PATHOLOGY OF DISEASE OF RABBITS

Rabbit physiological data

- **Heart rate:** 180–300/minute
- **Respiratory rate:** 30 – 60 /minute
- **Body temperature:** 38.5 – 40^0C
- **Daily food consumption (pellets):** 50g/kg
- **Daily water consumption:** 50–15–ml/kg
- **Daily urine production:** 10–35 ml/kg
- **Body weight adult:** 1–6kg
  new–born kit: 30–80g
- **Life expectancy:** 5–8 years
- **Diploid chromosome number (2n) =** 44

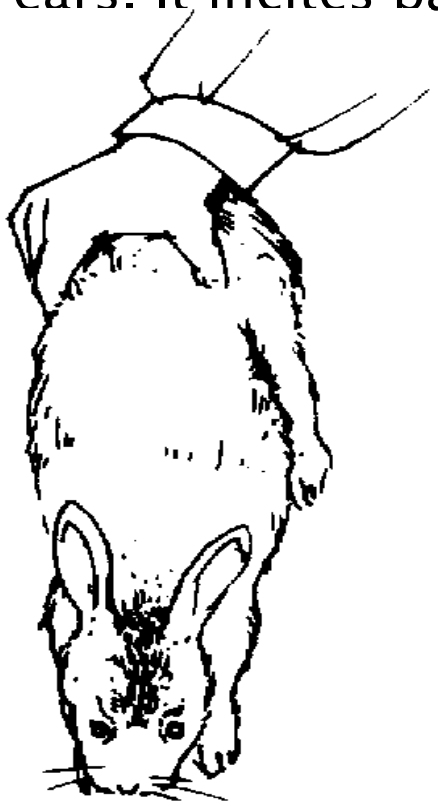
HEMATOLOGY

RBC X 10^{12}/L	Hb g/L	PCV L/L	Platelet X 10^9/L	WBC X 10^9/L	Neutrophils 10^9/L	Lymphocytes 10^9/L	Blood Vol. ml/ Kg
6.5 4.5-8.5	94- 175	0.40 0.31 -0.50	468 180-750	4.0-13	3.0-5.2	2.8-9.0	57-65

* The normal values may vary according to age, sex, breed and function of animals

Approach For Handling

- Rabbits must be approached & handled gently, otherwise rabbit may collapse. They should be lifted by neck scruff or abdominal haunches.
- They must *never* be lifted by their ears. It incites pain.



Continued....

- For carrying rabbits : pick them up by the skin of the shoulders and carry them by the saddle just above the hindquarters, using thumb and index finger. Support is essential.

BACTERIAL DISEASES

1. PASTEURELLOSIS

Etiology

- Pasteurella multocida
  - A gram-negative, non motile.

- Serotypes affecting rabbits
  - Capsular serotype – A or D
  - Somatic serotype – 3 or 12

Transmission

- Predisposing factor:
  - Change in temperature
  - Experimental manipulation
  - Pregnancy
  - Concurrent disease

- Routes of infection
  - Oral
  - Respiratory routes
  - Venereal transmission – less common

- Direct contact is most effective but aerosol transmission can also occur
- Morbidity – as high as 90%
- Susceptibility: Decrease as the age increase

Clinical forms of disease

- Rhinitis with or without sinusitis along with associated conjunctivitis
- Pneumonia
- Otitis media & otitis interna
- Abscess formation
- Septicemia
- Genital infection

Clinical Signs

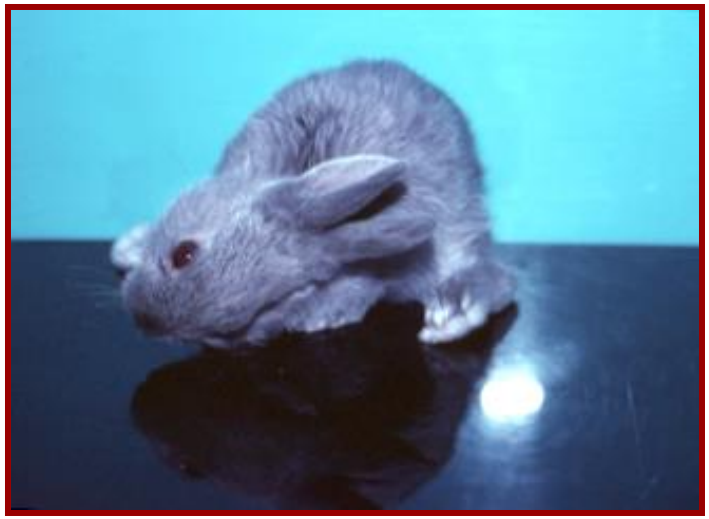
- Rhinitis with or with out sinusitis
  - Commonly known as “SNUFFLES”
  - Incidence may be as high as 60%
  - Serous to mucopurulent nasal discharge, coughing and exudate on the fur of the fore paws
  - If conjunctivities is present also reveals mucopurulent ocular exudate, chemosis, conjunctival reddening, swollen eyelids, epiphora and alopecia around the eyes

Clinical Signs (contd.)

- Pneumonia:
  - Chronic: Asymptomatic as cage housed animals have less demand of oxygen
  - Acute: anorexia, depression, dyspnea, moist rales and death
- Otitis media:
  - It is clinically silent condition progress to otitis interna which is manifested as torticolis
- Abscess formation:
  - Subcutaneous and visceral abscess may be clinically silent for long periods
  - Subcutaneous abscess often rupture spontaneously to external surface
- Septicemia:
  - Acute death without and clinical signs

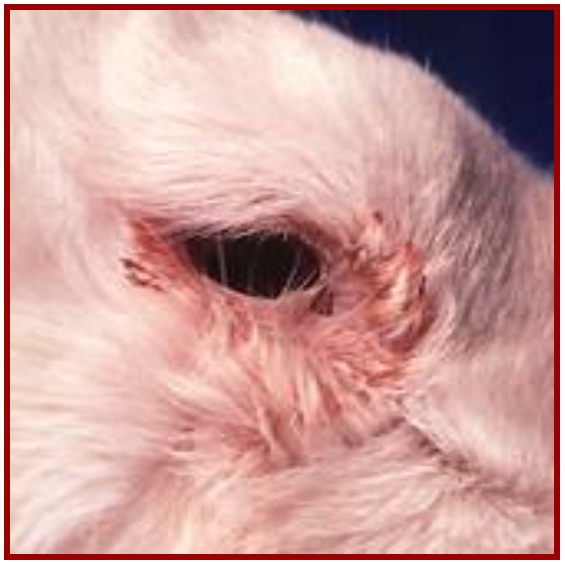
Clinical Signs (contd.)

- Genital tract infection:
  - Female:
    - Serous, mucous or mucopurulent vaginal discharge
    - Congenital infection is asymptomatic or manifested as decrease fertility or abortion
  - Male:
    - Orchitis or epididymitis
    - Decreased fertility
    - Enlarged firm testicles

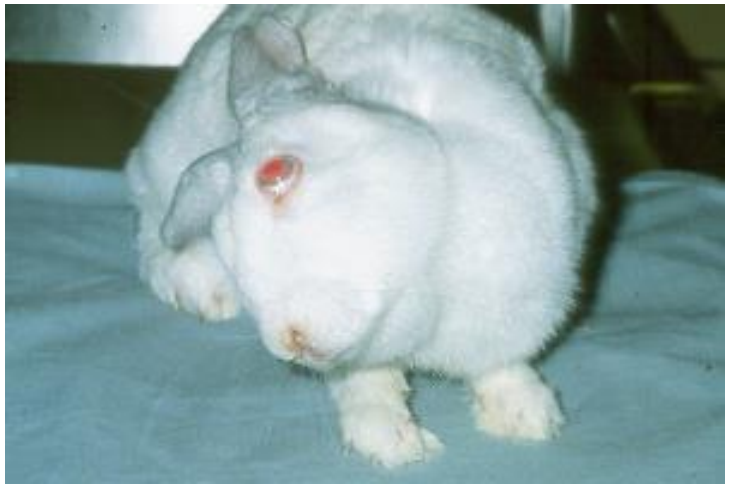


Otitis Media

Snuffle



Conjunctivitis



"Wry neck" = Torticollis
Otitis interna/media

- Exopthalmus

Entropion

Pathological findings

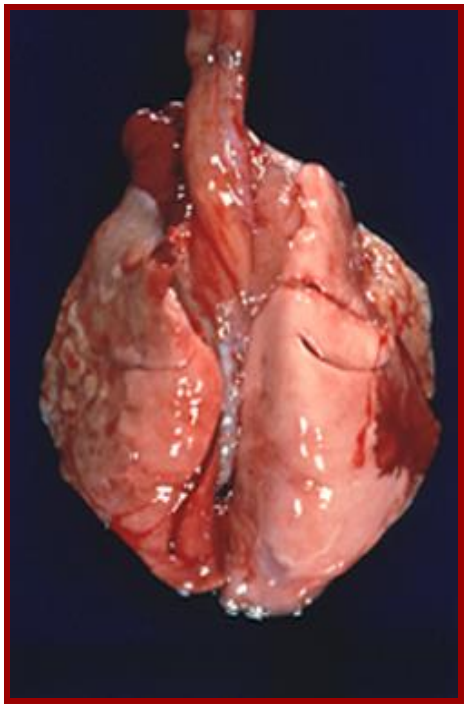
- Rhinitis with or with out sinusitis
  - Mucopurulent nasal exudate
  - Extensive neutrophil infiltration
  - Nasal passage – edematous, inflamed, congested and ulceration of mucosa
  - Atrophy of turbinate bone
  - Purulent conjunctivities
- Pneumonia:
  - Lungs exhibits consolidation, atelectasis and abscess
  - Purulent to fibrinopurulent exudate
  - Ares of hemorrhage and necrosis
  - Fibrinopurulent pleuritis and pericarditis along with acute hepatic necrosis and spleenic lymphoid atrophy

Pathological findings (contd.)

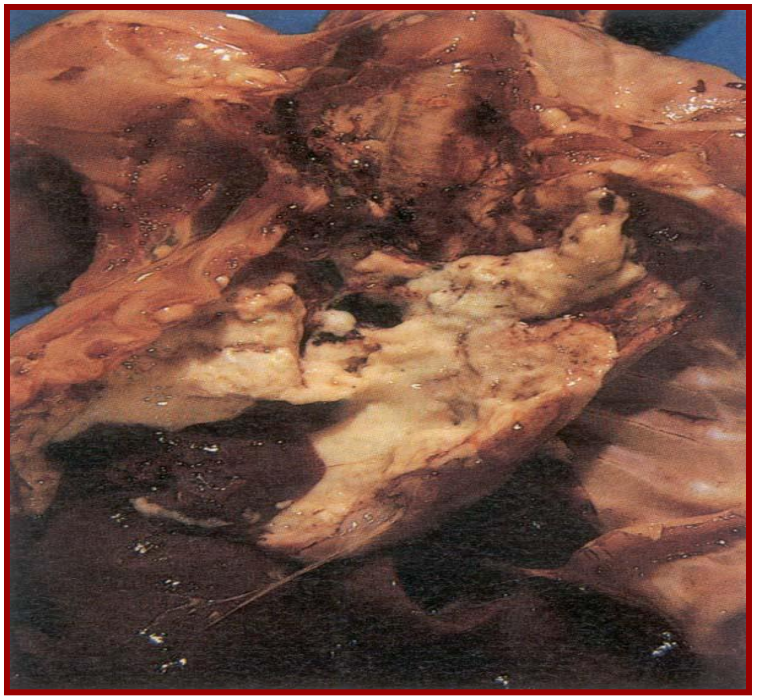
- Otitis media:
  - Suppurative exudate with goblet cell proliferation
  - Lymphocytic and plasma cell infiltration
- Septicemia:
  - Congestion and patechial hemorrhages in many organs.
- Abscess:
  - Necrotic center
  - Contents infiltrate containing polymorphonuclear neutrophils and fibrous capsule.

Pathological findings (contd.)

