African Journal of Basic & Applied Sciences 7 (6): 311-319, 2015
ISSN 2079-2034

DOI: 10.5829/idosi.ajbas.2015.7.6.96202

Coccidiosis in Small Ruminants

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Abstract: Coccidiosis disease caused by a coccidian parasite of genus *Eimeria* cause great economic loss in small ruminants. The disease mostly develops in epithelial cells of small and large intestine. The various species of *Eimeria* are host specific and there is no cross infection between sheep and goat. Although the disease affects all sheep and goats, young lambs and kids are more susceptible. *E.ovinoidalis*, *E.crandalis* of sheep and *E.arloingi*, *E.christenseni* and *E.ninakohlykimovae* of goats are the most pathogenic species of *Eimeria*. The disease may cause local edema and hemorrhage as a result of its effect on intestinal epithelium cells. Clinical Coccidiosis causes diarrhoea, dullness, abdominal pain, leading to dehydration and lose of weight. Necropsy examination offers more reliable indication of clinical Coccidiosis which are mainly characterized by multiple raised white nodules in the intestinal mucosa. The disease is more prevalent in highly overcrowded and animals under stress condition. Finding of a few oocysts in the diarrhea of a sick animal does not justify the diagnosis.Coccidiostat drugs have a limited value in eliminating the disease, but rather they inhibit the development of the causative organism.

Key words: Coccidiosis · Sheep · Goat · Diagnosis · Treatment

INTRODUCTION

Ethiopia has approximately 47.7million cattle, 26.7million sheep, 23.33million goats, 21.7 million horses, 5.57 million donkeys and 1million camels [1]. With an overall 48 million head of sheep and goats, Ethiopia is the third largest among African nations and ranks eighth in the world. Sheep and goats are widely adapted to different climates and are found in all production systems. They contribute quarters of domestic meat consumption, half of domestic wool production and 40% of fresh skin and 92% of semi processed skin export trade [2].

Coccidiosis is one of the most economically important infection threatening sheep and goat industry worldwide [3]. It is a protozoan infection caused by coccidian parasite of genus *Eimeria*. They are intracellular parasite in the epithelium of alimentary tract and it is a self limiting disease [4].

The severity of coccidiosis is dependent on both species of *Eimeria* and size of infecting dose of oocysts [5]. Of the 15 species of *Eimeria* of sheep, *E.ovinoidalis* and *E.ahsata* are highly pathogenic. *E. arloingi, E.christenseni* and *E. ninakohlykimovae* are highly pathogenic *Eimeria* species of goat [6]. It is often difficult to to identify individual species of coccidia due to their similarity in size and shape [7].

Their life cycle consists essentially of several asexual generations (Merogony) followed by sexual generation ending in the development of oocysts which are passed in the faeces [8, 9].

Clinical disease is common after conditions of stress namely weaning, feed change, shipping and in crowded condition which result in excessive manure and urine contamination [10, 11]. Diagnosis is based on microscopic examination, post mortem examination and symptoms. Finding of a few oocysts in the diarrhoea of lamb or kids does not necessarily justify the presence of coccidiosis. It is always adviceable to depend on necropsy findings than faecal examination [12]

It is mainly suspected when there is diarrhoea, poor body growth, weight loss and abdominal pain [5]. Treatment of affected animals with clinical signs includes, supportive care and administration of coccidostat drugs that inhibit the coccidian development [10].

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The objectives of the review are.

- To understand the taxonomical classification of Eimeria.
- The etiology and its different Eimeria species.
- To review on the life cycle, epidemiology, clinical signs, pathogenesis, diagnosis, treatment and prevention.

Coccidiosis in Small Ruminants

Definition: Coccidiosis is one of the most important diseases of sheep and goats which is caused by protozoans of genus *Eimeria* that develop in small intestine and large intestine and it affects young animals in particular [13]. They are highly species specific meaning the species of *Eimeria* that infect sheep will not infect goats or cattle and vice versa [14]. Coccidiosis is of significant economic importance because of losses caused by clinical disease and its sub clinical infection [13].

