Tick Borne Hemoparasitic Diseases of Ruminants: A Review

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Abstract: Tick borne diseases are caused by different microorganisms and transmitted by ticks. They are the most prevalent and devastating diseases in the developing countries all over the world. The objective of this paper is to review tick borne hemoparasitic diseases of ruminants. Tick borne hemoparasitic diseases of ruminants such as Anaplasmosis, Babesiosis and Theileriosis remain most important in tropics areas. Anaplasmosis, a rickettsial disease of blood caused by Anaplasma marginale and characterized by progressive anemia, jaundice and fever without hemoglobinuria. Babesiosis is the other tick-borne disease caused by protozoan of the genus Babesia and characterized by haemolytic anemia and fever, with hemoglobinuria. It is a disease with a world-wide distribution affecting many species of mammals with a major impact on cattle. Theilerioses is also a tick borne protozoal disease in ruminants caused by hemoprotozoan parasites belonging to the genus Theileria. Theileria parva, is the most pathogenic species in Africa cause a disease called East coast fever which is characterized by enlargement of superficial lymph nodes and a sustainable fever. Losses from tick borne hemoparasitic diseases of ruminants like reduction of milk and meat production, restricting the introduction of susceptible cattle breed with superior genetics, costs from death and abortion as well as costs for treatment and control purposes are taking away the benefits of livestock owner and nation. Effective diagnosis of tick borne hemoparasitic diseases of ruminants is helpful to implement appropriate prevention and control strategies. Tick control, chemoprophylaxis and immunoprophylaxis are the basic methods to control tick borne hemoparasitic diseases of ruminants.

Key words: Anaplamoisis • Babesiosis • Ruminant • Theileriosis • Tick Borne

INTRODUCTION

Hemoparasitic diseases have a global distribution, stretching from the polar circle to the equator. This is due to the fact that their vectors, ticks and blood sucking flies have a global distribution. Tick borne hemoparasites includes all tick-borne organisms which are visible with light microscope and which occur in the circulating blood as part of their normal life-cycle [1]. The most important hemoparasites are Babesia, Theileria and Anaplasma. These hemoparasites are transmitted through ticks [2].

Hemoparasites are of great economic impact on livestock affecting 80% of the world cattle population and causes economic loss due to morbidity and mortality. Hemoparasite is a major threat to food security especially among the livestock dependent communities within the sub saharan Africa [3]. Haemoparasites have generally been shown to cause destruction of red blood cells resulting in anemia, jaundice, anorexia, weight loss and infertility. The occurrence and importance of hemoparasite is a reflection of complex interaction involving the causative organisms, vector, the vertebrate hosts and the environment [4].

Arthropod transmitted hemoparasitic diseases are economically important vector-borne diseases of tropical and subtropical parts of the world including Ethiopia. Tick borne hemoparasitic diseases of ruminants are caused by the Babesia, Theileria and Anaplasma species and all are intracellular parasite species [5].

Anaplasmosis is a vector borne infectious blood disease in cattle caused by the rickettsial parasites, Anaplasma (A) marginale and A. central in cattle and A. ovis in small ruminants. It occurs primarily in warm tropical and subtropical areas. The disease is not contagious but transmitted most commonly by ticks. It can also be transmitted via contaminated surgical instruments, biting flies and mosquitoes. The intracellular
In cattle, this disease is caused by *A. marginale*. In six populated continents of the world, mostly in tropical and subtropical countries [7, 8]. Geographical distribution: Anaplasma is found endemic in tick borne hemoparasitic diseases and to increase the occurrence of the disease is associated with the abundance of vector ticks [10].

**Control of tick borne hemoparasitic diseases of ruminants using effective methods such as vector control, chemoprophylaxis and immunization is crucial to control tick borne hemoparasitic diseases and to increase the productivity of animals by improving their health conditions in the tropics and other parts of the world where tick borne diseases are prevalent [9]. Therefore, the objective of this paper is:**

- To review tick borne hemoparasitic diseases of ruminants

### Tick Borne Hemoparasitic Diseases of Ruminant Anaplasmosis

**Etiology:** Anaplasmosis is caused by the members of genus *Anaplasma* (Rickettsiales: *Anaplasmataceae*). In cattle, this disease is caused by *A. marginale* and *A. centrale*; later less pathogenic than former [10] where as in sheep and goats *A. ovis* is the important causative agent [11].

**Epidemiology:** Transmission: Anaplasmoiss is not contagious; numerous species of tick vectors (Boophilus, Dermacenter, Rhipicephalus, Ixodes and Hyalomma) can transmit *Anaplasma* species [12]. Not all of those are likely significant vectors in field and it has been shown that strains of *A. marginale* also co-evolve with particular tick strain. After feeding on an infected animal, transstadial transmission may occur. Transovarial transmission may occur although even in a single host Boophilus species [6]. Anaplasmosis may also be spread mechanically by infected hypodermic needle, castrating, spaying and dehorning instruments, blood transfusion and embryo transplant. Additionally intrauterine infections also occur in cattle but much less frequently in field cases than in experimental once. Anaplasma can transmit by biting flies of the family Tabanidae [9].

