Department of Pathology and Clinical Pathology

Clinicopathological studies on camels (*Camelus dromedaris*) infected with some blood parasites at Aswan Governorate

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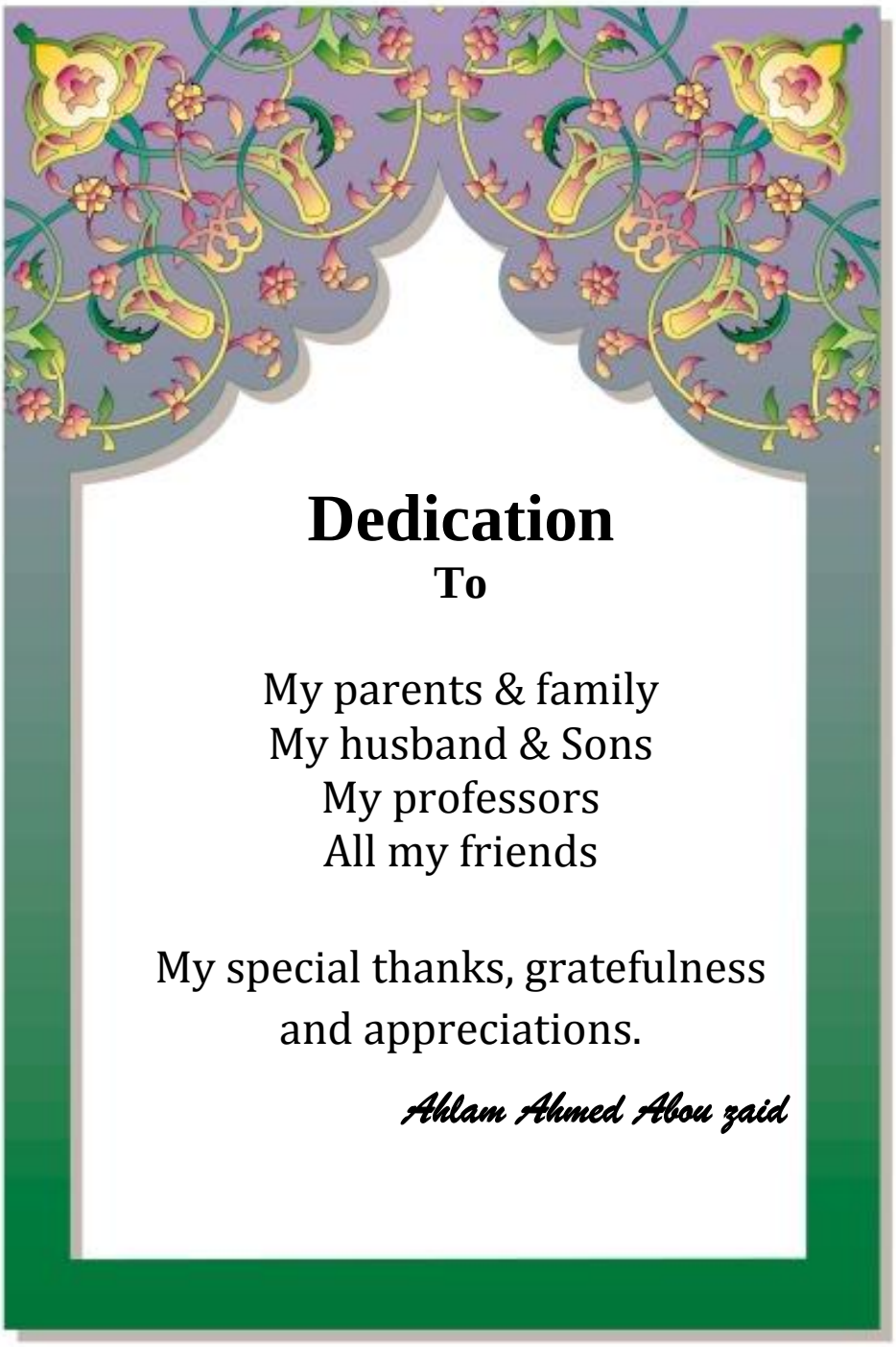

Dedication

To

My parents & family
My husband & Sons
My professors
All my friends

My special thanks, gratefulness
and appreciations.

Ahlam Ahmed Abou zaid

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LIST OF ABBREVIATIONS

A.	Anaplasma
A/G	Albumin/Globulin ratio
ALP	Alkaline phosphatase
ALT	Alanine aminotransferase
ANOVA	One-way analysis of variance
AST	Aspartate aminotransferase
B.	Babesia
BUN	blood urea nitrogen
EDTA	Ethylene Diamine Tetra-acidic Acid
H&E	Hematoxylin and eosin stain
Hb.	Hemoglobin
HCt.	Hematocrit
IFAT	Indirect fluorescence antibody test
LDH	Lactate dehydrogenate

MCH	Mean corpuscular hemoglobin
MCHC	Mean corpuscular hemoglobin concentration
MCV	Mean corpuscular volume
PCV	Packed cell volume
PLT	Platelets
RBCs	Red blood cells
rpm	Revolution per minute
SPSS	Statistical Package for Social Sciences
TEC	Total erythrocytic count
Th.	Theileria
TLC	Total leucocytic count
WBCs	White blood cell
GSBS	Giemsa stain blood smear
GSBF	Giemsa stain blood film
Spp.	Species

INTRODUCTION

Camel is a very hardy animal and well adapted anatomically as well as physiologically to harsh climatic conditions of desert, it suffers from various endo and ecto-parasitic diseases. These diseases cause economic losses in terms of decrease in working capacity, growth and productivity **(Parsani et al., 2008).**

Ticks are important vectors of pathogens of both humans and animals and transmit a broad range of bacterial, protozoal, rickettsia and viral pathogens **(Sonenshine 1991; Parola and Raoult 2001; Orkun et al., 2014).** In addition to acting as vectors, ticks also affect the well-being of their host directly through irritating bites, blood loss, damage to the skin and anorexia leading to reduced growth **(El Imam et al., 2015 ; Jabbar et al ., 2015)**.

Theileria, Anaplasma and Babesia are tick-borne blood parasites which globally impact on animal health and economic ***(*Imelda et al., 2015*).*** Theileriosis is an important hemoparasitic disease of animals inducing a variety of clinical manifestations ranging from a subclinical presentation to a fatal disease depending, on the animal species, host, age and the species of the microorganism. Tropical theileriosis caused by species of the genus *Theileria* has a wider distribution extending from North Africa to China **(Mukhebi et al., 1992)**. Anaplasmosis is an arthropod borne disease of ruminants caused by species of the genus Anaplasma (Rickettsiales: Anaplasmataceae) **(Kocan etal., 2004)** . *A. marginale* is the most virulent, characterized by a progressive hemolytic anemia, and is responsible for extensive economic losses in tropical and subtropical areas **(Wernery and Kaaden 2002).** A. marginale can be

distinguished from A. centrale by the location and the characteristics of the inclusion bodies in the erythrocytes **(Maghaddar, 2002; Al-Khaledi, 2008)**. Piroplasmids belonging to the genera *Babesia* are suspected of infecting dromedaries **(Egbe-Nwiyi et al., 1994)**, the significant effect of *Babesia* infections are reported in domestic animals, humans, and some wildlife species **(Uilenberg, 2006)**.

Blood is the mirror of the beings health, it is an index for several metabolic processes of the body and reflect the status of the functioning organs of the body and health, hence production of the animal. On the other hand, the blood hemogram and its biochemical constituents are considered important information in relation to the health status of the camel **(Maha 2015).** From the hematological aspect, blood parasites in camels caused some hematological alterations including decrease in packed cell volume (PCV), Hemoglobin concentration (Hb), Red Blood Cell Count (RBC) and Platelet (PLT) values and increase in Total Leucocytic Count (TLC) values. On the other hand, biochemically revealed an elevation in levels of Amino Alanine Transferees Enzyme (ALT), Amino Aspartate Transferees Enzyme (AST), while decrease in total protein and albumin values **(Azeem et al., 2019)**. Histopathology, there was destructive damage previously detected in many organs associated to blood parasites infection **(Coskun et al., 2012; Lima et al., 2019)**. Blood parasites attack the camel's health inducing anemia, wasting and death in heavy infection **(Mahran., 2004).**

There is paucity of information on haemoparasites of camels and their significance on health and productivity in Egypt. This study was undertaken to determine the prevalence of haemoparasites of camels slaughtered in Daraw and Aswan slaughter houses, belonging to Aswan Governorate, Egypt, and evaluation the effect of these blood parasite

infections of camels *(camelus dromedaris*) on biochemical and hematological parameters, in addition to histopathological examination.

REVIEW OF LITERATURE

Camel *(Camelus dromedaries)* is an important component of the desert ecosystem **(FAO, 2001)**. The dromedary camel plays an important role in the economy, especially in the culture of Arabian countries. Apart from being adapted to the harsh environment, these pseudo-ruminants, popularly known as "ship of the deserts" are multipurpose animals used for milk and meat production, hair/felt, racing, transportation and tourism **(Faye, 2014; Faye, 2015)**. Camel production is severely affected by various diseases, especially in the absence of adequate veterinary services. Many endo- and ectoparasites affect their health, productivity and performance **(Megersa, 2010).**

Theileriosis

Theileria camelensis and T. dromedarii are considered the two common Theileria spp. that has been reported in camels **(El- Metenawy, 2000)**. It is transmitted by the tick species called Hyaloma dromedarii, where camel is main host, but it is also detected on the skin of cattle, sheep goats, and horses **(Hamidinejat et al., 2008)**.

Glass et al., (2003) showed that Camels that immediately being recovered from the primary infection with Theileria annulata have become a persistent asymptomatic carrier state for many years, with a low parasitemia. The carrier plays an important role in the maintenance of parasite's life cycle.

Salem and El Olemy (2017) recorded that a total of seventy seven apparently healthy dromedary camels (Camelus dromedarius), of both sexes, introduced to El-Bassateen slaughterhouse in Cairo, Egypt , revealed that microscopic and molecular prevalence of T. annulata were 32.47% and 40.26 %,

respectively. Stained blood smears of infected camels showed the intra-erythrocytic piroplasms and the intra-lymphocytic schizogonies, with a parasitemia rate of 2.4%. Also, **Ibrahim et al., (2017)** showed about 75% of in the investigated camels were found to be positive for piroplasmosis. However, no pathognomic clinical signs of piroplasmosis. This means that we face problematic to cure these infected animals as they become carriers of the parasite and serve as reservoirs for transmission **(Abou El-Naga and Barghash 2016).**

Morphology:

Theileria spp. presented inside R.B.Cs possesses two forms the first one called Erythrocyte form which occur in blood inside R.B.Cs and its take several shapes such as ring, comma, rod and oval shape, while the second called lymphocyte form which present in lymph node and called a koch‘s blue bodies represented the schizont of parasite and appear in two form, macroschizont which consist of 8-12 nuclei and microschizont which consist of 50-100 nuclei **(Telford et al., 2002; Shaw 2003; Mans et al., 2015).** Insufficient information about the microschizont stage of this parasite detected in camel; since it has been reported only its piroplasm stage in erythrocytes **(Elghali and Hassan, 2010).**

Life cycle:

The life cycle of Theileria spp. is very complex and has many differentiation steps involves three typical phases: schizogony, gametogony and sporogony **(Nazifi et al., 2011)**. Like all intracellular parasites **(Shaw, 2003; Mans et al., 2015)**. The life cycle in vertebrate started after infection through tick’s bite; then the parasite was developed in lymphocytes and in this stage called macroschizont expressed as roundish cells with pale blue cytoplasm and

different numbers of red nuclei which refer to Koch's blue bodies **(Alim et al., 2012)**. The merozoites invade new host's cells, the microschizont appear after two weeks of infection is characterized by several spherical nuclei the microschizont invade RBCs that which the infective stage to the ticks **(Uilenberg and Babesia 2006; Mans et al., 2015)**.Figure (1) life cycle of *Thieleria spp.*

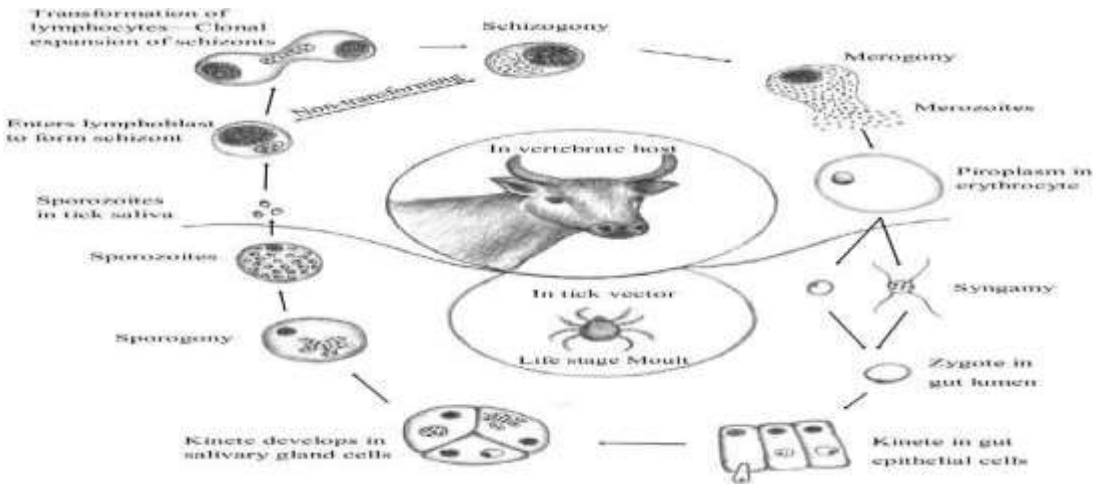

Figure (1): Life cycle of Theileria spp.
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Pathogeneses:

The pathogenesis of Theileria caused by macroscizonts and affect lymphocytes and reticular-endothelium system, the clinical signs and severity of the disease is depend on the degree of leukopenia due to arrested of maturation of these cells in bone marrow as result to toxic effect of parasite and increase the numbers of infected lymphocytes **(von Schubert et al., 2010; Ismael et al., 2013)** and production of a number of cytokine which may induce fever and play a role in anemia, muscle wasting and necrosis **(Dobbelaere and Baumgartner 2009)**.Moreover, **Tageldin et al., (2005)** recorded that, the hepatization of the lungs, and accumulation of excessive fluids and exudation in the chest cavity

(Tageldin et al., 2005). These fluids affect host respiration leading to serious tissue destruction and pulmonary edema **(El Imam, 2010)**.

