Foreign Animal Diseases with Equine Potential

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The amount of damage that a foreign animal disease will cause is directly proportional to the time between introduction and accurate diagnosis. In other words, we have to accurately recognize a foreign animal disease at first blush if we are to implement effective control measures. It is imperative that practitioners consider foreign animal diseases in their diagnostic rule-outs. Remember, “When you hear hoofbeats on the covered bridge, please think about the zebra!” Author’s address: College of Veterinary Medicine, University of Georgia, Athens, GA 30602-7388. © 2002 AAEP.

1. African Horse Sickness

African horse sickness (AHS) is an acute to subacute systemic illness primarily affecting horses. Mules and donkeys can also be infected but suffer a much less severe form of the disease. Dogs become infected by consuming meat from infected carcasses. The causative agent is AHS virus, an orbivirus, in the family Reoviridae. Transmission is through the bite of an infected Culicoides insect. The disease is currently restricted to Africa and is endemic in the dry tropical areas on either side of the equator, where it is maintained by prolonged, inapparent viremias in zebras. Periodically, the virus makes incursions into new areas, often with devastating results. The most recent example was a 4-yr epizootic in the Iberian peninsula, where it was introduced through the importation of viremic zebras. The disease persisted in Culicoides insects from 1987 until it was eradicated in 1990, but only after hundreds of horses had succumbed to the disease.

Incubation period is usually 3–5 days. Horses may present with a number of clinical signs, or disease may be largely inapparent until just before death. Animals are febrile and often depressed. Interestingly, colic is frequently noted. Characteristic supraorbital edema (Fig. 1) is seen in only about 10% of cases. Death occurs as a result of respiratory or cardiac failure. At post-mortem, there may be minimal gross lesions. Edema is often seen around the area of the ligamentum nuchae; however, this is an area that is rarely dissected during a routine post-mortem examination. Modest intermuscular edema may be seen anywhere in the carcass. Likewise, petechiation may be seen in many locations. Approximately one-half of the animals will have pulmonary edema to some extent (Fig. 2).

The pathogenesis of the disease is related to the virus’ ability to replicate in endothelial cells, especially those of lung and heart. Dysfunctional endothelium allows edema fluid to seep out in the case of the lung and focal necrosis of myocardium in the case of the heart. Histologic lesions are often very subtle.

Diagnosis of AHS can be problematic. Because the disease presents with such a myriad of non-specific clinical signs, AHS is not likely to be high on the list of rule-outs in non-endemic areas. Similarly, post-mortem lesions are inconsistent and without an a priori suspicion of AHS, the diagnosis...
will likely be missed at necropsy. Also, histologic lesions tend to be subtle and non-specific. Fortunately, the virus is easy to isolate. Whole blood from a live animal or spleen from a dead animal are the best specimens to submit to the diagnostic laboratory for isolation. Serology is also helpful except in acute cases.

The United States remains at risk for incursion of AHS. It is unknown if our own Culicoides vectors are competent vectors of the virus, because this specific research has never been done. However, based on what is known about orbiviruses in general, and abilities of various Culicoides to transmit, it seems likely that the virus could be transmitted by our indigenous Culicoides species. The U.S. quarantine restrictions regarding equine species are designed to protect against introduction of an infected equine. Consequently, the most likely route of entry would be from an infected insect that comes in through air travel.

Fig. 1. Edematous swelling in the supraorbital fossa occurs in about 10% of horses with African horse sickness.

Fig. 2. At post-mortem, most horses infected with African horse sickness virus will have some evidence of pulmonary edema.
2. Glanders

Glanders is a contagious disease primarily of horses and donkeys caused by Burkholderia (formerly Pseudomonas) mallei.\(^2\,^3\) The disease has great importance in history, because it was the scourge of cavalry aggregations and was used maliciously during World War I to infect the horses of enemy troops. Glanders was officially eradicated from the U.S. in 1937, and currently the disease is restricted to Asia and some parts of eastern Europe.

*B. mallei* is a gram-negative aerobic bacteria that gains entrance into the horse through ingestion or aspiration of material contaminated with exudates. The most commonly described means of transmission is through the water trough or feed bunk where bacteria from nasal exudates can accumulate to infect more animals. It should be noted that this bacteria will also infect and cause disease in humans.

