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Equine viral arteritis: Current status and prevention

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Abstract

Recently, there has been increased interest in equine viral arteritis (EVA) among veterinarians and horse owners. Outbreaks of the disease were identified initially in New Mexico, USA in 2006, and in the Normandy region of France in the summer of 2007. Both occurrences were associated with AI of cool-shipped semen. Each was linked to respiratory illness, neonatal death, abortion, development of carrier stallions, and cancellation of equestrian events. In light of the increased interest, this paper will present a brief case history, followed by a review addressing common concerns regarding EVA, current status, and control and prevention strategies, including vaccination, and recommended bio-security measures.

Keywords: Equine; Equine arteritis virus; Persistent infection; Transmission; Prevention

1. Case history

Early in the first week of June 2006 on an equine stud farm in New Mexico, USA, early pregnancy losses were detected in mares previously confirmed pregnant. The problem continued, and on June 16 the farm manager contacted the Maxwell H. Gluck Equine Research Center, University of Kentucky, to discuss probable causes [1]. Equine viral arteritis (EVA) was suggested as a likely cause of the abortions. Of the four stallions standing at this farm, the first developed fever, depression and dependant edema, especially of the scrotum and hind limbs. His fertility decreased, and he failed to impregnate any mares for the remainder of the season. Equine arteritis virus (EAV) was subsequently isolated from the semen of this stallion. Shortly thereafter, the stud manager reported that a second stallion had developed pyrexia; however, his fertility remained adequate to impregnate mares. Virus-positive semen was shipped from this stallion to multiple states within the United States prior to and during the second and third weeks of June and before the diagnosis of EAV infection by the Maxwell H. Gluck Center on June 26, 2006 [1]. During this time, two other stallions became infected. Stallion 3 developed a short-lived fever without substantial signs of disease, whereas Stallion 4 had neither fever nor clinical signs of disease. Both stallions subsequently tested positive for EAV in their semen. In early September 2006, Stallion 1 died from complications associated with laminitis. Stallion 2 remained persistently infected, shedding virus in his semen through January 31, 2007, tested negative on multiple virus isolation attempts in March 2007, and had three negative test breedings in July 2007. Stallion 3 tested positive through January 9, 2007, tested negative on multiple virus isolation attempts in February 2007, and had three negative test breedings in July 2007. Stallion 4 remains persistently infected, shedding

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infectious virus in his semen as of December, 2007 [Murray J. personal communication]. Also associated with the outbreak were 30 abortions, involving approximately half of the exposed pregnant mares on the farm with the index case.

The virus was disseminated to farms in 18 states, either via cool-shipped semen (48 mares) or mare transport (20 mares and foals) before the spread was controlled by quarantine and close surveillance [1]. A substantial spread point occurred in Utah via cool-shipped semen transport and subsequent animal movement; this resulted in respiratory illness, abortion, neonatal pneumonia, and death [1]. These outbreaks were finally controlled by quarantines and close surveillance.

2. Introduction

An extensive outbreak of equine viral arteritis that occurred in Kentucky Thoroughbreds in 1984 generated widespread interest, publicity and concern [2–5]. A number of other outbreaks of the disease have since been reported from North America and Europe [5–10]. Similarly, equine arteritis virus infection of horses has been identified in countries including Australia, New Zealand, and South Africa, previously thought to be largely or completely free of the virus [11–14]. Serological surveys have shown that EAV infection occurs among horses in North and South America, Europe, Australasia, Africa, and Asia [15], with considerable variation in seroprevalence of EAV infection among countries and within equine populations in some countries.

In the summer and fall of 2006, related to the use of cool-shipped semen for AI, EVA was identified in New Mexico and five other states, in association with abortion, respiratory disease, neonatal illness, and the development of the carrier state in an additional number of American Quarter Horse stallions (numerically the largest equine breed in the USA) reported at the 2006 Annual Meeting of the United States Animal Health Association, ''of overriding importance was the ease with which infection was very effectively spread among an immunologically naïve population through the use of semen from a stallion acutely and later, persistently infected with EAV. This occurrence of EVA was the first in which there was widespread dissemination of EAV in Quarter Horses, a breed essentially not previously exposed to this virus'' [1]. Most recently, in the summer and fall of 2007, the disease occurred on breeding farms in the Normandy region of France including the national stud, the Haras du Pin, where the infection was first diagnosed in a Percheron stallion. Within a week, other stallions developed clinical signs of the disease. This outbreak, which was also linked to AI of cool-shipped semen, eventually affected 26 farms, resulting in illness, neonatal death, abortion, development of persistently infected stallions, and cancellation of equestrian events [16]. The following review will address some of the common concerns with regard to EVA, its etiology, epidemiology, pathogenesis, pathology, clinical findings, diagnosis, transmission risks, control and prevention, including vaccination and recommended bio-security measures.

3. Etiology

Due to the distinctive inflammation in the muscle wall of small arteries in the acute phase of the infection, the virus is called equine arteritis virus and the disease it causes, EVA [15–17]. Equine arteritis virus is the prototype virus in the family Arteriviridae (genus Arterivirus, order Nidovirales), which also includes porcine respiratory and reproductive syndrome virus (PRRSV), lactate dehydrogenase-elevating virus, and simian hemorrhagic fever virus [18,19]. It is an enveloped, single-stranded, positive-sense, RNA molecule (Fig. 1) [19,20]. There is only one known serotype of EAV, but geographically and temporally distinct strains of EAV differ in the severity of the clinical disease they induce and in their abortigenic potential [15,21–29]. Strains of EAV from North America and Europe share at least 85% nucleotide identity; following phylogenetic analysis, these viruses generally segregate into North American and European geographical groups.

Survival of EAV is temperature dependant; although it may survive only 20–30 min at 56 °C and from 2 to 3 d at 37 °C, it can survive up to 75 d at 4 °C. Tissue culture fluid or tissue samples containing EAV can be stored at −70 °C for years without loss of infectivity [30]. However, the virus is readily inactivated by lipid solvents (ether and chloroform) and by common disinfectants and detergents [31].

4. Epidemiology

Although EVA is a disease almost exclusively of equids, antibodies to EAV have been identified in donkeys in South Africa [12,13] and in the USA [32]. Clinical, virological and serological responses of donkeys have been determined following intranasal inoculation with the KY-84 strain of equine arteritis virus. Equine arteritis virus has also been reported associated with a case of abortion in an alpaca in