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Sudan University of Science and Technology
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**Study of Some Hematological and Biochemical
Parameters of Babesiosis in sheep In Shikan
Locality – North Kordufan State – Sudan**

**دراسة بعض عناصر الدم والكيمياء الحيوية لمرض الباييزيا في الضأن في محلية
شيكان – ولاية شمال كردفان - السودان**

*A thesis submitted to the College of Graduated Studies in fulfillment
of the Requirement for the Degree of Master in Veterinary Medicine*

By

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DEDICATION

- ✚ To my father in his grave.
- ✚ To my dear Mother and my wife
- ✚ To My brothers and sisters
- ✚ With my all love and respect"
- ✚ Muzamil

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ABSTRACT

The aim of this study was to evaluate some hematological and biochemical parameters of ovine babesiosis in Shikan Locality North Kordufan State. A total of 150 samples of whole blood and blood for serum were taken from the jugular vein of suspected sheep which clinically examined for heart rate, respiratory rate, and body temperature. For identification of the protozoan, GIEMSA- stained blood smear method was used. Also homological (hemoglobin concentration, red blood cells, hematocrit red cell distribution width, platelet distribution width, mean platelet volume and platecrit) and biochemical (Total protein, albumin, globulin, urea, creatinine, cholesterol, and triglyceride) Parameters were measured. The results revealed that all health parameters were elevated in infected animals (20 animals) compared with negative infected animals (76) and control animals (54) to ovine babesiosis. There was decrease of hemoglobin concentration (4.83 ± 0.87), hematocrit (13.2 ± 5.77) and red blood cell counts (3.42 ± 1.10) compared with control animals (9.11 ± 1.24) (21.06 ± 6.55) (6.24 ± 2.54 respectively). But there was slight increase of red cell distribution width (19.1 ± 6.37) and mean platelet volume (7.23 ± 1.94) Compared with control animals (18.3 ± 4.05) (6.85 ± 1.38 respectively). Infected animals with parasite investigated for biochemical alterations revealed that an elevation in total protein (8.97 ± 0.80), globulin (5.40 ± 1.0), urea (34.5 ± 9.26) creatinine (1.14 ± 0.41), Cholesterol (55.0 ± 16.26) and triglyceride (59.9 ± 18.99). The albumin (3.19 ± 0.300) was lowered. Diminazene aceturate was given at dose rate 2mg/kg b.wt., I/M single dose followed by supportive therapy given based on requirement of the individual case and no death recorded. In conclusion sheep with babesiosis showed variable depletion in level of hemoglobin, red blood cells, hematocrit, platelets, plateletcrit and mean cell volume, in infected sheep babesiosis serologically total protein, urea, creatinine, globulin, cholesterol and triglyceride revealed variable increase compared to healthy sheep in all parameters evaluation of the

results leads to the suggestion that severe anemia resulted from *Babesia ovis* infection may cause kidney and liver damage in sheep with babesiosis. Consequently, the investigation of kidney and liver elements would be a useful way to follow the prognosis of the disease, as well as to provide supportive therapy to reduce potential kidney and liver effects.

المستخلص

هدفت هذه الدراسة الي تقييم بعض المتغيرات الدموية و الكيميائية الحيوية لداء بابيزيا الاغنام من مارس 2018 الي فبراير 2021 في محليه شيكان - ولاية شمال كردفان . تم اخذ ما مجموعه 150 عينة من الدم الكامل والدم للمصل من الوريد الوداجي لأغنام مشتبه بها طبيعيا تم فحصها سريريا لمعدل ضربات القلب ,معدل التنفس ودرجة حرارة الجسم .لتحديد الطفيل -جيمسا -ملطخ وطريقة مسحة الدم, وكذلك متمائل (تركيز خضبات الدم, خلايا الدم الحمراء , مكداس الدم , عرض توزيع خلايا الدم الحمراء , عرض توزيع صفيحة الدم , مكداس الصفيحة الدموية ومتوسط حجم الصفيحة) والكيمياء الحيوية (البروتين الكلي , زلال , يوريا , كرياتينين , كوليسترول وثلاثي الجليسريد) . اظهرت النتائج ارتفاع جميع المعايير الصحية في الحيوانات المصابة (20 حيوان) مقارنة بالحيوانات السالبة (76 حيوان) وكذلك الحيوانات غير المصابة (54 حيوان) لمرض البابيزيا في الاغنام . كان هناك انخفاض في تركيز خضاب الدم (0.87 ± 4.83) , مكداس الدم (5.77 ± 13.2) و كريات الدم الاحمر (3.42 ± 1.10) مقارنة بالحيوانات غير المصابة علي التوالي (6.24 ± 2.54 , 21.06 ± 6.55 , 9.11 ± 1.24) . لكن يوجد زيادة طفيفة في عرض توزيع الخلايا الدم (6.37 ± 19.1) ومعدل حجم الصفيحة (1.94 ± 7.23) مقارنة بالحيوانات المصابة (4.05 ± 18.3 , 1.38 ± 6.85) علي التوالي. اظهرت الحيوان المصابة بالطفيل التي تم فحصها من اجل التعديلات الكيميائية الحيوية ارتفاع البروتين الكلي (0.80 ± 8.97) , قولوبيولين (1.0 ± 5.40) , يوريا (9.26 ± 34.5) , كرياتينين (1.14 ± 0.41) , كوليسترول (16.26 ± 55.0) وثلاثي الجليسريد (18.99 ± 59.9) , بينما انخفض الزلال (0.300 ± 3.19) الاغنام التي اظهرت اصابة بداء البابيزيا تم اعطاء عقار دايمينزيين داي اسيتيورات بجرعة 2 مجم علي كجم من وزن الحيوان مع علاجات داعمه بناء علي الاحتياجات الفردية لكل حيوان ولم تسجل حالات نفوق بينها وفي الختام اظهرت الاغنام المصابة بداء البابيزيا استنفادا متغيرا في مستوي الهيموجلوبين او مكداس خضاب الدم وخلايا الدم الحمراء والهيماتوكريت والصفائح الدموية ومتوسط حجم الخلية , في داء بابيزيا الاغنام المصابة مصليا البروتين الكلي ,اليوريا ,الكرياتينين , الجلوبيولين , الكليسترول والدهون الثلاثية اظهرت زيادة متغيرة مقارنة بالصحة . تقييم نتائج الاغنام في جميع المعايير يؤدي الي اقتراح ان فقر الدم الحاد الناتج عن عدوي البابيزيا قد يتسبب في تلف الكلي والكبد في الاغنام المصابة ببابيزيا وبالتالي فإن فحص عناصر الكلي والكبد سيكون وسيلة مفيدة لمتابعة تشخيص المرض ,بالاضافة الي توفير العلاج الداعم لتقليل الاثار المحتملة علي الكلي والكبد.

ABBREVIATIONS AND ACRONYMS

ANOVA	-	Analysis Of Variance
BUN	-	Blood Urea Nitrogen
CFT	-	Complement fixation test
DN	-	Deoxy ribonucleic Acid
E	-	Eggs
E.G.	-	Example given
EDTA	-	Ethylenediaminetetraacetic acid
ELISA	-	Enzyme-linked immunosorbent assay
FAO	-	Food and Agriculture Organization
HB	-	Hemoglobin
FAT	-	Fluorescent antibody tests
G	-	Gametocytes
HDL	-	High-density lipoproteins
IFAT	-	The immunofluorescence antibody test
IHA	-	Indirect hemagglutination tests
K	-	Kinete
LDL	-	Low -density lipoproteins
MCHC	-	Mean corpuscular hemoglobin concentration
MCV	-	Mean corpuscular volume
OIE	-	World Organization for Animal Health

PCR	-	Polymerase Chain Reaction
PCV	-	Packed cell volume
RBC	-	Red blood cells
SG	-	Salivary glands
SK	-	Strahlenko reper
ST	-	Sporoblast
SZ	-	Sporozoites
T	-	Trophozoites
Z	-	Zygote
WBC	-	White blood cells
I.E.	-	In other words
H	-	Hour
HCT	-	Hematocrit
RDW	-	Red cell distribution width
PLT	-	Platelet
MPV	-	Mean platelet volume
PCT	-	Platecrit

INTRODUCTION

Babesiosis is a group of tick-borne diseases caused by several species of protozoa of the genus *Babesia*. These organisms are capable of infecting all species of domestic animals, and also found in some wild animals, which serve as reservoir of infection (Iosos, 1986). Sheep and goats are infected by *Babesia motasi* and *Babesia ovis*. Sheep babesiosis is of considerable economic importance in the areas which are infected with *Rhipicephalus bursa* (Radostis *etal.*.,2007). During infection with *Babesia*, the release of pharmacologically active substance and destruction of erythrocytes play a major role in the parasitemia of the disease. However, the proportion of each varies with the individual species of *Babesia* (Soulsby, 1982).

Developmental life cycle include merogony, gamogony and sporogony. During tick feeding the infection occurred by sporozoites which invade erythrocytes to then divide by binary fission to produce merozoites then to gametocytes which can initiate the infection in tick vector (Melhorn and Piekarski,2002). Diagnosis of the disease clinically by appearance of fever, malaise and restlessness, anorexia and anemia. Icterus hemoglobinuria and ascites may appear during late stages and progressive debility terminate to death (Smith *etal.*,1972; Soulsby,1982). Breathing is labored and rapid, the heart beat is fast and loud, nervous signs are hyperexcitability, moving the object, impaired vision and changing of urine to red color (Nyndo,1992). In ruminants ruminal movement ceased and abortion may occur (Urquhart *etal.*, 1996).

Babesiosis can be confirmed by performing Giemsa stained thin blood smears or Romanowsky – stained smear (Soulsby,1982; Urquhart *etal.*,1996) Serology includes enzyme – linked immunosorbent assay, complement fixation test (Salih *etal.*.,2005).

Molecular methods applied such as probes, polymerase chain reaction, reverse line hot hybridization and real time PCR (Mosqueda *etal.*,2012).

Currently antiprotozoal agents as deaminize accurate and imidocarb dipropionate were used (Enbiyale *etal.*, 2018). Main methods for prevention and controlling *Babesia* are immunization , chemoprophylaxis and vector control (Demessie and Derso ,2015) .

Piroplasmosis in the Sudan was early reported in the beginning of the past century. The disease was reported in 1902 and the research was done (Abdoun, 1984; Hashim, 1984).

Objectives

- 1- To demonstrate *Babesia spp* in sheep in shikan locality .
- 2- To evaluate some hematological and biochemical parameters of the disease in sheep.

CHAPTER ONE
LITERATURE REVIEW

CHAPTER ONE

LITERATURE REVIEW

1.1. Classification and characteristics of *Babesia spp*:

According to Soulsby (1982) *Babesia* species are classified :

Subclass : Piroplasmia

Order : Piroplasmidae

Family : *Babesiidae*

Genus : *Babesia*

Species : *B.motasi*

B.ovis

B.foliata

This family characterized by round to pyriform and amoeliod forms that occurring in erythrocytes. The vectors are ixodidae ticks.

1.2. Life cycle of *Babesia spp*:

The life cycles of the *Babesia spp* are very similar. All species of *Babesia* are naturally transmitted by the bite of infected ticks (almost all ixodids rather than argasids) and the main lifecycle difference amounts to the presence of transovarial transmission in some species (*Babesia spp.*) and not in others (*B. Microti*-like), during the tick bite, sporozoites are injected into the host and directly infect red blood cells .This phenomenon separates *Babesia spp* from *Theileria spp.*, where sporozoites do not readily infect red blood cells but initially penetrate a lymphocyte or macrophage in which development into schizonts takes place (Uilenberg, 2006). In the host, *Babesia* sporozoites develop into piroplasms inside the infected erythrocyte resulting in two or sometimes four daughter cells that leave the host cell to infect other erythrocytes until the host dies or the immunity of the host clears the parasites. The spleen with its lymphoreticular filter function is essential in resisting primary infections of *Babesia spp*. By specifically removing infected cells from circulation,

probably through a combination of spleen microcirculation and stimulated phagocytic cell activity (Devos *et al.*, 1987; Gray and Weiss, 2008). The life cycle of *Babesia spp* involves two different hosts: an Ixodidae tick, where sexual reproduction takes place, and a vertebrate, where the parasite exclusively experiences asexual reproduction within erythrocytes (Fig. 1).