Geographic distribution: Anaplasma is found endemic in all six populated continents of the world; mostly in the tropics and subtropics because of the broad range of vectors and the difficulties of efficient vector control [10]. The disease is an important economical factor even with a developed veterinary service [13].

Risk factor: Breeds; *Bos taurus* breeds are more likely to develop acute Anaplasmosis than crossbred Zebu, but *Bos indicus* are not commonly affected because of their resistance to heavy tick resistance [14]. Anaplasma species can cause infections in bovine population of all age categories where severity and mortality rate increases with augmentation of animal age. Anaplasmosis infection is higher in female than male animals due to hormonal disturbances, due to its use in milk production, draught power and breeding system which pose it to weakened immune system [5]. In temperate regions seasonal occurrence of the disease is associated with the occurrence of the vectors in which prevalence of Anaplasmosis is found higher in hot and humid weather associated with the abundance of vector ticks [10].

### Life Cycle: The developmental cycle of *A. marginale* in ticks is complex and coordinated with the tick feeding cycle (Figure 1). Infected erythrocytes taken into ticks with the blood meal provide the source of *A. marginale* infection for tick gut cells. After development of *A. marginale* in tick gut cells, many other tick tissues become infected, including the salivary glands, from where the rickettsiae are transmitted to vertebrates during feeding. At each site of infection in ticks, *A. marginale* develops within membrane bound vacuoles or colonies. The first form of *A. marginale* seen within the colony is the reticulated (Vegetative) form, which divides by binary fission, forming large colonies that may contain hundreds of organisms. The reticulated form then changes into the dense form, which is the infective form and can survive outside the host cells. Cattle become infected with *A. marginale* when the dense form is transmitted during tick feeding.
Fig. 1: Life cycle of *A. marginale*  
Source: Kocan et al. [14]

feeding via the salivary glands [14]. Once Anaplasma entered in to the blood; the organisms enters the red blood cells by invaginating the cell membrane so that a vacuole is formed; thereafter it divides to form an inclusion body containing up to eight initial bodies packed together [15].

**Pathogenesis:** The incubation period of infection varies with the number of organisms in the infective dose and ranges from seven to 60 days, with an average of 28 day [14]. *A. marginale* invades and multiplies in red blood cells. As the disease progresses, infected and even uninfected red blood cells are destroyed mainly in the liver and spleen, resulting in progressive hemolytic anemia and even death in severe cases, the number of infected erythrocytes increases drastically and phagocytosis by reticuloendothelial cells of parasitized erythrocytes lead to development of hemolytic anemia and icterus [11].

Depending upon the strain and the susceptibility of the host, from 10-90% of erythrocytes may be parasitized in the acute stage of the infection [9]. Cattle that survive acute infection develop persistent infections characterized by cyclic low-level rickettsemia. Persistently infected cattle have lifelong immunity and are resistant to clinical disease on challenge exposure. However, persistently infected cattle serve as reservoirs of *A. marginale* because they provide a source of infective blood for both mechanical and biological transmission by ticks [14].

**Clinical Sign:** Anaplasmosis is characterized by fever, weight loss, decreased milk production, pale mucous membranes, severe anemia, jaundice, hyper-excitability, abortion and mortality without hemoglobinemia and hemoglobinuria during acute phase of the infection [16]. The most marked clinical signs of Anaplasmosis are anemia and jaundice, the latter occurring late in the disease [17]. Affected animals may die suddenly from hypoxia if they are handled roughly [18].

**Diagnosis:** Clinical diagnosis of Anaplasmosis is performed by observing anemia, icterus and constipation. The presence of tick vectors or numerous biting Diptera may give an additional indication [19]. In endemic areas, Anaplasmosis in adult cattle showing chronic anemia without haemoglobinuria leading to cachexia [20].

Microscopic examination of Giemsa stained blood films Anaplasma inclusion in the red cells are usually sufficient for diagnosis. Under microscope *A. marginale* are seen as small, round, dark red ‘inclusion bodies’ located near the periphery of red blood cells as indicated in Figure. 2 [15].

Serological methods such as Enzyme-Linked Immunosorbent Assay (ELISA), complement fixations test (CFT), or card agglutination test have been used to detect antibodies in animals which have recovered from infection [21]. CFT has been used extensively in the past, but, it lacks sensitivity and doesn’t detect some carrier. Card agglutination test is sensitive, simple, rapid and can be used in the field. ELISA using a positive and negative antigen can eliminate non-specific reaction [19]. For the determination of the persistently infected cattle molecular diagnosis is mostly applicable [22].

