EQUINE PIROPLASMOSIS

Aetiology Epidemiology Diagnosis Prevention and Control References

AETIOLOGY

Classification of the causative agent

Equine piroplasmosis (EP) is a tick-borne disease of horses caused by the intraerytrocytic protozoan parasites *Babesia caballi* and *Theileria equi* of the Order Piroplasmida. *Theileria equi* was previously designated as *Babesia equi* but compelling evolutionary, morphologic, biochemical, and genetic evidence supports its reclassification as a *Theileria*.

Resistance to physical and chemical action

This agent does not survive outside its hosts and can only be transmitted through a tick vector, therefore, parameters associated with resistance to physical and chemical actions (i.e. temperature, chemical/disinfectants, and environmental survival) are not meaningful. Efficacy of medicines and biologics are described under "Prevention and control."

EPIDEMIOLOGY

This disease is a tick-transmitted disease of equids and its presence requires competent arthropod vectors. Infected animals may remain carriers of these blood parasites for long periods and act as sources of infection for other ticks. The introduction of carrier animals into areas where competent tick vectors are prevalent can lead to an epizootic spread of the disease.

Hosts

• Horses, mules, donkeys and zebra

Life Cycle and Transmission

- Babesia sporozoites invade red blood cells (RBCs) and transform into trophozoites which grow and divide into two round, oval or pear-shaped merozoites which, in turn, are capable of infecting new RBCs and the division process is then repeated
- Theileria equi sporozoites inoculated into horses via a tick bite invade the lymphocytes and these intralymphocytic forms undergo development and eventually form *Theileria*-like schizonts; merozoites released from these schizonts invade red blood cells (RBCs) and transform into trophozoites which grow and divide into pear-shaped tetrad ('Maltese cross') merozoites
- Twelve species of Ixodid ticks in the genera *Dermacentor*, *Rhipicephalus* and *Hyalomma* have been identified as transstadial vectors of B. caballi and *T. equi*, while eight of these species were also able to transmit *B. caballi* infections transovarially
 - Babesia spp. can be found in various organs of tick vectors and do transmit transovarially from egg to larva
 - Theileria equi develop in salivary glands of tick vector and not found in other tick organs; not transmitted transovarially from egg to larva
- Transmission is also possible through mechanical vectors contaminated by infected blood (e.g. contaminated needles)

Sources of infection

- Blood infected with causative parasites of piroplasmosis and associated vectors (i.e. ticks and mechanical vectors)
- Infected animals may remain carriers of these blood parasites for long periods and act as sources of infection for tick vectors

Occurrence

The parasites occur in southern Europe, Asia, countries of the Commonwealth of Independent States, Africa, Cuba, South and Central America, and certain parts of the southern United States of America.

Theileria equi has also been reported from Australia (but, apparently never established itself in this region), and is now believed to have a wider general distribution than *B. caballi*.

For more recent, detailed information on the occurrence of this disease worldwide, see the OIE World Animal Health Information Database (WAHID) Interface [http://www.oie.int/wahis/public.php?page=home] or refer to the latest issues of the World Animal Health and the OIE Bulletin.

DIAGNOSIS

Incubation period of equine piroplasmosis associated with *T. equi* is 12 to 19 days and approximately 10 to 30 days when caused by *B. caballi.*

Clinical diagnosis

The clinical signs of equine piroplasmosis are often nonspecific, and the disease can easily be confused with other similar hemolytic conditions presenting fever, anemia and jaundice. *Theileria equi* tends to cause more severe disease than *B. caballi*. Piroplasmosis can occur in peracute, acute, subacute and chronic forms. Documented case fatality rates vary from 10–50%. Most animals in endemic areas survive infection.

Peracute form

• Rare form of disease with only clinical observation being moribund or dead animals

Acute form

- Most common form of disease cases
- Characterised by fever that usually exceeds 40°C
- Reduced appetite and malaise
- Elevated respiratory and pulse rates
- Congestion of mucous membranes
- Production of a dark red urine; faecal balls that are smaller and drier than normal
- Affected animals may appear unthrifty; anemic and/or icteric

Subacute form

- Similar to acute form but accompanied by weight loss in affected animals and intermittent fever
- Mucous membranes vary from pale pink to pink, or pale yellow to bright yellow; petechiae and/or ecchymoses may also be visible on the mucous membranes
- Normal bowel movements may be slightly depressed and the animals may show signs of mild colic

Chronic form

Chronic cases usually present nonspecific clinical signs such as mild inappetence, poor performance and a drop in body mass

Lesions

- Lesions observed are those most often associated with an intravascular hemolytic condition
- Pale or icteric mucous membranes; blood may appear thin and watery
- Swollen liver with an orange-brown or paler coloration
- Enlarged, dark, friable spleen; palpable on rectal examination
- Kidneys may appear paler or darker than normal with possible petechial hemorrhages
- Subepicardial and subendocardial hemorrhages may be visible on cardiac tissue
 - Mild oedematous swelling of the distal part of the limbs sometimes occurs in subacute forms
- Secondary infections may lead to various non-specific lesions including oedema, emphysema or pneumonic condition of lungs

