
بسم الله الرحمن الرحيم

Pathology and pathogenesis of some important viral diseases of equine

I- African Horse sickness virus

African Horse sickness virus

- It is a highly **fatal infectious** disease caused by **insect borne virus** characterized by hydrothorax and pulmonary oedema, hydropericardium and cardiac degeneration with ascites.
- The virus of African horse sickness is **Endotheliotropic**, RNA-containing belong to genus – **Orbivirus**

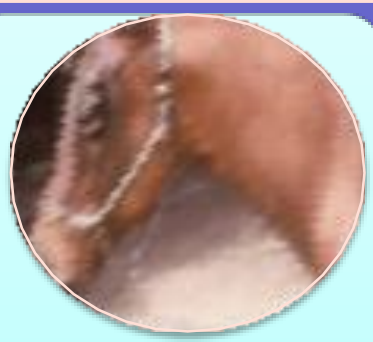
African Horse sickness virus

- **Susceptible hosts:**
  - Horse while other (mules, donkeys) are more resistant
- **Transmission:**
  - **Blood sucking insects (mosquitoes, culicoides(**
  - The second important factor in transmission of the disease is the reservoir animals **(dog may act as silent reservoir.(**

Clinical signs:

Pulmonary (acute) {[Dunkop

Pulmonary oedema, dyspnea, cough, and appearance of frothy fluid from the nose



Cardiac (subacute) { [Dikkop

bulging of the supraorbital fossa Oedema of eyelids, lips, cheeks, and tongue

Mixed

characterized by mixed symptoms of pulmonary and cardiac forms

Febrile

Fever for more days - 1-2 than Anorexia, dyspnea - Conjunctivitis -

This is the mildest form and is frequently .subclinical

Pathogensis

Inoculation of virus

Initial multiplication in regional LN

Primary viremia – disseminate to endothelial cells of target organs – endothelial cell damages

Effusion in body cavities, serosal hemorrhages

Secondary viremia

Foals develop – peracute pulmonary form

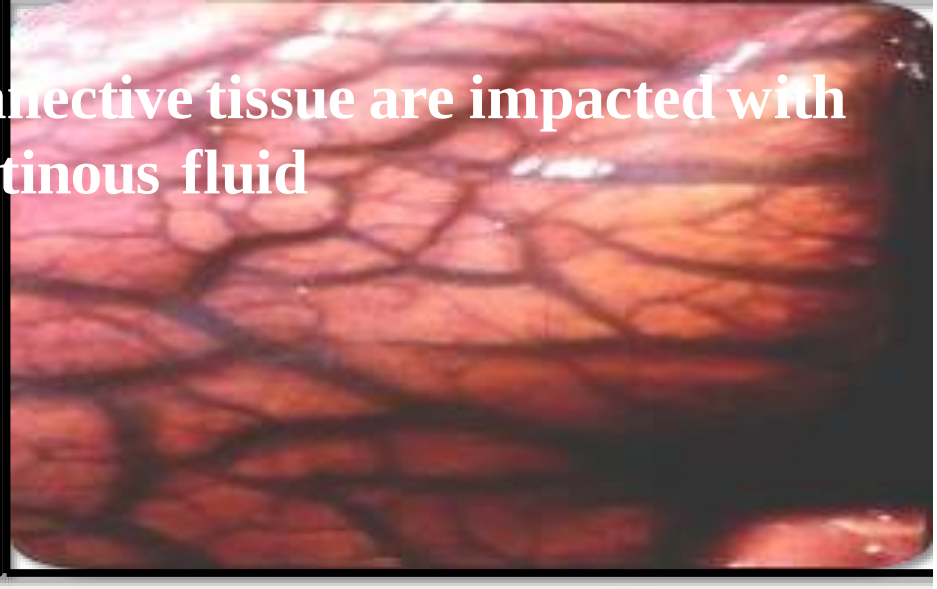
Cardiac form (serotype9)- degeneration and necrosis of myocardium, hydropericardium

Gross lesions

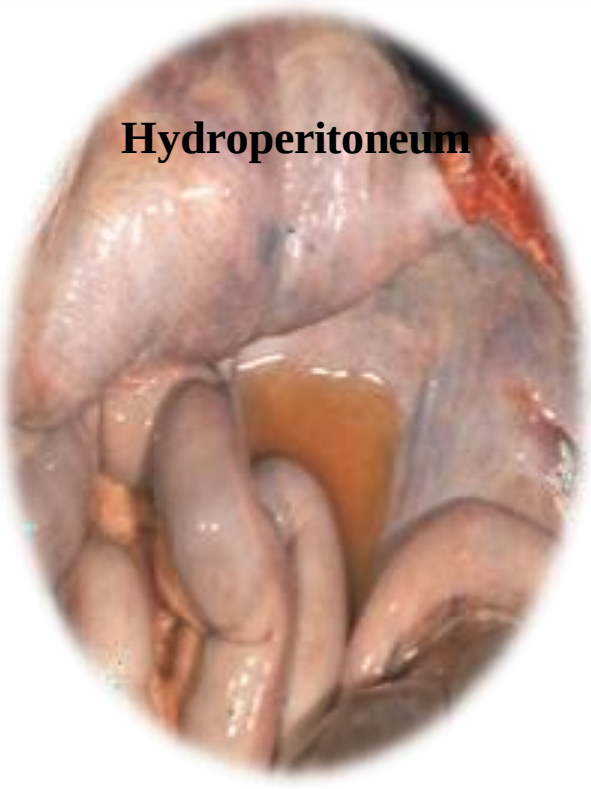
:A- Pulmonary form

- **Oedema** of the lung, **hydrothorax** and **hydroperitoneum.**
- The **interlobular** and **subpleural** connective tissue are impacted with yellow gelatinous fluid
- **On cut section,** the lung oozes a clear fluid and froth from bronchi.
- The draining **lymph nodes** appear soft and swollen.
- Colonic serosa, petechial and ecchymotic hemorrhages.

