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Protozoan Diseases in Farm Ruminants

Sima Sahinduran
*University of Mehmet Akif Ersoy,
Faculty of Veterinary Medicine,
Department of Internal Medicine,
Turkey*

1. Introduction

Protozoa are ubiquitous throughout aqueous environments and the soil, and play an important role in their ecology. Protozoa occupy a range of trophic levels. They also play a vital role in controlling bacteria population and biomass. Farm animals are usually infected with several species of parasites and they are also confined to pasture or pens. Parasite eggs, larvae, and cysts are intense in soil. One of the most important aspect of animal protozoology is transmission to humans. These are called zoonosis. Some of zoonotic diseases are common and important to public health. This chapter is about of the etiology, epidemiology, pathogenesis, clinical findings, diagnosis, treatment and control of the pathogenic protozoa in farm ruminants.

2. Babesiosis

2.1 Etiology

Babesiosis is an infectious tick- borne disease of livestock that characterised by fever, anemia, haemoglobinuria and weakness. The disease also is Known by such names as bovine babesiosis, piroplasmosis, Texas fever, redwater, tick fever, and tristeza (Zaugg, 2009). The disease also is a hemoparasitic disease caused by protozoa of the genus *Babesia* (Phylum: Apicomplexa), which infects mainly ruminants (Melendez, 2000). Infection of a vertebrate host is initiated by inoculation of sporozoite form of parasites into the blood stream during the taking of a blood meal (Radostits et al. 2008). The list of babesia species is shown in table 1.

2.2 Epidemiology

2.2.1 Bovine babesiosis

Bovine babesiosis disease is caused by at least six *Babesia* species (Table 1). Bovine babesiosis associated with *B. bigemina* and *B. bovis* is the most important disease of tropical and subtropical regions between 40°N and 32°S. Both species are transmitted transovarially by *Boophilus* ticks, but only tick larvae transmit *B. bovis*, whereas nymphs and adults transmit *B. bigemina*. (Radostits et al. 2008). In Europe, babesiosis is caused by *Babesia divergens*, an intraerythrocytic parasite that can persist for >13 months in the organs of

infected cattle. The distribution of *B. divergens* reflects its triphasic telotropic tick vector, *Ixodes ricinus*, which is widespread across Western Europe and North Africa (Jammes, 2009). *B. major* occur in Europe, North Africa and South America. *B. major* is transmitted by the three host tick *Haemaphysalis punctata* (Taylor et al., 2007).

Organism	Livestock affected	Geographic distribution
<i>B. bigemina</i>	Cattle	Americas, Europe, Africa, Australia, Middle East
<i>B. bovis</i> (<i>B. berbera</i> , <i>B. argentina</i>)	Cattle	Americas, Europe, Russia, Africa, Asia Australia
<i>B. divergens</i>	Cattle	Europe
<i>B. major</i>	Cattle	Europe, Russia, North Africa, Middle East
<i>B. jakimovi</i>	Cattle	Asia
<i>B. ovata</i>	Cattle	Japan
<i>B. motasi</i>	Sheep and goats	Southern Europe, Russia, southeast Asia, Middle East, Africa
<i>B. ovis</i>	Sheep and goats	Southern Europe, Russia, Asia, Middle East

Table 1. Babesia species (Babesiosis)

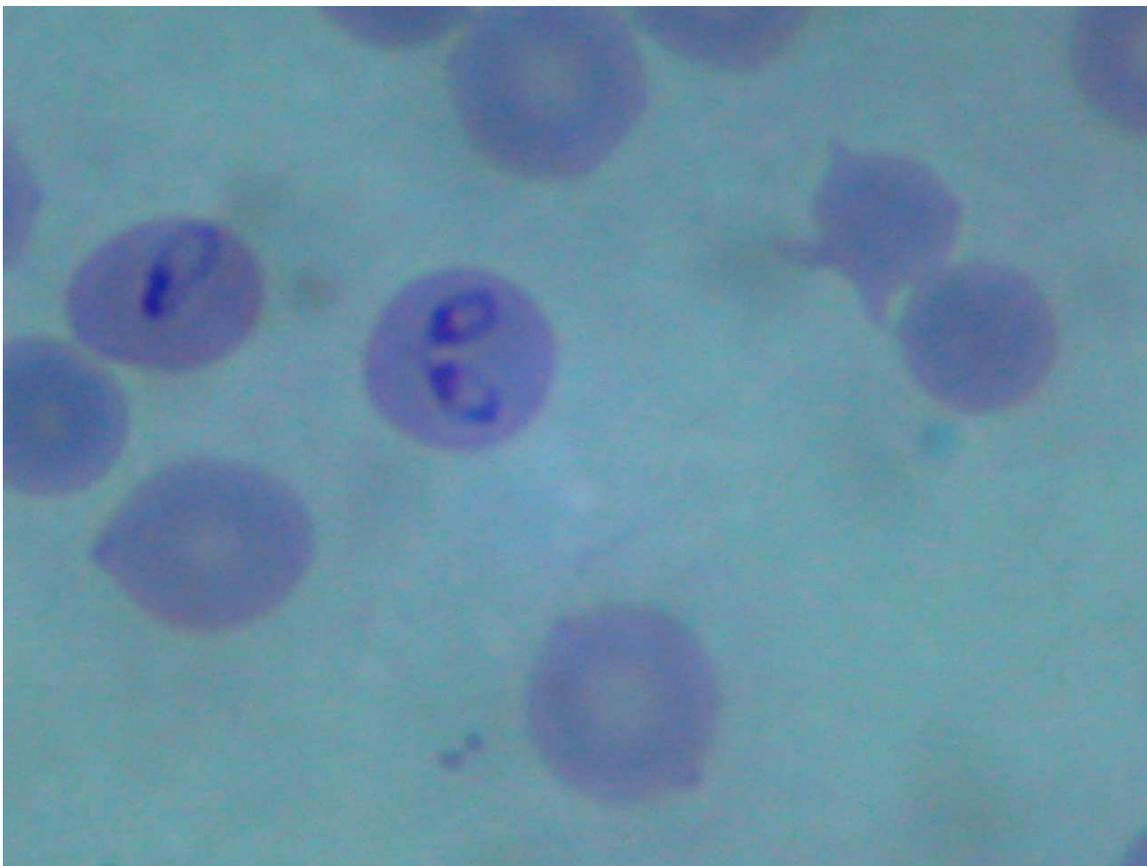


Fig. 1. Babesia bigemina in blood smear from a cow, Giemsa staining, immersion objective

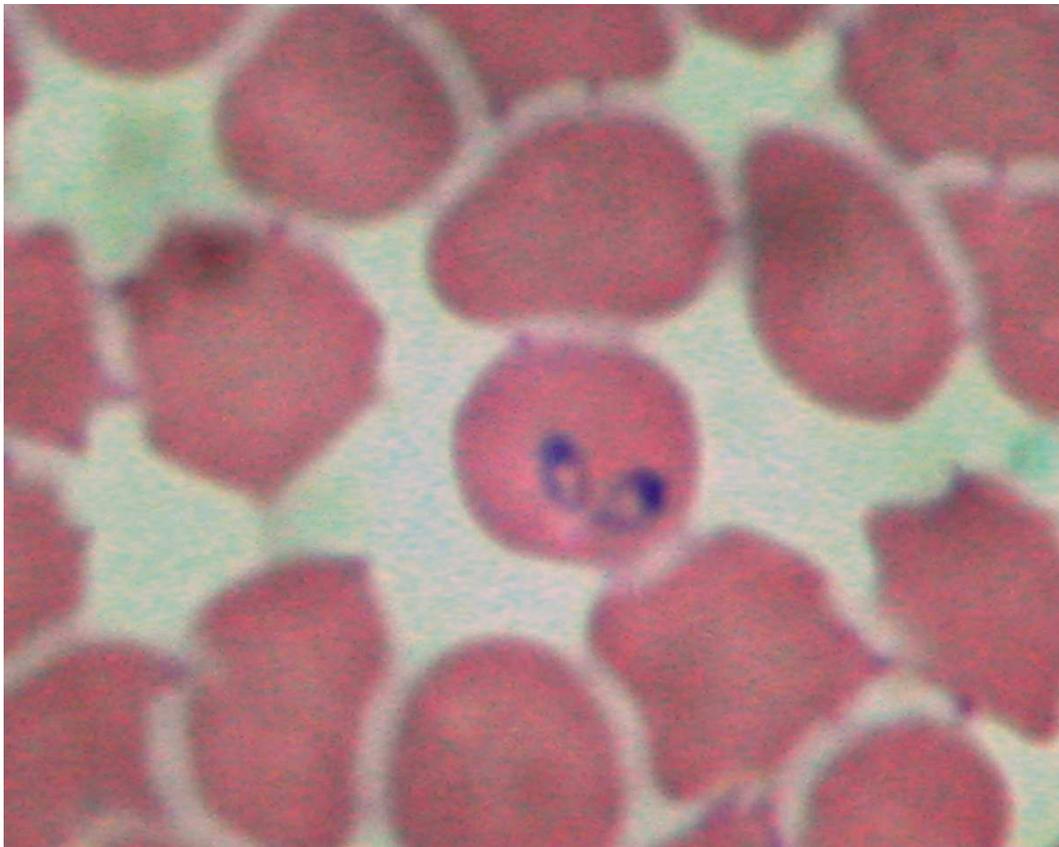


Fig. 2. *B. bovis* in blood smear from a cow, Giemsa staining, immersion objective