- Genital findings:
  - Female:
    - Enlarged and dilated uterus
    - In early stage exudate is watery which later on becomes thickens and cream in colour.
    - The exudate contains numerous neutrophils
  - Male:
    - Testes are enlarged and may contain abscesses

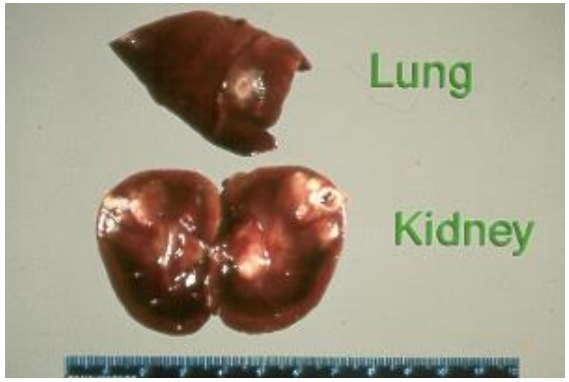


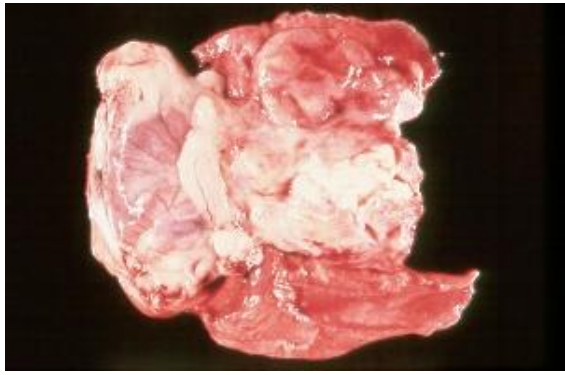
Enzootic Pneumonia

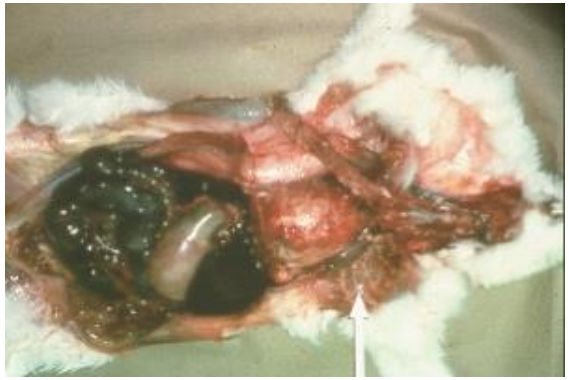


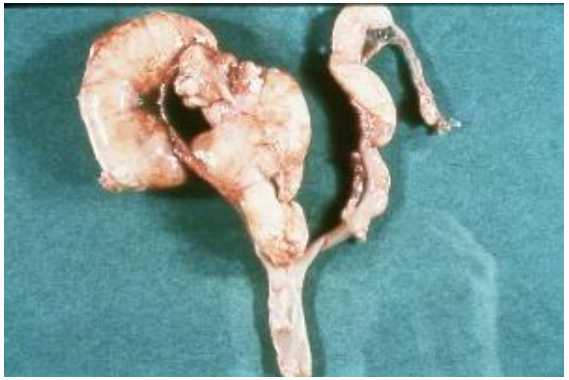
Pasteurelosis(Fibrinous exudates in abdominal cavity)

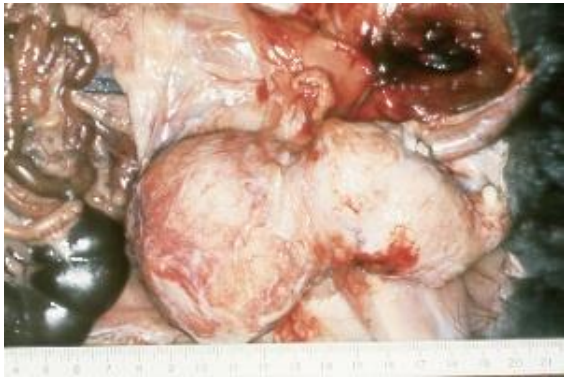
- Sepsis may result in multi organ abscesses











Diagnosis

- Clinical signs
- Post mortem lesions
- Cultural isolation of organisms
- Serological test
  - ELISA
  - Indirect HA
  - Gel diffusion precipitin test

2. TYZZER'S DISEASE

Etiology

- *Clostridium piliforme*
  - A gram-negative, bacillus shaped, spore forming bacteria
  - An obligate intracellular pathogen
  - Organism can not grow in artificial media
  - Cultured in embryonated eggs or tissue culture

Transmission

- Routes of infection
  - Oral – through ingestion of spores present in faeces
- Predisposing factor
  - Any type of stress to animal
- Susceptibility:
  - Out breaks usually occurs in animal of 6-12 weeks of age.

Clinical Signs

- Disease of weanlings
- Profuse watery diarrhea, anorexia, dehydration, lethargy and staining of hind quarters with feces.
- Death within 1-2 days after exhibiting clinical signs
- Mortality – As high as 90%
- Chronic infection usually characterized by weight loss and wasting

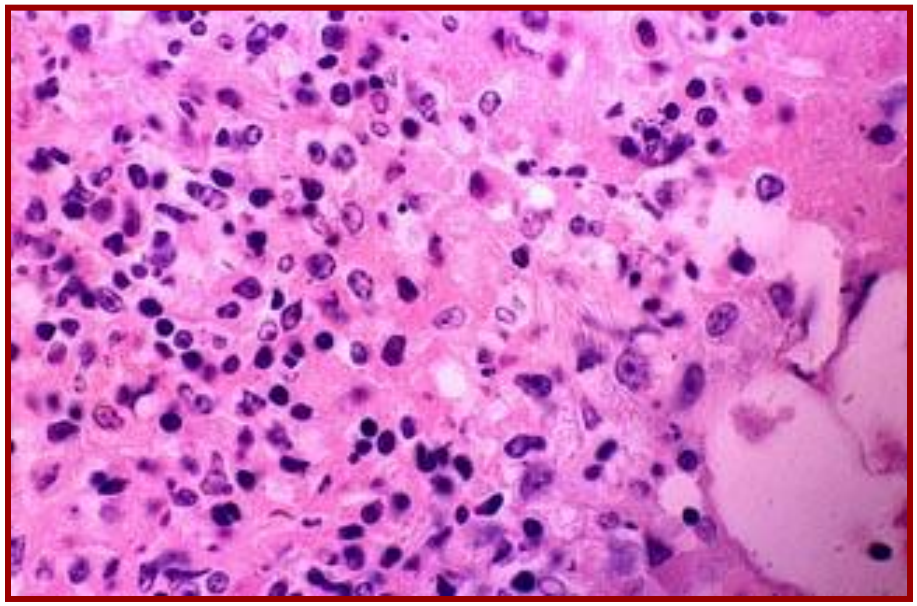
Pathological findings

- Gross findings
  - Hemorrhages on serosal surface of caecum
  - Thickened and edematous intestinal wall
  - Necrotic foci on intestinal mucosa
  - Ileum and colon may also be affected
  - Liver: Numerous pinpoint white foci through out the parenchyma
  - Myocardium: Numerous pinpoint white foci

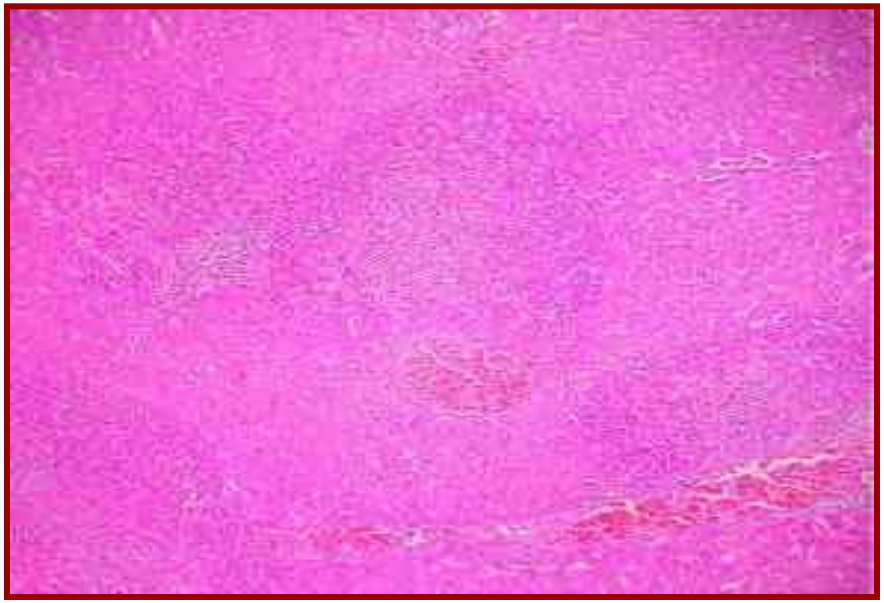
- Histopathological findings:
  - Caecum: Subserosal hemorrhages, oedema, mucosal necrosis may be extended upto deeper layers of caecal wall
  - Hepatic foci corresponds necrotic foci surrounded by polymorphonuclear neutrophils
  - Myocardium: Multifocal necrotic myocarditis
  - Presence of Tangled masses of rod-shaped bacteria is found periphery to lesions when used Warthin-Starry silver stain or Giemsa stain

Multi focal necrosis in liver

Typhlitis



liver, Tyzzer's disease, H&E stain. Necrotic and viable hepatocytes are present. Bacilli are faintly stained.



Multifocal areas of necrosis in liver

Diagnosis

- Clinical signs
- Post mortem lesions
- Demonstration of intracellular bacteria in tissue sections stained with Warthin-Starry silver stain or Giemsa stain
- Serological test
  - ELISA
  - Indirect immunofluorescence

3. ENTEROTOXEMIA

Etiology

- *Clostridium spiroforme*
- *Clostridium difficile*
- *Clostridium perfringens type A*
- *Clostridium welchii type A*

Transmission

-
  - Oral – Ingestion of contaminated feed and water
- Predisposing factor
  - Abrupt change in feed
  - Antibiotic administration
  - Co-infection with other bacteria
  - In older age animals disruption of normal gut flora leads to enterotoxemia
  - Other stress to animals
- Susceptibility:
  - Disease can be produced in all age group animal but weanling are more succeptible.

Clinical Signs

- Acute death without clinical signs
- Watery brown diarrhea
- Staining of perianal region
- Anorexia
- Dehydration
- Polydipsia
- Pyrexia or hypothermia
- Bloat
- Grinding of teeth

Pathological findings

- Gross findings
  - Caecum:
    - distended with excessive gas and dark brown fluid
    - May be serosal paint brush hemorrhages
    - Mucosal hemorrhage and ulcers
  - Colon & Ileum:
    - May contain mucus or gas and dark brown fluid
    - Also reveals the extension of the serosal hemorrhages

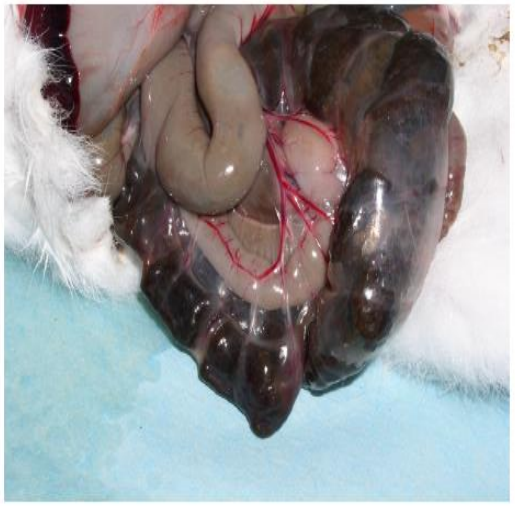
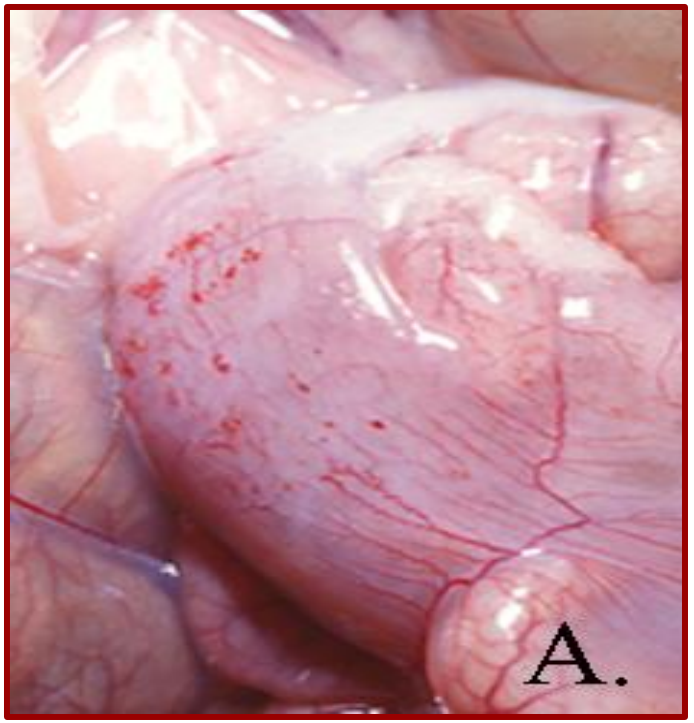
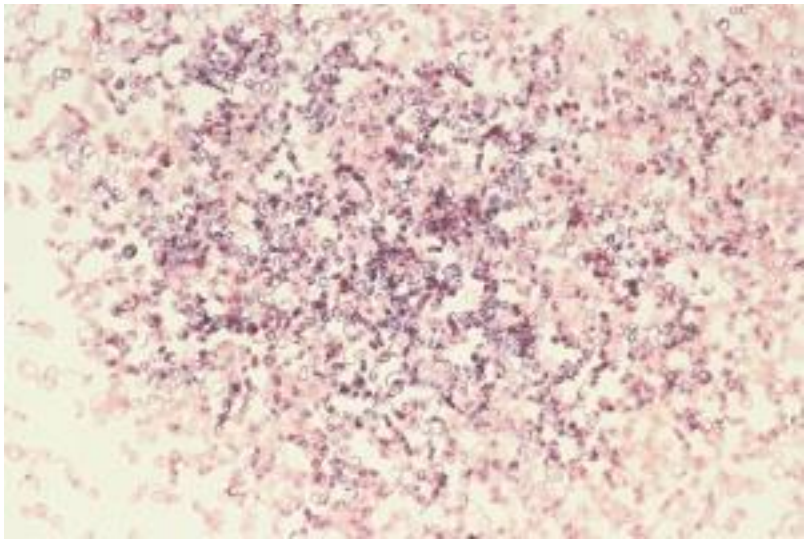


