

## Bovine Babesiosis: A Review

Adem Edao

College of Veterinary Medicine, Haramaya University, Ethiopia

**Abstract:** Bovine babesiosis is a febrile, tick-borne disease of cattle, caused by one or more protozoan parasites of the genus *Babesia* and generally characterized by extensive erythrocytic lysis leading to anemia, icterus, hemoglobinuria and death. In cattle, it's a major disease transmitted by ticks. The distribution of causative protozoa is governed by geographical distribution of insect vector that transmit the disease. Immunocompromised and stressed cattle are most susceptible to infection. Cattle are the principal hosts, but water buffalo, African buffalo and other ungulates may also become infected. Pathogenesis is related to rapid and massive intravascular haemolysis. The first clinical sign is usually a high fever with rectal temperatures reaching 41.5°C (106.7°F). Often the first visible appearance of infection is that the animal isolates itself from the herd, seeks shade and may lie down. This is usually associated with severe hemoglobinemia and hemoglobinuria. The most common diagnostic methods are; Serological test and microscopic examination of the agent using different techniques. The most commonly used compounds for the treatment of bovine babesiosis are diminazene aceturate, phenemidine disethionate, imidocarb dipropionate etc. As public health importance is concerned, cattle parasites seem to cause disease in people who are immunocompromised, in humans who have had splenectomies and can affect healthy people. The economic impact of babesiosis can be expressed in terms of mortality and morbidity, cost of disease control and, restrictions placed on the movement of animals. The control of the disease depends on the distribution of vector tick and effective quarantine. There are now clear grounds for considering new, integrated approaches which encompass: selection of tick-resistant cattle, exploitation of enzootic stability, use of effective vaccines when enzootic stability is not evident and use of acaricides only when economically justified in relation to the direct effects of ticks on livestock production. Based on the above points: implementing of proper prevention and control strategies; and epidemiological surveillance research should be performed in the country in order to establish the current status of the disease

**Key words:** Bovine • *Babesia bigemina* • *Babesia bovis* • Ticks

### INTRODUCTION

Bovine babesiosis is a febrile, tick-borne disease of cattle, caused by one or more protozoan parasites of the genus *Babesia* and generally characterized by extensive erythrocytic lysis leading to anemia, icterus, hemoglobinuria and death. In cattle, it's a major disease transmitted by ticks. This disease is caused by a protozoan parasite, *Babesia* species. Physical signs of infected cattle include fever, anorexia, depression, increased respiratory rate, muscle tremor, reluctance to move and behavioral changes such as circling, head pressing, mania and convulsions. Bovine babesiosis is a complex disease sharing the feature of being

predominantly transmitted by ticks. Worldwide babesiosis must count amongst the most important of all tick-borne diseases (TBDs). It impairs the development of livestock industries [1].

Three species, *Babesia bovis*, *B. bigemina* and *B. divergens*, are recognized as being of economic significance in cattle. Tick species are the vectors of *Babesia*[2] *Rhipicephalus* (formerly *Boophilus*) *microplus* is the principal vector of *B. bigemina* and *B. bovis* which is widespread in the tropics and subtropics. The vector of *B. divergens* is *Ixodes ricinus*. *B. bigemina* has the widest distribution but *B. bovis* is generally more pathogenic than *B. bigemina* and *B. divergens*. *Babesia bovis* infections are characterized

by high fever, ataxia, anorexia, general circulatory shock and sometimes also nervous signs as a result of sequestration of infected erythrocytes in cerebral capillaries. Anemia and hemoglobinuria may appear later in the course of the disease. In acute cases, the maximum parasitaemia (percentage of infected erythrocytes) in circulating blood is less than 1%. This is in contrast to *B. bigemina* infections, where the parasitaemia often exceeds 10% and may be as high as 30%. In *B. bigemina* infections, the major signs include fever, hemoglobinuria and anemia. Intravascular sequestration of infected erythrocytes does not occur with *B. bigemina* infections. The parasitaemia and clinical appearance of *B. divergens* infections are somewhat similar to *B. bigemina* infections [3]. In general it is essential to have adequate knowledge of the epidemiology and awareness of the disease before contemplating control programs. Therefore, the objective of this review was to revise bovine babesiosis and its related factors.

**Bovine Babesiosis:** Babesiosis is caused by intraerythrocytic protozoan parasites of the genus *Babesia* that infect a wide range of domestic and wild animals and occasionally man. Tick fever was the first disease for which transmission by an arthropod to a mammal was implicated at the turn of the twentieth century and is the first disease to be eradicated from a continent North America [4].

**Etiology:** The genus *Babesia* belongs to the phylum Apicomplexa, class Sporozoa, order Eucoccidiorida, suborder Piroplasmorina and family Babesiidae [5]. A group composed mainly by *Babesia* species from ungulates are *B. caballi*, *B. bigemina*, *B. ovis*, *B. bovis* and *Babesia* sp. from cattle; a second group of *Babesia* species including *B. canis* and *B. gibsoni* from canids. Bovine babesiosis is an infectious tick-borne disease of livestock that characterised by fever, anemia, hemoglobinuria and weakness. The disease also known by such names as bovine babesiosis, piroplasmosis, Texas fever, red water, tick fever and tristezza [6]. It is a hemoparasitic disease, which infects mainly ruminants [7]. Infection of a vertebrate host is initiated by inoculation of sporozoite form of parasites into the bloodstream during the taking of a blood meal [8].

