



Sudan University of Science and Technology



College of Veterinary Medicine

Investigation of Parasitic status of cattle in the Sudan University Dairy Farm

تفسي حالات الطفيليات في الأبقار هي مزرعة البان جامعة السودان

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Dedication

The reason of what we became today

*Thanks for your great support and
continuous care.*

To our Parents

We really grateful to both of your

*You have been my inspiration, and my
soul mates.*

To our Sisters & Brothers

*Who taught us to think, understand and
express.*

To our Teachers

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Abbreviation

Symbol	Abbreviation
Dir.	Direct
ELISA	Enzyme Linked Immunoassay
Flo.	Flotation
Kg	Kilogram
L.	Liter
mg	Milligram
No.	Number
PCR	Polymerase Chain Reaction
PCV	Packed Cell Volume
Premat.	Premature cows
Resp.	Respiration rate
Sed.	Sedimentation
Spp.	Species
Temp.	Temperature

Abstract

This studied was carried out to investigate the current status of parasite infection of dairy herd in the Sudan University farm. The farm contains 12 calves 18 premature cow and 34 adult cows. The conventional parasitological method for internal and blood parasite were used. These included direct, floatation and concentration method, together with Giemsa stain for blood parasites.

The samples collected were 64 fecal samples and 64 blood samples. The routine clinical examination was performed these included the general health parameters (temperature, pulse rate, respiratory rate).

The animals which were found negative to parasitic infection showed normal health parameters. The were *Eimeria ssp.* (84%), *Toxocara spp.*, *Trichostrongylus spp.* and *Trichuris spp.* These were with low occurrence (4%). *Monizia expansa* and *Schistosoma spp.* were the least (2%) the number of diseased animals were (78%) and the healthy animals were (22%).

The temperature in the infected animals with parasite varied from 38⁰C to 41⁰C and the pulse rate varied from 60 – 80 pulse \ minutes and the respiratory rate varied from 20 – 30 cycle\ minutes.

The most clinical signs observed in infected cattle included pale of mucous membrane, diarrhea, emaciation and rough coat. Examination of the environment revealed that the hygienic status of the farm was poor and this could explain the increased number of the cows infected by *Eimeria ssp.* and other parasites.

Key word: Dairy cow- Parasite- Fecal sample- Blood sample

الخلاصة

هدفت هذه الدراسة لمعرفة وجود اصابات طفيلية في مزرعة الجامعة، تحتوي هذه المزرعة على 12 من العجول الصغيرة و 18 من العجول غير الناضجة و34 من الابقار البالغة ' وقد استخدمت الطريقة التقليدية لفحص الطفيليات الداخلية والدموية . وقد استخدمت في هذه الدراسة طريقة الفحص المباشر والطفو و الترسيب لفحص عينات البراز ، والمسحة الدموية وصبغة جيمسا لفحص عينات الدم كان عدد العينات التي تم جمعها وفحصها 64 عينة براز و64 عينة دم . وكان الفحص السريري الروتيني يشتمل علي علامات الصحة العامة من (درجة الحرارة ، معدل النبض ، معدل التنفس) . واطهرت الدراسة أن العينات السليمة في الحيوانات الغير مصابة بالطفيليات أن علامة الصحة كانت طبيعية : وأنواع الطفيليات الداخلية التي وجدت في الحيوانات المصابة هي :

***Eimeria ssp.* (84%)**

***Toxocara spp* (4%)**

***Trichostrongylus spp* (4%)**

***Trichuris spp.* (4%)**

***Monizia expansa* (2%)**

***Schistosoma spp.* (2%)**

وقدرت نسبة الحيوانات المصابة ب (78%) و الحيوانات السليمة ب (22%)، وان درجة الحرارة في الحيوانات المصابة تتراوح من (38 الي 41 م⁰) و معدل النبض يتراوح من (60-80نبضة / الدقيقة) ومعدل التنفس يتراوح من (20-30 دورة / الدقيقة) .

ومعظم العلامات السريرية التي لوحظت في الحيوانات المصابة هي عبارة عن شحوب في الاغشية المخاطية والاسهال و الهزال وخشونة في الجلد الخارجي . لقد كشفت الدراسة ان الوضع الصحي لبئية المزرعة كان ضعيفا و هذا يفسر زيادة عدد الابقار المصابة بالكوكسيديا والطفيليات الاخرى.

كلمات استفتاحية: ابقار لبن - طفيليات - عينات دم - عينات براز

Introduction

Cattle are most common type of large domesticated ungulates. They are a prominent modern member of the subfamily *Bovinae*, are the most widespread species of the genus *Bos*, are the most commonly classified collectively. Cattle are raised as livestock for meat (beef and veal), and dairy animals for milk and other dairy products, and as draft animals (oxen or bullocks that pull carts, plows and other implements). Other products include leather and dung for manure or fuel (Bollongino *et al.*, 2012)..

Three fundamental factors that determine the health and productivity of a high-potential dairy cow are these nutrition, comfort and reproduction. Cows are resilient creatures but an uncomfortable environment comes at a cost to their health and productivity (John, 2011).

Good dairy farming practice also ensures that the milk is produced by healthy animals in a manner that is sustainable and responsible from the animal welfare, social, economic and environmental perspectives (FAO and IDF, 2011).

Animal can either mitigate or create adverse environmental conditions. Cows need a clean, dry, comfortable place to lie down. Cattle should have constant access to fresh clean water and feed with sufficient trough space for the number of animals in the group to avoid competition between animals. Good feed quality demands feed storage facilities that prevent feed spoilage. When cows have to stand for long periods because they cannot lie down in a clean dry area, they may become lame (John, 2011).

The large body mass of cows of temperate breeds and their high metabolism makes them susceptible to heat stress under hot conditions. Hot cows eat less, are more likely to become sick and more difficult to get pregnant (John, 2011).

Good hygiene will prevent transmission of disease, but waste management is a significant logistical problem where cattle are managed intensively. Udder health is largely influenced by the level of hygiene practiced before, during and after milking (John, 2011).

Cattle of all ages, but particularly young cattle, are affected by a diversity of internal parasites. Among these are the roundworms (Nematodes), which are primarily parasites of the gastrointestinal tract (a lungworm is included), the liver fluke (Trematodes), tapeworms (Cestodes) in the small intestine, and single celled protozoan parasites (Coccidia) in the lower intestinal tract (Williams and Loyacano, 2001).

Factors affecting parasitism to effectively control internal parasites in organic livestock systems must be known and manage the relevant risk factors. These include: Local climatic and environmental conditions strongly influence which parasites that present and in what number, warm and moist conditions generally increase the risk of significant infections. Breed and genetic selection, age, and stressors such as poor nutrition, pregnancy, birthing and weaning, can increase susceptibility to parasitism (Robyn and Berry, 2014).