Following uptake of intraerythrocytic parasites by an Ixodidae tick during a blood meal, parasites need to cross multiple cellular barriers in their migration through tick tissues and cavities, during which they undergo several—still only partially characterized—metamorphic changes (Kakoma and Mehlhorn 1994; Florin- Christensen and Schnittger *et al.*, 2003) Gametocytes, which are thought to be already present in the ingested blood, and this mature dimorphic elongated gametes—ray bodies—that fuse, yielding diploid zygotes (Ribeiro, 1998). Zygotes adhere to and invade midgut epithelial cells, and eventually transform into motile kinetes, which are released into the tick hemocoel and invade multiple tissues, including granular acini of salivary glands. Here, parasites replicate asexually by sporogony, forming sporozoite colonies. *Babesia* parasites undergo transovarial transmission, meaning that kinetes also invade tick ovaries and eggs, and infective sporozoites are formed in the salivary glands of the next-generation larvae as well. Sporozoites are injected into a suitable host together with tick saliva during feeding and invade erythrocytes. They then convert into hemoglobin-feeding trophozoites, which in turn transform into merozoites. Merozoites multiply by binary fission—merogony—and, eventually, they lyse host cells and invade new ones, repeating this asexual propagation cycle (Kakoma, Mehlhorn, 1994).

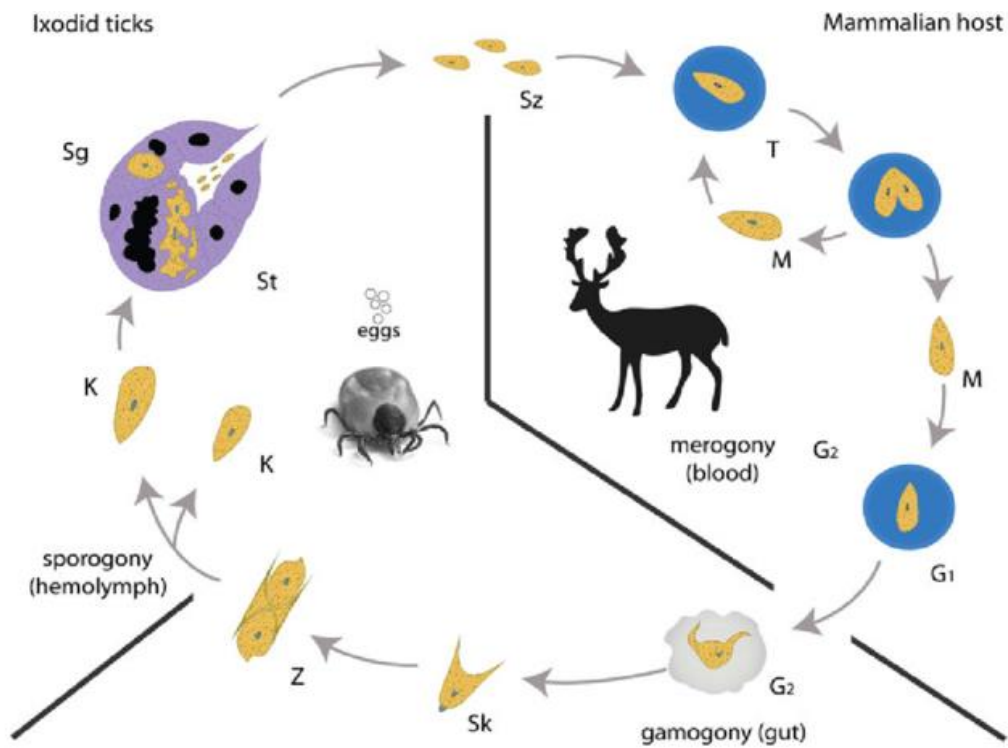


Fig.1. Simplified general life cycle of *Babesia* spp

https://www.researchgate.net/profile/Anke_Hildebrandt/publication/261839775/figure/fig1/AS:613919999664160@1523381342817/Simplified-general-life-cycle-of-Babesia-spp-modified-and-adapted-from-Hunfeld-etal.png

According to Mehlhorn and Piekarski (2002) *Babesia* life cycle consists of merogony, gamogony, and sporogony. Infection is acquired when sporozoites (Sz) are transferred during tick feeding. Sporozoites then invade erythrocytes and develop into trophozoites (T). Trophozoites divide by binary fission and produce merozoites (M) which continue infection and reinitiate the replicative cycle in the host.

Some trophozoites develop into gametocytes (G) which can initiate infection in the tick vector. In the tick gut gametocytes develop into “Strahlenkoörper” (Sk) which fuse to form a zygote (Z) developing into a kinete (K). Kinetes gain access to the hemolymph of the tick, replicate, and invade various organs.

Note that members of the *Babesia spp.* sensu stricto groups can infect the ovaries and be transmitted transovarially via the eggs (E), so that all stages (Larvae, nymphs and adults females) are potentially infective, whereas members of the *Babesia microti*-like groups are only transmitted from one stage to the next (transstadially), so that larvae are rarely if ever infected. Sporogony is initiated when kinetes invade the salivary glands (Sg). Here, the parasite forms a multinucleated sporoblast (St). Newly developed sporozoites (Sz) will then be injected into the host with tick saliva upon the next blood meal.

1.2.1. Life cycle in the vertebrate:

The cycle in the vertebrate, the transmitted sporozoites seem to infect the erythrocytes, except in the case of *Theileria* and some *Babesia* species, which invade *lymphocytes* first. These are differentiated further into Merozoites, which bud off from the schizont and lyse the cell. These merozoites or sporozoites (from *Babesia* species without a preerythrocytic stage) infect the host erythrocytes. The merozoite invades the host erythrocyte through a process of invagination (Rudzinska *et al.*, 1976). Forming a parasitophorous Vacuole. The vacuole membrane gradually disintegrates, and the parasite is left with the defining piroplasm feature of a Single membrane, in contrast to plasmodium species, which invade by a similar mechanism but retain the host membrane In addition to its own (Rudzinska *et al.*, 1976). Within the host erythrocytes, most merozoites become trophozoites and divide by binary fission; this asexual reproduction produces more merozoites, which lyse the cell and go on to infect additional erythrocytes. Four parasites can form at the same time, giving rise to a malarial cross form. Rapid reproduction destroys the Host cell and leads to hemoglobinuria in the host. Some trophozoites can, however, become potential gametocytes (Rudzinska *et al.*, 1976; Melhorn *et al.*, 1980). These trophozoites do not reproduce at this point but instead increase in size. Later, when they are in the Gut of the tick, these gametocytes will

develop into gametes prior to leaving the erythrocytes within the tick gut (Rudzinska *et al.*, 1976).

1.2.2. Life cycle in tick:

In the tick life cycle has been obtained from Studies with *B. Microti* (Telford, 1993). The organisms are first detectable in the tick about 10 h after the tick begins to feed on an infected vertebrate. After about 46 to 60 h of feeding, the Parasites are still detectable within the consumed erythrocytes, but some of them (the gametocytes) begin to develop new organelles most notable is the development of an arrowhead-shaped organelle at the anterior end of the organism called strahlenkörper (Koch, 1906) or ray bodies. Organisms containing this arrowhead structure within the tick host have been found in all infections with *Babesia* and *Theileria spp.* (Kakoma and Melhorn, 1993) that have been examined. These arrowhead forms are likely involved in the fusion of the gametes (Kakoma and Melhorn, 1993; Rudzinska *et al.*, 1983). The result zygote uses the arrowhead to enter the epithelial Cells of the tick gut approximately 80 h after the tick starts feeding. From the epithelial cells, the parasites move to the salivary acini via the hemolymph (Rudzinska *et al.*, 1983). Sporozoite development within the salivary gland can be divided into three stages. First, the parasite expands and fills the hypertrophied host cell, forming a multinucleate sporoblast which is a relatively undifferentiated, three-dimensional, branching meshwork from which the sporozoites will bud (Karakashian *et al.*, 1983). The second step starts only after the tick host begins feeding again, the specialized organelles of the future sporozoites (micronemes, rhoptries, and double membrane segments beneath the plasma membrane) develop within the meshwork. Finally, the mature sporozoites form through budding process. Mature sporozoites are approximately 2.2 to 0.8 mm in size and pyriform-shaped and contain a smooth endoplasmic reticulum, free

ribosomes, mitochondrion- like organelles, a single anterior rhoptry, and several Micronemes (Kakoma and Melhorn., 1993; Karakashian *etal.*, 1983). Approximately 5,000 to 10,000 sporozoites can be produced within a single sporoblast. It is estimated that several thousand sporozoites are deposited in the dermis around the tick's mouth during the final hours of attachment and feeding. This is smaller inoculum than the approximately 10,000 to 25,000 sporozoites needed to syringe-inoculate white-footed mice or hamsters (Piesman and Spielman, 1982). The efficiency of tick transmission is attributed to the tick saliva, which probably facilitates infection with its anti-inflammatory and/or immunosuppressive pharmacological activity (Ribeiro, 1987). "Large" *Babesia* species, like *B. Divergens*, can be transmitted transovarially. After the zygotes (also called ookinetes) have entered the hemolymph, they may invade other cells, such as Fat body cells or nephrocytes, and undergo a second cycle of division (Telford *etal.*, 1993). These secondary ookinetes can then invade the ovaries and be transmitted transovarially.

1.3. Ovine babesiosis:

1.3.1. Etiology:

Babesia infection is as result of infection with the protozoa of the genus *Babesia*, which belongs to the family *Babesidae*, and the order Piroplasmida. *Babesia spp* in sheep caused by three species namely *B.motasi* *B.carassa* *B.ovis* (Friedhoff, 1988). The parasite in sheep is transmitted by ticks of the genus *Haemaphysalis* (*H. punctata*, *H. otophila*), *Dermacenter* (*D. silvarum*), *Hyalomma spp* and *Rhipicephalus* (*Rhipicephalus bursa*) (Rehman *etal.* 2004). The ticks are sensitive to climatic condition and require a relative humidity of at least 80% to survive. Typical habitats of the ticks that transmit the infection include deciduous and coniferous woodland, heathland, moorland, rough pasture, forests and urban park (Gassner *etal.*, 2011).

1.3.2. Epidemiology:

The occurrence of the disease is dependent on the distribution of the ticks that transmit the disease. Many studies are available on associated risk factors of the protozoan infections in different countries. These countries include Iran (Bijan *etal.*, 2015), Kenya (Okuthe and Buyu, 2006), Greece (Theodoropoulos *etal.*, 2006), Rwanda (Bazarusanga *etal.*, 2007), Bolivia (Gonzales *etal.*, 2007), and Uganda (Magona *etal.*, 2008). Babesiosis is a haemoparasitic disease belonging to a complex of several tickborne diseases with different aetiological agents, such as protozoa, rickettsiae, and bacteria (Ranjbar-Bahadori *etal.*, 2011). Transmitted by ixodid ticks (Aktas *etal.*, 2007). The high lethality and morbidity caused by babesiosis explain its importance as a major constraint to livestock breeding development (Mehlhorn *etal.*, 1994; Ahmed *etal.*, 2002). Several species of *Babesia* (*Babesia ovis*, *Babesia motasi*, *Babesia crassa*, and *Babesia* spp) have been described in sheep; among them *B. ovis* and *B. motasi* are causative agents of sheep babesiosis (Uilenberg, 2001. Schnittger *etal.*, 2003., Liu *etal.*, 2007; Ranjbar-Bahadori *etal.*, 2011). *Babesia motasi* is moderately virulent, whereas *B. crassa* appears to have little or no pathogenicity (Hashemi-Fesharki, 1977). *Haemaphysalis punctata* is the vector of this species and is widespread in tropical Africa (Uilenberg *etal.*, 1980). The most important *Babesia* species infecting small ruminants is *B. ovis*, which has been reported in Europe, Africa, Asia, and the Far East (Ahmed *etal.*, 2006). *Babesia ovis* is highly pathogenic, especially in sheep; it causes severe infections characterized by fever, anemia, icterus, and haemoglobinuria. Mortality rates in susceptible hosts range from 30% to 50% in natural infections (Aktas *etal.*, 2005). *Babesia* infection is an emerging zoonotic disease that affects livestock and human with live-threatening implication, particularly in the immune compromised individuals. *Babesia* infection of sheep has been reported in several countries including Iran (Dekhordi *etal.*, 2010), China

(Guan *et al.*, 2012), Sudan (Osman, 1997) and Somalia (Ahmed *et al.*, 2013). *Babesia* infection of sheep causes losses in production of meat, milk, and other live-stock by-products with possibility of death in severe cases (Perez *et al.*, 2010). The infection causes severe economic losses to sheep farmers in tropical and sub-tropical regions.