The diagnosis:

Diagnosis of acute infection of Theileria is based on the clinical manifestations and microscopy detection of the intraerythrocytic piroplasms and intralymphocytic schizogony in the Giemsa stained blood and lymph node smears. In endemic areas, subclinical Theileria infection develops a chronic carrier, and act as a source of infection for ticks. In subclinical or chronic infections where the parasitemia is often very low, proficiency in microscopy detection of the piroplasm is essential **(Maharana et al., 2016)**.

Erythrocyte morphology:

Microscopical examination of the stained blood smears investigated various erythrocyte morphologic abnormalities in the T. annulata-infected camels. They comprise macrocytosis, poikilocytosis, anisocytosis, hypochromacia, metarubricytes and Howell-jolly bodies (**Salem and El Olemy., 2017).**

Histopathological lesions

Mohammed and Elshahawy (2018) reported that the *Theileria* infection in cattle caused prominent pathological findings including enlargement and swelling of the lymph glands and spleen, and ecchymotic and petechial hemorrhages of most of the internal organs, serous and mucous membranes and ulcers on the abomasal mucosa extended to intestines

OIE (Office of International Des Epizooties) (2014) recorded that Theileria infected cattle manifested by enlargement of superficial lymph nodes and subcutaneous edema.

Oryan et al. (2013) mentioned that T. annulata piroplasms in the cattle caused pale mucous membranes and petechial and ecchymotic hemorrhages in the lung, the mucosal and serosal surfaces together with lymphadenopathy. Moreover, the liver appeared friable, yellowish, and larger than normal. Hemorrhages and ulceration observed in the abomasal mucous membrane. Severe petechial hemorrhages found in the hairless areas of the skin. While histopathological examination revealed pulmonary edema and emphysema , proliferation of lymphocytes in the lymph nodes and proliferation of macrophages, lymphocytes and plasma cells in the spleen, Payer's patches, portal tracts of the liver, and interstitial tissue of the kidneys. Numerous multifocal areas of necrosis and ulceration noticed in the mucous membrane of the abomasum.

Sandhu (1996) noticed that T. annulata infections in calves induced noticeable necropsy lesions such as hepatomegaly, cutaneous hemorrhages, and abomasal ulcers. It was indicated severe damages to the hepatobiliary system due to hypoxia that resulted from hemolytic anemia and jaundice.

El-Refaii et al. (1998) detected large numbers of macro and microschizonts inside lymphocytes of the lymph nodes of T. annulata infected camels. Also, there was hyperplasia of lymphoblasts, besides lymphocytic depletion of the lymphoid follicles. Liver and lungs were intensely infiltrated with lymphocytes associated with intensive vacuolar degeneration of hepatocytes.

Fadly (2012) mentioned that blood parasites in cattle firstly affected lymph nodes resulted in lymph node enlargement and destructive disorders associated with fever, conjunctival petechia which are the most common signs.

Clinicopathological finding

Ayadi, et al. (2017) observed that Theileria annulata infection in cattle induced microcytic hypochromic anemia with significant decrease in erythrocytes, hemoglobin, and hematocrit, MCV, MCHC, MCH, levels. While level of total and direct bilirubin was significantly increased. While leucocytes, total proteins and albumin non-significantly changed.

Salem and El Olemy (2017) evaluated significant reduction in the mean values of the RBCs count, PCV, Hb concentration and MCHC of erythrogram of T annulata-infected camels as compared to non-infected camels. While there was anemia of macrocytic hypochromic type characterized by significant increase in MCV of T. annulata-infected camels in comparison with non-infected camels. Significant increase in the mean values of TLC, neutrophil, eosinophil and monocyte counts of T. annulata-infected camels as compared to non-infected camels, beside insignificant change observed in lymphocyte count of T. annulata-infected camels in comparison with non-infected camels. .

Youssef et al. (2015) exhibited that serum biochemical parameters related to the liver and kidney functions within insignificant changes in the T. annulata-infected camels when compared with non-infected camels. Serum total proteins and albumin were significantly lowered in the infected camels compared to control. However, significant increase detected in the serum globulins (α- and γ-globulins) and activity of AST in infected camels in comparison with the

control. The most common clinicopathological alterations associated with Thieleriosis are anemia and leukocytosis.

Abd-Elmaleck et al. (2015) detected a remarkable reduction in RBCs, Hb and Hct. levels with increase of WBCs in camels diseased with Theileria parasites. While MCV and MCH were decreased in infection with Thieleria infected camels.

Hussein et al. (2007) reported that cattle infected with Theileriosis is associated with some hematological and biochemical alterations particular in liver functions. Hematologically, it revealed normocytic hypochromic anemia. Furthermore, biochemically revealed decrease in serum levels of albumin and total proteins with increase in globulin level. Serum aspartate aminotransferase (AST) was significantly increased.

Boulter and Hall (2000) mentioned that tumor necrosis factor-α (TNF- α) has been involved in the pathogenesis of anemia in bovine Theileriosis by arresting haemopiotic progenitors.

Stockham et al. (2000) demonstrated that Thielosis resulted in signcificant increase in AST, besides hypoproteinemia, hypoalbuminemia. This is indication to the harmful effect of toxic metabolites of Theileria spp. on liver.

Babesiosis.

Babesiosis becomes a major concern in public health and animal production specialists in recent years both locally and internationally due to severity of out breaks, carrier animals and zoonotic nature. Babesiosis as an

emerging zoonotic disease and also causing heavy production losses due to the both clinical diseases and carrier animals **(Kirupananthana et al., 2016)**.

Babesia species are tick-borne hemoparasites infect erythrocytes inducing anemia in the host **(Aktas et al., 2012)**.

Multiple species exist by varying host specificity found all over the world **(Altay et al., 2008; Heidarpour et al., 2010; Razmi et al., 2003)**. Babesia caballi infect camel were recorded in Egypt and Sudan **(Abd Elmaleck et al., 2014: Abdelrahim et al., 2009).** Very few records are available about camel piroplasmosis lately in the one-humped camel zone, such as Iraq **(Jasim et al., 2015)**.

Babesia infected camels displayed changes in the blood constituents involving haemolytic anaemia, haemoglobinaemia, haemoglobinuria, anisocytosis, and polychromasia **(Wernery et al., 2002).**

Aktas et al., 2005 mentioned that infected animals after recovery from babesiosis become asymptomatic carriers. Moreover, **Kirupananthan et al., 2016** reported that no apparent clinical signs were noticed in Babesiosis carrier cattle. Diagnosing this carrier cattle population with Babesiasis is a challenge to the conventional diagnostic procedures due to low number of parasites in peripheral blood. However, diagnosis of carrier animals in herd is important for preventing outbreaks by transmission through vector ticks to healthy animals and for obtaining epidemiological data of the disease.

Morphology:

Babesia spp. was pear-shaped and arranged in pairs with variable angles near the margin of the infected RBCs. The Babesia cells varied in size from 1 to 2 μm for pairs with acute angles and from 3 to 4 μm for pairs with wide angles **(Swelum et al., 2014)**.

The parasite appears as a single or double pyriform and can take other forms depending on its type, round or oval or ring **(Bai et al., 2002)**.

Life cycle:

The life cycle of Babesia spp. within the vertebrate hosts is only multiplication by simple fission with highly destruction to 70% of the infected RBCs **(Duh et al., 2008; Silva et al., 2010)**. The life cycle of Babesia spp usually occur when the sporozoites injected into the blood stream of vertebrate with amount of saliva during the blood meal of infected ticks, the sporozoites invading erythrocytes then differentiate into trophozoites that asexually divided forming the merogony into two or sometime four merozoites, then the merozoites exit from erythrocytes invading new other one the replicative cycle is continuing in the host some of the merozoites are stop division and transform into gamonts or pregametocytes the gamogony and sporogony happened in the ticks when the ticks feed on an infected animals when they differentiate in the gut to form gametes that fuse forming a diploid zygote, the zygotes then transform to ookinetes which migrating through the hemocoel after that invade multiple ticks tissues like the salivary gland in the sporogony process and produce about 100000 sporozoites in the salivary glands tissues and transform to vertebrate body host during ticks blood meal and invade the red blood cells and this called transtadial transmission **(Schnittger et al., 2012)**. In invertebrate host

(ticks), the life cycle of development depending on environmental temperature, since occur very rapid with 28 c, high humidity and rainfall accommodate ticks carrying Babesia **(Joyner and Donnelly, 1979)**. Within the ticks gut, after 24-48 hrs the ray bodies (gametes) fused to each other to form motile zygote (ookinet) which appeared as cigar shape penetrate the epithelial cells of gut, then began to divided forming clap shape bodies (kinetes) and detected in gut 3-4 days after tick feed the zygot will be raptured and liberates the kinetes that will enter differnt organs like ovaries and salivary glands, in the ovaries the kinetes are lie in the yolk of ovum then stayed in the intestines of larval, so stages the infection occur by the infectious larve , nympha and this called ovarian transmission **(Alain et al., 2009)** Figure (2): Life cycle of *Babesia spp*.

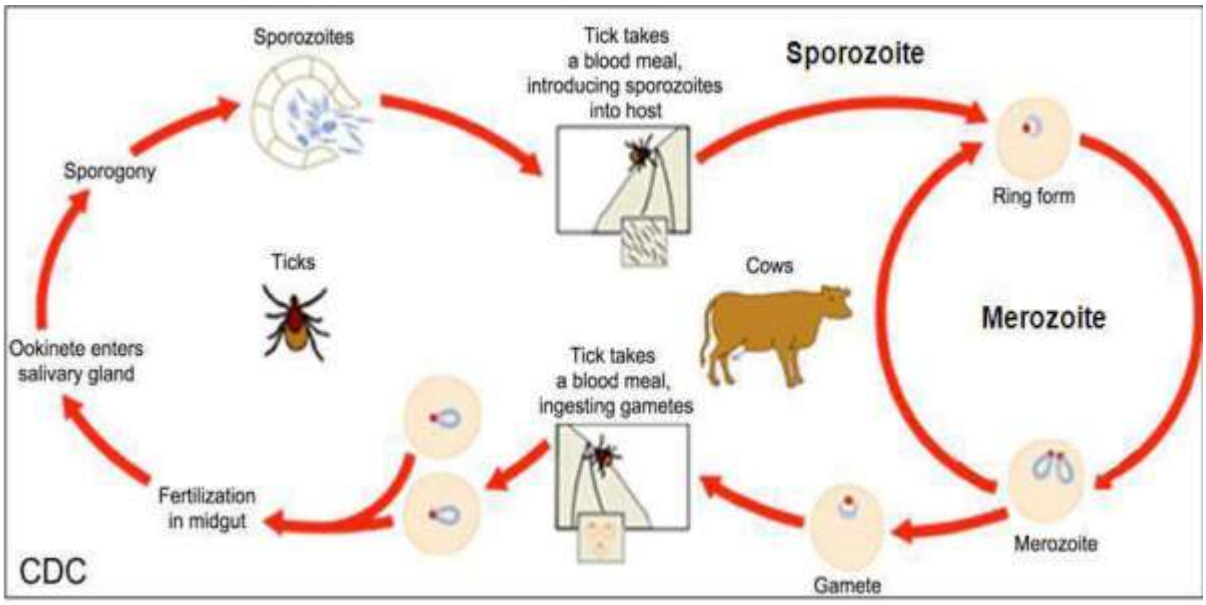


Figure (2): *Babesia spp.* Life cycle.

http://fullmal.hgc.jp/bb/icons/lifecycle.jpg

Clinical finding

The incubation period in babesiosis extended 1-2 weeks and the important signs fever 40-41c **(El Moghazy et al., 2014)**. The acute stage of the disease is

characterized by hemolytic anemia (**Talkhan et al., 2010; Ziapour et al., 2011).** Also there were ataxia, pale mucous membrane due to anemia, fatigue, weakness, muscle trembling, grinding of teeth, the feces are dry and bloody stained, and dehydration caused sunken eyes, falls of body temperature to subnormal level immediately before death, haemoglobinuria and enlargement of the prescapular and prefemoral lymph nodes **(Zobba et al., 2008)**. After recovery of camels from infection by piroplasmosis, become carrier and the value of such animals in the population is important in the epidemiology of Babesiosis **(Swelum et al., 2014)**.