Livestock management this includes grazing management (quality and quantity of feed) and stocking rates, pasture species selection and soil health also affected the susceptibility to parasites (Robyn and Berry, 2014).

Objectives:

- 1- To determine the type of internal parasites and blood parasites affecting dairy cows in Sudan University farm.
- 2- To assess the general health of dairy cattle in Sudan university farm.
- 3- To relate the parasitic infection to the general health of affected cattle's.

Chapter One

Literature Review

1-1. Blood parasite:

1-1-1. *Babesia* spp:

1-1-1-1. Etiology:

Babesiosis is an infectious tick-borne disease of livestock that is characterized by fever, anemia, haemoglobinuria and weakness. The disease is also known by such names as bovine babesiosis, piroplasmiasis, Texas fever, red water, tick fever, and tristezza (Zaugg, 2009). The disease 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 the sporozoite form of parasites into the blood stream during the taking of a blood meal (Radostits *et al.* 2008).

1-1-1-2. Pathogenesis:

Babesia spp. are a various group of tick-borne, 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. bigmina* infections are not as virulent as those of *B. bovis*, however the parasites may infect 40% of the red cells (Taylor *et al.*, 2007).

1-1-1-3.Clinical findings:

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). Sub-acute syndrome also occurs in young animals, but fever is mild and hemoglobinuria is absent (Radostits *et al.*, 2008). In cerebral babesiosis, hyper excitability, 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).

1-1-1-4.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; Inci *et al.*, 2010).

1-1-1-5.Treatment:

After the hemoglobinuria or cerebral signs, prognosis is not well. In acute cases that PCV 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, hematinic, 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 propionate (1-3 mg/kg), and amicarbalide diisethionate (5-10 mg /kg) (Taylor *et al.*, 2007; Radostits *et al.*, 2008; Zaugg, 2009).

1-1-1-6.Control:

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).

1-2.Gastrointestinal parasite:

1-2-1. *Eimeria spp:*

Coccidiosis is uncommon in adult cattle but occasional case and even epidemics do occur, especially in dairy cows that have calved 6-8 week (Radostits *et al.*, 2000).

1-2-1-1.Etiology:

The eimeriid coccidia are one of the more conversional groups of protozoa, and their taxonomy and classification have been debated for more than 50 years. The causative agent of coccidiosis by *E.zuernii* and *E.bovis* (Radostits *et al.*, 2008).

1-2-1-2.Life cycle:

The source of infection is feces of clinically affected or carrier animals, and infection is acquired by ingestion of contaminated feed and water, or licking the hair coat contaminated with infected feces.

The coccidial life cycle is self –limiting. The unsporulated oocysts are passed in the feces and develop into the infective stage in the environment. The original single cell divides, forming four sporoblasts, each of which develops into one sporocyst, and within each sporocyst to sporozoites develop. When ingested, the wall of the oocyst breaks down and the sporocyst are released. The sporocyst then enter epithelial cells. Once within the cell the sporozoites undergo asexual multiple fission (schizogony) and become **first-generation schizonts**, which form numerous merozoites. After the schizont matures, the merozoites released with by ruptures, of the epithelial cell. New epithelial cells are again invaded and **second-generation schizonts** occurs in the large intestine. This is followed by the release of another generation of merozoites, which invade epithelial cells and produce the sexual stages, the **macrogametocyte and microgametocyte**. The second-generation schizogony and fertilization of the macrogametocyte by the microgametocyte (**gametogony**) are the stages of the life cycle that cause functional and structural lesions of the large in intestine. As the second-generation schizonts or gamonts mature, the cells containing them slough from the basement membrane and cause

hemorrhage and destruction of the cecum and colon. The oocysts are the result of fertilization of the gametocytes and are discharged at the time of rupture of the cells, which usually coincides with the onset of clinical signs of dysentery (Radostits *et al.*, 2000).

1-2-1-3.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, because pathological changes, especially when large numbers of oocysts are ingested in a short period of time (Taylor *et al.*, 2007).

In healthy non immune 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)

1-2-1-4.Clinical signs:

The incubation period after experimental dosing varies in species of coccidia and animals infected. It ranges from 16 to 30 days in cattle infected with *E.zuernii* and *E.bovis*. A mild fever may occur in the early stage, but in most clinical cases the temperature is normal or sub normal. The first sign of clinical coccidiosis is the sudden onset of diarrhea with foal smelling, fluid feces containing blood and mucous and tarry. Blood may be appear dark, tarry staining of the feces or as streaks or clot, or the evacuation may consist entirely of large clots of fresh ,red

blood. The degree of hemorrhagic anemia is variable depending on the amount of blood loss, and in most naturally acquired cases in calves anemia is not a feature.

However, Dehydration is common but not, usually severe if affected animals continue to drink water. In appetite and in exceptional cases there may be anorexia and body weight gains are subnormal (Radostits *et al.*, 2000).

1-2-1-5.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 (see Appendix 5), direct examination of feces may not be adequate (Navarre and Pugh, 2002).

1-2-1-6.Treatment:

The chemo-therapeutic agent recommended for treatment and control of coccidiosis in calves. **Sulphadimidine** is used widely empirically for the treatment of acute clinical coccidiosis in calves. **Amprolium** is also used for treatment, and there may be a beneficial effect in terms of increased body weight gains and feed consumption compared to untreated controls recovering spontaneously (Radostits *et al.*, 2000).

1-2-1-7.Control:

Control of coccidiosis in feeder calves brought into a crowded feedlot depends on management of population density, or used of chemo-therapeutics, to control the number of oocysts ingested by the animals while effective immunity develops (Radostits *et al.*, 2000).

1-2-2. Toxocara spp:

This parasit is the largest intestinal parasite of cattle. It is a thick worm pinkish when fresh, and the cuticle rather transparent so that the internal organs

can be seen. The egg is subglobular, with a thick pitted shell and is almost colorless (Urquhart *et al*, 2007).

1-2-2-1.Life cycle:

The infection occurs by ingestion of L2 in the egg and non-migratory after transmammary infection with L3 or after ingestion of a paratenic host. However unlike the *T.canis* prenatal infection does not occur. The prepatent period from egg infection is about eight weeks (Urquhart *et al*, 2007).

1-2-2-2. Clinical sign:

The main effects of this infection appear to be caused by the adult worms in the intestines of calves up to six months old. Heavy infections are associated with poor thriving and intermittent diarrhea, and buffalo calves particularly, fatalities may occur (Urquhart *et al*, 2007).

1-2-2-3.Diagnosis:

The subglobular eggs, with thick pitted shells, are characteristic in bovine feces (see Appendix 7), (Urquhart *et al*, 2007).

1-2-2-4.Treatment:

The adult worms are susceptible to a wide range of antihelmintics including piperazin, levamisole and benzimidazoles. All these drugs are also effective against developing stages in the intestine (Urquhart *et al*, 2007).

