Equine Piroplasmosis

Equine Babesiosis, Equine Theileriosis, Biliary Fever
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Importance
Equine piroplasmosis is a tick-borne protozoal infection of horses. Piroplasmosis may be difficult to diagnose, as it can cause variable and nonspecific clinical signs. The symptoms of this disease range from acute fever, inappetence and malaise, to anemia and jaundice, sudden death, or chronic weight loss and poor exercise tolerance. Piroplasmosis is a major constraint to the international movement of equines. Although this disease was formerly endemic in Florida, the organisms were eradicated by the 1980s and piroplasmosis is considered to be an exotic disease in the United States. However, false negatives can occur in the complement fixation test, which was used for import testing until 2005, and there is a possibility that some horses in the U.S. might be inapparent carriers. In 2008, an outbreak occurred at a facility in Florida, highlighting the need to maintain constant vigilance for this disease.

Etiology
Equine piroplasmosis results from infection by the protozoa Babesia caballi or Theileria equi (formerly Babesia equi). Both organisms belong to the phylum Apicomplexa and order Piroplasmida. They can infect an animal concurrently. Other related protozoa such as Babesia bovis (the organism that causes bovine babesiosis) have been reported rarely in horses.

Species Affected
Equine piroplasmosis affects horses, mules, donkeys and zebras. Zebras are an important reservoir for infection in Africa.

Geographic Distribution
The parasites that cause equine piroplasmosis are endemic in many tropical and subtropical regions including parts of Africa, the Middle East, Asia, Central and South America, the Caribbean and Europe. To a lesser extent, they may be found in temperate areas. T. equi is thought to have a wider distribution than B. caballi. Australia, New Zealand, Canada, Japan and some other countries are free of these parasites. Equine piroplasmosis was eradicated from the United States by the 1980s, and it is considered to be an exotic disease. However, false negatives can occur in the complement fixation test, which was used for import testing in the U.S. until 2004/ 2005, and there is a possibility that some horses might be inapparent carriers. Other piroplasmosis-free countries that used this test could also have some carriers.

Transmission
B. caballi and T. equi are transmitted by ticks, which become infected when they ingest parasites in the blood of infected equids. Approximately 14 species of ticks in the genera Dermacentor, Hyalomma and Rhipicephalus can be vectors for these organisms; however, the epidemiological significance of some species is unknown. Potential tick vectors for T. equi and B. caballi exist in the U.S.

Although ticks are biological vectors for both T. equi and B. caballi, differences in these parasites' replication cycles can affect their methods of transmission. Inside the tick, Babesia zygotes multiply as 'vermicules,' which invade many of the tick's organs including the ovaries, and Babesia species are readily passed to the next generation of ticks in the egg (transovarial transmission). When an infected larval, nymphal or adult tick of the next generation attaches to a new host, the parasite is stimulated

to undergo its final maturation, allowing it to infect the host. In contrast, Theileria zygotes do not multiply in the tick, and transovarial transmission of T. equi is uncertain or absent. Ticks that transmit this organism can become infected as larvae and transmit the infection as nymphs, or they can become infected as nymphs and transmit the infection as adults (transstadial transmission). In some species of ticks such as Rhipicephalus microplus (formerly Boophilus microplus), T. equi can also be transmitted by the same tick stage that acquired the parasite (intrastadial transmission); whether this occurs in other species of ticks is unknown. Ticks infected with Theileria lose these parasites after transmission. Like B. caballi, T. equi parasites only stimulated to complete their maturation after the tick attaches to feed. For this reason, a tick infected with either organism must remain attached to the host for a time before it becomes infective; often, B. caballi and T. equi are transmitted after the tick has been attached for a few days. Equine piroplasmosis can also be transmitted directly between animals by contaminated needles and syringes, or by blood transfusions.

After recovery, horses may become carriers for long periods. Animals infected with B. caballi can remain carriers for up to 4 years, but might be able to clear the organism eventually. Equids infected with T. equi appear to remain permanently infected. Parasitemia is often absent in carriers, but can reoccur after immuno-suppression or strenuous exercise. T. equi can be passed to the foal in utero, and some foals can be healthy carriers. Transplacental transmission of B. caballi has rarely been reported, and some sources consider the evidence for this route to be unreliable.