The prevalence of *B. ovis* infection in Lohi sheep in Pakistan has been found to be 50% (Iqbal *et al.*, 2011). While a separate study reported a prevalence of 34% in infected sheep on detection by PCR (Shahzad *et al.*, 2013). In Nigeria, a prevalence of 7.5% has been reported for *B. ovis*- infection in sheep with prevalence's of 40% for Yankasa breed, 26.7% for Ouda breed and 33.3% for the Koraji breed (Biu *et al.*, 2009). An overall prevalence of between 45% and 95% has been reported in a study done in Machakos, Kenya. This prevalence was determined by using microscopy and ELISA to detect the presence of the *Babesia* (Wesonga *et al.*, 2010).

According to Mohammed Sayed Mohammed *et al.*, (2018) the prevalence of sheep babesiosis in Sudan were recorded in sennar state around three seasons in year (31.66%)

1.3.3. Pathogenesis:

The infective form of *Babesia* is known as sporozoites and is produced in the salivary gland of the tick vectors. The sporozoites are usually injected into the sheep by larvae or adult tick when feeding on the host. The parasite then attacks the host erythrocytes and then destroys them. This destruction leads to the release of hemoglobin in circulation (Alani *et al.*, 1988). The erythrocytic cycle continues until the animal dies or its immune system is overwhelmed. *Babesia* parasites may be present in the blood system in small numbers sometimes even for many years without causing the disease (Alani *et al.*, 1988). There are a number of changes in hematological and biochemical profiles linked to the destruction of erythrocytes by

Babesia following infection of sheep. As parasitemia advances, infected animals reveal a significant decrease in erythrocytes counts, packed cell volume (HCT) level, hemoglobin (HB)-concentration, mean corpuscular Volume (MCV), and mean corpuscular hemoglobin concentration (MCHC). The biochemical changes seen in this case include alterations of total serum protein, as well as changes in levels of albumin, urea, creatinine, triglyceride, and cholesterol, (Bijan *et al.*, 2012). The decrease in total proteins and albumin, and the increase in urea and creatinine might reflect the degree and severity of damage to the liver and Kidneys observed (Yeruham *et al.*, 1995). The pathological and histopathological findings in lambs which died of acute babesiosis were indicated of liver and kidney damage (Habela *et al.*, 1991) suggested that lesions observed in ovine babesiosis generally arise from vascular alterations such as vasodilation and blood stasis leading to anoxia of the tissues.

1.3.4. Diagnosis:

1.3.4.1. Clinical findings:

After infection with the parasite, sheep develop fever and Parasitemia within 2 to 4 days followed by clinical signs of the disease, which include anorexia, listlessness, anemia, moderate jaundice, and haemoglobinuria. In immunocompetent animals, hyperthermia, usually returns to normal level on the fourth day after the peak pyrexia. Thereafter, parasitemia may reduce to a low level or even zero during the course of the disease (Rahbari *et al.*, 2008). Most cases of *Babesia* infection are seen in adults and animals younger than nine months, which usually remain asymptomatic. The level of the parasitemia and the degree of anemia are not usually correlated. The decrease in HCT has been reported to range from 30 to 40%. In some studies, parasitized erythrocytes have not been observed to block capillaries in the brain and it has been postulated that the failure of the

cytoadherence to brain capillaries may contribute to the absence of nervous signs in acute babesiosis (Yeruham *etal.*, 1998).

Babesiosis is a disease complex caused by unicellular *Babesia* parasites, which are transmitted by ticks and invade and proliferate in the red blood cells of vertebrate hosts. They cause a febrile disease of domestic and wild animals characterized by extensive erythrocytic lysis leading to anemia, icterus and hemoglobinuria, which can be fatal. In contrast to *Theileria*, *Babesia* parasites do not have a pre-erythrocytic stage in the vertebrate host. *Babesia* parasites are among the most widely distributed blood parasites and have a considerable economic, veterinary and medical impact worldwide. (Hashemi-Fesharki ,1997).

1.3.4.2. Hematology:

In general the demonstration of *Babesia* in thin or thick smears made from peripheral blood and stained with Giemsa is confirmatory. The HB concentration decreased significantly in the affected ovine. These observations were similar with the findings of (Bhikane *etal.*,2005; Sulaiman *etal.*,2010; Zangana and Naqid ,2011) . In the affected goats the PCV was significantly lowered (Biu *etal.*,2012 ; Ijaz *etal.*,2013). Decreased, PCV and HB concentration may be associated with the intravascular hemolysis of erythrocyte, increased erythrophagocytosis by reticuloendothelial system and restricted erythropoietic activity of bone marrow (Lewis *etal.*, 1981).

Ibrahim *etal.* (2009) explained that breakdown of erythrocyte by *Babesia* organism stimulates the phagocytic cells such as lymphocyte to clean the toxic remnants of ruptured erythrocytes from the body as well as increased tissue demand of neutrophil that reduces its concentration in peripheral circulation. The deviation in these values might be due to degree of infection, tissue necrosis, hemolysis and other concurrent infections, a statistically significant decrease was recorded in RBC and HB (Benjamin, 2001). Microscopic detection is still the best and most

sustainable method for on-site diagnosis of acute babesiosis. Thin and thick blood films are made for examining the presence of piroplasms after staining with Giemsa's stain.

In the acute phase, diagnosis of ovine babesiosis infection is mainly based on the microscopic examination of Giemsa-stained blood smear and clinical symptoms (Aktas *et al.*, 2005). The technique of microscopy may not detect the parasite during subclinical infections because of low parasitemia experienced during this stage. Furthermore, examination of blood smear by microscopy may be influenced by the technical skills of the laboratory technician (Uilenberg *et al.*, 2001).

1.3.4.3. Biochemical findings:

According to Esmailnejad *et al.* (2012) elevation in BUN was due to kidney malfunctions in *Babesia* infection. The levels of creatinine were significantly higher in affected than non-infected sheep. Similarly the elevated level of creatinine was reported by Esmailnejad *et al.* (2012). The elevation in creatinine level might have resulted from kidney dysfunction in *Babesia* infection. Similar findings were reported by Sulaiman *et al.* (2010). This change may be attributed to the damage of liver (Jain, 1986). Significant decrease in the blood glucose level was observed similar findings were reported by Zulfiqar *et al.* (2012). The decrease in blood glucose concentration may be due to the utilization of glucose by parasites and damage to the liver in addition starvation may also reduce the blood glucose level (Zulfiqar *et al.*, 2012).

1.3.4.4. Serology:

Many serological diagnostic methods, including indirect fluorescent antibody tests (IFAT), fluorescent antibody tests (FAT), complement fixation tests (CFT), indirect hemagglutination tests (IHA), have been developed for the diagnosis of babesiosis (Liu and Zhanoxing, 1980; Wano and Znonouno, 1989; Liu *et al.*, 1993). DNA probes have also been developed (Wu and Xmiunoum, 1996). But have only been

used in the laboratory. Indirect hemagglutination, complement fixation tests and ELISA have been established for a number of *Babesia* species (Bakheit *etal.*, 2007). PCR-based molecular diagnostic tools have also been established for the detection and differentiation of parasites DNA in blood samples of affected animals (Criado-Fornelio, 2007). Molecular tools employing the detection of nucleic acids such as DNA have been used to detect the presence of *Babesia* species in sheep blood (Shayan *etal.*, 2008). For example, techniques such as PCR targeting specific genus of the parasite have been used to detect *Babesia* spp such as *B. ovis*, *B. crassa*, *B. motasi* (Almeria *etal.*, 2001). The specific types of PCR that have been used to detect the parasite in sheep blood include conventional PCR, nested PCR, and real-time PCR.

1.3.4.5. Necropsy findings:

The gross postmortem and microscopic lesions of ovine babesiosis have been reviewed (Morel, 1989). In hemolytic babesiosis, icterus is seen upon opening the carcass, by the color it gives to all the connective tissues, and internal and external mucosae. The bladder contains red urine. Splenomegaly is invariably present, with a dark red, mushy pulp due to degeneration of the hematopoietic centers. Prominent Malpighian corpuscles, due to hyperplasia of the reticular tissue, are observed in the middle of this pulp. The liver is enlarged and congested, with discolored patches on a brownish background. On section the lobule is seen to have a yellow center with a grey border. The bile is granular. In hypertrophic kidney the two zones, cortical and medullary, are not clearly distinguishable. In the case of pneumonia due to icterus, the lungs show hepatization and local congestion, with rust-red mucus, and sometimes small hemorrhages. Petechia may be present on the peritoneal and cardiac serosae. If icterus is slight, the muscles appear pale because of anemia and fever (Morel, 1989).

1.3.5. Differential Diagnosis:

There are several diseases and or conditions that could be confused with babesiosis because of the similarity of clinical signs. The blood-tinged urine may be taken as a tentative diagnosis but other causes leading to the appearance of 'coffee-colored' urine, like ovine hematuria and tumors of the urogenital system must be considered in differential diagnosis. Presence of tick/s on the carcass may aid in the diagnosis. Nervous signs may also occur in heart water (Cowdriosis) and Nagana (trypanosomiasis) (Nyindo, 1992). Acute hemolytic babesiosis can be confused with leptospirosis. In the latter case, the ocular mucosa is dark red, internal mucosae are hemorrhagic, and the general condition is more markedly affected (Morel, 1989).

Chronic babesiosis or long convalescence from babesiosis can be confused with anaplasmosis which is also tick-transmitted and hence occurs at the same time and in the same environment (Morel, 1989).

1.3.6. Treatment:

Delan (1988) reported that buparvaquone 2.5 mg/kg as single dose has spectrum of activity against *B. ovis* infection. Vidhya *etal.*(2011) treated a case of *babesia ovis* in an adult buck at Veterinary College Hospital, Bidar with diminazene aceturate 2.5 mg/kg b.wt., I/M single dose with supportive therapy. Ijaz *etal.*(2013) estimated the efficacy of various drugs in goat against babesiosis in Lahore and its peri-urban areas. The result showed that the efficacy of imidocarb dipropionate along with oxytetracycline, imidocarb dipropionate alone, diminazene aceturate along with oxytetracycline and diminazene aceturate alone in goats was 100%, 80%, 90% and 70% treated *Babesia* respectively. There are many drugs that have been used for the treatment of ovine babesiosis including quinuroium sulphate, acaprin, diminazene aceturate, (Berenil) and imidocarb dipropionate salt (Imizol) (Hashemi-Fesharki, 1977).

1.3.7. Prevention and Control:

Transmission and spread of infection of sheep with *Babesia* parasites is controlled mainly by using acaricides to control the tick-vectors and by practicing good pasture management (Ahlam *etal.*, 2014). The control of the piroplasmosis depends on effective quarantine to prevent the introduction of the vector tick by dipping or spraying animals at risk with recommended acaricide. In routine surgery, care should be taken to prevent accidental transfer of blood from one animal to another (e.g. castration, dehorning). Widespread use of tick vaccines may also have a significant influence on the incidence of infection in cattle (Taylor *etal.*, 2007)

CHAPTER TWO
MATERIALS AND METHODS

CHAPTER TWO

MATERIALS AND METHODS

2.1. Study area:

The study was carried out in North Kordufan state (Fig. 2) which lies between latitudes $11^{\circ} 15' - 16^{\circ} 45' N$ and longitudes $27^{\circ} 5' - 32^{\circ} 15' E$. It has an area of 185,302 km² and inhabited by nomads and pastoralists. The state is divided into administrative units called localities. Agriculture and livestock comprise about 70% of the economic activities. The state is covered by a wide range of vegetation and green grasses especially in the season of rainfall (Anonymous, 2008). However, soil types are about 55.0% sand or gouze, 20.0% gerdud, 15.0% alluvial land and 10.0% clay land. The annual rainfall is concentrated in a single relatively short summer season from June to September and the state enjoys an annual rainfall of up to 500 mm. The state falls in the grass-land and wood-land savannahs. It has abundant fodder and grazing areas in rainy seasons during which animals are trekked by pastoralists to the northern part of the state while during dry seasons animals are trekked to Bahar Al-Gazal River in South Sudan. In the state a mixture of farming systems are practiced including nomadic, sedentary and semi-sedentary animal production systems. Furthermore, Kordufan has an estimated livestock population of 24,665,761 animals (Anonymous, 2008; ILRI, 2009)

North Kordufan state is delimited by six states. They are northern state, Khartoum state, the White Nile state, south Kordofan state, West Kordufan state.