1-2-2-5.Control:

The prevalence of infection can be dramatically reduced by treatment of calves at three and six weeks of age preventing developing worms reaching patency (Urquhart *et al*, 2007).

1-2-3.*Trichuris spp.*:

The adults are usually found in the caecum but are only occasionally present in sufficient numbers to be clinically significant (Urquhart *et al*, 2007).

1-2-3-1.Etiology:

Species: *Trichuris globulosa* (Urquhart *et al*, 2007).

1-2-3-2.Clinical sign:

The clinical sign is generally negligible although isolated out breaks have been recorded. Spradic disease due to heavy infections is more common in pigs and dogs and is associated with watery diarrhea which usually contains blood (Urquhart *et al*, 2007).

1-2-3-3.Diagnosis:

Since the clinical signs are not pathognomonic, diagnosis may depend on finding numbers of *Trichuris* eggs in the faeces, (see Appendix8). However, since clinical signs may occur during the prepatent period, diagnosis in food animals may depend on necropsy and in dogs on a favorable response to anthelmintic treatment (Urquhart *et al*, 2007).

1-2-3-4.Treatment:

In ruminant the pro-benzimidazoles, modern benzimidazoles, the avermectins/ milbemycons or levamisole by injection are very effective against adult *Trichuris*, but less so against larval stages. In pigs, these drugs may be used, while in the dog, some of the benzimidazoles and milbemycons are the drugs of choice (Urquhart *et al*, 2007).

1-2-3-5.Control:

Prophylaxis is rarely necessary, particularly in ruminant, but in the case of pigs or dogs attention should be given to areas where eggs might continue to survive for long period. Such areas should be thoroughly cleaned and disinfected or sterilized by wet or dry heat (Urquhart *et al*, 2007).

1-2-4.Schistosoma spp.:

It is effected all domestic mammals, mainly important in sheep and cattle. The intermediate host water snails, *Bulinus* and *physopsis spp*, are particularly important in the transmission of bovine and ovine schistsomosis (Urquhart *et al*, 2007).

1-2-4-1.Etiology:

Species:

Major: *Schistosoma bovis*, *Schistosoma mattheei*, *Schistosoma japonicum*.

Minor: *Schistosoma leiperi*, *Schistosoma spindale*, *Schistosoma nasalis* (Urquhart *et al*, 2007).

1-2-4-2.Life Cycle:

The female in the mesenteric vein inserts her tail into a small venule and since the genital pore is terminal, the eggs are deposited, or even pushed, into the venule. There, aided by their spines and by proteolytic enzyme secreted by the unhatched miracidia, they penetrate the endothelium to enter the intestinal sub mucosa and ultimately the gut lumen; they are then passed out in the faeces. The eggs hatch minutes in water and the miracidia penetrate appropriate snails. Development to the cercarial stage occurs without a redial form and there is no metacercarial phase, penetration of the final host by the motile cercariae occurring via skin or by ingestion in drinking water. The developmental period in the snail can be as short as five weeks. After penetration or ingestion the cercariae lose their forked tails, transform to schistosomula, or young flukes, and travel via the blood stream through the heart and lungs to the systemic circulation. In the liver they locate in the portal veins and become sexually mature before migrating to their final site, the mesenteric veins .The prepatent period is 6-7 weeks (Urquhart *et al*, 2007).

1-2-4-3. Pathogenesis:

Schistosomosis is generally considered to be a much more serious and important infection in sheep than in larger ruminants, and even where a high prevalence of the parasite is detected in slaughtered cattle, clinical signs of the disease are seen only rarely. Acute disease characterized by diarrhea and anorexia occur 7-8 weeks after heavy infection and is entirely due to the inflammatory and granulomatous response to the deposition of egg in the mesenteric veins and their subsequent infiltration in the intestinal mucosa. Following massive infection death can occur rapidly, but more clinical signs abate slowly as infection progresses. As this occurs, there appears to be a partial shift of worms away from the intestinal mucosa and reactions to these migrating parasites and their eggs can occur in the liver. At necropsy during the phase of the disease there are marked hemorrhagic lesions in the mucosa of the intestine, but as the disease progresses the wall of the intestine appears greyish, thickened and edematous due to confluence of the egg granulomata and the associated inflammatory changes; on sections of the liver there is also evidence of egg granulomata inadvertently, been swept into small portal vessel. In sheep, anemia and hypoalbuminaemia have been shown to be prominent during the clinical phase apparently as a result of mucosal hemorrhage, dyshaemopoiesis and an expansion in plasma volume. The significance of low-level infection is not known. But it has been suggested that this may have a considerable effect on productivity. There is evidence Experimentally of acquired resistance to reinfection by homologous species and, from natural infections, that resistance may develop as result of prior exposure to a heterologous species (Urquhart *et al*, 2007).

1-2-4-4. Clinical Signs:

These are diarrhea, sometimes blood stained and containing mucus, anorexia, thirst, anemia and emaciation (Urquhart *et al*, 2007).

1-2-4-5. Diagnosis:

This is based mainly on the clinico-pathological picture of diarrhea, wasting and anemia. Coupled with a history of access to natural water sources. The relatively persistent diarrhea, often blood stained and containing mucus, may help to differentiate this syndrome from fasciolosis. The demonstration of the characteristic eggs in the faeces or in squash preparations of blood and mucus from the faeces is useful in the period following patency but less useful as egg production drops in the later stages of infection (Urquhart *et al*, 2007).

In general, when schistosomiasis is suspected, diagnosis is best confirmed by a detailed postmortem examination which will reveal the lesions and, if the mesentery is stretched, the presence of numerous schistosomes in the veins. In epidemiological surveys serological tests may be of value (Urquhart *et al*, 2007).

1-2-4-6. Treatment:

Care has to be exercised in treating clinical cases of schistosomiasis since the dislodgement of the damaged flukes may result in emboli being formed and subsequent occlusion of major mesenteric and portal blood vessels with fatal consequences. The drugs still widely used are the antimony preparations. Tartar emetic, antimony and stibophen. Although these are being superseded by niridazole and trichlorfon. All of which have to be given over a period of days at high dosage rates. Fatalities associated with the use of these drugs are not uncommon. Praziquantel which is used in the treatment of human schistosomiasis is also effective in animals (Urquhart *et al*, 2007).

1-2-4-7. Control:

This is similar to that outlined for *F.gigantica* and *Paramphistomum* infections. Since the prevalence of snail populations varies according to temperature, local efforts should be made to identify the months of maximum snail population, and cattle movements planned to avoid their exposure to dangerous stretches of water at these times (Urquhart *et al*, 2007).

