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DEPARTMENT OF VETERINARY PATHOLOGY

An Overview

EQUINE INFECTIOUS ANEMIA

Introduction

- Equine infectious anemia (EIA) is important viral disease of horse ,mules & donkey
  - Cause by EIAV of Lentivirus genus
    - Synonyms - Swamp fever,
      Mountain fever,
      Slow fever,and
      Coggins disease
- EIA has a worldwide distribution and has been around since the early 1800's.
  - OIE – List B disease

Cont…

- It is chronic persisting viral disease characterized by
  - Emaciation
  - Anemia
  - Intermittent fever ,
  - Generalized lymphoproliferative changes
  - Oedema and
  - Thrombocytopenia

(Leroux *et al*., 2004)

- Once the equine infected ,they remain as carrier as long as animal live.

(Coggins, 1984)

History

- EIA is first described in France in 1843.

(Ligné, 1843)

- Subsequently reported in many part of world including Europe,Africa ,North America, Asia,Oceania and U.K

(Ishii and Ishitani, 1975)

- First disease of horses proven to be caused by a "filterable virus".

(Vallee and Carre, 1904)

- EIA is the first animal disease to be assigned a viral etiology, preceding by several years the major discovery of the first tumor virus by Rous
- EIA is the first retrovirus-induced disease proven to be transmitted by insects

(Stein *et al.*, 1942)

- EIAV is the first persistent virus for which "antigenic drift" was defined .

(Kono, 1972).

- EIA is the first retrovirus-induced disease for which a diagnostic test was approved (Coggins and Norcross ,1972).
- EIAV was the first virus shown to be related to the HIV through cross-reaction in tests of blood serum (Montagnier *et al*., 1984).
- Swamp fever term was first used by Torrance in 1903 in Canada
- In India – first case reported from Karnataka in 1987 .

(Uppal and Yadav ,1989)

Epidemiology

- Worldwide distribution
  found on all continents with the exception of Antarctica.

  (Paquette , 1985)
- High incidence in central America and northen country of south America

  (Burns *et al*,.1987)
- In USA and Canada the average infection rate is 3 –6 % of all horse

  (Perryman *et al*,.1980)

- Most prevalent in northern and central region of Europe

(Hanson *et al*,.1985)

- Mostly occur during summer and autumn.
- Marshy area are more prone to infection
- The prevalence of infection depends - Density of the horse population,
  - Insect vector population, and
  - Control measures in place

ETIOLOGY

- **Equine infectious anemia virus (EIAV)**

Family : Retroviridae

Genus : Lentivirus

Size : 80-100 nm

Envelop : Present

Capsid symmetry: Icosahedral

Genom : Diploid linear positive sense ssRNA having Reverse transcriptase (RNA dependant DNA polymerase

(Van regenmortet *et al*.,2000)

- EIAV is Macrophage tropic virus

(Oaks *et al.*,1998)

- EIAV replication only in tissue macrophage and does not occur in circulatory monocyte

(Sellon *et al.*, 19

- Highest amount of virus in liver, spleen, lymph node and bone marrow .Macrophage serve as an reservior for persistent viral infection

(O'Rourke, 1991)

- EIAV shares antigenic cross reactivity with human and feline immunodeficiency virus

(Radostits *et al.*, 20

- (Radostits et al., 2000)

)

- Antigenic drift in nature - Antigenic drift in the surface glycoprotein (gp45 & gp90) and emergence of novel antigenic strain

(Montelaro,1984)

- One major group specific antigen, p26 that is conserved & basis of the AGID & competative ELISA diagnostic test

(Coggins, 1972)

Transmission

Source - Clinically affected animals (potent sources)

- Normal “inapparent” carrier

Transmission occurs almost exclusively through the transfer of contaminated blood or blood products

(Hensen *et al.*,1996)

Insect vectors transmission

All large biting flies including

Horse Flies (Tabanus species)

Stable Flies (Stomoxys species)

Deer Flies (Chrysops species)

Mosquitoes and Midges have been implicated in transmission although this has not been proven

(Issel *et al.*, 1992)

Insect transmission of EIAV is dependent on

- Number and habits of the insects
- Density of horse population
- Number of times the insect bites the same and other horses
- The amount of blood transferred between horses
- The level of virus in the blood of the infected horse from which the initial blood meal was obtained

(knaus *et al.*, 1993)

Insect factor that influence the likelyihood of spread :