**Epidemiology of Babesiosis:** *B. bigemina* and *B. bovis* are the most important disease of tropical and subtropical regions between 40°N and 32°S. Both species are transmitted transovarially by *Rhipicephalus* (formerly

*Boophilus*) ticks, but only tick larvae transmit *B. bovis*, whereas nymphs and adults transmit *B. bigemina* [9]. *B. major* occurs in Europe, North Africa and South America. It is transmitted by the three host tick *Haemaphysalis punctata*. *B. bovis* and *B. bigemina* are particularly important in Asia, Africa, Central and South America, parts of southern Europe and Australia [10].

The major *Babesia* spp known to infect domestic animals *B. bovis* and *B. bigemina* are present in many countries between 40°N and 32°S [1]. The main vectors of *Babesia* are *Rhipicephalus* (formerly *Boophilus*) ticks. *Rhipicephalus* (formerly *Boophilus*) *microplus* the most important and widespread vector, but in southern Africa, a closely related tick, *Rhipicephalus* (formerly *Boophilus*) *decoloratus*, interferes with its spread in drier and colder areas. Interbreeding between the two species produces sterile progeny which creates a zone through which *R.(B.) microplus* has difficulty passing [11]. Generally both parasites have the same distribution, but in Africa *B. bigemina* is more widespread than *B. bovis* because of the ability of *B. decoloratus* and *Rhipicephalus evertsi* to act as vectors [12]. *Rhipicephalus* (formerly *Boophilus*) *annulatus* is the principal vector of *B. bovis* and *B. bigemina* in Northern Africa [13]. Some areas of southern Europe *Babesia divergens* are transmitted almost exclusively by *Ixodes ricinus* in northern Europe and this probably explains its limited distribution [12, 14].

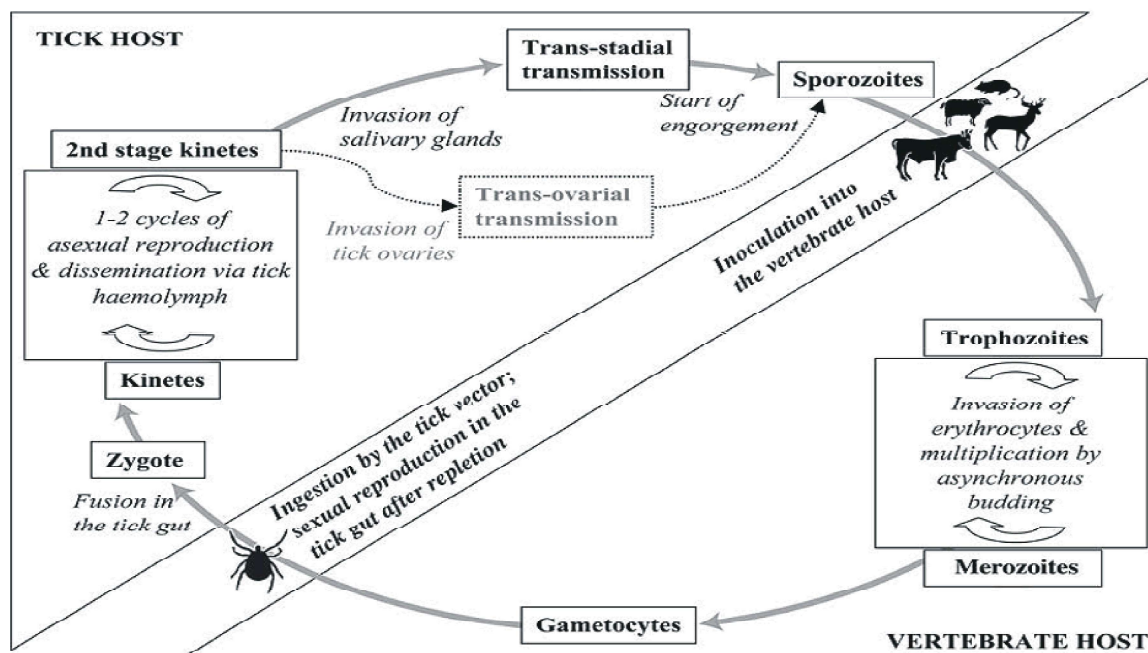
**Transmission:** Hard ticks of the family Ixodidae are the natural vectors of babesiosis. *Babesia* spp are generally transmitted transovarially in one-, two or three-host ticks, but stage-to-stage transmission "transstadial" may also take place. One-host ticks of the genus *Rhipicephalus* (formerly *Boophilus*) that has a worldwide distribution primarily transmit *B. bovis* and *B. bigemina*, the two species that are of most concern. Larvae, nymphs and adults feed on the same individual host until they mated, replete females drop to the ground to oviposit. *Rhipicephalus* (formerly *Boophilus*) *microplus* and *Rhipicephalus* (formerly *Boophilus*) *annulatus* transmit both *B. bovis* and *B. bigemina* whereas *Rhipicephalus* (formerly *Boophilus*) *decoloratus* (blue tick- Sub-Saharan Africa) can only transmit *B. bigemina*. The major vector of *B. divergens* is *Ixodes ricinus* [15].