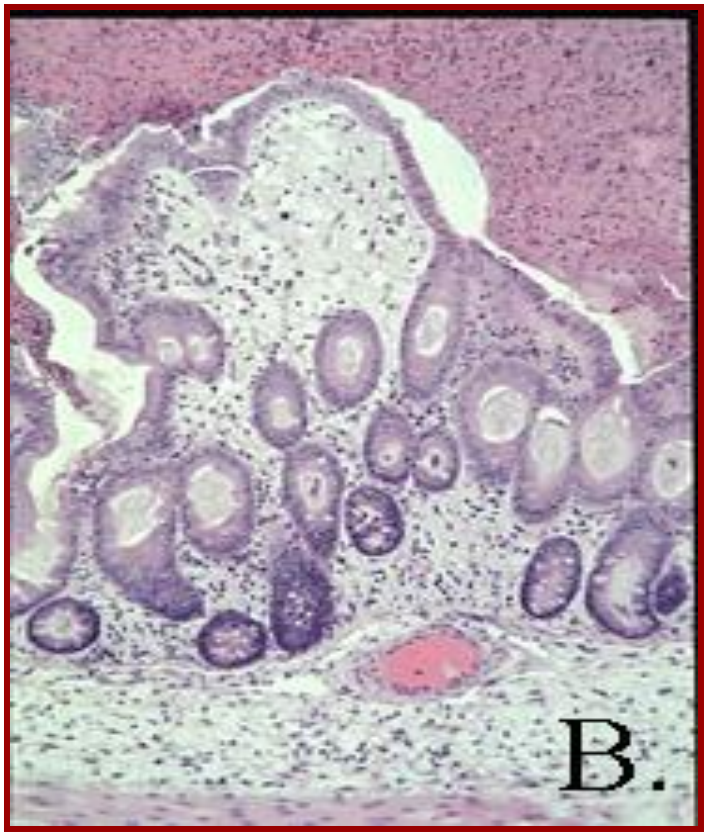
Figure 1. Six-week-old rabbit, dead 5 days after experimental reproduction of ERE with inoculum TEC3, showing total caecal impaction, one of the main gross lesions of the disease. There are no inflammation or congestion visible on the caecum wall.



fluid-filled edematous caecum with serosal congestion and hemorrhage.



Gram stain of a caecal smear
Helically coiled semicircular shaped organism



swelling and loss of enterocytes and pseudomembrane formation

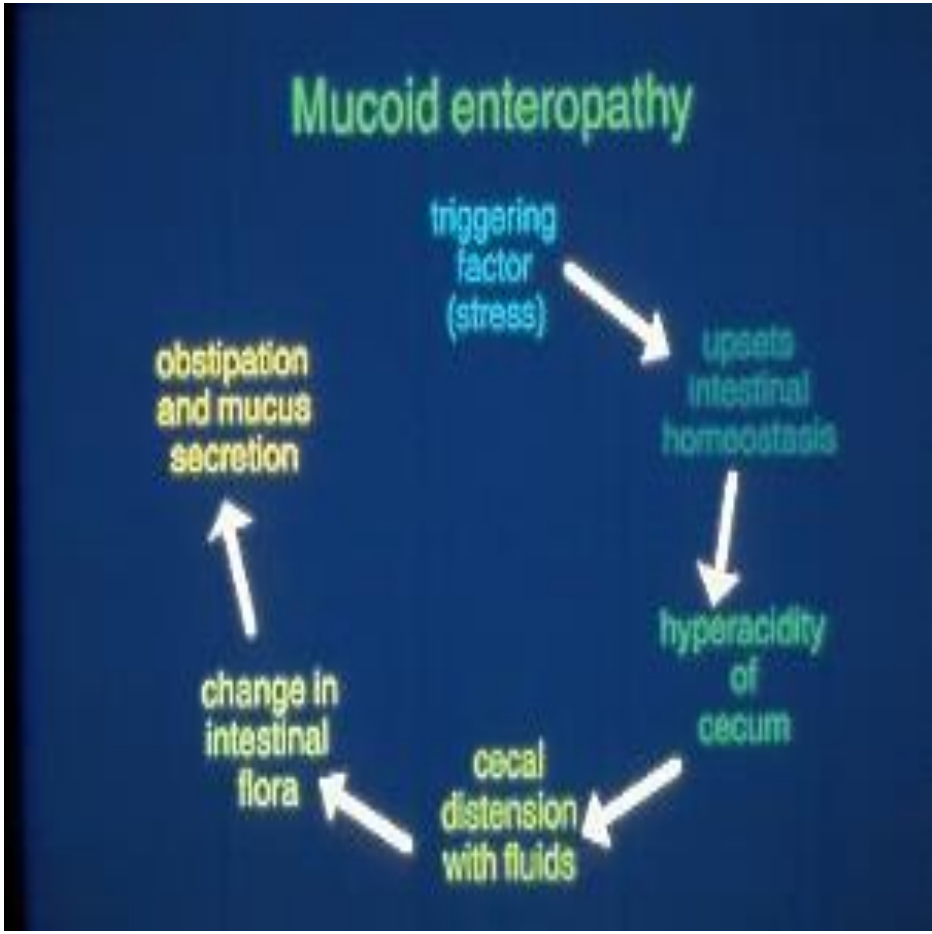
Diagnosis

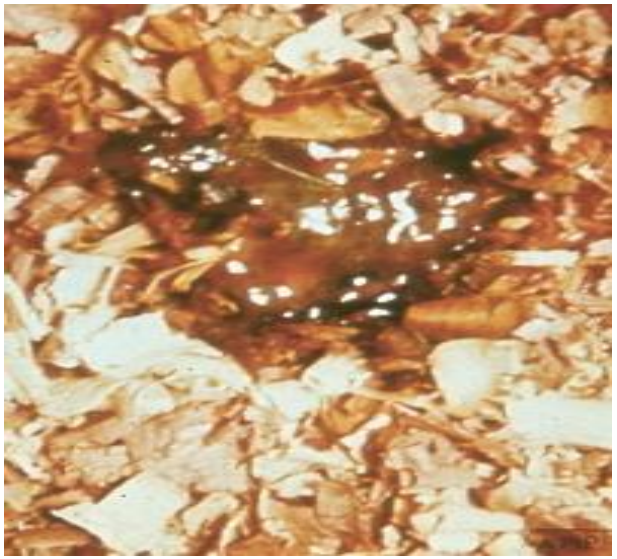
- Clinical signs
- Post mortem lesions
- Demonstration of intracellular bacteria in tissue sections stained with Warthin-Starry silver stain or Giemsa stain
- Serological test
  - ELISA
  - Indirect immunofluorescence

4. Mucoid Enteropathy

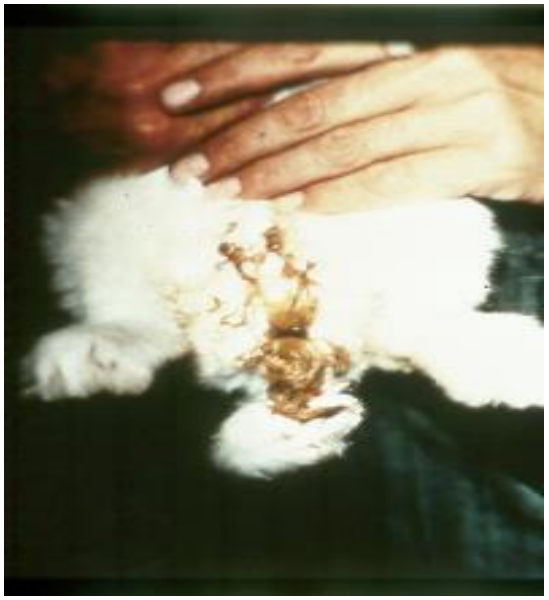
Mucoid Enteropathy

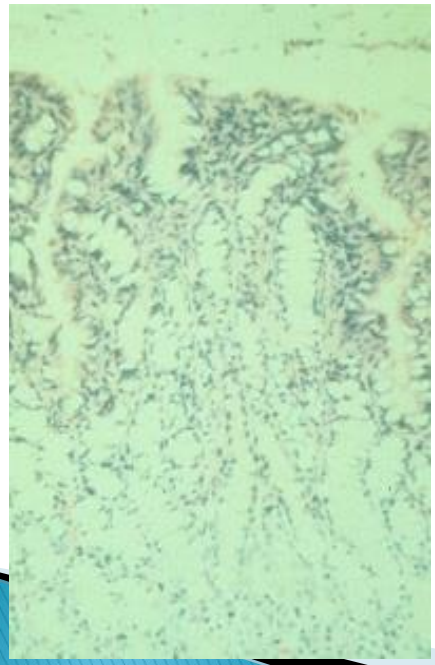
- Etiology
  - Similar to enterotoxemia
  - Stress induced
  - Suspect *E. coli* toxin
- Clinical signs
  - Anorexia, depression, mucoid diarrhea, dehydration
  - Abdominal pain
  - Teeth grinding
  - Hunched posture
- Diagnosis
  - Clinical signs and lesions
  - Goblet cell hyperplasia
- Treatment
  - Supportive care, +/- antibiotics
- Prevention
  - High fiber diet
  - Modulate stressful events



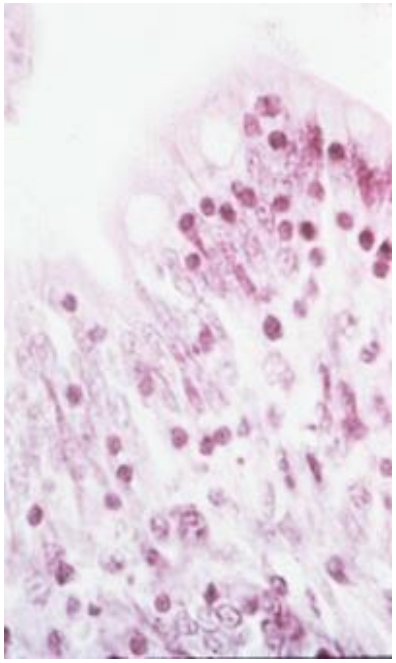


Mucoid diarrhoea





Goblet cell hyperplasia without inflammation



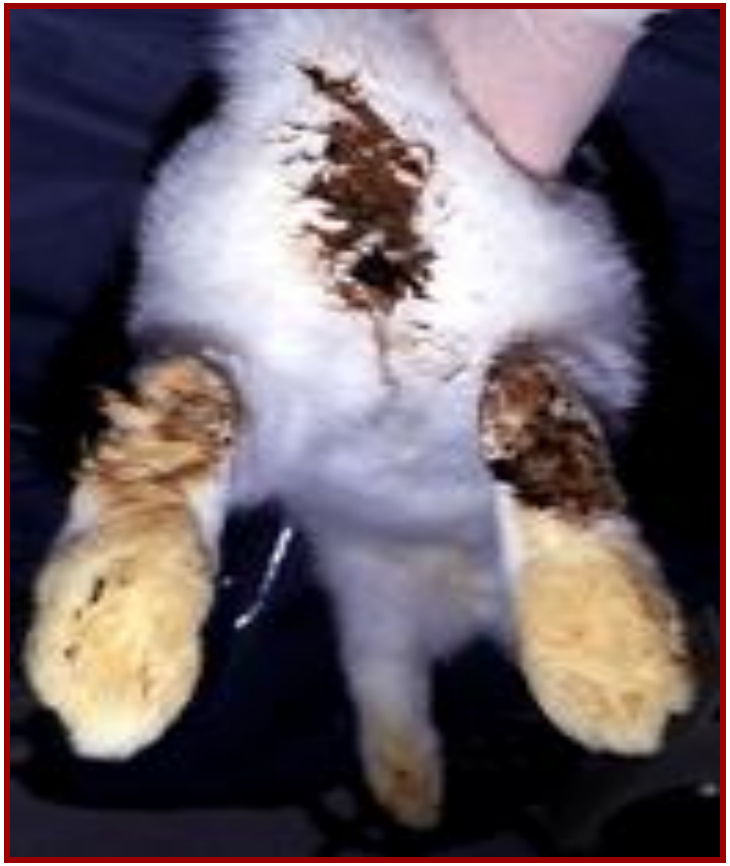
5.
Colibacillosis

Etiology

- *Escherichia coli*
  - A causative agent of diarrhea in rabbits
  - Strain:
    - RDEC-1
  - Serotype of RDEC-1 strain
    - O15:H – Most virulent and affects weanlings
    - O109:H2, O103:H2, O128 and O132

Clinical Signs

- Typically affects 4-6 weeks age of animals
- Also affects 1-2 weeks old suckling animals
- Occurs in 3 different Clinical forms based on infecting strain of bacteria
  - Neonatal diarrhoea with high mortality
    - Suckling rabbits reveals severe yellow diarrhoea and mortality
    - Associated with serotype O109:H2
  - Weanling diarrhoea with high mortality
    - Develops profuse, watery diarrhoea with dehydration, anorexia, weight loss, stunted growth and death if infecting strain highly virulent
    - Associated with serotype O103:H2 or O15:H
  - Weanling diarrhoea with low mortality
    - Mild diarrhoea in weanlings
    - Associated with serotype O123, O128 and O132