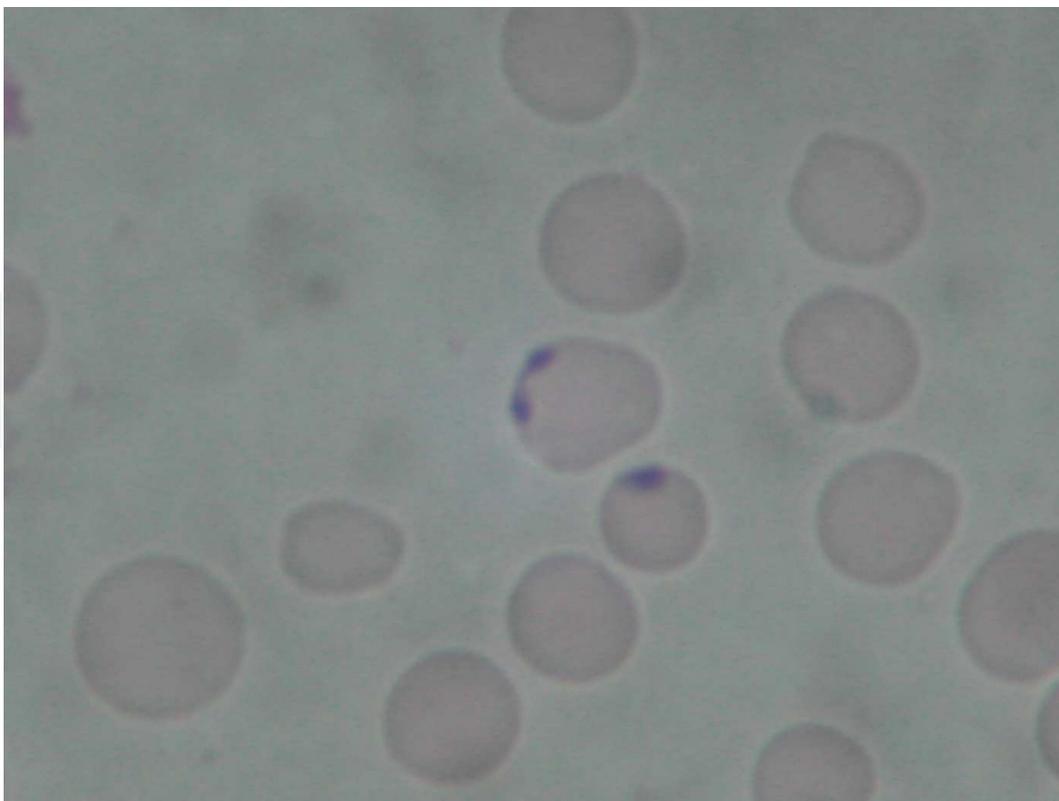


Fig. 3. *B. divergens* in blood smear from a cow, Giemsa staining, immersion objective

2.2.2 Sheep and goats

In sheep and goats, babesiosis is associated with *B. ovis* and *B. motasi*. *Babesia ovis* occur in Southern Europe, former Soviet State, Middle East and Asia. *Rhipicephalus bursa* has been shown to be a vector for *B. ovis*. *Babesia motasi* occur in Southern Europe, the Middle East, the former Soviet State, southeast Asia and Africa. The parasite transmitted by ticks of the genus *Haemaphysalis* (*H. punctata*, *H. otophila*), *Dermacentor* (*D. silvarum*) and *Rhipicephalus* (*R. bursa*) (Taylor et al., 2007).

2.3 Pathogenesis

Babesia spp. are a various group of tickborne, obligate, intra-erythrocytic Apicomplexan parasites infecting a wide variety of animals. 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 babesial agents reproduce within the tick's tissues. Some of the reproducing 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 (Zaugg, 2009).

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 (Taylor et al., 2007).

Babesia affecting small ruminants are generally less pathogenic than their bovine likes (Cebra, C., and Cebra, M., 2002a).

2.4 Clinical findings

2.4.1 Cattle

Incubation period is 2-3 weeks. *B. bigemina* and *B. bovis* produce acute syndromes which are clinically indistinguishable, and are characterized by high fever (41°C), anorexia, depression, weakness, cessation of rumination, and a fall in milk yield. Hemoglobinuria can be seen, 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 occur in pregnant animals (Radostits et al., 2000). Subacute syndrome also occurs in young animals, but fever is mild and hemoglobinuria is absent (Radostits et al., 2008).

In cerebral babesiosis, hyperexcitability, convulsions, opisthotonos, coma, and death, may be 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 (Zaugg, 2009).

2.4.2 Sheep

In sheep develop fever and parasitemia within 2 to 4 days; the clinical signs of the disease include anorexia, listlessness, anemia, moderate jaundice and hemoglobinuria. In intact animals, hyperthermia returned to normal on the fourth day after the peak pyrexia, and parasitemia is eliminated within the course of the disease (Rahbari et al., 2008).

2.5 Diagnosis

Blood smears and clinical findings are useful in acute cases of piroplasmosis, but are not sufficient in subclinical cases. The complement fixation test is used serological test for bovine babesiosis. The most commonly used tests are ELISA, PCR and a DNA probe, which can detect specific parasitemias at very low levels of infection (Radostits, 2008). Recently, the 'reverse line blot (RLB) is a versatile technique for simultaneous detection and identification of small ruminant piroplasm species, based on the recognition of specific gene regions by oligonucleotide probes (Nagore et al., 2004; Qingli et al., 2009; Inci et al., 2010;).

2.6 Treatment and control

After the hemoglobinuria or cerebral signs, prognosis is not well. In acute cases that PVC values are above 12%, treatment will be successful. Supportive therapy such as blood transfusions (4 L of whole blood per 250 kg of body weight), fluids, hematinics, and prophylactic antibiotics are important (Zaugg, 2009). Babesiosis can be treated using diminazene aceturate (3-5 mg/ kg), phenemidine diisethionate (8-13 mg/ kg), imidocarb dipropionate (1-3 mg/kg), and amicarbalide diisethionate (5-10 mg /kg) (Cebra, C., and Cebra, M., 2002a; Taylor, 2007; Radostits, 2008; Zaugg, 2009).

The control of the disease depends on effective quarantine to prevent the introduction of the vector tick. The control of ticks by dipping or spraying animals at risk with recommended acaricides. In routine surgery, Care should be taken to prevent accidental transfer of blood from one animal to another (e.g., castration, dehorning). In addition, in cattle, the selection and breeding of cattle which acquire a high degree of resistance to ticks is practiced. Widespread use of tick vaccines may also have a significant influence on the incidence of infection in cattle (Taylor et al., 2007; Radostits et al., 2008; Zaugg, 2009).

3. Theileriosis

3.1 Etiology

Theileriosis is caused by Theileria spp. in cattle, goats, sheep and wild and captive ungulates (Radostits et al., 2000). Theileriosis is a hemoparasitic disease caused by protozoa of the genus Theileria (Apicomplexa). Theileria species affect domestic and wild ruminants, especially in Africa, Europe, Australia, and Asia (Allison and Meinkoth, 2010). The parasites are transmitted by tick. These parasites, undergo repeated merogony in the lymphocytes ultimately releasing small merozoites, which invade the red cells to become piroplasms. Theileriosis, have a variety of tick vectors which cause infections ranged from clinically inapparent to rapidly fatal (Taylor et al., 2007).

The list of theileria species is shown in table 2.

3.2 Epidemiology

3.2.1 Bovine theileriosis

Bovine theileriosis is caused by at least six Theileria species (Table 2). In bovine, East coast fever caused by *T. parva*. The tick vector is *Rhipicephalus appendiculatus*. East coast fever generally occur in cattle but also buffalo (OIE., 2004), and occurs in East Africa. ECF is prevalent throughout the wetter areas, but is absent from the wet highlands in the horn of

Africa (Norval et al., 1991). In Africa, *T. parva* is a highly fatal disease in cattle (Lawrence et al., 2004; Carlson, 2009a) and this disease is a great threat on the development of the livestock industry, with losses of about 10,000 cattle per year in Zambia (Nambota et al., 1994).