- Climate and Season: Biting flies prefer hot and humid conditions for feeding and breeding
- Attractiveness of Host: foals are less likely to be bitten than older horses
- Distance between horses: Tabanids prefer to complete an interrupted meal on the initial host or nearby host
- Host Housing: Horse flies are less likely to enter closed shelter

(Issel *et al.*, 1992)

Other means of transmission

- Intrauterine infection can occur lead to:

Abortion of the fetus

Foals born infected with EIA often die within 2 months

(Kemen, 1971)

- Iatrogenic transmission

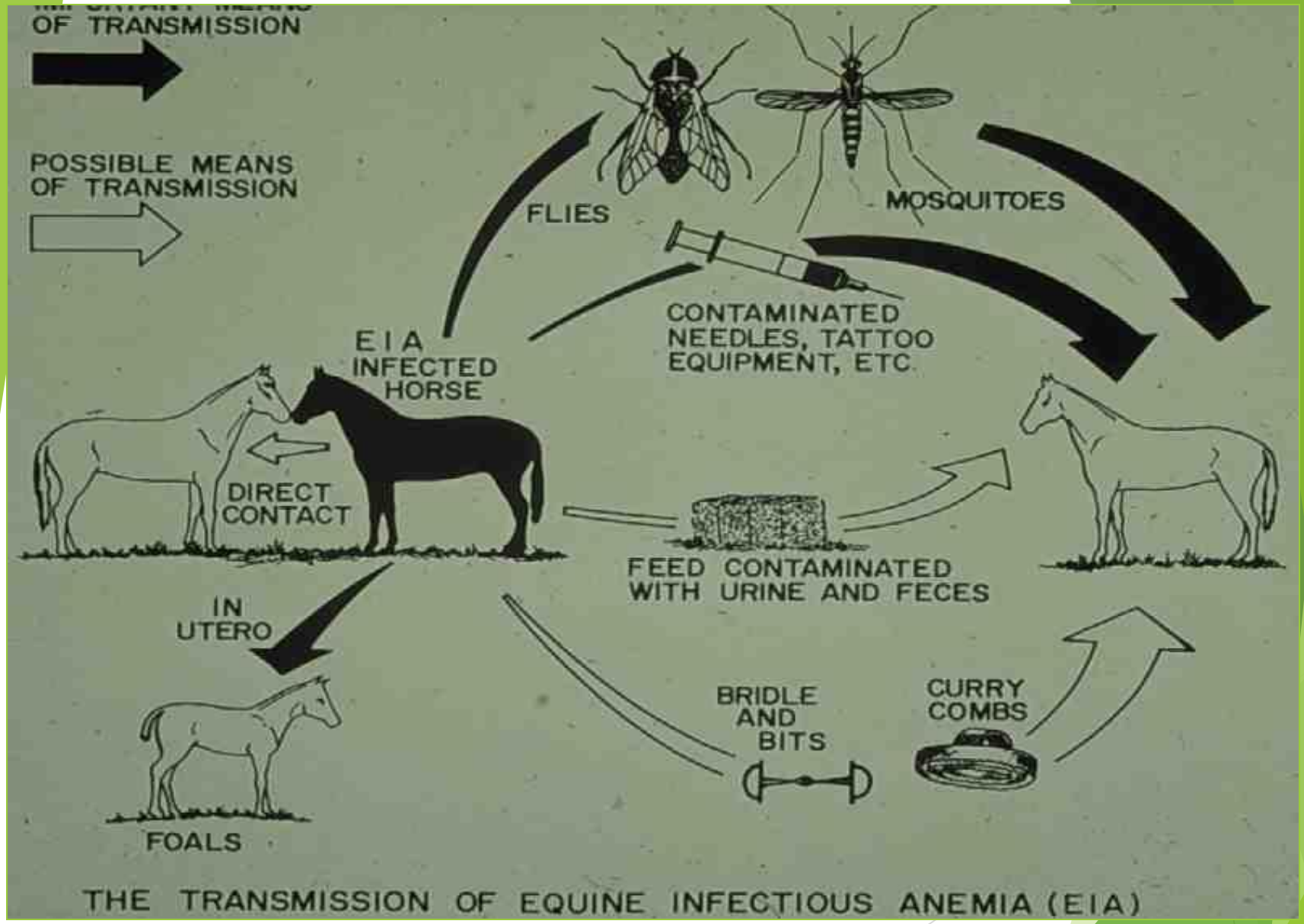
(Orrego,1982)

Possible means of transmission

- Contact with mucous, urine, semen, milk, and feces

of the acutely infected animal.

