Babesia sp.

Host Specificity and Ecology

 The babesias are one of the most widespread blood parasites in the world based on numbers and distribution of species in animals, second only to the trypanosomes.

 They generally have two classes of hosts, an **invertebrate** and a **vertebrate** host. The maintenance of *Babesia* spp. is dependent on both hosts; the specific tick vector must feed on a vertebrate reservoir that is competent in maintaining the *Babesia* organisms in an infectious state.

 Most *Babesia* spp. are highly host specific and often splenectomy is needed to establish an infection in an unnatural host.

Mode of transmission in *Babesia* spp

§ **Transovarian transmission**

§ **Transstadial transmission** (stage to stage transmission)

• Transovarian transmission though several generations, although this varies with the species of *Babesia* and the species of tick.

• *B. divergens* can survive in tick populations for **at least 4 years** even if cattle are not present.

• When an infected tick attaches to a new host, *Babesia* are stimulated to undergo their final maturation.

Development in the vertebrate host:

§ All species of *Babesia* are transmitted by ticks.

§ Sporozoites are injected into the host together with the saliva of the vector tick and directly infect RBCs.

§ The sporozoites develop into piroplasms.

§ Multiplication usually results in two (sometimes four) daughter cells, which leave the host cell and each enters another red cell. Multiplication continues either until death of the host, or more usually until the immune system of the host puts an end to the multiplication of the parasite.

§ *Babesia* parasites do not form pigment in the parasitized cell, which distinguishes them straightaway from genera such as ***Plasmodium*** and ***Haemoproteus***; apparently *Babesia* species digest hemoglobin well enough so as not to leave such residues

Development in the tick vector

§ The tick becomes infected when ingesting blood cells containing piroplasms, which should probably be considered as gametocytes.

§ They develop into male and female gametes in the tick gut. The microgametes fuse with macrogametes to form motile zygotes.

§ In *Babesia* the zygotes then multiply and the ‘‘vermicules’’ which result invade numerous organs of the tick, including the ovaries.

§ In this way, the infection passes through the ovary and the egg to the next tick generation. This is called **transovarial transmission**.

§ It is usually the female tick that becomes infected and sporogony takes place in the salivary glands of larval, nymphal and/or adult ticks of the next generation.

§ **Transstadial transmission** (the infection passed to the next stage) also occur.

§ When the tick attaches to a new host, maturation of the sporozoites takes place and the host is infected with saliva from the tick.

§ *Babesia* as well as *Theileria*, the transmitting tick is not immediately infective after attachment, the sporozoites first have to mature before they are infective, and often actual transmission only occurs a few days after the tick has attached.

***Babesia* in Cattle**

• **Babes** (**1888**) investigated disease outbreaks causing haemoglobinuria (red water fever) in cattle in **Romania** and was the first to describe piroplasms in the blood of cattle ***Haematococcus bovis* (=*Babesia bovis*)**

• Shortly afterwards investigations by **Smith and Kilborne** (1893) in the USA demonstrated the causative organism of 'Texas Fever' (babesiosis) which they called ***Pyrosoma bigeminum*** (=***Babesia bigemina***).

• They were the first to demonstrate transmission of a disease organism from an arthropod to a mammalian host when they showed the organism was transmitted by ***Boophilus annulatus*** to cattle (**Smith & Kilborne, 1893**).

• Four species of bovine *Babesia* are now recognized:

1. ***Babesia bovis*** (=*B. argentina; B. berbera; B. colchica*),
2. ***Babesia bigemina***
3. ***Babesia divergen***

Pathogenesis

 Release of pharmacologically active substances and the destruction of RBCs play a major role

 Plasma kallikrein levels rise markedly 3 days after infection and then fall to subnormal levels terminally.

 kallikrein production increases vascular permeability and vasodilation triggering coagulation leading to circulatory stasis and shock.

 PCV drop due to *B. bovis* infection is attributed to disturbances rather than RBCs destruction.