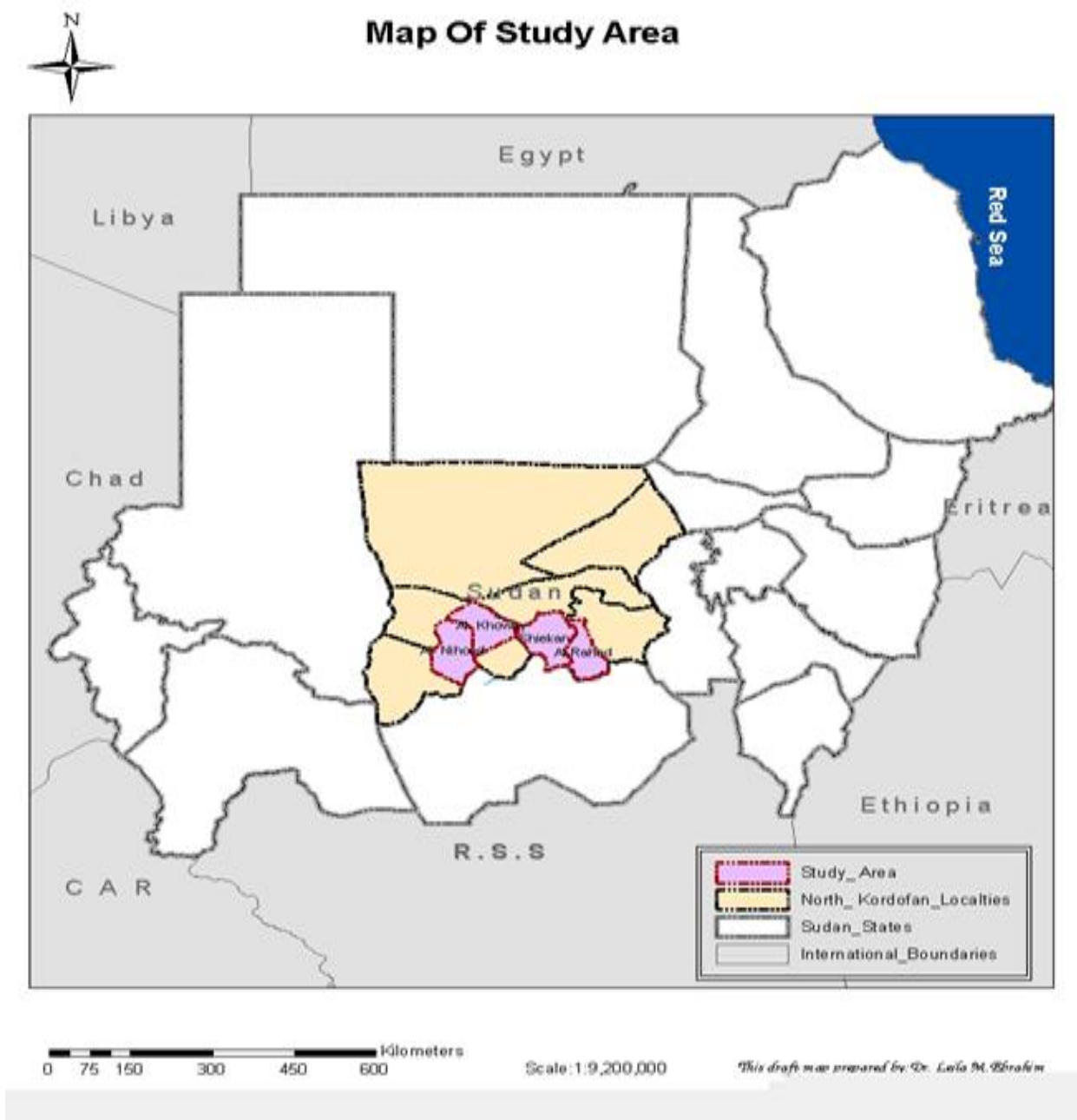


Fig. 2: Map of the Study Area Adopted from (Yassir Adam *et al.*, 2015).

2.2. Animals:

This study was conducted on March 2018 to March 2021 in Skikan Locality – North Kordufan State -Republic of Sudan .Whole blood sample were taken from 150 selected sheep in different herds located in west, east, north and south part of Shikan locality.

2.2.1. Clinical examination:

After visual inspection measured (Kelly,1984; Matijatko *etal.*,2007),clinical examination heart rate, respiratory rate and body temperature were also measured. Twenty sheep showed the clinical signs of fever, anemia, and occasional hemoglobinuria. Fifty -four sheep were clinically healthy were selected from the different herd as control animals.

2.2.2. Blood samples collection:

A total of 150 samples of whole Blood samples were taken from the jugular vein of all selected sheep. A volume of 3-10 mL blood was collected into newly labeled vacutainers containing EDTA- as anticoagulant for determination of hematological parameters and other tubes without EDTA for separation of serum samples for biochemical analysis.

2.3. Laboratory examination

2.3.1. Smear preparation for microscopic examination:

A total of 150 thin smear of the blood samples were made on newly labeled glass slide. The blood was taken from ear vein using sterilized needles. Care was taken to avoid any contamination of the samples. Then the dried blood smears were fixed with absolute methyl alcohol for one minute. To detect *Babesia*, Giemsa staining was done as described by Almeria *etal.* (2001). the stained smears examined by a microscope under oil immersion lens. Hundred Different microscopic fields were carefully examined per sample the photos of smears that revealed *Babesia*-infected erythrocytes were took directly by mobile camera and then saved for further documentation. The smears were recorded as negative for *Babesia* if no parasites were found in the oil-immersion field.

2.3.2. Hematological parameters:

Hematological parameters include hemoglobin concentration (HGBg/dl) , red blood cell counts (RBCs \times 10¹²) hematocrit (HCT %) red blood cell distribution

width (RDW-SDFL),platelet cell count (PLT $\times 10^9/L$) , mean platelet cell volume (MPV/FL) and palatocrit (PCT/M/L). All these parameters were determined immediately by Automated hematological analyzer (Mindary,3000) (Schalm *etal .*,1986; Shiono *etal .*,2003).

2.3.3. Biochemical parameters:

Biochemical examination of animal infected with *babesia* revealed that the following biochemical parameters albumin, globulin, urea, creatinine, cholesterol, and triglyceride were estimated by analysis of serum kits (Bio system kits 350).

2.4. Drugs:

Diminazene acetate was given at dose rate 2mg/kg b.wt., I/M single dose followed by supportive therapy given based on requirement of the individual case.

2.5. Statistical analysis:

Comparison of the haemato-biochemical parameters of infected sheep with healthy sheep was done by analyzing the data statistically. The results were analyzed by-way analysis of ANOVAs followed by pair –wise comparisons using the Duncan test. The computer software SPSS version 17.0 for windows was used for analysis.

CHAPTER THREE
RESULTS

CHAPTER THREE

RESULTS

THE RESULTS

The suspected sheep [96 animals] were examined clinically for heart rate, respiratory rate and body temperature as shown in table 1. But after microscopic examination of stained smears 20 animals (13.3%) were found infected by *Babesia ovis* (Table 2). And Table 3 showed that the hematological changes in hemoglobin concentration, red blood cell count and hematocrit, and these parameters were decreased in positive infected animals by *B. ovis* compared with Negative non-infected animals by the disease and control animals. But there was slight increase of red blood cell distribution width (19.1 ± 6.37) compared with control animals (18.3 ± 4.05). Platelet cell counts were decrease in infected animals (Table 3) compared with control animals, while the mean platelet cell volume was slightly increased (7.23 ± 1.94) compared with control animals (6.85 ± 1.38), whereas, plateletcrit was decreased (0.49 ± 0.53) in the infected animals compared with control animals (32.9 ± 119). There were lightly changes in mean corpuscular volume compared with control animals (Table 3), but the mean corpuscular hemoglobin concentration was decreased in sheep positive to *B. ovis*. As shown in table 4, results of biochemical tests of infected sheep indicated variable increase in total protein, urea, creatinine, globulin, cholesterol and triglyceride compared to healthy sheep, while albumin was decreased in infected animals.

Table 3.1: Estimation of clinical parameters of infected sheep (N=20) and non-infected sheep (N=76) for babesiosis in Skikan Locality–North Kordufan State.

PARAMETER	NONINFECTED INFECTED	INFECTED
HEART RATE	76.27±11.29	114.64±6.19
RESPIRATORY RATE	25.87±5.29	54.48±2.17
BODY TEMPERATUREC	39.17± 0.14	40.87±0.89

Table.3.2: Percentage of sheep babesiosis (N=20) in Shikan Locality – North Kordufan State.

Variable	Frequency	percent%
Control	54	36.0
Positive	20	13.3
Negative	76	50.7
Total	150	100.0

Table 3.3: Some hematological values of sheep babesiosis (N=150) in Shikan Locality – North Kordufan State.

Variable	Mean Std. Deviation Positive	Mean Std. Deviation Negative	Mean Std. Deviation Control
HGB g/DL	4.83±0.87	7.02±0.55	9.11 ±1.24
RBCs×10 ¹² /L	3.42±1.10	4.48±1.79	6.42±2.54
HCT%	13.2±5.77	15.4±4.55	21.0±6.55
MCV flu	33.4±2.50	35.2±3.70	33.8±3.78
MCHC g/do	42.1±15.2	49.3±15.3	48.5±21.0
RDW- SD Fl	19.1±6.37	17.3±5.69	18.3±4.05
PLT×10 ⁹ /L	346±203	475±317	531±390
MPV Fl	7.23±1.94	6.76±080	6.85±1.38
PCT M/L	0.49±053	52.1±182	32.9±119

Table 3.4: Biochemical values of sheep Bebeiosis (N=150) in Shikan Locality North Kordufan State.

Variable	Mean Std. Deviation Positive	Mean Std. Deviation Negative	Mean Std. Deviation Control
Total protein g/dl	8.97±0.80	5.93±0.92	7.45±0.51
Albumin g/dl	3.19±0.300	3.15±0.77	3.66±1.00
Globulin g/dl	5.40±1.0	3.16±0.97	3.72±1.03
Urea g/dl	34.5±9.26	25.0±9.92	25.1±8.96
Creatinine g/dl	1.14±0.41	0.72±0.24	0.83±0.25
Cholesterol g/dl	55.0±16.26	48.0±12.9	50.2±9.15
Triglyceride g/dl	59.9±18.99	57.3±7.83	58.1±12.7

