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Bovine babesiosis: A general review

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Abstract
The health and production in domestic animals are influenced by various factors; among them parasites constitute the most important pathogenic agents. Tick-transmitted hemoparasites of the protozoan genus *Babesia* (phylum Apicomplexa) are the second most common blood-borne parasites of mammals after the trypanosomes. In worldwide, Babesiosis is one of the most important tick-borne disease caused by *Babesia* spp. occur in cattle which causes economic losses to the farming community by reducing the milk and meat production Bovine Babesiosis is characterized by high fever, hemoglobinuria, anaemia, inappetence, pink pale to pale mucous membrane, swollen lymph node and suspended rumination with mild to moderate tick infestation. Detection and treatment of babesiosis are important tools to control babesiosis. Microscopy detection methods are still the cheapest and fastest methods used to identify *Babesia* parasites although their sensitivity and specificity are limited. For years, babesiosis treatment has been based on the use of very few drugs like imidocarb or diminazene aceturate. Controlling and removing babesiosis's vector, the Boophilus tick, is the most effective way to fight the disease. Tick prevention with acaricides, immunisation of vulnerable stock, chemoprophylaxis, treatment of infected livestock, stock movement control, and raising tick-resistant cattle are some of the latest control measures used.

Keywords: babesiosis, anaemia, hemoglobinuria, diminazineaceturate, cattle, tick-borne

Introduction
Bovine Babesiosis is a febrile, tick-borne disease that affects cattle and buffalo. The acute type is characterised by rapid parasite growth and multiplication in the blood, as well as severe erythrolysis, which causes anaemia, icterus, hemoglobinuria, spleen enlargement, and, in some cases, death (Gray *et al*., 1985 and Deepak *et al*., 2019) [1, 2]. The term "Babesiosis" refers to the subclinical and chronic infections that usually persist after the parasite has been attacked. Clinically, the chronic type is characterised by anaemia and varying weight loss. From an economic standpoint, Babesiosis has greatest impact on cattle. Many different species have been described since *B. bovis* was first described in Romania by Babes in 1888 [3]. Bovine babesiosis is a major impediment to livestock improvement programmes in endemic areas because the greatest losses occur in fully susceptible cattle introduced into enzootic areas. Abortion, loss of bull fertility, decrease in milk production, and treatment costs are all outcomes associated with an outbreak (Salem *et al*., 2016) [4]. The distribution of vector ticks, particularly in tropical and subtropical regions, is linked largely to bovine sickness. The disease is only spread by ticks, which pick up *Babesia* infections from infected animals and then pass them on to other healthy animals after a blood meal. Tick infections can be passed on to future generations through the eggs (Homer *et al*., 2000) [5]. Tick infections can be passed down to future generations via the eggs. This disease can be found almost anywhere on the planet. Ticks have a wide variety of hosts, ranging from reptiles, birds, and small animals like rodents to large domestic and wild animals like elephants, because of the climatic conditions in tropical countries like India. Ticks are difficult to eradicate from nature due to their wide host range, and their infestation on domestic animals will continue to be a problem.

Etiology
Protozoan parasites of the genus *Babesia*, order Piroplasmida, phylum Apicomplexa, cause bovine babesiosis. *Babesia bovis*, *B. bigemina*, *B. divergens*, and *B. majo*r are the most likely *Babesia* species to cause bovine babesiosis (Gray *et al*., 1985 and Homer *et al*., 2000) [1, 5].

B. bovis and *B. bigemina* are the most common and important species in tropical and subtropical areas, respectively. In the tropics and subtropics, *B. bovis* is more pathogenic.

Hosts

Cattle are the most common hosts, but other ungulates such as water buffalo, African buffalo, and other ungulates may also be infected. Such hosts are unlikely to be major infection reservoirs. While native cattle (*Bos indicus*) have a high level of resistance to babesiosis, crossbred cattle are susceptible enough to warrant the use of control measures. Susceptibility to *Babesisa spp*. infection reduces with age, but the incidence of clinical disease increases (Homer *et al*., 2000) [5]. Calves from immune mothers are shielded from babesiosis for up to six months due to the presence of innate immunity. Infection is rare in animals over the age of five years. *B. bigemina* is mainly transmitted to animals under the age of one year while *B. bovis* occur for those animals with age more than two years (Bhikane *et al*., 2001) [6]. The most vulnerable to infection are the cattle which are immune-compromised and stressed (pregnancy-poor conditioned).