Historical Background: The microscopic size of coccidian and difficulties encountered when interpreting the different stages of their development explain the complexity of the history.Antony van Leewenhock in 1674, was the first to observe oocysts of protozoan parasite in the bile of rabbit and Stidae 1865 named as *Eimeria stiedai*. The name coccidium appeared for the first time in 1879 in the writings of Leuckart. Its life cycle with alternating sexual and asexual phases, was later clarified due to the hard work of researchers like Balbiani, Eimer and Kauffman. Its classification in 20th century was made difficult due to worldwide distribution of Apicomplexa and diversity of its species [13].

Economic Importance: In intensive breeding conditions, coccidiosis can become an infection of great economic importance. Sub clinical Coccidiosis is probably not of major importance in comparison with other infections. In two studies conducted in East Africa Coccidiosis appear to be secondary cause of mortality amongst small ruminants in combination with other parasite or infectious disease (Pneumonia, Helminthic disease) [13].

Taxonomical Classification: Protozoa are unicellular organisms in which the various activities of metabolism, locomotion are carried out by organelles of the cell [15].

Kingdom…… Protozoa
Phylum…….Apicomplexa
Class…….Conoidasida
Subclass……Coccidiasina
Order--------- Eucoccidiidae
Sub order-----Eimeriorina
Family-------Eimeridae
Genus-------Eimeria

Fig. 1: Taxonomic classification of coccidia [15, 16]

Table 1: Pathogenic species of *Eimeria*in sheep and goat

Species	Coccidian name	Prepatent period	Pathogenesity
Sheep	*E.crandallis*	15-20 days	++
Sheep	*E.ovinoidalis*	12-15days	+++
Goat	*E.arloingi*	20 days	++
Goat	*E.christenseni*	14-23	++
Goat	*E.ninakohlyakimovae*	10-13	+++
Goat	*E.capria*	17-20	++

Source, Kusiluka and Kambarage [17]

Etiology

Species of Eimeria: The common species of *Eimeria* affecting goats in sub-Saharan countries are *E. alijevi, E. arloingi and E. ninakohlyakimovae* and *E. christenseni.* Other species are *E. hirci, E. caprovina, E. jolchijevi* and *E. aspheronica. E. arloingi, E. christenseni* and *E. ninakohylakimovae* are considered to be the most pathogenic species of *Eimeria* in goats [17]. The species affecting sheep include *E. crandallis, E. ahsata, E. faurei, E. intricataand E. ovina*. Other species are *E. ovinoidalis, E. pallidaand E. parva. E.ovinoidalis. E.Crandallis, E. ovina*and *E. ahsata* are known to be pathogenic in sheep [18].

Morphological Characteristics of *Eimeria* Species in Sheep: It has beenreported from many countries around the world. Fifteen *Eimeria*spp. were describedas etiological agents in sheep and 10 specieshave been found in central Europe (Table 2). In Central and Eastern Europe, *E. ovinoidalis*, *E. bakuensis*, *E. crandallis E. weybridgensis*, *E. parva*and *E. faurei*are the predominant species [19].

Life Cycle: The life cycle of coccidia can be divided into two phases: anexogenous phase and an endogenous phase. The exogenous phase takes place outside of the body in the environment and is called “Sporulation of oocysts“. During the endogenous phase, which occurs internally, the parasite undergoes numerous divisions in the intestinal cells. Life cycle takes between 2 and 4 weeks, depending on the species of *Eimeria*. (Fig. 2) [19].