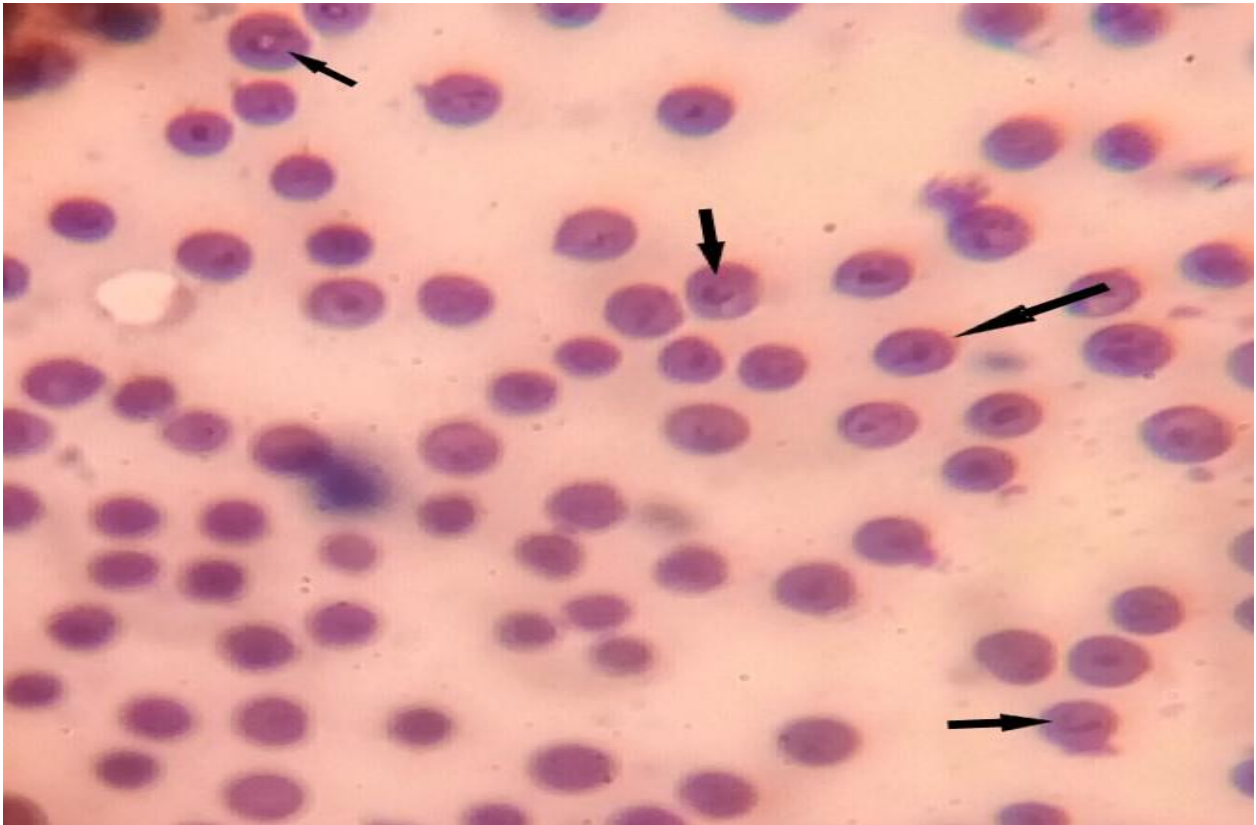


Fig.3 Blood smear from a naturally infected sheep with ovine babesiosis. In infected RBCs (Giemsa, 100 in shikan locality. The arrow heads in panels show erythrocytes infected with *Babesia* species. The smears were prepared from. Different blood samples.

CHAPTER FOUR
DISSCUSSION

CHAPTER FOUR

DISSCUSSION

According to the present study, different changes due to parasitemia were observed in the infected sheep. The observations were in accordance with the findings by Razmi *et al.*, (2003); Aktas and Altay (2007) and Sevine *et al.*, (2007). Decrease in red blood cells count, hematocrit, mean cell volume and hemoglobin concentration levels in infected sheep. These results were consistent with previous findings by Voyvoda *et al.*, (1997) and Hadadazadeh *et al.*, (2002). In addition decline in hemoglobin concentration and red blood cell count observed in other studies that was previously performed on clinicopathological changes induced by *B.equi* and *B. gibsoni* (Ambawat *et al.*, 1999; Trotta *et al.*, 2009). The present of anemia may be attributed to immunomediated phenomena by auto antibodies directed against component of membrane of infected and uninfected erythrocytes (Rubino *et al.*, 2006). Production of toxic hemolytic factors of the parasite due to (Rafaj *et al.*, 2007) . Mechanical damage by trophozoite causing intra erythrocytic binary fission (Zobba *et al.*, 2008) and erythrophagocytosis and through releasing vasoactive molecules such as kallikein (Soulsby, 1982; Brockus and Andresen, 2003). Concerning the erythrocyte indices, with parasitemia rates progression, a decrease was observed in level of mean cell volume and mean corpuscular hemoglobin concentration. As parasitemia increased, a depletion in mean cell volume and mean corpuscular hemoglobin concentration was evident that indicated microcytichypochromic anemia. The result was in accordance with reports of Uilenberg, (2006) Rubino *et al.*, (2006) and Zobba *et al.*, (2008) who recorded microcytic-hypohromic anemia in horse infected with *B.equi*. On the other hand polychromatophilic erythrocytes (Synonymous reticulocytes) in blood smears pointed out a hemolytic anemia. Reduction in mean cell volume level may be due

to decrease in hematocrit level and that attributed to the dilution of blood and subsequently mean cell volume. Also the most common abnormality of erythrocytes parameters is anisocytosis which was detected in infected animals and in reference to the value of mean cell volume which was below the normal values associate with spherocytosis (Zyner *et al.*, 2007). Polychromatophilic erythrocytes have a deficient component of hemoglobin concentration, therefore, the mean corpuscular hemoglobin concentration decreases in ovine/caprine babesiosis (Brockus and Andresen, 2003) as parasitemia of disease increased. The destruction of circulating red cells by auto antibodies is directed against infected and non-infected red cell membranes resulting in intravascular and extravascular haemolysis (Day, 1999; Irwin, 2005). However, Toboada and Lobetti (2005) proposed that direct parasitic damage contributes to anemia. Nevertheless, induction of serum hemolytic factors increased erythrophagocytic activity of macrophages and damage induced by secondary immune system after the formation of antierythrocyte membrane antibodies which prove the importance of the pathogenesis of anemia. Plateletcrit represents the percent of blood volume occupied by Platelets. It is well known that the surfaces of cells are essential for clotting reactions to take place (Khandekar *et al.*, 2006). Generally, Platelet indices could provide valuable information about the nature of thrombocytopenia, and that more attention should be paid to these indices in the diagnosis of thrombocytopenia. Despite the valuable information given by thrombocyte indices. *Babesia* initiates a mechanism of antibody – mediated cytotoxic destruction of circulating erythrocytes (Furlanello *et al.*, 1991). In this study there was elevation of total protein, urea, creatinine, and cholesterol and triglyceride level. The results are in consistent with findings reported by other researchers (Yeruham *et al.*, 1998; Rahbari *et al.*, 2008; Crongaj, 2010). It is known that renal involvement occurs in *B.ovis* infection (Habella *et al.*, 1991; Uilenberg, 2006) causing elevation in total protein, urea and creatinine level

and might have resulted from kidney dysfunction (Uilenberg, 2006). Muscle catabolism (Yeruham *etal.*, 1998) and colonization of *B. ovis* in the renal blood circulation (Habell *etal.*, 1991). It is suggested that in ovine babesiosis many potential factors leading to impairment of renal function e.g. acute diffuse proliferative glomerulitis, acute glomerular hemorrhage and acute tubular necrosis (Uilenberg, 2006; Habell *etal.*, 1991). Hypoalbuminemia in current study is in agreement with previous study (Elissalde *etal.*, 1983; Trotta *etal.*, 2009). Reduction of albumin level probably corresponds to disturbance in liver function, urinary loss of albumin associated with renal failure (Proteinuria) and anorexia in relation to high rise of body temperature. Also similar results have been reported previously (Irizarry-Rovira *etal.*, 2001; Diana *etal.*, 2007). Concerning the increase of total protein and globulins levels, these findings in all accordance to studies of Camacho *etal.*, (2005); Rubino and Cito, (2006); Trotta *etal.*, (2009). Generally the study of hyperproteinemia can be attributed to an increase in the globulin concentration in response to parasitic antigen and released hemoglobin from destructed erythrocytes. Elissalde *etal.*, (1983) and Camacho *etal.*, (2005) recorded the elevation in cholesterol and triglyceride concentration that expectable. The slight elevation of cholesterol and triglyceride concentration can be due to liver compensatory reaction to the loss of protein of adipose tissue metabolism during *Babesia* infection (Camacho *etal.*, 2005). In general, ovine babesiosis causes high morbidity rates among susceptible sheep, but the treatment by diminazene accurate (2mg/kg b.wt) followed by supportive therapy after accurate diagnosis giving successful results (Vidhya *etal.*, 2011; Mosqueda *etal.*, 2012). There was improvement effect after administration of diminazene aceturate (2mg /kg b.wt, I/M single dose with supportive treatment (multivitamins+ i.v.fluids)). These results were accordance to (Vidhya *etal.*,2011). Symptomatic therapy along with fluids was given based on requirement of the individual case. Visible improvement

efficacy of this drug was observed by negative blood smear examination of infected sheep. In conclusion sheep with babesiosis showed variable depletion in level of hemoglobin, red blood cells, hematocrit, platelets, plateletcrit and mean cell volume in infected sheep babesiosis. In blood chemistry, total protein, creatinine, globulin, cholesterol and triglyceride revealed variable increasement compared to control and negative sheep to babesiosis.

Conclusions and Recommendation's

Sheep infected by *babesia* showed variable depletion in level of hemoglobin, red blood cells, hematocrit, platelets, plateletcrit and mean cell volume .slight increase of red cell distribution width And mean platelet volume in infected sheep babesiosis were registered.

biochemical alterations revealed that an elevation in total protein , globulin ,Urea ,creatinine , Cholesterol and triglyceride. And the albumin was lowered. In conclusion evaluation of the results leads to the suggestion that severe anemia resulted from *babesia ovis* infection may cause kidney and liver Damage in sheep infected With babesiosis. Consequently, the investigation of kidney and liver elements would be a useful way to follow the prognosis of the disease, as well as to provide Supportive therapy to reduce Potential kidney And liver effects.

Recommendation's

1-from this study we recommend the prevention of sheep from the disease and finding a suitable way to eradicate ticks from *Babesia ovis* endemic area.

2-awareness of stock farmer about economic effect of *Babesia ovis* and a suitable way to minimize This high cost.

References

- Abdoun,A.M.O. (1984).*Studies on some aspect of equine piroplasmosis in Khartoum district Sudan* ,M.V.Sc thesis U of K.
- Ahlam, F; H.; Mervat, R.;Rabab, R.; Aziz, A. (2014). Toxic effect of *Babesia* infection in cattle and chemotherapeutic treatment in Egypt. *American Journal of infectious disease and microbiology*; 2(5):91-96.
- Ahmed A. H.; Abdalla M. I.; Rabab H. M.; Hussein H. A. (2013). Preliminary Assessment of Goat Piroplasmosis in Benadir Region, Somalia. DOI: 10.4236/ojvm.2013.36044.
- Ahmed, J.;Yin, H.; Schnittger L.; Jongejan, F. (2002). ‘Ticks and tickborne diseases in Asia with special emphasis on China’, *Parasitology Research* 88, 51–55.
- Ahmed, J.S.;Luo,J;Schnittger, L.; Seitzer,U.; Jongejan,F.; Yin, H. (2006). ‘Phylogenetic position of small-ruminant infecting piroplasmes’, *Annals of the New York Academy of Sciences* 1081, 498–504.
- Aktas ,M; Altay KJ ;Dumanli N. (2007) .Determination of prevalence and risk factor for infection with *Babesia ovis* in small ruminants from Turkey by polymerase chain reaction .*Parasitol Res* .100:797-802.
- Aktas M.; Altay, K.; and Dumanli, N. (2005). Development of a polymerase chain reaction method for Diagnosis of *Babesia ovis* infection in sheep and Goats. *Veterinary Parasitology*, 133, 277-281.
- Alani A.J, and .I.V. Herbert. (1988). the pathogenesis of *Babesia motasi* (Wales) infection in sheep. *Veterinary pathology*: 27(3-4):209-220.
- Almeria, S.; Castella, J.; Ferrier, D. (2001). Bovine Piroplasma in Minorca (Balearic Island, spain): a comparison of PCR based and light microscopy detection. *Veterinary parasitology*. 99(3):249-259.
- Ambawat,HK ; Malhorta;DV,Kumar,S, *etal* (1999) .Erythrocyte associated hemato-biochemical changes in *Babesia equi* infection experimentally produced in donkey .*Vet Parasitol*,85:319 -324.
- Anonymous (2008). Department of Statistic and Information. Annual Report. Ministry of Animal Resources and Fisheries, Khartoum, the Sudan.

Bakheit,MA; U Seitzer; PA Mbat;JS Ahmed. (2007). Serological Diagnostic Tools for the Major Tick-Borne Protozoan Diseases of Livestock. *Parassitologia*. 49(Suppl. 1): 53-62.

Bazarusanga, T.; Vercruysse, J.; Marcotty, T.; and Geysen D. (2007). Epidemiological studies on *Theileria parva* infections in Rwanda. *Veterinary Parasitology* 143, 246-221.