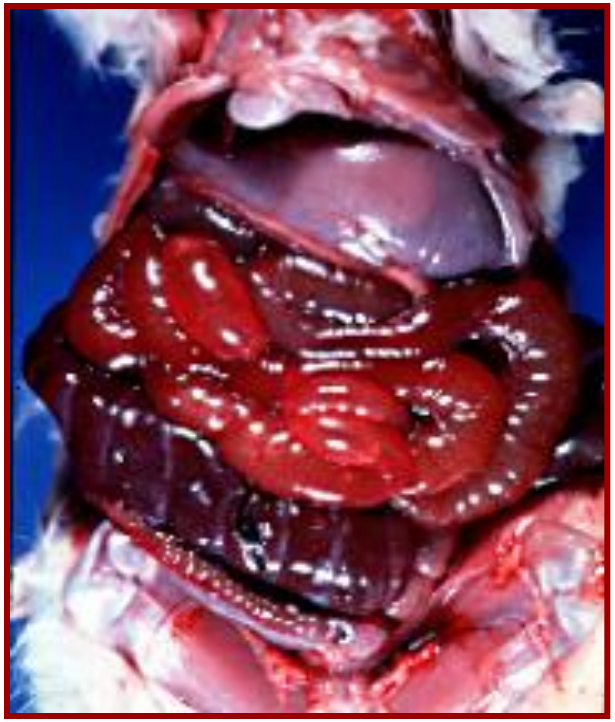


Diarrhoea

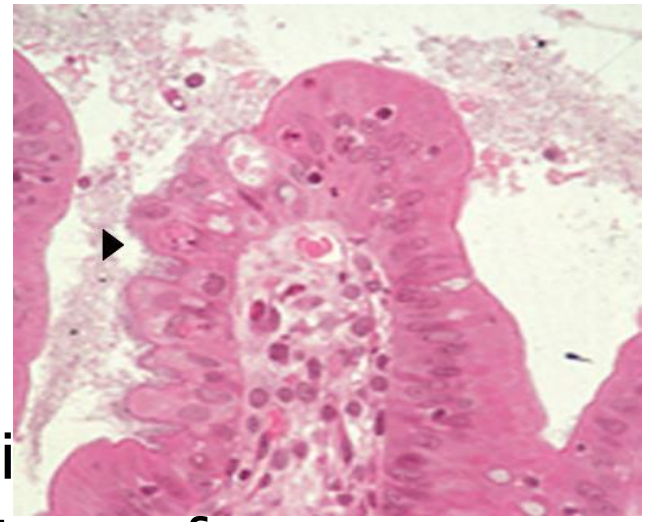
Pathological findings

Gross findings

- Oedematous, thickened and mucosal ulceration of ileal, caecal and colonic wall
- Content of the caecum may be watery and brown
- Also presence of serosal hemorrhages
- In neonates: Entire intestinal tract will be affected and content yellow – brown feces.
- Enlargement of mesenteric Lymph nodes



- Histopathological findings:



  - Ileum, caecum and colon:
    - Atrophy of villus and its fusi
    - Flattening and disorganizations of epithelium
    - Focal necrosis of mucosa
    - Presence of neutrophils in lamina propria
    - Edematous sub mucosa
    - Neutrophils and enterocytes may be present in the intestinal lumen.
    - Colonies of coliforms may be present on the intestinal mucosal surface.

Diagnosis

- Clinical signs
- Post mortem lesions
- Culturing of causative organisms from the faeces of rabbit with diarrhoea
- Definitive diagnosis based on somatic and flageller serotyping to correlate the strain with known enteropathogenic strains

6. TREPONEMATOSIS

Etiology

- Synonyms
  - Venereal spirochetosis
  - Rabbit syphilis

- *Treponema paraluis cuniculi*
  - A gram-negative, spiral shaped rod, spore forming bacteria
  - It is closely related to T. pallidum a causative agent of human syphilis.

Transmission

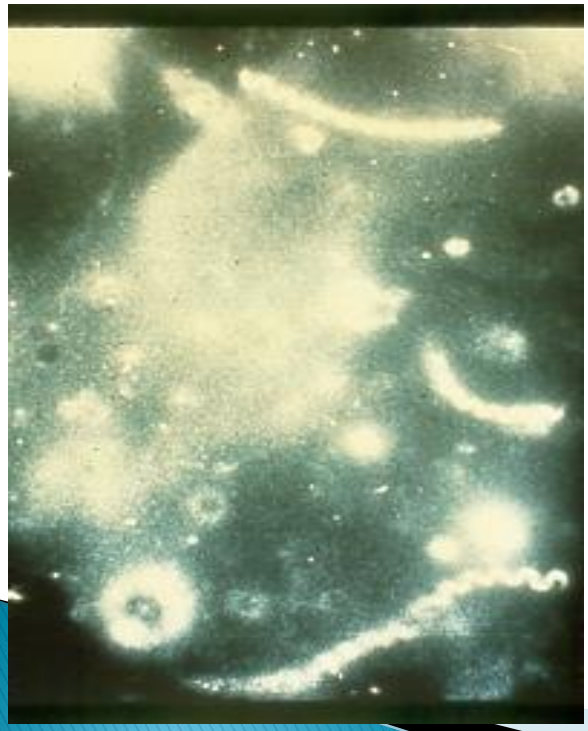
- Routes of infection
  - Venereal – Organisms are transmitted between animals during breeding
  - Very rarely the organisms are found in nonbreeding animals
  - Clinical signs do not appear for 3-6 weeks after exposure and sero-conversion may not occur untill 8-12 weeks after exposure

Clinical Signs

- Typical lesions occur in vulvular or preputial areas
- Lesions begin swelling and erythema, often with vesicles or papules
- Lesions may also occurs at other mucocutaneous junctions
- Lesions progress to ulceration, followed by scaling and crusting over the ulcer
- Regional lymphnodes may be enlarged
- Lesions are chronic in nature and may be resolve after many weeks

Mucocutanious lesions

Genital lesions



Darkfield microscopy
Visualize spirochetes

Pathological findings

- Histopathological findings:
  - Epidermal hyperkeratosis, hyperplasia and acanthosis with ulceration
  - An exudative crust over the ulcer
  - Macrophage and plasma cell infiltration
  - Hyperplasia of regional lymph nodes
  - Spirochetes may be found in the lesions with Warthin-Starry silver stain

Diagnosis

- Clinical signs
- Demonstration of spirochetes in the lesions
- Wet mounts of scrappings from lesions examined under dark field microscopy where the organisms are demonstrated as corkscrew motility.
- Serological diagnosis is made using the serology test used to diagnosis of T. pallidum infection in human as both the organism shares many antigens. The serological tests used are
  - Microhemagglutination
  - Venereal disease research laboratory slide test (VDRL)
  - Rapid plasma reagin card test (RPR)

7. PROLIFERATIVE ENTEROPATHY

Etiology

- *Lawsonia intracellularis*
  - A gram-negative, obligate intracellular bacterium
  - Plays key role in the development of proliferative bowel disease in Hamsters and pigs

Clinical Signs

- Weanlings are most commonly affected
- Diarrhoea, depression and dehydration which resolves within 1-2 weeks
- Rarely leads to death
- In some animals the disease is asymptomatic
- Leads to high mortality when associated with secondary infection with E. coli

Pathological findings

- Gross findings
  - Thickening and corrugation of ileum
  - Jejunum, caecum and proximal colon shows variety of lesions
  - Enlargement of mesenteric lymph nodes
  - Clinically affected animals shows watery content in caecum

- Histopathological findings:
  - Thickened intestinal mucosa
  - Crowded, elongated and some time branching crypts are seen in intestinal mucosa
  - Villi of small intestine are often blunted
  - Inflammation is not present in all the cases but in some cases the infiltrates consisting of plasma cells and histocutes is observed

Diagnosis

- Clinical signs
- Histological identification of rod-shaped to curved spiral, silver staining bacteria within the apical cytoplasm of crypt enterocytes.
- Immunochemistry
- Identification of nucleotide sequence by polymerase chain reaction for the sample collected from jejunum, ileal and colonic tissues of infected rabbits

VIRAL DISEASES

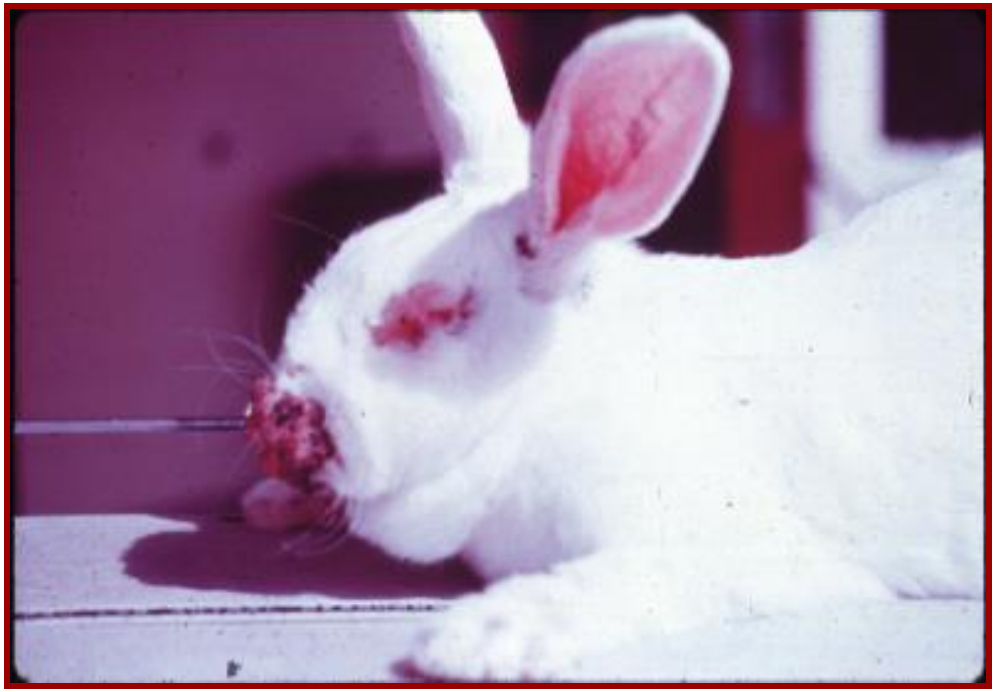
1. POXVIRUS INFECTION

Etiology

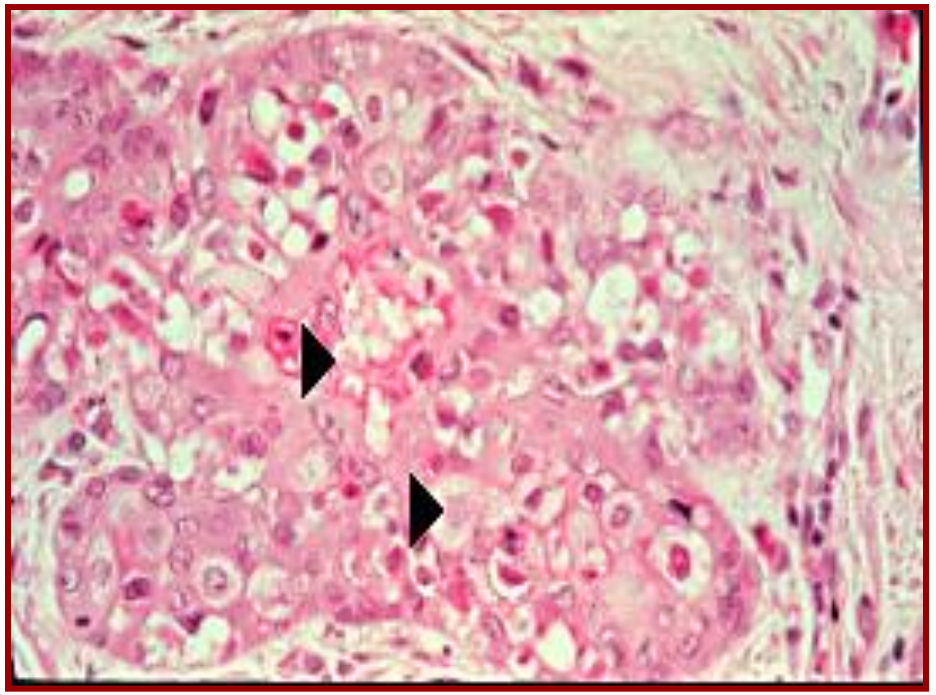
- Myxomatosis caused by Myxoma virus
- Rabbit fibroma virus
- Rabbit pox virus

Myxoma virus

- Produces Myxomatosis
- Oryctolagus genus is succeptible
- Develops a fatal disease characterised by numerous mucinous skin lesions
- Histology shows “Myxomas” to be composed of undifferentiated stellite mesenchymal cells embedded in a matrix of mucinous material and interespersed with capillaries and inflammatory cells
- Diagnosis depends on culture of the virus from infected tissues.
- Disease is spread
  - by fleas
  - by mosquitoes
  - by direct contact



Myxomas of mouth



Intracytoplasmic inclusions

Rabbit fibroma virus

- Fibroma virus is antigenically related to Myxoma virus
- Less virulent cause skin tumors
- It is spread by arthropods
- Fibromas are flat, subcutaneous, easily movable tumors
- Where as papilloma arise from the skin and are heavily keratinised and projected outward.

