FIELD VETERINARIENS BAFFLE: THEILERIOSIS AND BABESIOSIS

Introduction: Theileriosis and Babesiosis are the two major tick borne hemoprotezoan diseases of cattle, particularly of crossbreds. Bovine Tropical Theileriosis is a serious constraint in the upgradation programme as the high yielding European cattle and their crossbreds are highly susceptible to the disease. Annual economic losses due to Theileriosis alone have been estimated to the tune of $800 million. *T. annulata* also produces cutaneous form of Theileriosis in cow calves and it is responsible for heavy mortality. Babesiosis causes heavy mortality in the adult animals responsible for heavy economic losses to the cattle owner.

Transmission:

Theileriosis: Theileriosis in Indian bovines is mainly caused by *Theileria annulata* and the disease is known as Bovine tropical Theileriosis. The chief vector responsible for transmission is *Hyalomma anatolicum anatolicum* and other ticks species belonging to the said genera. The transmission of theilerial particles from one stadia to another stadia takes place called as transtadial transmission. The theilerial particles can not survive for more longer period in the tick and hence no transovarian transmission occurs.

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Life cycle of *Theileria*

- **Bite of tick (nymph/adult)**
- **Release of sporozoites**
- **Multiplication in lymphocytes and formation of Koch's blue body**
  - **Macrophages** (Agamonts)
- **Release of microshizonts in to RBCs**
- **Formation of Pyroplasms in RBCs**
  - **Leading to parasitaemia**
- **Bite to the affected host by tick**
  - **Formation of Male and Female gamete**
  - **Formation of Ookinete**
  - **Sporogony and Sporozoites in salivary glands**
  - **Bite to the another host causing infection**

**Diagnosis should be done at this juncture**

**Host cattle**

**Tick vector**

**Babesiosis**: In India bovine Babesiosis is mainly caused by two species

- *Babesia bigemina*  
  - large form
- *Babesia bovis (=B. argentina)*  
  - small form

The species are transmitted mostly by one host tick *Boophilus microplus*. The transmission is transovarian. Once the adult female tick is infected it can transmit the infection for 32 generations\(^8\). Hence *Babesia* are called parasites of ticks rather than of vertebrate host. Another important point is that no multiplication is known to occur in the vertebrate host. Whatever multiplication is known that is in ticks. Hence *Babesia* can survive and propagate without the vertebrate host. Male ticks, nymph and adult female can transmit the disease.
Life cycle of Babesia

Life cycle of Babesia

Bite of tick to the infected positive cattle (host)

Tick consumes Babesia pyroplasms

Blood is digested in gut epithelium of tick and Babesia released and enter in gut epithelium

Multiply through multiple fission in the gut epithelium cells

Cells resulting after fission body is called Vermicules

Vermicules crosses gut epithelium and enters in haemocoel

2nd schizogony occurs in haemocytes.

If it is one host tick then schizogony occurs in the oocytes of ovarian cells

Pathogenesis

Theileriosis: Incubation Period is 10-25 days. Due to schizogony in the lymphoid tissue hyperplasia occurs causing swelling of lymphnodes. Due to sudden release of toxins of macroschizonts high rise in body temperature and formation of the ulcers on the mucosal layers of abomasum. In acute cases there is disponea and death due to anaemic anaemia.

Disease runs peracute, acute, sub acute or chronic course. In exotic animals, the peracute form of the disease may be met with when the animal feeds and ruminates normally a day earlier and dies on the next day with a high rise of temperature. Death rate ranging from 10 to 90 percent may occur. Cases of cerebral involvement may be seen. The disease may commence with 40 to 41.7°C temperature which continues till the death or cure of the animal. The superficial lymph nodes are swollen.
Leucopenia is not characteristic of the disease. There is progressive leucocytosis which is entirely due to lymphocytes. The RBC infection is high. The low RBC count is attributed to the removal of infected erythrocytes by spleen and liver and not due to the destruction of erythrocytes by the parasite.