At necropsy, pale and icteric carcass and organs, splenomegally, constipation, congestion of the liver and hardening of the omasum are indicative of Anaplasmosis [19].
Anaplasma like structure is to detect in one of the strains [14]. The introduction of the disease into herds by carrier animals should be prevented by prior serological testing. Attention should also be given to preventing iatrogenic transmission with instruments used for injections or surgical operations by disinfection after use on each animal. The eradication of Anaplasmosis is not a practicable procedure in most countries at the present time because of the wide range of insects which are capable of carrying the disease, the long period of infectivity of carrier animals and, in some areas, the presence of carriers in the wild animal population [9]. In endemic areas, control measures are aimed at minimizing stress in indigenous reared cattle [18].

**Treatment:** Tetracycline compounds are effective in treatment if given early in the course of the disease and especially before the parasitaemia has reached its peak. Tetracycline is effective when injected at five to ten mg/kg, provided the treatment is repeated two times at 24 hour interval. The treated animal is not free from Anaplasma after treatment, but can be eliminated completely by a longer treatment [19]. More recently imidocarb has been shown to be effective and may also used to sterilize carrier animals [15].

Symptomatic treatment such as blood transfusion, drugs that stimulates erythropiosis, drugs which protects liver cells may help recovery [19].

**Prevention and Control:** Methods for the control of Anaplasmosis have consists of tick control with acaricides, chemotherapy for prevention and vaccination. Weekly dipping in an acaricide is used in tropical areas to control this disease. Animals that are to be introduced into an enzootic area should be vaccinated. Vaccination has been an economical and effective way to control bovine Anaplasmosis worldwide. Vaccines for the control of Anaplasmosis can be divided into two major types: live and killed vaccines. Both types of vaccines rely on the use of *A. marginale* from infected bovine erythrocytes. Both types induce protective immunity that reduces or prevents clinical disease, but these vaccines do not prevent cattle from becoming persistently infected with *A. marginale* [9].

Chemotherapy, probably used more often for prevention of Anaplasmosis in the United States than in other areas of the world, is expensive and often not applicable to range cattle and the intensive use of antibiotics bears the risk of causing selection of resistant strains [14].

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**Economic Importance:** Bovine Anaplasmosis causes important economic loss in most countries, mainly due to the high morbidity and mortality in susceptible cattle herds. The losses due to Anaplasmosis are measured through several parameters: low weight gain, reduction in milk production, abortion, the cost of Anaplasmosis treatments and mortality [14].

**Babesiosis:** The disease also called piroplasmosis, Cattle tick fever, Red water fever or Taxes fever [8].

**Etiology:** The causative agents of Babesiosis are specific for particular species of animals. In cattle: *B. bovis, B. bigemina, B. divergens and B. major* [9]. *B. ovis* and *B. motasi* are known to be pathogenic agents in sheep and goats [23].

**Epidemiology:** Transmission: Babesia species is transmitted by hard ticks in which Babesia passes transovarially, via the egg, from one tick generation to the next [24, 25].
Geographic occurrence: Babesiosis occurs throughout the world [23]. However, the distribution of the causative protozoa is governed by the geographical and seasonal distribution of the insect vectors [9]. The vector of Babesia, *Boophilus* (B) *microplus* is widespread in tropical and subtropical regions of the world. Both species are transmitted transovarially by *Boophilus* ticks, whereas nymphs and adults transmit *B. bigemina* and *B. divergens*. Babesiosis transmitted by *Ixodes ricinus* is widespread. Small ruminants Babesiosis is caused by *B. ovis* [27].

**Risk Factors:** Host factor: *Bos indicus* breeds of cattle are more resistant to Babesiosis than *Bos Taurus* [28]. This is a result of evolutionary relationship between *Bos indicus* cattle, *Boophilus* species and Babesia [9]. Because of natural selection pressure, indigenous populations, having lived for a long time with local ticks and tick-borne diseases, have developed either an innate resistance or an innate ability to develop a good immune response to the tick or tick-borne hemoparasitic disease in question. Sheep were highly susceptible to *B. ovis* than goats [29]. It is frequently stated that there is an inverse age resistance to Babesia infection in that young animals are less susceptible to Babesiosis than older animals; the possible reason is passive transfer of maternal antibody via colostrum. The severity of the clinical Babesiosis increases with age [15, 30].

**Pathogen Factor:** Many Intra-erythrocyte hemoparasites survive the host immune system through rapid antigenic variation which has been demonstrated for *B. bovis* and *B. bigemina* [9]. *B. bovis* is the most pathogenic organism, resulting in high mortality rates among susceptible cattle [31]. The effects of *B. ovis* are usually less severe than *B. motasi* [24].

**Environmental Factor:** There is a seasonal variation in the prevalence of clinical Babesiosis, the greatest incidence occurring soon after the peak of the tick population. Of the climatic factors, air temperature is the most important because of its effect on tick activity; higher temperatures increase its occurrence. Heaviest losses occur in marginal areas where the tick population is highly variable depending on the environmental conditions [9]. Babesiosis infection in cattle mostly reaches peak in summer [7].

