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Theileriosis: A Review Article

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Introduction

Theileriosis is a parasite infection spread by a vector. *Theileria* species are to blame, primarily *T. annulata* and *T. orientalis* in India. These intracellular parasites are obligatory. It has a complex life cycle in both vertebrate and invertebrate hosts and is primarily transmitted by ticks. They are quite similar to babesia; however, they are different in that babesia goes through an erythrocytic stage of development before moving on to leucocytic stage. One of the most serious hemoprotozoan parasitic conditions, it can cause productivity loss, economic loss, and, in extreme situations, death.

Theileria parasitic protozoan parasites are the source of a category of tick-borne disorders known as theilerioses. Theileria spp. are widely distributed throughout tropical and subtropical regions of the world in domestic and wild ungulates. The two most significant cattle-affecting species are T. parva and T. annulata, which produce acute diseases with substantial fatality rates. In addition to T equi occasionally causing clinical disease in horses, T lestoquardi, T luwenshuni, and T uilenbergi are significant sources of death in sheep. The infections are treatable with the medicine buparvaquone, although it is pricey, and the treatment of the diseases typically entails either preventing tick infestation or, in some locations, immunization.

Theileria and Babesia are both members of the Piroplasmorina suborder. Although Theileria employ WBCs and RBCs in succession to complete their life cycle in mammalian hosts, Babesia are primarily RBC parasites. The parasite's contagious sporozoite stage is spread by the saliva of infected ticks while they eat. Leukocytes are invaded by sporozoites, which quickly transform into schizonts. Theileria species that are more likely to cause disease, such as T parva and T annulata, mostly multiply inside the host's WBCs, whereas less dangerous species do so primarily in RBCs. Development of the pathogenic schizont stage When the host WBC divides as a result of the parasite, the parasite also divides. As a result, the population of

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parasitized cells increases and spreads throughout the lymphoid system as a result of migration. Some of the schizonts eventually go through merogony later in the infection, releasing merozoites that infect RBCs and give rise to piroplasms. The prologue to a complicated cycle of development that culminates in transfer of infection by ticks eating in their next instar (trans-stadial transmission) is the uptake of piroplasm-infected RBCs by vector ticks feeding on sick animals. As opposed to Babesia, there is no transovarial transmission.

Theileria spp comes under Phylum: Apicomplexa, Order:Piroplasmida, Family: Theileriidae, Genus: *Theileria* and Species: *Theileria annulata* and *Theileria orientalis*, preferably in India.

Species susceptibility

Theileria annulata and Theileria parva are more pathogenic and cause economic damage. Cattle of European (Taurine) breeds are more prone to theileria than those of native Zebu breeds. Cattle are the principal reservoir host for T. annulata. Although it can also harm sheep and goats, it rarely causes serious sickness in these smaller ruminants. There are less piroplasms in the erthrocyctes of sheep and goats, and they have fewer chances of spreading them to ticks. When it comes to large ruminants, T. lestoquardi, T. uilenbergi, and T. luwenshini are less susceptible.

Source of agent

In this infection the ticks contain sporozoites in the salivary glands. The parasite's life cycle includes an asexual developmental stage in mammalian hosts and a sexual phase in tick vectors. Through poorly understood molecular mechanisms, the parasites drive replication after infiltrating host leukocytes and reprogramming several host signaling pathways. T. annulata multiplication within red blood cells results in anemia, which may exacerbate the disease's pathogenesis.

Epidemiology

T. annulata and *T. orientalis* are more common in India. In India, one of the rising diseases is *T. orientalis*. Theileriosis is thought to be an endemic illness. It is not primarily present in places like Jammu and Kashmir and the Himalayan regions, but there is a chance for infection to spread through the movement of animals from endemic places like Punjab, Haryana, etc. Additionally, there are no hyalomma species ticks reported in the Himalayan regions.