Incubation Period

The incubation period for equine piroplasmosis is 12 to 19 days when it is caused by T. equi, and 10 to 30 days when it is caused by B. caballi.

Clinical Signs

The clinical signs of piroplasmosis are variable and often nonspecific. T. equi tends to cause more severe disease than B. caballi.

In rare peracute cases, animals may be found dead or dying. More often, piroplasmosis presents as an acute infection, with fever, inappetence, malaise, labored or rapid respiration and congestion of the mucus membranes. The feces may be small and dry, but diarrhea has also been reported. Anemia, thrombocytopenia, jaundice, hemoglobinuria, sweating, petechial hemorrhages on the conjunctiva, a swollen abdomen, and posterior weakness or swaying may also be seen. Subacute cases have similar but less severe clinical signs. The fever may be intermittent, and animals may show weight loss, signs of mild colic, and mild edema of the distal limbs. The mucus membranes in subacute cases can be pink, pale pink or yellow, and they may have petechiae or ecchymoses. In chronic cases, common symptoms include mild inappetence, poor exercise tolerance, weight loss, transient fevers and an enlarged spleen (palpable on rectal examination). Some infected mares, including carrier mares, may abort or pass T. equi to their offspring. Foals infected in utero may be weak at birth, and rapidly develop anemia and severe jaundice. In other cases, these foals can be healthy carriers.

Asymptomatic carriers can develop clinical signs after immunosuppression or strenuous exercise.

Post Mortem Lesions

In acute cases, the animal is usually emaciated, jaundiced and anemic. The liver is typically enlarged and may be either dark orange-brown or pale from anemia. The spleen is enlarged. The kidneys may be pale and flabby, or they may be dark red or black if the animal had hemoglobinuria. Petechial hemorrhages may be seen in the kidneys and subepicardial and subendocardial hemorrhages in the heart. Secondary infections may cause edema, emphysema or signs of pneumonia in the lungs.

Morbidity and Mortality

In some areas, equine piroplasmosis is most common in the summer and fall; however, even in these regions, cases can occur throughout the year. In some endemic areas where tick control is not practiced, nearly all horses are eventually exposed to B. caballi. Reported case fatality rates for equine piroplasmosis vary; one source suggests that the mortality rate can vary from less than 10% to as high as 50%.

Diagnosis

Clinical

Equine piroplasmosis should be suspected in horses with anemia, jaundice and fever; however, the clinical signs are often variable and nonspecific.

Differential diagnosis

The differential diagnosis for piroplasmosis includes surra, equine infectious anemia, dourine, African horse sickness, purpura hemorrhagica, and various plant and chemical toxicities.

Laboratory tests

Equine piroplasmosis can be diagnosed by identification of the organisms in Giemsa stained blood or organ smears. B. caballi merozoites are joined at their posterior ends, while T. equi merozoites are often connected in a tetrad or “Maltese cross.” T. equi can often be found in the blood in acute infections, but may be very difficult to find in carrier animals. B. caballi can sometimes be difficult to find even in acute disease. In carriers or other animals with low parasitemia, thick blood films can sometimes be helpful.

Because organisms can be difficult to detect in carriers, serology is often used for diagnosis. Serological tests include complement fixation (CF), indirect fluorescent antibody (IFA) and various enzyme-linked immunosorbent (ELISA) assays. Immunoblotting (Western blotting) can also be used, and an immunochromatographic test for T. equi has been described. The complement fixation test can be affected by natural anticomplementary activity in serum, as well as drug treatment or other factors; some carriers can be negative in this test. Animals do not become CF-positive for at least a month after inoculation. For these reasons, the IFA test and competitive ELISA (C-ELISA) have replaced complement fixation for import testing The IFA test can distinguish T. equi from B. caballi.

Polymerase chain reaction (PCR) assays are available in some laboratories. Additional molecular techniques