Equine infectious anemia (Swamp fever)

The virus causing equine infectious anemia (ElAV) is RNA retrovirus, a member of the subfamily Lentivirinae of the family Retroviridae.

Epidemiology…

1-Horses and ponies are susceptible to infection by EIAV and characteristically develop signs of the disease within days to weeks of infection. Mules also become infected and develop clinical signs similar to that of horses .

2-donkeys do not develop equine infectious anemia and there is suspicion that strains of the virus pathogenic to donkeys exist but it considered as carrier

3-The source of all new infections of ElAV is an infected horse, donkey or mule.

4-The insect vectors responsible for the transmission of ElAV between horses are all large biting flies including Stomoxys calcitrans (stable fly), Chrysops sp. (deer fly), and Tabanus sp. (horse flies) .

5-mechamical transmission can occur by Mosquitoes

6-Intrauterine infection can occur and result in abortion or the birth of infected foals that often die within 2 months. Mares can be infected by insemination with semen containing the virus.

7-Infection can 'be readily achieved by the use of contaminated surgical instruments or needles

Pathogenesis….

1-Viral multiplication

After infection, ElAV multiplies in tissues that have abundant macrophages such as spleen.

2-Immune reaction

a-The immune response to ElAV is responsible for controlling replication of the virus and also plays an important role in the pathogenesis of the disease and The initial infection is likely controlled by cytotoxic T-Iymphocytes before the appearance of neutralizing antibodies.

b-Most virus in viremic horses is a complex of virus and antibody. The virus-antibody complex is readily phagocytosed by cells of the reticuloendothelial system, including tissue macrophages,and is involved in the development of the fever, depression, thrombocytopenia, anemia and glomerulonephritis characteristic of the disease .

Neurogenic sings in horses with ElAV infection is attributable to viral infection of neural tissue.

3-Anemia

The anemia of horses with ElAV attributable to shortened life of red blood cells and decreased red cell production. As Infection with ElAV shortens the lifespan of circulating red blood cells to about 38 d, compared with the normal value of 130 d.

The reduction in red blood cell lifespan is likely due to the presence of virus-antibody complexes on the surface of red blood cells with sequent clearance of such cells by the reticuloendothelial system as evidenced by the presence of sideroleukocytes in peripheral blood of infected horses. EIAV also has a suppressive effect on erythroid cells in bone marrow.

4- Thrombocytopenia

ElA and has been attributed to the deposition of virus-antibody complexes on platelets with subsequent removal of affected platelets by tissue macrophages. However, others

have identified a primary production deficit due to an indirect, non cytocidal suppressive effect of ElAV on megakaryocytes(the platelets origin).

Clinical findings…

1- incubation period of 2-4 weeks with initial anorexia, depression, weakness and loss of condition. Ataxia, behavioral changes, hyperesthesia and blindness some time.

2- There is Intermittent fever up to 41°, sometimes ,Jaundice, edema of the ventral abdomen the prepuce and legs, and Petechial hemorrhages in the mucosa, especially under the tongue and in the conjunctivae .

3- There is a characteristic increase in rate and intensity of the heart sounds, Respiratory signs are not marked and there is no dyspnea until the terminal stages, but there may be a thin serosanguineous nasal discharge. There is considerable enlargement of the spleen which may be detectable per rectum. Pregnant mares may abort

4- Many animals recover from this acute stage after a course of 3 d to 3 weeks. Others become progressively weak, recumbent and die after a course of 10-14 d of illness.

5- Animals recovering from the acute disease may appear normal for 2-3 weeks and then relapse with similar but usually less severe signs. Death may occur during relapse.

6- in chronic stage, the appetite is usually good, although allotriophagia may be observed. Some affected animals appear to make a complete recovery but they remain infected and may suffer relapses in later years.

Clinical pathology…

1-anemia,thrombocytopenia and appearance of sideroleukocytes (leukocytes containing hemosiderin)

2- Hypergammaglobulinemia may be present. Serum biochemical examination may reveal an increase in bilirubin concentration and a decrease in serum iron concentration.

3-detection of Abs by Elisa test .

Differential diagnosis ….

Acute disease

- Purpura hemorrhagica
- Babesiosis
- Equine granulocytic ehrlichiosis
- Equine viral a rteritis
- Autoimmune hemolytic anemia
- Leptospirosis
- Parasitism
- Idiopathic thrombocytopenia,

Chronic disease

- Internal abscessation (metastatic Streptococcus equi infection)
- Chronic inflammatory disease, neoplasia and chronic hepatitis.