Benjamin, M.M. (2001). Outline of Veterinary Clinical Pathology, 3rd Edn., Kalyani Publishers, New Delhi, 351 p

Bhikane, A.U.;Ambore, B.N.; Narladkar, B.W.; Yadav, G.U. and Deshmukh, Y.T. (2005). Babesiosis in goat - a case report. *Intas Polivet*, 6(2): 215.

Bijan E.; Mousa T.;s and Siamak A-R. (2012). Investigation of hematological and biochemical parameters in small ruminants naturally infected with *Babesia ovis*. *Veterinary Research Forum*. 3(1): 31–36.PMCID: PMC4312816 .

Biu A A.; Gulani, I A.; Bulama, B. (2009). Prevalence of ovine *Babesia* infection in Maiduguri, Nigeria, *Sahel Journal of Veterinary Sciences*. Vol 8, No 1.

Biu, A.A.; Ibrahim, A.I.; Kumshe, H.A. and Ahmed, T.B. (2012). Prevalence of *Babesia ovis* in Maiduguri Metropolis, Nigeria. *Journal Science and Multidisciplinary Research*, 4: 84-88.

Brockus.Chw, and Andresen , CB.(2003). Clinical Pathology 4th ed Ames ,Iowa : Blackwell Publishing .pp.3-45.

Camacho,AT;Guitian,FJ;Palls,E, (2005) .Serum protein response and renal failure in canine *babesia annae* infection *vet res* . 36:713-722 *Vet Res*.

Criado-Fornelio A. (2007) A review of nucleic-acid-based diagnostic tests for *Babesia* and *Theileria*, with emphasis on bovine piroplasms. *Parassitologia*. 2007 49 Suppl 1:39-44.

Crongaj , M, Petlevski,R, Mrljak ,V, (2010) .Malondialdehyde level in scrum of dogs infected with *Babesia canis* *Vet Med* .55: 163-171.

Dehkordi Z.S.; Zakeri S; Nabian S;; Bahonar A.; Ghasem,i. F.; Noorollahi F. and Rahbari S. (2010). Molecular and bio-morphometrical identification of ovine *Babesia* infection in Iran. *Iranian Journal Parasitol.* ; 4:21–30.

Delan, T.T. (1988). Theileriosis in Eastern, Central and Southern Africa. In: Workshop on East Coast fever immunization, Lilongwe, Malawi 20-22 September, The International Laboratory for Research on Animal Diseases, pp 206.

Demssie,X.and Derso , S. (2015) . Tick borne hemoparasitic disease of ruminants; *Advance in Biological Research*, 9(4); 210-244 .

Devos, A.J.; Dalglish, R.J.; Callow, I.I. (1987). *Babesia*. In: Soulsby, e.j.l. (ed.), *immune Responses in parasitic infections*, vol. Iii. Protozoa. Crc press, boca raton, fl, pp. 183–222.

Diana ,A; Guglielmini , C.Candini, A. (2007). Cardiac arrhythmias associated with piroplasmosis in the horse: a case report. *Vet.J* .174:193-195.

Elissalde.GS;Wagner , GG; Criag , TM; *etal* (1983) . Hypercholesterolemia and hypocortisolemia in cute terminal *Babesia bovis* infection *Vet parasitol* .12:1-11.

Enbiyale, G.Debalke, D. Aman, E., edminm , B., Abebe, S. (2018). *Review on bovine babesiosis. Acta Parasitological Globalis*, 9(1); 15-26.

Esmailnejad, B.; Tavassoli, M.; and Asri-Rezaei, S. (2012). Investigation of hematological and biochemical parameters in small ruminants naturally infected with *Babesia ovis*. Urmia, Iran, *Veterinary Research Forum*, 3(1): 31-36.

Florin-Christensen M, Schnittger (2009) L.Piroplasmids and ticks: a long-lasting intimate relationship. *Front Biosci.*;14:3064–73 Florin-Christensen M, Schnittger L.Piroplasmids and ticks: a long-lasting intimate relationship.

Friedhoff K.T. (1988). Transmission of *Babesia*, in *Babesia infection of domestic animals and man*. Ristic M. (ed.), CRC Press, Boca Raton, Florida, 23-52.

Furlanella ,MA; Reina,D;Nieto,C.(1991) . Histopathological changes in sheep experimentally infected with *Babesia ovis* *Vet .;Parasitol* .;38:1-12.

Gassner, F; Van Vliet, AJ; Burgers, SL; Jacobs, F; Verbaarschot, P; Hovius, EK; Mulder, S; Verhulst, NO; Van Overbeek, LS; and Takken W. (2011). Geographic

and temporal variations in population dynamics of *Ixodes ricinus* and associated *Borrelia* infections in the Netherlands. *Vector Borne Zoonotic Disease*, 11(5):523-532.

Gonzales J. L.; Chacon, E.; Miranda M.; Loza A.; and Siles L. M. (2007). Bovine trypanosomosis in the Bolivian pantanal. *Veterinary Parasitology* 146, 9-16.

Gray, J.S., weiss, I.M., (2008). *Babesia microti*. In: Khan, n. (ed.), emerging protozoan pathogens. Taylor and francis, abingdon, uk, pp. 303–349.

Guan G, M.A. M; Liu A.; Ren Q.; Wang J.; Yang J, Li A.; Liu Z, Du P, Li Y, Liu Q, Zhu H .and Yin H, Luo J. (2012). A recently identified ovine *Babesia* in China: serology and sero-epidemiology. *Parasitol Int*, 61(4):532-7. Doi: 10.1016/j.parint.04.004.

Habella M.A.; Reina D.; Nieto C; *etal.* (1991). Histopathological changes in sheep experimentally infected with *Babesia ovis*. *Vet Parasitol.*;38:1–12.

Hadadazadeh ,H.;Khazraiiina, P.; Rahbari ,S.(2002) . Study on hematological change in experimentally infected lambs by *Babesia ovis* . J Fac , U of K.

Hashemi-Fesharki ,R. (1997). Tick-borne diseases of sheep and goats and their related vectors in Iran. *Parassitologia*. 39(2):115-7.

Hashemi-Fesharki, R. (1977). Studies on imidocarb dihydrochloride in experimental *Babesia ovis* infection in splenectomized lambs. *British Veterinary Journal* 133(6):609-14 PMID:608109.

Hashim, M, Abdella (1984). *Studies in Cale in Northern Sudan*. M.V.SC Thesis, U of K health and producon. 22:123-125.

Ibrahim, A.K.; EL Behairy, A.M.; Mahran, K.A. and Awad, W.S (2009). Clinical and laboratory diagnosis of piroplasmids in naturally infected cattle in Egypt. *Journal of Egyptian Veterinary Medical Association*, 69(2): 191- 203.

Ijaz, M.; Rehman, A.; Ali, M.M.; Umair, M.; Khalid, S.; Mehmood, K.; and Hanif, A. (2013). Clinico-epidemiology and therapeutical trials on babesiosis in Sheep and goats in Lahore, Pakistan. *The Journal of Animal & Plant Sciences*, 23(2): 666-669.

ILRI (2009). Constraints in the market chains for export of Sudanese sheep and sheep meat to the Middle East. *16th Research Report*. ILRI Publications Unit, Addis Ababa, Ethiopia, pp: 8-40.

Iqbal F.; Ali M.; Fatima M.; Shahnawaz S.; Zulifqar S.; Fatima R.; Shaikh R.S.; Shaikh AS, Aktas M.; Ali M.(2011).A Study on prevalence and determination of the risk factors of infection with *Babesia ovis* in small ruminants from southern Punjab (Pakistan) by PCR amplification. *Parasite*. 18:229–234.*Iran Journal Parasitol*. 5(4): 21 –30.PMCID: PMC3279853.

Irizarry –Rovira, AR; Stephens , J; Christian ,J. (2001) .*Babesia gibsoni* infection in a Dog from India .*Vet Clin Pathol* .30:180-188.

Irwin, P.J .(2005) . babesiosis and Cytauxzppnsis . Arthropode-Borne Infectious Diseases of Dogs and Cats .Manson publishing Ltd., Barcelone , Spain , 1st edition .

Jain, N.C. (1986). *Schalm''s Veterinary Haematology*. 4th Edn., Lea and Febiger, Philadelphia, pp 610-612.

Kakoma I; Mehlhorn H, (1994). *Babesia* of Domestic Animals, In: Krier JP, ed. *Parasitic Protozoa*. Volume 7. Academic Press Inc, 141-216 .

Kakoma I, and Mehlhorn H. (1993). *Babesia* of domestic animals. In: Kreier J P, editor. *Parasitic Protozoa*. 2nd ed. Vol. 7. San Diego, Calif: Academic Press;. pp. 141–216.

Karakashian S J; Rudzinska M A; Spielman A. (1983). Lewengrub S, Piesman J, Shoukrey N. Ultrastructural studies on sporogony of *Babesia microti* in salivary gland cells of the tick *Ixodes dammini*. *Cell Tissue Res*. ;231:275–287.

Kelly, W.R. (1984). *Veterinary clinical diagnosis*, 3th addition. Baillier Tindal. London .pp440.

Khandekar , M.M; A.S. Khorana, S.D . Deshmukh , A. I Kakrani ,A. D. Katdare , A. Khinamdar (2006) . Platelet volume indices with coronary artery disease and acute myocardial: an Indian scenario .*J clin . Pathol* 59,146- 149 . ied dogs.

Koch R. (1906). Kultivierungversuch der Hunde piroplasmen. *Z Hyg Infektionskr*. ;54:1–9. [[Google Scholar](#)].

Lewis, D.; Holman, M.R.; Purnell, R.E.; Young, E.R.; Herbert, I.V. and Bevan, W.J. (1981). Investigations on *Babesia motasi* isolated from Wales. *Research in Veterinary Science*, 31(2): 239-43.

Liu JUNHUA and ZHANOXING (1980). Complement fixation test for equine babesiosis. *Proceedings of the Second National Symposium on Veterinary Parasitology*, 305-307.

Liu ZHON~L~O, Zl.; OmmONG, MA LmUA.; and Y.o BAoma (1993). Study on babesiosis of buffalo in Hubei province of China. *Anaplasma and babesiosis Network Newsletter*, 2, 2.

Liu, A.H.; Yin, H.; Guan, G.Q.; Schnittger, L.; Liu, Z.J.; Ma, M.L. (2007). At least two genetically distinct large *Babesia* species infective to sheep and goats in China', *Veterinary Parasitology* 147, 246–251.

Losos, G.J. (1986). babesiosis , theileriosis, anaplasmosis and heart water in Infectious *Tropical Diseases of Domestic Animals* .pp. 3-831.

Day, (1999) "Antigen specificity in canine autoimmune hemolytic anemia," *Veterinary Immunology and Immunopathology*, vol. 69, pp. 215–224,. View at Google Scholar.

Magona, J W; Walubengo, J.; Olaho-Mukani, W.; Jonsson, N.N.; Welburn, S.; and Eisler, M.C. (2008). Clinical features associated with seroconversion to *Anaplasma marginale*, *Babesia bigemina* and *Theileria parva* infections in African cattle under natural tick challenge. *Veterinary Parasitology* 155,273-280.

Matijatko, V; V. Mrljak; I. Kis;N .Kucer and J. Forsek. (2007) . Evidence of an acute phase response in doge naturally infected with *Babesia canis* . *Vet Parasitol.*, 144:242-250.

Mehlhorn, H.; Schein, E. and Ahmed, J.S. (1994).*Theileria* ,in J.P.Kreier(ed.), *Parasitic Protozoa*, vol. 7, pp. 217–304, *Academic Press*, San Diego, CA.

Mehlhorn, H.; W. Peters; A. Haberkorn. (1980). the formation of kinetesAnd oocysts in *plasmodium gallinaceum* and considerations on

phylogenetic Relationships between *haemosporidia*, *piroplasmida*, and other *coccidia*. *Protistologica* 16:135–154.

Mehlhorn, H; piekarski , G.(2002). *Grundriss der parasitenkunde 6th revised edition* . Spectrum Akademischer Verlag GmbH, Heidelberg , Berlin, Germany pp.38-39.

Morel P, (1989). Tick-borne diseases of livestock in Africa. In: Mira Shah-Fischer, Ralph R. *Manual of Tropical Veterinary Parasitology*, 299-460.