Table 2: Morphological characterstics of *Eimeria* species

Eimeria species	Size	Shape	Color oocyst	Pathogenesis	Oocystresidium	Sporocystresidium	Sporulation day at 20°C
E. ahsata	29-44*17-28	Ovoid	Yellowish	++	-	+	2-3
E. bakuensis	23-36*15-24	Elongated to ellipsoidal	Yellowish	+++	-	+	2-4
E. crandallis	17-28*17-22	Ellipsoidal tospherical	Colourless	+/++	-	+	1-3
E. granulose	22-37*17-26	Jm-shaped ovoidAS	yellowish	-	-	+	3-4
8+k E.entricata	40-56*30-41	Ellipsoidal	Brown	-	-	+	3-7
E. marista	15-22*11-14	Ellipsoidal	colourless	-	-	+	3
E. punctata	18-28*16-21	Ovoid	-	-	+	+	2
E. ovinoidalis	17-28*12-23	Oval to ellipsoid	-	+++	-	+	1-3
E. palida	12-20*8-15	Ellipsoidal delicatwall	-	-	-	+	1-3
E. parva	10-22*10-19	Spherical to sub spherical	-	+/++	-	+	3-5

Source, BAHC[19]

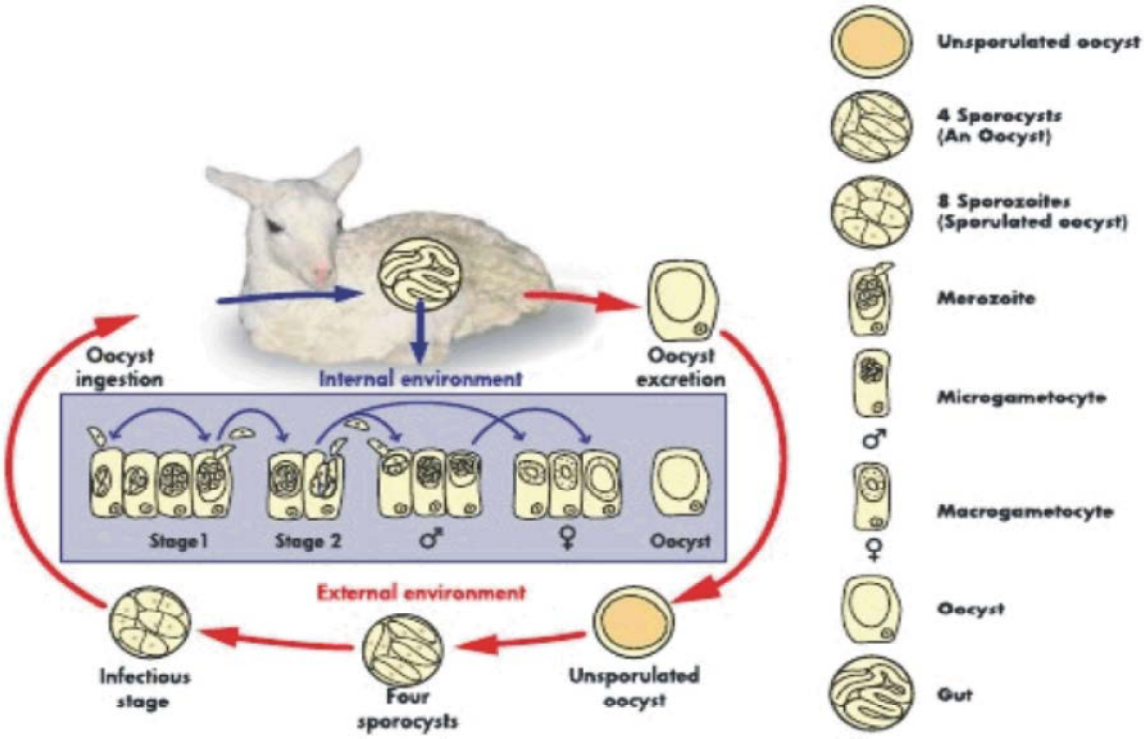

Fig. 2: Life cycle of ovine coccidian [19]

Exogenous Phase: The unsporulated oocysts are passed out in the faecal material of the sheep into the environment. Under optimal environmentalconditions such as: moisture, temperature (24 – 32°C) and oxygen. Unsporulated oocysts of most species sporulate in approximately 2 to 5 days. Oocysts are usually killed at temperatures over 40°C and below -30°C, sporulated and non sporulatedo oocysts can remain viable for more than a year [20]. Unsporulated oocysts are more susceptible toextreme changes in climatic conditions than sporulated oocysts [21]. Oocysts can withstand freezing at-5°C to -8°C for several months [22] and havebeen shown to be able to overwinter in Norway on pastures and be infective to grazing animals in the next grazing season [23].