**Life Cycle:** Babesia multiplies in erythrocytes by asynchronous binary fission, resulting in considerable pleomorphism (Figure 3). 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. The ovaries are also invaded, which leads to transovarial transmission [32]. The host gets the infection when the larva sucks blood. After one moulting the larva transforms into nymph which also infect as larva. Nymph transforms into adult after moulting and they transmit infection in similar way [30]. The infective stage of Babesia, sporozoite, enters in to the host when the tick sucks blood [19].

**Pathogenesis:** Babesia produces acute disease by two principle mechanism; hemolysis and circulatory disturbance [33]. During the tick bite, sporozoites are injected into the host and directly infect red blood cells. In the host, Babesia sporozoites develop into piroplasms inside the infected erythrocyte resulting in two or sometimes four daughter cells that leave the host cell to infect other erythrocytes [34]. It invades erythrocyte and cause intravascular and extravascular hemolysis [33]. The rapidly dividing parasites in the red cells produce rapid destruction of the erythrocytes with accompanying haemoglobinaemia, haemoglobinuria and fever. This may be so acute as to cause death within a few days, during which the packed cell volume falls below 20% which will lead to anemia. The parasitaemia, which is usually detectable once the clinical signs appear, may involve between 0.2% up to 45% of the red cells, depending on the species of Babesia [25].

**Clinical Sign:** Affected animals suffered from marked rise in body temperature, loss of appetite, cessation of rumination, labored breathing, emaciation, progressive hemolytic anemia, various degrees of jaundice (Icterus) from paleness in mild cases to severe yellow discoloration of conjunctival and vaginal mucus membranes in more progressive cases; haemoglobinuria, accelerated heart and respiratory rates, ocular problems and drop in milk production. The fever during infections in some cases cause abortion to pregnant cattle [7]. Coffee colored urine is the characteristics clinical feature of Babesiosis [30]. Babesia parasites cause both acute and persistent subclinical disease in cattle [35].

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Fig. 3: Life-cycle diagram of Babesia species  
Source: Gray [32]

Ovine Babesiosis is a progressive hemolytic anemia disease of sheep [36]. High rise of body temperature, anorexia, dyspnea, haemoglobinurea, emaciation, pale mucous membrane, jaundice, constipation and recumbency were the main clinical signs in both sheep and goats. Chronically infected sheep are usually symptomless, except for parasitemia and unthriftness [24]. Animals that recover from the acute disease remain infected for a number of years with B. bovis and a few months in the case of B. bigemina. No clinical signs appear during this carrier state [6].

Neurological signs are exhibited due to B. bovis infections as erythrocytes get adhered to the capillaries of the brain like nystagmus, circling movement, head pressing, hyperesthesia, convulsion, ataxia, teeth grinding and muscle tremor [30].

Diagnosis: Accurate and correct diagnosis of Babesial infections plays an important role in monitoring, management and control of infection [35]. Clinical symptoms, because of their unspecificity, cannot be used to make a correct diagnosis [37].

Microscopy Detection Methods: Microscopic examination still cheapest and fastest methods used to identify Babesia parasites [38]. Identification of the different stages of the parasite in mammalian or arthropod host tissues can be used for direct diagnosis purpose [39].

Thin and Thick Blood Smears: Blood smear examination has been considered to be the standard technique for routine diagnosis, particularly in acute cases, but not in sub-clinical infections where the parasitemia is usually much lower [35, 39]. Thin blood smears were the first method to detect Babesia parasites in clinical samples, a method still used today very effectively in most diagnostic laboratories. Blood is usually collected, combined with an anticoagulant and smeared on a glass slide, air-dried, fixed with methanol and stained with Giemsa or a similar stain for several minutes. The slide is then washed thoroughly and dried. Intra-erythrocytic parasites are observed under a microscope using a 100X objective and a drop of immersion oil. The observation of paired Intra-erythrocytic merozoites is indicative of infection but there are other stages of the parasite like the trophozoites, which present different forms and sizes depending on the species and these make their detection difficult and time consuming [39]. B. bigemina is larger in size and having paired structure at an acute angel to each other in the erythrocyte while B. bovis is smaller in size and having paired forming an obtuse angel to each other [7]. Merozoites of B. bovis are usually found in the center of erythrocyte where as merozoites of B. bigemina are pear-shaped [20]. B. motasi is pyriform in shape; B. ovis has rounded shape and marginally located inside the erythrocytes [40].
Fig. 4: Giemsa stained Babesia species inside the bovine erythrocytes (Thin arrow) where as thick arrow shows an uninfected bovine erythrocyte. **Source**: Chaudhry et al. [26]

Another technique developed to detect low levels of parasitemia, especially in cases where *B. bovis* is involved, is based on thick smears of infected blood stained with Giemsa (Figure. 4). The advantage of the thick smear is that a large amount of erythrocytes is analyzed in a reduced amount of space; therefore the probability of finding infected cells is ten times higher than in the thin smear [39].