Mosqueda, A., Olvera- Ramirez, G., Aguilar – Tipacamu, G, and G. J. Canto (2012). Current Advance in Detection and Treatment of babesiosis. *Current Medical Chemistry*, Pp. 1504-1518.

Mohammed S Mohamed, Sami M bukhari, Adem D Abakar , Adil EA Bala , Siddig E Idris ,(2018) Incidence and prevalence of tick –borne hemoparasites infecting sheep and goats in sennar state , Sudan, *international journal of biology Research* ,ISSN:2455-6548, Impact factor :RJIF 5.22 ,www.biologyjournal.in,volume 3;Issue 2; April 2018 page No .173-177.

Nyindo M, (1992). *Animal diseases due to protozoa and rickettsia*. Nairobi, Kenya: English Press, 67-77.

Okuthe, O.S., and Buyu, GE. (2006). Prevalence and incidence of tick-borne disease in smallholder farming system in the Western-Kenya highlands, [http://dx.doi.Org/10.1016/ Journal of Veterinary. Adv,3\(7\): 211-214](http://dx.doi.Org/10.1016/ Journal of Veterinary. Adv,3(7): 211-214).

Osman AM. (1997).Major tick-borne diseases of sheep and goats in the Sudan.*Parassitologia*39(2):143-4. *Review.PMID:9530699*.

Perez, de Leon, A.A; Strickman, D. P; Fish, D.; Thacker, E.; de la Fuente, J.; Krause, PJ.; Wikel, SK.; Miller, RS; Wagner, GG.; Almazan, C.; Hillman, R.; Messenger, M.T.; Ugstad, P.O.; Duhaime, 24 R.A.; Teel, P.D.; Ortega-Santos. A.; Hewitt, DG; Bowers, EJ; Bent, SJ; Cochran, MH; McElwain, TF; Scoles, GA; Suarez, CE; Davey, R; Howei, Freeman, JM; Lohmeyer, K; Li AY; Guerrero, FD; Kammlah, DM; Phillips, P. and pound JM. (2010). One Health approach to identify

research needs bovine and human babesioses: *Workshop report. Parasites and Vectors*; 3(1):36.

Piesman, j., and a. Spielman. (1982). *Babesia microti*: infectivity of parasites From ticks for hamsters and white-footed mice. *Exp. Parasitol.* 53:242–248.

Radostits, O.M., G. C. Gay; K.W. Hinchliffad, P.O. Constable, (2007). *Veterinary Medicine*, A text book of the diseases of cattle, sheep, goats, pigs and horses, 10th. Ed. London, Saunders Elsevier, pp. 1110- 1489, 1527-1530.

Rafaj, RB Mrljak , v; kucer, N.(2007) . Protein C activity in babesiosis of dogs. *Vetarhiv.*77:1-8.

Rahbari, S.; Nabian, S.; Khaki, Z.; Alidadi, N.; Ashrafihelan, J. (2008). Clinical, hematologic and pathologic aspects of experimental ovine *Babesia* infection in Iran. *Iran Journal Veterinary Research.*; 9(1):59-64. *Research of Veterinary Science*; 41(1):14-20.

Ranjbar-Bahadori, S.; Eckert, B., Omidian, Z.; Shirazi, N.S. and Shayan, P. (2011).*Babesia ovis* as the main causative agent of sheep babesiosis in Iran’, *Parasitology Research* 110, 1531–1536.

Razmi .GR; Naghibi a; Aslani MR.(2003). An epidemiological study on *Babesia* infection in small ruminants in Mashhad suburb , Khorasan province , *iran small Rumin Res.*50:39-44.

Rehman, W.U.; Khan I.A.; Qureshi A.H. ; Hussain S. (2004). Prevalence of different species of Ixodidae (hard ticks) in Rawalpindi and Islamabad. *Pakistan Journal of Medical Science*, 43, 42-46.

Ribeiro J M. (1987).Role of saliva in blood-feeding by arthropods. *Annu Rev Entomol.* ;32:463–478.

Ribeiro MFB., (1998). Patarroyo JHS.Ultrastructure of *Babesia bigemina* gametes obtained in “in vitro” erythrocyte cultures. *Vet Parasitol.*;76:19–25.

Rubino .G; Cito , Lacinio, .(2006) . Hematology and some blood chemical parametrs as a function of Tick –Borne Disease (TBD) signs in Hors. *J Equine . Vet . Sci* 26:475-480 .

Rudzinska M A; Lewengrub S; Spielman A; Piesman J.(1983). Invasion of *Babesia microti* into epithelial cells of the tick gut. *J Protozool.* ;30:338–346.

Rudzinska M.A.;Trager W.; Lewengrub S.J.;Gubert E.(1976) An electron microscopic study of *Babesia microti* invading erythrocytes. *Cell Tissue Res.* 1976;169:323–334.

Salih, D. A., A. M. El-Hussein and L. D. Singla (2005). Diagnostic approaches for Tickborne hemo-parasitic diseases in livestock. *J. Vet. Med. Anim. Health*, 7 (2): 45, 56.

Schalm , OW; Jain NV; Carrol ; EJ .(1986) . *Veterinary Hematology*. 3rd ed. Philadelphia : Lea and febiger. 20-86.19.

Schnittger, L.; Yin, H.; Gubbels, M.J.; Beyer, D.; Niemann, S.; Jongejan, F. (2003).Phylogeny of sheep and goat *Theileria* and *Babesia* parasites’, *Parasitology Research* 91, 398–406.

Sevine F; Turgut K; Sevine M, (2007). Therapeutic and prophylactic efficacy of imidocrab dipropionate on experimental *Babesia ovis* infection of lambs. *Vet parasitoal* . 149:64-71.

Shahzad W.; Haider N.; Mansur-U-D A.; Rashid M.; Muhammad S. S.; Muhammad H. M.; Nisar A.; Ghulam A. and Fayyaz M. (2013). Prevalence and Molecular Diagnosis of *Babesia ovis* and *Theileria ovis* in Lohi Sheep at Livestock Experiment Station (LES), Bahadurnagar, Okara, Pakistan. *Iran Journal of Parasitol* . 8(4): 570–578.PMCID: PMC4266121.

Shayan P.; Hooshmand, E.; Nabian, S.; Rahbari S. (2008). Biometrical and genetically characterization of large *Babesia ovis* in Iran. *Journal of Parasitol Research*; 103:217-221.

Shiono,H.;Yagi,Y.;Chikayama, Y.(2003) . Oxidative damage and phosphatidylserine expression of red blood cells in cattle experimentally infected with *Theileria sergenti* . *parasitol Res.*37:1181-1189.

Smith HA; Jones. TC; Hunt. RD.(1972) .*Veterinary Pathology*, Edition 4. Philadelphia USA; Lea and Febiger 723-726.

Soulsby ,E.J.L.(1982) . *Helminthes, Arthropods and protozoa of Domestic animals* .Seventh Edition .Bailliere Tindol, London,pp.706-707.

Sulaiman, E.G.; Arslan, S.H.; Al-Obaidi, Q.T. and Daham, E. (2010). Clinical, haematological and biochemical studies of babesiosis in native goats in Mosul. *Iraqi Journal of Veterinary Science*, 24(1): 31-35.

Taboada and R. Lobetti (2005) . " babesiosis" in infection Diseases of the Dog and Cat C.G.Greena, *Ed Elsevier 3rd edition* .

Taylor M.; Coop R. and Wall R. (2007). *Veterinary Parasitology. 3rd ed.* Blackwell Publishing, USA, pp.103-115

Telford S R. III; Gorenflot A; Brasseur P; Spielman A. (1993) *Babesia* infections in humans and wildlife. In: Kreier J P, editor. Parasitic protozoa. 2nd ed. Vol. 5. San Diego, Calif: Academic Press;. pp. 1–47.

Theodoropoulos, G.; Gazouli, M.; Ikonomopoulos,J.A.; Kantzooura, V; Kominakis, A. (2006). Determination of prevalence and risk factors of infection with *Babesia* in small ruminants from Greece by polymerase chain reaction amplification. *Veterinary Parasitology*, 135,104.

Trotta .M;Carli E; Novari , G,*etal* .(2009) . Clinic pathological findings, molecular detection and characterization of *Babesia gibsoni* infection in a sick dog from Italy . *Vet parasitol* .165:318-322.

Uilenberg G. (2001). Babesiosis. *Encyclopedia of arthropod-transmitted infection of man and domesticated animals* Wallingford M.W.(ed), CABI publishing, UK, 122-144.

Uilenberg, G. (2006). *Babesia* –a historical over view. *Vet Parasitol* .138:3-10.

Uilenberg, G.; Rombach, M.C.; Perié, N.M. and Zwart, D. (1980). Blood parasites of sheep in the Netherlands. II. *Babesia motasi* (Sporozoa, Babesiidae), *Veterinary Quarterly* 2, 3–14.

Urquhart , G; Armour J ; Duncan , JL. Dunan , AM; Jennings FW.(1996) *Veterinary Parasitology* Second edition . Oxford UK .Baclwell Science Ltd.,pp. 307.

Vidhya , M.K; Puttakshamma .G.C ;Halmandg S.C; Kasaralikal, V,R;Bhojar R.and patil.N.A.(2011) .*Babesia ovis* infection in a goat -A case report in proceeding of the 29th ISVM convention and national Symposium on "Recent Development in Diagnostics and therapeutic including application of nanotechnology in Veterinary Medicine .pp5.

Voyvoda , H; Selcun S; Kaya,A; *etal* (1997) . Modification in serum iron and copper concentration, total and latent iron binding capacity (TIBC-LIBC) and transferrin saturation (TS) in natural *Babesia ovis* infection in sheep Turk Veterinerlik .; 21;31-37.

WANO CHANGJIANG and ZnONOUNO (1989). Studies on diagnosis of babesiosis in buffalo with indirect fluorescent antibody test, *Journal of Huanzhong Agricultural University*, 4, 373-379.

Wesonga. F. D.; Kitala.P.M.; Gathuma.J. M.; Njenga. M.J.; and Ngumi.P.N. (2010). an assessment of tick-borne diseases constraints to livestock production in a smallholder livestock production system in Machakos District, Kenya, *Livestock* .

Wu JtANSA and XM MINGQUAN (1996). Development and application of DNA probe for *Babesia bovis*. *Acta of Parasitology and Acarology*, 3, 2.

Yeruham, I.; Hadani, A.; and Galker, F. (1998). Some epizootiological and clinical aspects of ovine babesiosis caused by *Babesia ovis*--a review. *Veterinary Parasitology*, 74(2-4): 153-63.

Yeruham, I.; Hadani, A.; Galker, F.; Avidar, Y. and Bogin, E. (1998). Clinical, clinicopathological and serological studies of *Babesia ovis* in experimentally infected sheep. *Journal of Veterinary Medicine Series B*, 45(7): 385- 94.

Yeruham, I.;Hadani, A.; Galker, F.; Rosen, S. (1995). A study of an enzootic focus of sheep babesiosis *babesia ovis*, babes, 1892.. *Vet. Parasitol.* 60, 349–354.

Yeruham. I.; Hadanu A; Galker, F; Avidar, Y; and Bogin, E. (1998). Effect of passage of *Babesia ovis* in the gerbil (*Acomys cahirinus*) on the course of infection in splenectomized lamb. *Veterinary Parasitol.* 65:157- 161.

Yassir .A .S .; Halima M. O .; Mohamed .O .H. ; Mohamed .A. B .; Rihab .A.O .; Ayman .A.; Siham .E. S .; Mohamed .A .A .; Ahmed .A. I. (2015) . Seroprevalence of *Babesia bigemina* antibodies in cattle in North Kordofan state, the Sudan. *ARC Journal of Animal and Veterinary Sciences (AJAVS)* Volume 1, Issue 2, October - December 2015, PP 1-11 www.arcjournals.org.

Zangana, I.K., and Naqid, I.A. (2011). Prevalence of piroplasmiasis (*Theileriosis* and *Babesiosis*) among goats in Duhok Governorate Al-Anbar. *Journal of Veterinary Science*, 4(2): 50-57.

Zobba , R; Ardu , Miccolini , S;*etal*(2008) .Clinical and laboratory Finding In equine pirolasmosis . *JEquine Vet Sci* 28:301-308 .