**Babesiosis**

Highlighted pathogenesis is

1. Anemia and icterus due to three fold loss of RBCs,
2. Hæmoglobinuria due to overload of the hæmoglobin in the kidneys
3. Glomerulonephritis and Hæmoglobinuric nephrosis due to the immunological complexes lodged in the glomerulli and death due to organic failure.

Besides the clinical manifestations, the disease can induce severe haemolytic anaemia due to heavy destruction of infected red cells, removal of non-infected erythrocytes by phagocytes, increased fragility of non infected RBC during *B. bovis* infection and adsorption of circulating antigen antibody complexes to the surface of RBC leading to RBC removal by phagocytosis. In addition to anaemia, detectable haematological changes include significant decrease in Hb, PCV and TEC within 48 hours of infection. Leucocytosis with neutrophilia occurs in the beginning which come down to normal after 12-15 days of infection. Parasitaemia is associated some what with the rise in body temperature.

Substantial increase in plasma kallikrein, kinin and kininogens has been observed. The increase in these values has been attributed due to activation of kallikrein system with an enzyme contained in *B. bovis* extracts. Kallikrein produces vasodilation intravascular coagulation and vascular permeability leading circulatory stasis and shock. Increase in albumin and bile pigment has been reported during *B. bigemina* infection in cattle.

Observations on blood glucose, urea nitrogen, bilirubin and transaminases have indicated significant increase suggesting considerable liver damage.
Central nervous system damage has been ascribed due to concentration of parasitized cells in brain capillaries leading to obstruction of blood flow and adherence of infected cells to one another and to the vessel endothelium probably due to a parasite born antigen which alters the surface charge of RBC.\(^6\)

Some of cross-bred calves died manifesting paddling of feet, grinding of teeth, convulsions and coma. An important observation in such animals was distension of cerebral capillaries due to clumping of \textit{B. bovis} parasitized erythrocytes. Haemoglobinuria and appearance of parasites in peripheral blood smears were not to be necessarily present in such cases. However, when haemoglobinuria was evident \textit{B. bovis} parasites were detected in varying concentrations in smears from heart, kidney, liver etc. in addition to brain.\(^25\). Natural cases of cerebral babesiosis due to \textit{B. bovis} in cross bred bovines have also been discovered\(^25\)\(^26\)\(^30\). The Babesiosis disease is intertransmissible from buffalo to cattle and vice versa\(^6\).

**Clinical signs**

**Babesiosis**: Following signs and symptoms are observed\(^1\)\(^20\)\(^23\)\(^33\).

- Fever - 41 - 45.5\(^\circ\)C, anoxeria, increase in respiratory and heart rate.
- Anaemia, jaundice, Haemoglobinuria (Coffee coloured urine)
- Either constipation or diarrhea, weight loss
- Abortion in pregnant animals.
- Nervous symptoms in \textit{Babesia bovis} infection in calves.
- Coma and death due to anaemic anoxia.
- In chronic cases become carrier

**Theileriosis**: Following are the symptoms

- High Fever after the bite of tick
- Capricious appetite (feeding till death), drop in milk production, cessation of rumination.
- Enlargement of superficial lymph nodes (may go upto coconut size).
- Rough hair coat, excessive salivation, dry muzzle.
- Respiratory distress, pulmonary oedema.
- Death due to anaemic anoxia.
- High mortality.
- Haemoglobinuria, terminal stages.

**Epidemiology**

**Babesiosis:**

1. The age of animal especially in enzootic areas shows that adult animals suffer more than the young animals. Young animals have preimmunition and mostly suffer from latent symptomless infection but reports of fetal infection in newborns are not uncommon.

2. Seat of predilection of the parasite e.g., *B. bovis (=B. argentina =B. berbera)* attacks more to capillaries of the brain and kidneys and thus will give rise to different set of symptoms.

3. Babesiosis is a four component system in which the host, environment, parasite and vector play their role in the spread of the disease.

4. During the summer season, when the environment is conducive for the development of ticks, the disease may raise its head. However, found that disease is more prevalent in monsoon season followed by summer and winter.