Other important vectors include *Haemaphysalis* and *Rhipicephalus* species. Transmission through these one-host ticks is transovarially. The engorging adult female ticks pick up sporozoite and pass it on to their progeny (larval or seed ticks) through eggs. Following the attachment to another host, the infection is transmitted by

Table 1: Major *Babesia* species infective to domestic animals, their ixodid tick vectors and geographical distribution

<i>Babesia</i> species	Major ixodid vectors	Known distribution	Domestic species Affected
<i>Babesia bigemina</i>	<i>Rhipicephalus</i> (formerly <i>Boophilus</i> ) <i>microplus</i> , <i>Rhipicephalus</i> (formerly <i>Boophilus</i> ) <i>decoloratus</i> , <i>Rhipicephalus</i> (formerly <i>Boophilus</i> ) <i>annulatus</i> ,	Africa, Asia, Australia, Central and South America	Cattle, buffalo
<i>Babesia bovis</i>	<i>Rhipicephalus</i> (formerly <i>Boophilus</i> ) <i>microplus</i> , <i>Rhipicephalus</i> (formerly <i>Boophilus</i> ) <i>annulatus</i> , <i>Rhipicephalus</i> (formerly <i>Boophilus</i> ) <i>geigy</i>	As for <i>Babesia bigemina</i> , but less widespread in Africa	Cattle, buffalo
<i>Babesia divergens</i>	<i>Ixodes ricinus</i> , <i>Ixodes persulcatus</i>	North-west Europe, Spain, Great Britain, Ireland	Cattle
<i>Babesia major</i>	<i>Haemaphysalis punctuate</i>	Europe, North west Africa, Asia	Cattle
<i>Babesia ovate</i>	<i>Haemaphysalis longicornis</i>	Eastern Asia	Cattle
<i>Babesia ovis</i>	<i>Rhipicephalus bursa</i>	South-eastern Europe, North Africa, Middle East Asia	Sheep and Goat
<i>Babesia motasi</i>	<i>Rhipicephalus bursa</i> , <i>Haemaphysalis punctuate</i>	South-eastern Europe, North Africa and Asia	Sheep and Goat
<i>Babesia caballi</i>	<i>Dermacentor</i> spp., <i>Hyalomma marginatus</i> , <i>Hyalomma truncatum</i> ,	Africa, South and central America and southern USA Europe, Asia	Horses, Donkey, Mule

Source: [10]

Fig. 1: Generic life cycle diagram of *Babesia* spp.

Source: [16].

the larval, nymph and adult stages in case of *B. bovis* or by nymph and adult stages in case of *B. bigemina*. The percentage of larvae infected can vary depending mainly on the level of parasitaemia of the host at the time the female ticks engorge. Many ticks are needed to infect a single animal, as an engorging female tick can produce more than 3000 seed ticks, but only a very small number of seed ticks, sometimes less than 1 in 1000 will carry the infection. Contaminated needles, surgical instruments and blood transfusion can transmit the infection [15]

**Lifecycle of *Babesia* spp.:** The life cycles of the *Babesia* parasites are very similar. All species of *Babesia* are naturally transmitted by the bite of infected ticks (almost all ixodids rather than argasids) and the main life cycle difference amounts to the presence of transovarial transmission in some species. During the tick bite, sporozoites are injected into the host and directly infect red blood cells [10]. *Babesia* spp multiply in erythrocytes by asynchronous binary fission, resulting in considerable pleomorphism. This replication eventually gives rise to

gametocytes that are ingested by the vector tick. Conjugation of gametocytes occurs in the tick gut followed by multiplication by multiple fission and migration to various tissues including the salivary glands. Further development occurs in the salivary glands before transmission [16]. The general *Babesia* parasites life cycle are described below as depicted in fig1.

#### Risk Factors

**Host Factor:** Cattle are the principal hosts, but water buffalo, African buffalo and other ungulates may also become infected. Such hosts are probably not significant reservoirs of infection. Susceptibility to infection with *Babesia* spp decreases with age, but the severity of clinical disease increases. Calves from susceptible dams are highly susceptible to infection with minimal clinical signs from birth to 2 months of age at which time they develop an innate resistance that persists to about 6 months of age. Calves from immune mothers receive temporary protective maternal antibodies from the colostrum, which prevent babesiosis. This protection lasts about 3-4 months and in most cases, is followed by an age resistance, which lasts until the animals are about six months old. After 6 months of age, the number of infected animals in enzootic areas increases. The greatest infection rate is in animals in the 6-12 month age group. Infection is uncommon in animals over 5 years. Animals under one year of age are infected predominantly with *B. bigemina* and those over two years by *B. bovis*. Immuno-compromised and stressed cattle (pregnancy, poor conditioned) are most susceptible to infection [15].