Rabbit pox virus

- Rarely occurring disease
- Affected rabbits may or may not present pox lesions on the skin
- Animal have fever and nasal discharge 2-3 days after infection
- Eye lesions are seen as blepheritis, conjunctivitis and keratitis with subsequent corneal ulcers
- Skin lesions when present are wide spread
  - Begins as a rash and progress to papules upto 1 cm in diameter by 5 day of infection
  - Enlargements of lymph nodes
  - Face is oedematous and there may be lesions in oral cavity
- Necropsy reveals extensive nodules in many organs and wide spread necrosis.
- Histologically characteristic cytoplasmic inclusion are rare in this disease
- Disease is spread by aerosols and difficult to control

2. HERPESVIRUS INFECTION

Etiology

- Two Herpes viruses are isolated from rabbit kidney cultures
  - Leporid herpesvirus 1 (Herpesvirus sylvilagus)
  - Leporid herpesvirus 2 (Herpesvirus cuniculi)

- No incidence of naturally occurring disease

3. PAPILLOMAVIRUS INFECTION

- The virus leads to horny warts primarily on the neck, shoulders and abdomen.
- In natural out breaks papillomas are more common on the eye lids and ears
- Small percentage of papillomas are transformed into squamous cell carcinoma indicating virus is ONCOGENIC and hence the virus is extensively used as a model for oncogenic study
- In naturally occurring disease the lesions observed are small, white, discrete growths on the ventral surface of the tongue.
- Microscopically it reveals typical papillomas
- Most of the lesions eventually regress spontaneously.

4. ROTAVIRUS INFECTION

- It is common infection in rabbits
- Many colonies are serologically positive for Rota virus
- Rota virus can be isolated from faeces
- Clinically it leads to mild diarrhoea but some studies have shown even mortality
- It is mildly pathogenic to rabbits and hence required secondary infection to produce clinical disease and mortality
- Secondary infection with E. coli will produce more serious disease
- Older age rabbits are more resistant to Rota virus and E. coli combination infection
- Very young rabbits are protected by passive immunity when present but if absent they are quite susceptible
- Rabits of weaning age are most susceptible

5. CORONAVIRUS INFECTION

- Clinically the rabbits developed severe diarrhoea and most died within 48 hours of onset of clinical signs
- Leads to death of the animal due to congestive heart failure
- Microscopically reveals wide spread necrosis of the heart muscle
- Unknown environmental factors contributes to the severity of disease out break

6. CALCIVIRUS INFECTION

- **Family**: *Caliciviridae*
- **Genus**: *Lagovirus*
- **Species**: *Rabbit hemorrhagic disease virus*

Transmission: Direct contact
contaminated fomites
Urine & Faeces

Pathogenesis:

- The virus has a predilection for hepatocytes where it replicates in the cytoplasm.
- Hepatic necrosis begins in periportal areas, then spreads to affect the entire lobule.
- In experimental infections, there is up to 90% mortality.

- The incubation period is short – 1 or 2 days

Clinical signs:

- Seen only in rabbits older than 40-50 days of age. Sudden death, fever, depression, CNS signs, serosanguineous nasal discharge.
- Sudden death with no previous signs is quite common
- A peracute disease of adult rabbits that results in hepatic, enteric and lymphoid necrosis and a terminal massive coagulopathy.

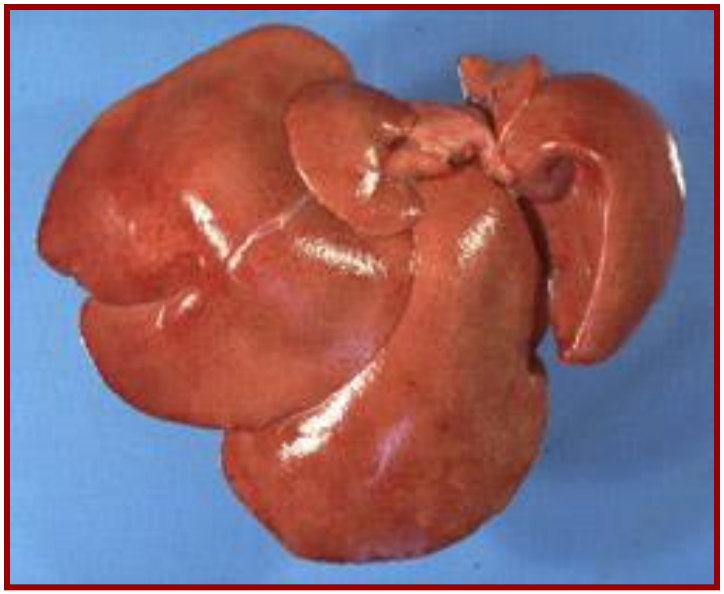
Rabbit. Severe epistaxis.

- Gross lesions:

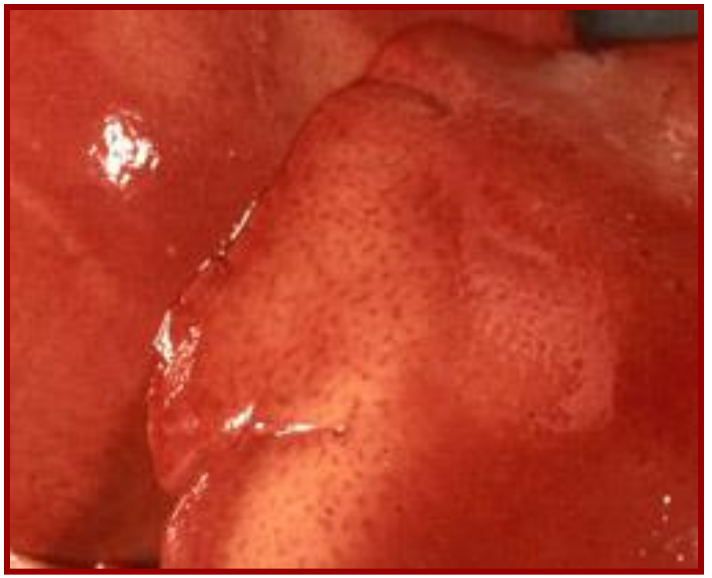
  Hepatomegaly
  splenomegaly
  Hemorrhage and serosal ecchymoses.

- Histopathology:

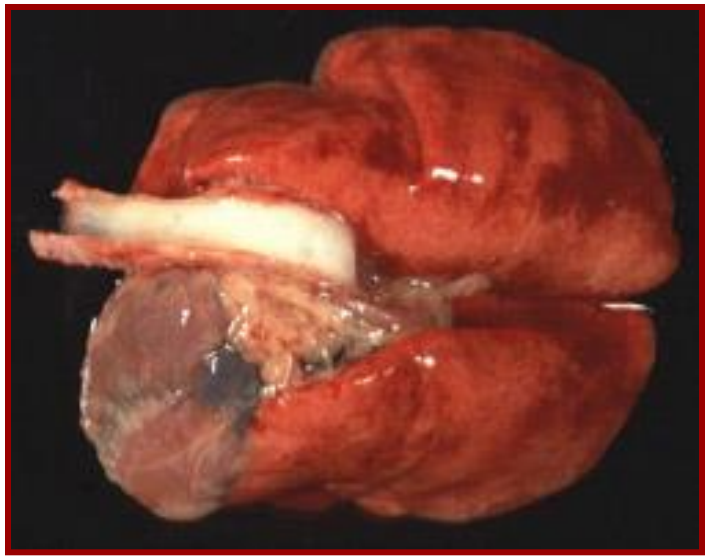
  Hepatic necrosis with polymorphonuclear cell infiltration Cryptal necrosis
  Pulmonary edema
  Hemorrhage
  Lymphocytolysis
  Fibrin thrombi in small vessels of multiple organs



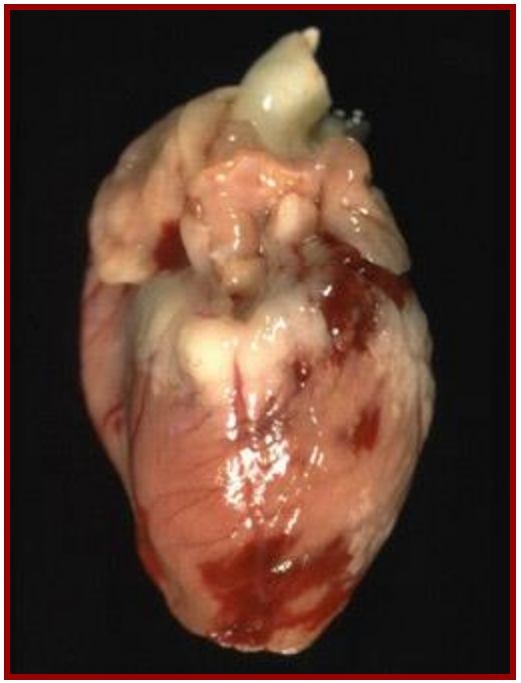
Rabbit, liver. All liver lobes are swollen, pale and have a reticular pattern.



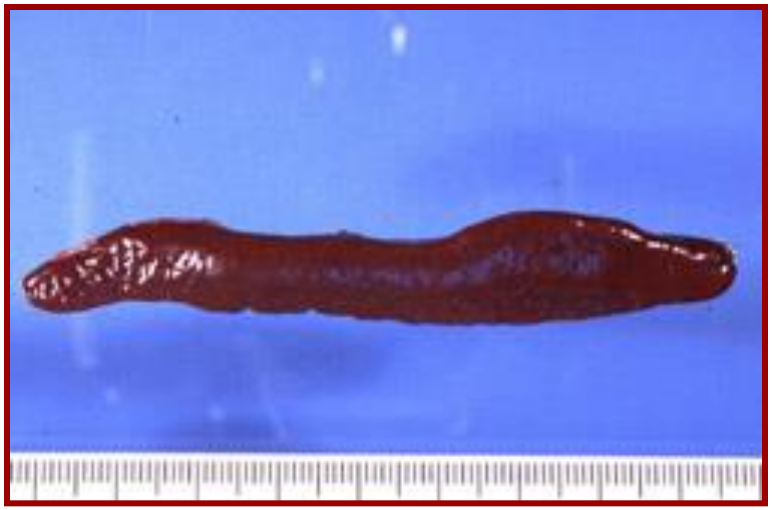
Rabbit, liver. There is a large area of pallor (necrosis) with a prominent reticular pattern.



Rabbit, lungs. The trachea is filled with foam, and the lungs are mottled and noncollapsed (severe pulmonary edema).



Rabbit, heart. There are multiple epicardial hemorrhages.



Rabbit, spleen. The spleen is markedly enlarged and congested.

Rabbit, kidney. There are petechiae throughout the cortex and the medulla is severely congested.

Diagnosis:

- ✓ **Hemagglutination test.**
- ✓ Negative contrast electron microscopy of liver tissue
- ✓ ELISA

PROTOZOAL DISEASES

1. HEPATIC COCCIDIOSIS

- Etiology:
  - Eimeria stiedae (Monocystis stiedae, Coccidium oviforme and Coccidium cuniculi)
  - Younger rabbits are more susceptible

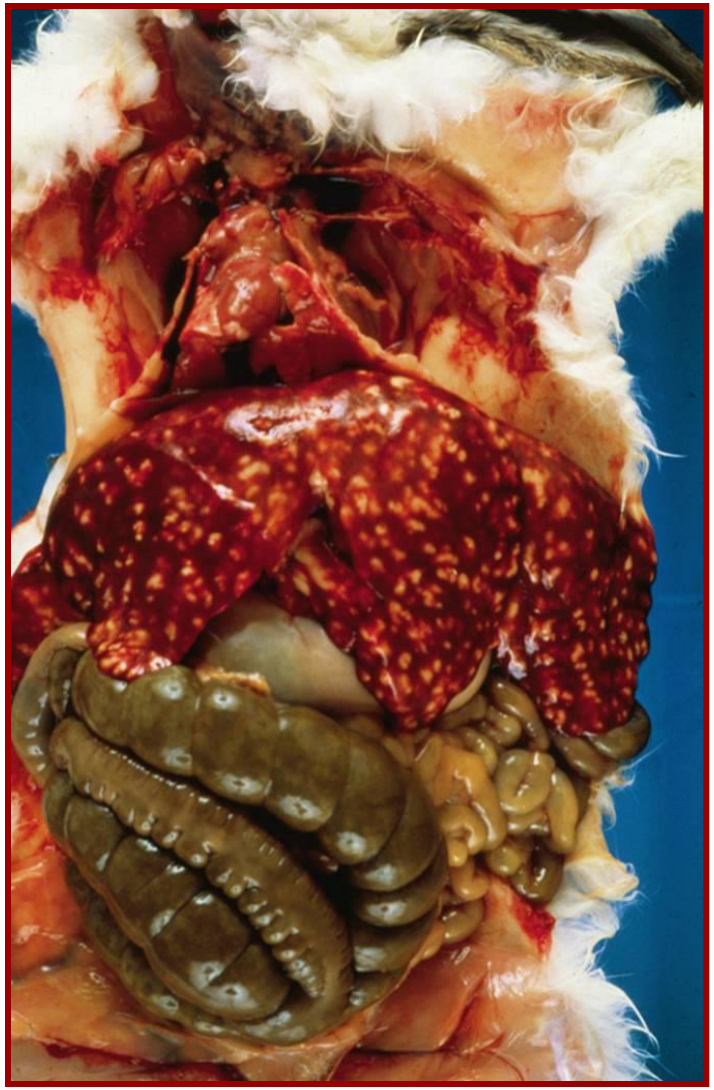
- Transmission:
  - Fecal-oral route
  - Experimentally also transmitted by intravenous, intraperitoneal and intramuscular route