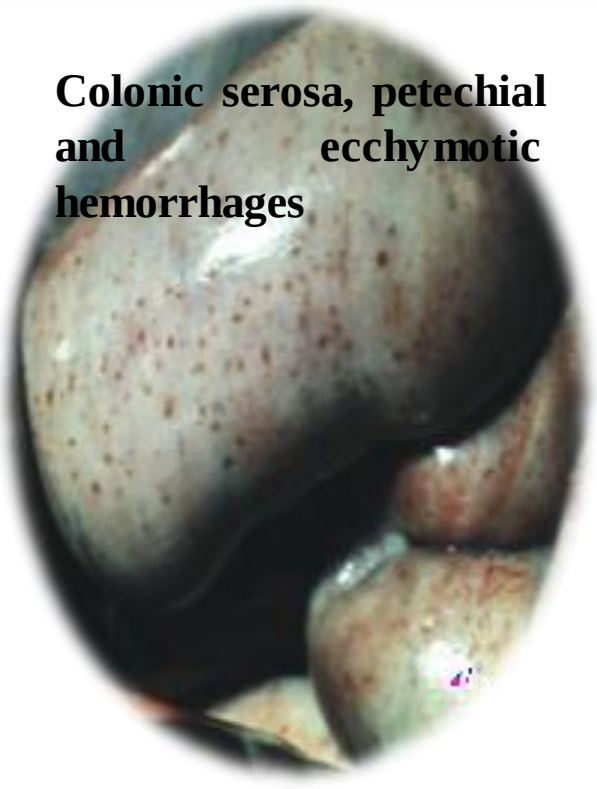
Interlobular and subpleural connective tissue are impacted with yellow gelatinous fluid



Froth from bronchi



Hydroperitoneum

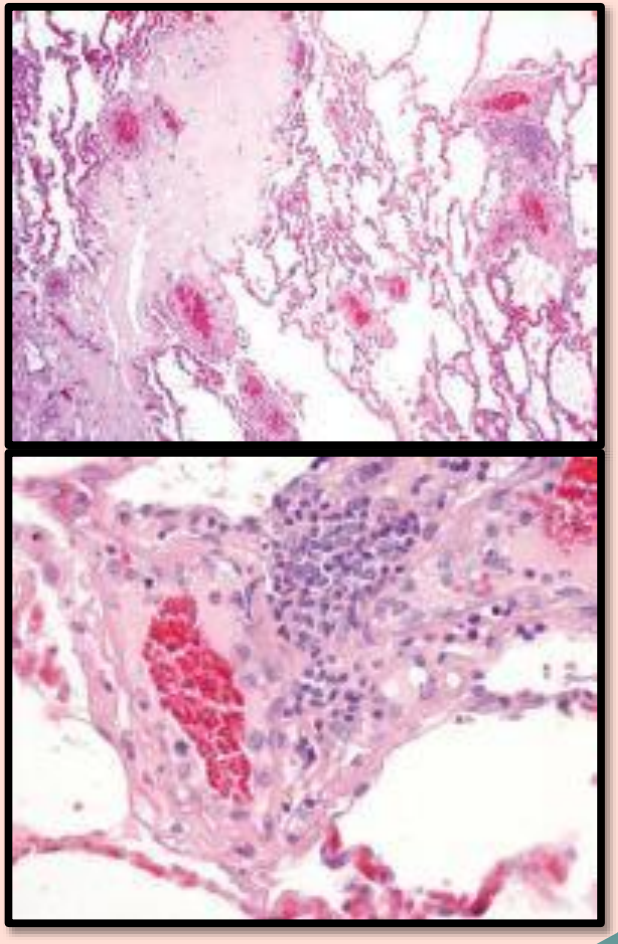


Colonic serosa, petechial and ecchymotic hemorrhages

Histopathology

:Lung *

- **Alveoli** contained moderate to abundant amounts of **fibrin** and **edematous fluid** mixed with low numbers of macrophages, lymphocytes and small foci of hemorrhage.
- **Interlobular septa**, perivascular and peribronchial **edema**, **fibrin**, few lymphocytes, plasma cells .and macrophages
- **Endothelial cells** within venules .are **swollen**