**Environmental Factor:** A heaviest loss occurs in marginal area where the ticks' population is highly variable depending on the environmental condition. In seasons when the tick population decreases infection may die out and immune be lost. Then in favorable season when ticks multiply the disease spread quickly amongst what has become susceptible population. There is also a seasonal variation in the prevalence of clinical babesiosis, the greatest incidence occurring soon after the peak of the tick population. The disease may have seasonal incidence if tick population varies with climate. For example, in England babesiosis is largely disease of spring, summer and autumn. The climatic factor which could have an important effect on seasonal prevalence are temperature, humidity and rain fall, of this temperature is the most important because of its effect on tick activity. High temperature increases tick survival [17].

**Morbidity and Mortality:** Babesiosis causes about 90% deaths in cattle when they were not provided health facilities while it was reported that babesiosis causes 30% deaths in cows and 70-80% in sheep [18]. In several countries of the world including Asia, Australia, Africa, South and Central America and United States the occurrence of Babesiosis in cattle is about 1.2 billion [19]. Babesiosis has a great monetary impact due to mortality, loss of meat, beef and milk productions of infected animals as well as this disease also have great influence on international dairy trade [20]. In naive cattle, susceptibility to disease varies with the breed. *Bos indicus* are more resistant than *Bos taurus*. Approximately 28% of a population of adult animals was susceptible to infection but resistant to clinical signs. In fully susceptible breeds, up to half or more of untreated adults and up to 10% of treated adults may die [21].

#### Pathogenesis of Most Common *Babesia* Species

***B. bovis* Infection:** Pathogenesis is almost entirely related to rapid and massive intravascular hemolysis [22]. *B. bovis* is the most pathogenic of the bovine babesia. *B. bigemina* infections are not as virulent as those of *B. bovis*; however the parasites may infect 40% of the red cells [10]. Cytokines and other pharmacologically active agents have an important function in the immune response to *Babesia*. Ticks are most often infected transovarially. The female tick becomes infected by the ingestion of parasites during engorgement. After it drops off the host, the *Babesia* agents reproduce within the tick's tissues. Some of their producing organisms are incorporated within developing tick embryos and the disease agents are transmitted to new hosts by the feeding of ensuing tick larvae, nymphs, or adults [7]. The outcome is related to the timing and quantity produced, but their overproduction contributes to disease progress causing vasodilation, hypotension, increased capillary permeability, edema, vascular collapse, coagulation disorders, endothelial damage and circulatory stasis [23].

Although stasis is induced in the microcirculation by aggregation of infected erythrocytes in capillary beds, probably the most deleterious pathophysiological lesions occur in the brain and lung. This can result in cerebral babesiosis and a respiratory distress syndrome associated with infiltration of neutrophils, vascular permeability and edema [24]. The acute disease generally characterized by; fever (>40°C), inappetence, depression, increased respiratory rate, weakness, a reluctance to move and hemoglobinuria. The fever during infections may cause pregnant cattle to abort and bulls to show reduced fertility lasting six to eight weeks [22].