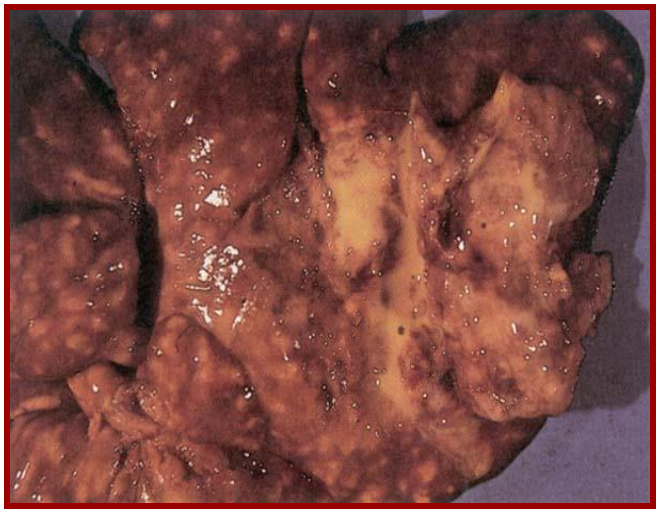
Clinical signs:

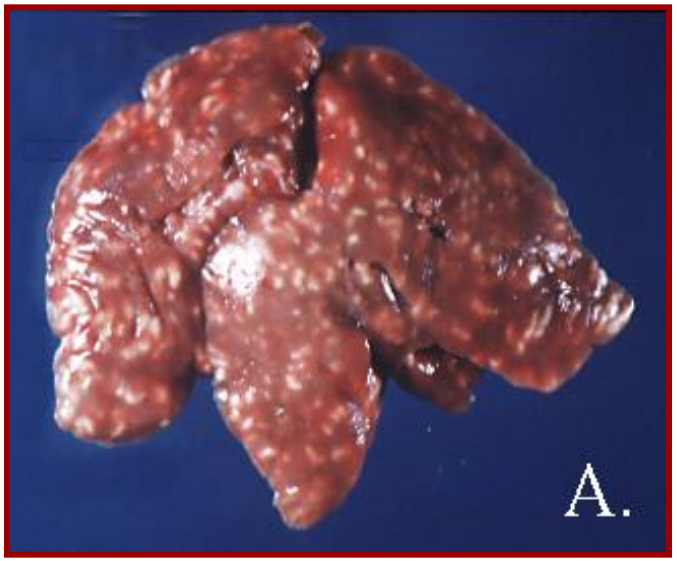
- Mild infection – No apparent disease
- Most of the clinical signs are due to interruption of normal hepatic function and blockage of bile duct
- Serum bilirubin increases upto 305 mg/dL within 6 days of infection
- Decreased growth rate and weight loss
- Diarrhoea is seen at terminal stage of disease

Pathological findings:

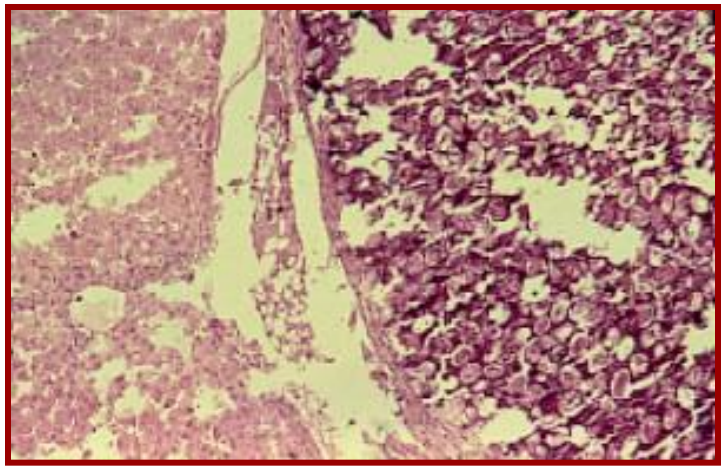
- Liver: Enlarged and discoloured with multifocal yellowish white lesions of varying size
- Exudate in biliary tree along with dilatation of bileduct
- Microscopically, papillomatous hyperplasia of the ducts along with multiple life-cycle stages of the organism in the biliary epithelium



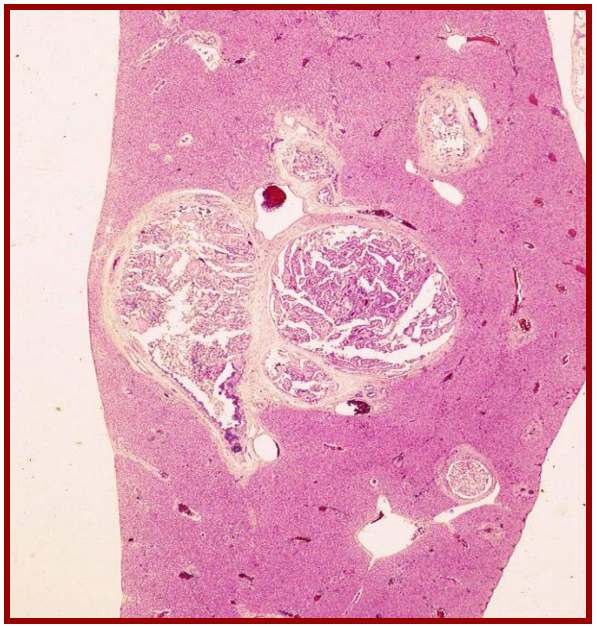




Coccidiosis in liver – rabbit



Haemorrhages in liver



Immature stage of Eimeria stiedae in liver

- Diagnosis:
  - Clinical signs
  - Examination of faecal material by floatation or concentration method
  - Detection of oocysts within the gallbladder and impression smear
- Potential research complications:
  - It is as a results of liver damage and decreased weight gains complicate both the supply of rabbits for research as well as adversely affected the research protocol

2. INTESTINAL COCCIDIOSIS

Etiology:

- *Eimeria intestinalis*
- *E. flavescens*
- *E. irresidua*
- *E. magna*
- *E. media*
- *E. piriformis*
- *E. neoleporis*
- *E. perforans*

Transmission:

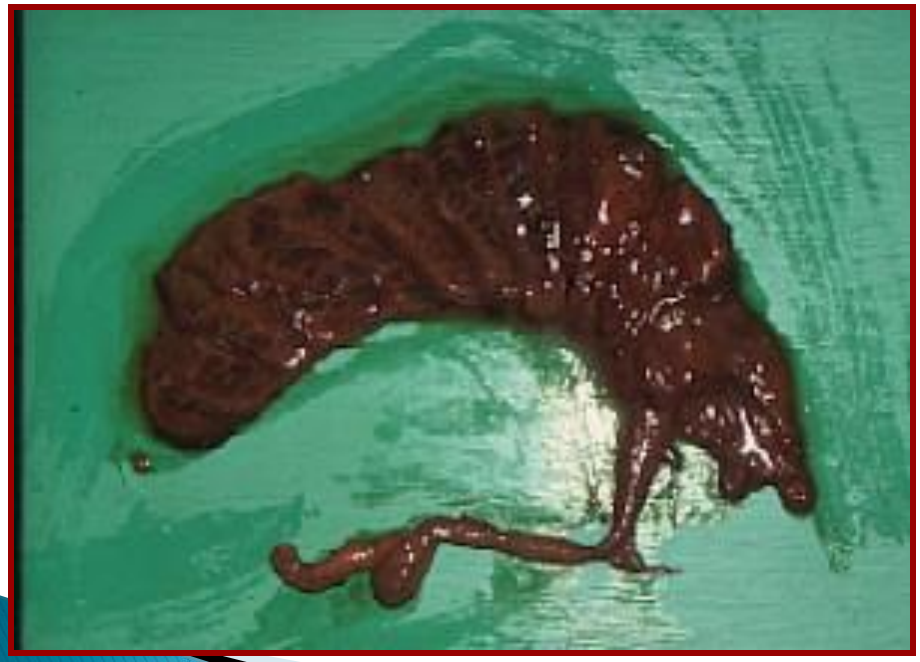
- Unsporulated oocysts are passed in the faeces and are NON INFECTIVE
- Such oocysts gets sporulated to an infective stage within 3 days after shedding
- Ingestion of infective oocysts leads to infection *ie* transmission through Faecal-oral route

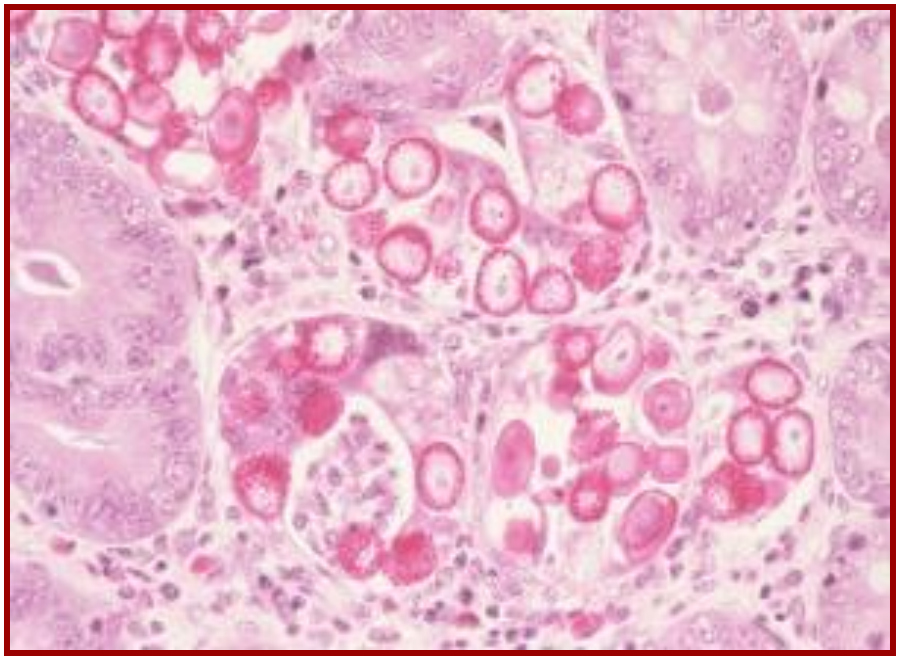
Clinical signs:

- It may be subclinical
- Symptoms ranges from mild to severe and resultant death
- Mortality is most likely to be observed in post weanling rabbits
- Clinical signs also depends on the presence of species of coccidia
- There may be severe diarrhoea, weight loss or mild reduction in growth rate
- Death is usually associated with severe dehydration due to diarrhoea

Pathological findings:

- Lesions are apparent in small and large intestine
- Necrotic areas of the intestinal wall appear as white foci
- Location and extend of the lesion depends on the species of coccidia





- Diagnosis:
  - Clinical signs
  - Examination of faecal material by floatation or concentration method
  - Using polymerase chain reaction (PCR) technology to detect Eimeria spp. In the faeces.

- Susceptibility:
  - Post weanling rabbits are most susceptible

3. CRYPTOSPORIDIOSIS

- Caused by *Cryptosporidium cuniculus*
- Organisms are found in the intestinal tract of rabbits
- Transmission is likely by ingestion of thick walled sporulated oocysts
- Clinical signs in rabbits is not well described
- Few signs and lesions described are dilatation of small intestine without diarrhoea
- Histologically it is characterised by presence of shortened, blunted villi and mild oedema of lamina propria
- Dilated lacteals of ileum
- Interestingly, inflammatory response is also observed

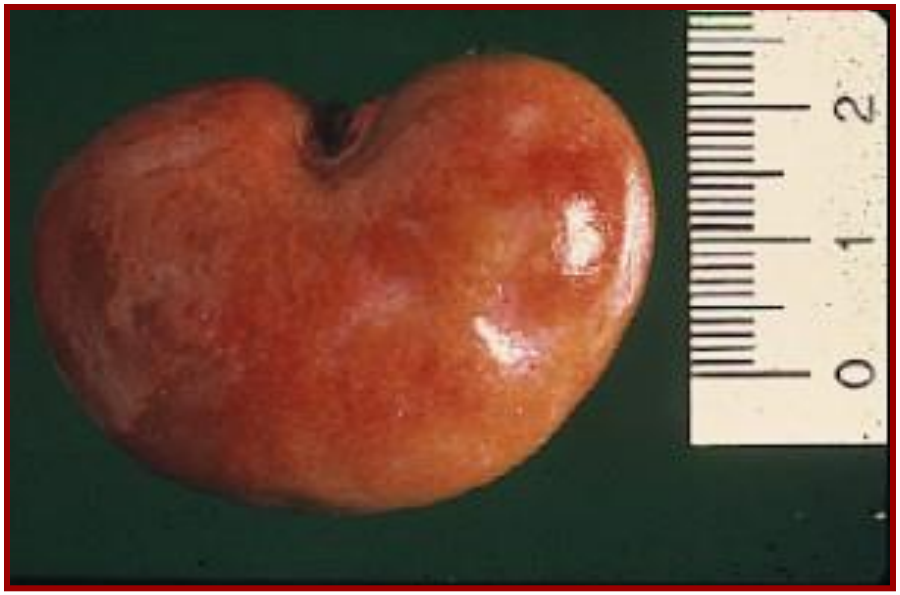
4. ENCEPHALITOZOONOSIS

- It has zoonotic importance
- Caused by *Encephalitozoon cuniculi*
- Clinically it cause motor paralysis in young rabbits and this disease is usually latent in rabbits
- Other clinical signs are convulsions, tremors, torticolis, paresis and coma
- Route of infection is not known but the organism are found in the urine of the infected animal
- Transmission occurred by oral administration of urine from infected rabbits
- Vertical transmission has also been reported in rabbits