Endogenous Phase: The endogenous phase of the lifecycle starts after uptake of thesporulatedoocysts. The sheep ingests the sporulated oocysts with contaminated feed orwater. Once into the digestive tract, the digestive tract enzymes exerttheir influence on the oocysts weakening the oocyst wall suficiently so that the active sporozoites escape into the lumen of the gut [24].

It includes: exystation, schizogony and gamogony. Once ingested by the host, the sporulated oocysts undergo process of exycytation which release infective sporozoites in intestinal lumen. In schizogony, each sporozoite (8 from one oocyst) actively penetrates into epithelial cells and transfers into trophozoite and then to schizont. Theschizonts will contain multitude of

merozoites. Each merozoite enter into another intestinal epithelium to produce secondary schizont. These secondary schizonts then create second generation merozoites by asexual multiplication. In gamogony, second generation merozoites penetrate into epithelial cell of large intestine to initate sexual development to form macro and micro gamonts [13].

Fertilization of the macrogamete bythe microgamete results in theformation of a zygote. The zygotelays a wall around itself to form an oocyst. The oocyst breaks out of thehost cell into the intestinal lumenand is excreted with faeces. The prepatent period (The time takenoral uptake of sporulated oocysts to excretion of oocyst) is 15-20 days for *E. crandallis* and 12-15 for *E. ovinoidalis* [19].

Epidemiology: Coccidia can infected animals of all ages and usually cause no clinical sign as immunity is quickly acquired and maintained by continuous exposure to re-infection. However, intensification may alter the delicate balance between immunity and disease with serious consequences for young animals may occur. It is one of the most important diseases of lambs and kids, particularly in the first few months of life. Development of disease is dependent on a number of factors, in particular husbandry and management (Fig. 3). Adult animals are highly resistant to disease, but not totally resistant to infection. As a result, small numbers of parasites manage to complete their life cycle and usually cause no detectable harm [25].

In the wild or under more natural extensive system of management susceptible animals are exposed to only low number of infective oocysts and acquire protective immunity. Under modern production system however, lambs or kids are born into potentially heavily contaminated environment and where the numbers of sporulated oocyts are high and disease often occurs [25].

Coccidiosis is widespread among small ruminants and has been reported in all subsaharan countries. Outbreaks of clinical Coccidiosis with mortality up to 86% have been reported in Nigeria. Studies carried out in Senegal, Ghana, Kenya, Tanzania, Zimbabwe andBotswana have indicated that Coccidiosis is an important subclinical disease which may beassociated with significant economic losses in the small ruminant industry [17].

Coccidia of small ruminant is present anywhere in the world. No data enable us today to confirm that there is a specific geographic distribution for any coccidia species, though surveys show varying prevalence rates [13].

Coccidiosis is likely to become a more important disease of small ruminants in sub-Saharan countries in future as the increasing landscarcity is forcing people to adopt more intensive management systems. Temperature, moisture and oxygen tension are the main factors which determine the survival and development of coccidial oocysts to the infective stage. The optimum temperature for the sporulation of most *Eimeria* spp. oocysts of sheep and goats is 28-31°C whiletemperatures below -40 and above 40°C are considered to be lethal.Sporulated oocysts are resistant to heat and desiccation and at 0-5°C oocysts may remain viable for up to 10 months in faecal sediments and moist pellets. Sunlight andlow oxygen tension are detrimental to the oocyst. The climatic conditions of the humid tropics are favorable for the survival and development of coccidian throughout the year [17].

Coccidiosis is a frequently diagnosed, but often misunderstood and neglected parasitic infection in sheep. Outbreaks of disease are known to occur from the tropics to the temperate zones, but little is known of the significance of coccidial infections in arctic zones [24]. Since sporulated oocysts are the infective stage of the parasite, the sporulation of oocysts excreted via feces in the environment is crucial. There is some evidence that some oocysts survive the winter in most areas of land. Lambs become infected in spring when they ingest the oocysts [20].