**Brain Smears:** When a bovine has died and it is presumed to be from Babesiosis caused by *B. bovis* due to presence of nervous clinical signs, identification of the parasite can be done by brain smears [26]. In this case, a small sample of grey matter of the cerebral cortex is placed on a slide and the tissue is smeared using another slide. The brain tissue is fixed and stained. The diagnosis is based upon observation of brain capillaries filled with infected erythrocytes. Almost one hundred percent of erythrocytes present in the brain capillaries are infected. Smears of other organs as the kidney or liver can also be carried out with good results [39].

**Immunological Methods:** Serological tests, including indirect fluorescent antibody test (IFAT) and ELISA are capable of detecting antibodies of Babesia in sub-clinical infections. Drawbacks of these tests are the occurrence of false-positive and false-negative results, involving cross-reactive antibodies and/or atypical specific immune responses [27]. Another drawback is that antibodies can be detected even months after recovery of infection though no active infection is prevalent, so these methods cannot help in revealing the exact picture of prevalence of infection at that particular point [35].

IFAT is the most widely used test for the detection of antibodies to *B. bovis* and *B. bigemina* but serological cross reactions make species diagnosis difficult [26]. IFAT is based on the recognition of parasite antigens by serum antibodies in the blood of the tested animal. It is easy to do but requires a good quality antigen, which is difficult to obtain [39].

**Molecular Diagnosis:** Molecular methods aimed to detect nucleic acids have been very useful when immunological methods do not work. Detecting nucleic acids is an indirect way of detecting the parasite so they are still considered indirect methods. However, the sensitivity and specificity of these methods are very high [39].

**Polymerase Chain Reaction:** PCR is more sensitive and specific technique and offers an alternative approach for the detection of Babesiosis [39]. An advantage of this method is that it allows identification of the parasite in the early stage of disease which enables early diagnosis, implementation of therapy and avoidance of complications [37].

**Treatment:** The success of the treatment depends on early diagnosis and the prompt administration of effective drug. The first specific drug used against bovine Babesiosis was Trypan blue, which is a very effective compound against *B. bigemina* infections, however, it did not have any effect on *B. bovis* and it had the disadvantage of producing discoloration of animal’s flesh, so it is rarely used. Diminazene aceturate, which is widely used currently in the tropics as a Babesiacide, was withdrawn from Europe for marketing reasons [41]. Imidocarb is the principal Babesiacide used in animals, the only one that consistently clears the host of parasites and for over 20 years, it has been used in the treatment and prophylaxis of Babesiosis and Anaplasmosi. Imidocarb retained in edible tissues of ruminants for long periods
after treatment [39]. The combination of imidocarb dipropionate and oxytetracycline is the most effective treatment of Babesiosis in sheep and goats [24].

In addition, supportive therapy such as blood transfusions, anti-inflammatory drugs, tick removal, iron preparations, dextrose, vitamin B complex, purgatives and fluid replacements, may be necessary in severe cases of Babesiosis [39]. Vitamin E also acts as supportive therapy as vitamin E ameliorates the oxidative effect of Babesia by increase antioxidant effect [42].

**Prevention and Control:** Epidemiological surveillance is the important aspect to control Babesiosis [43]. Active prevention and control of Babesiosis is achieved by three main methods: immunization, chemoprophylaxis and vector control. Ideally, the three methods should be integrated to make the most cost-effective use of each and also to exploit breed resistance and the development and maintenance of enzootic stability [44].

**Chemotherapy/chemoprophylaxis:** Several groups of compounds have been used in the chemical control of Babesiosis. Of these, only imidocarb dipropionate, diminazene aceturate and tetracycline antibiotics remain freely available in most endemic countries. Chemoprophylaxis is not a viable long-term alternative to effective immunization, but imidocarb and diminazene have been used to protect cattle for several months against Babesiosis [44]. At dosage of three mg/kg, imidocarb provides protection for Babesiosis for around four weeks and will eliminate *B. bovis* and *B. bigemina* from carrier animals [6].

**Immunization:** Using blood from carrier animals has been practiced for many years in tropical areas and more recently in Australia [15] Vaccination has been done with varying degree of success with live and dead whole parasite and isolated parasite antigen. Several findings support the development of vaccines against Babesiosis. First, cattle which recover from a primary Babesia infection or that have been immunized with attenuated parasites are resistant to challenge infection. Second, immunization of cattle with native Babesia antigen extracts or culture-derived supernatants containing secreted Babesia antigens elicit protective immunity against both homologous and heterologous challenge [9].

