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Potomac Horse Fever in Texas

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Potomac horse fever (PHF) is an infectious, noncontagious, sporadically-occurring and seasonal disease of unstressed pastured or stabled horses. Other names for this disease are equine monocytic ehrlichiosis, acute equine diarrheal syndrome and equine ehrlichial colitis. Ticks are theorized to be the vector for PHF spread. Currently the disease has been diagnosed in 22 states (including Texas) and one Canadian province. According to the Texas Veterinary Diagnostic Laboratory (TVMDL), clinical disease signs and serum antibody evidence for PHF have recently occurred in six herds in at least three counties (McLennan, Johnson and Taylor) of Central, North Central and West Central Texas.

Cause

A bacterium-like microorganism called a rickettsia is the cause of PHF. The name given this rickettsia is *Ehrlichia risticii.* Another ehrlichial microorganism, *E. equi,* has been documented to cause disease in horses in the United States. Other familiar rickettsial diseases in the U.S. are Rocky Mountain spotted fever and canine ehrlichiosis.

Transmission

Classic rickettsial diseases such as Rocky Mountain spotted fever and canine ehrlichiosis are transmitted by ticks. Because of the noncontagious and warm weather seasonal incidence (most cases in July and August) of PHF, blood-sucking arthropods are incriminated (not proven) in *E. risticii* transmission. As with any hemoparasitic disease, spread by blood-contaminated needles is always a possibility. Once a horse is infected and survives, a carrier state and continuous infection source may be established for at least 8 months. However, attempted transmission of PHF with blood from a recovered horse has been unsuccessful. Significantly more PHF has been found associated with horses: stabled in barns where the disease had occurred previously; pastured with cattle and kept on farms with dogs; pastured on lush forage with the known abundance of rodents; and horses bedded in sawdust. Although a positive association between these factors and possible tick transmission is tempting to make, their role is still under investigation. One recent study has partially disproven transmission of PHF by the American dog tick (*Dermacentor variabilis*). Experimental evidence is available to disprove fecal-oral transmission of PHF. This same experimental work has been used to strenghten the argument for arthropod-borne transmission.

Natural Disease Pattern

"Typical" cases display initial fever (102° to 106° F), depression and anorexia followed by colic, diarrhea, edema of the legs and ventral abdomen and/or laminitis (20 to 30 percent rate usually within 3 days of initial diarrhea). "Atypical" cases may be inapparent or display signs varying from only initial fever, depression and anorexia to severe colic, profuse diarrhea and/or laminitis. Although the attack rate of PHF per herd is usually low, the case mortality rate may be as high as 25 to 30 percent.

Disease Pattern from Experimental Infection

In experimental *E. risticii* infections in which horses display clinical disease signs, biphasic temperature elevation begins 7 to 10 days after inoculation with blood from diarrhea-stage, infected

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animals. Depression, anorexia, diarrhea and slight edema of the distal portion of the hind limbs were present by day 12 post inoculation (PI). Reisolation of *E. risticii* was accomplished on days 9, 10, 12, 13 and 16 PI. Decreased leukocyte and platelete counts were followed by increased leukocyte and rebound platelete count at day 15 to 20 PI. Indirect fluorescent antibody (IFA) serum titers of 1:20 to 1:80 occurred by days 10 to 12 PI and were 1:320 by PI day 15. IFA antibody titers 294 days PI were still 1:80. Stomach ulceration and edema below the linings (mucosa) of the small and large colon were gross post-mortem findings seen in one horse sacrificed 16 days PI (after diarrhea developed). Another experimentally infected horse was sacrificed on day 13 PI, after onset of clinical signs without diarrhea. On gross post-mortem examination, the stomach, small intestine (SI) cecum and large and small colon contained contents of watery consistency. The feces in the terminal small colon were normal. Ulcerations of the mucosal surface of the stomach and large colon were present. Microscopic examination of tissue from the affected large colon revealed degenerative changes and characteristic signs of inflammation. Rickettsial-like organisms were tentatively identified by electron microscopy of colonic specimens containing gross lesions. A third experimentally infected horse died 20 days PI. Gross examination showed: several stomach ulcerations; four small areas of hemorrhages in the mucosa of the SI; lack of SI contents except mucus; diffuse reddening of the entire SI mucosa; and watery brown fluid in the large colon, cecum and small colon with two small cecal ulcers.

