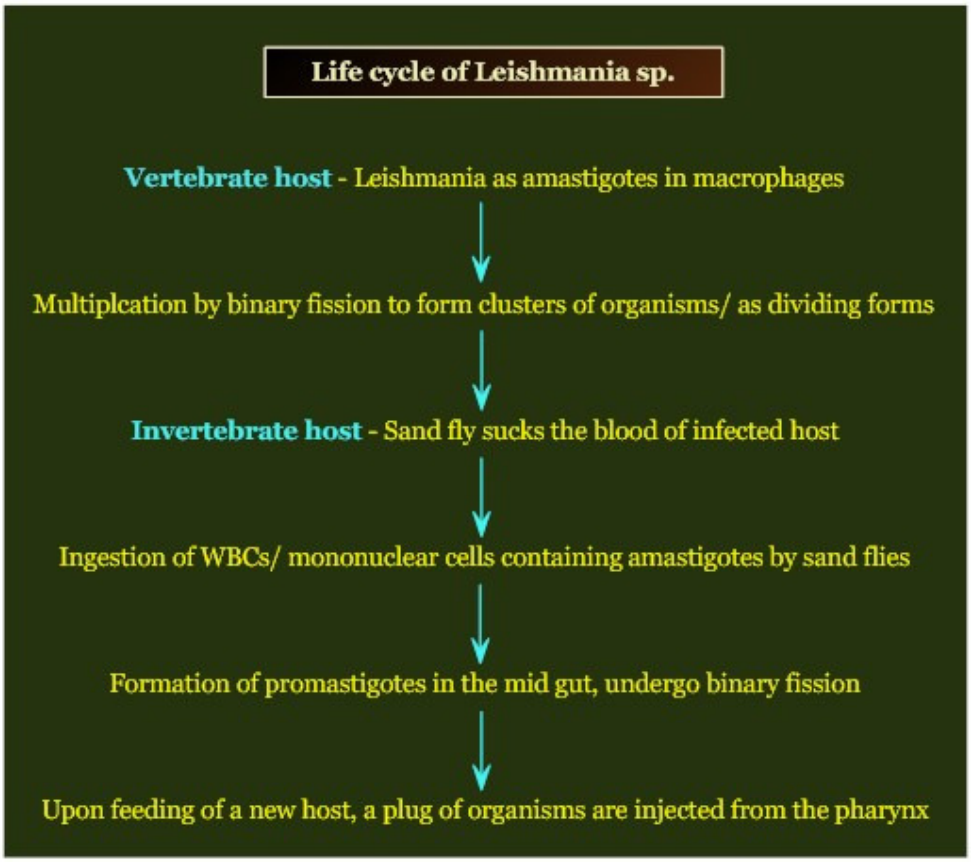
- *Vertebrate host:* Amastigote form in macrophages and other cells of RES in skin, spleen, bone marrow, lymph nodes, muscles and other sites or also in large mononuclear cells in blood.
- *Invertebrate host:* Promastigote form in gut.

Morphology

- Amastigote in vertebrate host is ovoid / round; only nucleus and kinetoplasts are seen in stained preparation.
- Romanowsky staining - Cytoplasm appears as blue, nucleus as red and kinetoplast as purple.

- Promastigotes in vectors are spindle shaped (3.5 µm).

Life cycle



EPIDEMIOLOGY

- Transmission
  - By feeding or inoculation on infected host by sand flies.
  - Infection also occurs when infected sand flies are crushed on the skin. Eg.Kala- azar fever.
  - Mechanical transmission by biting flies.
  - By needle passage as a possible method
  - Visceral leishmaniasis-Direct infection by means of excretion of infected individuals.
- Reservoir status: Dogs, cats, opossums and rodents are reservoirs.
- Season: Countries with hot and dry climate.
- Immunity:
  - In cutaneous form, spontaneous cure is followed by immunity for up to 20 years
  - In visceral form, Infection persists in the animal.

PATHOGENESIS

Cutaneous Leishmaniosis

- Differentiated as dry or moist or mildly erthymatous encountered in Old World and the muco-cutaneous form is encountered in New world.
- Dry form is more common and is urban in dogs; moist form is prevalent in rodents and gebrils. incubation period: 3 to 4 weeks after bite.
- *Man-Dry form:* Promastigotes in macrophages of skin undergo maturity and resultant amastigotes are released after rupture of cells and infect other cells.
- The first detectable lesion occurs. The first detectable lesion occurs after 3-4 weeks after bite by the fly as a reddish papule, developing into a crust, forming a shallow ulcer which enlarges gradually and reach several centimeters in diameter.
- Coalescence of ulcers occurs and spread over large areas. In uncomplicated cases, healing occurs in 2-12 months leaving a deeply pigmented and depressed scar. Infection is rarely fatal.
- *Dogs:* Similar to man; Ulcers on different parts of the body - lips and eyelids.

Visceral Leishmaniasis

- Indian Kala – azar fever is important & is highly fatal in man. incubation period: 10 days to more than year.
- *Man:* Clinical signs appear usually after 3-6 months beginning with irregular fever; malaise, head ache, occasional abdominal pain, dysentery or diarrhea and bleeding of mucus membrane of mouth and nostrils; Darkening of the skin and hairs become brittle; Progressive enlargement of the spleen and liver fills almost entire abdomen which is striking feature of the disease.
- In advanced cases, there is ulceration of the digestive tract and great emaciation.
- In untreated cases, mortality is 70- 90 %, higher in adults than infants due to intercurrent disease.
- Post Kala-azar dermal Leishmaniasis: Following recovery after treatment, whitish spots develop into lentil- sized nodules in the skin on the face and neck.
- *Disease in RES:* Increase in RES cells and the most affected organs are spleen , liver, bone marrow and lymph nodes; enlargement of malphigian corpuscles in spleen and fatty infiltration of Kupffer cells in liver; Macrophages , myelocytes and neutrophils of the bone marrow are filled with parasites; enlargement of lymph nodes, progressive leucopenia, monocytosis and anaemia.
- *Dogs:* Naturally, no involvement of dogs with Indian Kala- azar and otherwise pathogenesis is similar to man, either cutaneous or visceral lesions.
- Anaemia, emaciation and diarrhea resulting in death.

- In chronic cases - Cutaneous lesions like eczema, scurfy degeneration and loss of hair are characteristics of the disease, moreover Cutaneous ulcers in some cases around lips, eyelid and nostrils; spectacles due to depilation of hair around the eyes.

DIAGNOSIS

- Clinical signs
- Lesions in living condition
- Demonstration of parasites in peripheral blood smears, thick blood smears and centrifuged citrated blood for visceral form.
- In Cutaneous form- examination of skin scrapings from the edge of the lesion by Romanowsky staining is the method of choice in dogs.
- Examination of biopsy material from lymph nodes, spleen, and sternal/iliac crest puncture of bone marrow in visceral form.
- Culture of blood in NNN media for promastigotes.
- *Immunodiagnostic tests:* CFT, IFA, IHA and Indirect agglutination tes, Napier's aldehyde test, Chopra's antimony test, formal gel test and Urea stilbamide test.

TREATMENT AND CONTROL

Treatment

- In man, successful with organic antimony compounds and less effective in dogs**.**
    - *Cutaneous form:* Anthiomaline (lithium antimony thiomalate)- 1 ml rising by 0.5 to 2.5 ml on alternate days, 4-6 times.; I/ M.
    - *Both forms:* Pentavalent sodium stilbo gluconate(Pentastam) and trivalent sodium antimonyl gluconate(Triostan) are widely used.
    - *Visceral form:* Diamidines- Pentamide isothiocyanate – 2 mg /kg- 6 injections; 3 mg /kg - 7 injections; 4 mg/ kg 13 injections at 2-3 hrs interval. I/ V; very effective against L. donovani infection.

Control

- Control of sand flies by various CHC and OPC.
- Measures against breeding places of flies by removal of dense and decaying vegetation around houses,
- Fine mosquito nets in square should be used for human beings.
- Dogs with dermal lesions should be treated or destroyed.
- Control of stray dogs.

CHAPTER-34: THEILERIOSIS AND BABESIOSIS

Learning objectives

To know in detail about,

- Epidemiology, lifecycle, intermediate hosts, pathogenesis, clinical signs, prevention and control of Theileriosis and Babesiosis.

THEILERIOSIS

INTRODUCTION

Family	Theileridae
Genus	Theileria

- Members of this family occur in mammals.
- They have small, round, ovoid, irregular or bacilli form merozoites, as the parasites are pleomorphic and occur in lymphocytes, histocytes and erythrocytes of vertebrate host.
- Ixodid ticks are vectors in their life cycle transmitting the parasites to animals.
- The species are of great economic importance to dairy animals.
- Theileriosis is characterized by fever and lymphoproliferative disorders which may be associated with leukopenia/ anaemia.
- Location: Lymphocytes and erythrocytes (sometimes in monocytes).

THEILERIA SP. OF DOMESTIC ANIMALS

S. No.	Theileria	Vectors	Disease	Pathogenicity
Cattle and Buffalo				
1	*T.annulata* (India)	Hyalomma marginatum Hyalomma anatolicum (Transtadial transmission)	Tropical Theileriosis, Mediterranean coast fever, Tropical Piroplasmosis, Egyptian Fever	Highly pathogenic
2	*T. parva*	Rhiphicephalus	Bovine Theileriosis, East	Highly

		appendiculatus Hyalomma anatolicum (Transtadial transmission)	Coast fever, Turning Sickness, African Coast Fever, Corridor disease, Rhodesian tick Fever, Rhodesian Red Water disease	pathogenic
3	*T. mutans* (India)	Rhiphicephalus appendiculatus Haemophysalis bispinosa Haemophysalis punctata (Transtadial transmission)	Benign Bovine Theileriosis, Turning Sickness African Coast Fever	Non-fatal/ Slightly pathogenic
Sheep and Goat				
1	*T. hirci* (India)	Rhiphicephalus bursa Hyalomma anatolicum anatolicum (Transtadial transmission)	Malignant Theileriosis	Highly pathogenic
2	*T. ovis*	Rhiphicephalus bursa Haemophysalis spp (Transtadial transmission)	Benign Theileriosis	Non - pathogenic

TROPICAL THEILERIOSIS

- Parasite: *Theileria annulata*
- Morphology
  - Erythrocytic forms are indistinguishable from *T. parva*.
  - 80 % are round/ annular (1.5 mm) and rest are oval or comma shaped and are anaplasma like organisms.
  - One or more parasites are found in each RBC.
  - Multiple infection with 4-7 parasites in single RBC observed in severe cases.
  - Binary fission of erythrocytic forms result in formation of 2-4 daughter individuals.
  - Micro and Macro schizonts(Koch's Blue bodies) can be seen in the lymphocytes of spleen and lymphnodes.
  - Schizonts can also be seen monocytes or in free state lymph node biopsy smears. Schizonts may also be seen in peripheral blood smears.

