A perspective on equine viral arteritis
(infectious arteritis of horses) *

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Summary: The authors examine equine viral arteritis, emphasising occurrence
of the disease, distribution of the causal agent, clinical features, modes of
transmission, pathogenesis, the carrier state, diagnostic tests and control
measures including vaccination.

KEYWORDS: Arterivirus – Equine viral arteritis – Horse diseases –
Reviews – Viral diseases.

INTRODUCTION

The causative virus of equine viral arteritis (EVA) was first isolated in 1953, during
an outbreak of respiratory disease and abortion on a Standardbred breeding farm near
Bucyrus, Ohio, in the United States of America (USA) (2). The disease probably
existed in the horse populations of some countries for at least a century before that
occurrence.

International awareness of the disease increased in 1984, following an epizootic on
Thoroughbred farms in Kentucky, USA. Several earlier serological surveys had shown
that, while the presence of virus antibodies was widespread, few outbreaks of EVA had
been reported (11). Outbreaks of the disease have since occurred in the USA,
Switzerland, Austria, Poland, Canada, Italy, France and the United Kingdom (UK).
Sero logically positive animals have been found in most countries where testing has
been carried out. In many countries, there is a low level of infection among
Thoroughbreds but a high level in Standardbreds. Approximately 80% of Standardbred
stallions in the USA and in certain European countries are serologically positive.
Outbreaks of EVA have involved Standardbred, Thoroughbred, Arabian, Hanoverian,
Warmblood and Saddlebred breeds, as well as mixed breed horses and ponies.
Antibodies have also been found in donkeys and zebra.

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The causal agent of EVA, equine arteritis virus (EAV), is an enveloped, positive-stranded ribonucleic acid (RNA) virus of 45-70 nanometres (nm) in diameter. On the basis of physicochemical properties and virion architecture, EAV was originally classified as a Togavirus. However, it has since been shown that EAV is similar in terms of genetic structure and replication strategy to the virus of porcine reproductive and respiratory syndrome, lactate dehydrogenase-elevating virus of mice and simian haemorrhagic fever virus. These four viruses are now classified as Arteriviruses (1).

Horses affected with EVA may exhibit some or all of the following clinical signs: pyrexia; anorexia; oedema of the limbs, ventral trunk, scrotum, prepuce and periorbital regions; lachrymation; conjunctivitis; rhinitis; icterus; focal or diffuse urticaria; dyspnoea; coughing; submandibular lymphadenopathy; weakness and ataxia. Abortion may occur, as well as death in neonatal or older foals from interstitial pneumonia or pneumo-enteritis (2, 11).

The incubation period can range from three to thirteen days and varies, inter alia, with route of exposure, e.g. infection via the respiratory route can occur within 48-72 hours, whereas infection by the venereal route can require up to thirteen or fourteen days, with an average of seven days. In addition, a larger dose of virus may shorten the incubation period.

Abortion may occur with or without preceding clinical evidence of infection in the mare, and some mares which are severely ill with EVA do not abort (11). The abortion rate among susceptible pregnant mares can range from 10% to 70% of those that become infected. There is no evidence that mares can abort more than once due to EAV infection. The stage of pregnancy does not appear to influence the occurrence of abortion; foetuses from two to three months to full term are susceptible to infection. Abortions occur late in the acute phase or early in the convalescent phase of the infection.

Stallions affected with EVA may experience a reduction in sperm motility and an increased percentage of abnormalities in the sperm. This can last for 16 to 18 weeks after recovery from the acute phase of the infection. These changes are probably not due to the direct action of the virus, but are more likely to be secondary to the pyrexia and preputial and scrotal oedema.

The morbidity rate in outbreaks of EAV infection in adult animals varies greatly and depends on different virus, host and environmental factors, including virus dose, the age of the horse, intercurrent stress or disease and the time of year. Outbreaks of EVA at racetracks, where horses come into close contact and are subjected to the stress of training, can be associated with a relatively high morbidity rate. In some horse populations, clinical evidence of infection is seldom seen, even though infection rates may be high.

