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Babesiosis In Domestic Animals

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Introduction

Vector-borne protozoan diseases continue to pose significant challenges to both animal health and livestock productivity worldwide. Among these, **babesiosis** is one of the most important tick-borne protozoan diseases of domestic and wild animals, with occasional zoonotic transmission to humans. The disease is of particular concern in tropical and subtropical regions, where environmental conditions favor the survival and propagation of tick vectors.

Babesiosis is an intraerythrocytic protozoan infection affecting cattle, buffaloes, sheep, goats, pigs, dogs, cats, horses, wild animals, and humans. It is clinically characterized by fever, progressive anemia, hemoglobinuria, and jaundice, and in severe cases may lead to death. The disease was first reported in 1888 in South Africa and has since been recognized worldwide. The global prevalence of babesiosis has increased with the intensification of international livestock trade, transhumance, and the importation of animals into non-endemic regions. In addition to its veterinary and economic importance, babesiosis is increasingly recognized as an emerging zoonotic disease in humans.

Susceptible Hosts

Babesiosis has been reported in all breeds of cattle; however, the disease is more common and severe in crossbred and exotic breeds compared to indigenous ones. Among cattle, *Bos taurus* breeds are generally more susceptible than *Bos indicus*.

Age plays an important role in susceptibility. Calves aged 1–2 months exhibit a degree of innate tolerance, whereas older calves and adults are more prone to clinical disease. In addition to cattle, several other species are affected, including sheep, goats, pigs, horses, wild ruminants, and humans. Sporadic cases of human babesiosis have been reported in Europe, the

United States, and India (Rosenberg et al., 1981).

Mode of Transmission

Transmission of babesiosis is not merely a mechanical process, as *Babesia* organisms are actively injected into the host along with the saliva of infected ticks. During an infective bite, sporozoites are inoculated into the host bloodstream together with salivary secretions. The immature developmental stages of *Babesia* undergo part of their life cycle within the tick vector and are subsequently transmitted to susceptible animals through tick bites.

Ticks belonging to the genera **Boophilus, Rhipicephalus, Hyalomma, Haemaphysalis,** and **Ixodes** are recognized as the principal vectors of babesiosis. These ticks transmit different species of the parasite, including:

- **Bovine babesiosis:** *Babesia bigemina, Babesia bovis, Babesia divergens*
- **Equine babesiosis (piroplasmosis):** *Babesia caballi, Theileria equi* (formerly *Babesia equi*)
- **Canine babesiosis:** *Babesia canis, Babesia gibsoni*
- **Ovine babesiosis:** *Babesia ovis, Babesia motasi*

Transovarian transmission is a key epidemiological feature, occurring commonly in *Boophilus* spp., *Rhipicephalus evertsi*, *Rhipicephalus simus*, *Hyalomma marginatum*, and *Ixodes ricinus*. However, it is not observed in *Rhipicephalus sanguineus*, the principal vector of canine babesiosis. In addition to tick-borne transmission, the disease can also spread through **iatrogenic means**, including contaminated blood transfusions, surgical instruments, and hypodermic needles. Rare cases of transmission via the bites of infected carnivores (e.g., dog bites) and sexual transmission (*Babesia gibsoni* in dogs) have also been documented.

In humans, *Babesia microti* is primarily transmitted by *Ixodes scapularis* (the deer tick) in North America, whereas *Babesia divergens* is transmitted by *Ixodes ricinus* in Europe.

Pathogenesis

The incubation period of babesiosis varies depending on the *Babesia* species involved. Following inoculation of sporozoites by ticks, the parasites invade peripheral red blood cells, multiply asexually, and ultimately rupture the host cells. This destruction of erythrocytes results in hemoglobinemia, hemoglobinuria, and anemia.

Additional contributing factors include:

- Release of toxic metabolites by the parasite
- Increased vascular permeability, leading to edema and tissue anoxia
- Blockage of capillaries by parasitized erythrocytes
- Disseminated intravascular coagulation (DIC)

- Autoimmune hemolysis triggered by coating of erythrocytes with parasite antigens

Clinical Findings

Babesiosis is typically characterized by high fever, anorexia, depression, cessation of rumination, loss of appetite, ruminal stasis, and a marked decline in milk yield.