(Foil *et al*,.1987)


OF TRANSMISSION
POSSIBLE MEANS
OF TRANSMISSION
FLIES
MOSQUITOES
CONTAMINATED
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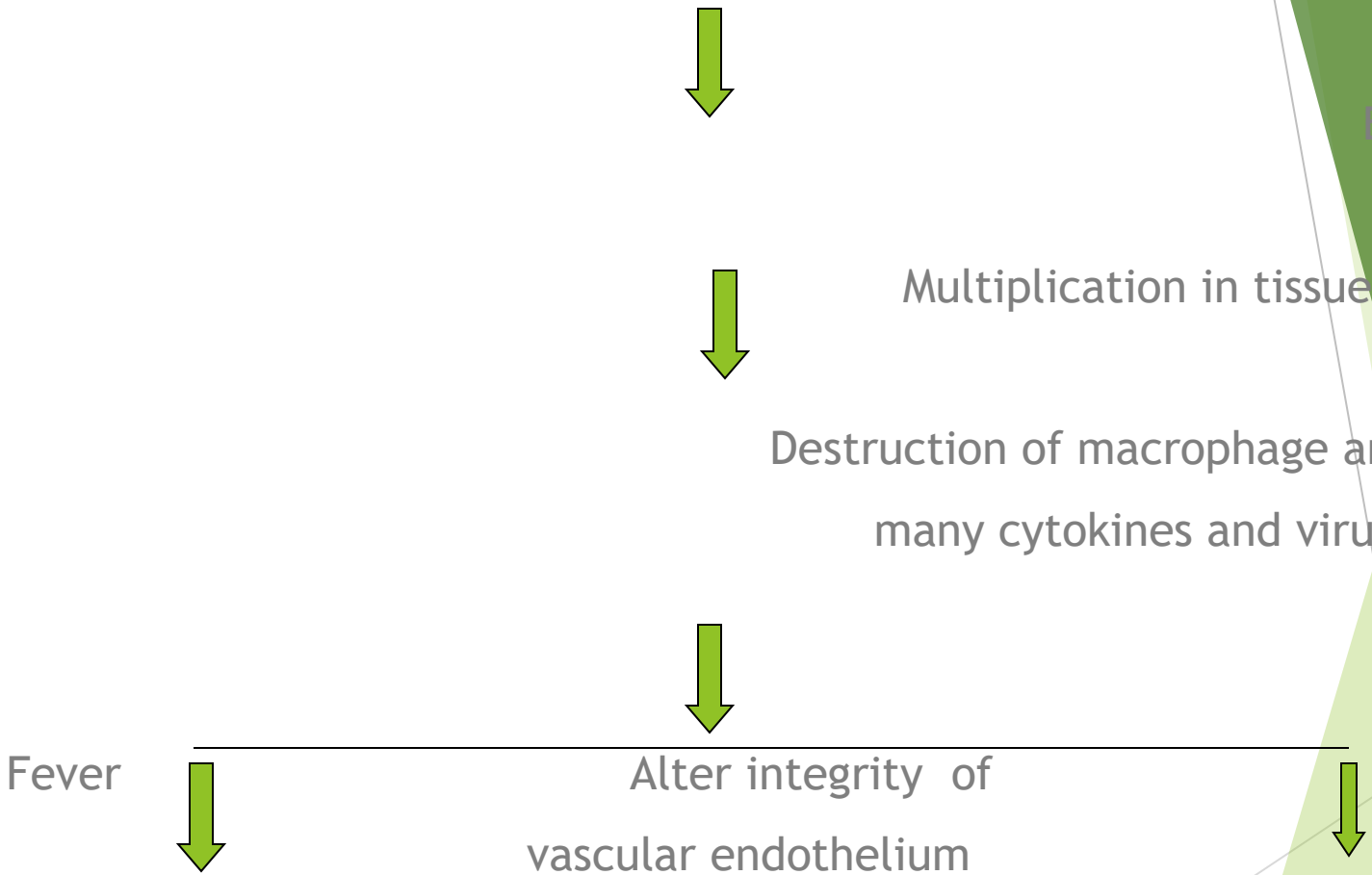
DIRECT
CONTACT
FEED CONTAMINATED
WITH URINE AND FECES
IN
UTERO
FOALS
BRIDLE
AND
BITS
CURRY
COMBS
THE TRANSMISSION OF EQUINE INFECTIOUS ANEMIA (EIA)