**Vector Control:** it is done by repeated treatment of cattle with acaricides in areas of high challenge; such treatment may require to be carried out twice weekly in order to kill the tick before the infective sporozoite develop in the salivary gland [25]. Significant factors currently affecting the control of Babesiosis and Anaplasmosis include increased resistance to acaricides by ticks and the numerous drawbacks of the current live vaccines [45].

**Economic Importance:** Recently Babesia becomes the most widespread parasite due to exposure of 400 million Cattle to infection through the world, with consequent heavy economic losses such as mortality, reduction in meat and milk yield and indirectly through control measures of ticks. Babesiosis, especially in cattle has great economic importance, because unlike many other parasitic diseases, it affects adults more severely than young cattle, leading to direct losses through death and the restriction of movement of animals by quarantine laws [8, 46]. The disease is also a barrier to improving productivity of local cattle by cross-breeding due to the high mortality of genetically superior but highly susceptible cattle, especially dairy cattle, imported from Babesia-free areas [47]. The consequence is that the quality of cattle in endemic areas remains low, therefore impeding the development of the cattle industry and the wellbeing of producers and their families [39].

**Theileriosis**

**Etiology:** Theileria species, tick transmitted, intracellular protozoan parasites infecting leukocytes and erythrocytes of wild and domestic large and small ruminants. In cattle: *T. parva*, *T. annulata*, *T. mutans*, *T. velifera*, *T. tarurotragi* and *T. buffeli* [19]. In small ruminants: *T. lestoquardi*, *T. ovis* and *T. separate* [30]. *T. annulata* and *T. parva* are considered to be the most pathogenic species of Theileria affecting cattle [48]. *T. lestoquardi* (formerly *T. hirci*) is the most virulent species in sheep and goats [6].

**Epidemiology:** Transmission: Economically important Theileria species that infect cattle and small ruminants are transmitted by ixodid ticks of the genera Rhipicephalus, Amblyomma, Hyalomma and Haemaphysalis [49]. Theileria sporozoites are transmitted to animals in the saliva of the feeding tick [30].

The most economically important species in Africa is *T. parva* [50]. Three subspecies are recognized in *T. parva*, namely *T. parva* *parva* causing classical East Coast Fever (ECF), *T. parva lawrencei* responsible for Corridor disease transmitted from buffalo to cattle and *T. parva bovis*, the causing agent of Zimbabwe Theileriosis, a more benign form also known as “January disease”[50].
Fig. 5: A generalized life cycle for the Theileria using *T. parva* as an example.
Source: Mans *et al.* [57].

### Table 1: A comparison of three species of Theileria of veterinary importance

<table>
<thead>
<tr>
<th>Species</th>
<th>Host</th>
<th>Vector</th>
<th>Disease</th>
<th>Distribution</th>
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<tbody>
<tr>
<td><em>T. parva</em></td>
<td>Cattle</td>
<td><em>Rhipicephalus</em></td>
<td>East Coast Fever</td>
<td>East and Central Africa</td>
</tr>
<tr>
<td><em>T. annulata</em></td>
<td>Cattle</td>
<td><em>Hyalomma</em></td>
<td>Mediterranean or Tropical Theileriosis</td>
<td>North Africa, South Europe, Middle East, Asia</td>
</tr>
<tr>
<td><em>T. hirci</em></td>
<td>Sheep and goats</td>
<td><em>Hyalomma</em></td>
<td>Malignant ovine (caprine) Theileriosis</td>
<td>North Africa, South Europe, Middle East. Asia</td>
</tr>
</tbody>
</table>

Source: Kaufaman [20]

It is important to emphasize that endemic region of *T. annulata* and *T. parva* do not overlap [51] however, there were reports of coexistence in southern Sudan [52].

**Host Sensitivity:** *T. parva* is highly virulent for European dairy cattle, however, the indigenous cattle breeds and African buffaloes in endemic areas have a natural resistance to this Theileria species [9]. The introduction of *T. parva* infection into a previously unexposed cattle population results in an epidemic situation with mortality up to 95% in all age categories of cattle. When the disease is established, the older animals that have survived primary infection become immune. In these situations, only calves and newly introduced stocks are at risk of primary infection and mortality is estimated high. *T. annulata* highly virulent for European dairy cattle but infection in local breed cattle is mild [19]. *T. hirci* causes clinical illness and mortalities in sheep than in goats [53].

**Environmental Factor:** Theileriosis occurs when there is much tick activity, mainly during summer but a single tick can cause fatal infection [55]. The pattern of seasonal occurrence of *Rhipicephalus (R) appendiculatus* (Vector of *T. parva*) is determined by climate [50]. *R. appendiculatus* is most active following onset of rain, outbreak of ECF may be seasonal or, where rainfall is relatively constant, may occur at any time [15].

**Host Factor:** Increasing age is associated with increased *T. parva* sero prevalence [56]. The frequency of occurrence of ECF is higher in female than in male cattle [55].