Treatment…

No specific treatment is available. Supportive treatment including blood transfusions and hematinic.

Control…
1-Control of EIA is based on identification and eradication or life-long quarantine of infected animals.
2- control of biting insects and prevent mechanical transmission
3-vaccination using Killed, whole virus vaccines

African horse sickness

African horse sickness (AHS) is associated with a viscero-tropic orbi-virus (family Reoviridae) of which nine antigenic strains (serotypes) are recognized.

Epidemiology..

1-African horse sickness is an infectious but not contagious disease of Equidae(horses, donkeys,mules, zebra). It is spread by the bite of blood-feeding insects.

2-dogs, elephants and perhaps other wildlife animals may also infected

3-the disease distributed in all the worlds .

4-African horse sickness virus (AHSV) is transrnitted by the bite of hematophagous insects including midges (Culicoides spp.), ticks (Hyalomma dromadarii and the brown dog tick, Rhipicephalus sanguineus),and mosquitoes .

5- mechanical transmission may also occur some times

6- The incidence of the disease is often seasonal because of the seasonal variations in the number of insects .

7- high morbidity and mortality rate specially in horses

8-Zoonotic disease African horse sickness caused encephalitis and chorioretinitis in eight workers in an AHS vaccine factory. Infection was likely be inhalation of freeze-dried virus

Pathogenesis…

1-AHSV affects vascular endothelium and monocytes!macrophagesresulting in a variety of 'forms' or clinical presentations of the disease.

2-After infection, the virus multiplies in local lymph nodes and a primary viremia started with dissemination of infection to endothelial cells and intravascular macrophages of lung, spleen and lymphoid tissues. Viral multiplication then results in a secondary cell-associated (red cell and white cell)

3-Localization of antigen depends on the form of the disease -horses with horse sickness have most of he antigen in the spleen whereas horses with the more severe cardiopulmonary form have abundant antigen in cardiovascular and lymphatic systems .

4-Infection of endothelial cells results in degenerative changes, increases in vascular permeability, impaired intercellular junctions,loss of endothelium, subendothelial deposition of cell debris and fibrin, Edema,hemorrhage, and microthrombi are associated ith the vascular lesions.

5-Abnormalities in the lungs include development of alveolar and interstitial edema, sequestration of neutrophils and platelet aggregates and formation of fibrinous microthrombi

Clinical findings….

The incubation period in natural infections is about 5-7 d. Three are 4 clinical forms of the disease occur, an acute or pulmonary form, a cardiac or subacute form, amixed form, and a mild form known as 'horse sickness fever'. An intermittent feverof 40-41"C (105-106°F) is characteristic of all forms.

Acute (pulmonary) horse sickness (dunkop)

1-This is the most common form in epizootics and has a case fatality rate of 95 % . Fever is followed by labored breathing, severe paroxysms of coughing and a profuse nasal discharge of yellowish serous fluid and froth.

2-Profuse sweating, profound weakness and a staggery gait progress to recumbency.

3-Death usually occurs after a total course of 4-5 d ,although it can be so acute as to be without observed signs in some horses. Severe respiratory distress persists for many weeks in surviving animals. This is the form of the disease that occurs naturally in dogs.

Subacute (cardiac) horse sickness (dikkop)…

1-is most common in horses in enzootic areas and has a case fatality rate of 50 % . The incubation period may be up to 3 weeks,and the disease has a more protracted course than does the acute, pulmonary form.

2-There is edema in the head,particularly in the temporal fossa, the eyelids and the lips, and the chest which may not develop until the horse has been febrile for a week

3-The oral mucosa is bluish in color and petechiae may develop under the tongue.

4-Examination of the heart and lungs reveals evidence of hydropericardium,endocarditis and pulmonary edema.

5-Restlessness and mild abdominal pain and paralysis of the esophagus, with inability to swallow and regurgitation of food and water through the nose,some time seen. Recovery is prolonged. A fatal course may last as long as 2 weeks.

A mixed form….

It characterized by both pulmonary and cardiac signs, is evident as an initial subacute cardiac form that suddenly develops acute pulmonary signs. Also, a primary pulmonary syndrome may subside but cardiac involvement causes death. This mixed form is not common in field outbreaks.

Horse sickness fever…

A mild form of horse sickness fever that may be easily detected , and is common in enzootic areas. The disease occurs in horses with some immunity or infection by serotypes of low virulence. This is the only form of the disease that occurs in zebra. The temperature rises to 40.5°C over a period of 1-3 d but returns to normal about 3 d later. The appetite is poor, there is slight conjunctivitis and moderate respiratory distress.