The course of the disease after experimental infection with the experimentally derived pathogenic variant of the Bucyrus strain of EAV is usually much more severe than that seen in cases of natural infection with the virus (6, 11). As many as 50% to 60% of mature horses may die after inoculation with this virulent variant of the Bucyrus strain. Deaths are associated with acute vasculitis, especially of the gastrointestinal tract.

EAV causes widespread necrotising arteritis affecting the smaller arteries and other blood vessels, and resulting in oedema and haemorrhage in many organs, as well as
infarctions in the intestine and lungs (4). The virus also causes widespread inflammatory lesions in several organs. Death of the foetus may result from in utero infection or from severe vasculitis of the placenta. Congenital infection of the foal occurs uncommonly. The virus can rarely, but apparently with increasing frequency, give rise to severe disease in very young foals, in which EAV causes a fulminating interstitial pneumonia or pneumo-enteritis (11).

During the acute phase of the infection, the greatest amount of virus is usually shed via the respiratory tract for 2 to 16 days after infection (6). Virus can also be shed in the urine for up to three weeks, but not as consistently as from the respiratory tract. While virus has been found in the kidney as late as day 28, this is considered to be exceptional. Virus is found in the blood macrophages for 1 to 21 days (with a mean of 7 days) during the acute phase of the infection. In the mare, virus can be found inconstantly in the uterine and vaginal secretions for 2 to 9 days after infection. Stallions in the acute phase of the infection shed virus in semen in high concentrations. Virus shedding may continue after the acute phase is over and last several weeks or months or many years (9, 11).

EAV is spread by respiratory and venereal routes (6, 11). EAV can also be transmitted by fomites. Equipment such as halters and twitches and the clothes and skin of the handler can all become contaminated. The chronically shedding stallion is probably the most important source of infection; infected teaser stallions or newly introduced nurse mares are less likely sources but have sometimes been implicated. The less physical contact between horses, the less risk of exposure by the respiratory route.

While only one serotype of the virus is recognised, there is evidence of genomic and antigenic variation between strains (11). Genomic variation has been observed among strains isolated in North America and Europe. There may be differences in pathogenicity among strains. Some strains of EAV are associated with more severe disease than others, and some appear to be more abortigenic. All strains are immunologically cross-reactive. There is no evidence for the emergence of more highly pathogenic strains in recent years. Some countries can trace the occurrence of infection to the introduction of imported animals (e.g. Australia, New Zealand, South Africa and Chile). The role of donkeys and zebra in the maintenance and spread of the virus is not yet established, nor is the virus currently recognised as a cause of naturally occurring disease in these species.

As many as 30% to 55% of stallions can become chronic carriers of EAV. Virus is found in several accessory sex glands of the male reproductive tract. Three categories of carriers have been defined, based on duration of virus persistence:

- short term for 2 to 5 weeks after acute infection
- medium term for 3.5 to 7 or 8 months after acute infection
- long term for at least 11 years.

Establishment and maintenance of the carrier state is androgen-dependent (11). Virus persistence does not occur in prepubertal colts, nor in mares or geldings. There is no evidence of intermittency of virus shedding, e.g. shedding associated with seasonal fluctuations in serum testosterone levels. Virus can be eliminated from the reproductive tract of persistently infected stallions following castration. Virus does not persist in the testis or epididymus and fertility appears unaffected in carrier stallions. All carriers are serologically positive, though some may have low antibody titres. Not
all stallions that are serologically positive, however, are necessarily persistently infected with EAV. Efficiency of transmission by way of semen of carrier stallions to seronegative mares is 85% to 100%.