Organism	Livestock affected	Geographic distribution
<i>T. parva</i>	Cattle, African buffalo	East and central Africa
<i>T. annulata</i>	Cattle, domestic buffalo	Mediterranean countries (Portugal and Spain, the Balkans), Middle East, Indian subcontinent and China
<i>T. orientalis</i> complex	Cattle	Southern Europe, Middle East, Asia, Australia
<i>T. velifera</i>	Cattle, zebu	Africa
<i>T. taurotragi</i>	Cattle, antelope, particularly the eland	Africa
<i>T. mutans</i>	Cattle, deer	Africa, Caribbean Islands
<i>T. hirci</i>	Sheep and goats	North and East Africa, Middle East, India
<i>T. ovis</i>	Sheep and goats	Europe, Africa, India

Table 2. Theileia species (Theileriosis)

In bovine, Tropical Theileriosis (Mediterranean coast fever), occurs in Mediterranean countries, Middle East, Indian and China, and is caused by *T. annulata* (Radostits et al., 2008). The tick vectors are *Hyalomma dentritum* in north Africa and in the Mediterranean countries, *H. dentritum* and *H. dromedarii* in central Asia, and *H. marginatum* in India (Taylor et al., 2007; Radostits et al., 2008). In endemic areas indigenous cattle are relatively resistant while modified cattle, especially European breeds, are highly susceptible (Taylor et al., 2007).

Theileria orientalis complex is a milder disease than East coast fever and Tropical Theileriosis, and called benign theileriosis in cattle (Radostits et al., 2008). The tick vectors are *Amblyomma variegatum*, *A. cohaerens*, *A. haebraeum*, *Haemaphysalis bispinosa* that these vectors are the probable vectors in Australia (Taylor et al., 2007).

Theileria velifera has been described in cattle and zebu and is very mild theileriosis in Africa (Taylor et al., 2007; Radostits et al., 2008). The tick vectors are *Amblyomma variegatum*, *A. lepidu* and *A. haebraeum* (Taylor et al., 2007).

Theileia taurotragi has been described in cattle, antelope and eland. Geographical distribution of this disease is in Africa. The tick vectors are *Rhipicephalus appendiculatus* and *Rhipicephalus pulchellus* (Taylor et al., 2007).

Thieleria mutans has been seen in cattle and deer (Carlson, 2009a).

Theileria hirci is malignant theileriosis of small ruminants (Lawrence, 2004;), and is enzootic in North Africa, Middle East, China and India. The tick vectors are *Rhipicephalus bursa* and *Hyalomma anatolicum* (Taylor et al., 2007).

Theileria ovis is benign theileriosis in Africa (Radostits et al., 2008). But in China *Theileria ovis* is a serious disease (Miling et al., 2009). The incidence and mortality rates of *Theileria ovis* in adult sheep and goats were have found 17.12% and 65.78%; 8.06% and 73.33% respectively in China (Shuzhen et al., 2002). In Turkey, prevalence of infection of *T. ovis* in sheep and goats were have been 64.19% and 12.36%, respectively (Sayın et al., 2009). The tick vectors of disease are *Rhipicephalus bursa* in the Mediterranean and *Rhipicephalus evertsi* in Africa (Taylor et al., 2007).

3.3 Pathogenesis

Pathogenesis of different form of theileriosis is based on the production of schizonts in lymphocytes and piroplasms in erythrocyte. *T. parva*, *T. annulata* and *T. hicri* produce highly schizonts and piroplasma and are very pathogenic; *T. mutans*, *T. buffeli*, and *T. ovis* unusually produce schizonts but may cause varying degree of anemia when piroplasms are highly in red blood cells (Radostits et al., 2008).

3.4 Clinical findings

Theileia spp. are classified in to 2 groups. In first group (*T. parva* and *T. annulata*), proliferate is seen in lymphocytes but in the second group (*T. orientalis*) it is seen in erythrocytes that causes hemolytic anemia (Magona et al., 2010).

In *Theileria parva* incubation period is approximately 1-3 weeks (Radostits et al., 2008). One or two days later, the first clinical sign is generalised swelling of the superficial lymph nodes, eyes, ears and submandibular regions. After few days there is anorexia, decreased milk production, loses condition, ceases rumination, rapid hearth beat, petechial haemorrhages under the tongue and on the vulva (Taylor et al., 2007). In cerebral theileriosis there are localized nervous signs and convulsions, tremor, salivation and head pressing (Radostits et al., 2008).

In *Theileria annulata*, pyrexia, anorexia, enlargement of superficial lymph nodes, nasal and ocular discharges and salivation is the most common signs. Constipation is recorded in some cases. Respiratory distress, coughing and pulmonary oedema are seen (Osman and Gaabary, 2007).

In *Theileria orientalis* clinical signs are associated with anemia, jaundice and lymphadenopathy (Taylor et al., 2007).

Clinical signs in *Theileria velifera* is not reported but in *Theileria taurotragi* mild fever and anemia are reported (Taylor et al., 2007).

In *Theileria hicri* which is very pathogenic in sheep and goats, in the acute form there is fever, inappetence, ceases rumination, weakness, enlargement of superficial lymph nodes, diarrhoea, jaundice, haemorrhage in submucous may occur. In chronic form there is fever, inappetence, emaciation, anemia and jaundice (Taylor et al., 2007). *Theileria hicri* is not reported in Turkey (Sayın et al., 2009).

Theileria ovis that is benign theileriosis in sheep and goats, clinically is mild (Taylor et al., 2007).

3.5 Diagnosis

Investigation of giemsa-stained blood smears and lymph node biopsy will reveal piroplasms in erythrocytes and schizonts in lymphocytes. For diagnosis, indirect fluorescent antibody test (IFAT) and indirect enzyme-linked immunosorbent assay (ELISA) are the most commonly used techniques. The ELISA tests is more sensitive than IFAT (Taylor et al., 2007; Radostits et al., 2008).

3.6 Treatment and control

Buparvaquone is the most effective drug and the recommended dose in cattle, sheep and goat is 2.5mg /kg BW (Taylor et al., 2007; Radostits et al., 2008).

In control of the disease use of genetically resistant breed, a judicious and selective application of acaricides at 3- week intervals and the use of vaccines are recommended (Radostits et al., 2008).

4. Sarcocystosis

4.1 Etiology

Sarcocystosis caused by *Sarcocystis* species in cattle, sheep and goats. The names of *Sarcocystis* species are according to their intermediate and final hosts (Taylor et al., 2007; Radostits et al., 2008). The list of sarcocystis species is shown in table 3.

Species	Synonyms	Intermediate host	Definitive host	Geographic distribution
<i>S. bovicanis</i>	<i>S. cruzi</i>	Cattle	Dog, wolf	Worldwide
<i>S. bovifelis</i>	<i>S. hirsuta</i>	Cattle	Cat	Worldwide
<i>S. bovihominis</i>	<i>S. hominis</i>	Cattle	Human	Worldwide
<i>S. ovicanis</i>	<i>S. tenella</i>	Sheep	Dog, fox	Worldwide
<i>S. ovifelis</i>	<i>S. gigantea</i>	Sheep	Cat	Worldwide
<i>S. capracanis</i>	-	Goats	Dog, wolf	Worldwide
<i>S. hircicanis</i>	-	Goats	Dog	Worldwide
<i>S. hircifelis</i>	<i>S. moulei</i>	Goats	cat	Worldwide

Table 3. *Sarcocystis* species (Sarcocystosis)

4.2 Epidemiology

Sarcocystosis occur in all of countries (Taylor et al., 2007; Radostits et al., 2008).

4.3 Pathogenesis

Sarcocystis spp, are protozoon parasites with a requisite two-host life cycle. Sexual reproduction phase occur in the intestine of a carnivore (dogs, cats) later, cysts in the muscles of cattle, sheeo or goats. Sporocysts are shedded by carnivore's feces and then ingested by cattle, sheep, or goats. Then sporocysts hatch in the ruminant small bowel and invade the vascular endothelium during three phases of asexual reproduction. After third phase, merozoites enter the ruminant's muscle tissue and encyst as sarcocysts (Smith and George, 2009; Reilly et al., 2002).