Coccidiosis affects mostly young animals. It is observed most frequently in lambs of 2 to 3 weeks age after weaning that are entering feedlots or experiencing a change in diet; or after severe periods of stress, such as shipping, bad weather, or with concomitant disease. The disease may also occur in ewes and lambs that are maintained for long periods of time on contaminated wet areas or heavily stocked irrigated pastures [20]. The susceptibility of lambs to coccidiosis increases with age until about 4 weeks. But, young lambs are most susceptible to coccidiosis [19].

In Ethiopia out of the 384 fecal samples collected in abattoirs, the overall prevalence of *Eimeria*infection is 59.6 percent.It also shows the prevalence rate of *Eimeria* infection between ovine and caprinewith more infection rate in ovine (66.8%) and relatively less in caprine (44.3%) [26].

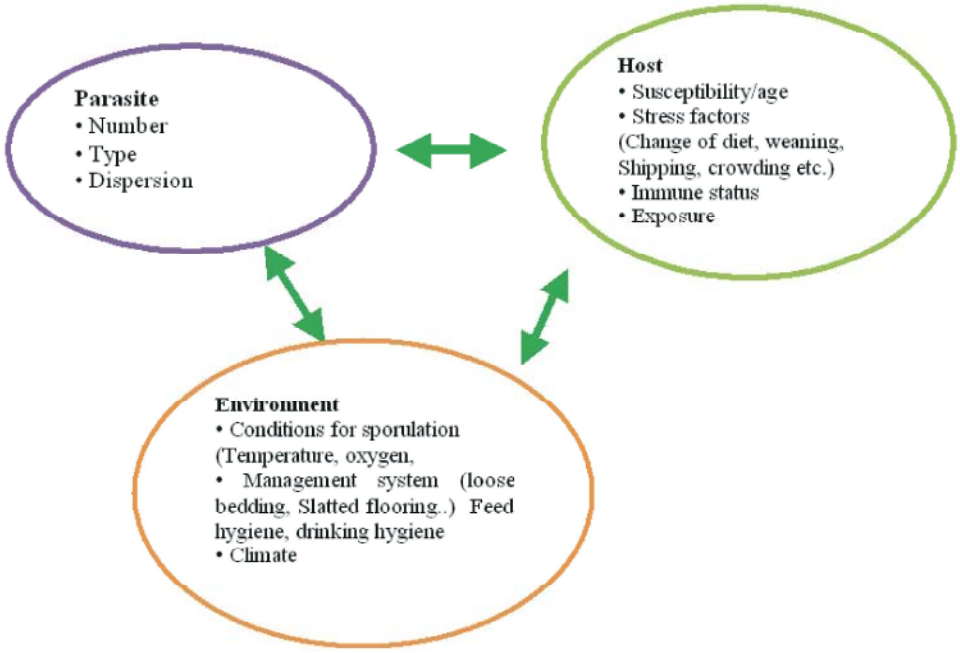

Fig. 3: Factors responsible tooccurance of Coccidiosis [19]

Pathogenesis: The two pathogenic species for lambs are *E. crandiallis* and *E. ovinoidalis* found in the ileum and may also affect caecum and colon. Both species have similar effect on the intestinal tract. Damage to the epithelial cells with subsequent reduction in their numbers is reflected histologically by villous atrophy, crypt hyperplasia and cellular infiltration [14].

The most pathogenic species of *Eimeria* are those that infect and destroy the crypt cells of intestinal mucosa. This is because the ruminant small intestine is very large, providing a large number of host cells and the potential for enormous parasite replication with minimal damage. If the absorption of nutrient is impaired, the large intestine is to some extent, capable of compensating [25].