5. The control of vector has direct bearing on the control of the disease. The control of ticks is bound to create instability in the clinical manifestation of the disease.

6. Sub clinical disease is more present in calves below 6 months and adults of 2 years and above.

**Theileriosis:**

1. Theileriosis is a four component system in which host, environment, parasite and vector play their role in the spread of disease.
(2) The occurrence of tropical Theileriosis (T. annulata) is seasonal and coincides with the incidence of ticks on the host, which is very high in summer i.e., from May to October. The stress due to extreme of host climate, transportation, intercurrent disease, vaccination etc., may be contributory factors.

(3) The vector of T. annulata is generally Hyalomma anatolicum anatolicum, found in desert, semi desert and steppe. It occurs from North West Africa eastward to India. It is a three host tick and both immature and adult stages occur on cattle. The adults are active in late spring. The immature stage of H. anatolicum feed on most of the animals and larvae and nymphs can acquire infection by feeding on infected cattle and transmit infection after molting when they feed as nymphs or adults. The season of adult feeding and thus of disease incidence in widely spread from March to November, but the nymphs have a more restricted season from July to September. The seasonal incidence of T. annulata is commonly in late spring and early summer.

**Diagnosis**

**Differential diagnosis**

<table>
<thead>
<tr>
<th>Theileriosis</th>
<th>Babesiosis</th>
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<tbody>
<tr>
<td>1 Diseases of young cross breeds</td>
<td>1 Diseases of adult dairy animals</td>
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<tr>
<td>2 Pyroplasms are very small</td>
<td>2 &gt; 2.5 – 5.0 m they are pyriform</td>
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<tr>
<td>&lt; 2.5 m they are ovoid, annular, ring or rod shaped.</td>
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<tr>
<td>3 Schizogony in lymphoid tissue of vertebrate host and gaemetogony and sprogony in tick.</td>
<td>3 Schizogony in tick, No multiplication in vertebrate host is known.</td>
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<tr>
<td>4 Trans-stadial transmission</td>
<td>4 Trans-stadial and trans-ovarian transmission.</td>
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<tr>
<td>5 Hb uria is occasional</td>
<td>5 Hb uria is characteristics</td>
</tr>
<tr>
<td>6 Tick transmission agency is Hyalomma anatolicum</td>
<td>6 Tick transmission agency is Boophilus microplus</td>
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</tbody>
</table>
7 Superficial lymphnodes are highly swollen and enlarged
8 Mucous membranes very pale
9 Skin occasionally urticarial
10 Nervous signs absent or occasional

Based on history of disease in the particular area and presence of ticks

Symptoms: Theileriosis: Enlarged lymphonde, fever
           Babesiosis: High fever, Hb uria, jaundice

Microscopic examination of blood smear:
1) Thin blood smear: During the acute febrile condition, blood smear should be collected and stained with Giemsa stain.
2) Thick blood smear: when low grade infection is there. Fix the smear with METHANOL, AIR DRY, STAIN WITH GIEMSA STAIN FOR 15-20 MINUTES AND OBSERVE UNDER OIL IMMERSION. THE WATER USED FOR STAINING SHOULD HAVE pH AROUD 7.2 OR SOFT DRINKING WATER SHOULD BE USED.
3) Direct wet-mount preparation using hypotonised erythrocytes for intraerythrocytic parasites like Theileria and Babesia can be employed. A small droplet of blood is taken on slide, allow to mix with a smaller droplet of distilled water under a cover slip. The distilled water makes the erythrocytes swell and lose their biconcave shape. This helps to prevent refraction of light which obscures the intra-erythrocytic parasites. The examination has to be done using the oil immersion lens in subdued light to appreciate the live and motile Theileria and Babesia organisms. Use of 'Anisole' or liquid paraffin instead of cedar wood oil will be more convenient and satisfactory.
4) Make fresh blood smears (both thin and thick) on clean slides, dry quickly and fix them with methanol before shipment to the laboratory.
5) Lymph node aspiration smears (For Theileria). Plunge a 18 gauge needle into the gland firmly held between the thumb and index finger. Aspirate a few drops of grey coloured liquid often mixed with a little blood) using a 2 ml syringe. Make smears on clean glass slides and dry. Fix with methanol stain and observe for KOCH BLUE BODIES. For diagnosis of Babesia bovis brain smears are preferred 5.