1-2-5. *Trichostrongylus spp.*:

Trichostrongylus spp. is rarely a primary pathogen in temperate areas. But is usually a component of parasitic gastroenteritis in ruminants. By contrast, in the subtropics it is one of the most important causes of parasitic gastroenteritis. One species *T.tenuis* has been implicated in outbreaks of severe enteritis in game birds (Urquhart *et al*, 2007).

1-2-5-1.Etiology:

Species: *Trichostrongylus axei*, *T.colubriformis* (Urquhart *et al*, 2007).

1-2-5-2.Life Cycle:

This is direct and the preparasitic phase is typically trichostrongyloid. Except that exsheathment of the L3 of intestinal species occurs in the abomasum. Under optimal conditions. Development from the egg to infective stage occurs in 1-2 weeks. The parasitic phase is non-migratory and the prepatent period in ruminants is 2-3 weeks. In the horse, *T.axei* has a prepatent period of 25days while in game birds infected with *T.tenuis* it is only 10 days (Urquhart *et al*, 2007).

1-2-5-3.Pathogenesis:

Following ingestion, the L3 of the intestinal species penetrate between the epithelial glands of the mucosa with formation of tunnels beneath the epithelium, but above the lamina propria. When the sub epithelial tunnels containing the developing worms rupture to liberate the young worms about

10-12 days after infection, there is considerable hemorrhage and edema and plasma proteins are lost into the lumen of the gut. Grossly, there is an enteritis, particularly in the duodenum; the villi become distorted and flattened, reducing the area available for absorption of nutrient and fluids. However many such areas appear normal. Where parasites are congregated within small area, erosion of the mucosal surface is apparent. In heavy infections diarrhea occurs, and this, together with the loss of plasma protein into the lumen of the intestine, leads to weight loss. A reduced deposition of protein, calcium and phosphorus has also been recorded. In the case of *T. axei* the changes induced in the gastric mucosa are similar to those of *Ostertagia* with an alteration in pH and an increased permeability of the mucosa. One difference is that the worms penetrate between the glands. Coalescence of the subsequent nodular lesions often result in plaques or ring-like lesions (Urquhart *et al*, 2007).

1-2-5-4.Clinical signs:

The principal clinical signs in heavy infections are rapid weight loss and diarrhea. At lower levels of infections, inappetence and poor growth rates, sometimes accompanied by soft faeces, are the common signs. It is often difficult to distinguish the effects of low infections from malnutrition (Urquhart *et al*, 2007).

1-2-5-5.Diagnosis:

This is based on clinical signs. Seasonal occurrence of disease and, if possible, lesions at post-mortem examination. Faecal egg,(see Appendix 9). counts are a useful aid to diagnosis although faecal cultures are necessary for generic identification of larvae (Urquhart *et al*, 2007).

1-2-5-6.Treatment and control:

Since the use of herbivore manure as fertilizer is a common practice preceding infection, through cleaning and cooking of vegetables is required for

prevention of infection. Alternative against include mebendazole and albandazole (Garcia., 2007).

Successful treatment with ivermectin has also been reported (Ralph *et al*, 2006).

1-2-6. *Moniezia spp.*:

This genus of cestodes is common in ruminants (Urquhart *et al*, 2007).

1-2-6-1. Etiology:

This genus of cestodes is common in ruminants, Specie are *Monizia expansa* and *M.benedeni* (Urquhart *et al*, 2007). *Moniezia expansa* is commonly known as sheep tapeworm or double-pored ruminant tapeworm. It is large a tapeworm inhabiting the small intestines of ruminants such as sheep, goats and cattle. It has been reported from Peru that pigs are also infected (Gómez *et al*, 2008).

1-2-6-2. Life Cycle:

This is similar to Anoplocphala. Mature proglottids or egg are passed in the faeces and on pasture the onchospheres are ingested by forage mites. The embryos migrate into the body cavity of the mite where they develop to cysticercoids in 1-4 months and infection of the final host is by ingestion of infected mites during grazing. The prepatent period is approximately six weeks, but the adult worms appear to be short lived, patent infections persisting for only three months (Urquhart *et al*, 2007).

1-2-6-3. Pathogenesis:

Although generally regarded as of little pathogenic significance there are a number of reports. Especially form Eastern Europe and New Zealand, of heavy infections causing unthreiftiness, diarrhea and even intestinal obstruction. However

Moniezia infections are so obvious, both in life, because of the presence of proglottids in the faeces. At necropsy, that other causes of ill health may be overlooked. It is interesting that experimental studies have failed to demonstrate substantial clinical effects even with fairly heavy worm burdens (Urquhart *et al*, 2007).

1-2-6-4.Clinical Signs:

While a great variety of clinical signs including unthriftiness, diarrhea, respiratory signs and even convulsions have been attributed to *Moniezia*, infection is generally symptomless. Subclinical effects remain to be established (Urquhart *et al*, 2007).

1-2-6-5.Diagnosis:

Diagnosis is done by analysis stool sample in which eggs can be detected (see Appendix 6), or often observation of the gravid proglottids in feces and anus (Bauer., 1990).

1-2-6-6.Treatment and Control:

In many countries a variety of drugs including niclosamide, praziquantel, buna and midline and a number of broad spectrum benzimidazole compounds, which have the advantage of also being active against gastrointestinal nematodes, are available for the treatment of *Moniezia* infection. If this is carried out in calves and lambs in late spring, in temperate areas, the numbers of newly infected mites on pasture will be reduced. Sloughing and reseeded or avoiding the use of the same pastures for young animals in consecutive years, may also prove beneficial (Urquhart *et al*, 2007).

Chapter Two

Materials and Methods

2-1.Study area:

This study was conducted in the farm of Sudan University of Science and Technology, College of Veterinary Medicine which located in Khartoum State particularly Hillat kuku area, Sharg El Neel locality.

2-2.Sampling:

A number of (64) fecal samples and (64) blood samples were collected.

2-2-1.Fecal sample:

The fecal sample (64) were collected from the rectum of cattle. Each samples was clearly labelled with animal identification in clean plastic container.

2-2-2.Blood sample:

The blood was collected in the morning from jugular veins and milk veins using heparinized tubes. The samples were labeled with animal number and transported as soon as possible to the laboratory.

2-3.Parasitological Techniques:

2-3-1.Fecal examination:

The following examination methods were done:

2-3-1-1.Direct method:

A drop of distilled water was placed in the middle of clean dry glass slide and a small amount of the faces was mixed, then cover slip was placed. The sample was examined under the microscope for detection of presence of parasitic ova (Thienpont *et al.*, 1979).

2-3-1-2. Floatation method:

Feaces (1gm) was mixed with distilled water and crushed, then filtered through a fine sieve (Thienpont *et al.*, 1979).

The filtrated was mixed with concentrated sodium chloride (NaCl) 4-5ml, poured in the test tubes and filled with the concentrated salt, then glass slide was placed. The sample examined microscopically for the presence of ova after 15 minutes (Thienpont *et al.*, 1979).