The incubation period can be weeks to months. Animals present with fever and then signs referable to the point of entry. If entry is through the pharyngeal area, there may be severe rhinitis and pneumonia. If entry is through a skin abrasion, animals usually present with suppurative inflammation of draining lymphatics. The organism tends to produce pyogranulomas and multiple nodules, which often spread through lymphatics, creating “cords.” When these occur in the skin, they are referred to as “farcy pipes.”

Diagnosis in areas of the world where this disease is endemic is easily accomplished through clinical and pathologic findings, with confirmation by bacterial culture. In a non-endemic area, disease might be mistaken for strangles (*Streptococcus equi*), rhodococcal pneumonia, or ulcerative lymphangitis (*Corynebacterium pseudotuberculosis*).

Although *B. mallei* is susceptible to a number of antibiotics, treatment is usually not undertaken. In the interests of disease control and eradication, affected animals are usually destroyed.

3. Hendra Virus

Hendra virus first emerged in 1994 at two racing stables in Australia, where it caused illness and death in both horses and humans. The virus was subsequently identified in two other incidents in 1995 and 1999. To date, there have been 17 equine and 2 human deaths from this newly described virus.\(^4\)

Hendra virus was originally called equine morbillivirus because of sequence similarities to other viruses in this genus. However, subsequent investigations revealed that it is significantly different, and now this virus, along with the also emerging Nipah virus, has been placed in a new genus in the family Paramyxoviridae.

Transmission seems to be by ingestion or inhalation. The virus reservoir is large fruit bats of the genus *Pteropus*, also known as “flying foxes.” These arboreal mammals are not clinically affected by the virus, but a significant serologic prevalence indicates widespread infection. It is thought that the virus is spread through urine or uterine secretions and that increasing urbanization caused by habitat destruction has brought these animals and their viruses into much closer contact with humans and domestic animals.

In all three outbreak cases, the virus did not spread beyond the initial focus, so it is probably not contagious from horse to horse. The two humans who died from this disease had very close contact with the horses—one was involved in nursing care and the second was probably infected through splashing of fluids at necropsy. Consequently, there are risks to humans from close contact with infected horses.

In horses, the incubation period is 5–10 days. Period of disease is short, approximately 2–3 days, followed by death or recovery. Clinical signs are related predominantly to pneumonia, but in some horses, neurologic signs have been evident. The most dramatic post-mortem findings are severe pulmonary edema and vascular congestion.

The virus replicates in endothelium throughout the body but especially in the lung. Syncytial cells are present in histologic sections and may help with the diagnosis.

The probability of Hendra virus entering the United States is minimal. However, the same urbanization forces at work in Australia are also operating on our continent. It is estimated that we know less than 10% of all the bacteria and viruses present in any of our animal species—and normal flora in one when transmitted to another may cause an emerging disease. We are constantly placing new species into evolutionarily incompatible zones and are always at risk of the development of new disease.

4. Epizootic Lymphangitis

Epizootic lymphangitis is a rare chronic disease of horses, present in Africa, Asia, and Eastern Europe. The causative agent, *Histoplasma farcinosum*, is a dimorphic fungus, with mycelial form present in soil and yeast form in the lesion. Most cases occur secondary to contaminated soil coming in contact with a dermal abrasion, although biting flies have been incriminated in taking the organism to mucosal surfaces, especially conjunctiva.\(^5\)

Clinical signs consist of a chronic ulcerative dermatitis, most commonly on the extremities, although as mentioned above, peri-conjunctival area may also be affected. Lymphatics frequently become filled with inflammatory exudates and cells, resulting in “cording.” Granulomatous or purulent nodules forming along these lymphatics may rupture. Infrequently, animals present with pneumonia because of inhalation of the organism. In this case, pulmonary lesions are multifocal purulent nodules or more extensive lobar involvement.
Diagnosis of the disease rests on laboratory confirmation. The yeast organisms can be readily visualized in direct smear or histologic section, appearing as pleomorphic, 2- to 5-μm organisms surrounded by a halo. These yeasts occur both within macrophages and in extracellular locations. Because the discharges are largely responsible for contaminating the environment with fungus, treatment is not usually undertaken. Rather, infected animals are destroyed. In the case of very valuable animals, surgical curettage may be done, with careful attention paid to the disposal of lesion material. Subsequent to treatment, lesions may recur as long as 1 yr later.

The probability of epizootic lymphangitis appearing in a domestic horse in the United States is extremely slim. It is possible that the disease could be imported in a live horse; however, the chronic nature of the disease makes this unlikely, because clinical signs or lesions would be noticed, if not before export, then hopefully during the quarantine period. If diagnosed within the United States, prompt and radical attention should be paid to the environment to prevent this fungus from becoming established.