The diagnosis:

The diagnosis of Babesia spp. done by using Giemsa staining of suspicious blood smears. Also, the morphology of the piroplasms in the RBC is decisive for the diagnosis of Giemsa stained blood smears. Diagnosis of Babesia spp. in acute phase by microscopic examination of Giemsa stained thin blood smears occurred when the number of the parasites is significant and high in the blood **(schnittger et al., 2004; Aktas et al., 2007)**. While in chronic infection, thick blood smears are characterized by presence of a little number of the parasite **(Abdullkadim, 2009; Schneider et al., 2011)**. Babesia identification in infected animals is difficult to be clinical signs dependent because there is much other disease that showed the same clinical signs; therefore it should identify parasites in the blood of the infected animal **(Abdullkadim, 2009; Perez-Llaneza et al., 2010)**.

Pathological lesions:

Mohammed and Elshahawy (2018) reported that, the macroscopic lesions of Babesia bovis in cattle include enlarged soft and pulpy spleen, distended liver, a gall bladder swollen with thick granular bile, congested dark-colored kidneys and generalized anemia and jaundice. Other organs exhibited congestion or petechial hemorrhages and occasionally pulmonary edema. The surface of grey matter of the brain appeared pink. Moreover, histopathological lesions of Babesia bovis included renal infarction, hepatic degeneration and lung edema.

Abd-Elmaleck et al., (2015) studied that the liver, kidney and lung of infected rats with Babesia spp. showed highly edematous with largely dilated appearance, it also suffered from dark-brown and extremely friable. Blood sinusoids were heavily infiltrated with macrophages and parasite-containing erythrocytes. The Kupffer cells are enlarged, in response to the process of phagocytosis of *B. cameli* infected erythrocytes. Moreover, there was inflaammation in liver characterized by predominant in infiltrations of lymphocytes, plasma cells, and histiocytes, which allied perivascular and parenchymal.

Habella et al., (1991) detected that Babesia infection induced renal hypoxia. As well, both renal infarction and disseminated intravascular coagulation in experimentally infected cattle with *B. ovis* was demonstrated.

Clinicopathological findings

Rubino et al., (2006) recorded that, high pathogenicity with Babesia occurs when the RBCs, packed cell volume (PCV) and hemoglobin concentration of animals was decreased resulted in anemia. Anemia occurs due to mechanical damage and destruction of RBCs by the binary fission of trophozoites (**Zobba et al., 2008**). Hemolytic anemia that occurs during acute stage is normocytic form and then become macrocytic with adequate reticulocytes and increased main corpuscular volume (MCV) (**Sulaiman et al., 2010**). At begging of infection Leukocytes were slightly decreased in number and then increase due to rise in lymphocytes (**Rahbari et al., 2008; Esmaeilnejad et al., 2014**). Other cause of anemia is decrease in number of RBCs lead to decrease of O_2 that induced tissues anoxia and damage especially in liver and kidney **(Turgut, 2000)**. Kidney damage occurs attributed to precipitation of immune complex in glomeruli initiated glomerulonephritis **(Rosenfeld and Dial, 2010).**

Abd-Elmaleck et al., (2015) detected a remarkable reduction in RBCs, Hb and Hct. levels with increase of WBCs in camels diseased with Babesia parasites. While MCHC non-significantly changed in Babesia infected camels.

Swelum et al., (2014) concluded that Babesiosis significantly affected the haematobiochemical parameters of dromedary camels, comprising the liver, kidney, and muscle functions. The affected animals showed significant reduction ($P<0.001$) in the RBC count, Hb Concentration, and MCV as well as significant reduction ($P<0.01$) in HCT and a significant reduction ($P<0.05$) in MCH. Additionally, significant increase ($P<0.01$) in white blood cell (WBC) count and platelets (PCT) count were recorded. However, other hematological

parameters were not significantly changed. Furthermore, there was a significant increases in total protein, albumin and aspartate aminotransferase (AST), were detected in the affected camels.

Esmaeilnejad et al., (2012) studied the hematological and biochemical parameters in small ruminants infected with *Babesia ovis*. The Hb concentration, PCV, RBCs, MCV and MCHC were significantly ($P<0.05$) decreased.Whilst total leukocyte counts, number of lymphocytes, monocytes, neutrophil and eosinophil revealed a significant ($P<0.05$) increase. Infected animals displayed significant ($P<0.05$) elevation in total proteins with significant ($P<0.05$) reduction in level of albumin and significant ($P<0.05$) increase in level of creatinine compared to non-infected animals.

Sulaiman et al., (2010) evaluated that *Babesia* infected goats resulted in significant decrease in total RBC, Hb concentration, PCV. Biochemically there was an increase in activity of alanine amino transferase (AST), aspartate amino transferase (ALT), blood urea nitrogen, with a significant decrease in levels of the serum total protein, albumin and globulin.

Hussein et al., (2007) reported that Babesiosis associated with some hematological and biochemical alterations particular in liver functions. Hematologically, it revealed normocytic hypochromic anemia, Biochemically, cattle infected with Babesiosis showed decrease in serum levels of albumin and total proteins with increase in globulin level. Serum aspartate aminotransferase (AST) was significantly increased in Babesiosis, while the serum level of alanine aminotransferase (ALT) was significantly elevated in case of Babesiosis.

Anaplasmosis

Anaplasmosis is infectious non contagious tick-borne diseases, it be considered one of the most important in domesticated and wild ruminant inducing significant economic losses in tropical and subtropical regions **(Kocan et al., 2000 ; Nazifi et al., 2011)**.

Anaplasmosis (Gall sickness) mainly prevalent in the tropics and subtropics has a peracute to chronic course in the large ruminants. It is a non-contagious, insect bite/tick borne haemoparasitic disease of domesticated and wild ruminants including camel **(Aiello and Mays 1998)**. The causative agent of Anaplasmosis in camls and cattle are Anaplasma marginale, **(Alsaad 2009; Boes and Durham 2017)**. Furthermore, Anaplasma centrale appears to be less pathogenic than Anaplasma marginal **(Kocan et al., 2003)**. Research on the effects of Anaplasma in camels is still not sufficient **(Al-Ani 2004)**.

The main characteristic findings of Anaplasmosis are fever, digestive disturbance, emaciation and progressive anemia **(Radostitis et al., 2000)**.

Anaplasmosis occurs more sporadically in temperate climate areas. Carrier animals are usually Reservoirs of infection **(Radostits etal., 2006)**. Moreover, biological transmission of A. marginale occurs between Anaplasmosis carriers to susceptible animals (**Kocan et al., 2004; Mohammed et al.,2007)**.

Morphology

Anaplasma known as black particles inside the red blood cells in Geimesa stained blood smear (**Ismael et al., 2014)**. Anaplasma remain in host

blood cells and located in central or marginal of red blood corpuscles according to species **(Kocan et al., 2003; Capucille, 2011)**. Under microscopical examination, Anaplasma marginale appear as spherical granules near periphery of infected erythrocytes **(Maghaddar, 2002; Al-Khaledi, 2008)**. While Anaplasma centrale is presented in central of erythrocytes **(Kocan et al., 2003; Liu et al., 2005)**.

Life cycle:

The evolution of the life cycle of Anaplasma is complex and coordinated with the tick feeding cycle **(Al-Khaledi, 2008)**. The tick takes the infected erythrocytes through blood absorption which is a main source of Anaplasma in the intestinal and tick cells. After the development of Anaplasma spp. in the intestinal cells of ticks, several tick tissues become infected, as salivary glands, during transmition of the parasite to host during feeding **(Futse et al., 2003; Amanda et al., 2006; Nazifi et al., 2011)**. Anaplasma spp. develops through gaps that enclosed by membranes or colonies in all infected tick tissues **(Kocan et al., 2010)**. The first form Anaplasma detected in the colonies is the vegetative form, where is divided by bilateral fission, forming large colonies **(Kocan et al., 2003)**.

Infected erythrocytes when disrupted, it release bodies which then can invade other erythrocytes. These bodies developed vacuoles within the cytoplasmic membranes of the red blood cells and then undergone binary fission forming dense blue-purple round/cube shaped inclusion bodies **(Rikihisa 2011)**. This amplifies infection within the host and increases the expectation of transmission when insect's blood feed **(Scoles et al., 2005)**. The second round of replication in the acinar cells of the salivary gland apparently depend on

resumption of tick feeding on a mammalian host is followed by transmission via the saliva **(Futse et al., 2003)**. Figure (3): life cycle of *Anaplasma spp*.

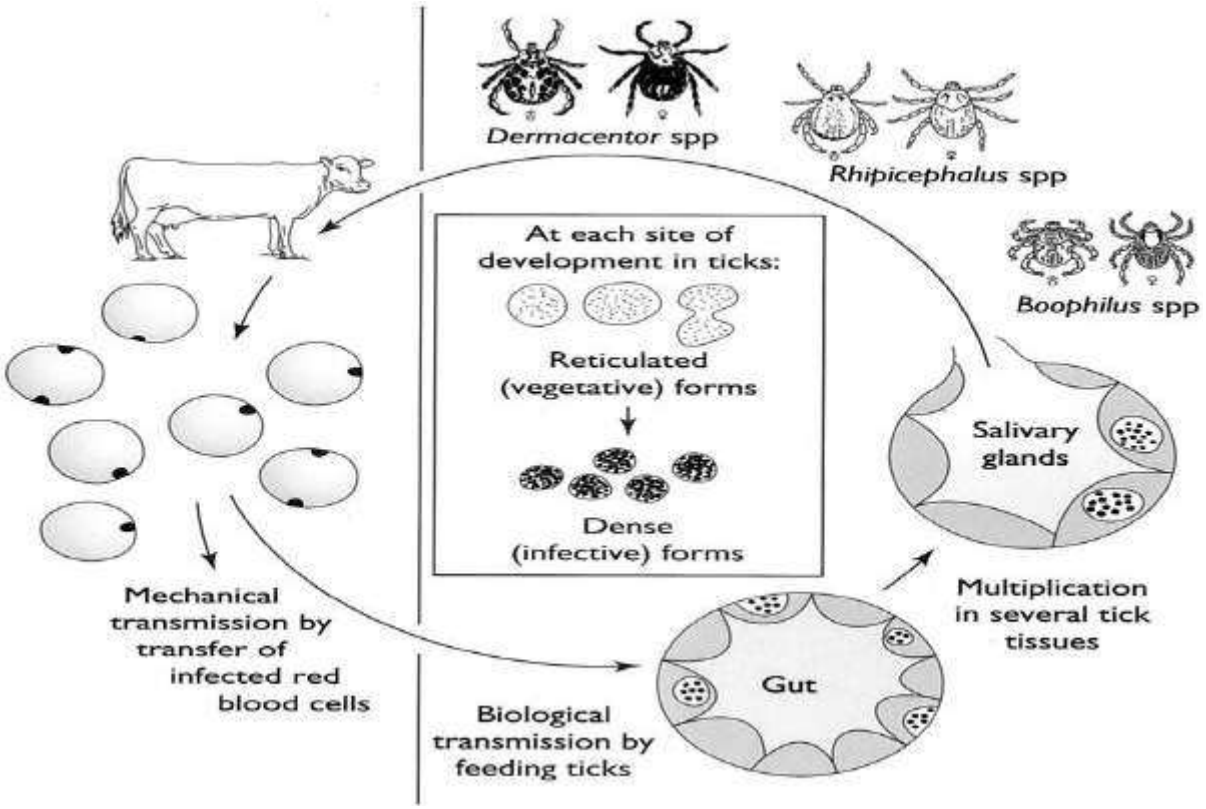


Figure (3): life cycle of *Anaplasma spp*. (Kocan et al., 2003)

Clinical signs

Clinically affected camels with anaplasmosis manifested clinical signs included fever, emaciation, anorexia, diarrhea, pale mucous membranes, lacrimation and anemia that persisted up to 87 days in some cases **(Hekmatimoghaddam et al., 2012)**. However, not all animals displayed the typical clinical finding, since some camels showed sub clinical anaplasma marginalis infection , with a clinical history of dullness, progressive loss of condition **(Sudan et al ., 2014).**

Diagnosis:

Microscopic examination for diagnosis of the blood protozoa including Anaplasma spp. through usage thin or thick Giemsa stained blood smears **(Tejedor-Junco et al., 2011**). Giemsa-stained blood films can be truly used as a proper method to detection of Anaplasma in the animals clinically suspected acute anaplasmosis, but it is not practical for the evaluation of presymptomatic or carrier animals **(Carelli et al., 2007).**

Pathological lesions:

Das et al., (2021) mention that the major histopathological changes noticed in cattle Anplasmosis included degenerative changes in hepatocytes and renal tubular epithelial cells, myocardial degeneration and necrosis, interstitial pneumonic changes, enlargement of red pulp area of spleen with histiocytic proliferation and hemosiderosis. Moreover, revealed inflammatory process characterized by infiltration of mononuclear cells in various organs as liver, kidney and heart.