- Life cycle
    - In vertebrarte host- Asexual reproduction - Schizogony
    - *Vectors*
        - Sexual reproduction- gamogony and sporogony
        - Transtadial transmission occurs in ticks. Infection from tick larvae to nymphs or nymphs to adults.
    - *Incubation Period:* 15 days following tick transmission.

EPIDEMIOLOGY

Age

- Both young calves (even 2-3 days old) and adults are affected.

Breed

- Highly virulent in European breeds of cattle, but all breeds are susceptible, especially in dairy animals and crossbred animals and less virulent in local zebu breeds; Mortality in exotic cattle is up to 70%.
- In endemic areas, all animals are virtually infected but the case- fatality is 10 -20% and is confined to calves.

Transmision:

- Transtadial transmission (stage to stage) by ticks of *Hyalomma spp.*
- Infection from tick larvae to nymph or nymph to adult occurs.
    - Sporogony & gamogony takes place in salivary gland if the tick.
    - Schizogony takes place in the host – lymphocytes & RBCs- erythrocytic stages.
- Congenital transmission occasional in calves.

Carrier status

- Indigenous cattle without clinical signs remain as a constant source of infection to susceptible animals; Buffaloes, may act as carriers; Recovered animals with long lasting immunity act as carriers; Wild animals may also transmit the disease.

Immunity

- Cell-mediated immunity is proved by leucocyte migration inhibition test.
- No cross immunity occurs between *T. parva* and *T. mutans.*

- Immunity is developed by all 3 stages- sporozoites, schizonts and erythrocytic stages; each developmental stage elicits a homologous immune response and may provide a partial protection against infection with all other stages.

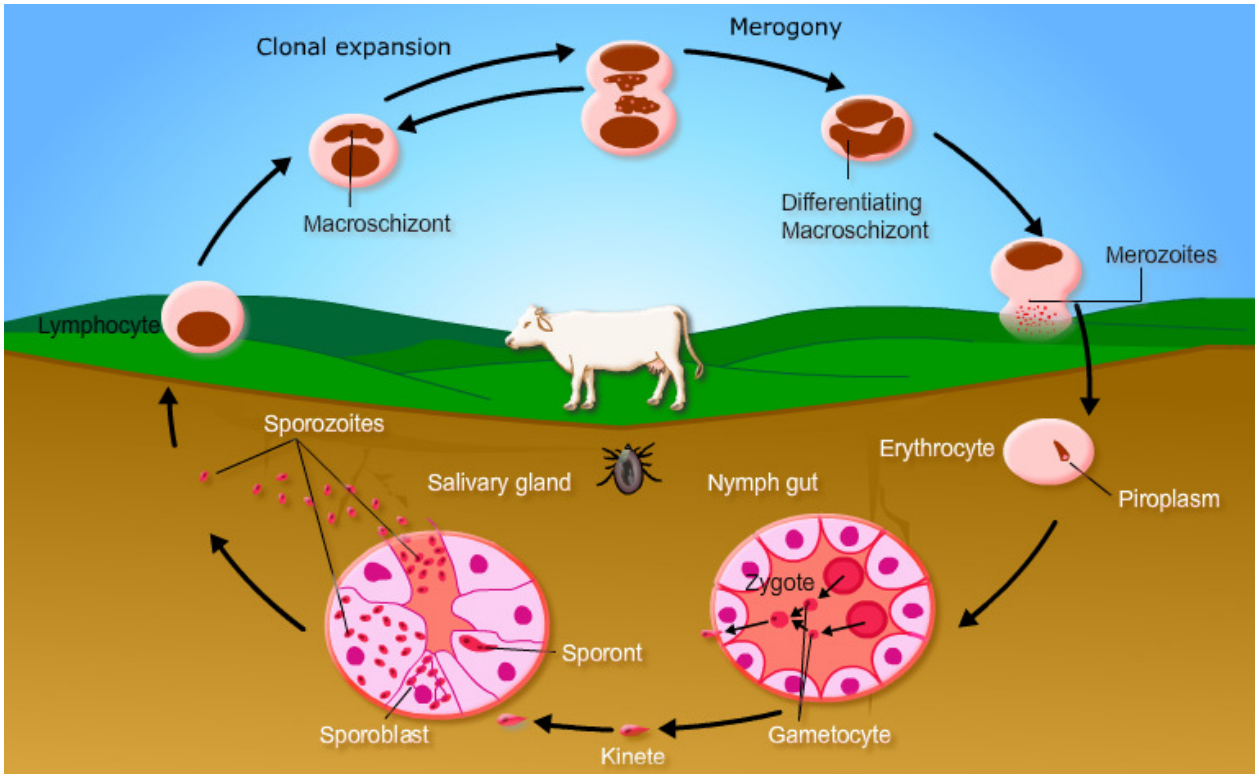
Season

- In India, mostly animals suffer during summer and rainy seasons (May - Oct) which is attributable to high incidence of tick vectors.; In tropical areas, ticks are active through out the year.
- Incidence due to stress of hot and humid weather.

Mixed infection

- Mixed infection with *Anaplasma/ Babesia spp* is common.

Lifecycle of *Theileria annulata*



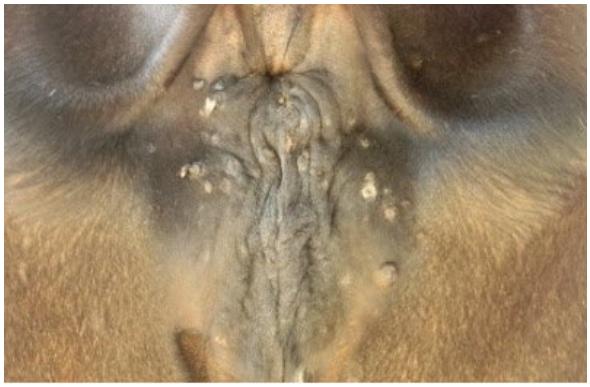
PATHOGENESIS AND LESIONS

Pathogenesis

- Mortality varies from 10- 90%.
- Incubation period: 9 to 25 days.
    - *Per acute form*

        - Common one lasting for 3 - 4 days or upto 20 days.
        - First sign is fever(40.5 –41.5°C), continuous or intermittent persisting for 3- 20 days.
        - The other signs after few days appear including inappetence, cessation of rumination, rapid heart beat, weakness, reduced milk production, enlargement of superficial lymph nodes and odema of the eyelids
        - Marked anaemia, haemoglobinuria, bilirubinuria and bilirubinaemia, icterus of conjunctiva with petechial haemorrhage, jaundice, high colored urine, prostration and death.
        - Laboured breathing, serous nasal discharge, coughing, restlessness and starry coat.
        - Diarrhoea with blood and mucus in faeces, greatly emaciated (RBCs < 1 million/ cmm); Death after 15 days.
    - *Sub acute form*
        - Fever is irregularly intermittent for 15 days, then the animal recovers.
    - *Chronic form*
        - Intermittent fever, in-appetence, marked emaciation , some degree of anaemia, icterus for 4 weeks or longer, In some cases, sudden death may occur within 1-2 days.
    - *Mild form*
        - Little is seen, but mild fever, in appetence, listlessness, slight digestive disturbances, lachrymation for few days and moderate anaemia.
- Cerebral theileriosis
    - Nervous signs predominate and brain impression smears reveal Koch's Blue bodies.
- Cutaneous form
    - Some cases reveal urticarial type of skin lesions and haemoglobinuria.

Cutaneous form of theileriosis



Cutaneous form of theileriosis

Lesions

- Emaciated body of the carcass, pale mucous membrane, enlargement of the superficial lymph nodes, spleen and liver; Distension of the gall bladder with thick bile; Congestion and petechial haemorrhages on the kidney and lungs; In few cases, necrotic infarcts of brain.
- Punched necrotic ulcers in the abomasum are pathognomonic; In young calves, abomasum is full of undigested clots of milk.
- Haematological changes
    - Fall of haemoglobin (< 2 gm/ml) , reduced PCV, reduced RBC count (1.5-2 million/ Cmm), leukocytosis followed by leukopenia.

DIAGNOSIS AND TREATMENT

Diagnosis

- Based on clinical signs.
- Demonstration of erythrocytic forms (piroplasms) in pheripheral blood smears
- Demonstration of Koch's blue bodies (schizonts) in lymphocytes from lymph nodes.
- For latent cases, serological tests can be employed CFT, Capillary tube agglutination test, IFA.
    - Others: IHA, AGID, Dot-ELISA, LMI test, Conglutination Complement Absorption test (CCA), Gel agglutination test, immunoelctrophoresis
- By autopsy, examination for lesions
- Differential diagnosis: Anaplasmosis, Babesiosis, Trypanosomosis, Ehrlichiosis.

Treatment

- Different drugs respond differently with schizonts and erythrocytic stages of the parasite and none of the drugs are fully effective; but buparvaquone is fully effective in clinical cases.
- To combat the anaemic changes, haematinics as supportive therapy.
    - Buparvaquone (Butalex) - Naphthaquinone drug and drug of choice.
        - @ 2.5 mg / kg, deep I/M against different stages of T. annulata, it is effective.
        - 2^{nd} dose at 48 hours intervals.
    - Parvaquone - 10 mg/kg, deep I/M, effective against piroplasmic stage.
    - Berenil (Diminazene aceturate) – 2.5 to 5 mg /kg, deep I/ M; Doubtful against erythrocytic stage.
    - Oxytetracycline - 20 mg /kg for 7 days; I/M; effective against of schizogony.
    - Halofuginone (stenorol*) – Potent against schizont stage but toxic.
        - 1 to 2 mg/kg as 2 doses, as tablets.
    - Others

- Methohexate, 3% Tryphan blue solution, Sulpha drugs, Diethyl carbamazine citrate, Babesan, Chloramphenicol, Sulphadimidine+ chloroquine phosphate, Nivaquin and Famaquin.