**Risk Factor**

**Agent Factor:** The two unique features of the leukoproliferative Theileria parasites in their vertebrate hosts are their ability to exist free in the host cell cytoplasm and their ability to transform the host cell reversibly, leading to uncontrolled proliferation of the parasite and the host cell [54].

**Life Cycle**

Generalized life cycles for the Theileria genus include secretion of infective sporozoites during tick feeding into the feeding site (Figure 5). Sporozoites then
petechia and cough are the most common symptoms [58]. Phases: Weight loss, weakness, anorexia, conjunctival, lateral recumbency and marked anemia in its late associated with fever, pronounced leukopenia, diarrhea, of the vector ticks and by lymph destructive disorders scapular lymph node as a result of the predilection sites phases. The first lymph nodes involved are the pre associated with lymph nodes enlargement in its early characterized by lymphoproliferative disorders.

Clinical Findings: Tropical Theileriosis of cattle characterized by lymphoproliferative disorders associated with lymph nodes enlargement in its early phases. The first lymph nodes involved are the pre scapular lymph node as a result of the predilection sites of the vector ticks and by lymph destructive disorders associated with fever, pronounced leukopenia, diarrhea, lateral recumbency and marked anemia in its late phases [15]. Weight loss, weakness, anorexia, conjunctival petechia and cough are the most common symptoms [58].

Clinical signs of ECF characterized, unlike Tropical Theileriosis, by the absence or limited intensity of anemia and high frequency of respiratory sign. Other symptoms include; diarrhea, corneal opacity leading to blindness and subcutaneous edema [19]. In susceptible cattle, ECF is characterized by enlargement of superficial lymph nodes starting with the parotid lymph node, a sustainable fever and diverse clinical signs associated with invasion of non lymphoid tissues with parasitized lymphoblast. ECF causes high mortality with death occurring approximately three weeks after infection, mainly as a result of severe pulmonary oedema [59].

Malignant Theileriosis of sheep and goats is usually acute and highly fatal disease of adult animals manifested by high fever associated with conjunctival and nasal discharge, jaundice, enlargement of superficial lymph nodes, emaciation, reduced appetite, labored breathing and sometimes hemoglobinurea [60, 61].

Diagnosis: Tentative diagnosis of Theileriosis in the field is mainly based on clinical signs and tick infestation on the infected animals. However, confirmation of the diagnosis depends on microscopic examination of blood and lymph node smears stained with Giemsa in acute cases [58, 62].

Microscopic Examination: The presence of multinucleate intracytoplasmic and free schizonts, in lymph node biopsy smears, is a characteristic diagnostic feature of acute infections with T. parva and T. annulata [59]. Piroplasm of T. hirci appeared as a small oval, round or dotes-like inside erythrocytes and macroryschizont in large lymphocyte [60]. Microscopic examination alone is not a fully reliable method of diagnosis unless coupled with other serological and molecular diagnostic assay [63].

Serological Diagnosis: The main weaknesses of serological tick-borne disease diagnosis have been reported as cross reactivity, low specificity and sensitivity and being poor at detecting low piroplasm levels in carrier animals [64].

Molecular Diagnosis: PCR technique: PCR technique can be used as a gold standard method and also can be used for screening of T. annulata carrier cattle. PCR is highly sensitive and specific method, which used to detect the rate of native carrier cattle harboring T. annulata [65].

Treatment: Chemotherapeutic agents such as parvaquone, buparvaquone which is given intramuscularly and halofuginone, coccidostat given per
Fig. 6: Giemsa stained blood smears of infected cattle reveal the presence of intra-erythrocytic forms (Arrows) morphologically compatible with theilerial piroplasms. Some of the infected erythrocytes showed morphological disorders represented by round-shaped appearance and irregular thorn-like protrusions. Source: Hassan [55]

Fig. 6: Giemsa stained blood smears of infected cattle reveal the presence of intra-erythrocytic forms (Arrows) morphologically compatible with theilerial piroplasms. Some of the infected erythrocytes showed morphological disorders represented by round-shaped appearance and irregular thorn-like protrusions. Source: Hassan [55]

orally are available to treat clinical *T. parva* and *T. annulata* infections. Treatments with these agents do not completely eradicate Theilerial infections leading to the development of carrier states in their hosts [20]. Oxytetracycline has a therapeutic effect if given at the time of infection; they are no value in the treatment of clinical cases [25].

**Prevention and Control:** The main methods in the prevention control of ECF include tick control, cattle movement control, host immunization and chemotherapy and integrated control that combines any of the methods Gachohi et al. [56].

**Tick Control Methods:** Includes direct application of acaricides to cattle through dipping, spray races, hand spray, pour-ons and hand dressing. However, acaricides have their own disadvantages; they are expensive, ticks can easily develop resistance to them and they can be detrimental to the environment [56].