The pathogenesis of the disease is dependent on the effect of developmental stages of the parasitein various regions of the intestine. The number of oocysts ingested, species of *Eimeria* present, age and immune status of the host, location of the parasite in tissues and number of host cells destroyed determine the severity of the disease. Severe damage tothe intestinal mucosa is caused by the second generation meronts and sexual stages of *Eimeria*. Destruction of capillaries in the intestinal mucosa may lead to hypoproteinaemia and anaemia. Secondary bacterial infection can occur and causesevere enteritis. The changes in the intestinal mucosa cause increased rate of peristalsis, malabsorption and diarrhea. Diarrhea is followed by dehydration, acidosis, anaemia and terminal shock. Coccidiosis is mainly a disease of kids andlambs up to 4-6 months of age and in adult animals the disease is usuallyasymptomatic or mild. The clinical disease occurs when young non-immune animals are exposed to massive challenge with sporulated oocysts [17].

E. ovinoidalis is seen as the mostpathogenic species occurring insheep. Affected animals showdiarrhea, which is usuallyhemorrhagic, abdominal painand anorexia. On postmortem the caecum is usually inflammed, emptyand contracted and the caecal wallis hyperemic, edematous andthickened. In some cases themucosa may be hemorrhagic. Ileumand colon may also be affected. Much of the damage to the caecum is associated with the gamonts because they are most numerous [27].

Table 3: Site of infection and prepatent period of *Eimeria* spp. in sheep [25]

Eimeria spp.	Infection site	Prepatentperiod in days
E. ovinoidalis	Ileum and caecum, colon	12-15
E. crandallis	Ileum and caecum, colon	15-20
E. bakuensis	Small intestine	18-29
E. ahsata	Small intestine	18-30
E. faurei	Small and large intestine	13-15
E. intricate	Small intestine	23-27
E. parva	Small intestine	12-14
E. weybridgensis	Small intestine	23-33

Table 4: Site of infection and prepatent period of Eimeria spp. in goats [25]

Eimeria spp.	Infectious site	Prepatent period (days)
E. alijevi	Small and large intestine	7-12
E. aspheronica	Unknown	14-17
E. arloingi	Small intestine	14-17
E. caprina	Small and large intestine	17-20
E. caprovina	Unknown	14-20
E. christenseni	Small intestine	14-23
E. hirci	Unknown	13-16
E. jolchijevi	Unknown	14-17
E. ninakohlyakimovae	Small and large intestine	10-13

Diarrhoea results from the disruption and inflammation of the intestinal mucosa. In the more typical acute form of the disease, fluid and electrolyte loss result from the compromise of the normal restorative potential of the intestinal epithelium as well as leakage of plasma and lacteal constituents from the inflammed and disrupted mucosa. When extensive, the loss can lead to fatal systemic sequelae of dehydration, acidosis and serum electrolyte derangement [28].

Clinical Signs: Subclinical coccidiosis should be suspected when complaints of poor growth, weight loss reduced feed intake, or loss of fecal pellet formation are reported from young susceptible animals in management situations conducive to the persistence and multiplication of coccidian.Peracute cases caused by severe blood loss in the intestinal lumen may be present at sudden death before signs of diarrhea or abdominal discomfort are seen [28].

In acute cases early signs will include decreased appetite, listlessness, weakness and abdominal pain that may be manifested by crying and frequent rising up and lying down. The feces may be first unpelleted and then pasty and then, a watery yellowish-green to brown diarrhea develop. Diarrhea which may be mucoid or bloody, abdominal pain, tenesmus, inappetance, debility, loss of weight and dehydration, persistent straining in an attempt to pass feces, rough hair coat, are the common features associated withcoccidiosis. Anaemia may also be encountered. In the acute disease, there may befever, ocular and nasal discharges. Clinical coccidiosis is observed commonly in lambs 4-6weeks old. In severe cases there may be acute, bloody diarrhea as a result of extensive damage to the gut epithelium [14].

Coccidiosis is self-limiting; however, other enteric pathogens can complicate the clinical picture. Exposure to low grade challenge results in development of strong immunity against the disease. Successive infections in young animals may cause animals to excrete large numbersof oocysts with subsequent heavy contamination of houses, pastures or wateringplaces [17].