Life cycle

Only animals in the active stages of infection have viable protozoa in their bloodstream. Ticks are the natural carriers of babesiosis, with the causative parasites remaining in the invertebrate host for a part of their life cycle. The tick *Boophilus microplus* (now known as *Rhipicephalus spp*.) hosts both *B. bovis* and *B. bigemina* for a portion of their lives. Babesiosis is mainly spread by *B. annulatus* and *B. microplus* (Homer *et al*., 2000 and Dipeolu *et al*., 1984) [5, 7]. Except for erythrocytes, *Boophilus spp.* and *Babesia spp*. parasitize no other vertebrate host cell. Each sporozoite penetrates the erythrocyte's cell membrane, where it begins to develop. Each sporozoite penetrates the cell membrane of an erythrocyte, from which two merozoites form. *Babesia*'s developmental stages are trophozoites. The creation of two populations of ray bodies from the gametocytes occurs during the passage of the host blood to the tick vector's mid gut. The ray bodies continue to multiply, and when division is complete, they fuse to form a zygote. The zygote infects the tick gut's digestive cell, where it multiplies and develops into kinetes that escape into the tick hemolymp. After escaping into the hemolymph, these motile club-shaped kinetes infect a number of cell types and tissues, including the oocyte. These motile club-shaped kinetes then escape into the hemolyph and invade a range of cell types and tissues, including the oocytes, where secondary schizogony processes repeat themselves. As a result, transovarial transmission happens as the larval stage develops. When kinetes invade the salivary glands and transform into sporozoites, the infected tick's development normally stops before the vertebrate host is infected (Achuthan *et al*., 1980) [8]. Infection can be spread physically by contaminated needles and surgical instruments.

Clinical signs

Calves are usually fairly immune to clinical disease. Medical symptoms can be very serious in older animals. The clinical symptoms of *B. bovis* and *B. bigemina* infections are similar, but *B. bovis* is more pathogenic and central nervous system involvement is more common, while *B. bigemina* infections are normally less serious, but red urine appears earlier and more frequently than *B. bovis* infections. Infections with *B. bigemina* rarely produce nervous symptoms. The severity of clinical symptoms develops in direct proportion to the level of parasitaemia. The incubation period lasts from 1-3 weeks, with a high mortality rate of up to 60% in fully susceptible animals (Radostits *et al*., 2000) [9]. The length of the course is usually between two and three weeks. Subclinical infections are fairly common in cattle, especially in young animals. Signs may be acute and serious in clinical situations, or mild with parasitemic episodes lasting 1-3 weeks (Homer *et al*., 2000) [5].

Acute infection

The first symptom is usually a high fever, with temperatures reaching 41.5 °C, which lasts several days before other symptoms appear. Anorexia, ruminal atony, elevated respiratory rate, and aversion to movement are all present. The conjunctiva and mucous membranes are initially congested and reddened, but the colour changes to the pallor of anaemia as erythrolysis occurs. Severe jaundice, dark red to brown urine, and a very stable froth characterise the final stages. It is possible to have either constipation or diarrhoea (Dwivedi *et al*., 1976) [10]. *Borrelia burgdorferi* causes CNS involvement due to parasitized erythrocytes sludging into brain capillaries, a condition known as "cerebral babesiosis". Infection with the *B. bovis* causes incoordination, mania, convulsions, posterior paralysis, and coma. When anxious signs occur, they almost always lead to death. Death can occur days after the onset of a fever, and seriously infected animals can die after just 24 hours of illness. The mortality rate can range from 50 percent to 100 percent, but most animals can survive if they are not overly stressed. Hemoglobinuria is rarely present and the fever is moderate. *B. divergens* infections have parasitemia and clinical appearances similar to *B. bigemina* infections; *B. divergens* induces anal sphincter spasm, allowing "pipe stem" faeces to move. And when there is no diarrhoea, the faeces are expelled with great force in a long thin stream. The febrile period typically lasts about a week in those animals that live, and there is usually significant weight loss, a decrease in milk production, the risk of abortion, and a long recovery. For several weeks, survivor bulls may have had decreased fertility. A sub-acute syndrome and minor infections can also occur; however, the symptoms are less apparent and can be difficult to identify. The fever is mild, and the haemoglobin level is normal (Knowles *et al*.,1982, Zintl *et al*., 2003, Rejitha *et al*., 2003 and Hemaswathy *et al*., 2021) [11-14].