Coetzee et al., (2005) recorded severe anemia, jaundice, hepatosplenomegaly, petechial hemorrhages, flabbiness and pallor in the heart of *Anaplasma marginale* infected beef cattle. Furthermore, Histopathology revealed inflammatory process characterized by mononuclears infiltration into various organs.

Lima et al., (2019) mentioned that cattle infected with Anaplasma grossly revealed pale carcass, ascites, and increased liver size` with rounded edges, full and distended gallbladder with thick bile, moderate jaundice and accumulation of dry stools in the rectum. Additionally histopathological examination of Anaplasma infected cattle, revealed focal infiltration of macrophages and

lymphocytes in the heart, liver and kidney, in addition hepatic sinusoidal dilatation, and necrosis of the large intestine.

Jaswal et al., (2015) recorded Fatty changes and bile retention in liver may be due to cholestasis and mild infiltration of mononuclear cells in portal triad and marked thickening of glission capsule or perihepatitis in Anaplasma infected cattle.

Egbe-Nwiyi et al., (1997) reported coagulative necrosis of the heaptocytes and the bile ducts hyperplasia in bovine Anaplasmosis.

Anderson and Hurtado (1989) detected centrilobular hypoxic hepatic necrosis and splenic hemosiderosis which may be due to erythrocyte destruction in Anaplasmosis.

DeVos et al., (2006) reported biliary retention in most cases of Anaplasmosis as there is accumulation of haemosiderin in cells of mono-nuclear phagocytic system, the lungs showed variable extents of interstitial pneumonia and emphysema and Spleen showed increased red pulp with massive proliferation of lymphocytes.

Clinico pathological findings:

Lima et al., (2019) studied that cattle infected with Anaplasma exhibited a distinct decrease ($p < 0.05$) in red blood cells, hemoglobin, PCV and platelets. In leukocyte count, there was significant increase in lymphocytes in comparison with non-infected camels.

Coskun et al., (2012) recorded that Anaplasma marginale associated parasitemia caused changes in biochemical and hematological parameters associated anemia and tissue damage in cattle. Significant decrease in red blood cell count, packed cell volume, and hemoglobin concentration was detected in infected cattle compared to control. Serum aspartate aminotransferase and creatinine concentrations were significantly elevated in infected cattle in comparison with the control.

Sudan et al., (2014) reported that, in subclinical anaplasmosis in camel (Camelus dromedarius) examination of the blood confirmed lower value of haematological indices (Haemoglobin 10.4 g/dl, TEC 5.5 9 106/mm3) besides, neutrophilia and eosinophilia(Neutrophils 65 %, Lymphocytes 28 %, Monocytes 2 %and Eosinophils 5 %). Moreover, the Giemsa stained thin blood smear with moderate parasitaemia (30 %) revealed intraerythrocytic small, polymorphic (oval to spherical) rickettsial bodies near to the periphery of erythrocyte cell membrane.

Azeem et al., (2019) noted that hemoparasitized camel with Thieleriosis ,Anaplasmosis and Babesiosis showed significant decrease in PCV, Hb, RBC and PLT values whereas TLC was increased significantly as compared to healthy camels. Biochemically, haemoparasitized camels have constant trend to considerable increase in activities of ALT and AST. On the contrary, the mean level of total protein and albumin was significantly decreased.

Trypanosomaisis

Trypanosomiasis is one of the more important animals diseases and severe pathogenic protozoan disease for camel in tropical and semitropical

regions, caused by Trypanosoma evansi **(Omer et al., 2004; Barghash et al., 2014).**

Trypanosoma evansi is transmitted mechanically by insect **(Paim et al., 2011).** The morbidity and mortality rates in a population of camels with no immunity can be high **(Kassa et al., 2011).** In addition to causes economic losses because decreased productivity in working animals, reduced weight gain, decreased milk yield, and the cost of treatment due to infected animals suffer from illness, anaemia and deaths, **(Eyob & Matios, 2013).**

Trypanosoma evansi is similar in shape in all mammals just different in size, Trypanosoma have a leaf-like or rounded body containing a vesicular nucleus ,and a varying number of sub pellicular microtubules lying beneath the outer membrane, There is a single free flagellum arising from a kinetosome or basal granule **(Laha &sasmal,2008; Abera et al .,2015)**. An undulating membrane is present in some genera and the flagellum **(Taylor et al., 2007)**.

The life cycle

The life cycle of T. evansi began from ingested by flies transmit T. evansi mechanically through their mouthparts when they feed on more than one host within a short interval because the trypanosoma remain infective for only a short period and then multiple in mid gut for 10 days and migrate to salivary glands in epimastigote form and return multiple in salivary glands convert to metacyclic form (infective form) injected by fly during bite to become trypomastigote in blood and lymph of the host **(Dawood, 2007; Nasir, 2015)**

MATERIAL AND METHODS

Materials:

1- Animals :

The present study was carried out on one hundred camels (3-7 years) slaughtered at Daraw and Aswan slaughter houses, belonging to Aswan Governorate during period from October 2019 to September 2020.

Chemical: Bio-Diagnostic Kits produced by Bio-Diagnostic Laboratories, Dokki, Giza, Egypt.

2- Samples :

I- Blood sampling:

Two separate blood samples were dragged from jugular vein of study camels from Aswan and Draw slaughter houses.

First blood sample

It was taken in vacuum tube with EDTA anti coagulated. Concerned for

A-Giemsa stained blood smears

B-hematological studies

The second blood sample

It was taken in plain tubes, centrifuged at 3000 rpm for 10 min. and the resultant clear serum was separated carefully and preserved in Eppendorf tubes at -20 °C for Biochemistry analysis.

II- Tissue samples :

Tissues samples from lymph node and liver were collected from the camels slaughtered at Aswan and Daraaw slaughtered houses,

Methodes

Giemsa stained blood smears

Thick and thin Giemsa stained blood smears were made for morphological examination of the protozoan blood parasites under an oil immersion lens**.**

Hematology

The anticoagulated blood samples was used for complete blood picture including white blood cells count (WBCs) count, red blood cells count (RBCs), total hemoglobin, hematocrit assays, differential leucocytic count, MCV, MCH and MCHC) which done by Automated Hematology Analyzer (Diff3) Mek-6410/Mek-6420.Animal Health Research Institute Doki, Giesa , Egypt.

Biochemical analysis

Biochemical analysis had been estimated spectrophotometry via usage standard test kits.

- **Assessment of liver function tests:**

A) Determination of alanine aminotranferease (ALT) and aspartate aminotransferase (AST):

Activities of AST and ALT were evaluated colorimetrically in serum according to **Reitman and Frankel, (1957)** using spectrophotometer Bio-Dignostic Kits produced by Bio-Diagnostic Laboratories, Dokki, Giza, Egypt.

Calculation:

From absorbance, the reading units of AST and ALT assessed in relation to the standard curves of enzymes activity.

B) Determination of Total Protein:

Total protein was measured through usage the colorimetric method according to **Gornal et al., (1949)**.

Calculation:

$$\text{Total protein (gm/dl)} = \frac{\text{A sample}}{\text{A standard}} \text{x } 5$$

C) Determination of Albumin:

Albumin was determined colorimetrically as described by **Doumas et al., (1971)**.

Calculation:

$$\text{Albumin (gm/dl)} = \frac{\text{A sample}}{\text{A standard}} \text{x } 4$$

D) Determination of Globulin:

Globulins were determined through subtraction the albumin value from the total protein value.

Calculation (g/dl) = Total Protein – Albumin

- **Assessment of kidney function tests :**

A) Determination of serum urea:

Level of urea was estimated using enzymatic colorimetric method which described by **Fawcett and Scott, (1960)** via Bio-Diagnostic Kits, Dokki, Giza, Egypt.

Calculation:

$$\text{Urea (mg/dl)} = \frac{\text{A sample}}{\text{A standard}} \text{ X } 50$$

B) Determination of serum creatinine:

Colorimetric method was done for evaluation of creatinine level in serum according to **Bartles et al., (1972)** using Bio-Diagnostic Kits, Dokki, Giza, Egypt.

Calculation:

$$\text{Creatinine (mg/dl)} = \frac{\text{A sample}}{\text{A standard}} \text{ X } 2$$

Tissues

Tissues samples from lymph node and liver were collected from the slaughtered camels and fixed in 10 % neutral-buffered formalin. The specimens were routinely processed for paraffin embedding technique **(Bancroft et al., 1996)**. Stained sections were examined under light microscopy (Olympus CX31, Japan).

Statistical analysis:

One-way analysis of variance (ANOVA) using SPSS (Statistical Package for Social Sciences) was performed for data analysis according to **Borenstein et al. (1997)**. The results were expressed in form of Mean ± Standard Deviation. The difference was significant when $P< (0.05)$.

RESULTS

HAEMATOLOGICAL RESULT

1- Blood smears

Prevalence & seasonal varation of blood parasites:

Microscopical examination of the Giemsa stained blood smears revealed Thieleria , Anaplasma, and Babesia sp. in (30%) out of the 100 asymptomatic camels examined throughout four different seasons and negative *Trypanosoma Spp.* result (From October 2019 till September 2020). Generally *Thieleria spp.* was represented the highest incidence of infection (15%) in the present study especially in autumn (8%), followed by *Anaplasma spp.* (10%) and the lowest incidence of infection was represented in *Babesia spp.* (5%). At the same time the lowest incidence through different seasons was represented in spring (1%) only *thieleria spp*. **(Table 1, Fig. 4).**

Table (1): Percentage of blood parasites prevalence of *Thieleria*, *Anaplasma* and *Babesia* recorded in camels examined throughout four differrant seasons.

Season	Thielerea	Babesia	Anaplasma	Total %
Autumn	8	3	5	16
Winter	2	1	2	5
Spring	1	-	-	1
Summer	4	1	3	8
Total %	15	5	10	30

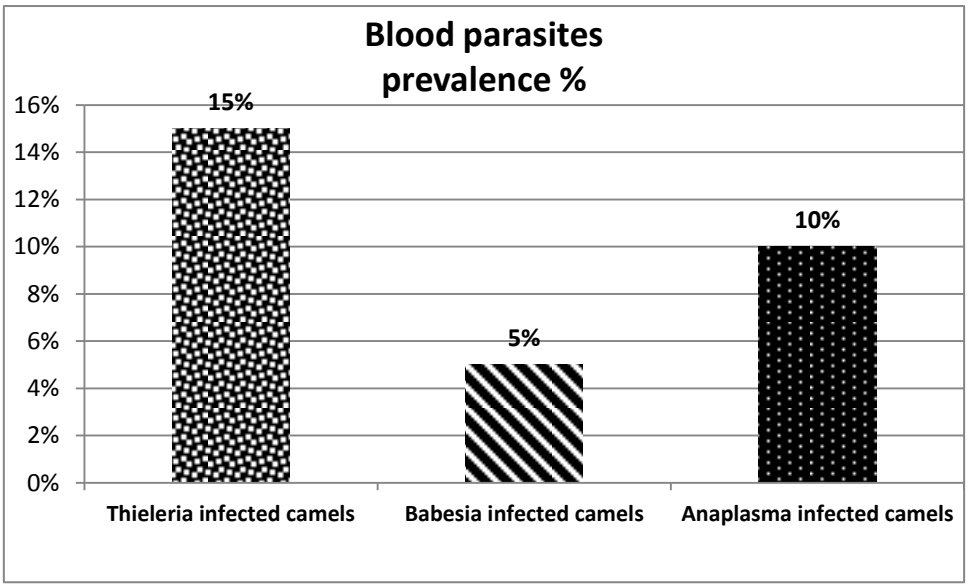


Fig. (4): Percentage of blood parasites prevalence of Thieleria, Babesia, and Anaplasma in the examined camels.

Microscopical examination of the Giemsa stained blood smears

Microscopical examination of the Giemsa stained blood smears revealed that:

In camels infected with *Theileria spp*., the intra-erythrocytic trophozoit signet ring and cocci, shaped piroplasms as well as the intralymphocytic schizogonies "Koch's blue bodies" and presence of dacrocyte cell in **Figure (5, 6, 7).**

In *Anaplasma* infected camels, dense, homogeneously staining blue-purple inclusions. *A marginale* inclusions were usually located toward the margin of the infected erythrocyte with presence of hawell jolly body and macrocytic hypochromic erythrocytes Figure **(8, 9, 10).**

In *Babesia* examined camel Babesia merozoite were located in the erythrocyte, it is usually found as pairs that are at an obtuse angle to each other. Hypochromic erythrocytes were a prominent finding.**Figure (11, 12)**.

Microscopic examination of the stained blood smears from camels with different types of hemoparasites demonstrated varied erythrocyte morphologic abnormalities which include macrocytosis, anisocytosis and poikilocytosis, hypochromacia **(Fig. 5, 6, 7, 9, 10, 11,12)**.