- **Bovine babesiosis:** Hemoglobinuria ("redwater") is a pathognomonic sign. In *B. bovis* infections, jaundice is uncommon, whereas in *B. bigemina* infections, jaundice is frequently observed.
- **Young animals:** The disease is associated with splenomegaly and anemia. Severe cases may progress to depression, hyperpnea, prostration, muscular tremors, and death.
- **Canine babesiosis:** The disease manifests in five clinical forms:
    - **Respiratory form:** dyspnea, pulmonary edema
    - **Cerebral form:** nervous signs, opisthotonus, coma
    - **Gastrointestinal form:** diarrhea, vomiting
    - **Renal form:** hemoglobinuria, uremia
    - **Muscular form:** muscular tremors, ataxia

In complicated cases, babesiosis may lead to hemoglobinuric nephrosis, icterus, hepatopathy, pulmonary edema, cerebral syndromes, pancreatitis, hypoproteinemia, cardiac involvement, and death.

Clinical Pathology

Hematology

- Severe anemia; packed cell volume (PCV) reduced to 8–15%
- Marked leukocytosis with monocytosis
- Moderate to increased total leukocyte count
- Hypoproteinemia due to decreased serum albumin, with compensatory hyperglobulinemia
- Elevated serum bilirubin as a consequence of hemolysis
- Icteric plasma due to bilirubin accumulation
- Elevated blood urea nitrogen and serum glutamic oxaloacetic transaminase (SGOT) (Roy & Choudhury, 1973)

Lesions

- Carcass pale and emaciated with icteric mucous membranes
- Subcutaneous tissues and muscles pale
- Petechial and ecchymotic hemorrhages in the endocardium and on serosal surfaces
- Liver enlarged, yellow, friable

- Gallbladder distended with bile
- Kidneys enlarged and congested
- Spleen enlarged, dark, friable
- Lymph nodes hemorrhagic
- Lungs exhibiting edema
- In dogs: splenomegaly and hepatomegaly are prominent

Diagnosis

Diagnosis is based on the following criteria:

1. **Clinical signs:** High fever, hemoglobinuria, anemia, and jaundice.
2. **Blood smear examination:** The simplest diagnostic method. Parasites are demonstrable in peripheral blood using Giemsa-stained thin smears observed under oil immersion.
3. **Clinical pathology:**
    - Low erythrocyte count, packed cell volume, and hemoglobin concentration
    - Increased serum bilirubin
    - Increased serum globulins
4. **Serological tests:**
    - Capillary agglutination test
    - Indirect hemagglutination test
    - Complement fixation test
    - Indirect fluorescent antibody test (IFAT)
    - Enzyme-linked immunosorbent assay (ELISA)

Treatment

- *B. bovis* and *B. major* (large species) are more sensitive to therapy and require lower doses.
- Commonly used drugs include:

1. **Diminazene aceturate** (Berenil, Veriben, Ganaseg, Azidin): 3.5 mg/kg b.w. intramuscularly; in severe cases, 7 mg/kg b.w.
2. **Imidocarb dipropionate** (Imizol, Forray-65, Imicarb): 1.2 mg/kg b.w. subcutaneously; in *B. equi* infection, 4.4 mg/kg b.w. deep intramuscular once; in dogs, 5–6 mg/kg b.w. intramuscularly or subcutaneously
3. **Quinuronium sulfate** (Trypan Blue): 0.5 mg/kg b.w. subcutaneously
4. **Phenamidine isethionate:** 10 mg/kg b.w. intramuscularly
5. **Acranil:** 3 mg/kg b.w. intramuscularly (Gradwohl & Roy, 1950)

6. **Pyrimethamine–sulfadiazine combination**: occasionally used in canine babesiosis

Supportive Treatment

- Antipyretics for control of fever
- Intravenous glucose saline or dextrose solutions to correct dehydration
- Liver extract and vitamin B-complex for hepatic support
- Iron supplementation (oral or parenteral); ferrous sulfate orally where liver extract with iron is unavailable
- Whole blood transfusion in cases of severe anemia

Control

- Tick control remains the cornerstone of babesiosis prevention. Regular acaricide application by dipping, spraying, or pour-on formulations is essential.
- Vaccination strategies include:
    - **Live vaccines:** Attenuated strains of *B. bovis* and *B. bigemina*
    - **Hyperimmune serum:** In India, calves have been immunized using hyperimmune *B. bigemina* antiserum
    - **Killed vaccines:** Provide partial protection, capable of stimulating protective immune responses
    - **Crude vaccines:** Reported by Schetter & Hildebrandt (1982)
    - **Canine vaccine:** *Pirodog* is available in France for prevention of canine babesiosis

Zoonotic Aspect

Babesiosis also has zoonotic importance. Human infections have been increasingly reported, particularly in immunocompromised or splenectomized individuals. *Babesia microti* is the predominant cause of human babesiosis in the United States, transmitted by *Ixodes scapularis*, while *B. divergens* is the major species implicated in Europe, transmitted by *Ixodes ricinus*. Sporadic human cases have been documented in India as well (Wester et al., 1996).