2-3-1-3.Sedimentation method:

The filtration of faces (1gm) after mixed with 30-40 volume of the distilled water. The filtration was mixed with 1-3ml sodium chloride in test tube and centrifuged at 1500 round per minutes for 5 minutes (Thienpont *et al.*, 1979).

The supernatant fluid was discarded and small amount of the sediment was mixed and place in the middle of clean dry glass slide, then cover slip was placed. The sample was examined under the microscope (Thienpont *et al.*, 1979).

2-3-2.Blood film:

The blood tests were made by collecting about 3ml of whole blood from the jugular vein of each animal in a vacationer tube. The tube were taken to the laboratory of the Department of Parasitology of Collage of Veterinary Medicine, Sudan University of the Science and Technology and examined as soon as possible

2-3-2-1.Thin blood film:

A small drop of fresh whole blood was dropped on the middle of one end of the slide and spread right across the slide and then air dried. The slide was labeled and blood films were fixed in absolute methyl alcohol for 2 minutes, stained in 5% diluted Giemsa stain for 30 minutes and wash in distilled water and then dried. Immersion oil was put on the blood film and examined microscopically (Zafar *et al.*, 2006).

2-3-2-2.Thick blood film:

Thick blood films were made after punctured ear veins of calves by sterile lancet. The slide was labeled and blood films were fixed in absolute methyl alcohol for 2 minutes, stained with 5% diluted Giemsa stain for 30 minutes, and washed in distilled water and then dried. Immersion oil was put on the blood film and examined microscopically (Zafar *et al.*, 2006).

2-4.Tick collection:

Each sample animal was carefully checked for ticks, tick specimens were placed into plastic container and examined grossly.

2-5.Clinical examination: Clinical signs were investigated and recorded according to (Kelly, 1984) and including temperature, respiratory and pulse rate.

Chapter Three

Results

Table (1): Showed the total number of the cattle in the university farm distributed according to age, adult were (53%), premature were (24%) and calve were (19%).

Table (2): Showed the result of the parasitological test and health parameter in the apparently health cattle in the University farm. All animals were negative to blood and internal parasites. Respiratory value (10-30 cycle\min), temperature (37.8-39.2⁰C) and pulse vale (55-80 pulse\min) were within the normal level.

Table (3): Showed that only one calve was positive to *Babesia spp.* with increased body temperature (41.2⁰C) and presence of ticks.

Table (4): Showed that only one adult cattle was positive to *Schistosoma spp.* with fecal mixed with blood and mucous.

Table (5): Showed that there were two calve positive *spp.* both calve showed diarrhea and pale mucous membrane.

Table (6): Showed that one premature cattle and one calf were to *Trichuris spp.* with rough hair coat and skinning body condition.

Table (7): Showed that two cows affected by *Trichostrongylus spp.*, the first case was adult cow which showed they were clinical signs of diarrhea and died after one weak form collected to sample, the case was calve diarrhea and no clinical signs appearance.

As show in **Table (8):** Premature which was infected by ticks and showed positive fecal sample test to *Moniezia expansa.*

Table (9): Showed that a large number of the cattle were infected by **coccdiosis** and the main clinical signs of was diarrhea.

Table (10): showed total number and total percentage to diseased cattle form in other healthy cattle.

Fig (1): Showed the percentage of the diseased and healthy cattle among the total number of cattle, it was noted that the percentage of diseased cattle were (78%) and healthy cattle were (22%).

Fig (2): Showed the percentage of each parasite detected in fecal samples that coccidiosis was of large *percentage* (84%), *Toxocara spp.*, *Trichostrongylus spp.* and *Trichuris spp.* were with low occurrence (4%) *Moniezia expansa* and *Schistosoma spp.* were the least (2%).

Table (1): Total number of cattle in Sudan university farm according to age

Age	No. Of cattle	Percentages
Adult	34	53%
Premature	18	28%
Calve	12	19%
Total	64	100%

Table (2): Health parameters of cattle with negative of result to the parasite infection in sdanu university farm

No.	Cattle No.	Cattle Age	Specimen				Health Parameters		
			Blood	Fecal			Resp. (cycle/min)	Temp. °C	Pulse (pulse/min)
				Dir.	Sed.	Flo.			
1	Bull	Adult	-	-	-	-	24	39.7	80
2	906	Adult	-	-	-	-	20	38.5	55
3	907	Adult	-	-	-	-	25	39.2	75
4	908	Adult	-	-	-	-	28	39.0	78
5	344	Premat.	-	-	-	-	24	39.2	70
6	345	Premat.	-	-	-	-	25	38.6	75
7	346	Premat.	-	-	-	-	22	38.4	68
8	348	Premat.	-	-	-	-	28	38.0	65
9	342	Premat.	-	-	-	-	26	39.2	72
10	416	Premat.	-	-	-	-	20	38.7	80
11	2	Calf	-	-	-	-	30	39.3	69
12	6	Calf	-	-	-	-	27	39.5	66
13	10	Calf	-	-	-	-	29	38.8	60
14	12	Calf	-	-	-	-	25	37.5	70
Normal Health Parameter							10-30	37.8-39.2	55-80

Table (3): Calf positive result to *Babesia spp* in Sudan university farm .

No.	Cattle No.	Cattle Age	Specimen				Health Parameters			Note
			Blood	Resp. (cycle/min)	Temp. °C	Pulse (pulse/min)				
1	5	Calf	+	32	41.2	80	Present tick and coccidian			

Table (4): Cattle positive to *Schistosoma spp* Sudan university farm.

No.	Cattle No.	Cattle Age	Specimen				Health Parameters			Note
			Blood	Fecal			Resp. (cycle/min)	Temp. °C	Pulse (pulse/min)	
				Dir.	Sed.	Flo.				
1	Sondos	Adult	-	+	+	+	24	39.1	70	Blood and mucous mixed fecal

Table (5): Calve positive to *Toxocara spp* in Sudan university farm.

No	Cattle No	Cattle Age	Specimen				Health Parameters			Note
			Blood	Fecal			Resp. (cycle/min)	Temp. °C	Pulse (pulse/min)	
				Dir.	Sed.	Flo.				
1	1	Calf	-	+	+	+	24	39.4	60	Diarrhea & pale mucous membrane
2	9	Calf	-	-	+	-	25	39.7	62	Diarrhea & pale mucous membrane

Table (6): Cattle positive to *Trichuris spp* Sudan university farm.

No	Cattle No	Cattle Age	Specimen				Health Parameters			Note
			Blood	Fecal			Resp. (cycle/min)	Temp. °C	Pulse (pulse/min)	
				Dir.	Sed.	Flo.				
1	412	Premat.	-	+	+	+	21	38.7	60	Rough hair coat and thin
2	11	Calve	-	+	+	-	24	39.3	62	Thin

Table (7): Cattle positive to *Trichostrongylus spp* in sudan university farm.