Post-mortem lesions

In general, prolonged infections cause anaemia and jaundice in the carcass, while acute infections cause severe congestion in most organs, with haemorrhages under the membranes of many internal organs. The spleen is swollen, often several times its usual size, friable, black, with a cut surface that looks like raspberry jam and a soft pulp. The kidneys and liver are both deteriorating. The gallbladder is distended with thick, dark green bile, and the liver is swollen and icteric. The gallbladder may have haemorrhage on the mucous surface. Congested, swollen, and dark-coloured kidneys are present. Urinary bladder distention is common, and urine is dark, reddish-brown in colour. Edema and congestion of the lungs are common. Icteric abomasal and intestinal mucosa with subserosal haemorrhages are possible. Petechial haemorrhages can occur both subepicardial and subendocardial. Congestion or petechial haemorrhages may occur in other tissues and organs, including the brain. The grey matter in the brain has a distinctive cherry-pink hue in

cerebral babesiosis (Bhikane *et al*., 2001 and Radostits *et al*., 2000) [6, 9].

Diagnosis

Fever, anaemia, jaundice, hemoglobinuria, and an enlarged dark pulpy spleen in cattle in enzootic areas where Boophilus ticks are present are all symptoms of babesiosis. The presence of *Babesia* on blood smears, positive serologic tests, transmission studies, or both can be used to validate this. Thick and thin blood smears, ideally from capillaries in the ear or tail tip, should be prepared from the live animal (Homer *et al.*, 2000) [5]. Hematologic research includes jugular blood in EDTA, and serological tests necessitate both acute and convalescent sera. The transmission test uses blood with citrate. Smears of the heart muscle, kidney, liver, lung, and brain, as well as a blood vessel in an extremity, should be obtained during necropsy. Ticks should be removed from diseased livestock or stables. The most common method for verifying tick fever is a microscopic examination of blood and organ smears from sick or dead animals. The best place to gather capillary blood is at the end of the tail. Organ smears, which can be made from animals that have been dead for up to 24 hours, are also a valuable diagnostic tool. *B. bigemina* is commonly seen on Giemsa-stained thin blood smears in cases of acute infection. Detonation is more likely with thick smears. While thick smears improve the chances of detecting the causative organism, they make identifying the characteristic morphology more difficult. The low parasitaemia in the circulating blood of *B. bovis* infections makes brain biopsies a valuable tool for detecting and diagnosing the infection. Since the organism disappears or is present in exceedingly low numbers soon after the acute infection, diagnosis of chronic infection is usually made using a variety of serologic tests for the detection of particular antibodies (Bose *et al.*, 1995) [15]. Serological serum testing is used to validate a diagnosis and to provide further information. The Indirect Fluorescent Antibody Test (IFAT) is a laboratory test that detects antibodies to *B. bovis* and *B. bigemina*. Antibodies to *B. bovis* and *B. bigemina* can be identified using an enzyme-linked immunosorbent assay (ELISA). Enzyme-linked immunosorbent assay (ELISA) for *Babesia bovis*: The ELISA is a valuable diagnostic and epidemiological instrument for testing the presence or absence of antibodies in a large number of samples. The microtitre plates are coated with crude *B. bovis* antigen in an indirect ELISA. The *Babesia bigemina* enzyme-linked immunosorbent assay (ELISA) is a competitive ELISA that coats microtitre plates with a recombinant antigen. While DNA probes capable of detecting extremely low parasitemias, such as those found in carrier animals, are being produced, they are not yet widely used (Woodford *et al.*, 1990) [16]. *Babesia* infection of ticks is detected using hemolymph and egg smears. A transmission test can be used to confirm infection in chronic cases that are difficult to diagnose due to the lack of hemoglobinuria and low parasitaemias, as well as in suspected carrier animals. Blood (500 mL) is inoculated into a completely susceptible animal, preferably a splenectomized cow, and the recipient is monitored for infection.