PROPHYLAXIS AND CONTROL

- By Chemoprophylaxis + Immunoprophylaxis + Tick control.
  - Wide spread and strict application of acaricides
  - Use of genetically resistant breeds.
  - Immunoprophylaxis
    - Tissue culture vaccines (Rakshavac-T*)
      - Attenuated Schizonts of *T. annulata* developed by in – vitro cultivation of bovine lymphocytes infected with *T. annulata* schizonts. A dose of 2 x 10^6 infected cells suspended in serum free tissue culture remains viable at 4 ^{0}C for 1 week and used to vaccinate cattle.
      - Age: Cross–bred and exotic cattle of 2 months / above.
      - Dose: 3 ml , S/c, annually.
  - Infection and Treatment method
    - A potentially lethal known dose of stabilate of ground up infected ticks together with a long acting formulation of OTC or Buparvaquone is developed. It suppresses the development of the parasite and allow the effective CMI response with minimal disease response.
  - Subunit vaccines:
    - Antigen to be included are sporozoites and Schizonts that induce CD^4 + T cells and antigen stimulating specific response like cytotoxic cells.
  - Recombinant antigens
    - Immunization with recombinant sporozoites / merozoites surface antigen partially protect against sporozoite which are poorly immunogenic. For *T. annulata*, it induces the innate immune response.

EAST COAST FEVER

Life cycle: Similar to *T. parva.*

Pathogenesis

- Serious disease in cattle; High rate of mortality.

- *Acute form*
  - More common; fever (41. 7 ^{0}C) , enlargement of superficial lymph nodes, swelling of eyelids and ears, diarrhoea with blood and mucus, marked emaciation.
  - Edema of lungs causes death immediately.

- *Sub acute*
    - Frequent in calves; less pronounced symptoms.
- *Turning Sickness*
    - In east and Central Africa. signs are circling movements and abduction of limbs; immunity is solid and specific.

Diagnosis

- Demonstration of Schizonts in lymph node biopsy.
- Demonstration of erythrocytic forms are difficult.
- Differentiation from the *Theileria spp* is difficult.
- Serological tests: IHA, IFA, Capillary agglutination test

Treatment

- Oxytetracycline and Chlortetracycline, Buparvaquone.

THEILERIOSIS IN SHEEP AND GOAT

- Morphology: Oval and rod shaped.
- Life cycle: Similar to cattle.
- Pathogenesis
    - Similar to Bovine Tropical theileriosis
    - Acute: High fever, listlessness, nasal discharge, anaemia, jaundice, atony of rumen, weakness, transitory haemoglobinuria, petechial haemorrhage on abomasal mucosa, edematous lungs, enlargement of lymph nodes,
    - No cross immunity between T. hirci and T. ovis.
- Diagnosis: Examination of stained blood, lymph nodes or spleen.

BABESIOSIS

INTRODUCTION

- *Family:* Babesidae
- First vector borne disease caused by *Babesia spp* in cattle, sheep, pigs, horses and dogs and is characterized by fever, intravascular haemolysis causing a syndrome of anaemia, haemoglobinuria, and haemoglobinaemia and are transmitted by ticks.
- *Morphology*
    - In RBC- seen singly as ovoid, round, elongate or amoeboid trophozoites ; as pyriform merozoites (pairs)/ cruziform (tetrads) merozoites; Characteristically they are pear

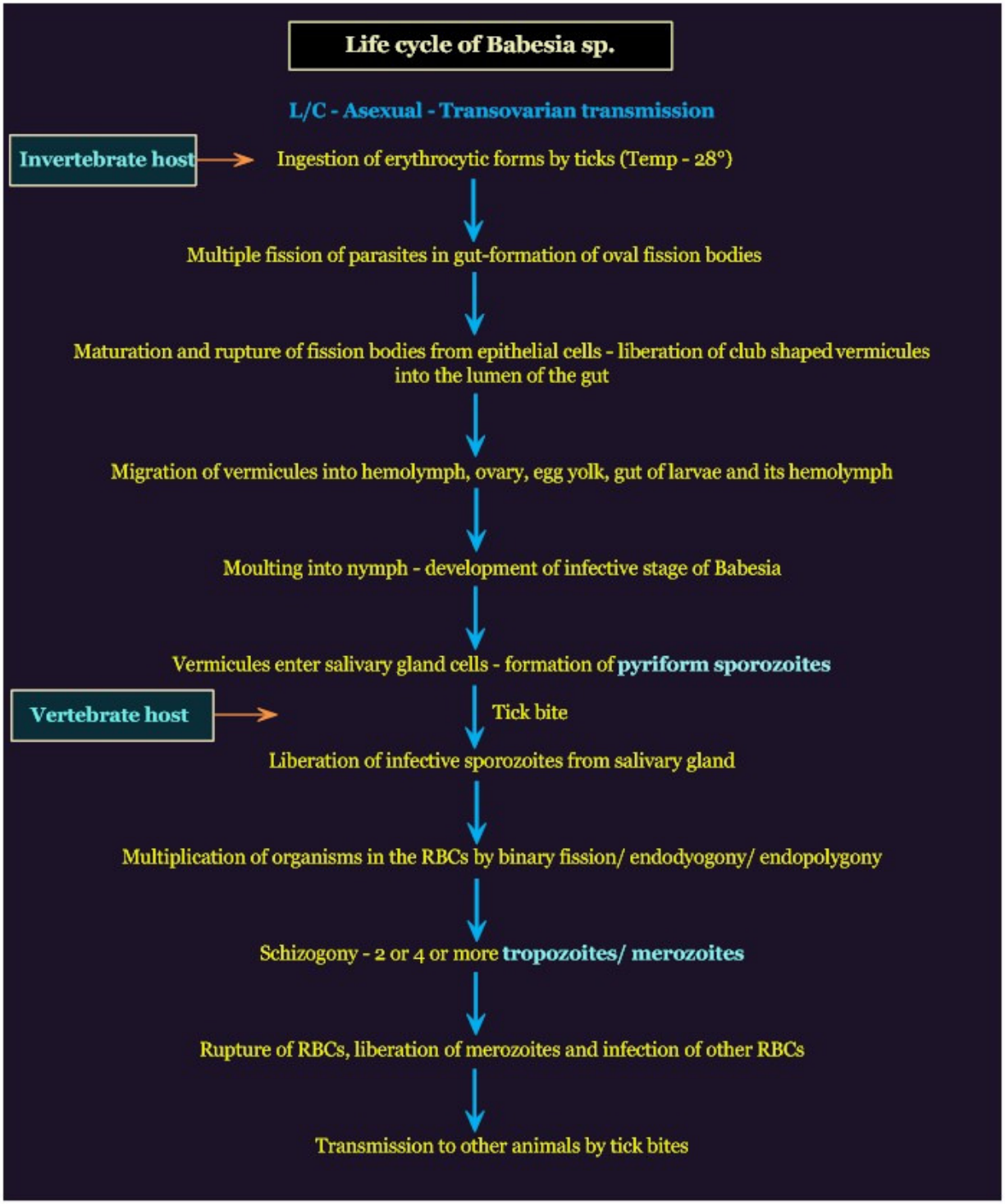
shaped; large forms lie with their narrow ends at an acute angle and small forms at obtuse angle.

BABESIA SP. IN LIVESTOCK

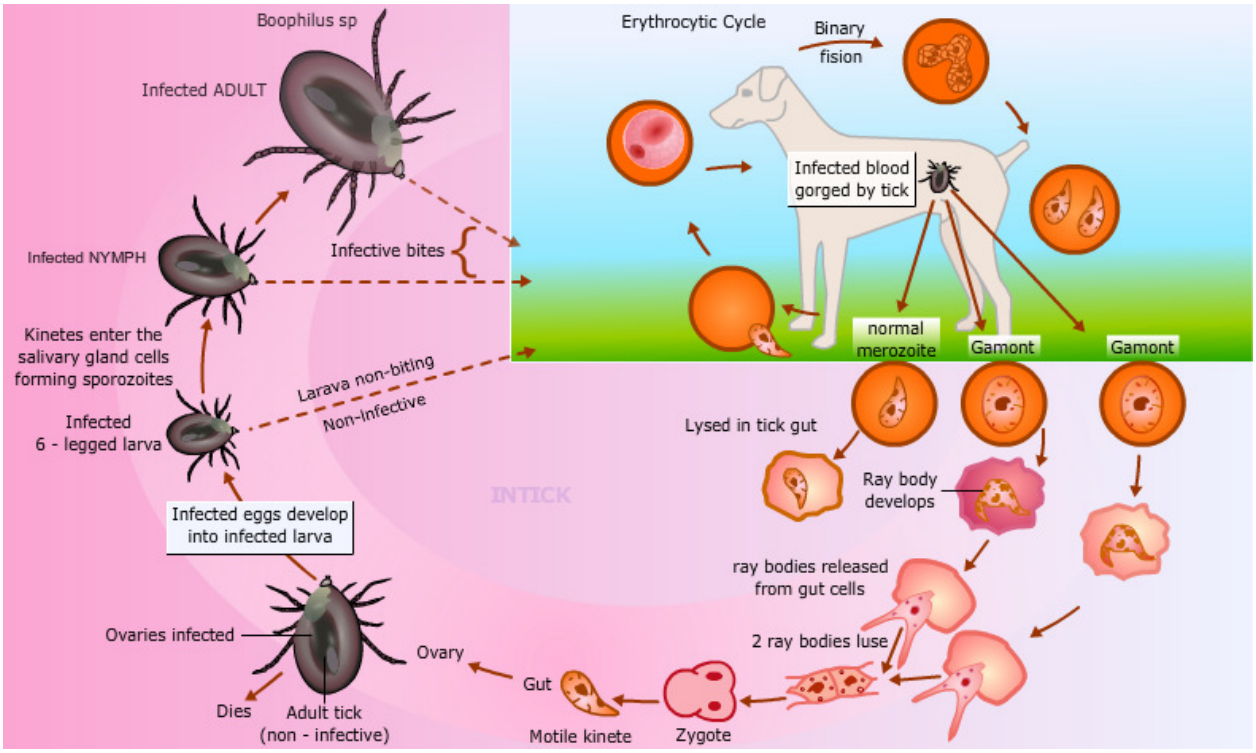
S.No.	Babesia spp	Vectors	Transmission	Disease
Cattle, Buffalo and Zebu				
1	*B. bigemina* (India)	*Boophilus annulatus* *B. microplus* *R. appendiculatus* *R. bursa* *Haemophysalis punctata*	Transovarian and Transtadial, also Intra uterine	Cattle Tick Fever Red Water Disease Bovine Piroplasmosis Texas Fever Triteza
2	*B. bovis* (India)	*B. microplus* *B. calcaratus* *R. bursa* *Ixodes ricinus* *I. persulcatus*	Transovarian and Transtadial, also Intra uterine	Bovine Babesiosis Piroplasmosis Red Water Disease
3	*B. major*	*Ixodes ricinus*	Transovarian and Transtadial	Less severe than B. bigemina
4	*B. divergens*	*B. calcaratus* *Haemophysalis punctata*	Transovarian	Less severe than B. bovis
Sheep and Goats				
1	*B. motasi*	*Dermacenter silvarian,* *Haemophysalis punctata* *R. bursa*	Transovarian and Transtadial	Pathogenic
2	*B. ovis*	*R. bursa*	Transovarian and Transtadial Transplacental	Less severe than B. motasi
Sheep				
1	*B. foliata* (India)	Not known	Not known	Not known
Goat				