No.	Cattle No.	Cattle Age	Specimen				Health Parameters			Note
			Blood	Fecal			Resp. (cycle/min)	Temp. °C	Pulse (pulse/min)	
				Dir.	Sed.	Flo.				
1	909	Adult	-	-	+	+	24	39.5	66	Diarrhea and poor condition
2	3	Calf	-	+	-	+	20	38.8	58	Diarrhea

Table (8): Cattle positive to *Moniezia expansa* in sudan university farm

No.	Cattle No.	Cattle Age	Specimen				Health Parameters			Note
			Blood	Fecal			Resp. (cycle/min)	Temp. °C	Pulse (pulse/min)	
				Dir.	Sed.	Flo.				
1	350	Premat.	-	+	+	-	24	39.6	72	Present tick

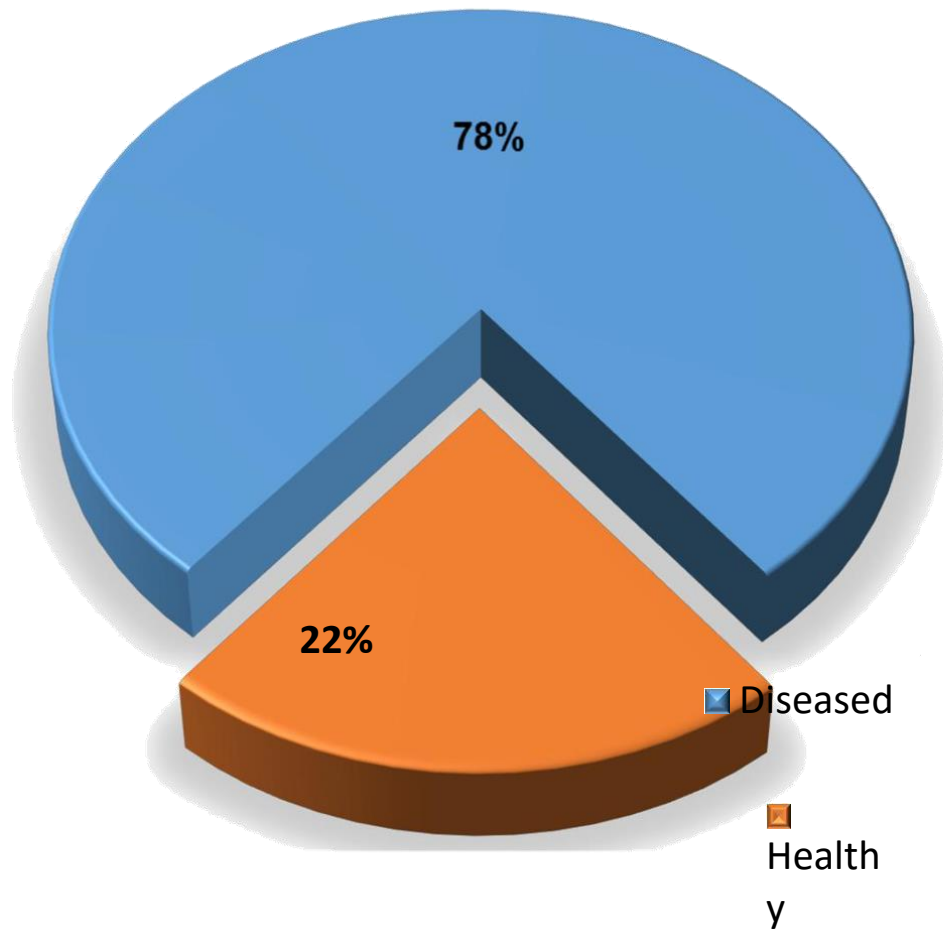
Table (9): Cattle tested for *Coccidia spp* sudan university farm.

No.	Cattle No.	Cattle Age	Specimen				Health Parameters			Note
			Blood	Fecal			Resp. (cycle/min)	Temp. °C	Pulse (pulse/min)	
				Dir.	Sed.	Flo.				
1	426	Adult	-	+	+	+	25	39.2	70	---
2	458	Adult	-	+	-	+	30	39.6	80	---
3	461	Adult	-	+	+	-	24	39.3	58	---
4	462	Adult	-	-	+	+	20	38.4	55	---
5	463	Adult	-	+	-	+	26	39.1	60	---
6	464	Adult	-	+	+	+	22	39.7	64	---
7	904	Adult	-	+	+	+	26	38.6	72	---
8	905	Adult	-	-	-	+	30	40.1	78	---
9	910	Adult	-	+	+	+	22	39.2	60	---
10	911	Adult	-	+	+	+	25	39.6	64	---
11	912	Adult	-	+	+	+	27	38.4	67	---
12	913	Adult	-	-	+	+	28	40.1	80	---
13	915	Adult	-	+	-	+	23	38.6	58	---
14	916	Adult	-	+	-	+	24	38.4	68	---

