Equine Viral Arteritis

Equine Typhoid, Epizootic Cellulitis—Pinkeye, Epizootic Lymphangitis Pinkeye, Rotlaufseuche

Importance

Equine viral arteritis is an economically important viral disease of equids. Stallions can transmit this virus during breeding and sometimes become long-term virus carriers in semen. Although carrier stallions can be bred if precautions are taken, the need to mate them with seropositive or vaccinated mares decreases their desirability as breeders. Acute illnesses are also seen occasionally, though many infections in nonpregnant animals are subclinical. While deaths are very rare in healthy adults, pregnant mares that become infected may abort, and very young foals may die of fulminant pneumonia and enteritis. Some reports suggest that equine viral arteritis may be increasing in prevalence due to the increased transportation of horses and semen.

Etiology

Equine viral arteritis is caused by equine arteritis virus (EAV; official species name Alphaarterivirus equid), an RNA virus in the genus Alphaarterivirus, family Arteriviridae and order Nidovirales. Only one serotype is recognized; however, there are a number of antigenic variants, and some isolates of EAV are more virulent than others. There are two known viral lineages, North American and European, and several clades within these lineages. EAV strains that differed significantly from either lineage were reported in some donkeys in South Africa, and other divergent strains may also exist, particularly as there is only limited information about the viruses circulating in Africa, Asia or the Middle East.

Species Affected

Equine arteritis virus infects equids. Clinical cases have been reported mainly in horses and ponies; however, antibodies to EAV are also found in donkeys and zebras, and experimentally infected donkeys developed clinical signs. EAV might also be able to cause disease in South American camelids, as a PCR test detected viral nucleic acids in an alpaca that had aborted; however, this remains to be confirmed by further studies and virus isolation.

Zoonotic potential

There is no indication that EAV can infect humans.

Geographic Distribution

Serological and/or virological evidence for the presence of EAV has been found in North and South America, Europe, Asia, Africa and Australia, as well as in most individual countries where testing has been done. However, this virus is reported to be absent from a few locations including Iceland and Japan. New Zealand appears to have successfully eliminated EAV circulation in horses, and the general population of equids there was reported to be virus-free in 2013.

The North American and European lineages are not limited to the continents where they originated, and can occur in other locations.

Transmission

Acutely affected horses excrete EAV in respiratory secretions, and this virus can be transmitted in aerosols where susceptible horses are in close contact. It has also been found in other secretions and excretions, including urine and feces, and occurs in the reproductive tracts of mares (vaginal and uterine secretions, ovary, oviduct) and stallions (semen). Venereal transmission is common, with stallions spreading the virus via both artificial insemination and natural service. Semen remains infectious after freezing. EAV was also shown to be transmissible via embryo transfer if the donor mare was inseminated with infected semen. Mares infected late in pregnancy may give birth to infected foals.

Mares, geldings and sexually immature colts eliminate EAV completely after an infection; however, stallions can continue to carry the virus subclinically in the male reproductive tract and shed it in semen for periods ranging from weeks to years. Some of these carriers eventually clear the infection, but others remain infected for life.
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EAV can occasionally be found for up to six months in the reproductive tract of older prepubertal colts, but they do not become true carriers.

EAV can be transmitted on fomites (e.g., equipment) and mechanical vectors such as humans or animals. Under some conditions, this virus can withstand heat of 37-38°C (99-100°F) for 2-3 days, or cold of 4-8°C (39-46°F) for up to 75 days; however, it is sensitive to sunlight and low humidity.

Disinfection

EAV is readily destroyed by detergents, common disinfectants (e.g., sodium hypochlorite, iodides and quaternary ammonium compounds) and lipid solvents such as ether or chloroform. It is inactivated in 20–30 minutes at 56-58°C (133-136°F).

Incubation Period

The incubation period ranges from 2 days to 2 weeks. Infections transmitted venereally tend to become apparent in about a week.

Clinical Signs

Equine viral arteritis is generally more severe in old or very young animals, and in horses that are immunocompromised or in poor condition. Most infections, including those that occur in mares bred to long-term carriers, are subclinical.