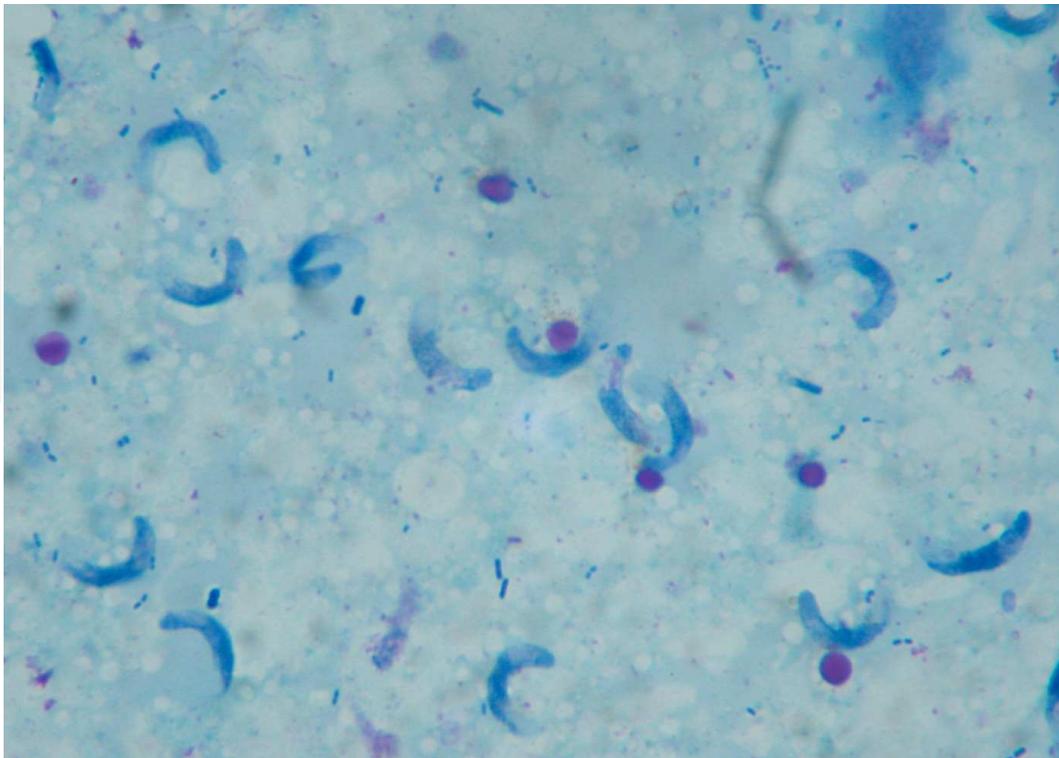


Fig. 4. *Sarcocystis* spp. bradizoit smear, impression smear, Giemsa staining, immersion objective

4.4 Clinical findings

In cattle is usually asymptomatic, but in heavy infections and nonimmune cattle, clinical signs include fever, anorexia, anemia, weight loss, lameness, abortion and diarrhea (Taylor et al., 2007; Radostits et al., 2008). Neurologic signs are occasionally seen in cattle that include ataxia, tremors, muscular weakness, hypersalivation, blindness, opisthotonos and nystagmus (Radostits et al., 2008; Smith and George, 2009). In infections that caused by *Sarcocystis bovicanis* in cattle there is usually loss of hair at the end of the tail (Taylor et al., 2007).

In sheep and goats clinical signs may be asymptomatic. In heavy infections there is fever, loss of weight, anemia and weakness. Abortion may occur (Taylor et al., 2007).

In chronic infections clinical signs include poor weight gain, edema of the limbs, anemia and abortion (Smith and George, 2009). Neurological disorder that is described in sheep caused by *S. Tenella* and *S. Arieticanis* (Radostits et al., 2008). Neurological symptoms, such as depression, in coordination, hind leg paralysis and coma can be seen in Encephalitic Sarcocystosis (Ozmen et al., 2009).

4.5 Diagnosis

Generally *Sarcocystis* infections are diagnosed at meat inspection with grossly visible sarcocysts in the animal's muscle. When infection is very heavy in intermediate hosts, clinical signs and histological evidence of schizonts in the blood vessels of organs and the presence of cysts in the muscles at necropsy will be used for diagnosis (Urquhart et al., 1987).

Indirect hemagglutination test (IHA) and ELISA test are used for serological diagnosis. In acute form of disease titers of antibodies are not high but 1 week to 3 months later will be at diagnostic levels. For certain diagnosis immunohistochemistry, electron microscopy and PCR techniques are available (Radostits et al., 2008).

4.6 Treatment and control

There is no effective treatment for sarcocystosis described in the literature (Urquhart et al., 1987; Radostits et al., 2008; Smith and George, 2009). Treatment of infected calves and sheep with salinomycin (4 mg /kg and 1-2 mg /kg BW; respectively) has been recommended. Amprolium 100 mg /kg, for 30 days reduces the severity of infection (Radostits et al., 2008; Smith and George, 2009).

After beginning nervous system symptoms in sheep, Encephalitic Sarcocystosis recovery has not been observed (Ozmen et al., 2009).

Control of disease is based on protection the food supply of ruminants. Feed bunk should be kept clean, also farm dogs and cats that have access to the feed or pastures should not be fed uncooked meat (Urquhart et al., 1987; Smith and George, 2009).

5. Neosporosis

5.1 Etiology

Neosporosis caused by *Neospora caninum* in cattle, sheep and goats. *Neospora caninum* is a protozoan parasite of the phylum Apicomplexa in the family Sarcocystidae (Radostits et al., 2008; Matthewa, 2009).

The list of sarcocystis specie is shown in table 4.

Specie	Intermediate host	Definitive host	Geographic distribution
<i>N. caninum</i>	Cattle, sheep, goats	Dog	Worldwide

Table 4. Neosporosis

5.2 Epidemiology

The protozoa *Neospora caninum* is an important parasite that cause abortion in cattle, sheep and goats (Gumber et al., 2002). The majority of *N. caninum*- positive cattle prenatally infected via their dams. Transplacental transmission is considered the major route of transmission of *N. caninum* in cattle (Schaes and Conraths, 2007).

In nonfatal infection in the fetus, the fetus is born with neurologic disorder (Smith and George, 2009).

N. caninum has a worldwide distribution, the prevalence of infection in cattle and sheep approaches 100% with a lower ((Taylor et al., 2007; Radostits et al., 2008).

5.3 Pathogenesis

Definitive host: Dogs are the final host and sexual phase occur in them but, they are also intermediate host in prenatal infections (Taylor et al., 2007; Matthews, 2009).

Intermediate host: Cattle are the major intermediate hosts and asexual reproduction phase occur in them (Matthews, 2009). Infection can be transmitted from dam to calf in utero and lactogenically. Infection of cattle can also occur from the ingestion of food or water contaminated with dog feces containing *Neospora caninum* oocysts (Taylor et al., 2007; Radostits et al., 2008). *Neospora caninum* is a major cause of abortion in cattle, however, sporadic abortions can occur in beef cows that have been infected congenitally (Dubey et al., 2006; Taylor et al., 2007; Radostits et al., 2008; Matthews, 2009).

Asexual phase has 2 stages:

1. Tachyzoites: Tachyzoites penetrate host cell like central nervous system, muscles, macrophages and other cells, where they divide rapidly. Tachyzoites can also be transmitted either with contaminated food and water or transplacentally to the fetus in pregnant animals. Tissue cyst containing bradyzoites that these are found only in the nervous system. After the asexual phase, sexual phase occur in definite host. It results in production of oocysts, which is shed in the dog feces.
2. Tissue cysts (Taylor et al., 2007).

Infection in sheep and goats is infrequently (Radostits et al., 2008).

5.4 Clinical findings

In cattle, Neosporosis causes stillbirth, fetal resorption, mummification, abortion and decreases in their milk production (Gumber et al., 2002; Radostits et al., 2008). Abortions in cows are seen between 5-7 month gestations (Gumber et al., 2002). Fetus may be born alive but congenitally diseased (Gumber et al., 2002; Radostits et al., 2008).

Neurological symptoms are different because of the widespread distribution of the parasite in the central nervous system. Calves are born with neurological symptoms, which these symptoms initially are mild but after birth become progress. In calves with neurologic dysfunction clinical signs are included of unable to stand, unable to suckle, domed skull and torticollis (Smith and George, 2009).

5.5 Diagnosis

The diagnosis of neosporosis is based on the examination of maternal and fetal sera ideally combined with the examination of fetal tissues.

- Immunofluorescent antibody test (IFAT) and indirect enzyme-linked immunosorbent assay (ELISA) are used for diagnosis.
- Histopathology of fetus: In histopathological examination of brain characteristic non-suppurative encephalitis is suggestive of *Neospora* infection and also the lesions in the heart are characteristic for diagnosis (Gumber et al., 2002; Taylor et al., 2007; Matthews, 2009).