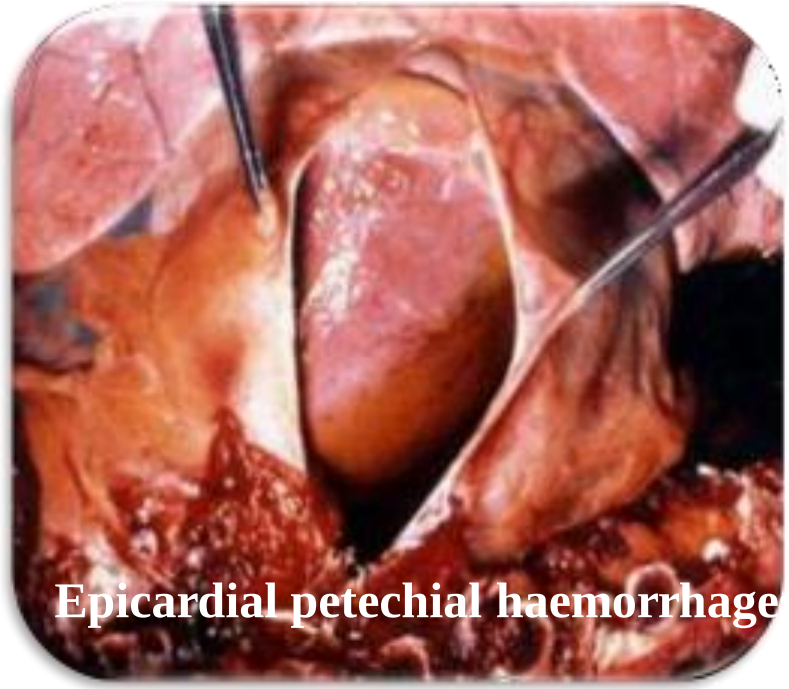


Gross lesions

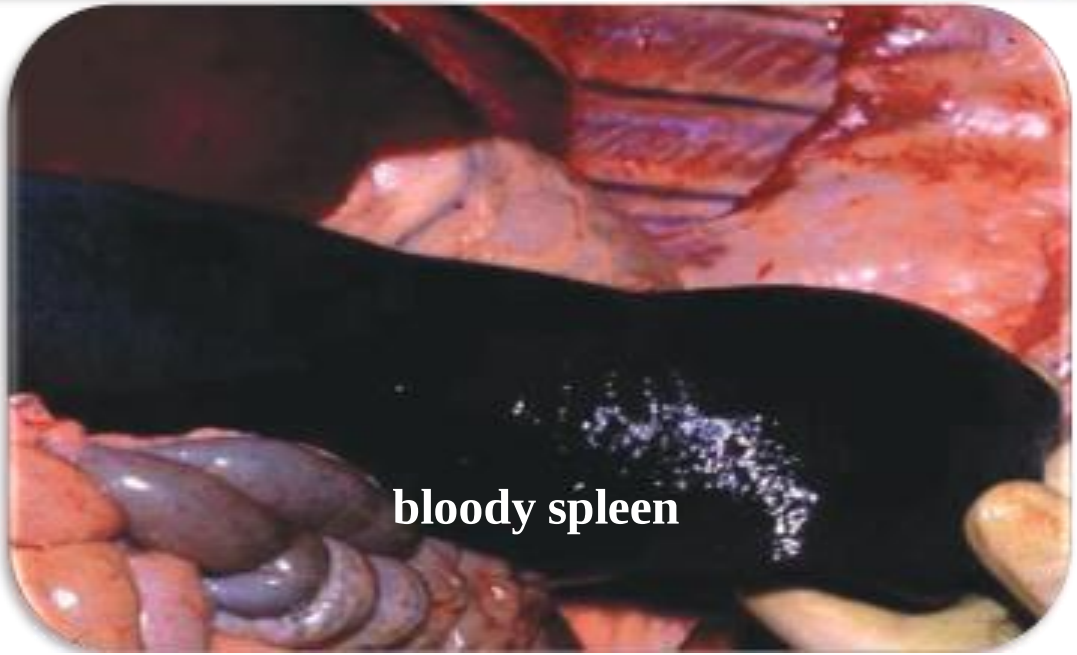
:B- Cardiac form

- Fibrinous effusion in the pericardial cavity.
- Subendocardial and epicardial petechial haemorrhages
- Haemorrhage in the myocardium
- Extensive subcutaneous oedema
- Supraorbital fossa Oedema
- The spleen, liver, and kidneys are congested.
- Splenomegaly, bloody spleen. The spleen is congested with blood and friable as a result of vascular damage caused by the virus.

Subendocardial haemorrhages



Epicardial petechial haemorrhage



bloody spleen

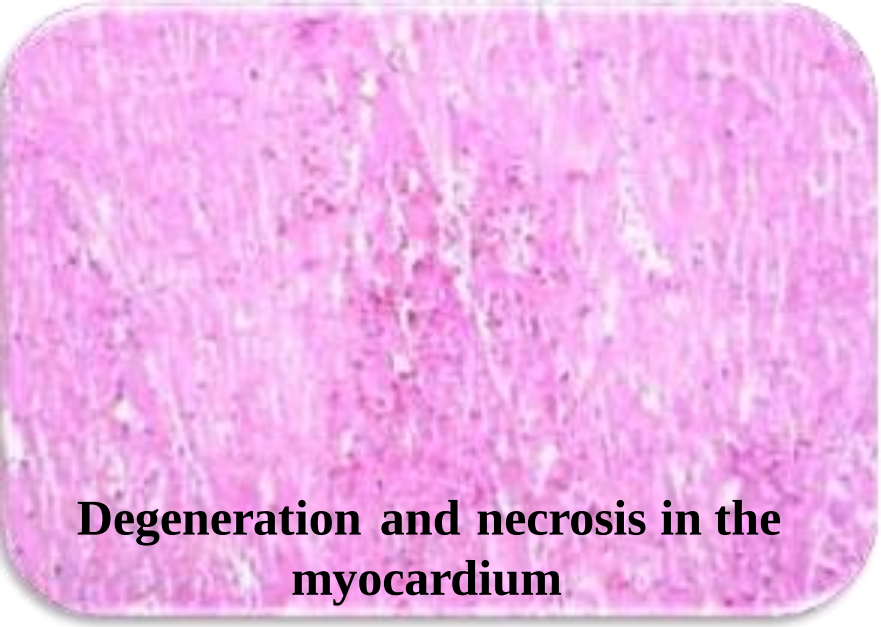
Histopathology

Heart:

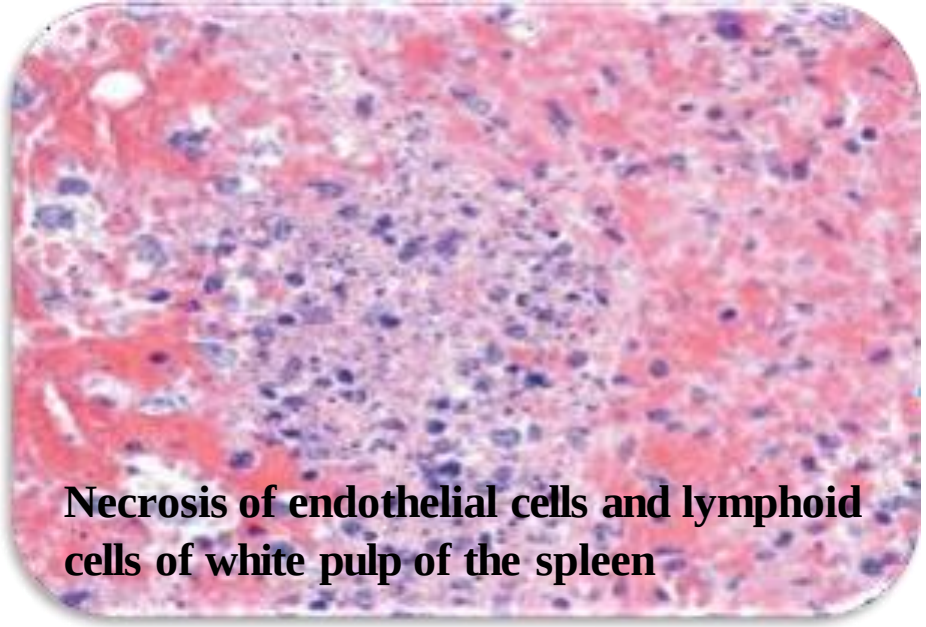
- Focal degeneration and even necrosis in the myocardium.
- Spleen and lymph nodes:

Depletion of lymphocytes necrosis of endothelial cells and lymphoid cells of white pulp of the spleen

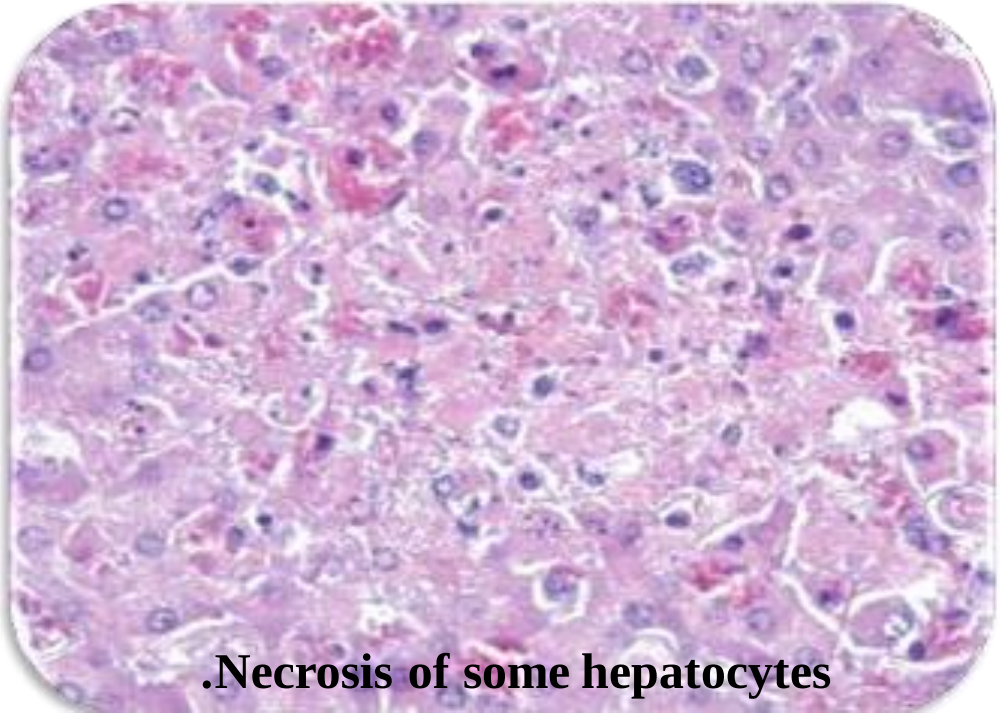
- Endothelial cells lining sinusoids of the liver are necrotic. Also note the necrosis of some hepatocytes.