1	*B. taylori* (India)	Not known	Not known	Benign type disease
Dogs				
1	*B. canis*	*R. sanguineus*	Transtadial	Biliary Fever Malignant jaundice Canine Piroplasmosis Canine babesiosis
2	B. gibsoni (India)	*R. sanguineus, Haemophysalis bispinosa*	Transtadial and Transovarian	Slightly Pathogenic
Cats				
1	*B. felis* (India)	Not known	Not known	Less pathogenic than dog
2	*B. cati* (India)	Not known	Not known	Less pathogenic than dog
Equines				
1	*B. equi* (India)	*Dermacenter spp* *R. sanguineus* *R. bursa* *Hyalomma anatolicum*	Transovarian and Transtadial, also Intra uterine	More pathogenic
2	*B. cabbali*	*Dermacenter spp* *R. sanguineus* *R. bursa* *Hyalomma anatolicum*	Transovarian and Transtadial, also Intra uterine	Less pathogenic
Pigs				
1	*B.trautamanni*	*Boophilus spp* *R. sanguineus* *Dermacenter* spp	Transovarian	Mild/ Fatal
2	*B. peroncittoi*	Not known	Not known	Mild/ Fatal

LIFE CYCLE OF BABESIA SP.



Lifecycle of Babesia canis



BABESIOSIS IN CATTLE

Disease

- Cattle tick fever
- Red water disease or Piroplasmosis or Texas Fever.

Morphology

- A large piroplasm, round, pyriform, oval or irregular form or characteristically pear shaped and lie in pairs forming an acute angle in RBCs.

Life cycle

- The spherical forms in the ovary of *B. microplus* occur with parasitophorous vacuole formed by host cells where they are transformed into folded kinetes.
- The sporozoites in tick salivay gland is pyriform.

Epidemiology

- *Virulence of parasite*

- *B. divergens* and *B. canis* – relatively pathogenic; *B.major* and *B.ovis* – mildly pathogenic producing transient anaemia.

- *Age of the host*
  - Inverse age resistance; Greatest infection rate in animals in the age 6-12 months age group and infection is uncommon in animals over 5 years of age.
  - *B. bigemina* : Under 1 year age are susceptible and *B. bovis* - Over 2 years of age are susceptible.
- *Breed*
  - *B. bigemina*- All races of cattle are equally susceptible; *B. bovis*- Zebu and Africander cattle are more resistant than British and European breeds and Santa Getrudis- intermediate position.
- *Transmission*
  - Transovarian transmission-one host tick & Transtadial transmission- two or three host tick. Transmission by several generations of ticks is possible.
  - Intra uterine transmission- B. bigemina and *B.ovis*.
  - Physical-By contaminated needles and surgical instruments. eg. *B. bigemina* and *B. equi*. also *B. bovis*.
- *Carrier status*
  - Infected adult animals like sheep act as carriers for over 2 years; if the are constantly infected , they act as carriers for life.
- *Reservoir status:* White tailed deer and African buffaloes.
- *Premunity*
  - Cattle are immuned to reinfection due to exposure to continuing low grade infection.
  - Eg: Premunity occurs for more than 1 year. Immunity is species specific. In endemic areas, calves acquire colostral immunity.
- *Risk factors*
  - Endemic areas-Outbreak is associated with stress due to parturition, tick borne fever/ starvation.
  - Break down of immunity occurs if there is superimposed infection with *A.marginale*/ FMDV in cattle and canine distemper in dog.
- *Season:* Greatest incidence occurs soon after the peak of tick population which varies with temperature, humidity and rainfall.
- *Enzootic instability:* It occurs when there is sudden increase in the tick population due to favourable climatic conditions, the incidence of the clinical cases may increase sharply.

PATHOGENESIS

- Highly pathogenic and death rate is higher in adults.
- Intra vascular haemolysis is the principal pathogenic effect due to the multiplication of *B. bigemina* and *B.bovis* in peripheral and visceral blood vessels respectively thus resulting in profound anemia, (hypochromic normocytic), jaundice and haemoglobinuria.
- Death is due to destruction of RBCs by auto-antibody mechanism, failure to recoup blood loss, cerebral anoxia and accumulation of toxic by-products.
- *B. bovis*
    - Production of vaso active substances, kinin and kallikrein results in vasodilatation accompanied by increased vascular permeability leading to circular stasis and shock.
    - Disseminated intravascular coagulation (DIC) due to break down products resulting in fatal pulmonary thrombosis in calves.
- Ischaemic changes in skeletal and cardiac muscles in survivors.
- *Haematology:* TEC - less than 2 million/ cmm and neutrophilia.
- *Biochemical changes:* Reduced calcium, phosphorous, total serum protein; Increase in blood glucose, total serum bilirubin and SGOT in liver damage
- *Necropsy findings:* Urinary bladder containing reddish, pink or coffee colored urine; Enlargement and congestion of lymph nodes.

CLINICAL FINDINGS AND DIAGNOSIS

Clinical findings

- Incubation period: 2-3 weeks; Sub clinical infection infection in young cattle.
- Fever, anorexia, brick red conjunctiva which becomes anaemic, depression, weakness, cessation of rumination, salivation, dryness of muzzle, lachrymation, dropped milk yield.
- Profound diarrhoea followed by constipation.
- Advanced stages: Severe anaemia, haemoglobinuria, jaundice, dark red to brown urine with a very stable froth and death after 24 hours.
- In survivors - course is 3 weeks. Abortion in pregnant animals.
- Cerebral form (*B.bigemina*): In coordination, posterior paralysis, mania, convulsions, coma, fever, death within 12-36 hours after clinical signs.
- Chronic cases: extend for several weeks with intermittent temperature.
- *B.divergens*: similar to above. Also spasm of anal sphincter muscles causing the passage of Pipe-stem liver.

Diagnosis

- Based on clinical signs.
- Detection of piroplasms in peripheral blood smears which is not always possible. Both thick and thin blood smears may be used for demonstration of organisms in RBCs.
- Presence of heavy infestation with ticks.
- FAT- Acridine orange fixed with blood smear appeared red with yellow nucleus; DNA probes.
- Necropsy: Splenomegaly, jaundice, haemoglobinuria, myocardial ecchymoses –highly suggestive.
- Immunodiagnosis in sub clinical infections
    - IHA- 80% effective in field infections
    - IFA, CFT, IHA and Capillary agglutination tests (specific and no cross reaction).
    - Others: Latex agglutination tests, Card agglutination test, ELISA, Microplate enzyme immuno assay. IFA differentiates antibodies due to vaccination and natural infection.

DIFFERENTIAL DIAGNOSIS

- Epierythrozoonoses -anemia
- Theileriosis-haemoglobinuria
- Leptospirosis- severe in young ones
- Anaplasmosis
- Trypanosomosis
- Bacillary haemoglobinuria
- Rabies
- Copper toxicity.

TREATMENT AND CONTROL

Treatment

- Aromatic diamidines:
    - Imidocarb dipropionate (Imizol)
    - @ 1 mg/kg, S/C; 2 mg/kg, Most effective against B. bigemina; therapeutic and prophylactic; completely eliminates the parasites with some residual activity.
    - Merit:
        - Non- infected cattle develops one month resistance;
        - To protect cattle during pregnancy where vaccination is contraindicated.
- Quinuronium derivatives (Acaprin, Babesan, piroplasmin. Piruvan)

- Effective against B. bigemina and other larger spp; 1 ml/ 50 kg but with a maximum dose of 6 ml, S/C.

- Other Aromatic diamidines
  - Diamprone -@ 10 mg. kg, deep I/M.
  - Phenamidine-@ 12 mg/kg, S/C in 40 % aqueous solution.
  - Pentamidine- Chemo immunizing agent against B. bigemina in cattle.
- Others
  - Tryphan blue solution (1-2 %) - 100 ml , I/V.
  - Acriflavin (5%)- 20 ml.
- Amicarbalide - effective in cattle and horses; @ 5-10 mg/kg, but I/M at 24 hrs interval.
- Primaquine - 0.5 mg/ kg; I/M.

Control

- Control of ticks- acaricides, repellants;dipping of animals at regular intervals
- Segregation and treatment of animals harbouring infection.
- Chemoprophylaxis- Imidocarb for 2 months which allows animals infected gradually.
- Killed vaccine- with incomplete Freund's adjuvant and irradiated Babesia spp.
- Chemoimmunization- vaccination with virulent organisms and simultaneous subsequent challenge with Babesia spp.
- Live attenuated vaccine- less virulent and attenuated babesia are suspended in a cell free plasma are used after passing through spleenectamized calves.
- Vaccine contains 10^3 B. bovis organisms / dose are the minimum infective dose.
- Live attenuated tissue culture vaccines are also effective.
- Vaccination with non-living antigens, antigenic proteins produced in blood stream or culture medium as a part of the growth process of Babesia spp.
- Sub unit vaccine from monoclonal technology
- Blood transfusion- Oldest; immunization by inoculation of infected blood followed by babesidal drugs but transmission of bovine leucosis and other diseases.
- Diminazene aceturate-@ 3-8 mg/ kg, but deep I/M; safe and effective to treat all spp.

BABESIOSIS IN HORSES

Life cycle: Similar to *B. bigemina*

- *B. equi*
  - Incubation Period: 10-21 days
  - Severe in young and new born foal

- Course-8-10 days
- Chronic cases survive for months and carries persists for 4years.
- Mixed infections with *B. cabbali* can occur
- Fever, reluctance to move, edema of fetlock, head and ventral abdomen, frequent colic, jaundice, anaemia.

- *B. caballi*
  - Nervousness
  - Walking in circle
  - Incoordination and paralysis of hind quarters
  - Haemoglobinuria - rare.

Treatment: Imidocarb-@ 4mg/ Kg on 4 occasions at 72 hrs intervals.