 Anaemia

 Glomerulonephritis

 Central nervous system damage (*B. bovis* and *B. canis*)

Clinical signs in cattle

§ Anorexia

§ High fever 41-42 °C

§ Haemolysis and anaemia

§ Haemoglobinuria

§ Cardiac palpitation

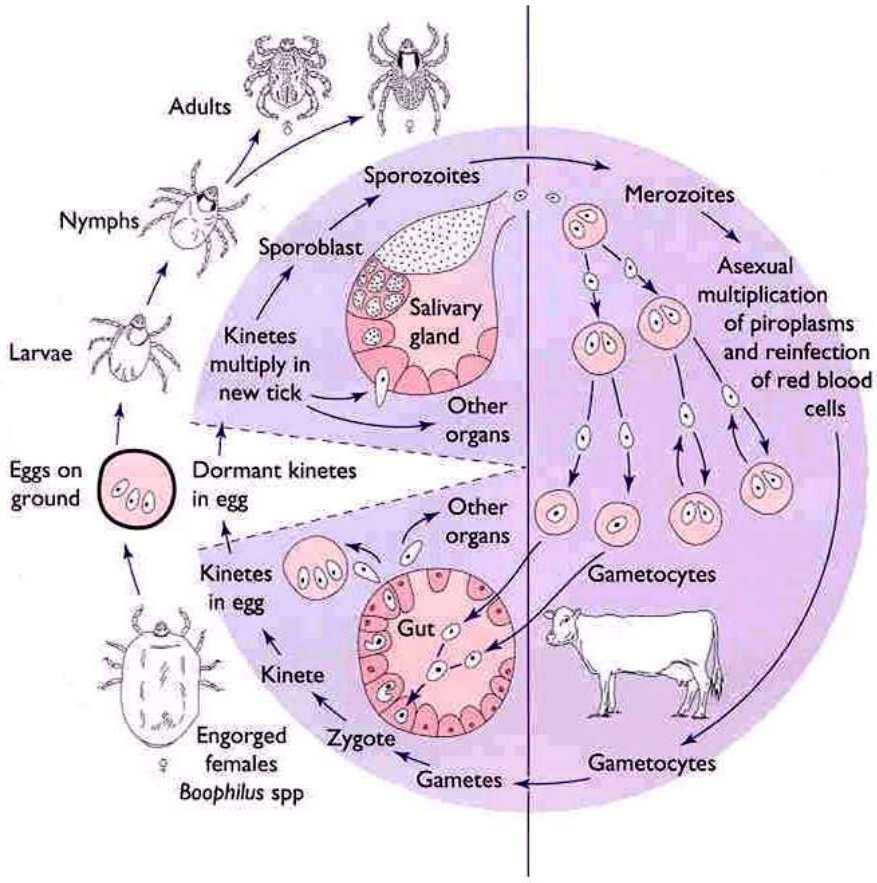
§ Intitially profuse diarrhoea followed later by marked constipation

§ Mortality is very high in acute cases

§ In chronic cases there are irregular intermittent temperature rises

§ Animal become thin and emaciated

§ Animal may recover



Diagnosis

- stained blood smear
- serological examination（including：IHA、ELISA）
- post mortem examination
- gene diagnosis（for example: PCR)

Treatment

- 1.diminazene
- 2.acriflavine
- 3.imidocarb

Theileria sp.

Theileria are tick transmitted intracellular protozoan parasites of domestic and wild animals.

Pathogenic *Theileria* species

Large ruminants

T. Parva : East Coast Fever (ECF)

T. annulata: Tropical theileriosis or Mediterranean Coast Fever (MCF)

Definitive host

Cattle - Buffalo - Sheep and goat

Transmission

Rhipicephalus appendiculatus is the main field vector

Rhipicephalid are three (3) host ticks, & transmission occur from stage to stage, transovarian transmission dosenot occur.

Tick sremains infected on pasture for two(2)years.

Theliria spoozitearetransmittedfromsalivaoftickstothedefinitivehost.

The parasite dies out faster in hot climate &in nymphs compared with adults.

