

Equine Viral Arteritis

*Equine Typhoid,
Epizootic Cellulitis–Pinkeye,
Epizootic Lymphangitis Pinkeye,
Rotlaufseuche*

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- OIE Collaborating Centres:
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Importance

Equine viral arteritis (EVA) is an economically important viral disease of equids. Stallions can become long term carriers of the virus, and transmit it during breeding. 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 illness also occurs in some horses. Although deaths are very rare in healthy adults, pregnant mares that become infected may abort, and very young foals may die of fulminating pneumonia and enteritis. Equine viral arteritis has recently increased in prevalence, possibly due to increased transportation of horses and semen.

Etiology

Equine viral arteritis is caused by equine arteritis virus (EAV), an RNA virus in the genus *Arterivirus*, family Arteriviridae and order Nidovirales. Isolates vary in their virulence and potential to induce abortions. Only one serotype has been recognized. Limited genetic analysis suggests that EAV strains found among donkeys in South Africa may differ significantly from isolates in North America and Europe.

Species Affected

Equine arteritis virus is found in the Equidae. Antibodies to this virus have been reported in horses, ponies, donkeys and zebras. Illness occurs mainly among horses and ponies, but clinical signs have also been reported in experimentally infected donkeys. EAV might also be able to cause disease in South American camelids: polymerase chain reaction assay (PCR) detected viral nucleic acids in an alpaca that had aborted.

Geographic Distribution

Antibodies to EAV have been found in most countries where testing has been done. Seropositive horses have been reported in North and South America, Europe, Asia, Africa and Australia. Infections are common among horses in continental Europe, but rare in the United Kingdom. This virus appears to be absent from Iceland and Japan.

Transmission

Equine arteritis virus can be transmitted by the respiratory and the venereal routes. Acutely affected horses excrete the virus in respiratory secretions; aerosol transmission is common when horses are gathered at racetracks, sales, shows and other events. This virus has also been found in urine and feces during the acute stage. It occurs in the reproductive tract of acutely infected mares, and both acutely and chronically infected stallions. In mares, EAV can be found in vaginal and uterine secretions, as well as in the ovary and oviduct, for a short period after infection. Mares infected late in pregnancy may give birth to infected foals. Stallions shed EAV in semen, and can carry the virus for years. Transmission from stallions can occur by natural service or artificial insemination. Some carriers may eventually clear the infection. True carrier states have not been reported in mares, geldings or sexually immature colts; however, EAV can occasionally be found for up to six months in the reproductive tract of older prepubertal colts.

Equine arteritis virus can be transmitted on fomites including equipment, and may be spread mechanically by humans or animals. This virus is inactivated in 20–30 minutes at 56–58°C (133–136°F), but can remain viable for 2 to 3 days at 37–38°C (99–100°F) and for up to 75 days at 4–8°C (39–46°F). Semen remains infectious after freezing.

Incubation Period

The incubation period varies from 2 days to 2 weeks. Infections transmitted venereally tend to become apparent in approximately one week.

Clinical Signs

Most EAV infections, especially those that occur in mares bred to long-term carriers, are asymptomatic. The clinical signs are generally more severe in old or very

Equine Viral Arteritis

young animals, and in horses that are immunocompromised or in poor condition. Fulminant infections with severe interstitial pneumonia and/ or enteritis can be seen in foals up to a few months of age. Systemic illness also occurs in some adults. In adult horses, the clinical signs may include fever, depression, anorexia, limb edema (particularly in the hindlimbs), and dependent edema of the prepuce, scrotum, mammary gland and/or ventral body wall. Conjunctivitis, photophobia, periorbital or supraorbital edema and rhinitis can also be seen. Some horses develop urticaria; the hives may be localized to the head or neck, but are sometimes generalized. Abortions or stillbirths can occur in mares that are pregnant when they are exposed. Abortions are not necessarily preceded by systemic signs. Temporary decreases in fertility, including reduced quality sperm and decreased libido, may be seen in stallions 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 little information on the clinical signs in these species. Fever, sometimes accompanied by mild depression, mild conjunctivitis, a slight oculonasal discharge and/or mild dependent edema were reported in donkeys inoculated with an EAV strain that is moderately virulent for horses. The clinical signs were mild, and could readily have been missed if the animals were not closely monitored. Donkeys inoculated with a South African donkey strain developed mild to more severe clinical signs including fever, depression, serous ocular and nasal discharge, and conjunctivitis. Pregnant donkey mares did not abort, and their foals were clinically normal when born. Horses inoculated with this donkey strain had very mild clinical signs.