BABESIOSIS IN DOGS

- Disease: Biliary fever/ Malignant jaundice.
- Pathogenesis
  - *B. canis*
    - Incubation period: 10-20 days; fever, anaemia, icterus, inappetence, marked thirst, weakness, prostration and frequent death.
    - Peracute cases: Haemoglobinuria, bilirubinuria.
    - Acute cases: Acute respiratory failure, extensor spasms and death within 4-5 days.
    - Atypical signs : Catarrhal bronchitis, pneumonia, subcutaneous edema, ascites, purpura, red urine or blood clots in faeces.
    - CNS signs: Locomotor disturbances, paresis and epileptic fits.
    - Lesions: Petechial hemorrhages in various organs and mucous membrane; icterus; edema in pleural and peritoneal cavities.
  - *B.gibsoni*
    - Chronic type of disease with relapses of fever, progressive anaemia, haemoglobinuria, jaundice - less common, staggering gait and death.
- Treatment: Berenil

CHAPTER-35: ANAPLASMOSIS AND EHRLICHIOSIS

Learning objectives

To know in detail about

- Epidemiology, lifecycle, intermediate hosts, pathogenesis, clinical signs, prevention and control of Anaplasmosis and Ehrlichiosis.

ANAPLASMOSIS

INTRODUCTION

- *Order:* Rickettsiales
- *Genus: Anaplasma*
- It is an infectious haemoparasitic disease caused by *Anaplasma spp* in cattle, sheep and goat and characterised by fever, anaemia, jaundice and emaciation.
- It is usually subclinical in sheep and goats.
- *Distribution:* Worldwide in tropics and subtropics; also in temperate areas of USA.

HOST, LOCATION AND MORPHOLOGY

Host and Species

S.No.	Parasite	Host	Pathogenecity
1	*A. marginale*	Cattle, wild ruminants, sheep and goat (inapparent)	Pathogenic
2	*A. centrale*	Cattle, wild ruminants, sheep and goat (inapparent)	Mild anaplasmosis
3	*A. ovis*	Sheep and Goat	Non- pathogenic

Intermediate Host/ Vectors

- One host tick *Boophilus* spp- transtadial transmission and little evidence on transovarian transmission.
- Tabanids- Mechanical transmission.
- 20 tick species are involved.

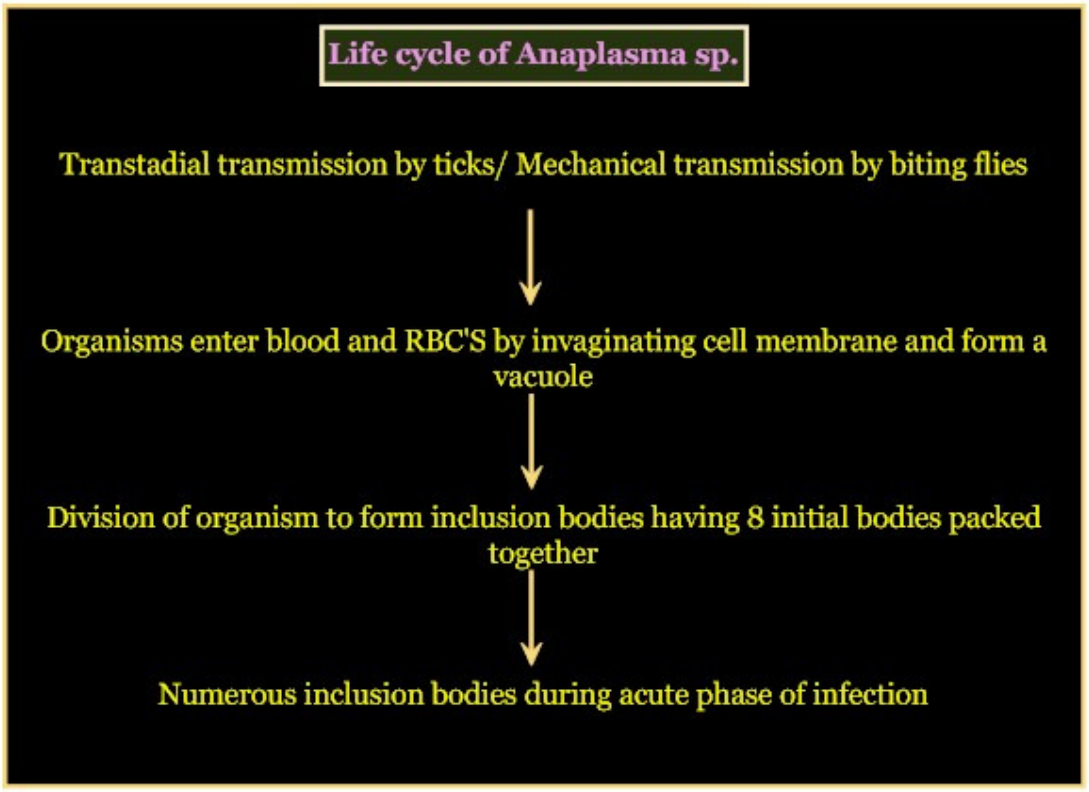
Location

- Red blood cells.

Morphology

- *A. marginale:* A small round, dark red inclusion bodies within RBCs on Giemsa staining.One organism in the RBC characteristically in the outer margin.
- *A. centrale:* Similar but are commonly found in the centre of RBCs.

LIFE CYCLE AND EPIDEMIOLOGY



Epidemiology

- *Age:* Over 3 years of age group are commonly affected; Young animals are relatively resistant; Mortality-80%.
- *Breed:* British breeds and Zebu cattle are equally susceptible but are not commonly affected due to their relative resistance to the ticks.
- *Transmission*
    - By developmental transtadial transmission by Ixodidae ticks- *Boophilus spp.*
    - Mechanical transmission by tabanid flies
    - Mechanical transmission by infected hypodermic needles, castration, dehorning, spaying, blood transfusion, vaccination, embryo transfer and blood sampling.
    - Congenital transmission: Less frequent.
- *Carrier status*

- Infected cattle are carriers for many years or for life.
- Sheep, goats, wild ruminants may act as carriers. Cattle in endemic areas are less susceptible due to previous exposure.

- *Immunity:* Acquired immunity in cattle due to previous infection.
- *Risk factors*
  - Low plane of nutrition- clinical disease is less severe; Thin hungry cattle – less susceptible than fat and well fed cattle
  - Cattle introduced in feed lots –highly susceptible.

PATHOGENESIS AND CLINICAL SIGNS

Pathogenesis

- Incubation period: 15 to 36 days; mortality-80% in imported cattle. In endemic areas, seasonal death rate - 10%.
- *Acute cases*
  - 10 – 90% RBCs are parasitized resulting in anaemia and death. Phagocytosis of a parasitized RBCs with the release of inflammatory reactants and development of fever.
  - Appearance of anti-erythrocytic antibodies in late acute stage exacerbates the anaemia.
- Fall in haematocrit level, RBC level and appearance of immature RBCs.

Clinical signs

- Very mild in cattle of less than 1 year.
  - *Per acute*
    - Fatal in cattle of less than 3 years of cattle and death within a day of onset of clinical signs. Sudden onset of high fever, anaemia, icterus, severe dyspnoea, hyper excitability and death within 24 hours; Abortion in pregnant cows.
    - Urine –normal colored; Milk yield-reduced.
  - *Sub acute*
    - In young animals and recovery is slow in more chronic cases
    - Sheep and Goats: Usually sub clinical, but may be severe in goats with the clinical signs similar to goats.

DIAGNOSIS AND TREATMENT

Diagnosis

- Clinical signs
- Detection of organisms in peripheral blood smear

- Presence of insect vectors
- History of occurence of outbreak
- Nucleic acid probes.
- Serological tests
    - Capillary tube agglutination test, CFT, IFA, Rapid card agglutination test, Dot- ELISA (highly sensitive and specific) and Antigen capture ELISA.

Differential diagnosis

- Babesiosis- More acute and haemoglobinuria.

Treatment

- *Tetracyclines*
    - OTC and CTC @ 6-10 mg/kg, single dose or daily for 10 –16 days; effective in elimination of carriers.
    - Long acting TC-20 mg/ kg, I/V, every week , 2-4 injections.
- *Imidocarb:* 3 mg/ kg, effective for classic cases, No sterility of carriers.
- *Amicarbalide:* 20 mg/ kg, S/c, No sterility of carriers.

CONTROL

- Control is not practicable because of
    - Wide spread insects
    - Long period of infectivity of carrier animals; inability to detect infection and carrier status in wild animals.
- Control of ticks by acaricides.
- Control of vector flies.
- Prevention of artificial transmission by instruments/ surgical operations by disinfection.
- Elimination of carriers by screening the animals entering into areas by capillary agglutination tests/ CFT.
- Elimination of carriers by a series of 4 injections at 3 days intervals of long acting TC.
- Immunization
    - *Killed A. marginale in an adjuvant vehicle as a vaccine (Anaplaz) in U.S.A*
        - Dose- 2 vaccinations at 6 weeks apart.
        - Immunity- atleast for 5 months.
        - Demerit- Not completely protective and development of carriers and isoerythrolysis in neonates.

- *Live virulent vaccine*
  - Limited to relatively resistant age group below 1 year of age. Demerit- development of carrier status.
- *Live attenuated vaccine*
  - Use of attenuated virulent strain, irradiated organisms and a virulent strain adapted for growth in sheep/ deer and the vaccine is frozen in liquid nitrogen.
- *Modified live vaccines:* Effective & limited to mature cattle; induces resistance to clinical diseases.
- *Infection and Treatment*
  - Administration of susceptible stock with small quantities of small blood containing mildly pathogenic *A.centrale* or a relatively avirulent *A. marginale* with or without subsequent administration of drug (OTC) is practiced in several countries.

EHRLICHIOSIS

MORPHOLOGY

- Ehrlichiosis is the rickettsial disease caused by the genus of *Ehrlichia* found in blood WBCs as intracytoplasmic inclusions and is charecterized by a short febrile illness associated with leuopenia and transmitted by ticks.

Morphology

- Small pleomorphic, coccoid to ellipsoidal intracytoplasmic forms in circulatory WBCs of various mammals.
- Organisms occur singly or in compact colonies as a morula which is characteristic of the organism.