5.6 Treatment and control

At present, there is no effective treatment for bovine neosporosis.

Control of abortion in infected cattle depends on saving food and water sources and the grazing environment from feces of any animal. Aborted fetuses and placentas should be removed or incinerated. The feces of dogs should be prevented from contaminating animal foodstuffs.

Congenitally infected cows are at high risk for abortion thus seropositive animals should be culling from a herd (Taylor et al., 2007; Radostits et al., 2008).

6. Coccidiosis

6.1 Etiology

Coccidiosis is a protozoan parasitic (genus *Eimeria*) disease that cause diarrhea in calves, lambs and kids (Radostits et al., 2008).

There are 12 species of *Eimeria* in cattle but all of them may not be pathogenic (Table 5).

The species that identified in sheep and goats is shown in table 6 and table 7.

Specie	Affinity site	Hosts
<i>E. zuernii</i>	Small and large intestine	Cattle, zebu, water buffalo
<i>E. bovis</i>	Small and large intestine	Cattle, zebu, water buffalo
<i>E. auburnensis</i>	Small intestin	Domestic cattle, zebu, buffalo
<i>E. alabamensis</i>	Small and large intestine	Domestic cattle, zebu
<i>E. bukidnonensis</i>	unknown	Domestic cattle, zebu, buffalo
<i>E. cylindrica</i>	unknown	Domestic cattle, zebu, buffalo
<i>E. canadensis</i>	unknown	Domestic cattle, zebu
<i>E. ellipsoidalis</i>	Small intestine	Domestic cattle, zebu
<i>E. pellita</i>	unknown	Bovine
<i>E. subspherica</i>	unknown	Domestic cattle, zebu, water buffalo
<i>E. wyomingensis</i>	unknown	Cattle, zebu, water buffalo
<i>E. brasiliensis</i>	unknown	Cattle, zebu, water buffalo

Table 5. *Eimeris* species identified in cattle.

Specie	Affinity site	Hosts
<i>E. crandallis</i>	Small and large intestine	Rocky Mountain big-horn sheep, domestic sheep, moufflon
<i>E. ahsata</i>	Small intestine	Domestic sheep, Rocky Mountain big-horn sheep, moufflon
<i>E. faurei</i>	Small and large intestine	Sheep, Rocky Mountain big-horn sheep
<i>E. intricata</i>	Small and large intestine	Domestic sheep, Rocky Mountain big-horn sheep
<i>E. ovinoidalis</i>	Small and large intestine	Domestic sheep, big-horn sheep, moufflon
<i>E. pallida</i>	unknown	Domestic sheep
<i>E. parva</i>	Small and large intestine	Domestic sheep, Rocky Mountain big-horn sheep, Barbary sheep
<i>E. granulosa</i>	unknown	Domestic sheep, Rocky Mountain big-horn sheep
<i>E. marsica</i>	unknown	Domestic sheep
<i>E. bakuensis</i>	Small intestine	Domestic sheep
<i>E. weybridgensis</i>	Small intestine	Domestic sheep

Table 6. *Eimeris* species identified in sheep.

Specie	Affinity site	Hosts
<i>E. alijeви</i>	Small and large intestine	Domestic goat
<i>E. caprina</i>	Small and large intestine	Domestic goat
<i>E. ninakohlyakimovae</i>	Small and large intestine	Domestic goat, Ibex, persian gazelle
<i>E. arloingi</i>	Small intestine	Domestic goat, Ibex, angora
<i>E. christenseni</i>	Small intestine	Domestic goat
<i>E. aspheronica</i>	unknown	Domestic goat
<i>E. caprovina</i>	unknown	Domestic goat
<i>E. hirci</i>	unknown	Domestic goat
<i>E. jolchijevi</i>	unknown	Domestic goat

Table 7. *Eimeris* species identified in goats.



Fig. 5. *Eimeria* spp. sporulated oocyst, calve, 40X



Fig. 6. *Eimeria* spp. sporulated oocyst, lamb, 40X



Fig. 7. *Eimeria* spp. sporulated oocyst, kid, 40X

6.2 Epidemiology

6.2.1 Bovine coccidiosis

Coccidiosis occurs universally in young animals normally in calves between 3 weeks and 6 months of age and caused important economic losses but also has been reported in cattle aged 1 year or more (Radostits et al., 2008; Ballweber, 2009). The disease occur in animals that kept in crowded and contaminated pens. The prevalence in calves is 46%, in yearling is 43% and in adult cows is 16% (Radostits et al., 2008). *Eimeria zuernii* is the most pathogenic species that causing haemorrhagic diarrhoea (Taylor et al., 2007).

In Turkey the most prevalent species are *E. bovis* (17.6-58.5%), *E. zuernii* (12.7-47.3%), *E. auburnensis* (4.5-64.7%) and *E. canadensis* (4.1-64.7%) (Ozer and Saki, 2001). The prevalence of infection in calves 60-90% have been reported in Turkey (Arslan and Sari, 2010).

6.2.2 Sheep and goats coccidiosis

Coccidiosis in sheep and goats is caused by protozoa of the genus *Eimeria*. Coccidiosis is one of the most common, important and higher infection rates disease in lambs and kids that kept in small areas contaminated with oocysts (Radostits et al., 2008).

In Turkey, the identified species in sheep are *E. ahsata*, *E. bakuensis*, *E. crandallis*, *E. granulosa*, *E. intricata*, *E. marsica*, *E. ovinoidalis*, *E. pallida*, *E. parva*, *E. punctata* and *E. weybridgensis* and in goats are *E. arloingi*, *E. christenseni*, *E. alijevei*, *E. hicri*, *E. caprina*, *E. caprovina*, *E. jolchijevi*, *E. apsheronica*, *E. crandallis*, *E. fauri*, *E. granulosa*, *E. ninakohlyakimovae*, *E. pallida*, *E. parva* and *E. punctata* (Arslan and Sari, 2010).

6.3 Pathogenesis

The pathogenesis of the disease is dependent on the destroy of the crypt cells of the intestinal mucosa because in the ruminant small intestine is very long and providing a high number of host cells and is a potential for parasite replication with minimal damage. Some *E.* species that invade the large intestine, cause pathological changes, specially when large numbers of oocysts are ingested in a short period of time (Taylor et al., 2007).

In healthy nonimmune animals when number oocysts ingested is low, the animals show no clinical signs of disease but in many oocysts condition, rupture and exfoliation of intestinal cells triggers intestine function and causes loss of blood, fluid, albumin and electrolytes into the intestine. Disaster of mucosal capillaries of intestine can cause to hypoproteinemia and anemia. Secondary bacterial infection may cause severe enteritis (Ballweber, 2009).

6.4 Clinical findings

In most clinical cases the temperature is normal or subnormal. Disaster of epithelial cells of intestine cause sometimes bloody diarrhea. Dehydration may occur but anemia occasionally may be seen. In severe infections, diarrhea which may be mucoid or bloody, tenesmus, abdominal pain, anorexia, dehydration and weight loss are the common clinical signs in coccidiosis (Radostits et al., 2008; Ballweber, 2009).

In the calves that infected with high number of oocysts may developed dysentery. Clinical coccidiosis occurs rarely in adult cattle (Radostits et al., 2008).

Coccidiosis in lambs is widely similar to that in calves, but dysentery do not usually occur. In infected kids clinical signs are associated with watery diarrhea, dehydration, anorexia, and weight loss (Ballweber, 2009).

6.5 Diagnosis

Diagnosis is based on history, clinical and necropsy findings, and microscopic examination of feces. Acute coccidiosis can be diagnosed by direct examination of feces but in chronic coccidiosis that very low oocysts number are seen in feces, direct examination of feces may not be adequate (Navarre and Pugh, 2002).

6.6 Treatment and control

Coccidiostats drugs are used for treatment. All animals in the flock should be treated. Trimetoprim+Sulfadoxine (16-24 mg/kg) can be used for three days by intramuscularly in lambs and kids (Ozmen et al., 2004). Amprolium (10 mg/kg BW) for 5 days or 65 mg/kg BW one dose can be effective in calves (Radostits et al., 2008). Diclazuril and toltrazuril are also coccidiocidal drugs (Matthews, 2009). Ghanem et al., (2008), suggested that toltrazuril is more effective than sulphadimine and amprolium in *E. Bbovis* and *E. zuernii* oocysts shedding in buffalo calves.

Because of the low serum vitamin C levels in lambs with coccidiosis, administration of vitamin C is suggested to be useful combined with the classical treatment (Sahinduran et al., 2006).