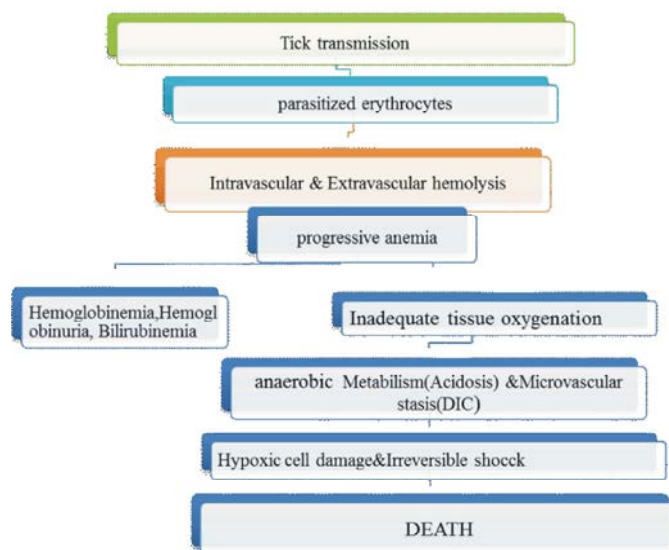


Diagram 1: Pathogenesis showing diagram of *Babesia species*

*B. bigemina* infection: *B. bigemina* is moderately pathogenic for adult cattle, but it is the most widely distributed of the *Babesia* spp., as it transmitted by *R. (B.) microplus* and *R. (B.) annulatus*, as well as *R. (B.) decoloratus* and probably *R. (B.) geigy*. *Babesia bovis* is highly pathogenic for European breeds of cattle. This parasite also occurs on all continents, but its distribution in Africa is restricted, as *B. bovis* cannot be transmitted by *R. (B.) decoloratus* [25]. Coagulation disorders, cyto-adherence and the hypotensive state seen in acute *B. bovis* infections are not features of *B. bigemina* infections [26]. With most strains of *B. bigemina*, the pathogenic effects relate more directly to erythrocyte destruction. Hemoglobinuria is present earlier and more consistently than in *B. bovis* infections and less fever. Acutely affected cattle are usually not as severely affected as those with *B. bovis* infections. There is no cerebral involvement and recovery in non-fatal cases is usually rapid and complete. Animals that recover from *B. bigemina* remain infective for ticks' for 4 to 7 weeks and carriers for only a few months. Erythrocytic parasitism by *Babesia* leads to anemia, hyperbilirubinuria, hemoglobinuria possibly followed by kidney failure, adult respiratory distress syndrome (ARDS) and central nervous system impairment [27].

**Clinical Signs:** Calves normally are reasonably resistant to *babesia*. In older animals, clinical signs can be very severe. However, differences in pathogenicity may occur with different geographic areas. The first sign is usually a high fever with rectal temperatures reaching 41.5°C (106.7°F). There is anorexia and ruminal atony. Often the

first visible appearance of infection is; isolation from the herd, becomes uncomfortable, seeks shade and may lie down. Cattle may stand with an arched back, have a roughened hair coat and show evidence of dyspnea and tachycardia. The mucous membranes are first injected and reddened, anemia, severe weight loss, drop in milk production, possible abortion and a protracted recovery [9].

As clinical finding hemoglobinuria, the color of urine is dark-red to brown, Respiratory and heart rates are increased and the red conjunctivae and mucous membranes change to the extreme pallor of severe anemia. Abortion occurs in pregnant animals and subacute syndrome also occurs in young animals, but fever is mild and hemoglobinuria is absent. In cerebral babesiosis, hyper excitability, convulsions, opisthotonos, coma and death, maybe observed in cattle infected with either *B. bigemina* or *B. bovis*, but especially with the *B. bovis*. Central nervous system signs are caused by brain anoxia resulting from severe anemia [7, 9].

**Diagnostic Methods:** Diagnoses of babesiosis are made by examination of blood and/or organ smears stained with Giemsa [28]. The most commonly used tests are ELISA, PCR and a DNA probe, which can detect specific parasitaemia at very low levels of infection [9]. For the best results, blood films should be prepared from capillary blood collected. The temptation to use blood of the general circulation should be resisted as these specimens may contain up to 20 times fewer *B. bovis* than capillary blood [22]. In *B. bigemina* infections, parasitised cells are evenly distributed throughout the blood circulation.

Thick blood films are 10 times more sensitive and are therefore very useful for the detection of low level *B. bovis* infections. These films differ from thin ones in that the blood is not spread over a large area and is not fixed before staining, thus allowing lysis of the red blood cells and concentration of the parasites [28].

**Direct Microscopic Examination:** This technique is usually adequate for detection of acute infections, but not for detection of carriers where the parasitaemias are mostly very low. Parasite identification and differentiation can be improved by using a fluorescent dye, such as acridine orange, instead of Giemsa [28]. Samples from dead animals should consist of thin blood films, as well as smears from cerebral cortex, kidney (freshly dead), spleen (when decomposition is evident), heart muscle, lung and liver [3].

**In vitro Culture:** *In-vitro* culture methods have been used to demonstrate the presence of carrier infections of *Babesia* spp and *B. bovis* has also been cloned in culture [29].

**Animal Inoculation:** Confirmation of infection in a suspected carrier animal can also be made by transfusing approximately 500 ml of jugular blood intravenously into a splenectomised calf known to be *Babesia*-free and monitoring the calf for the presence of infection. This method is cumbersome and expensive and obviously not suitable for routine diagnostic use. [4].

**Serological Tests:** Serodiagnosis is an invaluable tool in epizootiological studies of bovine babesiosis. Cattle which have been infected may have antibodies which are detectable for many years after infection, the parasite has been eliminated. The presence of antibody shows that animal has been infected and it is immunized [24].

**Indirect Fluorescent Antibody (IFA) Test:** Was widely used in the past to detect antibodies of *Babesia* spp, but the *B. bigemina* test has poor specificity.

**Complement Fixation (CF) Test:** Has been described as a method to detect antibodies against *B. bovis* and *B. bigemina*. This test has been used to qualify animals for importation into some countries [30].

**Treatment:** The most commonly used compounds for the treatment of babesiosis are diminazene aceturate (3-5 mg/ kg), phenmedin diisethionate (8-13 mg/ kg), imidocarb dipropionate (1-3 mg/kg) and

amicarbalid diisethionate (5-10 mg /kg) [4, 7]. At the high dose, imidocarb also eliminates *Babesia*. Treatment with long-acting oxytetracycline following vaccination significantly reduces parasitaemia and red blood cell destruction without inhibiting the development of immunity [31]. Supportive therapy such as blood transfusions (4 L of whole blood per 250 kg of body weight), fluids, hematinic and prophylactic antibiotics are important [7].

