

## Bovine Babesiosis: An Overview

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### Introduction

Bovine babesiosis is an economically important tick-borne haemoprotozoan disease affecting cattle and buffaloes. The disease is recognized as a major economic threat to the upgrading of the country's wealth system. This apicomplexan parasites "*Babesia*" mainly attack the host erythrocytes and characteristically mainly found in in naïve cattle older than one year, which result in either death or the development of persistent infection in animals, often known as "premunity" that have recovered from the acute phase. Persistently infected animals become reservoirs for transmission by competent ticks.

Taxonomically, *Babesia* stands as:

<b>Kingdom</b>	Protista
<b>Sub Kingdom</b>	Protozoa
<b>Phylum</b>	Apicomplexa
<b>Class</b>	Sporozoea
<b>Sub class</b>	Piroplasmia
<b>Order</b>	Piroplasmida
<b>Family</b>	Babesiidae
<b>Genus</b>	<i>Babesia</i>
<b>Species</b>	<i>B. bigemina</i> <i>B. bovis</i> <i>B. major</i> <i>B. divergens</i>

### History

1746



In the year 1888, Babes found out the symptoms of hemoglobinuria in cattle of Romania which he investigated further and became the first to discover piroplasm in the blood of cattle.

Initially, he thought it to be a bacterium and named as *Hematococcus bovis* and later it was changed to *Babesia bovis*.

In 1893. Smith and Kilbourne described under natural condition, *Babesia* species are transmitted by ticks and the causal agent was *B. bigemina* of Texas fever.

In 1932, Dennis described that the sexual reproduction of *Babesia* spp. occurs in *Boophilus annulatus*

Reik mentioned that the life cycle of *B. bigemina* is maintained in the tick *Boophilus microplus* and regarded the “vermicules” the infective forms of *Babesia*

Later, in 1956. the first case of human lethal babesiosis caused by *Babesia divergens* was recorded.

## Characteristics

Species	Host	Vector	Characteristics
<b><i>B. bigemina</i></b>	Principally bovine, zebu, water buffalo. Rarely deer and white-tailed deer	One host tick- <i>Boophilus annulatus</i> <i>B. calcaratus</i> , <i>B. decoloratus</i> Two host tick- <i>Rhipicephalus bursa</i> , <i>R. evertsi</i> . Three host tick- <i>Haemaphysalis punctate</i> , <i>R. appendiculatus</i>	Large piroplasm (4-5 µm in length and 2 µm in wide)  Pear shaped and lie in pairs, forming an acute angle in the red blood corpuscles.
<b><i>B. bovis</i></b>	Mainly Cattle. Rarely roe deer and stag.	<i>Ixodes ricinus</i> <i>I. persulcatus</i> <i>B. calcaratus</i> <i>B. microplus</i> <i>R. bursa</i>	Small piroplasm, lying superficially in RBC Signet ring formation is common consisting of a centrally placed vacuole with a nuclear mass at one pole
<b><i>B. major</i></b>	Cattle	<i>B. calcaratus</i> <i>H. punctata</i>	Small piroplasm, lying in the centre of RBC and angle formed is less than 90
<b><i>B. divergens</i></b>	Cattle	<i>I. ricinus</i> <i>D. reticulatus</i>	Remain in pairs, divergent, lying superficially in RBC



## Life Cycle

### In vertebrate host

The parasite undergoes schizogony in the bovine erythrocytes to form two, four or more trophozoites which will be liberated and attack other red cells. This process continues and large number of RBCs get parasitized. This stage will be taken by a susceptible tick. If the tick is one host tick, transmission will be transovarian and in case of two or three host tick, transmission will be stage to stage.

### In the tick vector

**Transovarian transmission:** After repeated multiplication in the RBC of host, ultimately micro and macro gametes are formed and during tick's blood feeding these micro and macro enter the gut cells of the tick and unite together to form zygote which becomes motile and forms vermicules and finally reach the acinar cells of the salivary gland of larva and on subsequent feeding to a healthy animal, the infective organisms are released into the blood of the host.

**Stage to stage transmission:** Multiplication occur in phagocytes. Pseudocyst containing club shaped organism are formed in about 7 days in nymph and migrate to muscle sheaths and divide repeatedly. When the nymph moult to adult, migration to salivary glands occurs and they remain in the salivary gland. Subsequent biting to the susceptible host will release the sporozoites.

## Pathogenesis

- 1) Release of pharmacologically active substance and destruction of erythrocytes are the main pathological condition.
- 2) *B. bigemina* resembles a hemolytic anaemia while *B. bovis* shows kinin production
- 3) In acute infection, there is rapid mobilization and activation of kallikrein which increases the vascular permeability and vasodilation causing circulatory stasis and shock.
- 4) There is initial fall of packed cell volume which is due to kallikrein level disturbance.
- 5) Anaemia is pronounced which is due to mechanical destruction of RBC and adsorption of Ag-Ab complexes to the surface of RBC leading to their removal by phagocytosis.
- 6) Glomerulonephritis occurs due to glomerular deposits of IgG and C3 component of complement.

## Clinical Signs

- 1) First sign to be showed by the affected animal is high fever (40- 41 C) with depression, lethargy, anorexia, weakness and cessation of rumination.
- 2) There will be dryness of muzzle and continuous lacrimation.
- 3) Initially there will be profuse diarrhoea followed by constipation.
- 4) The conjunctiva become brick red in the initial period, then it becomes anaemic.



- 5) With advancement of infection, anaemia becomes severe, coffee coloured urination (haemoglobinuria), jaundice, and the animal is in the stage of collapse.
- 6) Death is mainly due to excessive destruction of red blood cells, cerebral anoxia, and accumulation of toxic by-products.

### **Post Mortem Changes**

#### **Macroscopically,**

- 1) Subcutaneous and intramuscular edema
- 2) Icteric organ
- 3) Fat become yellow and gelatinous
- 4) Blood become watery and thin

#### **Microscopically,**

- 1) Liver shows congestion, centrilobular necrosis, deposition of haemosiderin in kupffer cells
- 2) Kidney shows degeneration of tubular epithelium, deposition of haemosiderin and cast formation.
- 3) Lymph node and spleen shows depletion of germinal centre , hyperplasia of reticular tissue, and macrophage having haemosiderin.

### **Diagnosis**

1. History
2. Clinical signs: most prominent signs are high fever, anaemia, haemoglobinuria, jaundice.
3. Thick and thin blood smear examination.
4. Immunodiagnostic test: important in subclinical cases where soluble antigen is obtained from parasite
5. Serodiagnostic test:
  - ✚ CFT (Compliment Fixation Test)
  - ✚ IFA (Indirect Fluorescent Antibody)
  - ✚ IHA (Indirect Haemagglutination) shows 80% efficacy
  - ✚ CTA (Capillary tube Agglutination) specific for *B.bigemina*

### **Treatment**

- 1) Diminazene aceturate @ 3-5 mg/kg body weight subcutaneously/ intramuscularly for 2 consecutive days
- 2) Imidocarb dipropionate/dihydrochloride @ 1.2 mg/kg body weight subcutaneously once.
- 3) Phenamidine isothianate @ 12 mg/kg body weight subcutaneously once.
- 4) Trypan blue @ 50-100 mg/kg body weight subcutaneously/ intramuscularly/slow intravenously.



## Control

- 1) Protection of healthy animal from infected stock.
- 2) Animal if suspected to have infection should be immediately segregated and should be treated.
- 3) Newly purchased animals should not be mixed with the existing stock and should undergo a quarantine period.
- 4) Control of vectors: Regular dipping of animals can get rid of the tick infestation.