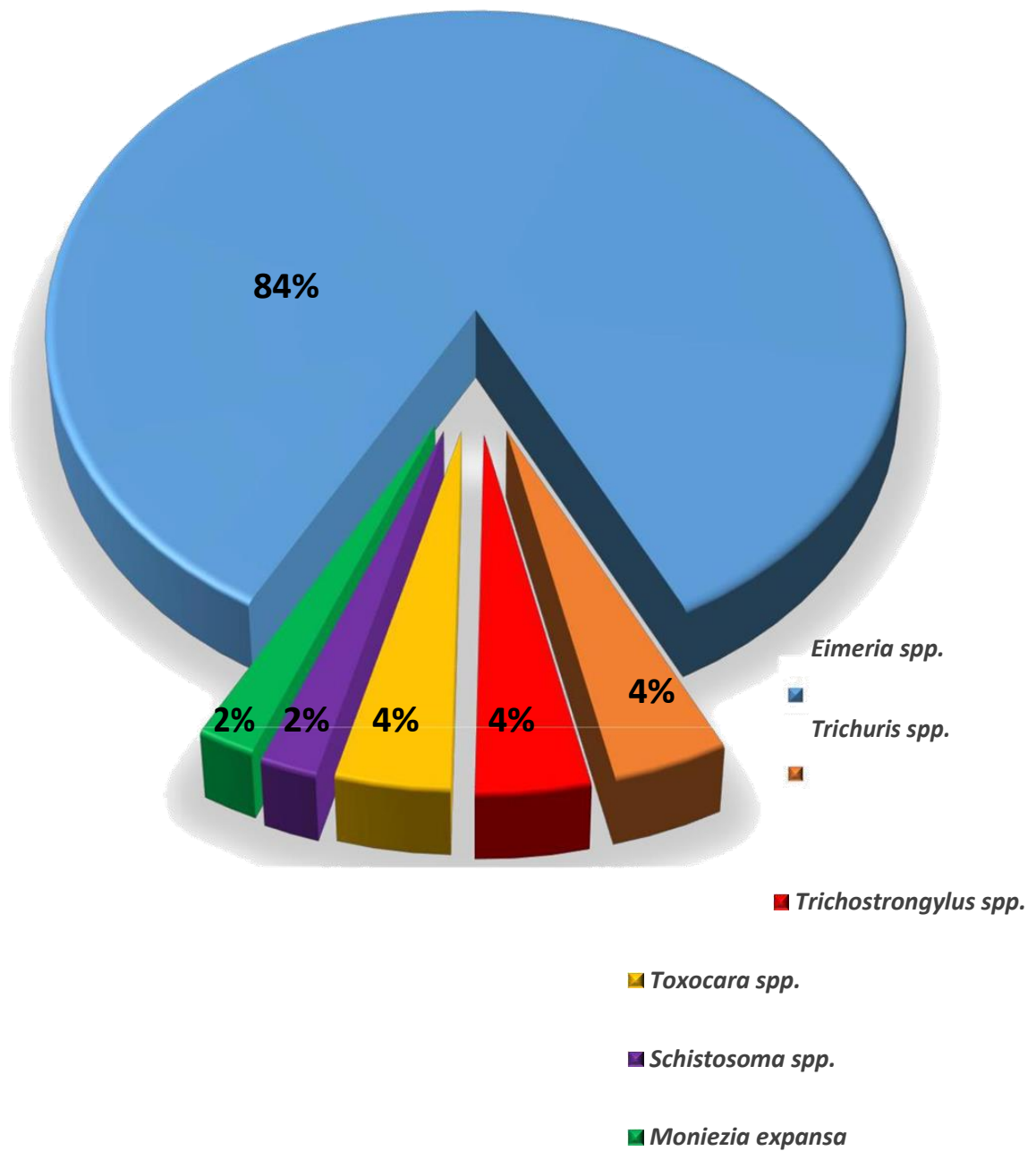
15	917	Adult	-	+	+	+	26	39.0	68	---
16	918	Adult	-	-	+	-	24	39.1	65	---
17	919	Adult	-	-	+	+	22	38.6	60	---
18	922	Adult	-	-	+	+	27	39.3	76	---
19	923	Adult	-	-	+	+	25	39.7	72	Rough hair coat
20	924	Adult	-	+	+	+	23	40.0	59	Emaciation
21	925	Adult	-	+	+	+	24	40.2	64	Emaciation &diarrhea
22	926	Adult	-	-	+	-	25	38.6	64	---
23	927	Adult	-	+	-	+	22	39.1	68	---
24	928	Adult	-	-	+	-	28	38.0	68	---
25	929	Adult	-	+	-	+	30	39.2	74	---
26	Essra	Adult	-	+	+	+	28	38.7	70	---
27	Amina	Adult	-	+	+	+	20	38.5	57	---
28	Marwa	Adult	-	+	+	+	22	38.5	55	---
29	341	Premat.	-	+	-	+	25	39.4	69	---
30	343	Premat.	-	+	+	-	29	39.7	78	---
31	347	Premat.	-	+	-	+	26	39.3	70	---
32	349	Premat.	-	+	+	-	22	38.5	59	---
33	360	Premat.	-	+	+	+	28	40.2	80	Bad smell diarrhea
34	410	Premat.	-	+	-	+	23	39.1	58	---
35	413	Premat.	-	-	+	-	27	39.4	69	Rough coat
36	414	Premat.	-	+	+	-	28	40.0	76	---
37	415	Premat.	-	+	-	+	26	39.5	67	Rough coat &thin
38	417	Premat.	-	+	-	-	20	38.6	58	Diarrhea
39	4	Calf	-	+	+	-	25	39.3	66	Diarrhea
40	5	Calf	+	-	+	+	32	39.7	64	Present tick & babesia
41	7	Calf	-	+	+	-	20	39.1	60	Diarrhea
42	8	Calf	-	+	+	-	22	39.4	62	Diarrhea

Table (10): The total number of healthy and infected cattle in the Sudan University farm

Age	No. Of cattle	No. Of healthy cattle	No. Of Diseased Cattle	Diseased cattle %
Adult	34	4	30	88.2%
Premature	18	6	12	66.7 %
Calve	12	4	8	66.7 %
Total	64	14	50	78.1%



Fig(1) The precentage of healthy and diseased cattle among the total number of cattle in sudan university farm



Fig(2)Percentage of each parasite found in feecal samples of examined cattle in sudan university farm

Chapter Four

Discussions

This study came out to investigate the parasitic infection in university farm at Hilat kuku. The result, indicated the presence of 50 cattle infected among the total number of animal in the farm 64.

Babesiosis was found in one calf, were also founded to be infected with Babesiosis representing as reported by 8.9 % Hazem *et al.*, (2014).

Schistosoma spp. with blood and mucous in feces was detected in one animal also this parasite were reported to occur in 2.7% of cattle which were heavily infested (Sumanth *et al.*, 2004).

Toxocara spp. was detected in two calves with diarrhea and pale mucous membrane the presence of this in dairy cattle and calves. The prevalence of *Toxocara spp.* in cattle was 4.6% in Ankara (Güralp *et al.*, 1985).

Trichuris spp. during the investigation was detected in one premature cow and one calf. This presence of *Trichuris spp.* 5.27% was also documented by Belem *et al.*,(2001).

In the investigation of *Trichostrongylus spp.* was detected in one adult dairy cow and one calf. This parasite was the most prevalent in cattle 3.83% and reported by Belem *et al.*,(2001).

Moniezia expansa although it is a ruminant parasite, but during study was detected in one premature cow. The prevalence of *Moniezia expansa* in cattle were 0.65% reported by Ken and Rex.,(2001)

In this study *Eimeria spp.* was found to be the most parasite detected in all age group of the dairy herd (42/84%). with clinical signs of rough hair coat, emaciation and diarrhea. The prevalence in calves is 46%, in yearling is 43% and in adult cows is 16% (Radostits *et al.*, 2008).

It is worth to mention that the hygienic status at the University farm during this investigation was very poor and this may explain the general parasitic infestation in the dairy herd.

Conclusion and Recommendations

Conclusion

The mixed parasitic detected in the University farm were *Eimeria spp.*, *Schistosoma spp.*, *Trichuris spp.*, *Toxocara spp.*, *Trichostrongylus spp.* and *Moniezia expansa*, and blood parasite was *Babesia spp.*

In the present study there are three group of cattle according to age (adult, premature and calve) were investigation in infection in adult animal was heavy, moderate in premature cows and calves. Highly prevalence in the coccidiosis in farm due to status in farm.