Hygiene in the house, minimisation of predisposing factors, avoiding the overcrowding of animals in the pens, control of the pastures for parasite are important factors for the control of coccidiosis.

7. Cryptosporidiosis

7.1 Etiology

Cryptosporidium are protozoan parasite with at least 13 species occurring in mammals, birds, and fish, but only 2 of these species are important in livestock animals (Taylor et al., 2007; Radostits et al., 2008). *Cryptosporidium* spp. are intracellular protozoan parasites causing gastrointestinal disease and diarrhea, and they are important infection in young ruminants.

The list of cryptosporium specie is shown in table 8.

Specie	Affinity site	Hosts
<i>Cryptosporidium parvum</i>	Small intestine	Cattle, sheep, goats, deer, man
<i>Cryptosporidium andersoni</i>	Abomasum	Cattle

Table 8. Cryptosporidia species

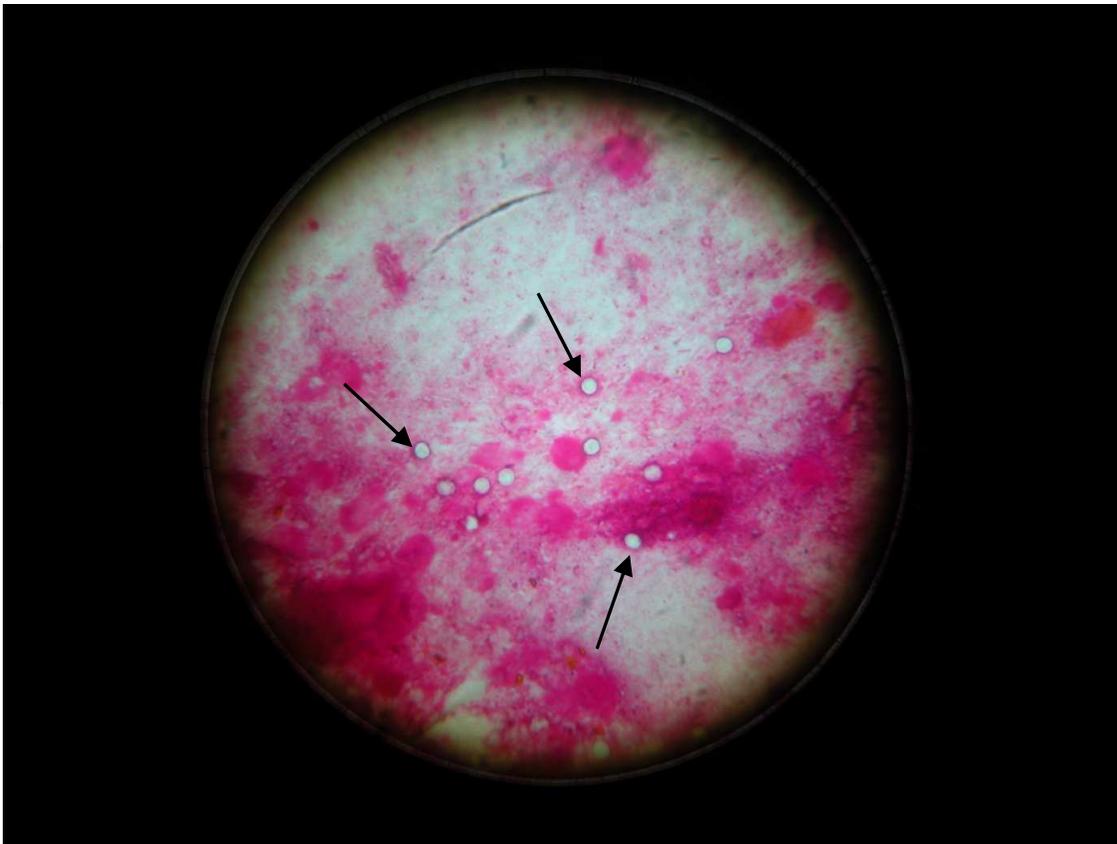


Fig. 8. *Cryptosporidium* spp. oocyst, calve, carbol- fuchsin staining, 40X

7.2 Epidemiology

Cryptosporidiosis occur primarily in neonatal calves, but also in lambs and kids (Radostits et al., 2008). One major species, *C. Parvum*, infects both farm animals and humans (Jones et al., 1997). *Cryptosporidiosis* is a fairly prevalent disease in many countries, and the disease is one of the most economically important diseases especially in calves and kids in Turkey (Sevinc, 2004). Calves at 1-15 days are at the highest risk (El- Khodery and Osman, 2008). The higher percentage of oocysts excreted is observed in 7-day-old calves (Feitosa et al., 2008). Animals of all ages can be infected, but diarrhea occur only in young animals (Gunn et al., 2009). In young calves there is a significant relationship between season and infection (El- Khodery and Osman, 2008). Infection peaks occur in spring and autumn (Taylor et al., 2007). Castro-Hermida et al.,(2007), suggested that asymptomatic adult sheep and goats are the source of environmental contamination by *C. Parvum*.

7.3 Pathogenesis

Cryptosporidium parvum infection, cause a malabsorptive diarrhea (Navarre and Pugh, 2002). *Cryptosporium* life cycle consist of six developmental stages. After ingestion of the oocyt there is excystation, merogony, gametogony, fertilization, oocyst wall formation, and sporogony. In contrast to other protozoal agents, cryptosporidia do not require fecal excretion for sporulation to infective stages, and they sporulate in the intestine (Navarre and Pugh, 2002; Radostits et al., 2008).

7.4 Clinical findings

Infections that caused by *Cryptosporidium andersoni*, are usually asymptomatic, although depressed weight gain in calves and milk yields in milking cows have been reported (Taylor et al., 2007).

In *Cryptosporidium parvum* infections, clinical signs are characterised by anorexia, depression, weight loss, diarrhea, dehydration, high morbidity and possibly death because of dehydration. *Cryptosporidium parvum* can cause diarrhea in calves 5 to 15, and in lambs and kids 5 to 10 days of age (Navarre and Pugh, 2002; Radostits et al., 2008). Relapses of diarrhea are quite common, and *Cryptosporidium parvum* usually occurs as a component of mixed infections (Navarre and Pugh, 2002).

7.5 Diagnosis

Oocysts can be detected using Ziehl-Nielsen stained fecal smears. *Cryptosporidia* can be diagnosed by fecal flotation. A number of molecular and immunological assays are available for diagnosis for example immunofluorescence (IF) or enzyme-linked immunosorbent assays (ELISA) (Taylor et al., 2007).

Cryptosporidiosis is a zoonotic disease, and human can become infected from handling infected animals or feces.

7.6 Treatment and control

There is no effective drug for treatment of cryptosporidiosis in ruminants (Navarre and Pugh, 2002).

Halofuginone has efficacy in calves with diarrhea due to *Cryptosporidium parvum*. Halofuginone is reported to reduce oocyst shedding and the intensity of diarrhea. Oral dose of 0.1 mg / kg BW daily for 7 days can reduce oocyte excretion (Klein, 2008).

Decoquinate, 2.5 mg/ kg, can be effective in prevention of cryptosporidiosis in kids and goats (Navarre and Pugh, 2002; Matthews, 2009).

Cryptosporidiosis in young animals should be treated with fluid therapy and correction of acid-base disturbance.

The control of disease is based on the minimize transmission between the source of the organism and young animals. During the course of the diarrhea, diarrheic young animals should be isolated from healthy animals (Radostits et al., 2008). The best control of cryptosporidiosis comes from calves, lambs, and kids getting adequate immunity through colostrum after birth.

8. Giardiasis

8.1 Etiology

Giardiasis is the infection caused by the flagellate intestinal protozoan, *Giardia intestinalis*, which is also known as *G. duodenalis*, or *G. lamblia*. Infection occur in a wide range of domestic and wild animals and also humans. *G. intestinalis* is a potential pathogen in

livestock, causing diarrhea, weight loss, poor condition and lethargy (Hunter and Thompson, 2005). The parasite is a noninvasive and colonizes the mucosal surface of the small intestine.

The *Giardia* specie is shown in table 9.