Zulfiqar, S.; Sadia, S.; and Furhan, I. (2012). Detection of *Babesia bovis* in blood samples and its effect on the haematological and serum biochemical profile in large ruminants from Southern Punjab. *Asian Pacific Journal of Tropical Biomedicine*, 2(2): 104-108.

Zyner , w; Cojska, Q; Rapacka , G; *etal* .(2007) . Hematological changes during the course of canine babesiosis by large *Babesia* in domestic dogs warsa (Poland) *Vet parastol* .145:146-151.

https://www.google.com/url?sa=i&url=https%3A%2F%2Fwww.researchgate.net%2Fpublication%2F283301222_Seroprevalence_of_Babesia_bigemina_antibodies_in_cattle_in_North_Kordofan_state_the_Sudan&psig=AOvVaw3nlPaKKVX5seNcQlFJRyZ2&ust=1619857326827000&source=images&cd=vfe&ved=0CAMQjB1qFwoTCNIt4PnEpfACFQAAAAAdAAAAABAD.

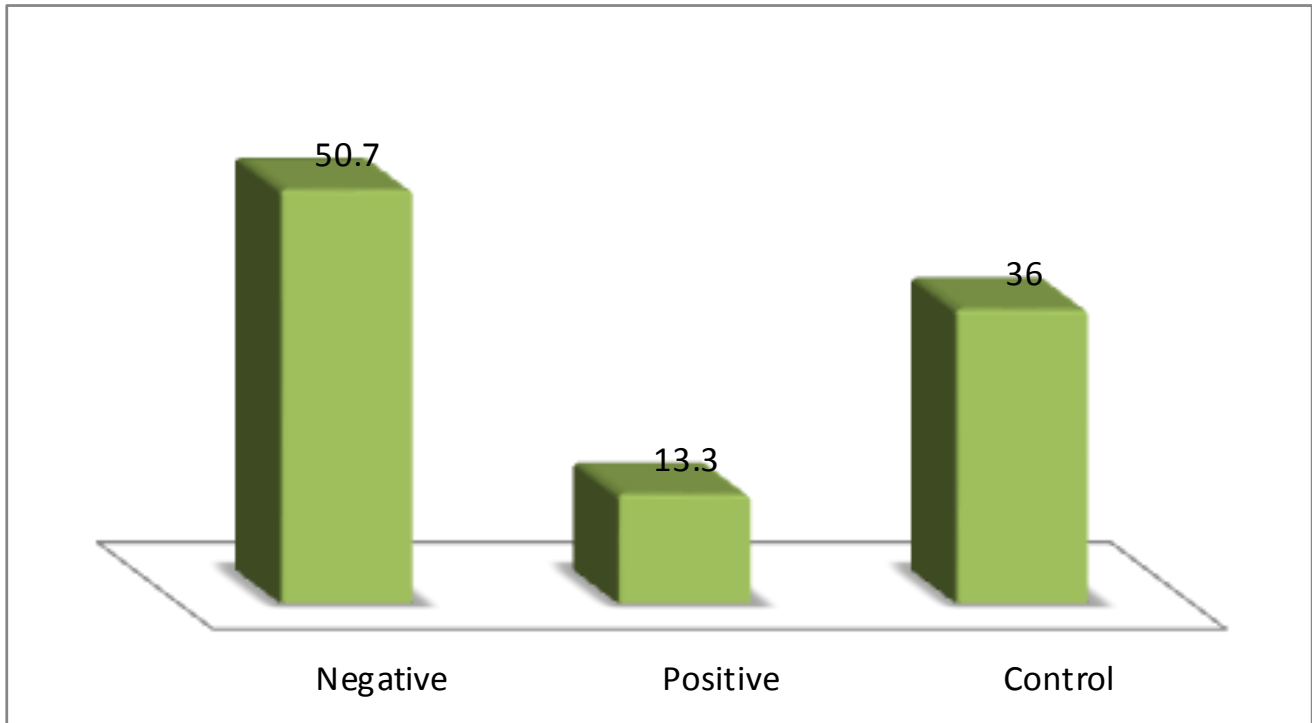
APPENDICES

Appendix.1. Percentage of sheep babesiosis (N=20) in Shikan Locality – North Kordufan State.

Appendix.2. some hematological value of sheep babesiosis (N=150)in Shikan Locality – North Kordufan State.

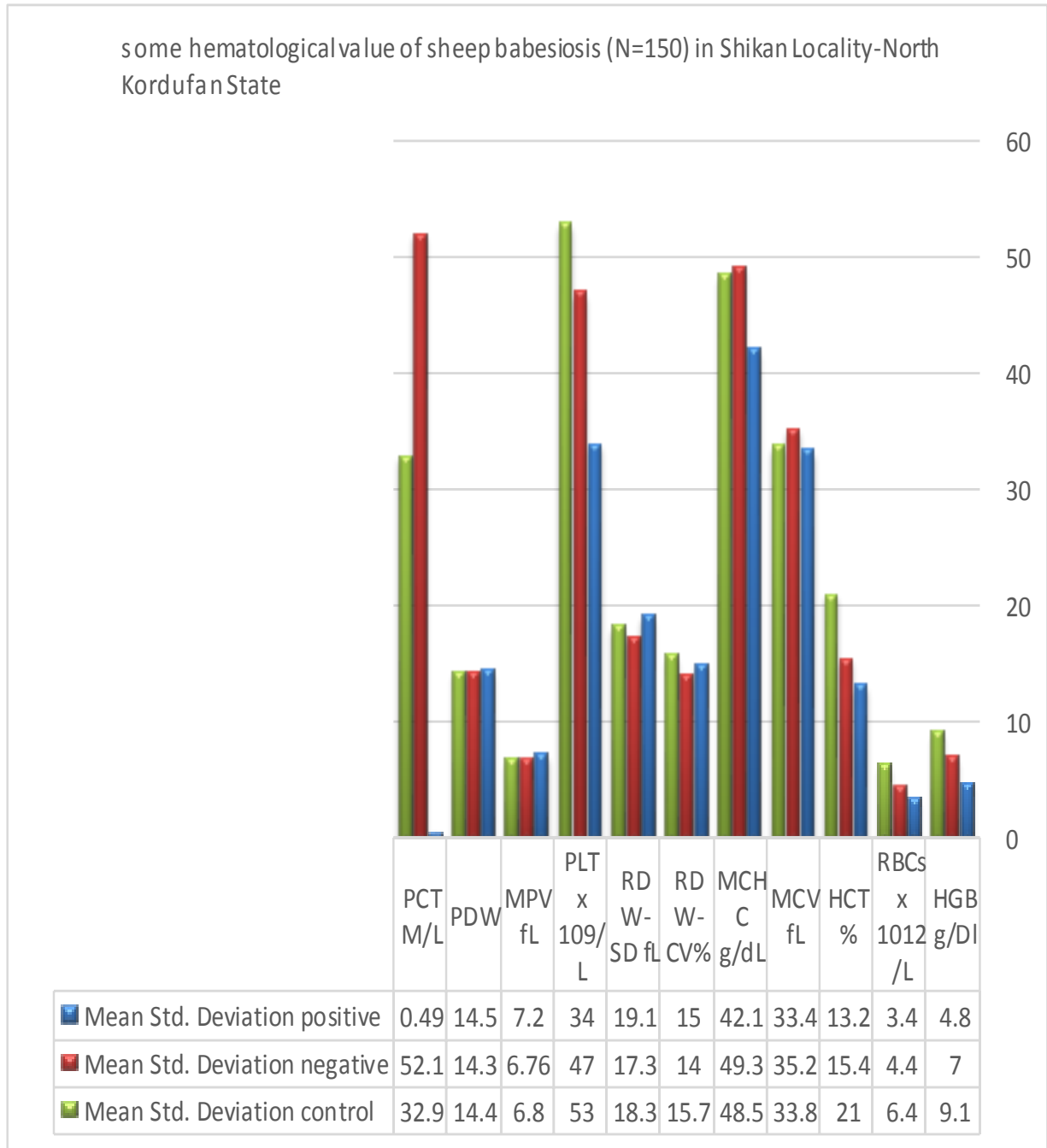
Appendix.3. biochemical value of sheep babesiosis (N=150) in Shikan Locality – North Kordufan State.

Appendix 1



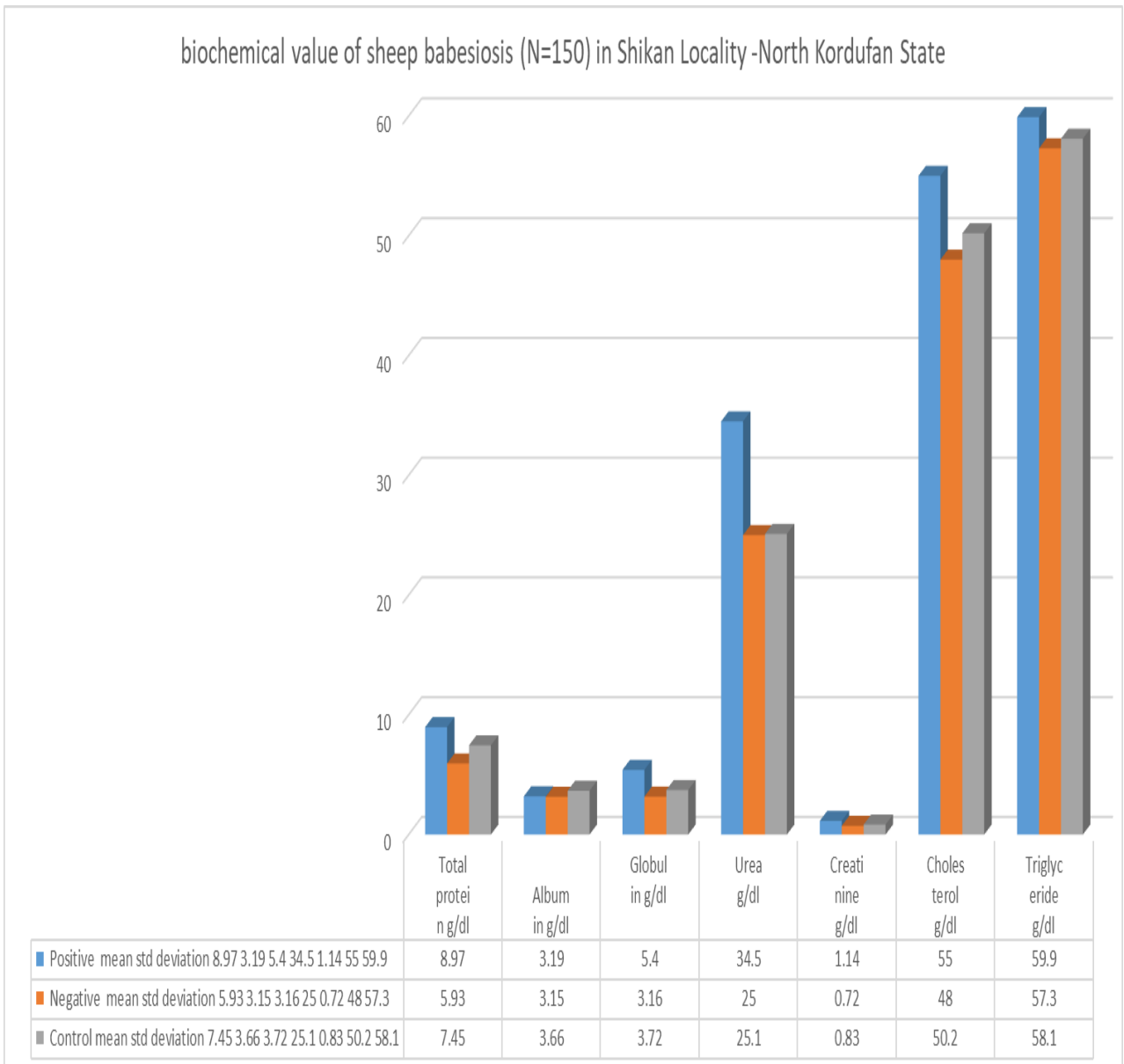
Percentage of sheep babesiosis (N=20) in Shikan Locality – North Kordufan State.

Appendix 2



Some hematological values of sheep babesiosis (N=150) in Shikan Locality – North Kordufan State.

Appendix 3



Biochemical values of sheep babesiosis (N=150) in Shikan Locality – North Kordufan State.