Necropsy Findings: Necropsy examination offers more reliable indication of clinical coccidiosis. Grossly signs of enteritis may range from mild catarrhal to hemorrhagic or necrotic in nature. In per acute cases, the intestinal lumen may contain fresh blood. Thickening of the intestinal wall caused by edema iscommon. The most consistent and characteristic lesion is the occurrence of multiple raised, white nodules measuring between 1 and 6 mm in diameter on the intestinal mucosa, which may be apparent even when the intestine is viewed from the serosal side. These nodules represent sites of active gametogony and histological examination with the history must be considered in establishing coccidiosis as cause of disease [28].

Atnecropsy, gross lesions were seenmostly in the jejunum, ileum, caecum and sometimes in the proximalcolon. Three cases had minimallesions including a few scattered, whitish, non pedunculated to pedunculated nodules on the mucosa of the jejunum and ileum [29]. Eighteen cases had marked lesions including numerous small whitish non-pedunculated nodules on the mucosa of the jejunum, ileum, caecum and proximal colon. Advanced caseshad adenomatous like mucosa and acerebriform or gyrate pattern on the serosal surface. The most commonlesions were in the jejunum, ileum and caecum, observed grossly as non-pedunculated whitish nodules and microscopically, as proliferative enteritis with presence of developmental stages of the *Eimeria* in the hyper plasticenterocytes [29].

Diagnosis: Faecal samples from lambs (Pastyor watery) can be takendirectly from rectum. Most of the ovine *Eimeria* spp. canbe differentiated by an experiencedexaminer by the morphology of the un sporulatedoocysts. For thespecies *E. crandallis* and *E. weybridgensis* this is not possibleand therefore they are differentiatedby the shape and position of the sporocysts and sporozoites. Additionally *E. parva* and *E. palida*are difficult to separate from eachother because of their similarity and therefore sporulated oocysts can behelpful [19]. Following recovery from coccidia infection an animal is relatively immune to re-infection with the same species. But immunity is not solid and the infection will be of low grade and will not harm the host. Hence the presence of oocysts of even highly pathogenic species in the faeces does not necessarily mean that the animal has clinical coccidiosis. On the other hand it may cause severe symptom and even death before oocysts has been produced [30].

Coccidia are probably present in all sheep and the mere presence of oocysts in the faeces is not ground for a diagnosis of coccidiosis. Therefore it is adviceable to conduct post mortem exam- nation on representative members of flock before definitive diagnosis is reached [15]. The only sure way to diagnose coccidiosis is by finding lesions containing coccidia at necropsy. Scrapings from lesions should be mixed on aslide with little physiological saline solution and examined microscopically. Study of sporulatedoocyst is more desirable than unsporulatedoocyst [30].

Sporulation of Faces:

- Weigh 4 g of faces in a petridish, flatten it and add 2% potassium dichromate solution (K2Cr2O7) to avoid overgrowthof fungi and bacteria that kill protozoa.
- Store the samples at 28-30°C with daily aeration, because oxygen is necessary for sporulation of oocysts; if possible permanent aeration is advisable [19].

Differential Diagnosis: Coccidiosis should be differentiated from other diseases such as cryptosporidiosis, colibacillosis, enterotoxemia, salmonellosis, viral enteritis and dietary diarrhea in the case of diarrhea and abdominal pain. In the case of abdominal pain alone with absence of diarrhea, abomasal bloat and other intestinal accidents are suspected. In the case of per acute death due to coccidiosis the differential diagnosis includesenterotoxaemia, bacterial septicaemia, nematodiasis and paramphistomiasis for weaning lambs of 2 to 5 months of age [18, 30].

Treatment: Supportive care is principle therapeutic intervention in acute case of coccidiosis. Diarrheic animal should be removed from group and give oral or parentral balanced electrolyte solution. In pre weaned kids, milk should be fed only in small amounts, as disruption of intestinal mucosa will produce mal digestion and promote osmotic diarrhea from undigested lactose. Severely anaemic kids with acute intestinal blood loss may require blood transfusions [28]. Broad spectrum antibiotics are indicated in severe cases to prevent bacterial septicemia secondary to disruption of intestinal mucosa. Use of anti coccidian drugs in individual active cases may have limited value. Most are used as coccidiostats that inhibit the development but do not eliminate. Therefore the main goal of coccidiostat drug is to reduce the number of additional cases from developing in group of animals [28].