Specie	Affinity site	Hosts	Geographic distribution
<i>Giardia intestinalis</i>	Small intestine	Cattle, sheep, goats, man	Worlwide

Table 9. Giardiasis

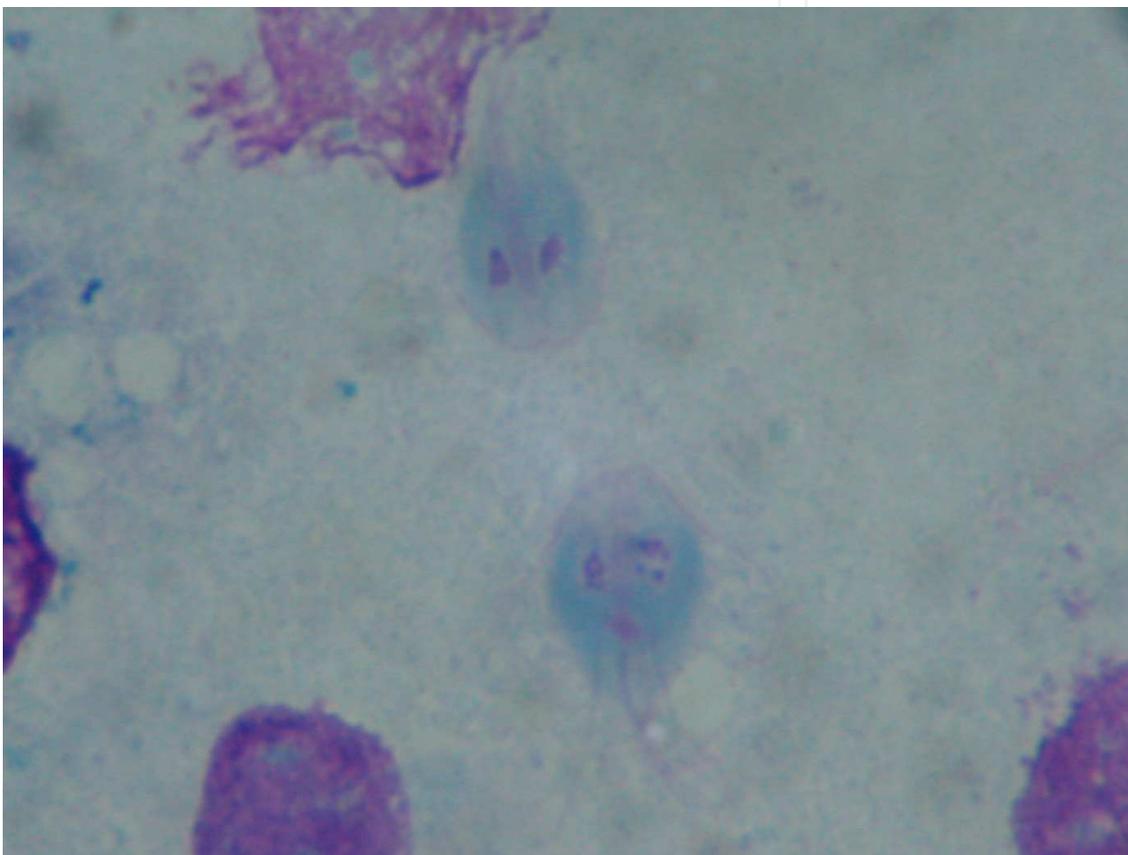


Fig. 9. *Giardia* spp. trophozoit, lamb, Giemsa staining, immersion objective

8.2 Epidemiology

Giardia intestinalis is an important parasite in domestic ruminants also, in human with high levels of infection. Asymptomatic adult animals may act as healthy transporters and may be a source of environmental contamination and infection for young animals. Infection is commonly exhibited by the animals within 2 weeks of birth (Xiao, 1994). Diarrhea may be transient, but infected animals can continue to shed cysts for long times.

Olson et al., (2004) reported a high levels of infection of *Giardia* in young livestock, specially calves. *Giardia* occur in both beef and dairy cattle, and studies have demonstrated prevalence rates of 100% (Xiao and Herd, 1994).

8.3 Pathogenesis

The pathogenesis of giardiasis is similar to cryptosporidiosis. The parasite cause diffuse injure to the small intestinal mucosal surface. Giardia infection cause a reduction in the height of the villus and microvillus, and loss of the absorptive surface area throughout the small intestine. If disease occur in animals, it is believed to result from nutrient malabsorption and due to diarrhea.

8.4 Clinical findings

Clinical signs of giardiasis include acute or chronic diarrhea, dehydration, weight loss, abdominal pain and reduction in weight gain. The diarrhea may be continuous or intermittent.

8.5 Diagnosis

Diagnostic methods for introduce of giardia include direct microscopy, fluoresent antibody staining, ELISA, and PCR(Gunn et al., 2009).

8.6 Treatment and control

Infected sheep and goats can be treated with fenbendazole (5-10 mg /kg) for 3 days (Navarre and Pugh, 2002). Fenbendazole (10 mg /kg BW daily for 3 days) and albendazole (20 mg /kg BW daily for 3 days), have been shown also to be effective in calves (Thompson, 2004; Radostits et al., 2008). Geurden et al., (2006), demonstrated the efficacy and safety of a 5 day treatment with paromomycin against an experimental Giardia infection in calves. Ozmen et al., (2006), suggested that five day treatment with Trimethoprim + Sulfadoxine and multivitamin complexes can be effective in the prevention and treatment of Giardia intestinalis in lambs and kids.

Re-infection frequently occurs if the sources of contamination are not eliminated and the frequency of transmission is high (Thompson, 2004).

9. Besnoitiosis

9.1 Etiology

Besnoitia are protozoan parasite with classified 7 species, which only 2 of these species are important in livestock animals. Besnoitiosis is a emergent parasitic disease that caused by Besnoitia besnoiti in cattle, and Besnoitia caprae in goats. These parasites are in the family Sarcocystidae (Radostits et al., 2008), and they are an obligate intracellular apicomplexan protozoan parasites (Majzoub et al., 2010).

The Besnoitia species are shown in table 10.

Specie	Affinity site	Intermediate hosts	Definitive host
Besnoitia besnoiti	Skin, conjunctiva	Cattle	Cat, wild cats (Lion, leopard)
Besnoitia caprae	Skin, conjunctiva	Goats	Cat, wild cats (Lion, leopard)

Table 10. Besnoitia species

9.2 Epidemiology

Disease occur as outbreaks in tropical and subtropical countries and sporadically in other countries (Radostits et al., 2008). Bovine besnoitiosis is common in Africa, Asia and in the South of Europe (Jacquet et al., 2010), Caprine besnoitiosis in Kenya, Uganda, Iran, and Kazakhstan. In livestock, cattle and goats are intermediate host, and cats are their definitive hosts (Radostits et al., 2008). It is thought that agent is transmitted mainly by hematophagous insects (Jacquet et al., 2010).

9.3 Pathogenesis

Following infection in cattle or in goats, the tachyzoites proliferate in macrophages, fibroblasts, and endothelial cells and causing vasculitis. Subsequently they develop to form bradyzoite cysts in fibroblasts in the dermis, subcutaneous tissues, fascia, nasal and laryngeal mucosa (Taylor et al., 2007; Radostits et al., 2008). After developing cysts in the skin, painful subcutaneous swelling, thickening of the skin, alopecia, and necrosis can be occur (Taylor et al., 2007).

9.4 Clinical findings

9.4.1 Bovine besnoitiosis

Clinical signs occur in two stages: acute and chronic. In acute stage clinical signs include fever, increase in pulse and respiratory rates, painful swelling on the ventral aspects of the body, edema of the skin, sometimes diarrhea, lacrimation and nasal discharge. In chronic stage, there is a severe dermatitis over most of the body (Radostits et al., 2008).

9.4.2 Caprin besnoitiosis

Thickening of the skin over the lower limbs, around the eyes, nose, face and scrotum, alopecia in all of these areas, and white granular cysts in the sclero-conjunctiva can be seen (Oryan and Azizi, 2008).

9.5 Diagnosis

Besnoitiosis can be diagnosed by demonstration of *B. bradyzoites* in skin biopsy smears or scleral conjunctival scrapings (Radostits et al., 2008).

9.6 Treatment and control

There is no known treatment for this disease (Taylor et al., 2007).

Control is effected by the elimination of infected animals and carriers, and early diagnosis (Antonio Castillo et al., 2009).

10. Toxoplasmosis

10.1 Etiology

Toxoplasma gondii is a systemic coccidian, a universal parasite that causes rarely abortion in cattle but is a major abortifacient in sheep and goats (Radostits et al., 2008; Christensen et

al., 2009). Felids are the definitive hosts, and there are wide range of intermediate hosts in this disease (Radostits et al., 2008).

Toxoplasma specie is shown in table 11.

Specie	Affinity site	Intermediate hosts	Definitive host
Toxoplasma gondii	Muscle, liver, lung, reproductive system, central nervous system	Cattl, sheep, goats, man, and all warm blooded animals	cats

Table 11. Toxoplasmosis

10.2 Epidemiology

Toxoplasmosis is one of the most common parasitic infections of man and other warm-blooded animals in most parts of the world (Hill et al., 2002). The disease is a major cause of ovine abortion in many countries (Christensen et al., 2009). Seroprevalence studies show high rates of infection in farm animals. Major importance of disease in farm animals is its zoonotic potential. Source of infection for sheep, and cattle is the oocyst passed in the feces of the cats. Cats shed oocysts by their feces. Cats infected by ingesting tissues of intermediate hosts (Radostits et al., 2008).