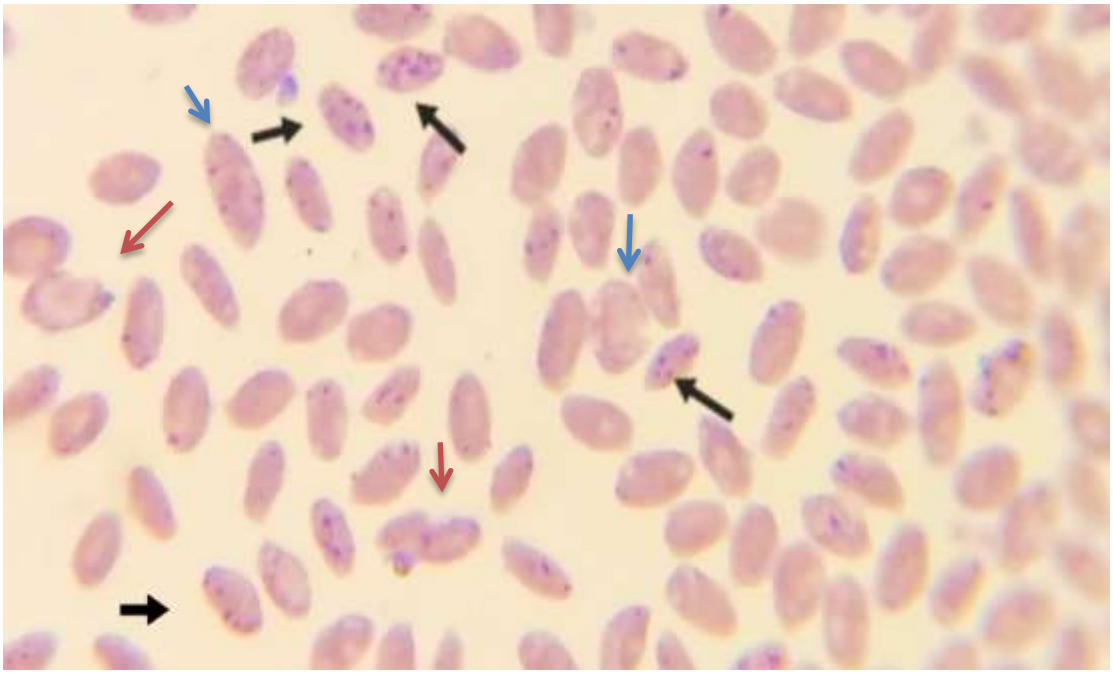
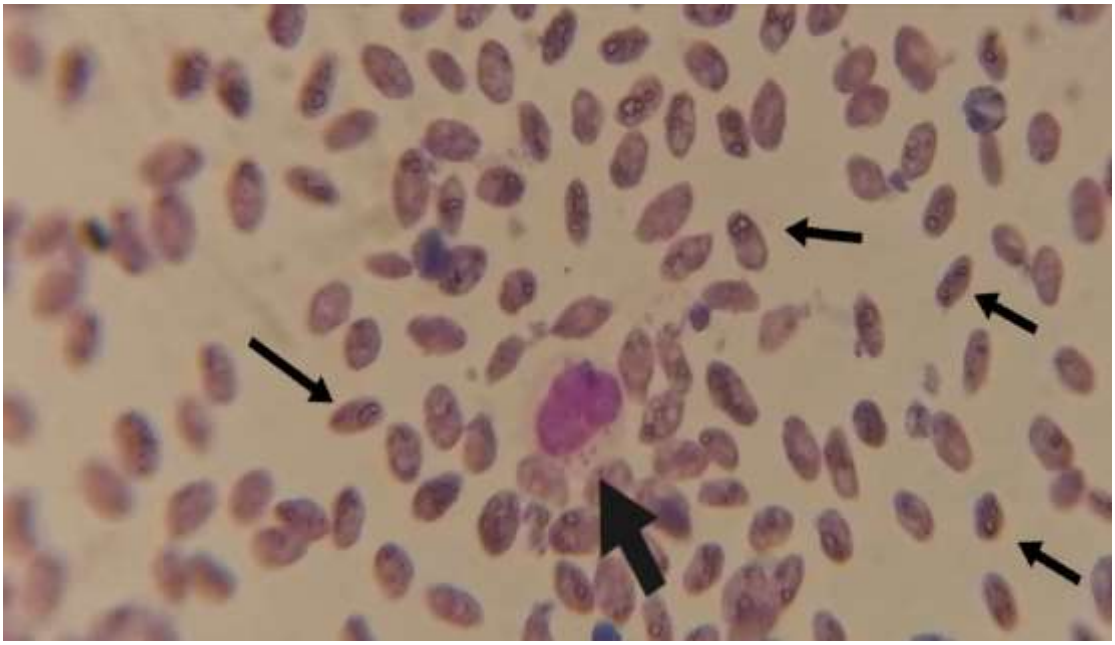


figure (5): Giemsa-stained blood film from examined camel showing Cocci form of Thieleria (black arrow) located in erythrocyte of camel. Note also the hypochromic(red arrow) and macrocytic(blue arrow) erythrocytes **(X1000).**



Figure(6): Giemsa-stained blood film showing schizont of Thieleria in lymphocytes (thick arrow) and signet-ring form of Thieleria piroplasm (thin arrow)in erythrocyte of examined camel.**(X1000).**

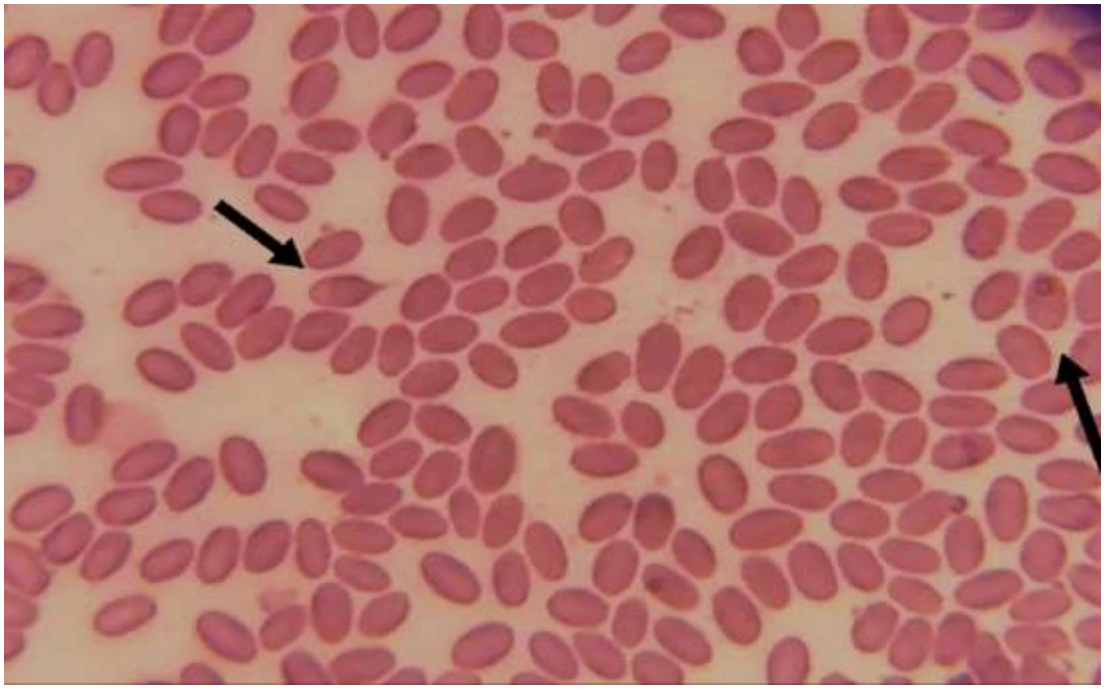


Figure (7): Giemsa-stained blood film from examined camel showing dacrocyte cell (arrow) andThieleria infected RBCs in form of (signet ring) **(X1000).**

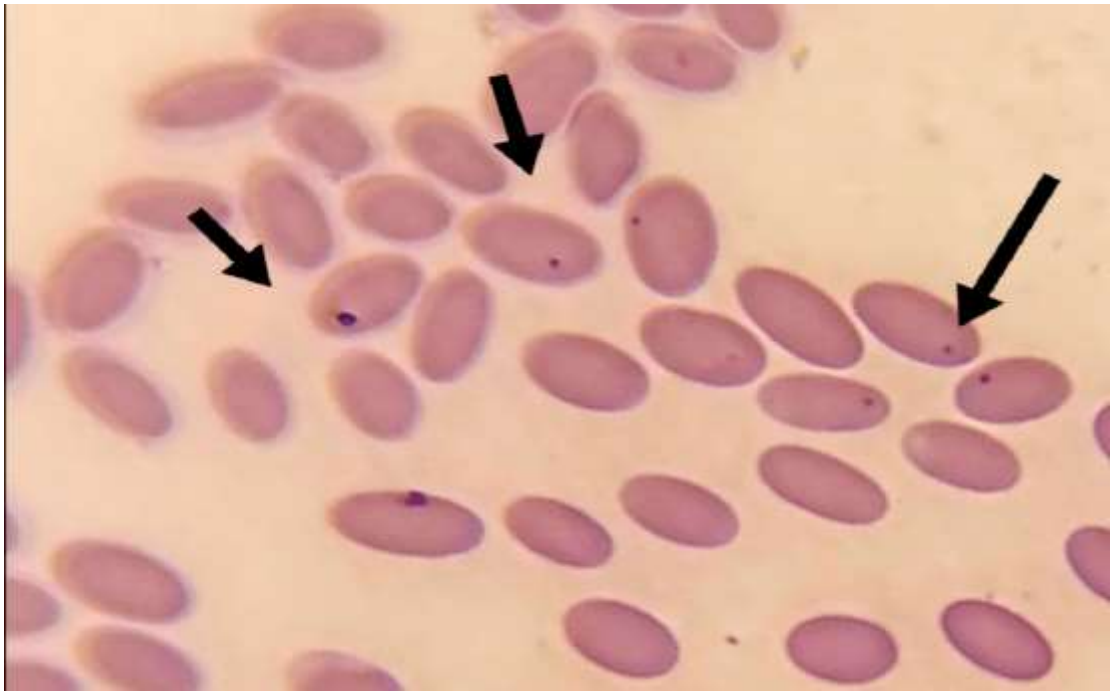


Figure (8): Giemsa-stained blood film from examined camel showing dense, homogeneously staining blue-purple inclusions *A. marginale* inclusions are usually located toward the margin of the infected erythrocyte **(X1000)**.

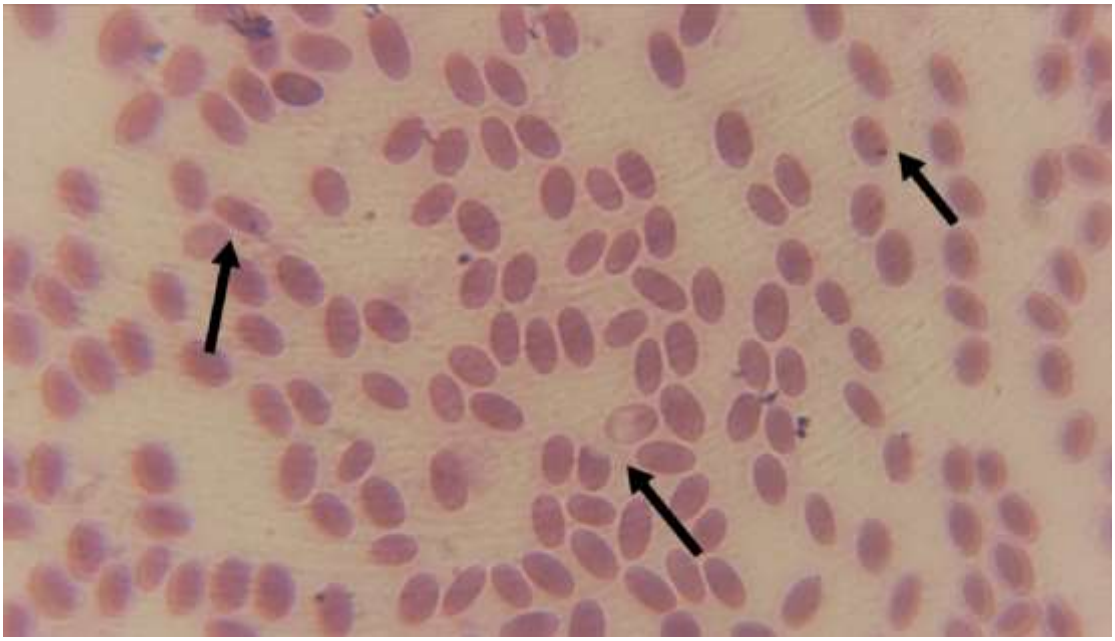


Figure (9): Giemsa-stained blood film from examined camel showing Anaplasma in erythrocyte of camel . Note also the hypochromic erythrocytes. **(X1000)**.