Drugs used for treatment of coccidiosis include sulfonamides, nitrofurans, gonophores' antibiotics and quinolons [30].

Sulfonamides: These are old drugs and are to be used for treatment only. There are several types with efficacy against coccidia: sulfamethazine, sulfaquinoxalineand sulfadimethoxine are usually given as a drench or in feed orwater. Toxicity is a real risk from overdose or long-term treatment and toxic signs are depression and kidneyfailure.

- Sulfadimethoxine:75mg/kg orally for 4-5 days
- Sulfadimidine:135mg/kg orally for 4-5 days
- Sulfa guanidine:280mg/kg orally for 4 days
- Sulfamethazine:50mg/ton of feed[28].

Nitrofurans:

- Amprolium: 50-62.5mg/kg given in drinking water or feed for sheep, 100mg/kgfor goats for 4 days.
- Nitrofurazone:Effective against *Eimeriafaura* at dose of 7-10 mg/kg daily for 7 days[15].
- Ionophores antibiotics:Monensin: 15-20g/ ton of feed, Lasalocid:20-30 g/ton of feed Salinomycin:100ppm in concentrated feed for 3 weeks for Weaning[28].
- Quinolones: Decoquinate-0.5 to 1.0mg/kg orally in feed continuously

Other Drugs: Toltrazuril, clopidol and methyl benzoquate.

Note: Sulfonamides and nitrofurazones offer added benefit of control of secondary bacterial infection [28].

Prevention: Feed lots should be kept dry and clean. The feed trough should be constructed high enough so that there is no wastage from it and they are not contaminated by faeces. Proper drainage of the feedlot is necessary. Where nursing ewes are fed concentrates at pasture, the feeding area should be changed regularly. If inclement weather necessitates that lambs and ewes are to be kept in yards or barns, then the bedding should be changed regularly to avoid accumulation of large numbers of sporulatedoocysts [15].

Animals particularly at risk from coccidiosis are those that are kept indoors on damp bedding, or those on contaminated heavily stocked pastures, particularly in cold wet weather. The incidence of disease can be reduced through avoidance of overcrowding and stress

and attention to hygiene. Raising of feed and water troughs can avoid contamination by reducing the levels of infection. Good feeding of lambs prior to parturition and creep feeding of their progeny will also help to boost resistance to coccidiosis. The control measures are:

- Avoid overcrowding.
- Keep bedding as clean and dry as possible (Coccidiasurvive best in moist conditions).
- Avoid following-on young lambs behind older lambs when at pasture, as the older ones will transmit the infection to younger ones.
- Ensure lambs have sufficient colostrum and nutrition [31].

CONCLUSION

Coccidia of small ruminants, which is caused by *Eimeria* species is the most important disease that cause morbidity and mortality in both sheep and goats. It is a self limiting disease which is widely distributed throughout the world. Clinical coccidiosis which is more sporadic incurs economic losses linked to direct consequences of diarrhoea, retarded growth of animals and mortality. It is the secondary cause of mortality in small ruminants in combination with other parasitic or infectious diseases. It mostly occurs and affects young lambs and kids which are in intensive husbandry and under stress condition. The occurrence of coccidiosis is due to massive ingestion of sporulated oocysts in highly contaminated environment. Necropsy examination offers most reliable indication in for diagnosis of clinical coccidiosis. Based on the above conclusion the following points are recommended:

- There should be proper management of sheep and goats to avoid pre disposing factors for the occurrence of the disease.
- Try to reduce the disease incidence through avoidance of overcrowding and stress.
- Attention should be given for the hygienic system of housing and feeding.
- Prevention should be given more attention than treatment.
- Lambs should be feed with sufficient colostrum feeds.

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