A case of *Ehrlichia equi* in an adult horse in British Columbia

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Ehrlichia equi is an obligate intracellular rickettsial parasite that infects granulocytic cells in horses and burros. Experimentally, dogs, goats, sheep, and nonhuman primates may be infected. The disease has its greatest prevalence in California, particularly northern California, where it occurs predominantly in the fall, winter, and spring, with the highest incidence in March. The exact mode of transmission is unknown, although ticks are thought to be the vector (1,2). In North America, the disease has been reported in Florida, Colorado, New Jersey, Illinois, and Washington (2–4).

In experimental infection, the incubation period ranges from 3 to 10 d. The disease is often mild and self-limiting in endemic areas, with horses recovering in 2 to 3 wk; no carrier state is apparent. The severity of the clinical signs tends to be age dependent, with horses older than 4 y exhibiting more severe clinical signs. The clinical signs are fever, depression, and anorexia, often accompanied by marked ataxia (2). There is usually severe hyperbilirubinemia, and petechiae can be present. Limb edema may occur, particularly in older horses. Many of the signs appear to be the result of a necrotizing vasculitis, which can be demonstrated histologically (1,2).

A 4-year-old, Thoroughbred cross mare was referred from Vancouver Island with a vague history of discomfort, thought to reflect colic. Upon arrival at the clinic, the horse was profoundly depressed and the mucous membranes were extremely icteric. The rectal temperature was 37.5°C. Rectal palpation revealed a mildly distended, nonpainful, pelvic flexure of the large colon. Her packed cell volume at this time was 0.26 L/L (normal, 0.32 to 0.53 L/L)(7). Intravenous fluids were given overnight, and blood was collected the following morning for a complete blood count (CBC) and chemistry panel. An abdominal tap was also performed. The horse at this time had a fever of 39.5°C and was restless. Differential diagnoses based on clinical findings were primary liver disease, equine infectious anemia, and hemolytic anemia (autoimmune, Heinz body).

Abnormal laboratory findings included a low hematocrit, 0.235 L/L, and a low total white cell count, 3.3×10^9/L (normal, 5.5 to 12.5×10^9/L), characterised by a neutropenia, 2.4×10^9/L (normal, 2.7 to 6.7×10^9/L) and lymphopenia, 0.8×10^9/L (normal, 1.5 to 5.5×10^9/L). The neutrophils did not exhibit toxic change. There was hyperbilirubinemia, 141 μmol/L total bilirubin (normal, 17 to 77 μmol/L); increased fibrinogen, 8.0 g/L (normal, 1.0 to 4.0 g/L); and mild hypoproteinemia, 57 g/L total protein (normal, 60 to 77 g/L). There were no changes in the liver enzymes. A review of the blood film revealed small basophilic cytoplasmic inclusions in approximately 6% of the neutrophils. These inclusions were consistent with the morulae of *E. equi* [Figure 1]. The morulae were also present in the neutrophils in the cytocentrifuge preparation of the abdominal fluid. The organisms varied from circular collections of small curved rods to individual or multiple rod-shaped structures scattered in the cytoplasm. The horse was treated that evening with oxytetracycline (Liquamycin LP, rogar/STB, London, Ontario) at a dose of 3 g, IV; by morning, the temperature was normal, and the horse was eating. The medication was continued for 4 d, when the horse was returned to the owner. A CBC and chemistry panel were repeated 3 mo after the initial illness, when all parameters were within normal reference range. Sera from the 1st laboratory specimen and from a sample taken 6 wk after the original illness were tested for antibodies to *E. equi* (University of California, Davis, California, USA) and were found to have titers of zero and a 1:80, respectively.

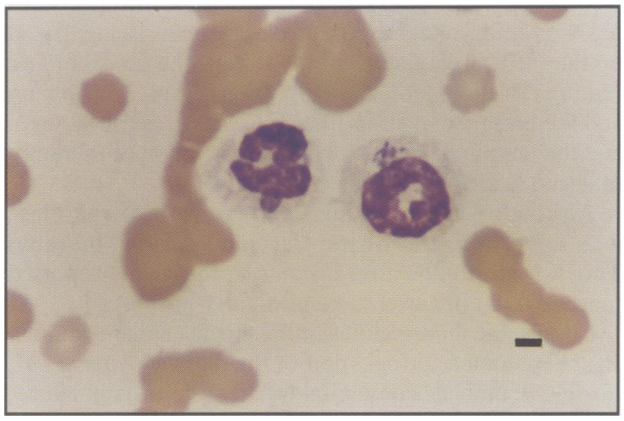

Figure 1. Neutrophils with morulae of *Ehrlichia equi*, one morula is intact, the other is breaking into individual elementary bodies. Wright-Geimsa stain. Bar = 3 μm.

This horse was native to British Columbia and had not travelled outside the province. She developed the infection in March, which is comparable to the seasonal peak seen in California. She was initially diagnosed as suffering from colic because of the history of abdominal discomfort. Since fever and severe hyperbilirubinemia are uncommon clinical findings with colic, we searched for other causes of the condition. In this case, liver disease was a primary consideration, but viral infections were another possibility. Since ehrlichiosis had not been recognized in this area, it was not considered until the characteristic morulae were discovered on the blood film. The discovery of the organism was fortuitous. Morula or elementary bodies are present for only 3 to 7 d and the degree of parasitism is variable. Buffy coat smears are often necessary to provide a concentration of cells sufficient to detect the organism. The presence of organisms tends to parallel

Can Vet J 1996; 37: 174–175

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the fever, and may persist for 2 to 4 d after the fever abates (1). This horse did not have signs of ataxia or limb edema, which may reflect the horse's young age or the rapidity of the diagnosis and treatment. The horse made an uneventful recovery and the response to oxytetracycline was excellent.

Granulocytic ehrlichiosis is not the same as Potomac horse fever, which is caused by *E. risticii* and manifests as severe diarrhea and laminitis. *Ehrlichia equi* infects neutrophils and occasionally eosinophils, whereas *E. risticii* organisms infect mononuclear leukocytes and are rarely found on a blood smear.

It is not known if this was an isolated case, or if the condition is present but unrecognized in this area. Since most horses usually recover spontaneously, the disease may go undetected. The potential for self-inflicted injury from ataxia and the considerable discomfort experienced by these animals warrants appropriate therapeutic intervention. This horse's clinical signs improved dramatically with appropriate antibiotic therapy. An awareness of ehrlichiosis may result in a better understanding of its prevalence in our area and may justify serologic surveys in the future.

There are also concerns regarding the potential for transmission of this organism to humans. Mortality caused by human ehrlichiosis has resulted in active investigation of the similarities between *E. equi* and organisms that have been identified in the human disease (6).

We hope that this report will prompt interest in this disease condition and a better understanding of *E. equi* within western Canada. CVJ

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