Degeneration and necrosis in the myocardium



Necrosis of endothelial cells and lymphoid cells of white pulp of the spleen



.Necrosis of some hepatocytes

II- Equine Arteritis virus

Equine Arteritis virus

- It is an infectious disease caused by Equine arteritis virus belong to genus **Arterivirus, family Togaviridae**
- **Characterized clinically** by depression, edema of limbs, red conjunctiva, enteritis, pneumonic complications and abortions
- **Principal lesion:** degenerative of endothelium and inflammatory changes of tunica media of small arteries

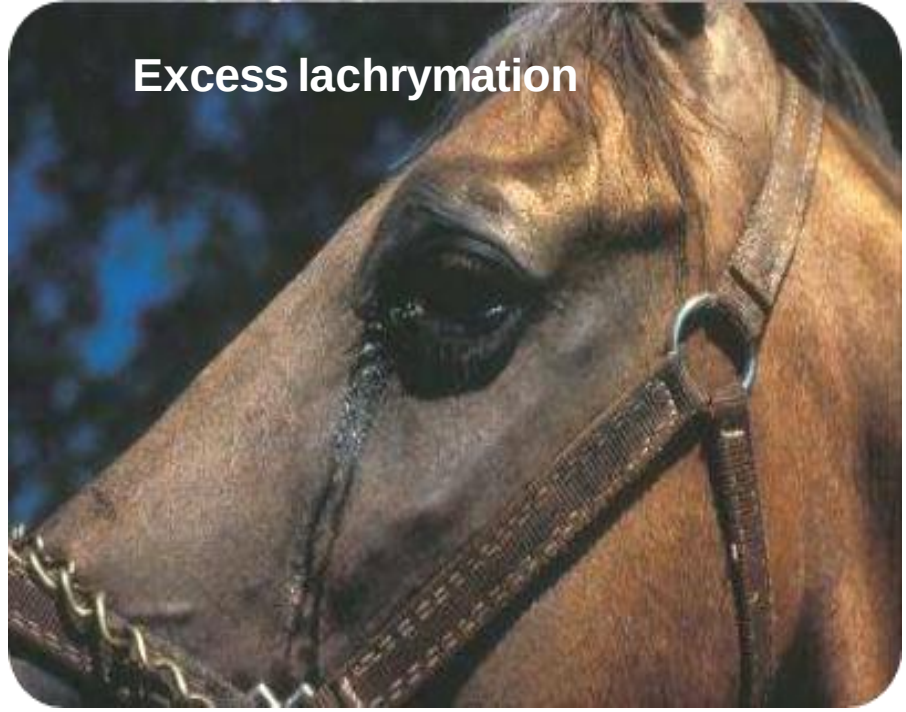
Transmission of EAV

- **Horizontal respiratory transmission** of EAV occurs after inhalation of infected respiratory tract secretions, urine and other body secretions from acutely infected horses and aborted fetuses and their membranes.
- **Venereal transmission of EAV** contained in the semen of stallions , vaginal and uterine secretions

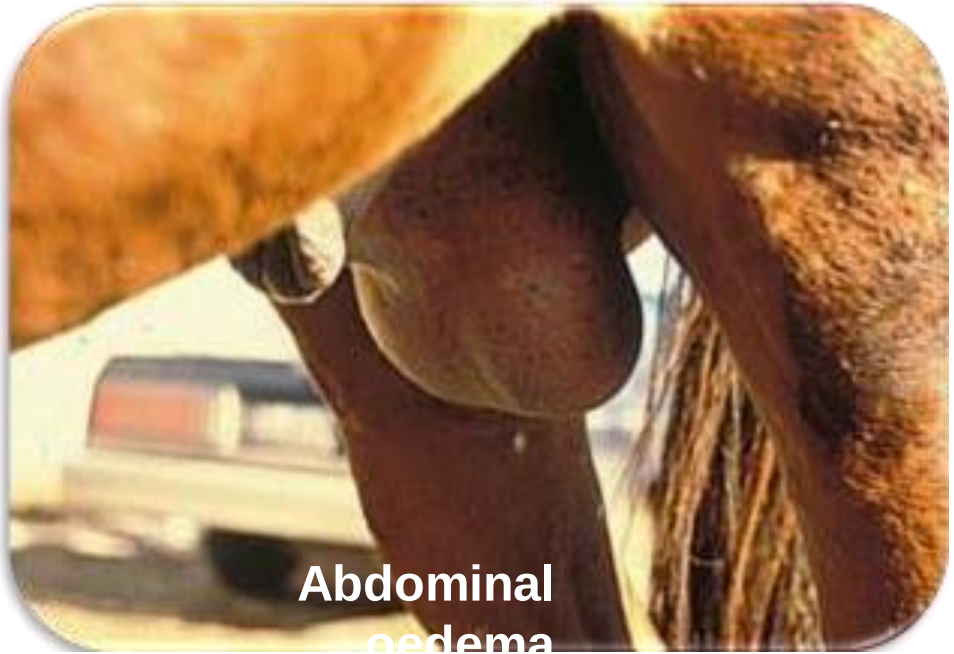
Clinical signs

- **Depression**
- **Oedematous swelling of the limbs**
- **Pink or red discoloration of the conjunctiva**
- **Abortion**
- **Haemorrhagic enteritis**
- **Pneumonia**
- **Urticaria**