Fulminant infections with severe interstitial pneumonia and/or enteritis can be seen in foals up to a few months of age, including some congenitally infected newborns. Adult horses occasionally develop a systemic illness, with clinical signs that often include varying degrees of fever, depression, anorexia, rhinitis, and dependent edema of the prepuce, scrotum, mammary gland, ventral body wall and/or limbs, particularly the hindlegs. Some horses may also have conjunctivitis, photophobia, periocular or supraorbital edema, urticaria and petechiae on the mucous membranes. Urticaria may be localized to the head and/or neck or generalized. Pregnant mares may abort or give birth to a stillborn foal, with or without any other clinical signs. Stallions may experience a temporary decrease in fertility, with reduced quality sperm and decreased libido, during the acute stage of the disease. The decrease in sperm quality has been attributed to increased scrotal temperature and edema, and can persist for up to 4 months. The quality of the semen is not decreased in carrier stallions. Except in cases of severe disease in foals, deaths are rare.

Outbreaks have not been reported among donkeys or mules, and although antibodies have been found, there is relatively little information about the clinical signs in these species. Fever, sometimes accompanied by mild depression, mild conjunctivitis, slight ocuolonasal discharge and/or mild dependent edema, was reported in donkeys inoculated with an EAV strain that is moderately virulent for horses. These signs were mild enough that they could readily have been missed if the animals were not closely monitored. Donkeys inoculated with a South African donkey strain had similar clinical signs; however, while some cases were mild, others were more severe. Pregnant donkeys did not abort in this study, and their foals were clinically normal when born. Horses inoculated with the South African donkey strain developed only very mild signs.

Post Mortem Lesions

Acutely ill animals may have edema, congestion and hemorrhages in the subcutaneous tissues, particularly those of the limbs and abdomen, and in various visceral organs and lymph nodes. The thoracic and abdominal lymph nodes, and the small and large intestines (especially the colon and cecum) are frequently affected, and the peritoneal cavity, pleura and pericardium may contain clear, yellowish fluid. Severely affected young foals often have pulmonary edema, interstitial pneumonia, emphysema, splenic infarcts and/or enteritis.

Aborted fetuses tend to be partially autolyzed, though they are sometimes well preserved. The only gross lesions in some fetuses may be excess fluid in the body cavities and signs of interlobular interstitial pneumonia. The endometrium of mares that aborted may be swollen and congested, and sometimes contains hemorrhagic lesions.

Diagnostic Tests

Equine viral arteritis can be diagnosed by virus isolation, the detection of viral nucleic acids or antigens, and serology.

Culture and/or RT-PCR may detect EAV in nasopharyngeal swabs or washings, conjunctival swabs, blood and semen from recently infected animals, as well as in a number of tissues at necropsy. The placenta, fetal fluids and fetal tissues (particularly lymphoid tissues, lung and spleen) are generally sampled in aborted fetuses or stillborn foals, while organs and lymph nodes associated with the respiratory and gastrointestinal tracts (e.g., lungs, liver, spleen) are collected in young foals that died of a systemic illness. In live animals, samples for virus detection, other than semen, should be collected as soon as possible after the onset of clinical signs. Blood samples intended for virus isolation should use acid citrate dextrose or EDTA as an anticoagulant, as heparin may inhibit the growth of RK-13 cells, the most commonly used cell line. The identity of the recovered virus can be confirmed by serum neutralization, RT-PCR, immunofluorescence or immunohistochemistry. An antigen-capture ELISA for use with culture supernatants has also been described.

Immunohistochemistry may identify viral antigens in tissue samples, including some taken from organs with no apparent lesions. It has also been used to identify acutely infected horses from skin biopsies, though this was not completely reliable. Histopathology is helpful in clinical cases, particularly abortions. Tissues likely to contain typical histopathological changes include the cecum, colon, spleen and associated lymph nodes, and adrenal cortex.

Carrier stallions can be identified by virus isolation or RT-PCR on semen. Samples should contain the sperm-rich fraction of the ejaculate, as EAV is not found in the pre-sperm fraction. Carriers can also be detected by breeding the
stallion to two seronegative mares, which are checked for seroconversion 4 weeks after breeding. Secretions and excretions other than semen do not contain EAV in carriers.

In recently infected horses, serology should demonstrate a fourfold increase in titer in paired acute and convalescent samples. Virus neutralization (e.g., the complement enhanced microneutralization test) and ELISAs are the most commonly used tests, but other assays such as complement fixation, the fluorescent microsphere immunoassay (MIA), agar gel immunodiffusion and indirect fluorescent antibody may also be available.

**Treatment**

Treatment is supportive, and most healthy horses recover on their own. Although some antivirals appear promising against EAV *in vitro*, there are no reports of their use in live animals.