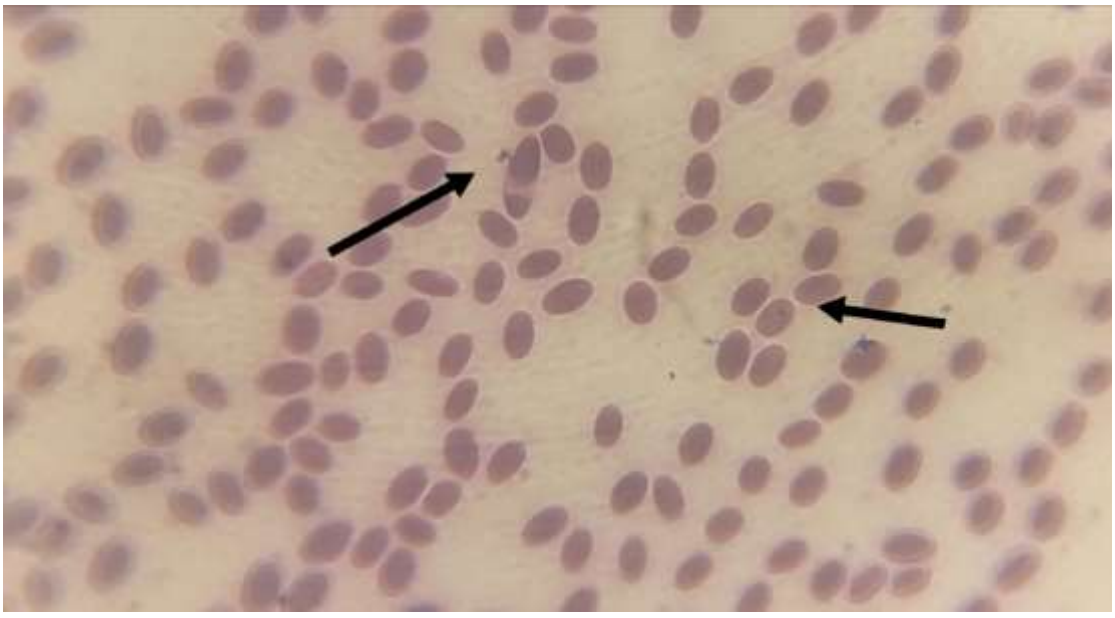


Figure (10): Giemsa-stained blood film from examined camel showing . Macrocytic hypochromic erythrocyte (arrow) in Anaplasma infected camel **(X1000)**.

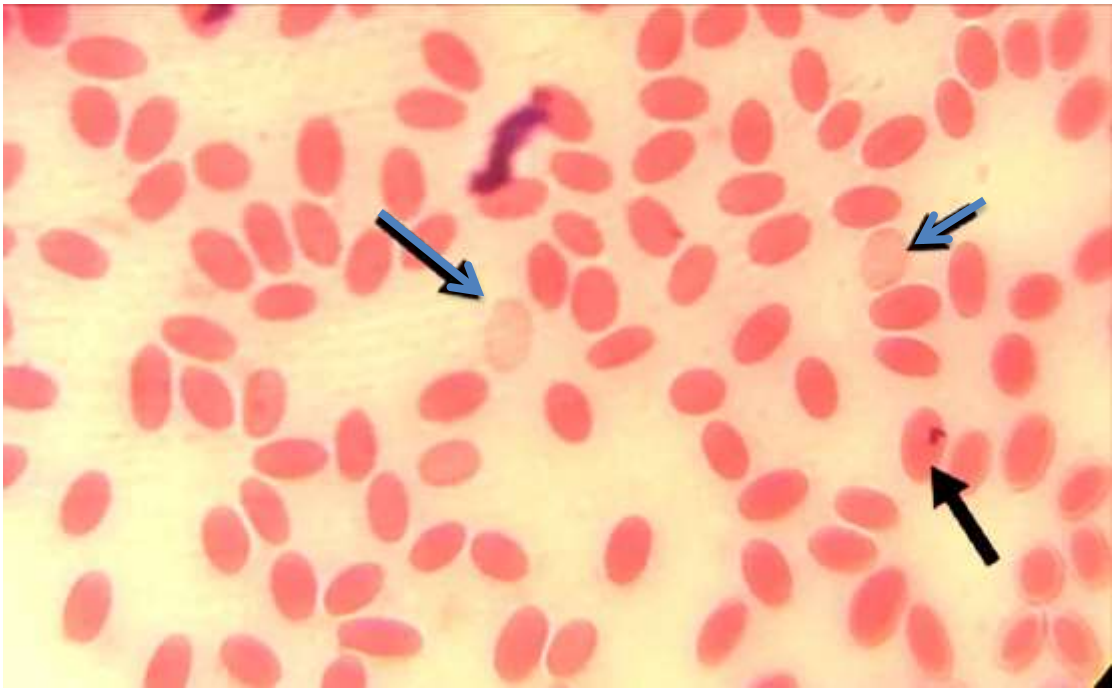


Figure (11): Giemsa-stained blood film from examined camel showing Babesia merozoite located in the erythrocyte(black arrow), found as pairs that are at an obtuse angle to each other. Note also hypochromic erythrocytes (blue arrow) (**X1000).**

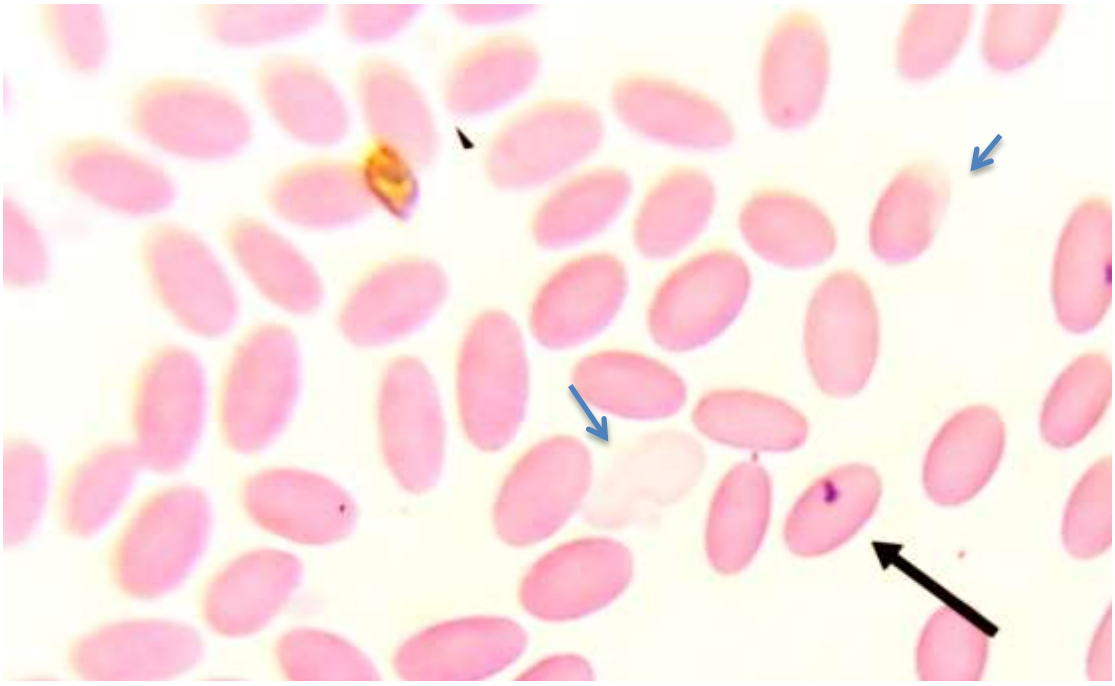


Figure (12): Giemsa-stained blood film from examined camel showing pronounced hypochromic erythrocytes(blue arrow) in Babesia infected camel(black arrow) **(X1000)**.

2- Hematological Parameters:

There was significant decrease ($P<0.05$) in RBCs count, Hb. Concentration, and PCV % in Thieleria, Babesia, and Anaplasma infected camels when compared with non-infected camels. Additiotionally, There was non-significant increase ($P<0.05$) in MCV in Thieleria, Babesia, and Anaplasma infected camels when compared with non-infected camels. While other parameters including WBC count, MCH, and MCHC values showed non-significant changes in Thieleria, Babesia, and Anaplasma infected camels when compared with non-infected camels **(Table, 2 & Figs.13, 14, 15, 16, 17, 18 &19).**

3- Differential leucocytic counts:

There was significant increase in lymphocytes % ($P<0.05$) with T. annulata, and Babesia infected camels when compared with non-infected camels. Also, significant increase ($P<0.05$) in eosinophil % in T. annulata, Babesia and Anaplasma infected camels when compared with non-infected camels. Monocytes showed increase ($P<0.05$) in Anaplasma infected camels. While, other parameters including Neutrophil, basophil values showed non-significant changes with non-infected camels **(Table, 3 & Figs. 20, 21, 22, 23 &24)**.

Table (2): Level of **hematological parameters** including RBCs, WBCs, hemoglobin, PCV, MCV, MCH, and MCHC of Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

Parameters	Blood parameters						
	RBC's ($x10^3$)	**WBCs ($x10^6$)**	**Hb. (gm/dl)**	**PCV (%)**	**MCH (Pg)**	**MCV (Fl)**	**MCHC (%)**
Thieleria infected camels	7.7±0.9*	10.6±0.3	11.5±0.6*	25.2±1.5*	14.8±0.5	32.4±0.6	45.7±0.5
Babesia infected camels	8.5±0.7*	9.9±0.87	13.9±0.5*	28.4±0.8*	15.5±0.5	33 ±0.9	47.8±0.3
Anaplasma infected camels	7.9±1.2*	10.1±0.4	12.6±0.8*	26.3±2.3*	15.9±0.6	30.03±0.8	48.1±0.5
Non infected camels	11.0±0.8	11.2±0.5	14.3 ±1.1	32 ±0.9	13.0±1.1	29.09 ±0.9	44.6±0.4

*→ is referring to significant changes in comparison with non- infected camels when $P<0.05$ %.

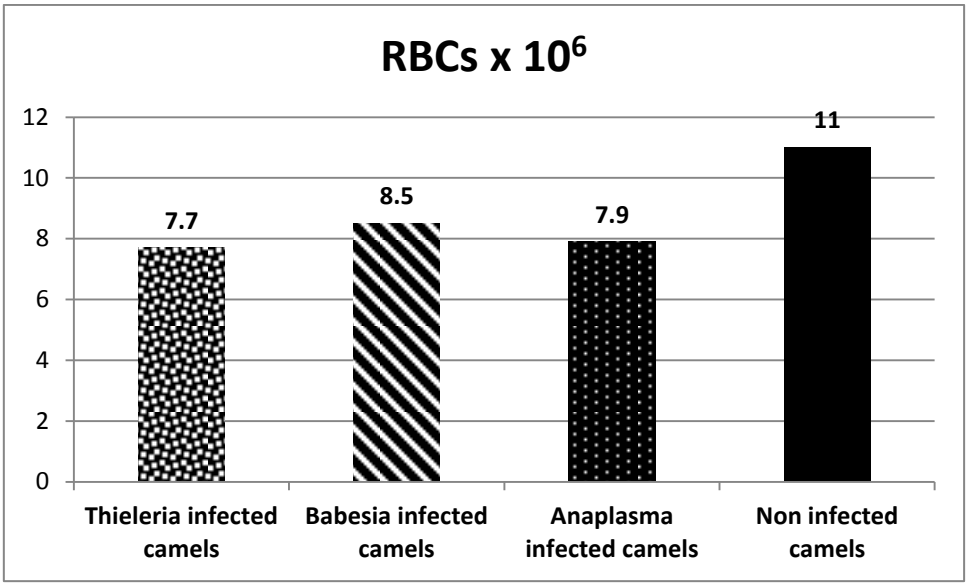


Fig. (13): Level of RBCs (x 10^{12}) of the camels infected with Thieleria, Babesia, Anaplasma and non-infected camels.

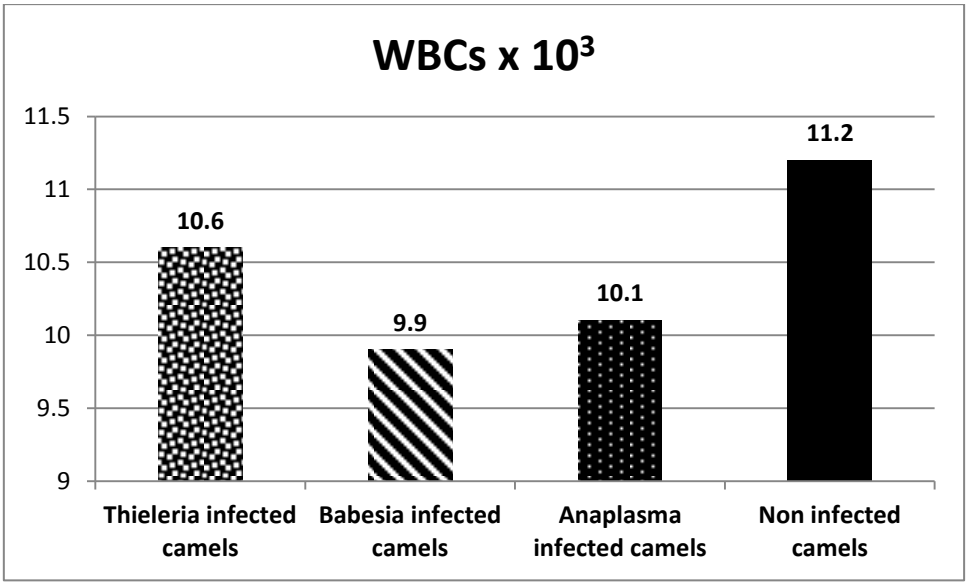


Fig. (14): Level of WBCs (x 103) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