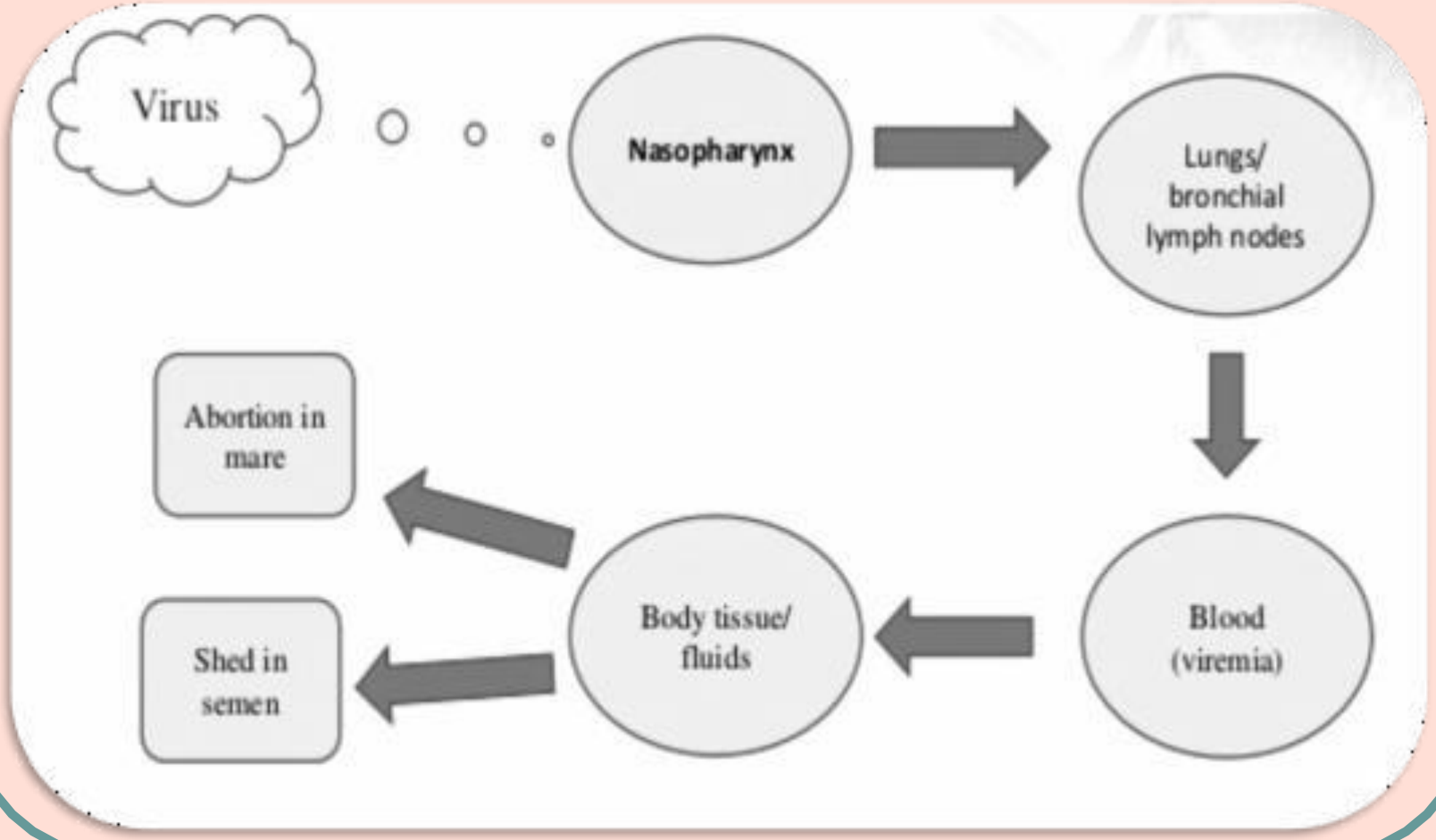

Ocular edema and conjunctivitis


Excess lachrymation


Urticaria type skin reaction -
due to lesions in blood


Abdominal

Pathogenesis



Pathogenesis

Infection by the respiratory route

Virus is disseminated via alveolar macrophages to regional lymph nodes.

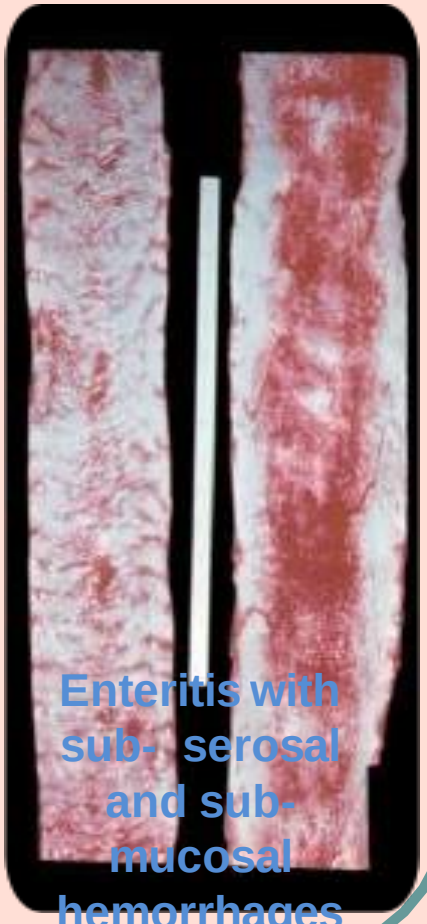
Viral replication occurs in vascular endothelial cells, which stimulates the influx of neutrophils into the internal elastic lamina

Fibrinoid necrosis of arterial blood vessels

Abortion appears to result from a diffuse vacuolation of endometrial BVS endothelium and a necrotizing vasculitis

Gross lesions

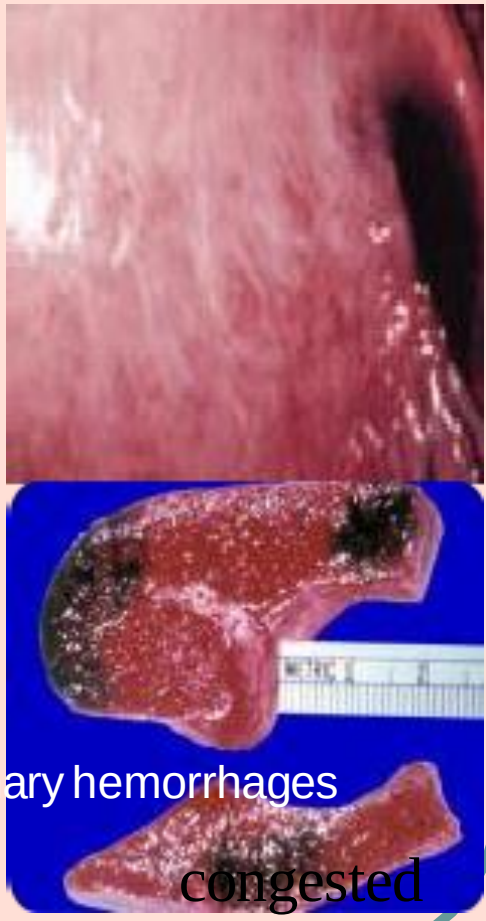
- **Edema**, **congestion**, **and** **hemorrhage** especially in the subcutis of the limbs and abdomen are the most frequent gross lesions.
- The **body cavities** may contain moderate to abundant amounts of **yellowish clear exudate.**
- Congestion and edema, hemorrhages can be observed along the course of the **colonic and cecal vessels**.
- Enlargement of LN