Differential diagnosis

- Surra
- Equine infectious aonemia
- Dourine
- African horse sickness
- Purpura haemorrhagica
- Plant and chemical toxicities

Laboratory diagnosis

Samples

- Several thick and thin blood smears collected from superficial skin capillaries of live animals during the acute phase of the disease (appearance of fever); organ smears can be acquired at necropsy (cerebral cortex, kidney, liver, lung, bone marrow)
 - o slides with blood or organ smears should be air-dried and then fixed in methanol
- Serum samples should also be collected

Procedures

Identification of the agent

- Microscopic examination of blood
 - o demonstration of parasites in stained blood; using Giemsa staining method
 - o thick blood smear technique also used in instances very low parasitaemia
 - as co-infections of *T. equi* and *B. caballi* occur, accurate identification of the species of parasite is sometimes desirable
- Identification of equine piroplasmosis in carrier animals by means of blood smear examination is difficult, inaccurate and not practical on large-scale; serological methods are preferred
- Molecular techniques for the detection of *T. equi* and *B. caballi* have been described
 based on species-specific polymerase chain reaction

Serological tests

- Indirect fluorescent antibody (IFA) test (a prescribed test for international trade)
 - IFA test has been successfully applied to the differential diagnosis of *T. equi* and *B. caballi* infections
 - recognition of a strong positive reaction is relatively simple, but any differentiation between weak positive and negative reactions requires considerable experience in interpretation
 - detailed description of the protocol of the IFA test is available from published sources and an example of an IFA protocol is provided in the OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animal.
- Enzyme-linked immunosorbent assay (a prescribed test for international trade)
 - indirect ELISAs using recombinant *T. equi* and *B. caballi* proteins have shown high sensitivity and specificity in detecting antibodies in infected horses
 - a competitive inhibition ELISA (C-ELISA) using recombinant protein and a specific monoclonal antibody (MAb) that defines merozoite surface protein epitope overcomes problems associated with antigen purity; 94% correlation was shown between the C-ELISA and the CF test in detecting antibodies to *T. equi*
- Complement fixation (CF) test
 - o CF test has been used by some countries to qualify horses for importation
 - The test may not identify all infected animals, especially those that have been drugtreated or that produce anti-complementary reactions, or because of the inability of IgG(T) (the major immunoglobulin isotype of equids) to fix guinea-pig complement
 - Therefore, the IFA test and C-ELISA have replaced the CF as the prescribed tests for international trade

For more detailed information regarding laboratory diagnostic methodologies, please refer to Chapter 2.5.8 Equine piroplasmosis the latest edition of the *OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals* under the heading "Diagnostic Techniques".

PREVENTION AND CONTROL

Sanitary prophylaxis

- EP is most commonly introduced into an area by means of carrier animals or infected ticks
- Thus, movement of equids requires testing (by IFA or ELISA as described above)
- Reducing exposure of equids to ticks
 - o repellents, acaricides and regular inspection; animals and premises
 - control and eradication of the tick vector; including removal of nearby vegetation that could harbour ticks
- · Any detected EP-positive animals should be quarantined from surrounding horses and vectors
- Special care in possible mechanical infection of horses with contaminated blood

Medical prophylaxis

- No biological products are available currently
- Antiprotozoal agents only temporarily clear *T. equi* from carriers

For more detailed information regarding safe international trade in terrestrial animals and their products, please refer to the latest edition of the OIE *Terrestrial Animal Health Code*.

REFERENCES AND OTHER INFORMATION

- Brown C. & Torres A., Eds. (2008). USAHA Foreign Animal Diseases, Seventh Edition. Committee of Foreign and Emerging Diseases of the US Animal Health Association. Boca Publications Group, Inc.
- Coetzer J.A.W. & Tustin R.C. Eds. (2004). Infectious Diseases of Livestock, 2nd Edition. Oxford University Press.
- Homer M.J. & et al. (2000) Clin. Microbiol. Rev., 13 (3): 451.
- Kahn C.M., Ed. (2005). Merck Veterinary Manual. Merck & Co. Inc. and Merial Ltd.
- Spickler A.R. & Roth J.A. Iowa State University, College of Veterinary Medicine http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.htm.
- World Organisation for Animal Health (2009). Terrestrial Animal Health Code. OIE, Paris.
- World Organisation for Animal Health (2008). Manual of Diagnostic Tests and Vaccines for Terrestrial Animals. OIE, Paris.

*
*

The OIE will periodically update the OIE Technical Disease Cards. Please send relevant new references and proposed modifications to the OIE Scientific and Technical Department (<u>scientific.dept@oie.int</u>). Last updated October 2009.