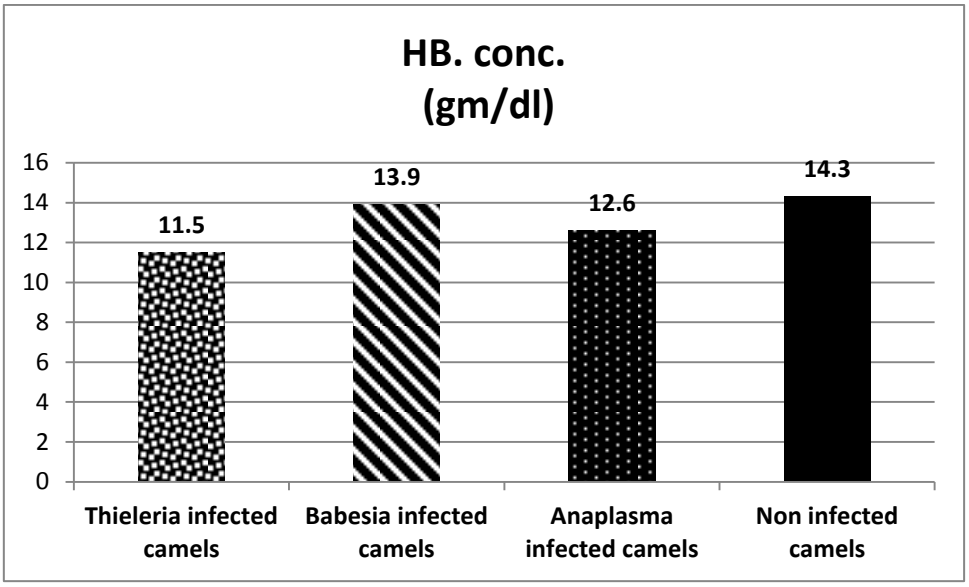


Fig. (15): Level of hemoglobin conc. (mg/dl) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

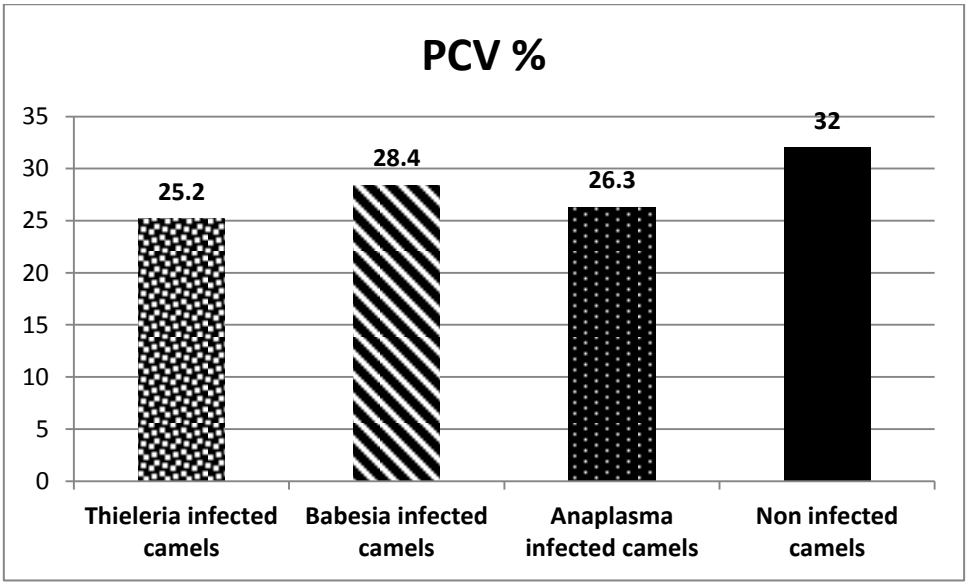


Fig. (16): Level of PCV (%) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

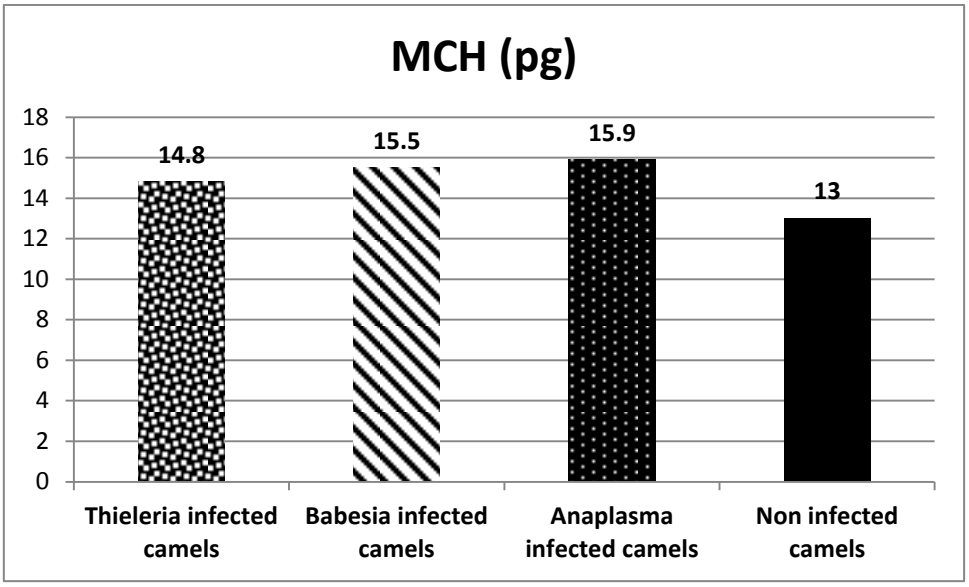


Fig. (17): Level of MCH (pg) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

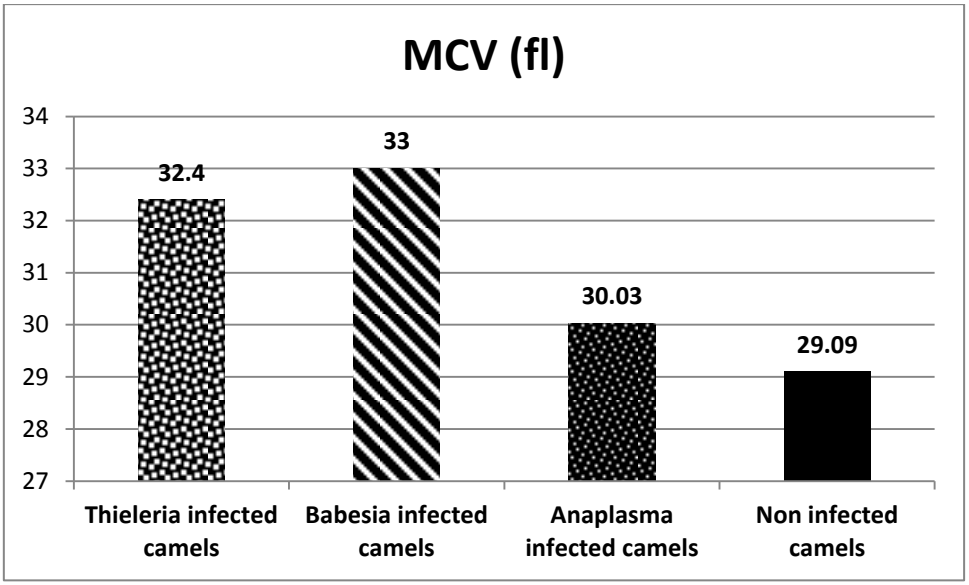


Fig. (18): MCV (fl) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

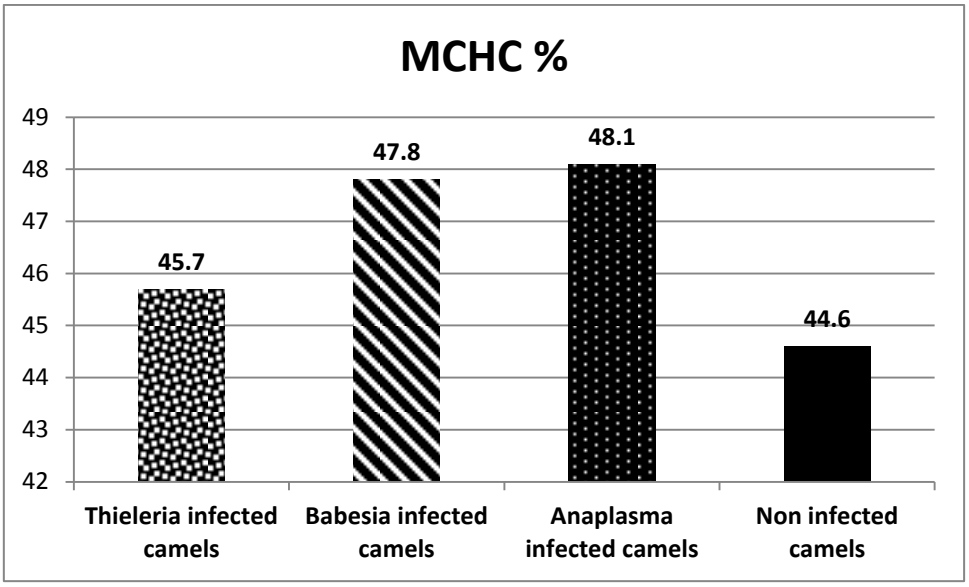


Fig. (19): Level of MCHC (%) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

Table (3): Differential leucocytic count of Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

Parameters	Differential leucocytic count				
	Lymphocytes %	**Neutrophil %**	**Monocytes %**	**Eosinophil %**	**Basophil %**
Thieleria infected camels	82.0±3*	7.0	2.0	9 .0±0*	0.0 ±0
Babesia infected camels	84.0±1*	2.8±1.5	7 .0±0.5	6.2 ±0*	0.0 ±0
Anaplasma infected camels	71.6±2.3	5 .0±1.5	10.6±0.5*	8.0 ±0*	0.6±0.5
Non infected camels	75.0 ±0.5	16.4±1.5	8.3±.5	0.3±0.5	0.0 ±0

*→ is referring to significant changes in comparison with non- infected camels when P<0.05 %.

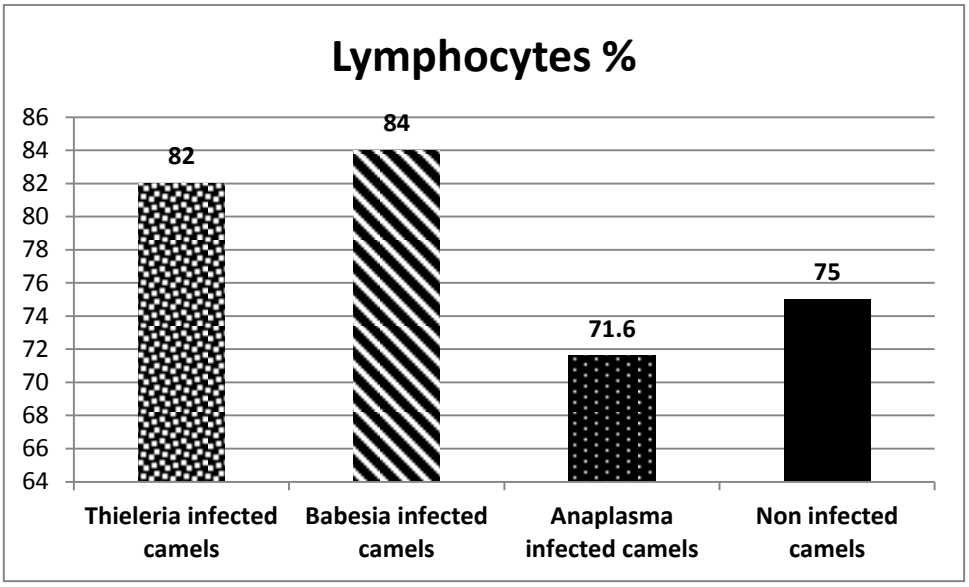


Fig. (20): Level of lymphocytes (%) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

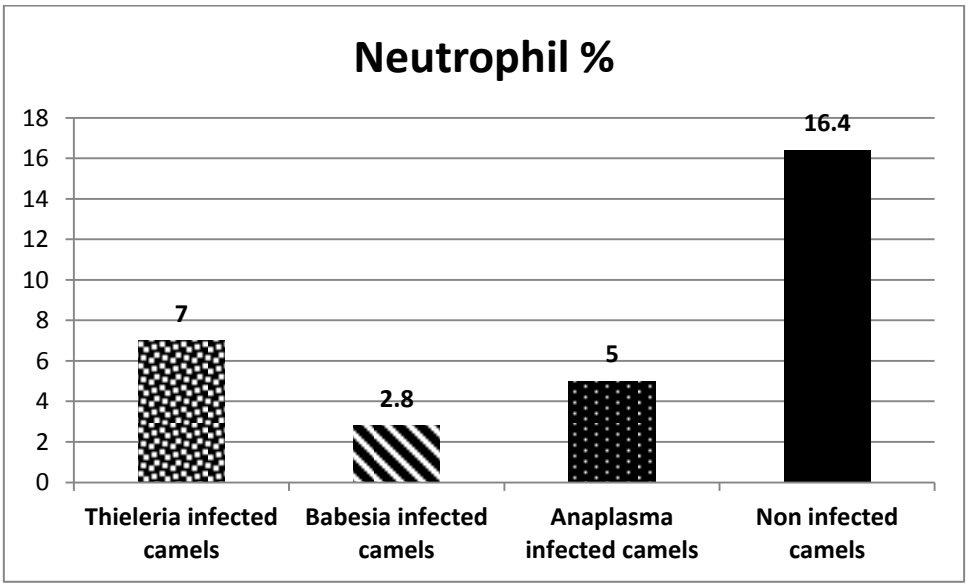


Fig. (21): Level of Neutrophil (%) of the Thieleria infected camels Babesia infected camels, Anaplasma infected camels and non-infected camels.