EVA is difficult to diagnose on the basis of clinical signs alone, as many of these signs are similar to those seen in other respiratory infections, so laboratory confirmation is essential (11). The appropriate diagnostic tests to use are listed in the OIE Manual of Standards for Diagnostic Tests and Vaccines (8). Virus can be isolated from various organs and secretions. Virus isolation from semen using rabbit kidney (RK-13) cells can, in experienced hands, identify over 99% of persistently infected stallions detected by test breeding suspect carrier stallions to seronegative mares. Serological surveillance can be conducted using the virus neutralisation test or enzyme-linked immunosorbent assay (ELISA). Freedom from infection of a country or region is difficult to establish because of the frequency of movement of many horses around the world. Japan is currently considered free of the virus.

In considering measures to prevent or control this infection, a balance has to be struck between facilitating the movement of horses and minimising the risk of spread of the virus. The importation of carrier stallions or infective semen has been a source of infection for outbreaks of EVA in some countries. Disease in eventers and performance horses caused by inter-country trade has not been commonly observed but has occurred. Codes of practice have been developed by the industry in some countries, e.g. France, Germany, Ireland, Italy and the UK, with recommended procedures for controlling EVA. It is advisable that serologically positive stallions be kept isolated until diagnostic tests are carried out to detect virus in semen. Serologically positive mares are perceived as less of a risk as they do not become chronically infected with the virus. The import regulations of different countries vary greatly; some do not permit the introduction of any serologically positive animals, while others exclude only serologically positive stallions, or accept such stallions based on confirmation of their non-carrier status by attempted virus isolation in cell culture from semen or by test breeding to seronegative mares. Serologically positive mares are sometimes accepted if it can be shown that the titre to EAV of these animals is stationary or decreasing.

A modified live virus (MLV) vaccine against EVA is commercially available in North America (3, 5). An inactivated vaccine is available for controlled use in stallions and mares in Ireland and the UK. The MLV vaccine was first used in 1984 in the USA in response to an extensive outbreak of EVA in Kentucky (11). Under controlled experimental conditions, there was no evidence that the vaccine virus was shed for up to five weeks after vaccination in stallions. Furthermore, mares bred to these stallions did not seroconvert (7, 10). With the exception of the first year of use of the vaccine, it has been mandatory to vaccinate all Thoroughbred breeding stallions in the states of Kentucky and New York in the USA since 1986. There have been no safety problems following use of this vaccine in stallions and non-pregnant mares. Based on the screening of 151 Thoroughbred stallions, there has been no evidence of vaccinal virus shedding, either by attempted virus isolation from semen or by serological testing of the mares bred to these stallions. There appears to be no evidence of localisation of the virus in the male reproductive tract and no evidence of recombination with field strains of EAV (11). Vaccinated stallions develop high antibody titres to the virus. Although vaccination is not recommended for foals under six weeks, the vaccine has been used in this age group with no untoward effects. Vaccination is also not recommended for use in pregnant mares, especially in
the last two months of pregnancy, because of one experimental case where vaccine virus was recovered from a weak, newborn foal after vaccination of the dam during the final stages of pregnancy. Vaccination of pregnant mares has been carried out in the field where there was high risk of natural infection with EAV, with no clinical sequelae. The recommendations are for vaccination of serologically negative stallions to prevent establishment of the carrier state. Mares should be vaccinated three to four weeks before breeding to an EAV-shedding stallion, and such mares should be isolated for a further period of three weeks after being bred to a carrier stallion for the first time. There is no evidence that vaccination of chronically infected stallions will aid elimination of the carrier state. An inactivated vaccine has been produced in Japan but has only been evaluated under experimental conditions.

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Résumé : Les auteurs étudient l’artérite virale équine en mettant l’accent sur la fréquence de la maladie, la répartition géographique de l’agent de l’artérite, les caractéristiques cliniques, les modes de transmission, la pathogénie de l’affection, le portage du virus, les épreuves de diagnostic et enfin les mesures de prophylaxie dont la vaccination.


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Resumen: Los autores examinan diversos aspectos de la arteritis viral equina, en especial la frecuencia de la enfermedad, la distribución del agente causal, las características clínicas, los modos de transmisión, la patogénesis, el estado de portador, las pruebas de diagnóstico y las medidas de control incluyendo la vacunación.


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REFERENCES