Pathogenesis



Production of neutralizing antibodies

This restrain the virus multiplication in macrophage

Appearance of new antigenic varient of the virus

New cycle of virus replication in macrophage & recurrence of clinical sign

Antigen – antibody complex formation
Deposition of this complex on RBC , kidney thrombocyte

Cont

Leads to anemia ,glomerulonephritis and thrombocytopenia

Subsequently achieve strong immune response against all antigenic Epitope that are common to all EIA virus strain & become asymptomatic

Clinical Presentation

Incubation period – 2-6 weeks

ACUTE FORM

- Rapid on set of high fever (up to 108f.) ,
- Depression
- Anorexia
- Ataxia
- Epistaxis
- Oedema of the legs, chest and ventral abdomen
- Extreme weakness
- Petechial hemorrhages on mucus membrane

Roughly 30% of animals become very sick and may die within 3-4 weeks

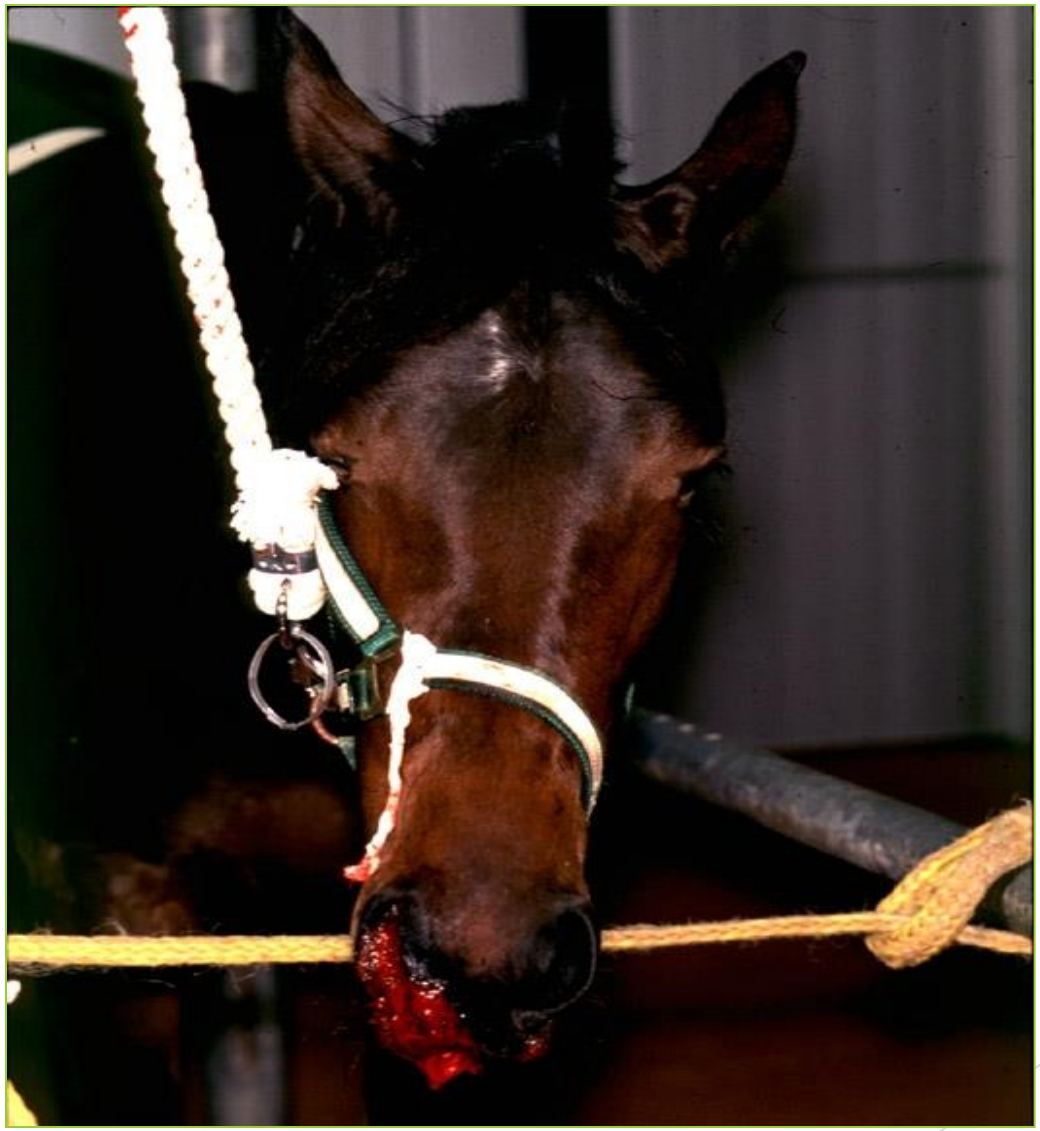
Animals that survive - recovery stage of 5 to 30 days