Enteritis with sub- serosal and sub-mucosal hemorrhages

Gross lesions

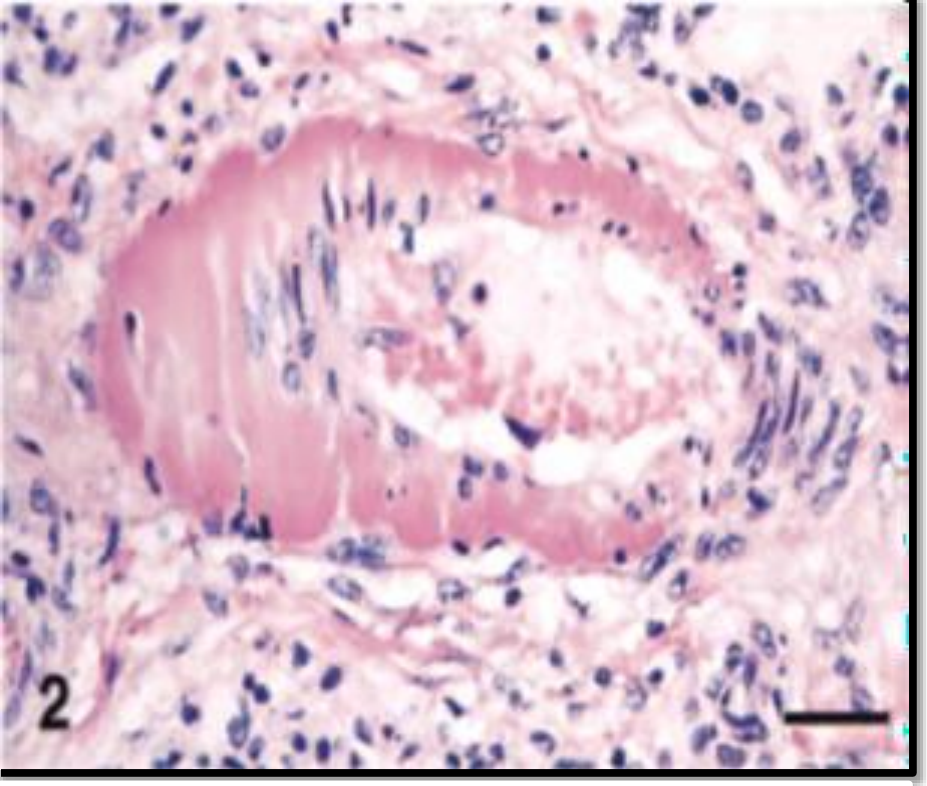
- **Lungs**, especially those of infected neonates are wet and increased in weight, with a prominent lobular pattern.
- The **trachea** may contain froth.
- Lungs showed multifocal or diffusely reddish areas because of congestion and hemorrhages.
- Hydrothorax, petichae in pleura، pericardium and lungs
- The **uterine endometrial** surface of aborted mares swollen and diffusely congested



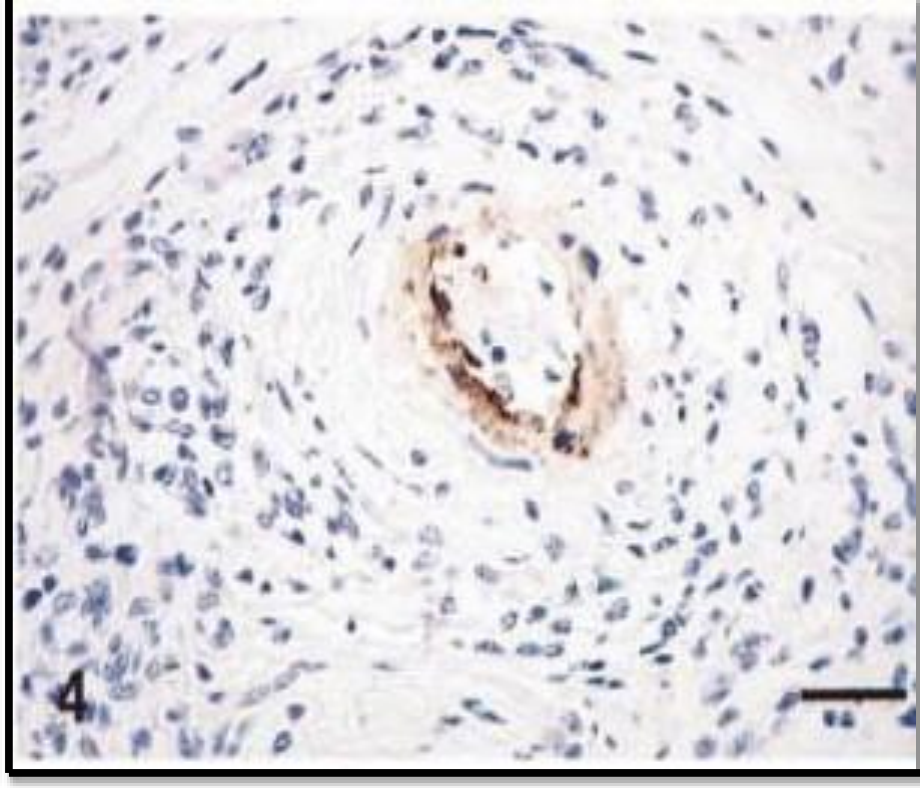
Pulmonary hemorrhages

Microscopic lesions

- **Blood vessels.**
- **Mild lesions** include vascular and perivascular oedema, lymphocytic infiltrate and endothelial cell hypertrophy.
- **Severe changes** include vasculitis with fibrinoid necrosis of the tunica media, abundant perivascular lymphocytic and lesser granulocytic infiltration
- Formation of large fibrinocellular thrombi.
- Within vessels, **EAV Ag** localizes in **endothelium** and **medial myocytes**.



Fibrinoid necrosis of the tunica media and perivascular edema with lymphocytic infiltrate.



EAV antigen is diffusing from the endothelium to the tunica media. (Immunohistochemistry(

Microscopic lesions

Lungs.