**Control and Prevention Measures:** Control measures that are currently applied to control ticks by use of acaricides, immunization of susceptible stock, chemoprophylaxis, treatment of infected animals, control of stock movement and raising cattle resistant to ticks. In endemic areas, where all indigenous cattle are infected as calves, no control is usually necessary [15]. The control of ticks by dipping or spraying animals at risk with recommended acaricides. In addition, cattle selection and breeding which acquire a high degree of resistance to ticks is preferable [7]. The long prophylactic effect of imidocarb against *B. bovis* and *B. bigemina* has been used to protect newly introduced cattle in the hope that the animals will become exposed to natural infections and develop immunity while still partially protected by the drug [32].

#### Tick Control

**Dipping Cattle:** Synthetic chemical pesticides specific for ticks (acaricides) are suspended in water for application to the hair coat of domestic animals. Cattle can be immersed in dip-baths containing 15, 000 liters of dip wash, or soaked using a pressurized spray-race made of metal tubing and nozzles. Sheep can be treated in smaller dips or showers [33]. Modern acaricides belong to the general classes of organophosphates (example chlorfenvinphos), formamidines (example amitraz) and benzyl phenylurea's (example fluazuron) [10]. When correctly applied they can be highly effective. Problems with acaricides are: danger of acute poisoning of treated animals and human staff; residues contaminating meat and milk; environmental contamination especially water sources; resistance that ticks acquire to acaricides; and cost of application. Cost and contamination can be reduced by seasonal timing of application (strategic treatment) based on ecological knowledge. Prediction of best times for treatment can be made using computerized models of the population dynamics of ticks [34].

**Vaccination:** Vaccination will prevent future outbreaks. There are many live vaccines for *B. bovis* and *B. bigemina* are available in many countries, as monovalent, bivalent

and sometimes trivalent vaccines. The vaccines consist of live organisms made avirulent by repeated rapid syringe-passage through splenectomized calves. Single 2 ml dose injected either subcutaneous or intramuscular and animals can be vaccinated at any age, but it is best to vaccinate animals at 3-9 months of age, it gives immunity after 8 weeks, which usually life long. Cattle that have been treated with prophylactic drugs, such as imidocarb dipropionate, are not responsive to vaccination for at least 4-8 weeks after the treatment. Keep the vaccinated animals tick-free for at least 4 weeks after vaccination, as solid immunity takes time to be produced. Vaccinate all 'at risk' animals in the affected area with tick fever vaccine once the situation has been assessed, except those treated with Imidocarb or showing symptoms of tick fever starting with groups that are most at risk. Start a long-term risk management strategy that includes annual vaccination of calves 3-9 months of age and vaccination of introduced cattle [15].

**Public Healthy Importance of Babesiosis:** To date, seven distinct *Babesia* parasites have been found to cause human babesiosis. *B. microti* and related organisms, *B. divergens*, *B. bovis*, *B. canis*, *B. duncani*, *B. venatorum* and a novel type of *Babesia* spp. [16]. Most patients infected with *Babesia* spp sensu stricto share splenectomy as a risk factor for acquiring the disease. In addition, for all *Babesia* infections advanced age and depressed cellular immunity are associated with a higher risk of symptomatic infection and more severe illness [27]. This is why the rising number of human immune virus positive individuals and the increasing population of immunocompromised individuals may serve to boost the number of human babesiosis cases [35]. Humans acquire the disease through tick bites or transfusion of contaminated blood products [36]. An exceptional way of infection which is rarely observed in humans is transplacental transmission [37].

In immune competent individual's parasitaemia can hardly be detected. Patients may show non-specific symptoms like fever, flu-like disease, headache, chills, sweats and myalgia. Clinical symptoms in immunocompromised patients include high fever (up to 40°C), high parasitaemia (20–80%) diaphoresis and severe anemia, shortness of breath, weakness and fatigue. *Babesia microti* infections may persist despite multiple courses of treatment and may be associated with relapsing symptoms for more than a year in immunocompromised individuals as described in a recent case control study [38].

**Economic Impact of Babesiosis:** Most of the cattle in the world are exposed to babesiosis [1]. Breeds of cattle that are indigenous to *Babesia* have a certain degree of natural resistance to these diseases and the consequences of infection are not as serious as when exotic *Bos taurus* breeds are involved. In addition, in tropical areas with a high vector population, natural exposure usually occurs at an early age and cattle are therefore immune to subsequent challenge as adults. Costs due to babesiosis are incurred not only from mortality and morbidity and from control measures (such as acaricides, treatments, purchase of vaccines and therapeutics), but also through its impact on international cattle trade [39].