Recommendations

- 1- The routine examination of the herd for healthy status and parasitic analysis.
- 2- To follow the documented rules for the hygienic and healthy environmental in farm.
- 3- Prophylactic control of the parasitic infestation.

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Appendices



Appendix (1): An overview of the University (SUST) dairy farm



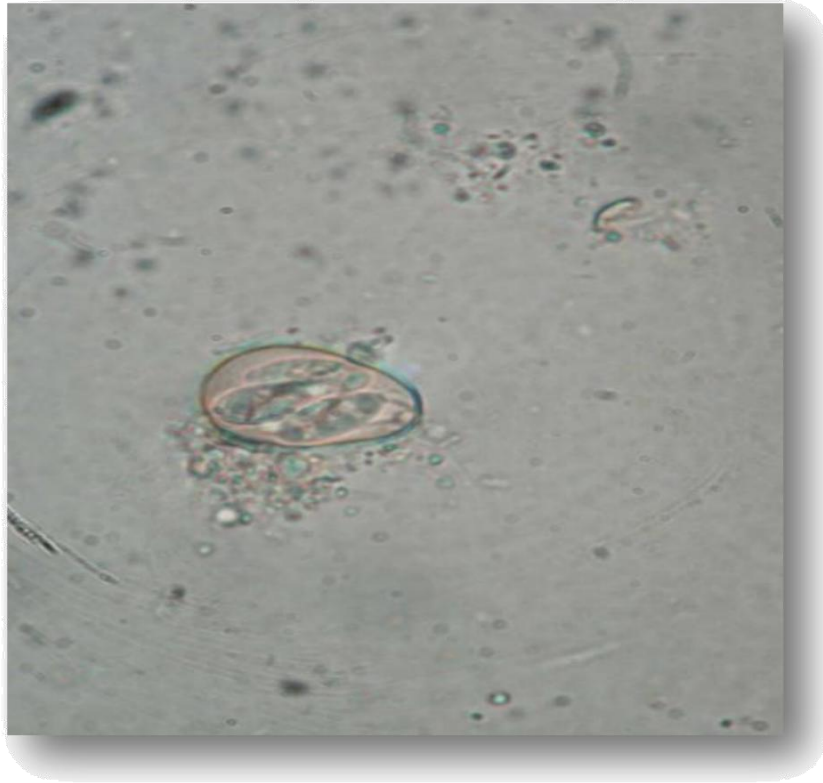
Appendix (2): The side of taking the blood samples from the animals



Appendix (3): A case affected by coccidiosis and appearance emaciation and rough hair coat.



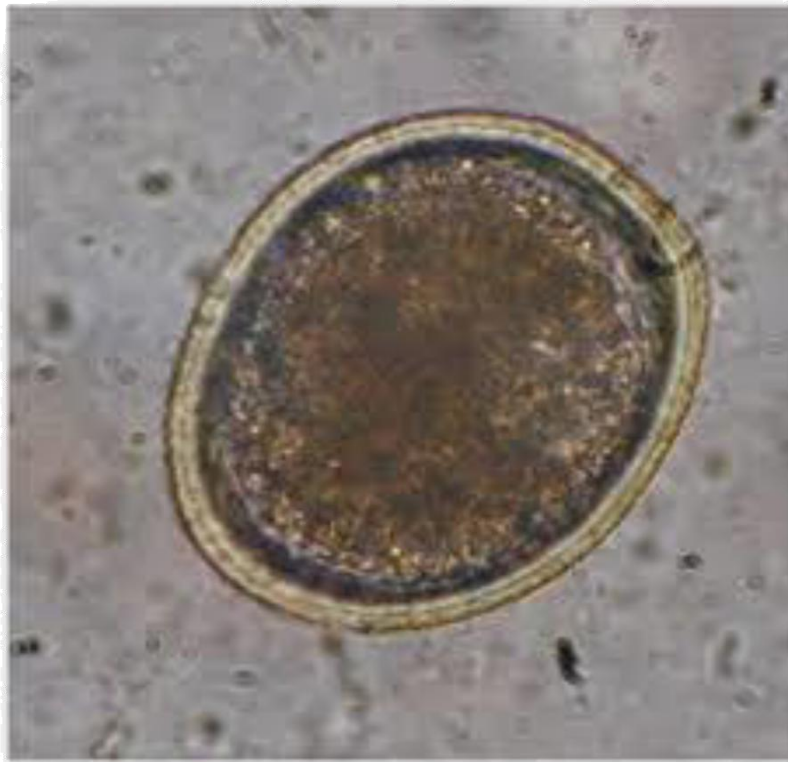
Appendix (4): A cow affected by coccidiosis (Emaciation due to severe diarrhea)



Appendix (5): *Eimeria* spp. sporulated oocyst in calf



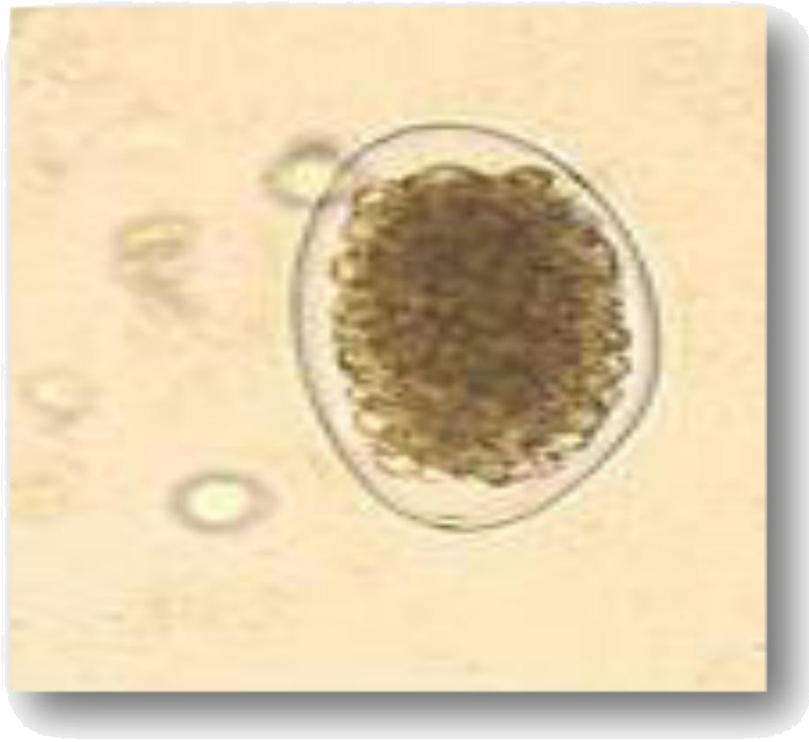
Appendix (6): Egg of *Moniezia* spp.



Appendix (7): Egg of *Toxocara* spp



Appendix (8): Egg of *Trichuris* spp



Appendix (9): Egg of *Trichostrongylus* spp