Hematology

In addition to increased bleeding and accelerated red blood cell sedimentation, clinical haematology revealed a substantial reduction in erythrocyte count, packaged cell volume, and haemoglobin concentration. In clinical conditions, haemoglobin levels are reduced to 3g/dL (Pandey *et al.*, 1978) [17].

Differential diagnosis

Anaplasmosis, trypanosomiasis, theileriosis, leptospirosis, bacillary hemoglobinuria, post-parturient hemoglobinuria, enzootic bovine pyelonephritis, and chronic copper poisoning are several other disorders to remember. The clinical symptoms of cerebral babesiosis must be distinguished from other central nervous system disorders such as rabies and plant toxicosis (Pandey *et al.*, 1978) [17].

Immunity

Animals who recover from natural infection have co-infectious immunity (premunity) for years with *B. bovis* and a few months with *B. bigemina*. During this carrier condition, no symptoms are evident, but stress factors like parturition, malnutrition, or concurrent diseases may break down the protective barrier, causing clinical signs to reappear. Infections that are repeated result in lifelong immunity. There is no immunity if the infection is treated quickly and effectively, and the protozoa are killed before antibodies are formed. There appears to be little connection between humoral and cellular immunity to babesiosis. Babesiosis immunity is both humoral and cellular, and the degree of immunity tends to be unrelated to the amount of serum antibodies. Many *Babesia* spp. infected animals develop immunity to the same species of *Babesia*. In *B. bigemina* resistant animals, there is evidence of some cross-protection against subsequent *B. bovis* infections. Regardless of the *Babesia* species involved or the dams' immune status, calves rarely exhibit clinical symptoms of illness after being infected (Radostits *et al*., 2000 and De Vos, 1979) [9, 18].

Treatment

Treatment offered early in the course of the infection has a high chance of success. If treatment is delayed, supportive therapy and blood transfusions can be needed to save the animal. Chemotherapy resistance is usually higher in small *Babesia*s. Treatment options include: Even if parasitemia is removed, treatment should be attempted early (before the animal becomes anaemic); caution should be exercised to prevent full parasitemia elimination before adequate antibodies are formed to provide long-term immunity (Shastri *et al.*, 1981) [19].

At a dose of 1 mg/kg BW, imidocarb dipropionate salt (Imizol 12 percent - 1 ml /100 kg by s/c injection) is effective. Imidocarb has been successfully used as a chemoprophylactic at a high dose rate of 3 mg/kg BW (Imizol 12 percent - 2.5 ml/100 kg by s/c injection) to avoid clinical infection for up to 2 months while allowing mild subclinical infection to occur as the drug level wanes, resulting in premunition and immunity. Since the use of imidocarb will cause an animal's immunity to tick fever to be disrupted, animals infected with Imizol should not be vaccinated for at least 8 weeks after care. Thick smears improve the odds of detecting the causative organism, but they make recognising the characteristic morphology more difficult. Because of the low parasitaemia in the circulating blood in *B. bovis* infections, brain biopsies are very useful in increasing the chances of detecting and diagnosing the infection (McHardy *et al.*, 1979) [20]. Since the organism disappears or is present in exceedingly low numbers soon after the acute infection, diagnosis of chronic infection is normally made using a variety of serologic tests for the detection of particular antibodies. Serological serum testing is

used to validate and provide further information regarding a diagnosis. The Indirect Fluorescent Antibody Test (IFAT) is a laboratory test that detects antibodies to *B. bovis* and *B. bigemina*. Antibodies to *B. bovis* and *B. bigemina* can be identified using an enzyme-linked immunosorbent assay (ELISA). *Babesia bovis* enzyme-linked immunosorbent assay (ELISA) is a useful diagnostic and epidemiological instrument for testing the presence or absence of antibodies in a large number of samples. The microtitre plates are coated with crude *B. bovis* antigen in an indirect ELISA. The *Babesia bigemina* enzyme-linked immunosorbent assay (ELISA) is a competitive ELISA that coats microtitre plates with a recombinant antigen. Anaplasmosis, trypanosomiasis, theileriosis, leptospirosis, bacillary hemoglobinuria, post-parturient hemoglobinuria, enzootic bovine pyelonephritis, and chronic copper poisoning are several other disorders to remember. The clinical symptoms of cerebral babesiosis must be distinguished from other central nervous system disorders such as rabies and plant toxicosis (Gray *et al*., 1985 and Homer *et al*., 2000) [1, 5].