Cont…


ataxia
and
head pressing


icteric mucous membranes

Vulva - Pale mucus membrane & Petechial haemorrhage



Epistaxis

Chronic form

- Intermittent fever (105°F) (Oxer,1995)
- Anemia
- Edema of the legs, chest and ventral abdomen
- Petechial hemorrhages on mucus membrane
- Increased respiration rate
- Increased heart rate
- Progressive weakness & emaciation
- Pregnant mare may abort

(Kemen, 1971)

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- Signs may occur randomly or in a pattern recurring roughly every weeks or month
- Animal showing temporary recovery may appear normal for weeks or month & then again relapse

(Montelaro *et al*.,1993)

- These repeat episodes of sickness usually decrease in intensity and frequency over time

(Oaks *et al*,.1992)

- Most recurrent clinical episodes occur within one year and usually cease after one year

(Clabough ,1990)

Chronic Emaciation

Oedema at ventral wall of the abdomen

Inapparent form

- No any recurrent episodes of clinical signs
- Exist as carriers of the virus, allowing continued spread of the disease
- Stressful situations may result in viral replication and the onset of clinical signs in these animals, moving them into the chronic phase

(Payne,1998)

- Dexamethasone-induced immune suppression of inapparent carriers results in the reoccurrence of disease associated with markedly increased virus replication

(Craigo *et al*., 2002)

Haematological changes

Anaemia -

Decreased amount of red blood cells due to:

- Immune mediated hemolysis

  (Messer and Arnold, 1991)

- Decreased red blood cell survival time (38days)

  (McGuire, 1971).

- Decreased production by bone marrow

  (Swardson *et al*,.1992)

- Thrombocytopenia

(Clabough *et al.*,1991)

- Hypergammaglobulinemia

(McGuire,1971)

Biochemical changes –

- Increase bilirubin concentration and Decrease serum iron concentration

(Evans *et al.*,1994)

Pathology

- The major clinical sign & lesion of EIA are attributable to the host response to virus & not direct damage to tissue

(Sellon , 1993)

- Main gross finding is jaundice ,oedema & petechial haemorrhage on pleura and peritoneum

- The nature of lesions depends to a large extent on clinical type of disease & the duration of illness

Immunity

1. Humoral immunity
2. Cell- mediated immunity

1. Humoral immunity

- Antibody against p26 detectable after 45days
- Antibody against gp45 and gp90 detectable after 60 days.
- Antigenic drift of the gp45 and gp90 antigen, allows virus to escape immunesurveillance
- Antibodies against gp45 and gp90 antigen do not neutralize the virus

(Cook ,1998)

- Most virus in viremic horse remain as virus – antibody complex

(Macguire.,1969)

2. Cell- mediated immunity

Cytotoxic T lymphocytes are associated with control of EIAV.

(Kuroda *et al.*, 1999)

Diagnosis

On the basis of

- HISTORY
- Clinical sign
- Positive Serological tests
- Postmortem lesions.
- Histopathological lesions
- Haematological changes

Serological tests

- Due to the persistence of EIA virus in infected equine, detection of serum antibody to EIA virus confirms the diagnosis of EIA virus infection

(1) A Coggins test (Agar gel immunodiffusion)

(Coggins *et al*.,1972)

(2) Enzyme-linked immunosorbent assay

(Suzuki et al.,1982)

Differential Diagnoses

Subacute and acute stages:

- Equine viral arteritis
- Equine erhlichiosis
- Purpura hemorrhagica
- Autoimmune hemolytic anemia
- Babesiosis
- Leptospirosis
- Parasitism

Control

- No specific treatment & vaccine available

(Kumar and Kumar ,2005).

- Control of EIA by -
  - Identification & eradication
  - Life –long quarantine of infected animal
  - Quarantine and testing of new stock
  - Compulsory testing of imported horse
  - Continued surveillance and monitoring of the disease by Coggins test.
  - Control of biting insects
  - Iatrogenic transmission is avoided by careful hygiene