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Transmission

It mostly happens because of ticks. It functions as a biological vector, which indicates that the parasite's developmental stage occurs there. The saliva of infected ticks allows *Theileria* to enter the host's body. The attachment of ticks does not transmit T.parva and T. annulata to the host. After the adhesion of infected ticks, the development of the sporozoites takes 48–72 hours (or a few days). On the ground, *T. annulata* might enter the infectious stage when the temperature is higher. Transovarial transmission does not take place. The animals that were found may be carried for months or perhaps a year. In this medical condition, transplacental transfer is a possibility.

Life cycle

In mammalian host, Sporozoites in salivary gland vomits infection in blood which enters leucocytes forming macroshizonts, proliferation of parasitized cells followed by merogony and then merozoites released from leukocyte forming piroplasms in erythrocyctes

In tick vector: male and female micro and macrogametes formed which fuses to form zygote which ultimately leads to formation of sporozoites in salivary glands of ticks invading mainly acinar cells in salivary glands. Scizonts stage is found in leukocyctes and piroplasm stage is found in erytrocyctes.

Clinical signs

Lymadenopathy, or lymph node enlargement, typically affects the parotid, pre-scapular, and pre-femoral lymph nodes. The vector's preferred feeding location is, in essence, the ear. It is easy to see and feel swollen lymph nodes. Ticks showing up in the ear flaps, valvular lips, neck, and dewlap region of infected cattle The skin of buffalo is unusual because it is relatively thicker than that of cattle, which allows ticks to cling to ear flaps and cause the parotid and supraorbital lymph nodes to grow. Pyrexia is observed and persists during the infection. Petechial and ecchymotic hemorrhages in the buccal cavity and conjunctival mucous membrane. Petechial hemorrhages in the valvular region in outdoor conditions. Bilateral exophthalmas with scleral hemorrhages. Pale color of the mucous membrane, which will vary depending on the disease's severity and progress. Anoxia. Ocular and nasal discharge, lacrimation, and cornal opacity are further clinical symptoms. Additionally described symptoms of tropical theileriosis include anemia and jaundice. Later on, hemorrhagic diarrhea is detected. Weight loss brought on by muscular atrophy. All of the clinical symptoms of T.

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tremor on the east coast are the same, and infected cells begin to clog brain capillaries, causing turning sickness in fetuses, which then causes neurological symptoms.

Postmortem lesions

In macroscopic examination, the spleen and lymph nodes are edematous and enlarged. Petechial and ecchymotic hemorrhages on the serosal and mucosal surfaces of the visceral organs. Yellow colouring of tissue from jaundice (particularly in buffalo, where white fat turns yellow in cases of *T. annulata*. Additionally, the liver may expand and white infiltration foci may be discovered. The accumulation of fibrin in visceral organs. Punched out ulcers in the abomasum are a key pathognomic feature of theileriosis. Frothy fluids in the trachea, bronchi, and nasal passages. Congestion, hemorrhages, and malacia in the area of the brain are symptoms of turning sickness.

In, Microscopic examination, Lymphocytic cell proliferation in the liver, kidneys, spleen, peyer's patches, and lymph nodes. Parasitic lymphocytes are seen inside the blood arteries of affected animals. It also contains the blood vessels in the brain. Various organs' tissue portions may include Koch's bodies.

Molecular methods like reverse blot assay and polymerase chain reaction are used in laboratory diagnostics together with blood smears and microscopic examination for Koch blue bodies. In the event of a serological test, indirect fluorescent antibody tests (IFA) and enzyme-linked immunosorbent assays (ELISA) are used.

Treatment

Intramuscular injection of buparvaquone 2.5 mg/kg body weight. The medicine is antiprotozoal. Oral supplementation with ECA durability in cases of anemia. Oxalated lactic acid is preferable for milk production that is producing at a slower rate. The liver is also affected, thus a livertonic is required. Anabolizing drugs. The anti-histamine CPM, commonly known as chlorphenamine or chlorpheiramine, is also administered. However, oxytetracycline induces a serological reaction and an immunological response to a 50-fold larger challenge when combined with an immunization stabilizer, whereas buparvaquone does not have this kind of impact. If in case of oxytetracycline is used in the treatment the best way of giving it in IM and not in IV it may lead to cardiac problems.