Post Mortem Lesions

In acute cases, the lesions are characterized by edema, congestion and hemorrhages of the subcutaneous tissues, visceral organs and lymph nodes. These changes are often found in the subcutaneous tissues of the limbs and abdomen, the thoracic and abdominal lymph nodes, and the small and large intestines (especially the colon and cecum), but may occur throughout the body. Accumulations of clear, yellowish fluid may be found in the peritoneal cavity, pleura and pericardium. Foals may also have pulmonary edema, interstitial pneumonia, emphysema, splenic infarcts and enteritis. In mares that abort, the endometrium may be swollen and congested, and can contain hemorrhages. Aborted fetuses are often partially autolyzed, but may be well preserved. In some fetuses, the only gross lesions may be excess fluid in the body cavities and signs of interlobular interstitial pneumonia.

Morbidity and Mortality

The prevalence of equine arteritis virus varies significantly among horse breeds. In the U.S., this infection is particularly common among Standardbreds. In a survey done in 1998, 24% of unvaccinated Standardbreds, 4.5% of Thoroughbreds, 0.6% of Quarter horses, and 3.6% of Warmblood horses had antibodies to this virus. In Europe, the seroprevalence is particularly high among Warmbloods. Breed-related differences in seroprevalence might be due to genetic differences, but they are more likely to be caused by different management practices. In experimentally infected horses, the breed has no apparent effect on susceptibility to infection or the establishment of carriers. Overall, approximately 10-70% of infected stallions become carriers of EAV.

Outbreaks are much less common than infections, and tend to occur when horses have been moved or semen shipped. The clinical signs are generally more severe in old or very young animals, and in horses that are immunocompromised or in poor condition. They also vary with the route of exposure, and the strain and dose of the virus. Asymptomatic infections are common, and deaths are very rare in healthy adults; young foals are more likely to die. The abortion rate varies from less than 10% to as high as 50-60%.

Diagnosis

Clinical

Equine viral arteritis should be considered when the clinical signs include fever, depression, edema, conjunctivitis, nasal discharges and abortions. This disease is difficult to differentiate from other systemic and respiratory illnesses of horses.

Differential diagnosis

The differential diagnosis includes equine influenza, equine infectious anemia and African horse sickness, as well as infections with Getah virus, Hendra virus, equine rhinitis A and B viruses, equine adenoviruses, and equine herpesviruses 1 and 4. Equine viral arteritis also resembles purpura hemorrhagica and other streptococcal infections, as well as poisoning from the toxic plant *Berberoa incana* (hoary alyssum).

Laboratory tests

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

In recently infected animals, equine arteritis virus may be recovered from nasal secretions, blood and semen as well as from a number of tissues and fluids at necropsy. Carrier stallions can be identified by isolating the virus from semen; EAV is not found in the respiratory secretions, blood or urine of carriers. This virus can be isolated in rabbit, equine and monkey kidney cells or cell lines. RK-13 (rabbit kidney) cells are the system of choice. The identity

Equine Viral Arteritis

of the virus can be confirmed using serum neutralization, reverse-transcription PCR (RT-PCR) assays, immunofluorescence or immuno-histochemistry.

RT-PCR can also detect viral nucleic acids in clinical samples from live horses or tissues taken at necropsy. Immunohistochemistry can identify viral antigens in necropsy samples, as well as in skin biopsies from acutely infected horses; the latter technique is not completely reliable. Histopathology is helpful in clinical cases.

Serological tests include virus neutralization, complement fixation, agar gel immunodiffusion, indirect fluorescent antibody, fluorescent microsphere immunoassay (MIA) and enzyme-linked immunosorbent assays (ELISA). A complement-enhanced micro-neutralization test is often used to detect antibodies to EAV. In acute cases, a fourfold increase in titer should be seen in serological tests.

Carriers can also be detected by breeding the stallion to two seronegative mares, which are checked for seroconversion four weeks after breeding.