Life cycle

1.*Theileria* sporozoites are inoculated into a mammalian host when an infected tick takes a meal Sporozoites invade lymphoid cells.

2.The schizont stage induces host cell transformation and proliferation leading to clonal expansion.

3.Schizonts differentiate into merozoites,

4.which invade erythrocytes.

5.Gametogenesis and fertilization takes place in the gut lumen of feeding tick

6.The resulting zygote invades a gut epithelial cell and develops into a single motile kinete.

7.The motile kinete egresses the gut cell

8.and invades the salivary glands.

9.Tick feeding initiates rapid sporozoite development in the salivary glands, and infective sporozoites are emitted during the later stages of feeding.

Clinical signs

ThefirstclinicalsignofECFincattleappears7to15daysafterattachmentofinfectedticks.Clinicalsignsare:

1)Swelling of the drainin glymphnode, usually thep arotid.

2)Generalized lymphadenopathy.

3) Fever[103ºF(39.5ºC)]

4) Other clinical signs are: Lacrimation, Cornealopacity ,Nasal discharge ,Terminaldyspnea & Diarrhea.

Gross lesions

1)A frothy exudates is frequently seen around hen ostrils of an ECF-infected animal.

2)Lymph nodes are greatly enlarged & maybe hyperplasic, hemorrhagic & edematous.

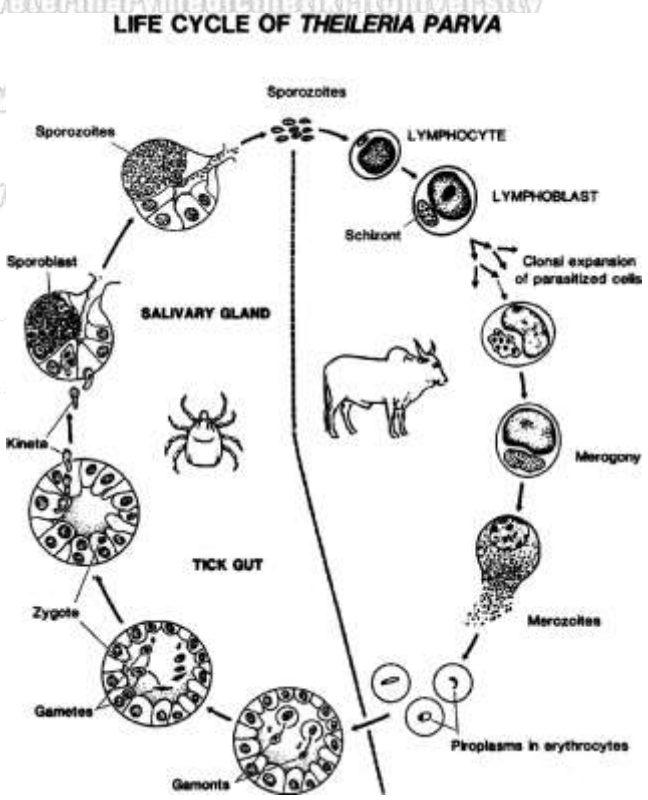
3)In acute cases of ECF ,lymph nodes are edematous &hyperemic.

5)Sero salsurfaces have petechial & ecchymotic hemorrhages.

6)Ulceration may be seen through out the GIT.

7)Lymphoid cellular infiltration appears in the liver &kidney, inter lobular emphysema & sever pulmonary edema appear.

8)The lungs are reddened & filled with fluid,& the trachea & bronchi are filled with fluid and froth



Diagnosis

a) Field Diagnosis

- In field, diagnosis is usually achieved by finding Theileria parasites in Giemsa-stained blood smear & lymph node needle biopsy smear.

b) Specimens for Laboratory

- Specimens consisting of buffy coat smear air-dried.
- Lymph node impressions air-dried.
- Lymph node, spleen, lungs, liver and kidney samples for histopathology.

Treatment

- There are three effective drugs for the treatment of ECF:

1) Parvaquone (Clexon)
2) Buparvaquone (Butalex)
3) Halofuginone lactate (Terit)