Clinical pathology…

1-Leukopenia, with lymphopenia, neutropenia and a left shift,

2-mild thrombocytopenia and hemoconcentration are characteristic of the acute forms

3-Serum biochemical abnormalities include increases in creatine kinase, lactate dehydrogenase,and alkaline phosphatase activities and creatinine and bilirubin concentrations**.**

4-abnormalities of clotting factors indices

5-serological testes using IFA and Elisa

Control…

1-Prevention of introduction of infection by clinically ill or in apparently infected animals

2- Slaughter of viremic animals where animal welfare and economic considerations permit this course of action

3-Management changes to reduce exposure to midges and Vector control

4- Induction of active immunity in animals at risk of disease using vaccination of attenuated vaccine . after vaccination is protective for at least 1 year but annual revaccination of all horses, mules and donkeys is recommended.

Equine viral arteritis (EVA)

Viral arteritis of horses, donkeys and mules is associated with an arterivirus, member of the coronavirus-like superfamily, The virus is single-stranded RNA.

Epidemiology…

1-Horizontal transmission of virus by nasal fluid, but also by urine, feces, lacrimal fluid and vaginal discharge of infected mares

2 Venereal transmission from stallions to susceptible mares.

3-recovery from natural infection results in the development of a strong immunity

Pathogenesis..

1-After inhalation of the virus it binds to the respiratory epithelium and infects alveolar macrophages and is detectable in bronchial lymph nodes by 48 hours after infection. Three days after infection the virus is detectable in circulating monocytes with subsequent systemic distribution of infection.

2-the virus localized in vascular endothelium and medial myocytes by days 6-9 and there is significant damage to blood vessels by day 10.

3-The virus infects renal tubular epithelium and can persist there for up to 2 weeks.

4-Medial necrosis of blood vessels might cause anoxia of associated tissues

5-Abortion is caused by a severe necrotizing myometritis

Clinical findings…

1-Infection by EAV is usually clinically inapparent, especially after venereal infection of mares. Abortion is not necessarily associated with clinical disease in the mare

2-Systemic disease is characterized by an incubation period of 1-6 days followed by the appearance of fever 39-41°C.

3-A serous nasal discharge which may become purulent and be accompanied in some horses by congestion and petechiation of the nasal mucosa, urticaria, conjunctivitis, excessive lacrimation developing to purulent discharge,keratitis, palpebral edema, and blepharospasm.

4-Opacity of the aqueous humor and petechiation of the conjunctiva may also occur

5-Signs of pulmonary disease, such as respiratory distress and coughing are attributable to pulmonary edema and congestion, but are uncommon.

6-The appetite is reduced or absent and, in severe cases, there may be abdominal pain, diarrhea and jaundice

7-Edema of the limbs is common and more marked in stabled horses than those at pasture

8-In stallions, edema of the ventral abdominal wall may extend to involve the prepuce and scrotum.

9-dehydration, muscle weakness develop quickly.and deaths may occur without secondary bacterial invasion

10-Clinical disease in neonatal foals is characterized by fever, depression,weakness, limb and facial edema,and respiratory distress. Severely affected foals usually die. Foals can be affected at birth, or be born apparently normal and develop disease 1-19 days after birth.

11-Abortion occurs within a few days of the onset of clinical illness, although itis not usually associated with clinically apparent disease. Abortions may occur in 10-60% of at-risk mares during an outbreak and during the 3rd-l0th months of gestation.

Clinical pathology…

1-leukopenia and thrombocytopenia

2-virus isolation
3- serological test such as Elisa
Treatment …
There is no specific treatment for equine viral arteritis. Most horses recover without specific care. Severely affected foals require intensive care

Control….
1-. Isolate all new arrivals (and returning horses) to farm or ranch for 3 to 5 weeks
2. If possible, segregate pregnant mares from other horses
3. Blood test all breeding stallions for EAV antibodies
4. Check semen of any unvaccinated, antibody-positive stallions for EAV to identify carriers before breeding
5 Once tested negative for EAV antibodies, vaccinate all breeding stallions annually
6. Physically isolate any EAV carrier stallions
7. Restrict breeding EAV carrier stallions to vaccinated mares or mares which test positive for naturally acquired antibodies to the virus
8. Vaccinate mares against EVA at least 3 weeks prior to breeding to a known carrier stallionVaccination with a modified live virus vaccine
9. Isolate mares vaccinated for the first time against EVA for 3 weeks following breeding to an EAV carrier stallion
10. In breeds or areas with high rates of EAV infection, vaccinate all intact males between 6 to 12 months of age.