- Lungs showed mild to severe **interstitial pneumonia** characterized by alveolar infiltration with macrophages and lesser numbers of neutrophils, hyaline membrane formation, and fluid-filled alveoli.
- **Hypertrophy** and **hyperplasia** of alveolar pneumocytes
- Pulmonary arteritis and phlebitis.
- **EAV Ag** localizes within the cytoplasm of **pneumocytes** and **alveolar macrophages**.

Microscopic lesions

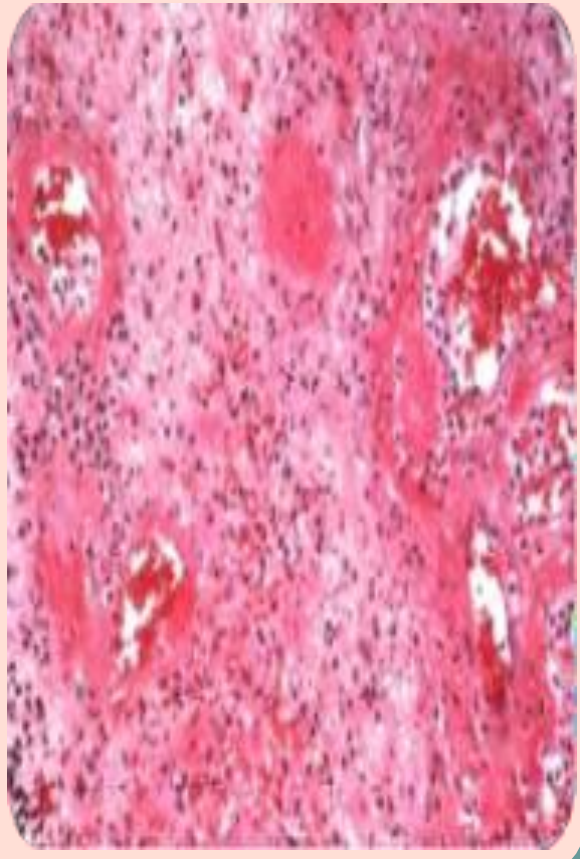
Lymphoid tissue.

- Lymphoid follicle necrosis, edema, and slight hemorrhage
- Lymph node sinuses contained highly pleomorphic and cells histiocytic lymphocytes.
- EAV Ag is contained within stromal dendrite like cells and within the macrophages of the lymph node sinuses and spleen.

Microscopic lesions

Female reproductive tract and fetus.

- **Fetus**
  - Mild interstitial pneumonia.
  - Vasculitis involving the allanto- chorion, brain, liver, spleen, and lung. EAVAg is inconsistently detectable within tissues of aborted fetuses.
- **Mares**
  - Uterine epithelial cells swollen
  - Uterine propria submucosa oedematous with infiltration of neutrophils and macrophages.
  - Necrotizing myometritis



Microscopic lesions

Male reproductive tract.

- Necrotizing vasculitis involving testes, epididymides, vasa deferentia, ampullae, prostate glands, and bulbourethral glands.

- This vasculitis was characterized by severe fibrinoid necrosis of small muscular arteries with edema and hemorrhage.

III- Equine infectious anemia

Equine infectious anemia

- Equine infectious anemia (EIA) is a disease, caused by a virus genus ***Lentivirus*** Family ***Retroviridae*** ***characterized clinically*** *by* anemia, intermittent fever, and severe weight loss.
- **Susceptible animals:**

Horses, ponies, mules, zebras and donkeys

- **Mode of transmition:**

➢ Virus Transmitted mechanically by horseflies, stable flies or by infected blood

➢ Vertical transmission – in utero/ colustrum feeding

- **N.B** Once an animal is infected with the virus, it is infected for life and animals may become asymptomatic carriers

Clinical signs

- **Acute phase:**
- fever, anorexia, extreme weakness, anemia,
- thrombocytopenia,
- **Chronic phase:**
- No or minimal anemia and thrombocytopenia

Pathogenesis

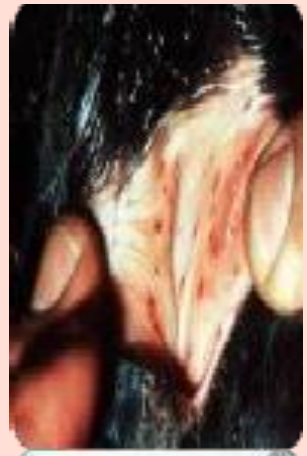
- **Virus inoculated** by fly or mosquito bites or needles.
- In the blood, **virus infects monocytes**, virus cannot fully replicate because they are not fully differentiated macrophages.
- After monocytes enter tissue, they differentiate into tissue macrophages and **virus can replicate** in these cells and serve to infect other macrophages and lymphocytes, especially in spleen and lymph nodes.
- Infected macrophages produce **pro-inflammatory chemokines and cytokines** that recruit additional monocytes and lymphocytes into organs.

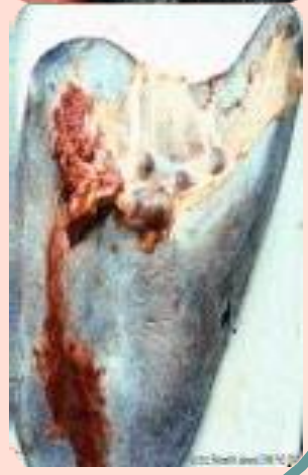
Pathogenesis

- Pulmonary intravascular **macrophages**, **Kupffer cells**, and **fixed macrophages** lining vascular sinusoids in the **spleen** and **lymph nodes** are **reservoirs** for virus and release it into the bloodstream.
- **Anemia** occurs from phagocytosis and complement-mediated lysis of erythrocytes that have had their membranes altered by virus, antibody, and complement.
- **Thrombocytopenia** occur because of activation of platelets and concurrent binding of fibrinogen to the surface of platelets during acute viremic phases of the disease. It is likely that activated platelets are quickly phagocytized by macrophages leading to thrombocytopenia.