EHRLICHIA IN LIVESTOCK

S.No.	Species	Host	Location	Vector	Disease
1	*E. bovis* (India)	Cattle	Mononuclear cells	*Hyalomma spp, Rhipicephalus sp*	Bovine Ehrlichiosis (Nofel/ Nobi)
2	*E. canis*	Dog	Monocytes	*R. appendiculatus, R. sanguneus*	Tropical canine Pancytopenia, Canine Rickettsiosis, Nairobi bleeding disease, Lohore disease, Lahopi caninme fever
3	*E. ovina*	Sheep	Mononuclear cells	*R. bursa*	Mild disease

4	*E. equi*	Horse	Neutrophils and Eosinophils	Not known	Rarely fatal
5	*E. risticii*	Horse	Monocytes and Macrophages	Dermacentor sp?	Potomoc Horse Fever, Equine Ehrlichial Colitis

EPIDEMIOLOGY AND PATHOGENESIS

Epidemiology

- *Transmission:* Non contagious and is transmitted by bite of ticks. Experimentally by parenteral inoculation of organisms in horses.
- *Age:* Very young lambs; Adult sheep and cattle newly entered in endemic areas.
- *Complications:* Disease predisposes sheep- lambs to Louping ill, tick pyaemia (Enzootic staphylococcosis) and pasteurellosis.
- *Carrier status:* Puppies for long time and stress precipitate the disease; Recovered animals.

Pathogenesis

- Organisms invade in monocytes, macrophages and epithelial cells and they multiply and the entire cytoplasm is filled with organisms causing destruction of leucocytes an thrombocytes.
- This results in leukopenia, thrombocytopenia, anaemia, bone marrow depression.
- Horse - Invasion of intestinal epithelial cells by organisms causing colitis and typhilitis.

CLINICAL SIGNS AND LESIONS

Clinical signs

- *Cattle*
    - Asymptomatic
    - Incubation period: 1-6 weeks
    - Acute cases: Fever, tremor, incoordination, convulsion, anorexia, pale visible mucous membrane, lymph node enlargement, salivation and lacrimation, diarrhoea, frequent micturition, ocular and nasal disharge, nervous signs, death during convulsive phase of illness; abortion or temporary sterility in males may result.
- *Horses*
    - Inapparent disease
    - In severe cases, high fever, anorexia, congested mucous membrane, severe diarrhoea of projectile in nature, colic, signs of dehydration, shock and signs of laminitis.

- Sub acute- Mild colic and subcutaneous oedema.

- *Sheep*
  - Long incubation period
  - High fever persisting for long time, depression, anorexia, profound anaemia, ataxia, paraphlegia, sternal recumbency followed by death.
  - Abortion may occur in sheep newly introduced in endemic areas.
- *Dogs*
  - Undulating fever, lymph node enlargement, anorexia, depression, pale mucous membrane, haemorrhage and clotting abnormalities result in haematemesis, epistaxis, haematuria and haemorrhagic diarrhoea; Polyarthritis, polydypsia, polyuria and uraemia occur.
  - Eye- haemorrhagic ophthalmitis is common.
  - Death is due to secondary infection resulting from lekopenia, mucosal/ serosal haemorrhages due to platelet deficiency (ecchymoses); also due to anaemia.

Lesions

- Subcutaneous edema, enlarged spleen and lymph node, haemorrhage in liver and kidney, alimentary tract and many organs including joints and eyes.

DIAGNOSIS, TREATMENT AND CONTROL

Diagnosis

- History of ticks
- Clinical signs
- Detection of organisms in cytoplasm of monocytes as morulae (intracytoplasmic inclusion bodies) from peripheral blood smears stained with Giemsa stain.
- Histopathological examination of tissues by staining for organisms.
- Serological tests: IFA and ELISA.

Differential diagnosis

- Cattle- Babesiosis, Theileriosis and Anaplasmosis.
- Dog- Canine ehrlichiosis from Canine Distember and auto immune haemolytic anaemia.

Treatment

- Tetracyclines-@ 10 mg/kg, I/V; effective (not in anaemic cases).
- Imidiocarb dipropionate.

Control

- Animals from endemic areas should not be introduced into a new herd , as they are carriers.
- Control of vector population by dipping of lambs.
- Inactivated whole cell adjuvanted vaccine is recommended.

CHAPTER-36: COCCIDIOSIS AND CANINE EPERYTHROZOON INFECTIONS

Learning objectives

To know in detail about

- Morphology, epidemiology, lifecycle, pathogenesis, clinical signs, prevention and control of coccidiosis in livestock and poultry.

COCCIDIOSIS

INTRODUCTION

Sub phylum	Sporozoa
Class	Coccidia
Family	Eimeridae Sarcosystidae

Eimeridae

- Organisms commonly called coccidia are typically intracellular parasites of epithelial cells of intestine of vertebrates with few exceptions.
- All forms have a single host in which they undergo asexual (Schizogony or merogony) and sexual (Gametogony) cycle of reproduction.
- Sporulation of fertilized zygote usually takes place outside the host.
- Genera of importance
    - Eimeria
    - Isospora
    - Cryptosporidia
- Coccidiosis is caused by Eimeria spp and Isospora spp.

S. No	Species	Location	Pathogenicity
Chicken			
1	*E. tenella*	Caeca	Most pathogenic - Caecal Coccidiosis
2	*E. brunetti*	Lower small intestine, Caeca, rectum, Cloaca	Most pathogenic- Rectal Coccidiosis
3	*E. necatrix*	Jejunum, mid-gut and other parts of large intestine	Most pathogenic- Intestinal coccidiosis
4	*E. maxima*	Mid-gut	Medium/ highly pathogenic intestinal Coccidiosis
5	*E. acervulina*	Duodenum	Mildly pathogenic
6	*E. mivati*	Duodenum and Rectum	More pathogenic
7	*E. mitis*	Duodenum and jejunum	Mild pathogenic
8	*E. praecox*	Duodenum	Mild/ Non- pathogenic
9	*E. hagoni* (uncommon)	Duodenum	Slightly pathogenic
Turkey			
1	*E. adenoides*	small intestine and large intestine	Most pathogenic
2	*E. gallopavonis*	Lower small intestine, Caecum, Rectum	Moderately pathogenic/ Non-pathogenic
3	*E. meleagridis*	Small intestine, Caecum and Rectum	Mildly pathogenic
4	*E. meleagrimitis*	Small intestine	Markedly pathogenic
5	*E. dispersa*	Small intestine	Mildly pathogenic
Geese			
1	*E. truncata*	Kidney	Highly pathogenic
2	*E. anseris*	Small intestine	Low pathogenic
3	*E. nocens*	Posterior small intestine	Moderately pathogenic

Cattle			
1	*E. zuernii*	Small intestine and large intestine	Highly pathogenic
2	*E. bovis*	Ileum, caecum and colon	Common pathogenic - clinical coccidiosis
3	*E. alabamensis*	Small intestine, caecum and upper colon	Non- pathogenic
Sheep			
1	*E. ovinoidalis*	Caecum and colon	Highly pathogenic
2	*E. crandallis*	Caecum and colon	Mildly pathogenic
Goat			
1	*E. arloingi*	Caecum and colon	pathogenic
Rabbit			
1	*E. steidae*	Liver	Pathogenic
2	*E. intestinalis*	Intestine	Pathogenic
3	*E.flavescens*	Intestine	pathogenic
Horse			
1	*E. leukarti*	Small intestine	Pathogenic in heavy infections
Pigs			
1	*I. suis*	Jejunum and small intestine	Pathogenic, not frequent
2	*E. debliecki*	Jejunum and small intestine	Pathogenic, not frequent
Dog			
1	*I. canis*	Small intestine and large intestine	Mildly Pathogenic
Cat			
1	*I. rivolta*	Small intestine and large intestine	Pathogenic
2	*I. felis*	Small intestine and caecum	Mildly Pathogenic

Morphology and Life cycle

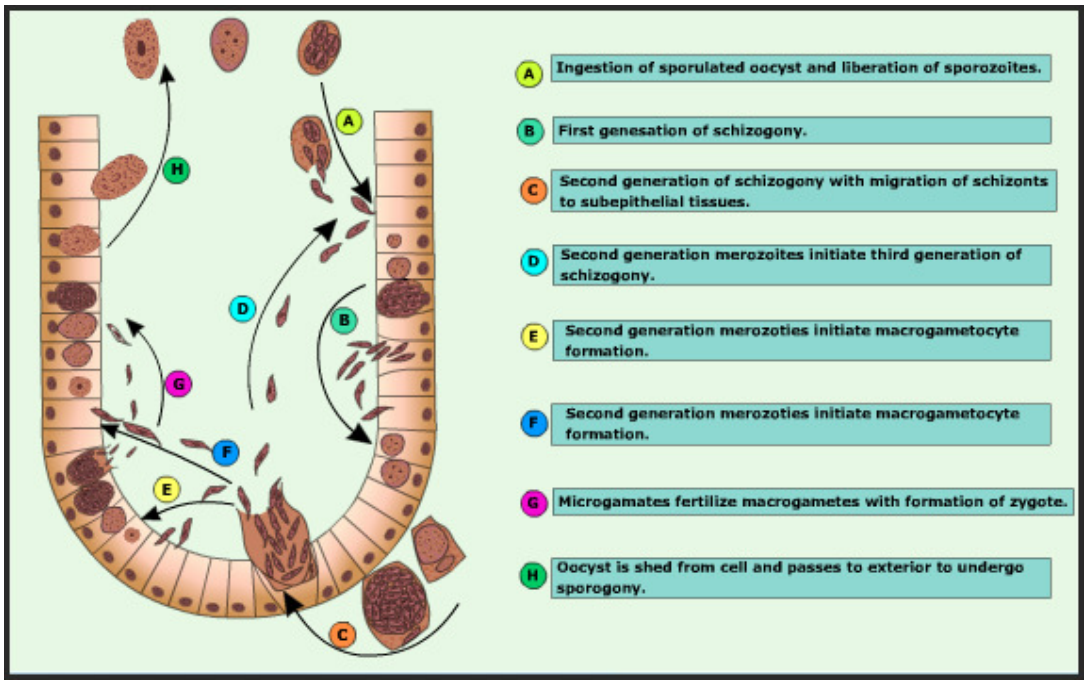
Morphology

- *Oocysts*
  - Most common shapes are spherical, ovoid/ ellipsoidal
  - It have refractile shell and some species with a small pore at one end called micropyle covered with a prominent polarcap.
  - An outer wall enclosing 4 sporocysts each containing 2 sporozoites in the sporulated oocyst which is the infective stage.
- *Tissue stage*
  - Mature schizonts identified by their location histologially, size and number of merozoites they contain which are arranged in a crescent shaped organism like a sliced onion.
  - In mature microgametocyte, the microgamates are arranged around the periphery in the cell.
  - Macrogametocyte has a large central nucleus and with small granules arranged around the periphery of the cell.

