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Tick Borne Trouble: Uncovering Babesiosis in Cattle

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INTRODUCTION:

Viktor Babes (1888) in Rumania was the first to describe *Babesia sp.* in the blood of cattle and sheep. By 1930, *Babesia sp.* had been described in all of domestic animals.

Babesiosis is caused by the intraerythrocytic protozoan parasite of the genus Babesia that infects a wide range of domestic animals, wild animals & occasionally man. The disease is a tick borne disease, mainly transmitted by Rhipicephalus (Boophilus) ticks (in *B. bovis, B. bigemina*), *Ixodes Ricinus* (in *B.divergens*). The geographical distribution of the parasite is world-wide (tropical & subtropical region). The major economic impact of babesiosis is on the cattle industry.

MORPHOLOGY:

They are large, pyriform seen in Red Blood Cells. They are round or amoeboid.

All the developmental stages occur inside the RBCs.

Vector: Ixodid ticks

DIFFERENT BABESIA SPECIES IN CATTLE:

- *Babesia bigemina*
- *B.bovis*
- *B.divergens*
- *B.major*

TRANSMISSION:

Babesia sp. are transmitted by ticks. Transmission of *Babesia sp.* by ticks is through eggs (transovarian) or stage to stage transmission (transtadial).

The transovarian transmission happens only in one host tick after attachment of larvae and the rest of the tick development occurs in the same animal.

With 2 or 3 host ticks, transtadial transmission is common.

Babesia bigemina in *Rhipicephalus (B) microplus*, one-host tick; *Babesia canis* in *Rhipicephalus sanguineus*, three -host tick;

Large forms of babesia are generally transmitted by adults and nymphs of ticks and small forms by larvae and adults of ticks.

***BABESIA BIGEMINA*:**

Disease: Cattle tick fever, red water fever, Texas fever, Piroplasmosis.

Vectors: *Rhipicephalus (B) microplus* & *R. annulatus* in India.

They are the main vectors of *Babesia bigemina* and *B. bovis*

Transmission occurs transovarian.

LIFECYCLE:

Life cycle: Indirect and heteroxenous

Vector: *Rhipicephalus (B) microplus*-transovarian transmission

Developmental cycle

1. Schizogony/Merogony- is seen in RBCs of Cattle
2. Gamogony- in tick – intestinal/gut epithelium
3. Sporogony- in tick salivary gland acinar cells- after hatching

Life cycle in the cattle

When infected tick bites the cattle, the sporozoites from tick saliva pass into the host, and inside the RBC it becomes a trophozoite, which undergoes binary fission and produces 2 merozoites.

Further division occurs which results in the rupture of RBC, and release of the merozoites which in turn infect fresh RBCs.

They develop into ovoid forms – gamonts – precursors of sexual phase.

Life cycle in the tick

When adult ticks feed on the infected host, the passage of blood to the midgut stimulates the production of ray bodies -stralenhorper bodies or spiky ray bodies-gametes. They fuse in pairs- syngamy and form spherical zygote. Zygotes transform into a motile vermiform kinete.

They penetrate and infect the tick digestive cells and undergo fission- Primary fission bodies-which give rise to primary kinetes or vermicules or sporokinetes. These are motile and enter into haemolymph.

They enter variety of cells/tissues hemocyte, muscle, malphigian tubule, female ovarian cells including oocyte to form secondary fission body.

Development in salivary gland

Kinetes remain dormant in the egg until the larvae hatches from the egg.

When the larva hatches, they enter salivary gland and multiply further

Kinetes enter salivary gland-acinar cell undergo sporogony each giving rise to minute sporozoites. These vermiform sporozoites enter to host during feeding of nymphal/adult ticks.

Pathogenesis

Death is due to over destruction of RBC – Multiple organ failure due to anaemic anoxia, oedema, icterus and haemoglobinuria, and also due to clogging of blood vessels supplying important organs by parasitic cells -causes anoxia and hemorrhagic shock-death. Glomerulonephritis due to haemolytic crisis-Circulatory Antigens form circulatory complexes

with antibody and complement which lodges in kidney. This reaction depletes body which disposes anaphylotoxin- augment shock.

Babesiosis is a highly pathogenic disease. Mortality is more seen in adults than young calves (inverse age resistance). The calves are resistant between 9 and 12 months. The disease mainly causes haemolytic anaemia due to the erythrocyte destruction. There is intravascular hemolysis- RBC ruptures due to the piroplasms multiplying and releasing from it.

Clinical signs:

The symptoms are more marked in exotic and cross breeds.

The incubation period is 1-2 weeks.

Acute form:

High Fever – 41-45.5° C

Anorexia, Rumen atony, lacrimation, salivation, dyspnea, tachycardia and rough hair coat. Initially the mucous membrane will be reddened, later mucous membrane is pale to icteric.