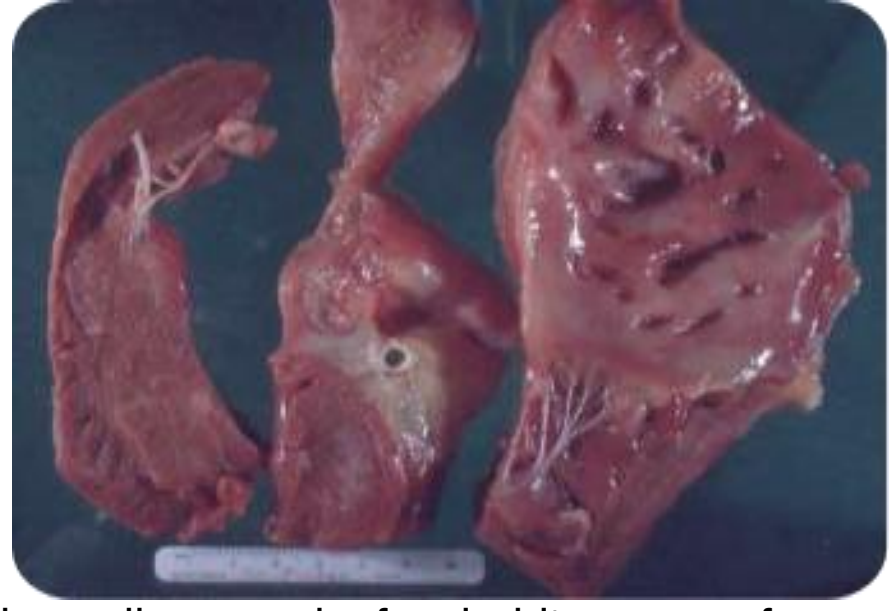
Gross lesions of Equine infectious anemia

- **Acute disease**
- Icterus, haemorrhages on serous membrane
- Paleness of mucous membranes
- Edema in the limbs and along the ventral abdominal wall
- Hepatomegaly, splenomegaly, lymphadenopathy
- **Chronic disease**
- Splenomegaly
- The **bone marrow** is dark red as a result of replacement of fat by hematopoietic tissue; the extent of replacement is an indication of the duration of the anemia.
- Heart – hemorrhage in epicardium and pericardium



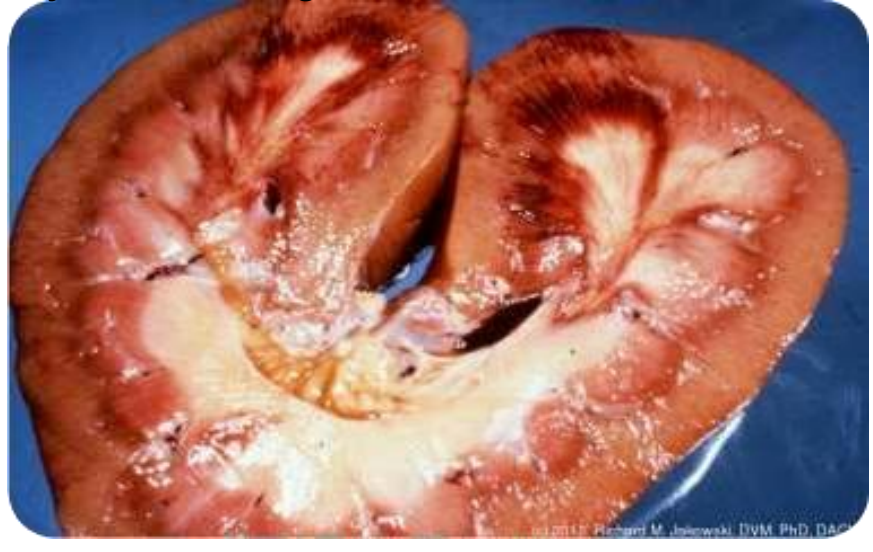


Enlarged grey red liver showing lobular pattern



Pale cardiac muscle, focal white areas of myocardial degeneration

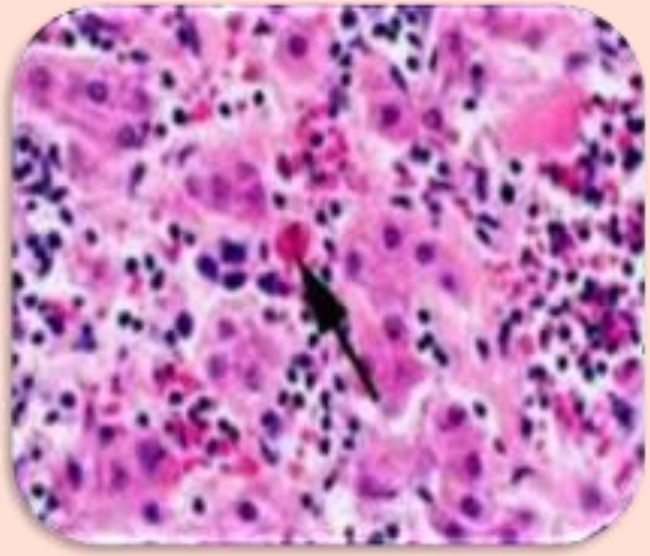
Replacement of BM fat with dark red hemopoietic tissue - erythroid hyperplasia



Kidney – infracts

Microscopic lesions

- Liver showed congestion of' the central vein and sinusoids
- Infiltration of lymphocytes in the portal areas
- Kupffer cells are enlarged and filled with hemosiderine.

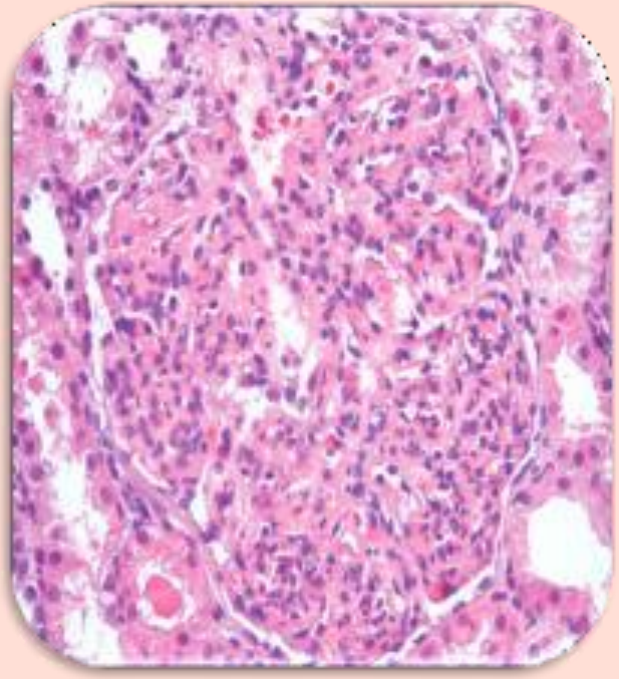


Microscopic lesions

- **Spleen** and **lymph nodes showed** infiltration and proliferation of lymphocytes and immature mononuclear cells in the red pulp of the spleen and trabeculae, and capsule of the lymph nodes
- **kidney** infiltratered by lymphocytes in the cortex and medulla especially around the blood vessels.
- The **cardiac muscle** infiltrated by lymphocytes with hyaline changes of the muscle bundles.
- Hyperplasia of bone marrow

Microscopic lesions

- Chronically infected horses have no gross lesions, but some animals may have glomerulonephritis characterized by thickened basement membrane and mesangium with neutrophilic infiltration and contained deposits of immune complex

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