Life cycle

- Prepatent period: In poultry- 5 days.
- Three phases
  - Sporulation
  - Infection and schizogony
  - Gametogony and Oocyst formation

Lifecycle of Eimeria tenella



EPIDEMIOLOGY

Agent

- *Production of oocysts*
  - Immediately after prepatent period, oocysts are passed in very large number in the faeces, eg: *E. tenella*.
  - Number of oocysts voided falls rapidly and cannot be found easily unless reinfection.
  - Occasionally small number of oocysts are found in the faeces several months after a single infection.
  - Reproduction potential of a species of coccidium after ingestion by a susceptible host influences the number of oocysts. Eg. high biotic potential of *E. tenella*.
  - Number of viable sporulated oocysts ingested by a host influences the oocyst production.
  - Light infection produces relatively more number of oocysts.
  - Increased resistance of host influences depressed oocysts production.
  - Caecal plugs in caecal coccidiosis retard the passage of oocysts in the faeces.
- *Causation of disease*
  - It depends on,
    - Virulence of coccidium, eg: *E. tenella and E. necatrix*

- Number of oocysts ingested.
- Oocysts introduced in empty crop causes a more severe infection.
- Feeding calcium carbonate in excess of 3% in feed.
- Age of the host.

Host

- *Age*
  - 1 to 2 weeks- less susceptible
  - 4 to 6 – most susceptible
  - More than 6 weeks – resistant
  - Susceptibility increases with age in *E.tenella.*
- *Breed*
  - White Leghorn, Rhode Island and Newhampsire are resistant; Barred Plymouth Rock, Gersey White Giant and Light Sussex are susceptible; Breeder and Layer pullets are at greatest risk.
- *Transmission*
  - Depends on the type of housing and management and by ingestion of oocysts.
- *Risk factors*
  - In intensive management system, incidence is more common.
  - Reduced incidence in birds kept on wire floors and solid floor houses.
  - Dirt condition, overcrowding of stock results in greater accumulation of oocysts and spread of infection.
  - In broiler production, overcrowding with high temperature.
- *Carriers*
  - Mechanical carriers- sparkling beetles, migratory birds and birds recovered from infection.
- *Immunity*
  - Previous exposure results in species - specific resistance. Some species induces a solid immunity, other species need several infections and is self – limiting in nature. Both CMI and humoral immunity are elicited.
  - Immunity is due to 2^{nd} generation schizogony;
  - Trickle infection: Continuous level of ingestion of oocysts induces very strong immunity but cause damage the intestine.
  - Severity of disease increases with concurrent infections; Marek's disease increases the severity of coccidiosis.

Environment

- *Favourable conditions*
    - Optimum temperature and RH: Temperature: 25- 32◦C; RH – 90%.
    - Survival of oocysts in the soil is about a year and during winter.
- *Unfavorable conditions*
    - Both unsporulated oocysts and sporulated oocysts are extremely sensitive to desiccation (greater than 56◦C is lethal) Direct sunlight is lethal to the parasite.
    - Below 10◦C, cool and dry conditions, freezing kill the oocysts.
    - Bacterial or fungal growth kills / lethal to parasite.
    - Chemical disinfectant: 10 % ammonia or methyl bromide kills the oocysts.

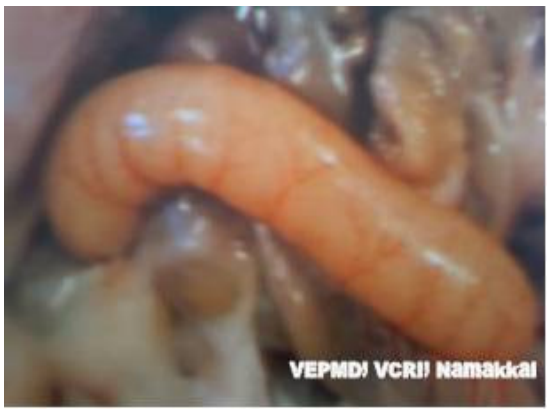
PATHOGENESIS AND CLINICAL SIGNS

Caecal Coccidiosis

- *Pathogenesis*
    - *E. tenella* is primarily responsible for caecal coccidiosis but gametogenous stage of *E. necatrix* and occasionally some stage of *E. brunetti* can cause; Morbidity-100%; Mortality- 80 %.
    - Principally in chicks of 3-7 weeks of age after 72 hours of infection.
    - Mortality is produced by a dose of $2x10^5$ oocysts produces mortality in 1-2 weeks age chicks.
    - Severity of the disease varies from indistinct infection to acute / highly fatal depending upon the no. of oocysts and virulence; Mortality- upto 100%
    - Second generation schizonts migrate to the lamina propria and sub mucosa and rupture of schizonts leads to massive haemorrhage, detachment of mucosal surface, diarrhoea and death within 4-6 days.
- *Clinical signs*
    - Clinical form: Occurs due to the ingestion of large number of oocysts over a short period and is characterized by the presence of soft faeces with blood, dullness, listlessness, drooping feathers, anaemias, paralysis and death.
    - Sub clinical form: Poor weight gain and poor conversion rate.
    - Lesions: Presence of dilated caeca with mixture of clotted and unclotted blood, caseous caecal contents adherent to mucosa, caseous plugs detached from mucosa and shed in the faeces; White spots and petechiae in caeca.

Intestinal Coccidiosis

- *Pathogenesis*
    - Both acute and chronic coccidia can occur by *E. necatrix* but sub clinical form is more common. Death can occur within 5-7 days.
- *Clinical signs*
    - Similar to caecal coccidiosis.Chronic watery diarrhoea with blood (mucous droppings in *E. maxima* infection); stunted growth, reduced egg production, listlessness, anorexia, soiling of vent and feathers; recovered birds are unthrifty and emaciated for a long time.
    - Lesions in the midgut
        - Second generation schizonts accumulation in deep mucosa as minute grayish white spots.
        - Balooning of intestine filled with clots of blood/ fresh blood and severe haemorrhage at 5-6 days after infection.
        - Intestine greatly thickened, dull red with fragile lining and friable with gangrene.
        - *E.maxima* - Salmon pink exudates and haemorrhage.



Rectal Coccidiosis

- Occur by *E.brunetti* in small intestine to cloaca.
- White fluid droppings mixed with blood and mucosa.
- Coagulative necrosis and slight haemorrhage.
- Loss of body weight and reduced feed intake and in appetence.

COCCIDIOSIS IN TURKEYS AND GEESE

Pathogenesis

Turkeys

- *E. adenoids* is the primary cause of turkey coccidiosis.
- *Age:* Young birds are highly susceptible especially at 4-5 weeks.
- *Risk Factors*
    - Intensive breeding (result in heavy loss)
    - Overcrowding.
    - Poor sanitation- dampness of house.
- Sporulation period- 1 day; P.P- 5 days.
- Pathogenicity by gametogony and schizogony.
- *Clinical cases:* Intestines with catarrhal inflammation with or without haemorrhage; Swollen and oedematous intestine with petechial haemmorhage on the mucosa of caeca; Intestinal contents are pasty and orange coloured, then become compact, caseous, mucoid with blood; tucked head under wings in affected cases.

Geese

- Haemorrhagic enteritis by *E. anseris.*
- *E. truncata*- Mortality- 100 %, marked emaciation, muscular in coordination, enlargement of kidney with light colour, numerous white nodules, streaks and lines and destruction of kidney tubular cells; acute nephritis may be seen.

Diagnosis

- *Correct and early diagnosis depends on 2 factors*
    - History of the flock
    - Finding various types of lesions and their locations by P.M. examination.
- *Others*
    - Faecal examination for the presence of oocysts is of little significance in severe out breaks and clinical cases, as the mortality starts before the discharge of oocysts.
    - Examination of gut scrapings for the presence of schizonts and unsporulated oocysts.
    - Study of sporulated oocysts by allowing sporulation of oocysts after mixing faeces with several volumes of 2.5 % Potassium dichromate solution for 1 day to 2 weeks.

TREATMENT

- Aim of the treatment is to allow sufficient schizogony develop in unaffected birds to stimulate their resistance.
- Eg: Sulphonamides have the great effect on II stage schizont without inhibiting I stage schizogony and increases the resistance.
- Fully recommended dose should be given and withdrawn gradually by reducing half of the dose, otherwise it is lethal.

S.No.	Drug	Dose	Coccidia	Stage of coccidia
Coccidiostats				
1	Amprolium	125 ppm in feed / water for 6 days	*E. tenella, E. necatrix* and also *E. maxima*	I and II Schizonts
2	Amprolium + Ethopabate	125 ppm + 8 ppm in feed/ water for 6 days	Extended spectrum	I and II Schizonts
3	Sulphaquinoxaline	0.5 % in feed, 0.043 % in drinking water for 2 days with 3-5 days interval.	*E. tenella, E. necatrix* and *E. acervulina*	II Schizonts
4	Sulphaquinoxaline + Pyrimethamine	Synergistic; 14 ppm + 45ppm in feed/ water	*E. tenella, E. necatrix* and *E. acervulina*	I and II Schizonts
5	Nitrofurazone + Furazolidone	55 mg/kg + 55 mg/ kg	*E. tenella* and *E. necatrix*	II Schizonts
6	Ionophore compounds			
	a) Monensin	0.121 % in feed	Superior to amprolium	
	b) Lasolacid	0.005 - 0.0075% in feed	High degree of activity	
	c) Salinomycin	0.01% in feed	Significant activity	
Coccidiocidals				
1	Robenidine	0.0066 % in feed	-	I Schizonts

CONTROL

- Primary prophylaxis in vaccination
    - *Coccivac* - vaccine with live attenuated oocysts of *E. tenella*, *E. maxima, E. acervulina*, *E.mivati* and chicken isolates
    - *Route* - Eye spray/cabinet spray/ in feed
    - *Age* - day old healthy chicken
    - *Precaution* - vaccine sensitive to anticoccidials
    - *Other vaccines are Livacox* - Q (attenuated quadrivalent vaccine), Immunocox
    - Coccivac-D (8 species) for breeders and layers
- Initial infection of 100 -700 oocysts/ each species followed by repeated daily doses of 1-5 oocysts for 20 days produces immunity by trickle infection which is reinforced by reinfection of oocysts in the litter.
- Selective breeding of domestic fowl is effective control method to increase the genetic resistance of the host.
- Nutritional supplement/ antagonism- Vitamin- A for recovery of the host; Vitamin-k to reduce the mortality due to haemorrhage and a high protein diet.
- Isolation of sick birds.
- Reduced drug rate to older birds allows limited exposure to developing coccidia so that it leads to acquired immunity.
- Recommendation of Switch programme where drugs are switched between batches of broilers and Shuttle programme where drugs are switched within life-span of each batch.
- Good ventilation to reduce humidity in the house to keep the litter dry.
- Heaping the litter for 24 hours to reach a temperature of 50◦C and forking for destruction of oocysts.
- Avoidance of contamination of feeders and waterers with droppings.
- Continuation of anticoccidials in water for 5 days.
- Burning the infected materials and sprinkling the quick lime @2-3 kg / 10 sq.feet over the litter.
- Avoidance of overcrowding in poultry houses.
- Isolation of young chicks from adults.
- Keep the waterers and feeders at higher level.
- Feeding chicks with coccidiostat in chick mash.
- Thorough disinfection and fumigation (Ammonia + Potassium permanganate) in the premises before the entry of newly hatched chicks.