**Control**

**Disease reporting**

Veterinarians who encounter or suspect equine viral arteritis should follow their national and/or local guidelines for disease reporting. State regulations should be consulted in the U.S., where this disease is reportable in many states.

**Prevention**

Isolating infected horses during an outbreak may reduce transmission. Precautions should also be taken to avoid spreading the virus on fomites. Equine viral arteritis vaccines appear to reduce virus shedding, in addition to preventing clinical signs, and may help contain outbreaks. However, live attenuated vaccines may cause some abortions in pregnant mares (probably mainly or exclusively during the last 2-3 months of gestation) and they are not recommended for any pregnant animals unless a high risk of abortion from EAV exposure outweighs this consideration. Live attenuated vaccines are also not recommended for young foals < 6 months of age, or the dams of these foals.

Venereal transmission and abortions can be controlled by good management and vaccination. Newly acquired horses should be isolated for 3-4 weeks, and pregnant mares should be separated from other horses and maintained in small groups according to their predicted foaling dates. To prevent venereal transmission, stallions should be tested for EAV, and carrier stallions should only be bred to well vaccinated or naturally seropositive mares. Because first-time vaccinates may shed the virus for a short time after being bred to a carrier, these mares should be isolated from seronegative horses, particularly pregnant mares, for 3 weeks. Naturally infected mares and those that are not first-time vaccinates are isolated for 24-48 hours. Excellent hygiene and decontamination of fomites should be practiced when breeding infected horses or collecting semen.

Live attenuated vaccines appear to prevent uninfected stallions from becoming long-term carriers, and stallions are regularly vaccinated before the start of the breeding season in some counties. Prepubertal colts are given the vaccine when they are 6-12 months old. The efficacy of killed vaccines in preventing carriage is not as well characterized. Although uninfected stallions have been kept near carriers for years without becoming infected, in one case the virus was apparently transmitted by indirect exposure to semen, and it has been recommended that carrier stallions be housed where they can be physically separated from uninfected horses. While there is no proven method to eliminate carriage in stallions, initial studies of anti-GnRH vaccines seem to be promising. One or more doses of a vaccine appeared to clear the virus from the semen of 15 carriers within 1-2 years, though it also caused persistent deficits in reproductive function in a minority of these animals.

New Zealand used annual testing, the identification of carrier stallions and control of transmission from these animals, and selective vaccination to eradicate equine viral arteritis.

**Morbidity and Mortality**

The prevalence of EAV in horses varies, and it is known to be higher in some breeds than others. Particularly high exposure rates, with approximately 25-70% or higher seroprevalence in unvaccinated individuals, have been reported in Standardbreds in the U.S., Warmbloods in Europe and Hucul horses in Poland, among others. In contrast, antibodies were found in less than 5% of unvaccinated Warmbloods in the U.S. Persistent carriage in stallions is likely to influence virus prevalence, and has ranged from < 10% to 70% in different outbreaks. Whether a stallion has a susceptible or resistant phenotype in the EqCXCL16 gene has been shown to strongly influence whether or not it becomes a long-term carrier.

EAV infections are frequently inapparent, and clinical outbreaks are uncommon. In many cases, outbreaks have been associated with horses being moved or semen shipped. The clinical signs caused by most field strains are often mild, though they may be more severe in old or very young animals, and in horses that are immunocompromised or in poor condition. Reported abortion rates in outbreaks have ranged from < 10% to ≥ 50%. Deaths primarily occur in very young foals, and are very rare in healthy adults. While immunity to EAV is thought to be long-lasting, some horses can become reinfected.

**Internet Resources**


The Merck Veterinary Manual

United States Department of Agriculture (USDA) Animal and Plant Health Inspection Service (APHIS). Equine viral arteritis

World Organization for Animal Health (WOAH)
Acknowledgements

This factsheet was written by Anna Rovid Spickler, DVM, PhD, Veterinary Specialist from the Center for Food Security and Public Health. The U.S. Department of Agriculture Animal and Plant Health Inspection Service (USDA APHIS) provided funding for this factsheet through a series of cooperative agreements related to the development of resources for initial accreditation training.

The following format can be used to cite this factsheet. Spickler, Anna Rovid. 2022. Equine Viral Arteritis. Retrieved from http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php.

References


BroadusCC, Balasuriya UBR, White JLR, Timoney PJ, Funk RA, Hodyoag GR. Evaluation of the safety of vaccinating mares against equine viral arteritis during mid or late gestation or during the immediate postpartum period. JAVMA. 2011;238(6):741-50.


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