Equine abortions

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Numerous and frequent are the etiologic agents able to cause abortion, stillbirth in horses.

Equine herpesvirus (EHV)-1 causes epidemic abortion, stillbirth, respiratory disease and myeloencephalopathy with paralysis in *Equidae* and *Camelidae*, whereas EHV-4 induces sporadic abortion and mainly respiratory disease**.** Considering the value of some foals, it is clear that both sporadic and epidemic abortions are of great economic importance to the horse industry. Rapid diagnosis is paramount. Often the mares abort in the last few months of pregnancy with no particular premonitory signs. The fetus is often contained within the fetal membranes with the red chorion on the external part. The fetus may show the following suggestive, but non-specific, features and lesions: meconium staining on the body surfaces, mucosal petechiae, straw colored fluid in the serosal cavities, edematous atelectatic lungs with rib impressions and thymic necrosis. Unfortunately, the pathognomonic macroscopic gray foci representing hepatic necrosis are not always present. Histologically, if lung, liver and thymus are collected, is always possible to observe the suggestive multifocal coagulative necrosis associated with the intranuclear acidophilic pathognomonic viral inclusions. It is also possible to examine frozen sections and detect lesions shortly after postmortem examination. Equine herpesvirus-1 antigen (EHV-1 Ag) can be detected in fixed tissues using peroxidase immunohistochemistry, which allows the simultaneous evaluation of lesions and EHV-1 Ag localization. Equine herpesvirus-1 Ag can also be abundant in areas where lesions are mild or absent. The antigen is intranuclear and intracytoplasmic and can be mainly detected in endothelium, epithelium and monocyte-macrophages, but also in myocytes and pericytes. Target organs are mainly lung, liver, reticular cells of lymphoid tissue and cortices of adrenal gland, but also intestine, kidney, pancreas and fetal membranes. The virus grows well in RK-13 cells where it produces a cytopathic effect, generally within 24 hours. Equine herpesvirus-4 is a slow growing herpesvirus, which replicates in a few days on equine lung cells. Virus identification can also be obtained with immunohistochemistry using fluorescent antibodies. Considering the latency of herpesviruses, polymerase chain reaction (PCR) techniques may be able to identify the infection, but without the contemporary evaluation of the presence of the lesions it is not possible to differentiate between latency, productive infection and disease. Fetal and maternal serology may be of benefit. However, vaccinated dams may not have a representative humoral immune response and fetal death can be very rapid, with insignificant fetal serological response, even in immunocompetent fetuses.

Equine arteritis virus (EAV) is an arterivirus, which is also able to induce respiratory disease, stillbirth, foal mortality and abortion. The aborted fetus can be fresh or moderately autolyzed and pathognomonic lesions are not observed. Occasionally there is mild interstitial pneumonia and vasculitis. Although with immunoperoxidase histochemistry it may be possible to identify EAV Ag in fetuses and the allantochorion, serological evaluation of the dam and virus isolation are, at the moment, the most powerful tools used to diagnose EAV abortion. On the other hand, lesions are highly suggestive in fatal cases involving adults and foals: interstitial pneumonia, systemic vasculitis, and, in terminal phases of the disease, tubular necrosis. Getah virus and equine influenza are also potential causes of abortion. Perhaps a vesivirus is able to cause equine abortion, but additional studies are needed to verify this.

Other rather common infectious etiologic agents of abortion in horses are bacteria and fungi. *Streptococcus equi var. zooepidemicus* induces severe progressive cervical chorionic necrosis and thickening with neovascularization and allantoic hyperplasia lesions progress from the cervical area toward the placental body inducing progressive placental insufficiency. Sometimes this is not followed by fetal septicemia. Similar gross and histologic findings are observed in cases of *Aspergillus spp.* mitotic placentitis. Gram-negative bacteria like *Escherichia coli*, *Klebsiella* spp and others are often able to induce fetal septicemia with dissemination of bacterial emboli in various organs. *Leptospira interrogans* serogroup *pomona*, serovar *kennewicki* and *gryppotyphosa* have been identified as a cause of abortion and neonatal sepsis in horses in Kentucky. In this case, placentitis does not ascend from the cervical area, but is most likely hematogenic in origin. Examination of the placenta may reveal edema

and necrosis of the chorion, enlarged liver and swollen kidneys with pale streaks. Histologically there is placental thrombosis, vasculitis and villitis with necrosis followed by hepatocellular dissociation, giant cell hepatopathy, nephritis, pneumonia and myocarditis. It is possible to identify the spiraliform bacteria using Steiner and Whartin Starry silver stains and or immunoperoxidase histochemistry, which allow to simultaneous evaluation of the lesions and bacterial antigens. This technique has made the use of fluorescent antibodies to detect leptospira in the placenta obsolete, where the specificity of the staining is difficult to interpret. Higher bacteria may cause equine placentitis and other bacteria have been identified as sporadic causes of abortions in mares. *Neorickettsia risticii*, the etiologic agent of Potomac horse fever induces enteritis, splenitis and hepatitis in the fetus. In addition to the relatively common *Aspergillus*, other causes of mycotic abortion include *Mucor spp*., *Histoplasma capsulatum*, *Candida spp*., whereas *Allescheria boydii* and *Coccidioides immitis* are rare.

Amongst the noninfectious causes of abortion there are umbilical cord abnormalities, which may induce fetal hypoxia and death. Umbilical cord torsion is predisposed by an abnormal increase in length. Acute and recurrent chronic torsions have been observed. The first is associated with severe segmental to diffuse edema and hemorrhage of the cord, whereas the latter may be characterized by urinary bladder distention, urachal dilatations, chorionic vessel thrombosis and intraluminal mineralization. Twinning often ends in abortion. Fetal loss appears to be caused by a combination of factors including inadequate nutrition, interfetal incompatibility and immunologic reactions. A sharp line of demarcation can readily identify the placentae from twins where the chorioallantois of the two fetuses comes in contact with each other. Other non-infectious causes of abortion include: early placenta separation, placenta edema, contracted foal syndrome, congenital anomalies, atrophy of placental villi, body pregnancy, and fetal diarrhea syndrome. Fescue toxicosis is an important cause of perinatal death of foals. It is associated with the endophyte *Neotyphodium* (previously *Acremonium spp*.). In these cases placenta membranes are congested and very edematous.

It is proposed that the mare reproductive loss syndrome observed in Kentucky, USA, which includes early fetal loss, late fetal loss, uveitis, pericarditis, and encephalitis, is associated with tissue penetration by septic barbed setal fragments (septic penetrating setae) from Eastern tent caterpillars (*Malacosoma americanum*). Once ingested, these barbed setal fragments migrate through moving tissues, followed by rapid hematogenous spread of bacteria, bacterial emboli, and/or septic fragments of setae (septic penetrating setal emboli).

There are other suggested but unproven causes of equine abortion.

The dissection of the aborted fetus and placenta and the following ancillary procedures including histopathology, immunohistochemistry, virology, bacteriology, and serology will allow us to obtain a rapid diagnosis and to prevent other fetal and neonatal losses.

Several control measures are required for the management of an epizootic situation, but of primary importance is the proper diagnosis of the disease involved. Once the offending agent is identified, control measure must be implemented. Control measures include isolation of affected animals, isolation and examination of any newly introduced animals, maintaining separate implements for sick and healthy animals and separating farm workers into those working with sick animals and those working with healthy animals. Depending on the infectious agent, vaccination in the face of an outbreak may be warranted, particularly in previously unvaccinated animals considered at risk. Transport of any animals to and from the farm should be prevented during the disease outbreak and for several weeks after the last affected animals are normal.

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