Samples to collect

In live horses with acute infections, EAV can be isolated from nasopharyngeal swabs or washings, conjunctival swabs and unclotted blood samples. Because heparin may inhibit the growth of RK-13 cells, blood samples should be anticoagulated with acid citrate dextrose or EDTA. Clinical samples can also be tested by RT-PCR. Samples should be collected as soon as possible after the onset of the clinical signs. Samples for virus isolation should be refrigerated and shipped cold. Carriers can be identified by detecting EAV in semen using virus isolation or RT-PCR. Semen samples should contain the sperm-rich fraction of the ejaculate; virus is not found in the pre-sperm fraction.

At necropsy, tissues should be collected for histopathology; the cecum, colon, spleen and associated lymph nodes, as well as the adrenal cortex, are especially helpful. In aborted fetuses or stillborn foals, samples for virus isolation should be taken from the placenta, fetal fluids and fetal tissues, especially the lymphoid tissues, lung and spleen. In young foals with fatal disease, necropsy samples should include a variety of organs and lymph nodes associated with the respiratory and gastrointestinal tracts; they should include the lungs, liver and spleen. Tissue samples can also be collected for RT-PCR and immunohistochemistry. EAV antigens can be found in organs with or without lesions. Immunoperoxidase staining can detect viral antigens in formalin-fixed or frozen tissues.

Paired acute and convalescent serum samples should be collected to diagnose acute disease. Serology is also helpful in surveillance and to confirm the absence of infection before vaccination.

Recommended actions if equine viral arteritis is suspected

Notification of authorities

Equine viral arteritis is a reportable disease in many states. Each state should be checked for specific regulations.

Federal Area Veterinarians in Charge (AVIC):

https://www.aphis.usda.gov/aphis/ourfocus/animalhealth/contact-us/CT_Vet_Acred_Asst_DD

State Animal Health Officials (SAHOs):

http://www.usaha.org/upload/Federal%20and%20State%20Health/StateAnimalHealthOfficials_rev.pdf

Control

Acutely infected horses should be isolated to prevent transmission in secretions and excretions. Precautions should also be taken to avoid spreading the virus on fomites. EAV is readily inactivated by detergents, common disinfectants and lipid solvents. No specific treatment is available; however, most healthy horses other than young foals recover on their own. Good nursing and symptomatic treatment should be used in severe cases. Vaccination can also help contain outbreaks.

Venereal transmission can be controlled by good management and vaccination. To protect pregnant mares from abortion, they should be separated from other horses and maintained in small groups according to their predicted foaling dates. Newly acquired horses should be isolated for 3 to 4 weeks. Vaccination appears to prevent uninfected stallions from becoming long term carriers. Stallions that are not carriers should be vaccinated before the start of the breeding season. Prepubertal colts are given the vaccine when they are 6-12 months old. Carrier stallions are identified and bred only to well vaccinated or naturally seropositive mares. Similarly, semen that contains EAV should be used only in these mares. Because first-time vaccinates may shed field viruses for a short time after exposure, these mares should be isolated from seronegative horses, particularly pregnant mares, for three weeks after breeding. Naturally infected mares and those that are not first-time vaccinates are isolated for 24-48 hours, to protect other horses from the viruses present in semen.

Carrier stallions should be housed where they can be physically separated from uninfected horses; in one case, stallions apparently became infected by indirect exposure to semen. However, this appears to be rare. EAV is sensitive to sunlight and low humidity, and uninfected stallions have been kept near carriers for years without infection. Excellent hygiene and decontamination of fomites should be practiced when breeding infected horses or collecting semen. Although some experimental methods may be promising, there is no proven method to eliminate carriage in stallions.

Mandatory notification, annual testing, the identification of carrier stallions and control of transmission

Equine Viral Arteritis

from these animals, and selective vaccination are being used in New Zealand to eradicate equine viral arteritis.

Public Health

There is no indication that equine arteritis virus can infect humans.

Internet Resources

Food and Agriculture Organization of the United Nations (FAO). Manual for the Recognition of Exotic Diseases of Livestock

<http://www.spc.int/rahs/>

The Merck Veterinary Manual

<http://www.merckvetmanual.com>

USDA APHIS. Equine viral arteritis

http://www.aphis.usda.gov/animal_health/animal_diseases/eva/

World Organization for Animal Health (OIE)

<http://www.oie.int>

OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals

<http://www.oie.int/international-standard-setting/terrestrial-manual/access-online/>

OIE Terrestrial Animal Health Code

<http://www.oie.int/international-standard-setting/terrestrial-code/access-online/>

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