COCCIDIOSIS IN CATTLE

Morphology

- Oval oocysts
- Presence of micropyle and absence of micropyle cap and residual cysts
- Sporocysts are elongate and ovoid with banana shaped sporozoites.

Life cycle

- P.P.P- 15 to 21 days
- Schizonts are seen in the central lacteals of the intestinal villi.

Epidemiology

- *Agent*
    - Delay in the lifecycle may occur due to the arrestment of schizogony stage and resumption occurs several months later with subsequent shedding of oocysts; Pathogenesis is by gamont stage of oocysts.
- *Age of the host*
    - Primarily in young calves of 21 days old to 6 months age; adults are also affected.
- *Risk factors*
    - Massive intake of oocysts
    - Overcrowding in unhygienic yards.
    - Pasture where congregation of livestock around the water holes.
    - Lack of sanitation.
    - In calves, which are recently turned out to permanent calf-paddocks.

PATHOGENESIS AND CLINICAL SIGNS

- *E. bovis*
    - Diarrhoea, dysentery, tenesmus, increased body temperature and death.
    - Lesion -Thickened, edematous, congested mucosae with haemorrhage of caecum, colon and terminal ileum with large amounts of blood.
- *E. zuernii*
    - Most pathogenic; causes catarrhal enteritis.
    - Lesions: caecum and colon are filled with semi fluid haemorrhage material or fresh blood with mucus.

- Clinical signs: Watery faeces with unpleasant odour, soiling of posterior part of animals.
- Severe cases: animals succumb and die after 7 days of clinical signs.
- Chronic cases: Bloody/ bloodless pasty mucus faeces, anorexia, convulsions, tremor, weakness, emaciation, death due to pneumonia (secondary bacterial infection).

Winter Coccidiosis

- By *E. zuernii* in cattle during cold/ stormy weather during winter where concentration of sporulated oocysts increase.

Mixed infections

- Abdominal pain, foul smelling diarrhoea, with blood or with soiled strings, drooping ears, rough coat, soiled hind quarters, tenesmus and anaemia.
- Inability to raise on legs, partial paralysis of anal sphincter; recovery is rare at this stage and death occurs in young calves.
- Secondary pneumonia, emaciation in severe cases.

Neurological coccidiosis

- Caused by *Eimeria sp.*
- Mortality-70%;
- Clinical signs are encephalopathy, diarrhoea, tenesmus, haematochezia, CNS dysfunction, twitching, hyperesthesia followed by nystagmus, opisthotonus, tremors, bellowing, snapping of eyelids, occasionally blindness, death within 1 month.

DIAGNOSIS AND TREATMENT

- Characteristic signs - Bloody diarrhoea and tenesmus.
- Faecal examination for oocysts.
- Post mortem examination if no oocysts are found.
- Differential diagnosis
  - Bovine winter dysentery - Epidemic and acute.
  - Johne's disease - chronic diarrhoea
  - Necrotic enteritis - Sudden death
  - Hypomagnesemia - Hyperaesthesia, prolapse of 3rd eyelid.
  - Cobalt deficiency - Pica
  - Salmonellosis - Septicaemia.

Treatment

- Amprolium - 20 -25mg/ kg for 4 to 5 days.
- Sulphamezathine - 0.125 mg/ kg for 13 days.
- Neurological coccidiosis - Calcium boro gluconate and amprolium @ 50mg/ kg

COCCIDIOSIS IN SHEEP AND GOATS

Epidemiology

- *Agent:* P.P.P – 15 days; Pathogenicity is by gametogony and schizogony
- *Age of the host*
    - Upto 6 months of age in lambs and kids; usually 4-7 weeks of age.
- *Transmission:* Newborn lambs / kids pick up the infection by suckling their soiled dams through soiled litter, drinking water and infected pastures.
- *Carrier status:* Adults are symptomless and transmit infection to the young ones.
- *Season:* Flare-up during onset of rains and winter.
- *Risk factors*
    - Unhygienic condition
    - Intensive grazing
    - Oocysts contamination around feeding troughs
    - Confinement in feedlots
    - Weaning

Pathogenesis

- Diarrhoea with foul smelling and with or without streaks of blood.
- Constipation, severe abdominal pain, tenesmus, anaemia, inappetence, unthriftiness, loss of weight, slight rise of body temperature, ailing kids lie down due to abdominal pain, soiling of hind quarters with faeces attracting blow flies.

Diagnosis, Treatment and Control: Similar to cattle.

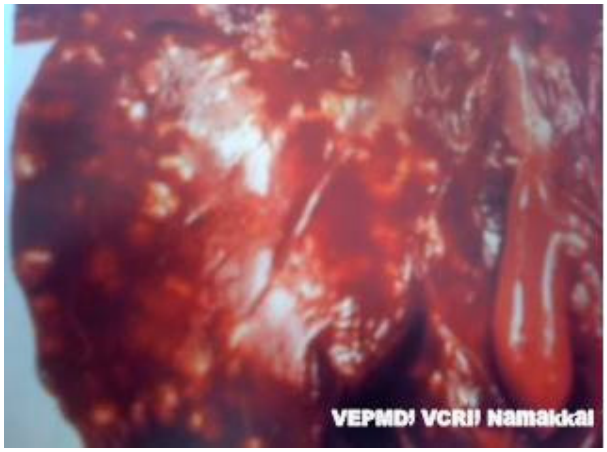
COCCIDIOSIS IN RABBITS

Epidemiology

- *Age:* Commonest around weaning and young rabbits are highly susceptible
- *Risk Factors:* Intensive breeding and rearing under poor sanitation**.**

Pathogenesis

- *Hepatic Coccidiosis*
    - P.P.P- 18 days; Appear 12 days after infection, rabbits die without sign in heavy infections;
    - Clinical signs -Diarrhoea, distension of the abdomen, meteorism, inappetence, constipation, icterus and oedema of the body.
    - Lesions: Enlargement of bile ducts; haemorrhage and enlargement of liver.



- *Intestinal Coccidiosis*
    - P.P.P- 7 days; Occurs frequently.
    - Clinical signs –Diarrhoea, indigestion, bloating, inappetence, reduced weight gain, sudden death without symptoms or convulsion and paralysis.
    - Lesions: Acute/ sub acute, rarely chronic, thickened wall with grayish white deposits, contents are pasty, diarrhoeic and with blood streaks.

Diagnosis

- Demonstration of oocysts in the faeces and postmortem examination.

Treatment

- Sulphonamides like sulphamezathine in drinking water @ 0.2 % effective**.**

COCCIDIOSIS IN DOGS, HORSES AND PIGS

Coccidiosis in Dogs

- Young puppies and kittens are more prone.
- Risk factors - Overcrowding and poor sanitation.
- Infection by predator- prey relationship.
- Usually carry coccidiosis infections but rarely severe disease occur; causes diarrhoeic syndrome
- Treatment - rarely practiced.

Coccidiosis in Horses

- Causes diarrhoea and death in heavy infections only.

Coccidiosis in Pigs

- Clinical condition is not frequent.
- Enteritis in piglets (1-2 weeks age groups) and diarrhoea.
- Older animals are carriers.

CANINE EPERYTHROZOON INFECTIONS

INTRODUCTION

- Canine eperythrozoon infection is caused by *Eperythrozoon* canis. It comes under family Rickettsiaceae genus *Eperythrozoon* .
- Based on phylogenetic similarities, several members of the genus *Eperythrozoon* have recently been transferred to the genus *Mycoplasma* .
- *Eperythrozoon* spp. are transmitted mechanically by arthropods. Ticks are vectors for dogs (*Rhipicephalus sanguineus*).
- Transmission may occur via surgical procedures through blood contamination of instruments.

CLINICAL SIGNS

- Eperythrozoonosis in dogs regarded as an innocuous disease and usually causes only mild anemia. Animals with clinical disease have inappetence, wasting, anemia, malaise, and depression. Body temperature may be elevated but is often normal.
- Haematology shows a macrocytic hemolytic anemia, with anisocytosis, poikilocytosis, and a marked left shift in erythrocyte maturation. The leukocyte count is normal or slightly elevated.
- Lesions includes the dead dogs showing a large area of subcutaneous haemorrhage, congestion, all kinds of mucosa and serosa and mild jaundice intra-abdominal fat, with scattered small bleeding points; muscle oedema, pale or yellowish brown, thin blood , solidification bad, ascites, splenomegaly, soft texture, colour white, hepatomegaly, bleeding points, were yellowish brown, crisp texture, enlarged gallbladder, bile thick; emphysema, abscess, and diffuse bleeding, gastrointestinal mucosal hyperemia, oedema, a large number of point-like or stripe-like bleeding or haemorrhagic spots; the body of the lymph nodes shows bleeding, renal cortex and medulla are a bit like bleeding.

DIAGNOSIS

- *Eperythrozoon spp* are usually coccoid (0.5-1.0 µm diameter). In Giemsa-stained peripheral blood smears, they appear attached to the surface of erythrocytes.
- Occasionally, rod-shaped forms (1-3 µm diameter) can be seen. It is important to differentiate between acute disease, in which the organism is readily identified in peripheral blood smears, and chronic, subclinical disease, in which animals present with a secondary infection and the organism can be difficult to detect in blood smears.

TREATMENT AND CONTROL

- In severely affected animals, transfusion may be required before treatment can begin.
- Tetracyclines used at recommended dose rates have proved effective. The use of disposable needles and correct sterilization of surgical instruments will minimize accidental transmission. Control of arthropod parasites on pets is recommended.