The most cost effective and sustainable control method is immunization. Vaccines against Theileriosis are of two types; the sporozoite vaccine and the schizont vaccine but the latter is preferred [30]. Vaccination against *T. parva* is based on a method of infection and treatment in which cattle are given a subcutaneous dose of tick-derived sporozoites and a simultaneous treatment with a long-acting tetracycline formulation. This treatment results in a mild or in apparent ECF reaction followed by recovery [66]. Efforts have been made to produce anti tick vaccination to immunize animal by using whole somatic antigen of tick and protein of salivary gland [30].

**Chemotherapy:** Oxytetracyclines are effective in controlling Theileriosis if given at the same time as infection as applied to block both parasite and disease development [50].

Quarantine measures, particularly with respect to importation of livestock from endemic areas into regions where suitable tick vectors exist, are of great importance [9].

Another method of Theileriosis control is cattle movement restriction from Theileriosis-specified areas. Movements within endemic areas are allowed. However, movements from endemic areas to non-endemic areas are allowed on the following conditions: the animals to be moved must test negative serologically by IFAT, they are treated with acaricide before they are moved to insure that they are tick-free, they are subjected to compulsory quarantine under close veterinary supervision [50].

**Economic Importance:** Tropical Theileriosis threatens an estimated 250 million cattle and acts as a major constraint on livestock production and improvement in many developing countries [67, 68]. Total economic losses because of Tropical Theileriosis include three main parameters: production losses, control costs and other indirect economic losses [69].

*T. parva* infection poses a significant threat to the livestock sector in two ways: through the economic impact of the disease from cattle morbidity and mortality and production losses in all production systems, as well as from the costs of the measures taken to control ticks and the disease. The costs of acaricide application, which is the primary means of tick control, is estimated to range between US$6 and US$36 per adult animal in Kenya, Tanzania and Uganda. The disease further prevents the introduction of the ECF susceptible but more productive exotic breeds of cattle, hampering the development of the livestock sector considerably [56]. The financial losses incurred, directly and indirectly, by ECF are extremely high. With more than 38% of the African total bovine population affected and an estimated mortality of 1.1 million cattle per year. ECF remains probably the most
important cattle disease in terms of economic losses and restriction of livestock development in affected countries of eastern, central and southern Africa [19]. ECF was estimated to have been responsible for 170 million US dollars worth of economic loss in 1989 alone and limits introduction of more productive exotic (Bos taurus) cattle in much of eastern, central and southern Africa [49].

Epidemiology of Tick Borne Hemoparasite of Ruminants in Ethiopia: Tick borne hemoparasitic diseases transmitted by the major tick species in Ethiopia are Anaplasmosis, Babesiosis and benign Theileriosis. The principal biological vector of A. marginale namely B. decoloratus, Hyalomma marginatumrufipes, Rhipicephalus evertsi evertsi are prevalent in Ethiopia. The vectors of B. bigemina in Ethiopia are B. decoloratus, B. annulatus and Rhipicephalus evertsi evertsi. The vectors of T. mutans; Rhipicephalus evertsi evertsi and Amblyomma variegatum are also commonly found in different regions of Ethiopia [70].

In Cattle: T. velifera, T. mutans, T. orientalis complex and T. annulata were found in northern Ethiopia(Addis Zemen, Humera and Sheraro) in which T. annulata, the cause of Tropical Theileriosis, was the first report in Ethiopia, Humera. In small ruminants: T. ovis and T. separata found also in these areas [71]. Indigenous cattle breeds such as Horro and Borna are more resistance to tick infestation than others [70].

CONCLUSION

Tick borne hemoparasitic diseases are the major bottleneck of livestock development in the tropics especially in developing countries. The major tick borne hemoparasitic diseases with heavy economic losses are mainly of Protozoal and Rickettsial diseases such as Theileriosis, Babesiosis and Anaplasmosis. These tick borne hemoparasitic diseases impair the export and import trade of live animal and animal products (Meat, milk, hide and skin) by down grading their quality and fear of the co-traders. Control of tick borne diseases is crucial in improving livestock health services and animal productivity. There are different control strategies which vary from region to region as well as from area to area. Potential control methods for tick borne pathogens include tick control, vaccines (Against ticks and parasites) and drugs (Against ticks and parasites). Successful application of control strategies will be dependent upon thorough understanding of parasite developmental cycles, biology of the tick vectors and the immune response of cattle to ticks and parasites. Therefore, based on this conclusion the following recommendations are forwarded:

- Economic losses from tick borne hemoparasitic diseases ruminants are very high so that concerned organization should give attention to control and eradicate them.
- Since chemical control can result in resistance and environmental contamination, environmentally friendly control mechanisms like vaccination and biological methods should be further developed.
- Awareness should be created on mode of transmission, control and prevention methods of tick borne hemoparasitic diseases to livestock owners.
- Proper identification and characterization of ticks involved in the transmission of disease should be done to implement particular control strategy.
- New vaccines and drugs should be designed that eliminates carrier states of hemoparasites in ruminants.

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