Order: Piroplasmida **By: Dr. Sanku**

2 families

1. Babesidae
2. Theileridae

Genus: *Babesia*

Blood protozoa of vertebrate which lack of pigment granules. These are the minute parasite multiplying in erythrocyte by asexual division, producing two, four or more non-pigmented amoeboid parasites. They multiply by binary fission or schizogony in red blood cell. The vectors are ixodid. When stained with Romanowsky stain, they show blue cytoplasm and red chromatin mass, usually at one pole. *Babesia* parasite generally fall in to two major groups-

a. Large form= average length is more than 3µm
b. Small form= average length is less than 2.5µm

Life cycle:

Multiplication of *Babesia* organism in the vertebrate host occurs in the erythrocyte by budding process (schizogony) to form 2,4 or more trophozoites. These are liberated from erythrocyte and invade other cells and like this way large percentage of RBCs are parasitized. Blood forms are rapidly transmissible to other host by mechanical transmission.

Smith and Kilborne (1893) first demonastrated *Babesia bigemina* causing **Taxes fever** is transmitted by tick *Boophilus annulatus.*

Development and transmission in tick either by

-transovarian transmission- one host tick or by

-stage to stage transmission- 2 or 3 host tick.

Sexual reproduction occurs in tick and asexual reproduction in vertebrate host in RBC. *Babesia* is not strickly host specific.

Transovarian Transmission:

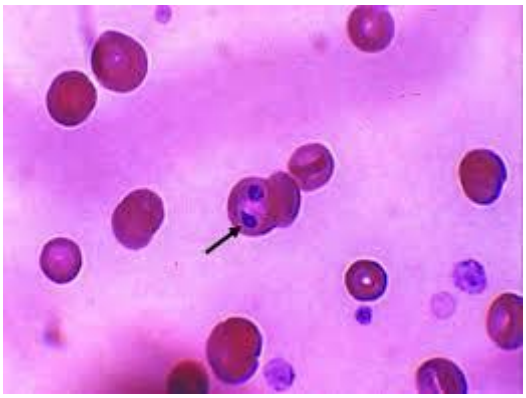
After repeated multiplication of parasite inside erythrocytes of vertebrate host, the stages are ultimately differentiated as micro and macrogametes providing that further development takes place in tick vector are sexual. These stage are taken by suitable tick and further development occurs in the gut of tick. Gamets are free by lysis of erythrocyte and unite to form zygote. Zygote undergoes development and multiplication. It,s a motile and come to serous surface and rupture. The vermicles thus released invade eggs and finally reach to gut epithelium of the larvae emerging out of infected eggs. The infective organisms finally reach the salivary gland of larvae and after maturation, are able to infect vertebrate host during feeding on blood. Subsequent developmental stages viz nymph, adult tick continue to transmit infection.

Stage to stage Transmission:

Multiplication of developmental form was seen in phagocyte. ‘Pseudocyst’ of organisms occur about 7 days after the nymph drops off the infected host. Club shaped organisms of 9µmX2µm are formed by 11-15 days in the cyst. These are liberated from the host cell and migrate to the muscle sheaths of the nymphal tick where they penetrate muscle cells and

divide repeatedly to form large nos of small, ovoid forms of about 1.2µm in length. Further development and migration to the salivary gland occurs when, after moulting, the adult tick feed on a dog. On reaching salivary glands, the parasiteenter the cells of the acini and undergo repeated binary fission to form a large no. of small, ovoid infective stage.

Babesia spp. in red blood cells are seen singly as round or ovid elongate, in pair as pyriform, in tetrads as cruciform. Characteristically they are pear formed lying at an angle with narrow end apposition.



Pathogenesis of *Babesia* infection:

2 factors play an important role.

1. Release of pharmacologically active substances
2. Destruction of erythrocyte

Disease caused by *B. bigemina* resembles a haemolytic anaemia, while *B. bovis* infection, kinin production is more important.

A. Plasma kallikrein raise markedly three days after infection then decrease subnormal. Kallikrein produce increase vascular permeability and vasodilatation.
B. In *B.bovis* infection, initial fall in PCV rather than destruction of erythrocyte.
C. Kallikrein also trigger intravascular coagulation.
D. Anaemia is associated withemerging parasites from RBC. Erythrocyte loss exceeds that attributable to mechanical rupture of cells by parasite.
E. Direct removal of non-infected erythrocyte by phagocytosis and osmotic fragility of non-infective RBC- predispose to spontaneous lysis in *B.bovis.*
F. Glomerulonephritis due to glomerular deposition of IgG+C3
G. Central nervous system damage- selective concentration of parasitized cell occurs in brain capillaries leading to obstruction of blood flow.
H. Adsorption of Ag-Ab complex (circulatory) to the surface of RBC leading to RBC removal by phagocyte.

***Babesia* of cattle:**

1. *B. bigemina:* Haemolytic anaemia
2. *B.bovis:* Kinin production is important
3. *B. divergens:*

4. *B.major:*

Babasia bigemina:

Causes cattle tick fever/red-water fever/piroplasmosis/ North American Texas fever/ Texas fever. It causes haemolytic anaemia. Found principally in bovine,zebu, water buffalo, alsodeer, white-tailed deer. This is a large piroplasm. Pear shaped and lie in pair forming an acute angle. Vectors are –

One host tick- *Boophilus annulatus*
B. calcaratus
B. microplus
Two host tick- *Rhipicephalus evertsi*
R. bursa
Three host tick- *Haemaphysalis punctata*
Rhipicephalus appendiculatus

Pathogenesis:

Young are symptomless, higher mortality in adult. Natural resistance of young calf disappears at 9-12 months of age. In young animal, parasite density is low parasite density. Incubation period is 1-2 weeks. First evidence of disease is rise in body temperature to 41-42^{0}C. High fever lasts from 2-7days or more. During this period profuse profound anaemia occurs. There is haemoglobinuria and cardiac palpitation. Profuse diarrhoea followed by constipation. Up to 75% RBC may destroy at the height of fever. Mortality is high in acute cases and death occurs 4-8 days after the onset of infection. If animal survive the acute phase, then go into chronic disease symptom. There is intermittent temperature rise. Temperature reaches 40-40.6^0 C. Animal become thin and emaciated, no marked haemoglobinuria and finally animal recovered. On post-mortem, subcutaneous and intramuscular oedema, icterus, fat is yellow in colour and gelatinous and blood is thin and watery. Urinary bladder is frequently red or dark brown. A inverse age susceptibility occur in *Babesia* infection. Young animals being naturally resistant while older animals are fully susceptible. Passive transfer of maternal antibodies via colostrums probably responsible for the resistance. Breed also plays an important role. *Bos indicus* has been suggested to be more resistant than *Bos Taurus*. Spleen plays an important role in maintaining the immune state to *Babesia* infection. Immunity may break down by removal of spleen.

Diagnosis:

1. Clinical sign-high fever and coffee coloured urine.
2. Detection of parasite in peripherial blood.
3. Immunological test like- Indirect Fluorescent Antibody test.
4. ELISA

Treatment:

1. Trypan blue (100ml of 1-2% solution in normal saline given I/Vly
2. Acriflavine -20ml of 5% aquous solution I/Vly

3. Pirevan (Babesan)= quinuronium sulphate :1ml of 5% solution s/cly per 50kg B.Wt. Intra venous injection is contraindicated.
4. Phenamidine: 12mg/kg s/cly in a 40% aquous solution.
5. Berenil: (4,4-diamidinodiazoaminobenzene aceturate)-2-3.5mg/kg by deep I/M injection.
6. Imidocarb
7. Recently exoantigen vaccine developed through MASP culture technique.

Control:

1. Controlof tick by regular dipping of cattle prevent clinical disease, give protection for 13 months,vaccine is antigenically strong for 3 yrs at 4^0C

Babesia bovis:

It is a small piroplasm. It occurs in cattle and buffalo of India. Transmitted by-

- ***Ixodes ricinus***
- ***I. Persulcatus***
- ***Boophilus calcaratus***
- ***Boophilus microplus***
- ***Rhipicephalus bursa***

Clinical Signs:

-High fever about a weeks-10days after infection
-Haemoglobinuria
-Perivascular perineural and interstitial edema occurred throughout brain and spinal cord.
-Convulsion, incordinatiion ad coma

Immunity is preimmunity: i.e. acquired immunity persists for several years in the absence of demonstrable organism and termination of a subclinical infection by drug treatment resulting in immunity to reinfection atleast for 6 month.

Spleen plays a important role in maintaining immune state to *Babesia* infection. Immunity may break down by removal of spleen. Antibody plays a critical role. It transmits thrugh colostrums to spread infection to young one.

Babesiosis in Sheep and Goat

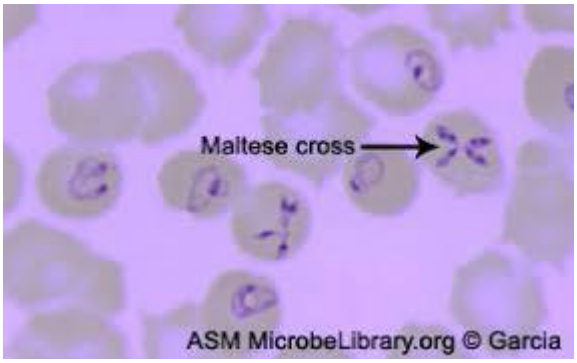
- *Babesia motasi*
- *Babesia ovis*

Are the predominant species causing high fever, haemoglobinuria, anaemia and even death. Diagnosis, treatment, control same with *B. bigemina*

Babesiosis in equine

-*B caballi* and *B equi*

B. caballi is large form found in pair inside the RBC. *B. equi* (small form), characteristically it remains as a cross which is known as “Maltose cross”. In this condition 4 nos of parasite (daughter) produce inside the RBC at a time from the mother organism.



Differentiation

B. caballi	***B. equi***
Less pathogenic	Highly pathogenic
Large form	Smaller form. Parasit divide in to 4 daughter form known as Maltose cross
High fever, anaemia, icterus and CNS involvement causing posterior paralysis. Haemoglonimuria is rare	Anaemia and haemoglobinuria are commonly marked bu CNS involvement is not a common feature.

Diagnosis is same with *B. bigemina*

Babesiosis in Dog and Cat:

- *B. canis*: large form
- *B. gibsoni*: small form

Both are transmitted by common dog tick *Rhipicephalus sanguineus.* The infection of *B. gibsoni* is much more chronic than *B canis* infection.

B. canis:

Following introduction of parasite by infected tick, they show different clinical sign. Involvement of circulatory system causing edema, ascitis, stomatitis, gastritis, nasal catarrah and dyspnoea. Affected dog may also show keratitis. In case of CNS involvement locomotor disturbances, epileptic form fits with other nervine signs are common. There is anaemia and jaundice. In advance case, haemoglobinuria observed. Faeces are highly yellowish in colour due to excess bile pigment. Urine also dark red in colour due to presence of lot of Billirubin. Puppies may also show clinical disease as severe as adults.

B. gibsoni:

Infection is more chronic which is characterised by intermittent fever, jaundice and haemoglobinuria. Death may take place after several weeks or even after several month of infection. Like *B canis,* infection there is also enlargement of spleen which is palpable during examination.

Treatment: Berenil is highly effective.

Control:
Treatment to affected dogs

Control of tick
Immunization