Prevention and control

Tick prevention with acaricides, immunisation of vulnerable stock, chemoprophylaxis, treatment of infected livestock, stock movement control, and raising tick-resistant cattle are some of the latest control measures used. There is generally no need for control in endemic areas where all indigenous cattle are infected as calves. Calves must be exposed to the infection when they are most resistant to maintain the enzootically stable state. If the problem persists, the calf crop will need to be vaccinated year after year. In order to enter an enzootic environment, susceptible cattle must be vaccinated first. Vaccination before outbreaks begin, as well as chemoprophylaxis after outbreaks have begun, is the recommended technique in marginal areas near enzootic areas. Controlling and removing babesiosis's vector, the Boophilus tick, is the most effective way to fight the disease. Tick regulation, rather than eradication, is the aim in most enzootic areas to achieve an equilibrium in which tick numbers are adequate to maintain low-level infection in cattle and thus tolerance to acute babesiosis, allowing prevalence to be limited to economically viable levels. Dipping can be done once a week in one-host ticks that stay on the host for three weeks, but twice a week in two- and three-host ticks. Dipping can be done once a week in one host ticks that stay on the host for three weeks, but twice a week in two and three host ticks. Chlorinated hydrocarbons, carbamates, organophosphates, natural and synthetic pyrethrins, and avermectins are some of the most popular acaricides used to kill ticks. Dips or sprays are the most popular ways to apply acaricides to livestock, with dips being more effective. Other methods of acaricide application, such as "pour-ons" and "spot-ons" have been introduced in recent years. The growth of resistance in tick populations necessitated the introduction of new compounds. If the vector distributio is set to zero, the vector distribution is zero. Because of the emergence of resistance in tick populations, the introduction of new compounds has become important. When a country's vector distribution is highly concentrated, infected areas must be identified, and stock movement both in and out must be managed (Bhikane *et al*., 2001, Radostits *et al*., 2000 and Shastri *et al.*, 1981) [6, 9, 19].

Chemo - immunization

Where babesiosis is widespread, chemo-immunization is the only way to achieve premunity, as treatment that sterilises the infection inevitably leaves the animal vulnerable to reinfection. Chemo-immunization may also be used to introduce susceptible cattle into an endemic environment, but it must be used in conjunction with an effective tick control programme to avoid significant losses in the face of a major tick vector challenge. Premunition is a mild, painless infection in which the parasite is dormant; its presence in the body stimulates defence against virulent infection with the same *Babesia* species. After recovering from a premunizing parasitemia, cattle would have a brief period of sterile immunity. If a carrier infection is followed by reexposure, as is typical in endemic areas, the animal will develop immunity that will last for the rest of its life. Even variants will not induce a clinically detectable reaction when animals are immunised. Inoculating live organisms (attenuated or virulent) into susceptible young cattle, followed by chemotherapy, as required, to change the clinical effects, induces coinfectious immunity or a state of premunition. If older animals are to be vaccinated, extra caution may be needed to prevent complications (Radostits *et al*., 2000) [9].

Vaccination

In several countries, live vaccines for *B. bovis* and *B. bigemina* have been available as monovalent, bivalent, and sometimes trivalent with Anaplasma. Live species are used in the vaccines, which are rendered avirulent by repeated rapid syringe passage through splenectomized calves. Animals can be vaccinated at any age, although it is safer to vaccinate them between the ages of 3 and 9 months, since it offers protection after 8 weeks, which is normally lifetime. Imidocarb has been successfully used as a chemoprophylactic, preventing clinical infection for up to two months while allowing mild subclinical infection to occur as the drug level declines, resulting in premunition and immunity. To reduce losses, handle ill cattle as soon as possible and keep track of those that have been treated. If sick animals are treated too late, they cannot recover. Remove ticks from all animals with an acaricide to help avoid a secondary outbreak. Surveillance of cattle on neighbouring farms, collection of blood smears from any suspicious cases, and treatment if possible. Vaccination is the only way to avoid potential outbreaks. If the condition has been evaluated, vaccinate all 'at risk' animals in the infected region with tick fever vaccine, except those treated with Imidocarb or displaying signs of tick fever (Radostits *et al*., 2000 and Woodford *et al.*, 1990) [9, 16].

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