Severe anaemia

Weight loss, abortion, drop in milk production

Infected cattle may stand with an arched back, staggering gait

Urine will be dark red in colour (haemoglobinuria – dark red or coffee coloured urine). When haemoglobinuria is seen, the temperature becomes subnormal.

Constipation commonly develops in the later stages and animals may become recumbent due to weakness.

Chronic form:

Several weeks with intermittent temperature. Animal becomes thin and emaciated.

No marked haemoglobinuria, finally animal recovers, loss of weight, icterus, hard yellow faeces.

Cerebral form:

Onset is sudden-Temperature-41.7°C in few hours, death in 12-36 hrs. Parasites appear to accumulate and multiply in cerebral capillaries since organisms are rarely seen in blood smears.

Haematological changes:

Haemoglobin (Hb), packed cell volume (PCV), differential leucocytic count (DLC), total leucocytes count (TLC) and total erythrocytic count (TEC) were significantly decreased.

Decreased blood glucose

In acute phase, anaemia is normocytic later become macrocytic with increased MCV. WBC decreases first, increase to 2-3 folds after recovery.

Serum level of aspartate aminotransferase (AST), alanine aminotransferase (ALT) and gamma glutamyltransferase (GGT) were increased.

Post mortem lesions:

The entire carcass may be very icteric and/or pale

Blood is very thin watery.

Urinary bladder contains dark colour urine.

Spleen shows enlargement with soft dark pulp

Liver enlarged with yellow colour distension of gall bladder with dark bile.

Kidney congested.

Diagnosis:

Based on clinical signs and presence of tick infestation

Blood smear examination- merozoites inside the RBC-Pear shaped

The parasite also detected by PCR.

Serological Testing

ELISA (Enzyme-Linked Immunosorbent Assay)

IFAT (Indirect Fluorescent Antibody Test)

Treatment:

Diminazine aceturate: 2-3.5 mg/kg Body weight deep I/M,

Imidocarb: Therapeutic and Prophylactic. 5mg/kg S/C.

Quinuronium derivatives ,. Quinuronium sulfate, - 1 ml 5% solution S/C for 50 kg Body wt.

Diampron amicarbalide: 10 mg kg I/M or S/C.

Trypan blue 100ml of 1-2% solution in normal saline given I/V.

Acriflavin: 20 ml 5% solution I/V

Phenamidine 12 mg kg S/C in 40% aqueous solution.

Supportive therapy: Calcium, blood transfusion, lot of water and saline.

Control & prevention:

TICK CONTROL-By using acaricides to cattle as pour-on with high residual activity during the greatest exposure, Such as flumethrin &deltamethrin to use protect the cattle from ticks for 2 and3 weeks respectively.

Chemoprophylactic effects of imidocarb against bovine babesiosis.

Environmental concerns over the continued use of insecticides has led to use of integrated strategies for tick control.

Anti-tick vaccine also available in some countries.

Immunization with mild strain, Exoantigen

BABESIA BOVIS:

Disease: Bovine Babesiosis, Piroplasmosis, Red water (except USA), Canada.

Morphology: Small, pyriform, round, irregular, vacuolated signet ring forms are common. Merozoites are in centre of erythrocytes.

Pathogenesis:

Characteristic feature-It produce shock syndrome which occurs even with 1% parasitaemia

Main pathogenesis is due to activation of kinin system

Within 3 days of infection, the plasma kallikrein system is activated. Prekallikrein transforms into kallikrein which causes increased vascular permeability, vasodilatation, circulatory stasis and shock.

Also, there is a massive release of pharmacologically active substances-prostaglandins, and macrophage migration inhibition factor which contribute to shock. Infected RBCs adhere to vascular endothelium which results in stasis of blood and congestion

Increased osmotic fragility – spontaneous lysis of RBCs

Above all *B. bovis* causes a condition called cerebral babesiosis- in which the parasitized RBCs sludge in the microcirculation of the brain which leads to hypersensitivity, trembling, weakness, grinding of teeth, coma and death due to circulatory collapse.

Clinical signs:

- High fever
- Inappetance/depression
- Increased respiratory rate
- Weakness and reluctance to move
- Haemoglobinuria may be present-Not a pathognomonic sign
- Anaemia and jaundice
- Muscle wasting and tremors
- Recumbency
- Pregnant cattle abort, decrease fertility
- CNS symptoms in case of cerebral form. Convulsion, incordination, coma, congestion of white mater, dilatation of capillaries packed with RBC. Perivascular neuronal interstitial oedema through brain.

Babesia divergens:

- Northern Europe form in cattle, small 1.5x0.5µm, appear as pairs diverted from centre in RBC, pyriform, circular up to 2 µm seen.
- Transmission by *Ixodes ricinus, Dermacenter reticulatus*.
- Pathogenesis: Less severe than *B.bigemina*. Hemoglobinuria, jaundice, Severe infection, death. Gastro-intestinal upset with thin watery diarrhoea followed by constipation, progressive appetite loss-death or recovery.