## CONCLUSION AND RECOMMENDATIONS

Livestock and livestock products play an important role in the socio-economic development of the world. Bovine babesiosis is a febrile, tick-borne disease of cattle, caused by one or more protozoan parasites of the genus *Babesia* and generally characterized by extensive erythrocytic lysis. The etiology, epidemiology and prevalence of the disease is governed by geographical distribution of insect vector that transmit disease. Diagnosis and treatment of bovine babesiosis are related to history of tick distribution, clinical sign, necropsy finding and differential diagnosis that give us hint for control and prevention measure. The main problem of tick fever involves economic and zoonotic importance that leads to loss in production, foreign trade, treatment cost and human force since it is zoonotic. The other problems includes: drug resistance, lack of disease management and control, insufficient veterinary service, low government attitude for veterinary professionals and poor social understanding on healthy and value of their livestock. Based on the above points we can generalize that bovine babesiosis is a zoonotic and economically significant disease which is harmful to cattle industry. So, the following points have been recommended.

- To implement proper prevention and control strategies; an epidemiological surveillance research should be performed in the country in order to establish the current status of the disease.
- Traditional method of controlling ticks continuously and widely in Africa, mainly by using acaricides by dipping or spraying.
- There should be strong biosecurity measures in cattle farm

- To implement proper use of drug and vaccine to reduce economic losses and avoiding drug resistance.
- Strengthening social and government altitude for veterinarians to change their understanding on healthy and importance of livestock in the country.

## REFERENCES

1. Mccosker, P.J., 1981. The global importance of babesiosis. In Babesiosis (ed. Ristic, M. & Kreier, J. P.), pp: 1-24. New York, *Academic Press*.
2. Levine, N.D., 1988. Predator-prey coccidia: The sarcocystidae. The Protozoan Phylum Apicomplexa, Vol. II. Boca Raton, Florida: CRC Press, pp: 1-10.
3. Bock, R., L. Jackson, A.J. De Vos and W. Jorgensen, 2008. Babesiosis of cattle. In: Ticks: Biology, Disease and Control. Cambridge University Press, Cambridge, UK, pp: 281-307.
4. Zulfiqar, S., S. Shahnawaz, M. Ali, A.M. Bhutta, S. Iqbal, S. Hayat, S. Qadir, M. Latif, N. Kiran, A. Saeed and F. Iqbal, 2012. Detection of *Babesia bovis* in blood samples and its effect on the hematological and serum biochemical profile in large ruminants from Southern Punjab. *Asian Pacific Journal of Tropical Biomedicine*, pp: 104-108.
5. Angus, B., 1996. The history of the cattle tick *Boophilus microplus* in Australia and achievements in its control. *International Journal of Parasitology*, 26: 1341-1355.
6. Allsopp, M.T., T. Cavalier-Smith, D.T. De Waal and B. A. Allsopp, 1994. Phylogeny and evolution of the piroplasmids. *Parasitology*, 108: 147-152.
7. Zaugg, J.L., 2009. Babesiosis. In: Smith, B.P. (Eds) *Large Animal Internal Medicine*. Mosby, Elsevier, St. Louis, pp: 1157.
8. Melendez, R.D., 2000. Babesiosis: An emerging zoonosis in temperate and tropical zones. A review. *Revista Científica- Facultad De Ciencias Veterinarias*, 10(1): 13-18.
9. Radostits, O.M., C.C. Gay, K.W. Hinchcliff and P.D. Constable, 2008. Diseases associated with protozoa. 10<sup>th</sup> edn. In: *Veterinary Medicine*.
10. Taylor, M.A., R.L. Coop and R.L. Wall, 2007. *Veterinary Parasitology*. Third. Edn. Blackwell Publishing.
11. Sutherst, R.W., 1987. The dynamics of hybrid zones between tick (Acari) species. *International Journal for Parasitology*, 17: 921-926.
12. Friedhoff, K.T., 1988. Transmission of Babesia. In *Babesiosis of Domestic Animals and Man* (ed. Ristic, M.), pp: 23-52.
13. Bouattour, A., M.A. Darghouth and A. Daoud, 1999. Distribution and ecology of ticks (Acari: Ixodidae) infesting livestock in Tunisia: an overview of eight years field collections. *Parasitology*, 41: 5-10.
14. Caeiro, V., 1999. General review of tick species present in Portugal. *Parassitologia*, 41: 11-15.
15. El Sawalhy, A.A., 1999. "Veterinary Infectious Diseases" 2<sup>nd</sup> edn. Ahram Distribution Agency, Egypt.
16. Gray, J.S. and L.M. Weiss, 2008. Babesiamicroti. In: Khan, N. (Ed.), *Emerging Protozoan Pathogens*. Taylor and Francis, Abingdon, UK, pp: 303-349.
17. Barandical, I.F., E.B. Berriatus, R.A. Juste and A.L. Garciaperez, 2006. Risk factor with Ixodid tick species Distribution, 37: 11-12.
18. Durrani, A.Z. and N. Kamal, 2008. Identification of ticks and detection of blood protozoa in Friesian cattle by polymerase chain reaction test and estimation of blood parameters in district Kasur, Pakistan. *Tropical Animal Health Production*; 40: 441-447.
19. Ziapour, S.P., B. Esfandiari and M.R. Youssefi, 2011. Study of the prevalence of babesiosis in domesticated animals with suspected signs in Mazandaran province, north of Iran, during 2008. *Journal of Animal Veterinary Advance*, 10: 712-714.
20. Mosqueda, J., A. Olvera-Ramírez, G. Aguilar-Tipacamú and G.J. Cantó, 2012. Current Advances in Detection and Treatment of Babesiosis. *Current Medicinal Chemistry*, 19: 1504-1518.
21. Barros, Y. and R. Figuera, 2008. Babesiosis In: *Foreign animal diseases*. 7<sup>th</sup> edition. Boca Raton, FL: United States Animal Health Association, pp: 147-158.
22. Callow, L.L., R.J. Rogers and A.J., 1993. Tick-borne diseases: cattle-pathology and serology. In *Australian Standard Diagnostic Techniques for Animal Diseases* (ed. Corner, L. A. & Bagust, T. J.), 1-16. East Melbourne, CSIRO Information Services.
23. Ahmed, J.S., 2002. The role of cytokines in immunity antimicrobial pathogenesis of piroplasmoses. *Parasitology Research*, 88: 48-50.
24. Brown, W.C. and G.H. Palmer, 1999. Designing blood-stage vaccines against *Babesia bovis* and *B. bigemina*. *Parasitology Today*, 15: 275-281.