- Grossly
  - commonly kidneys demonstrate the lesions
  - There are multiple white, pinpoint areas or gray, intended areas on the renal cortical surface

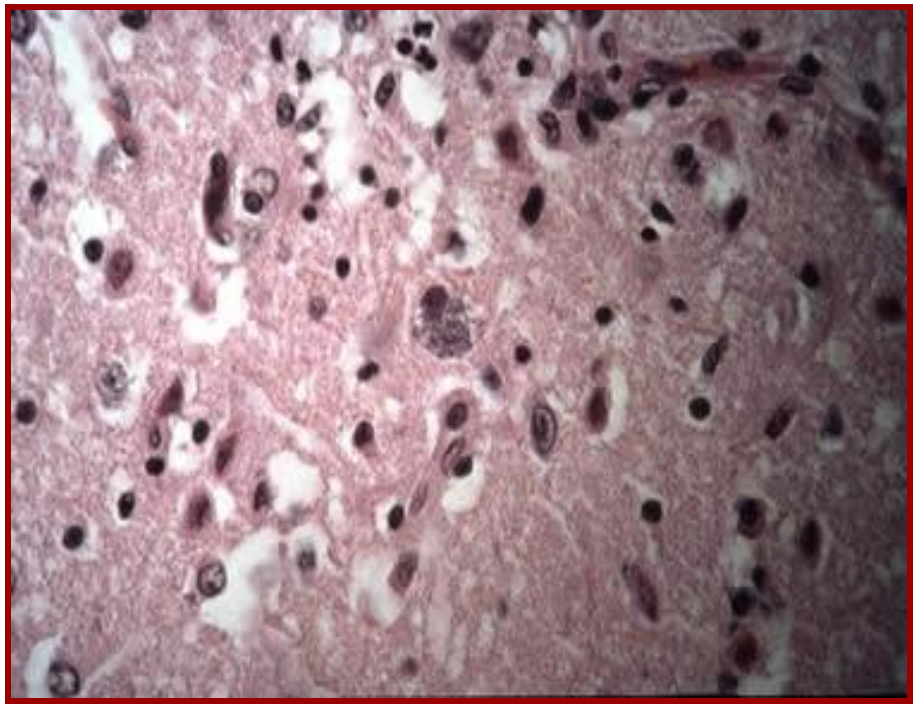
- Histological examination

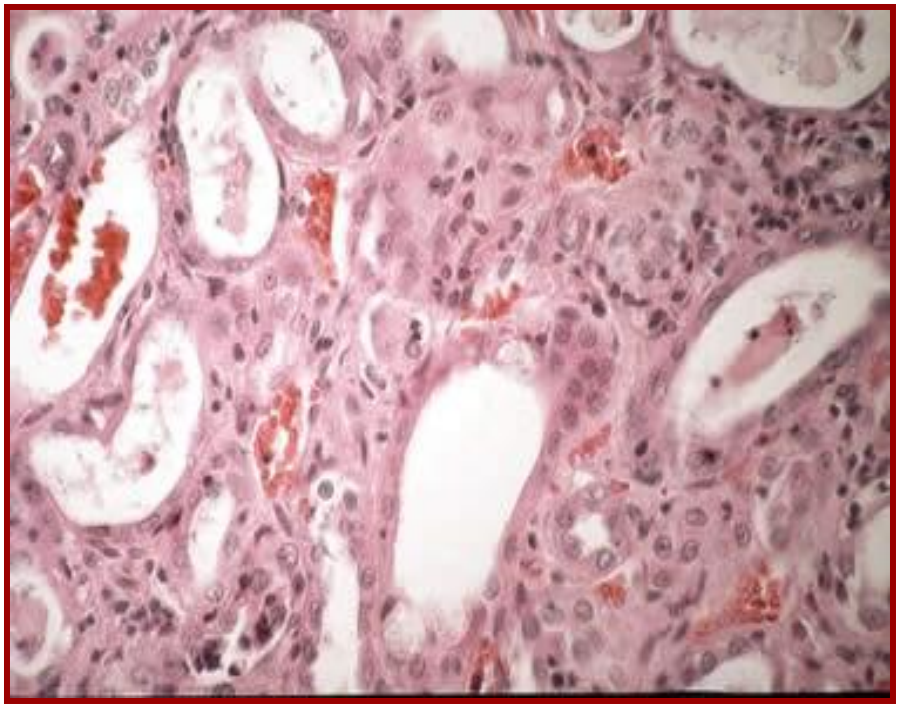
  - Microscopically it is characterised by granulomatous inflammation
  - Interstitial infiltration of lymphocytes and plasma cells along with tubular degeneration
  - Observation of organisms using Giemsa stain, Grams stain or Goodpasture carbol fuchsin stain in tissue



Kidney (Nosematosis)

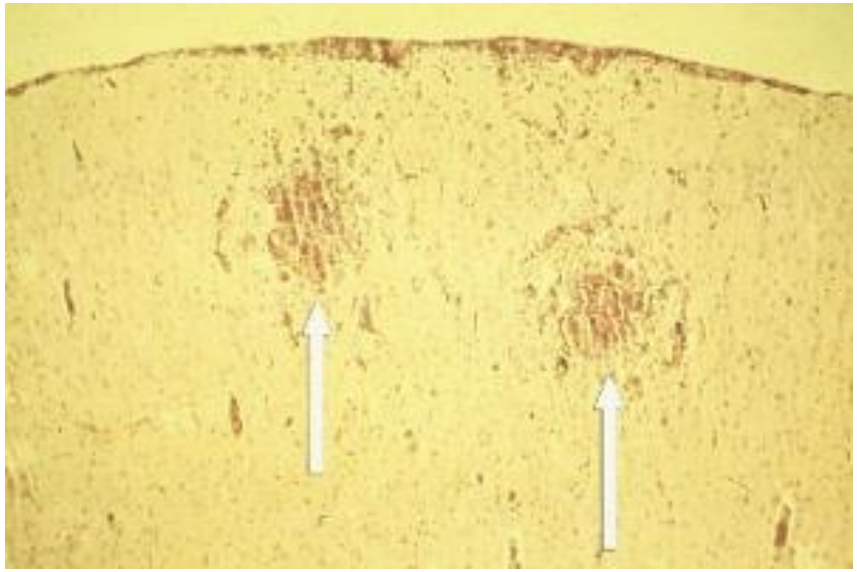
chronic interstitial nephritis



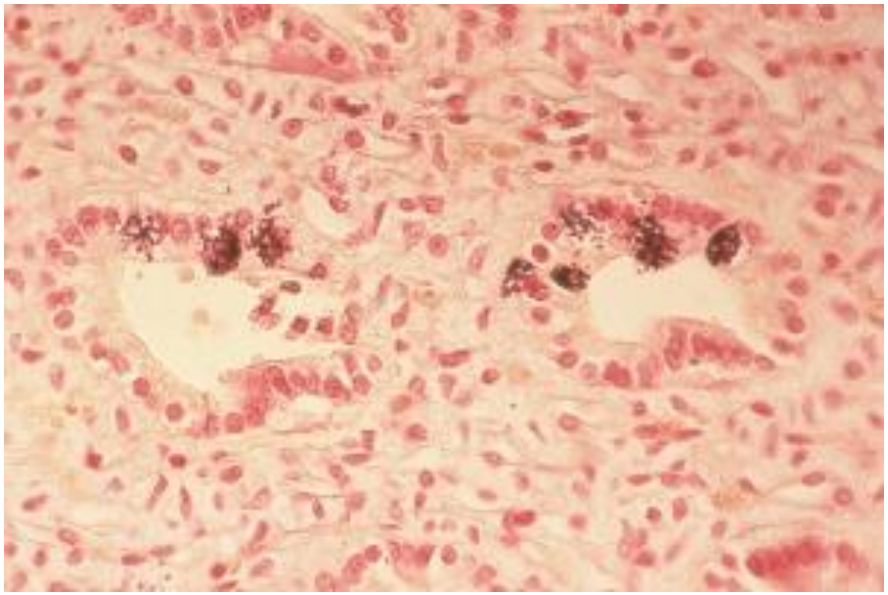


Brain **Liver**

Intracellular cysts of Encephalitozoon cuniculi, H&E, 40X.



Focal granulomas – brain



Organisms in pseudocysts
Renal tubule
epithelium

- Definitive diagnosis
  - Serological tests
    - Indirect fluorescence antibody technique – usefull in screening of large colonies of rabbits
    - Complement fixation test
    - Immunoperoxidase test
    - Enzyme immunoassay
- Research complications:
  - Granulomatous reactions would obviously complicate renal physiology and neurologic research

ARTHROPOD & HELMINTH DISEASES

1. EAR MANGE / PSOROPTIC MANGE / EAR CANKER / OTOCARIASIS

- Etiology:
  - Non-barrowing mite - Psoroptes cuniculi (Rabbit ear mite)
- Clinical signs:
  - Lesions primarily occurs on the inner surface of the external ears
  - Lesions are pruritic and results in scratching, head shaking, pain and even self mutilation
  - A tan, crusty exudate accumulates in the ears over the lesions
  - Exudate becomes quite extensive and thick
  - The skin under the crust is moist and reddened
  - The ear may become malodorous

Psorotes cuniculi (Ear lesions)

- Pathogenesis:
  - All the stages of mite (egg, larva, protonymph and adult) occur on the host
  - During early stage mites feed on sloughed skin cells and lipids. As local inflammation increases, they ingest serum, Hb and RBCs.
- Pathological findings:
  - Histologically, chronic inflammation, hypertrophy of the Malpighian layer, parakeratosis and epithelial sloughing
- Diagnosis:
  - Exmination of material scrapped from the inner surface of the ear using dissecting microscope
  - Mites are oval shaped with well developed legs that project beyond the body margin

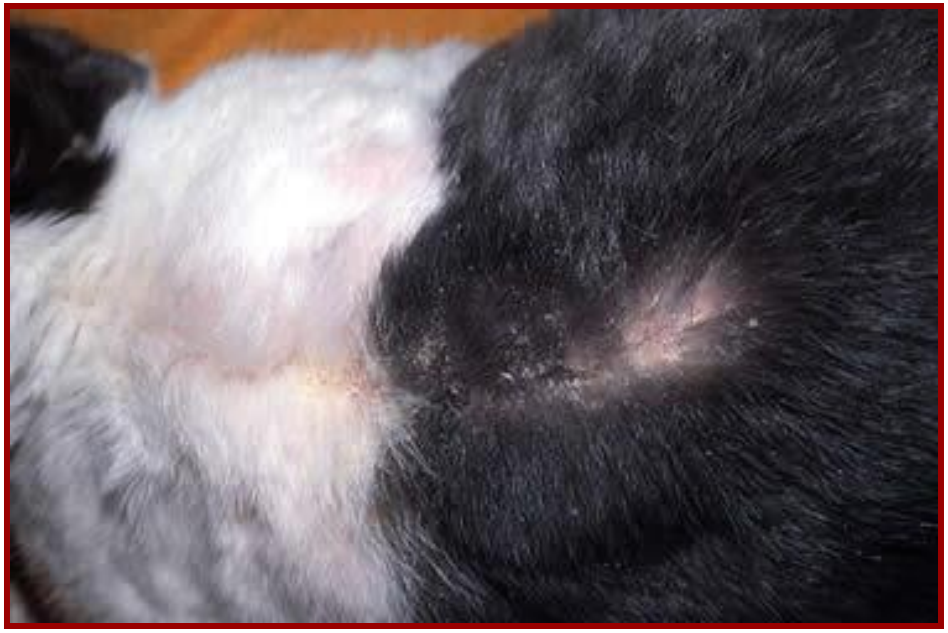
2. SKIN MITES OF RABBITS

- Etiology:
  - Non-barrowing mite –
  - Cheyletiella parasitovorax, C. takahasii, C. ochotonae, C. johnsoni
  - It has zoonotic importance

Pathogenesis:

- Transmission is probably by direct contact
- All the stages ie egg, larvae, pupa and adult occur on the host
- Mites remain in association with the keratin layer of the skin and feed on tissue fluid

- Clinical signs:
  - Mainly affect area over the scapulae
  - Leads to mild hair loss and also have gray-white scale
  - Affected animals do not scratch and no evidence of pruritis



heavy skin flaking and scaling

- Lesions:
  - Skin lesions are mild or nonexistent
  - Lesions are characterized by mild dermatitis, hyperkeratosis and inflammatory cell infiltrate
- Diagnosis:
  - Examination of skin scrapping and brushing fur from affected areas under dissecting microscope
  - Samples may be cleaned with 5-10% potassium hydroxide
  - The mites have a large, distinctive curved claw on the palpi