5. Trypanosomal Diseases

There are three trypanosomal diseases of horses and all of them are foreign to the United States—dourine, surra, and nagana.2,6

Dourine
Dourine, caused by Trypanosoma equiperdum, is currently present in many parts of the world, predominantly Asia, South America, and Europe. It has been eradicated from the United States. Dourine is a venereal disease that may become systemic. Organisms reside in the urethra of infected stallions and in the vaginal discharge of infected mares. In the early stages of the disease, genitalia may be swollen and indurated. Subsequently, characteristic skin plaques develop. These plaques are referred to as “silver dollar” lesions because of their characteristic size and shape. The disease may progress to CNS involvement, including weakness, incoordination, and paralysis.

Treatment is not usually undertaken. Although the organism is susceptible to certain trypanocidal drugs, these tend to favor the production of a carrier state, and so euthanasia rather than treatment is often the preferred option. The most likely mode of entry for dourine into the United States would be in an infected animal. Because all imported animals are examined for signs of disease, incursion is highly unlikely.

Surra

Trypanosoma evansi is the causative agent of surra, a disease that affects horses and other equidae, as well as cattle, buffalo, and camels, in parts of Asia, Africa, and South America. Transmission is through biting flies, although vampire bats may serve as vectors as well. Clinical signs may be quite nonspecific and consist of malaise, weakness, anemia, and ultimately, death. At post-mortem, there may be lymphoid hyperplasia and occasionally meningoencephalitis. Because of the nonspecific nature of the disease, laboratory confirmation is essential. Anti-trypanocidal drugs may be used for treatment in endemic areas. Without treatment, the mortality rate is 100%. Introduction of T. evansi into the United States would most likely be through an infected animal. Because this is a vector-borne disease, immediate recognition is imperative to institute sufficient control measure to keep flies from becoming infected and disseminating the disease.

Nagana

Nagana, or African animal trypanosomiasis (AAT), is caused by three different trypanosome species—Trypanosoma congolense, Trypanosoma vivax, and Trypanosoma brucei brucei. All are transmitted through the bite of the tsetse fly (Glossina sp), although T. vivax may be moved from animal to animal by biting flies. AAT is prevalent throughout sub-Saharan Africa, and although primarily a disease of cattle, the parasites will also infect and cause disease in a number of other hoofed stock, including horses. Clinical signs are similar in all—weakness, lethargy, and anemia. Pathologic lesions are nonspecific. Confirmation of the disease requires laboratory testing. Introduction of any of these agents of AAT into the United States is unlikely. Establishment of the disease within our borders is even less likely because of the absence of the primary fly vector (tsetse fly).

6. Venezuelan Equine Encephalitis

Of the three equine encephalitides—Eastern, Western, and Venezuelan—only the latter is a foreign animal disease. The last incursion of Venezuelan equine encephalitis (VEE) in the United States was in 1971, but its continued presence in northern South America and periodic outbreaks in Mexico are worrisome. VEE is clinically similar to its endemic relatives, Eastern and Western equine encephalitis.2,7 Diffuse cerebral involvement results in a variety of clinical signs, usually progressing to dementia and recency. Herd morbidity tends to be very high with VEE and mortality ranges from 40% to 90%. Effective vaccines exist for the equine encephalitides. Definitive diagnosis requires viral isolation in the laboratory or demonstration of an acute rise in antibody titer specific for VEE. If the disease were introduced into the United States, it would be very important to effectively quarantine affected horses to decrease spread, because horses are important amplifiers of the virus, and the high level of viremia is effectively carried to other susceptible animals through hematophagous insects, primarily mosquitoes.
7. Conclusion

There are two ways a veterinarian can become “famous” when a foreign animal disease enters the neighborhood. One way is to diagnose it; the other way is to miss it! The amount of damage that a foreign animal disease will cause is directly proportional to the time between introduction and accurate diagnosis. It is imperative that we accurately recognize potentially foreign animal diseases when they first present to practitioners in the field if we are to implement effective control measures. Practitioners should consider foreign animal diseases in their diagnostic rule-outs and not hesitate to seek the assistance of their state or local federal veterinarian or a FAD (foreign animal disease) diagnostician. Remember, “When you hear hoofbeats on the covered bridge, please think about the zebra!”

References