In experimental *E. risticii* infection in which a horse displayed no clinical disease signs, no temperature elevation occurred and attempted isolation of the organism was unsuccessful. A similar leukocyte, platelete and IFA titer pattern were the only evidence for *E. risticii* infection.

Experimental *E. risticii* infections in horses substantiate the fact that a range of inapparent infection to fatal disease does occur.

Diagnosis

Diagnosis is based on factors such as history, clinical disease signs, blood count, serum IFA analysis, *E. risticii* identification, gross post-mortem and microscopic findings, electron microscopy and elimination of other disease causes.

Affected horses may have a history that includes out-of-state origin or horse circuit travel. Almost 100 percent of normal equine sera recently screened for *E. risticii* antibodies by the TVMDL have been negative. However, several PHF herds in Texas seem to have their origin of infection within the state.

The variability of PHF clinical disease signs is an important point. Serum IFA analysis usually demonstrates infected herds may have many times more inapparent or less apparent cases than acute PHF cases. It is also more likely that premises having infected horses previously will continue to have more cases than premises never before having infected horses.

The previously discussed leukocyte and platelete values are a possible aid to diagnosis but are not specifically diagnostic.

The TVMDL currently offers an IFA test for PHF ($\geq$1 ml serum required). When a horse has clinical disease signs, a titer of $\geq$1:160 is thought to be diagnostic. Titers of $\geq$1:20 are thought to be positive for inapparent or less apparent cases of PHF.

A blood sample obtained during the initial diarrheal stage of PHF is the submission of choice for *E. risticii* identification. A citrate-type anticoagulant should be used to preserve the sample. Mice that develop *E. risticii* antibodies after intraperitoneal inoculation with leukocytes from a suspect blood sample indicate positive results.

Although the gross post-mortem and microscopic lesions have been previously described for uncomplicated experimental infections, lesions in naturally occurring cases may vary according to disease severity and stage and the presence of other infectious agents (i.e. salmonella).

As in experimental infection, electron microscopy might also be used to identify the *E. risticii* microorganisms involved in natural infection.

Salmonellosis, colitis X, intestinal clostridiosis and other conditions should be considered in a differential diagnosis of PHF. The TVMDL requests feces submission to help eliminate salmonellosis and other infectious causes.

Treatment

Antibiotic therapy is thought to be of most value in the acute stages of PHF. This is, of course, the most difficult time to make the correct diagnosis. No antibiotics are currently approved for treatment of PHF. Oxytetracycline has been tried but is not recommended because colitis and diarrhea become intensified in some cases. By the time a diagnosis is usually made, treatment to correct dehydration, shock and diarrhea are indicated. Horses recovered from initial infection are thought to be protected from reinfection for at least 13 months and usually return to previous performance levels except for laminitis complications. Appropriate preventive therapy for laminitis is a necessary consideration in every PHF case.

Prevention

Since little concrete data is available on PHF transmission, prevention recommendations are very general. Consideration of the blood-borne and seasonal nature of the disease emphasizes the control of ectoparasites on horses, other livestock and pets. Isolation of infected or suspect horses in combination with animal and premise ectoparasite control should minimize exposure to uninfected horses. Only sterile or sanitized needles should be used to give injections or collect blood. The use of blood-contaminated needles between horses or between other animal species and horses may lead to the increased incidence of PHF in Texas.

Summary

Horse owners should be aware of the characteristic pattern, various clinical disease signs and possible transmission factors associated with PHF. If a horse displays clinical signs suspicious of PHF, a veterinarian should be consulted for appropriate action. Information on the control of ectoparasites of livestock and pets is available from veterinarians or county Extension agents.

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