3. SARCOPTIC MANGE

- Etiology:
  - Barrowing mite – Sarcoptes scabiei, also referred as itch or scab mites
  - It has zoonotic importance
- Clinical signs:
  - Lesions consists of intense pruritis, hair loss and abrasions as a result of scratching
  - Serous encrustations on the skin and secondary bacterial infection are common
  - Lesions are generally seen on the head
  - Affected rabbits also exhibits anemia and leukopenia

- Pathogenesis:
  - All the stages of sarcoptic mites occur on the animals
  - The female burrow into the skin to lay eggs
  - Young larvae can be found in the skin while older larvae, nymphs and male resides on the skin surface
  - Mites feed on lymph and epithelial cells
- Pathological findings:
  - Rabbits with severe infection reveals amyloidosis of the liver and glomerulus
- Diagnosis:
  - Examination of skin scrapping. Samples may be cleaned with 5-10% potassium hydroxide
  - The body of the mites is round and the legs are very short

4. PINWORM INFESTATION

- Etiology:
  - Oxyuris ambigua found in caecum and colon of rabbits
  - Also known as Passalurus ambiguus
- Clinical signs:
  - In heavy oxyurid burdens even clinical signs are not usually apparent except in one case unsatisfactory breeding performance and poor condition

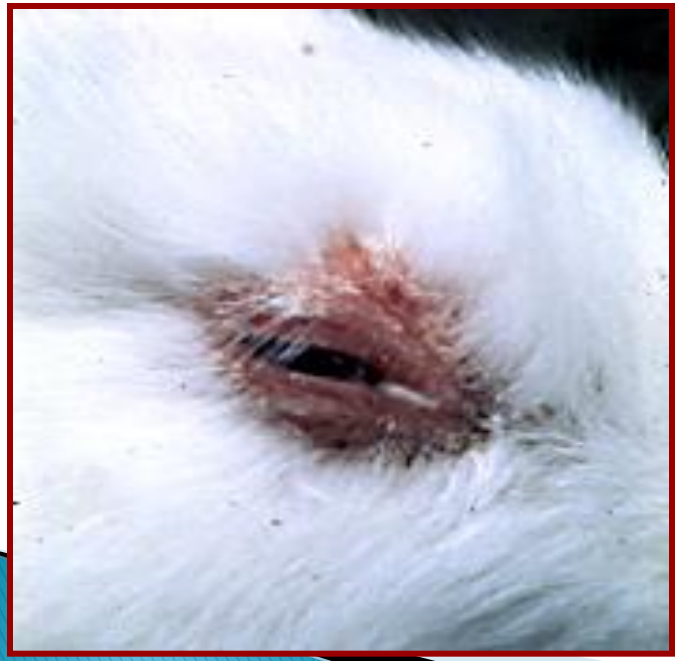
- Transmission:
  - Readily found in wild and domestic rabbits
  - Embryonated eggs are passed out in feces and are immediately infective
- Pathogenesis:
  - Mature worms are found in lumen of caecum or colon
  - After ingestion the eggs hatch in the small intestine and larvae molt
  - Development continues and maturation occurs in caecum
  - Prepatent period is between 56-64 days

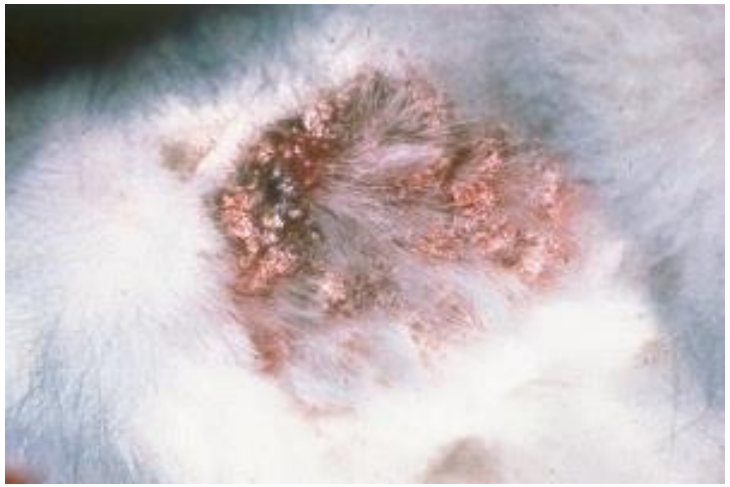
MYCOTIC DISEASES

1. SUPERFICIAL MYCOSES / RING WORM

- Etiology:
  - Trichophyton mentagrophytes – Most commonly seen
  - Microsporum canis – Occasionally seen
  - It is common in pet rabbits but seen infrequently in laboratory bred rabbits
  - This disease has zoonotic importance
  - Predisposing factors:
    - Poor nutrition
    - Environmental stressors
    - Overcrowding
    - Excessive heat or humidity
    - Extermes age
    - Pregnancy

- Transmission:
  - Through direct contact with affected animals
  - Contact with macroconidia and arthropores
  - Fomites can be significant source of infection perticularly objects like hairbrush or other equipments
  - Asymptomatic carriers





Dermatophytosis

- Clinical signs:
  - Patchy alopecia with crusting
  - Lesions are often erythematous
  - Disease may spread to paws, ear and other sites
  - Lesions are typically pruritic, circular and 1-2 cm in diameter with surrounding raised rim of acute inflammation and broken hairs
  - The lesions expand radially with central healing
  - Hyperkeratosis and acanthosis

- Pathological findings:
  - Hyperkeratosis and acanthosis
  - With acute and chronic inflammatory cells diffusely infiltrating the underlying dermis
  - Focal abscesses of the hair follicles within the perimeter of the lesion because of secondary bacterial infection
  - Special stain such as
    - Periodic acid–Schiff
    - Gridley fungus stain
    - Gomori methenamine–silver stain are used to visualize mycelia and artropores

- Diagnosis:
  - Differential diagnosis should be done to differentiate from acaritic mange, fur pulling, moist dermatitis, malnutrition, spirochetosis, seasonal molting, behavioral vice and bacterial dermatopathy
  - Skin scrapping examination after treating with 10% KOH and mineral oil
  - Scrapping should be collected from the periphery of the lesions
  - Microsporum fluoresce under Wood's lamp illumination

2. DEEP & SYSTEMIC MYCOSES

- Etiology:
  - Aspergillosis flavus or Aspergillosis fumigatus
  - Lesions contained hyphae surrounded by eosinophilic “astroids bodies”
  - Isolation of A. fumigatus from the reproductive trace of an adult female rabbit that aborted at an advanced stage of pregnancy and the associated placenta has also been reported.
  - Pneumocystis carinii is a microorganism present in the lungs of many mammal species.
  - It is a generally a harmless microorganism in immunocompetent individuals and has been identified in clinically normal rabbits.
  - Animals with a less then fully functional immune system are susceptible to more severe infection

- In rabbits respiratory disease accompanied by pulmonary lesions has been reported in young or debilitated animals.

- Saverely affected animals have histological lesions characterized by extensive interstitial pneumonia with infiltration lf mononuclear cells.

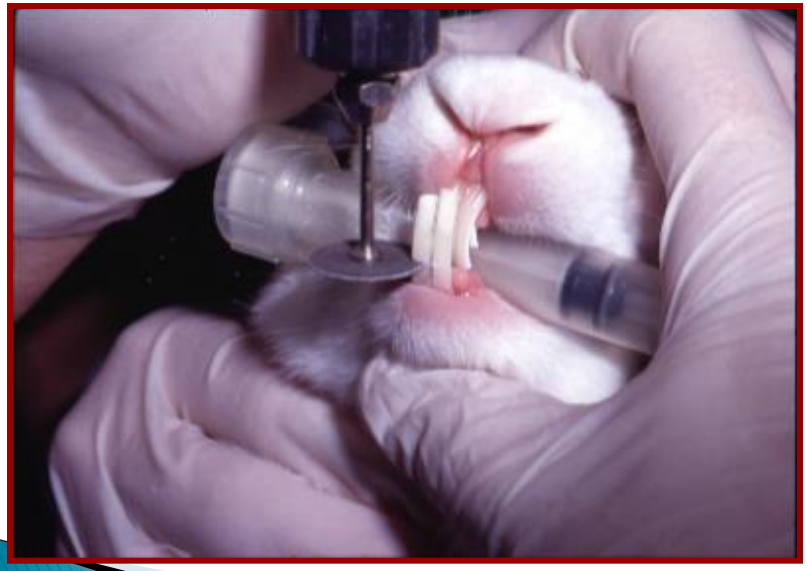
Miscellaneous Conditions

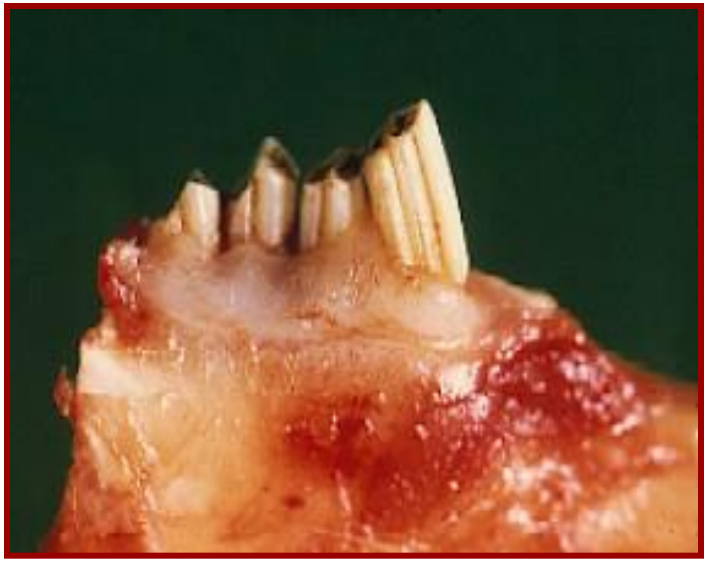
1. Cachexia and overgrown incisor

- Rabbit’s incisors, or front teeth, grow continuously throughout their life.
- Normally, chewing on their food and on wood blocks keeps them a normal length.
- Sometimes this is not enough and the incisors become overgrown.
- The rabbit will be unable to eat properly and unable to groom.
- Its coat will become ragged and you will notice excessive drooling.

2. Malocclusion

- There are both hereditary and environmental causes of malocclusion. Hereditary mandibular prognathism is apparent by 3 weeks of life and is due to an autosomal recessive trait with incomplete penetrance. Malocclusion of premolars and molars has been reported in older rabbits. As a rabbit's incisor teeth grow 4" per year, it is necessary to clip them (but not too close) every 6 to 8 weeks. Dog nail clippers (Rescoe or White styles), bone or wire cutters and a rotary tool with disc attachment (shown in photo) may be used to trim teeth. Care should be taken to prevent shattering the incisors.



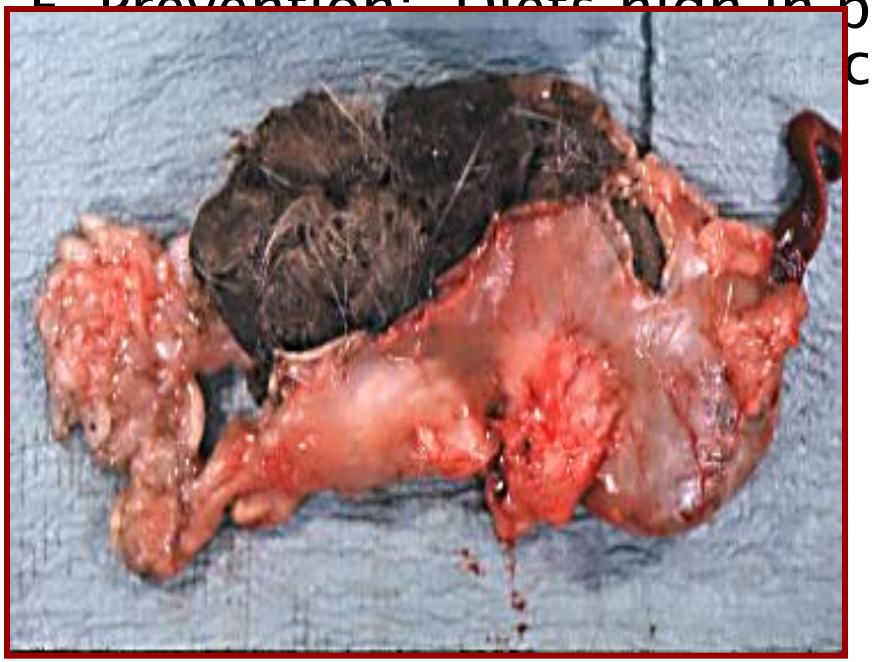


3. Hairballs (Trichobezoar)

- A. Etiology: Hairballs can form in the stomach as the rabbit grooms itself or a cagemate. Insufficient fiber in the diet can also lead to increased hair consumption. Incidence of hairball formation is high although abnormal digestive function pribably rarely occurs unless the hair ball is extensive.

- B. Clinical Signs: Generally, hairballs are not a problem and are found incidentally at necropsy. Occasionally, they will cause a partial or complete obstruction; these hairballs may be palpable, and the rabbit will stop eating and lose weight. The rabbit may be bright, alert and afebrile with a history of anorexia and lack of feces excretion of several days duration.

- C. Diagnosis: Palpation and contrast radiography may be used.
- E. Prevention: Diets high in plant fiber has dramtically cli




THANK YOU