Serological examination:
1) CFT, FAT, formol gel
2) ELISA
3) PCR

Post mortem changes

Babesiosis:
- Generally the carcasses are pale and emaciated
  - Thin and watery blood
  - Edematous and yellowish subcutaneous, sub serous and IM tissue.
  - Icteric discoloration of organs.
  - Enlarged liver and distended gall bladder with dark green bile
  - Kidneys enlarge and dark
  - Edematous lungs.
  - Distended urinary bladder with coffee coloured urine.
  - Characteristics lesion ..... intravascular clotting.
  - In chronic cases Hb uria is absent.

Theilerosis 1
- Generalized enlargement of lymph nodes (edematous / haemorrhagic)
- Enlargement and haemorrhagic spleen.
- Haemorrhages on serous and mucous membrane.
- Enlarged yellowish and mottled liver.
- Ulcer on mucosa of abomasm
- Massive pulmonary oedema hyperemia and emphysema of lung.
- Hydrotharax, hydropericardium
- Pulmonary edema.

**Treatment:**

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<tr>
<th>Sr. No.</th>
<th>Name of the drug</th>
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<tr>
<td></td>
<td><strong>Theileriosis</strong></td>
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<tr>
<td>1</td>
<td>Buparvaquone 2.5 mg/kg Bw deep I/M</td>
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<tr>
<td>2</td>
<td>Oxytetra cycline (LA) 20 mg/kg BW deep I/M</td>
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<tr>
<td></td>
<td><strong>Babesiosis</strong></td>
</tr>
<tr>
<td>1</td>
<td>Diminazine Aceturate (3-8 mg/kg BW) deep I/M</td>
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* Do not use any other route than intra muscular
* Therapy can be repeated after 48-72 hours depending on severity of infection
* Initiate the therapy as early as possible; otherwise animal succumbs to anaemia despite sterilization of blood.
* Supportive therapy with liver extract, Vitamin B-complex, iron preparations
* Blood transfusion if Hb level drops below 6g/dl

**Control:** It can be done in three ways

- Chemotherapy
- Control of Babesiosis and Theileriosis
- Chemoprophylaxis
- Vaccination
- Tick Control
Chemoprophylaxis: For Theileriosis buparvaquone is the current drug of choice given at the dose rate of 2.5 mg/kg IM at the age of 7-10 days or 8-25 days or at the age of 30 days.

Vaccination: Raksha vac T vaccine to be given at the age of 2 months.

Tick Control: Tick control is important step in the control of both diseases.

Physical Control of Ticks:
1. Burning of pasture at the end of grazing season.
2. Burning of cracks and crevices in the cattle shade
3. Hunting of bushes in the grazing land.
4. Rotational grazing.
5. Zero grazing for calves
6. Good management of calves during first 3-6 months of age
7. **Tick Proof House**: When valuable crossbred animals are reared under confinement, a tick proof house can be constructed. No cracks & crevices in building. An acaricide channel should encircle the entire building and should be kept always filled with suitable acaricide solution.
8. **Fumigation** of building before or beginning of monsoon season.

Chemical control of ticks: Following acaricides can be used.
1. Amitraz, Chlorfenvinphos, Crotroxyphos, Chlorpyrifos, Coumaphos, Cypermethrin, deltamethrin, diazinon, permethrin, phosmet, dichlorovos, trichlorophon

Spray, dips, slow release ear tags, leg tags, tail tags, should be used.

2. Injection of Ivermectin at the dose rate of 200 μg/kg body weight S/C.

3. At the same time the acaricide spraying on the animal and house should be carried out.

Bio-pesticides control of ticks:

1. Neem products (Neem oil, NSKE, neem extract)
2. Karanj oil
3. Combination of these two in 1:1 proportion

References