25. Jacquiet, P., N.M. Perie, F. Jongejan, G. Uilenberg and P.C. Morel, 1990. Présence de *Theileria annulata* en Mauritanie. Rev. Elev. Méd. Vét. Pays. Trop., 43: 489-490.
26. Dalgliesh, R.J., J.B. Molloy, W.K. Jorgensen and R.E. Bock, 1995. Technical Cooperation Programme. AG: DP/ETH/83/023. Technical Report. Food and Agriculture Organization of the United Nations (FAO), Rome, Italy, pp: 4-24.
27. Telford S.R. and J.H. Maguire, 2006. Babesiosis. In: Guerrant, R.L., Walker, D.H., Weller, P.F. (Eds.), Tropical Infectious Diseases: Principles, Pathogens and Practice. Churchill Livingstone, New York, USA, pp: 1063-1071.
28. Bose, R., W.K. Jorgensen, R.J. Dalgliesh, K.T. Friendhoff and A.J. De Vos, 1995. Current state and future trends in the diagnosis of babesiosis. Vet. Parasitology, 57: 61-74.
29. Holman, P.J., K.A. Waldrup, R.E. Droleskey, D.E. Corrier and G.G. Wagner, 1993. In vitro growth of *Babesia bovis* in white-tailed deer (*Odocoileus virginianus*) erythrocytes. J. Parasitology, 79: 233-237.
30. Anonymous, M., 2006. Complement fixation test for detection of antibodies to *Babesia bigemina* and *Babesia bovis* Microtitration test. United States Department of Agriculture (USDA), Animal and Veterinary Services, National Veterinary Services Laboratories, Ames, Iowa, USA.
31. Jorgensen, W.K., R. E. Bock, T.G. Kingston, A.J. De Vos and S.J. Waldron, 1993. Assessment of tetracycline and *Babesia* culture supernatant as prophylactics for moderating reactions in cattle to live *Babesia* and *Anaplasma* vaccines. Australian Veterinary Journal, 70: 35-36.
32. De Vos, A.J., R.J. Dalgliesh and W. McGregor, 1994. Effect of imidocarb dipropionate prophylaxis on the infectivity and immunogenicity of a *Babesia* vaccine in cattle. Australian Veterinary Journal, 63: 174-178.
33. Willadsen, V., 2006. "Tick control: thoughts on a research agenda". Veterinary Parasitology, 138: 161-168.
34. Randolph, S.E. and D.J. Rodgers, 1997. "A generic population model for the African tick *Rhipicephalus appendiculatus*". Parasitology, 115(3): 265-279.
35. Haselbarth, K., A.M. Tenter, V. Brade, G. Krieger and K.P. Hunfeld, 2007. First of human babesiosis in Germany-clinical presentation and molecular characterisation of the pathogen. Int. J. Medicine Microbiology, 297: 197-204.
36. Homer, M.J., I. Aguilar-Delfin, S.R. Telford, P.J. Krause and D.H. Persing, 2000. Babesiosis. Clin. Microbiol. Rev., 13: 451-469.
37. Kjemtrup, A.M. and P.A. Conrad, 2000. Human babesiosis: an emerging tick-borne disease. Int. J. Parasitol., 30: 1323-1337.
38. Krause, P.J., B.E. Gewurz, J.A. Hill, G.P. Wormser, K. Dickason, M. Coleman, J.E. Giroto and A. Spielman, 2008. Persistent and relapsing babesiosis in immunocompromised patients. Clin. Infect. Dis., 46: 370-376.
39. McLeod, R. and P. Kristjanson, 1999. Final report of jointesys/ILRI/ACIAR Tick Cost project - Economic impact of ticks and tick-borne diseases to livestock in Africa, Asia and Australia. International Livestock Research Institute, Nairobi.