10.3 Pathogenesis

The whole developmental process of *Toxoplasma gondi* requires two hosts, the definitive host, cat and the intermediate host (all warm blooded animals).

There are three stages of *Toxoplasma gondii* that are infectious for all hosts: tachyzoites, bradyzoites, and oocysts.

- Tachyzoites: This form of the parasite present during the acute stage of infection in the intermediate host.
- Bradyzoites: present in the tissue cysts. tissue cysts can develop in lungs, liver, and kidneys, and they are more prevalent in muscular and neural tissues, including the brain, eye, skeletal, and cardiac muscle.
- Oocysts: Present only in cat feces and containing sporozoites. Cats shed oocysts after ingesting tachyzoites, bradyzoites, or oocysts. Oocysts are the infective form of importance in livestock animals (Dubey et al., 1998; Radostits et al., 2008).

10.4 Clinical findings

There are a few reports of clinical toxoplasmosis in cattle associated with fever, dyspnea, nervous signs and abortion (Taylor et al., 2007). *Toxoplasma* is not significant in bovine abortion (Radostits et al., 2008). In small ruminants, particularly in sheep clinical signs include fever, abortion, prenatal mortality in lambs, generalized tremor, and dyspnea (Taylor et al., 2007; Radostits et al., 2008).

10.5 Diagnosis

Diagnosis is based on the clinical signs, presence of the characteristic small white necrotic foci in cotyledons. ELISA test is also used to detect antibodies in fetal and maternal serum. Indirect fluorescent antibody test can be used for fluid collected from the fetal thorax or abdomen. Serological examination of dam can be used for diagnosis (Matthews, 2009).

10.6 Treatment and control

Combination of sulfamethazine (SMZ) and pyrimethamine (PMA) can be effective in pregnant ewes. The drugs are effective against the proliferating parasites in the acute stage of the disease (Radostits et al., 2008). The combined dose of (SMZ) and (PMA) is administered over 3 days for 3 periods, commencing on days 110, 115 and 130 of gestation. SMZ is injected s.c. at an initial dose of 5 ml/ 10kg body wt on the first day of a given treatment period with subsequent doses of 2.5 ml/10kg on each of the next 2 days. PMA is administered at 2 mg/kg on the first day and at 1mg/kg on the subsequent 2 days of each treatment period (Buxton et al., 1993).

Because the cats are the only definitive host of toxoplasmosis, it should be kept away from animals farm, and also feeding and drinking water should be prevented from contamination by cat feces. Aborted fetus and excreta, must be strictly disinfected.

It is important to reduce the risk for human disease associated with consumption of infected meat (Radostits et al., 2008).

11. Trypanosomosis

11.1 Etiology

Member of the genus *Trypanosoma* are lived in the bloodstream and tissues of cattle, sheep, and goats in all of the world. These are flagellated protozoal agents that produce a variety of very serious disease of human and animals, but many of them are nonpathogenic (Carlson, 2009b). Trypanosomosis is one of the most important diseases of animals and human. Most African species are transmitted by the tsetse flies (Taylor et al., 2007).

There are 7 species of *Trypanosoma* species in cattle, sheep, and goat but all of them may not be pathogenic (Table 12).

Specie	Affinity site	Hosts
<i>T. brucei brucei</i>	Blood, myocardium, central nervous system, reproductive tract	Cattle, sheep, goats, camel
<i>T. brucei evansi</i>	Blood	Cattle, zebu, goats, camel, deer
<i>T. congolense</i>	Blood	Cattle, sheep, goats, camel
<i>T. vivax</i>	Blood	Cattle, sheep, goats, camel, antelope
<i>T. theileri</i>	Blood	Cattle
<i>T. simiae</i>	Blood	Sheep, goats, camel
<i>T. melophagium</i>	Blood	Sheep, goats

Table 12. *Trypanosoma* species identified in cattle, sheep, and goats.

11.2 Epidemiology

11.2.1 African trypanosomosis (Nagana)

African trypanosomosis is caused by *T. brucei brucei*, *T. congolense*, *T. vivax*, and *T. simiae*, these species affect all domestic mammals and transmitted by tsetse flies.

The most pathogenic species in cattle are *T. congolense* and *T. vivax*. *T. congolense* and *T. vivax* caused severe disease in cattle, sheep, and goat but *T. brucei brucei* causes a subclinical infection in cattle, and severe disease in sheep, and goat (Radostits et al., 2008). *Trypanosoma evansi* causes surra disease of cattle in India.

In Ethiopia the prevalence of infection in bovine which is caused by *T.* species are reported as below:

T. vivax 90.5%; *T. congolense* 4%; and mixed infection of *T. vivax* and *T. congolense* 5.5% (Mihret and Mamo, 2007).

T. theileri is nonpathogenic and reported in North America but occasionally occurs parasitemia that causes fever, depression, and decreased milk production (Carlson, 2009b).

11.3 Pathogenesis

In African Trypanosomosis, after entering through the skin, parasites reach to the bloodstream via the lymphatic system. Infection is characterized with parasitemia. Some trypanosoma spp. invade extravascular spaces such as the ocular aqueous humor and cerebral spinal fluid. Some trypanosoma spp. may produce hemolysin that causes anemia in the host. Then, phagocytic activity is increased because of the massive erythrocyte failure. (Cebra, C., and Cebra, M., 2002b).

11.4 Clinical findings

Clinical findings are based on the speed of onset of anemia and the grade of organ impairment. Trypanosomosis can be acute, subacute, or chronic. In acute form abortion, drop in milk, depression, anorexia can be seen. Hyperemic mucous membranes and lacrimation also can occur. In subacute form clinical signs include weight loss, enlargement of lymph nodes and dry hair coat. In chronic form dull, dry hair coat, inelastic skin, lethargy, pale mucous membranes and exercise intolerance may be seen (Cebra, C., and Cebra, M., 2002b).

T. theileri rarely causes clinical signs, but sometimes parasitemia may develop (Carlson, 2009b).

11.5 Diagnosis

Diagnosis can be based on the clinical findings, presence of vectors, appearance of trypanosomes on a fresh blood smear, or a Giemsa-stained blood smear. Indirect fluorescent antibody test (IFA) and the enzyme-linked immunospecific assay (ELISA) test are used for diagnosis (Cebra, C., and Cebra, M., 2002b).

11.6 Treatment and control

The most common drugs that are used for treatment of trypanosomosis are shown below.

- Diminazene aceturate 3.5-7 mg/ kg BW.
- Homidium bromide and chloride 1mg /kg BW -Pyrithidium bromide 2mg / kg BW.
- Isometamidium 0.25-1mg / kg BW (Radostits et al., 2008).

Vector control can help to control or prevent trypanosomosis. Insecticides can be used for prevent bites by tsetse flies and other flies (Cebra, C., and Cebra, M., 2002b) .

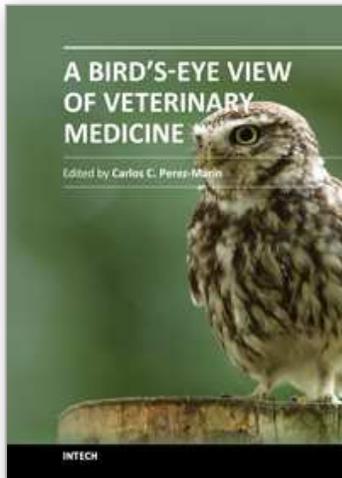
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Veterinary medicine is advancing at a very rapid pace, particularly given the breadth of the discipline. This book examines new developments covering a wide range of issues from health and welfare in livestock, pets, and wild animals to public health supervision and biomedical research. As well as containing reviews offering fresh insight into specific issues, this book includes a selection of scientific articles which help to chart the advance of this science. The book is divided into several sections. The opening chapters cover the veterinary profession and veterinary science in general, while later chapters look at specific aspects of applied veterinary medicine in pets and in livestock. Finally, research papers are grouped by specialisms with a view to exploring progress in areas such as organ transplantation, therapeutic use of natural substances, and the use of new diagnostic techniques for disease control. This book was produced during World Veterinary Year 2011, which marked the 250th anniversary of the veterinary profession. It provides a fittingly concise and enjoyable overview of the whole science of veterinary medicine.

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Phone: +86-21-62489820
Fax: +86-21-62489821

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