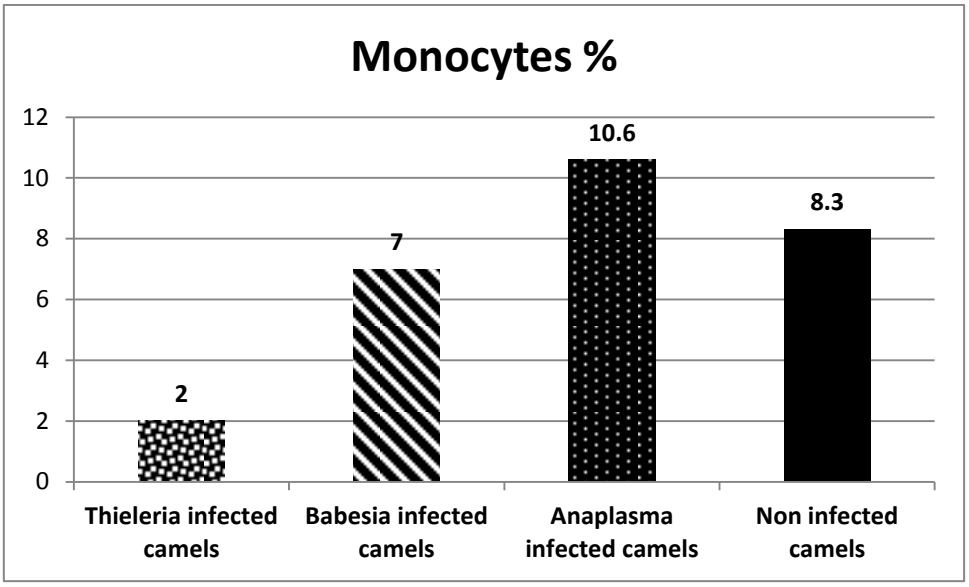


Fig. (22): Level of Monocytes (%) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected

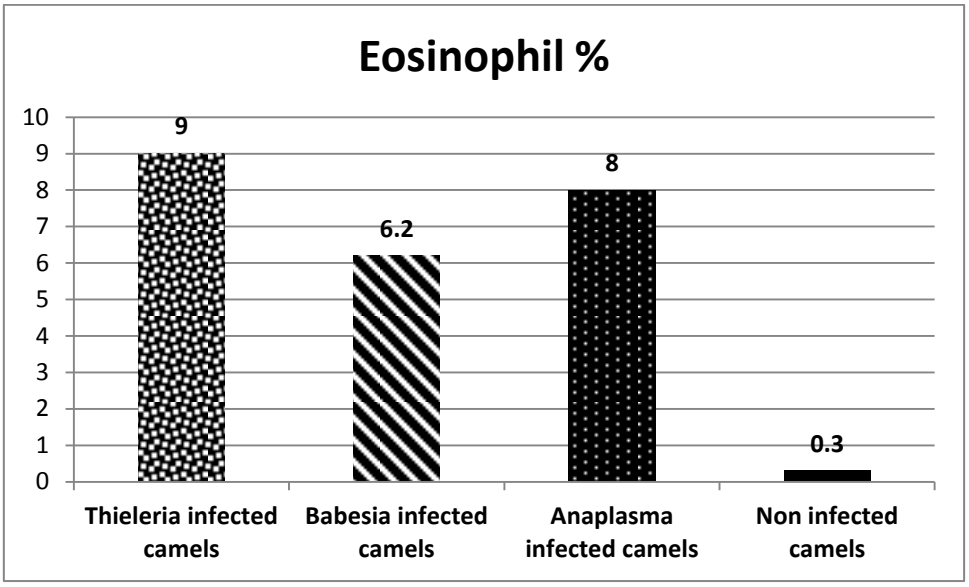


Fig. (23): Level of Eosinophil (%) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

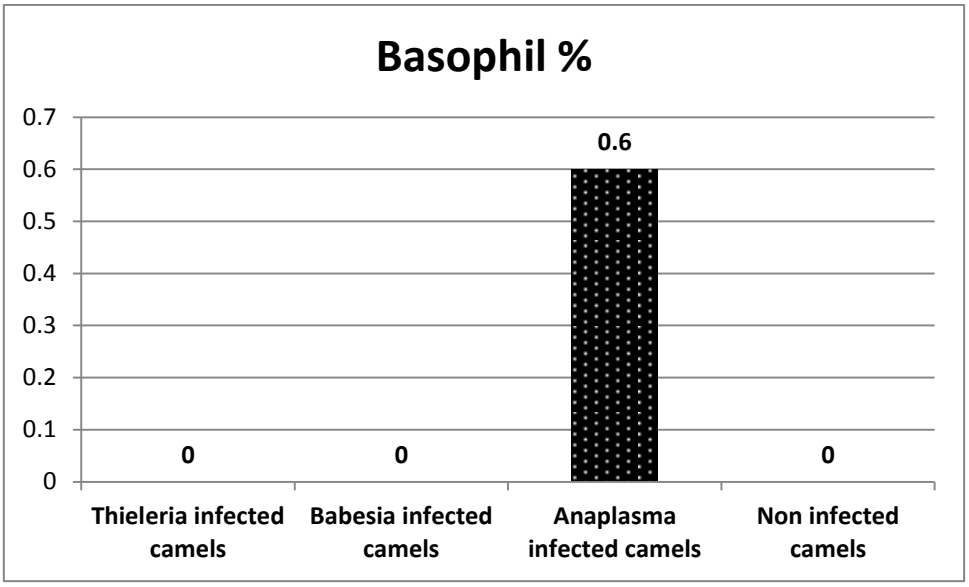


Fig. (24): Level of Basophil (%) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

BIOCHEMICAL ANALYSIS:

1- Liver Function Tests:

A- Aspartate aminotransferase (AST) and Alanine aminotransferase (ALT):

Level of AST revealed significant increases (P<0.05) in Thieleria, Babesia, and Anaplasma infected camels in compare with non-infected camels at P<0.05. While, ALT revealed significant increase (P<0.05) only in Anaplasma infected camels in comparison with non-infected camels at P<0.05 **(Table, 4, Figs. 25 & 26)**.

B- Total protein, Albumin, and Globulin

Level of total protein, albumin and globulin exhibited non-significant decrease in Thieleria, Babesia, and Anaplasma infected camels in comparison with non-infected camels at P<0.05 **(Table, 5, Figs. 27, 28&29)**.

Table (4): level of liver function tests of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

Groups	Liver Function Tests	
	GOT/AST (IU/l)	**GPT/ALT (IU/l)**
Thieleria infected camels	121.0±2.0*	11.0±2.1
Babesia infected camels	114.6±2.0*	13.5±1.0
Anaplasma infected camels	103.0±3.6*	14.2±1.5*
Non infected camels	69.6±2.5	11.0±1.0

*→ is referring to significant changes in comparison with non-infected camels when $P<0.05$ %.

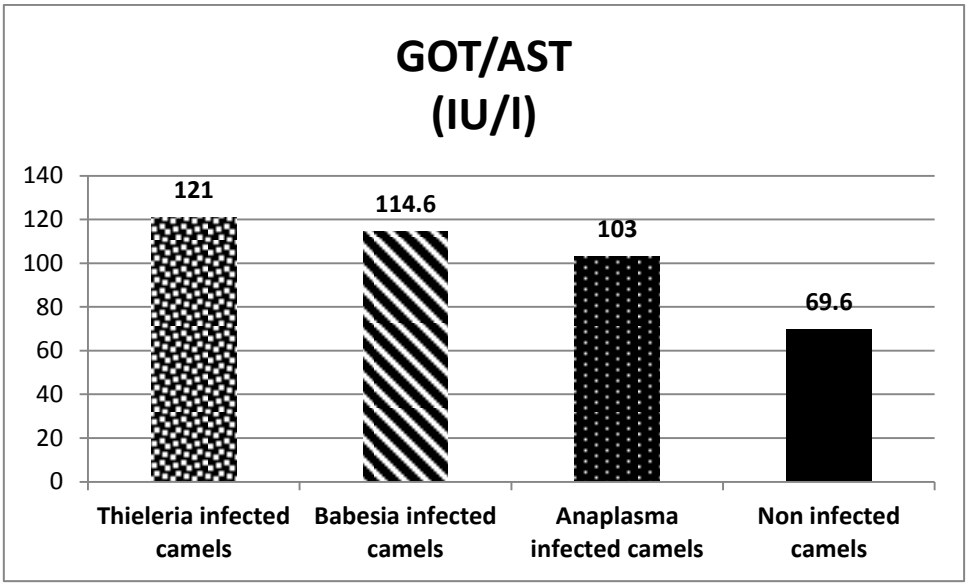


Fig. (25): Level of GOT/AST (IU/l) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

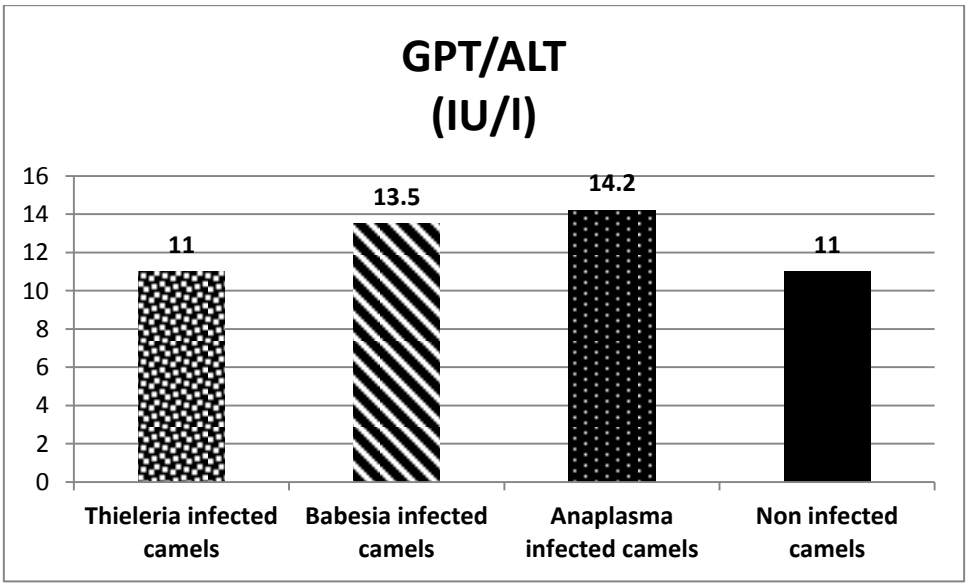


Fig. (26): Level of GPT/ALT (IU/l) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

Table (5): Level the Total protein, Albumin, and Globulin (gm/l) of Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

Groups	Protein profile		
	Total Protein (mg/dl)	**Albumin (mg/dl)**	**Globulin (mg/dl)**
Thieleria infected camels	7.07±1.5	2.5±0.8	4.5±0.8
Babesia infected camels	7.3±1.2	2.4±1.1	4.9±0.7
Anaplasma infected camels	8.2±0.9	2.7±0.6	5.0±0.7
Non infected camels	9.53±2.1	3.14±0.5	6.4±1.7

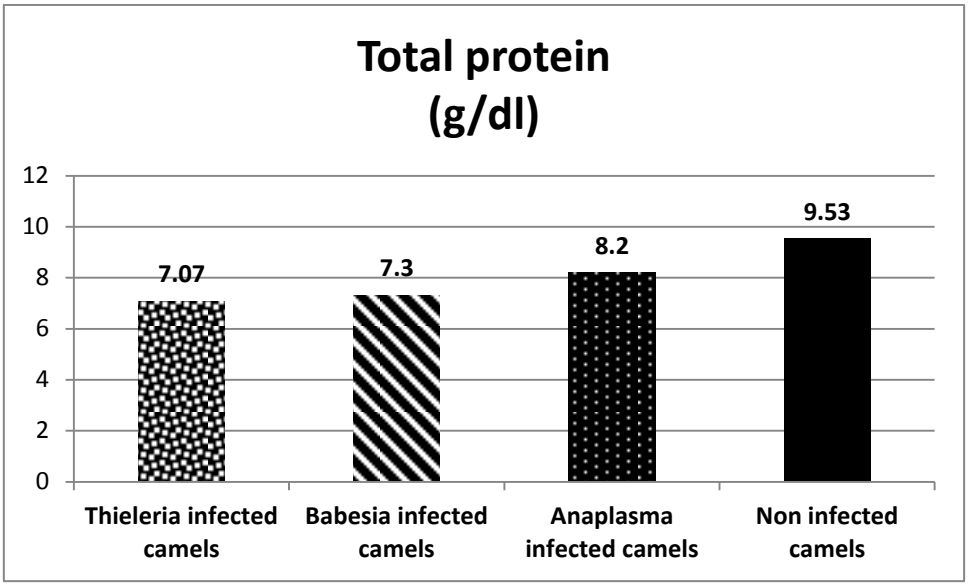


Fig. (27): Level of total protein (mg/dl) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

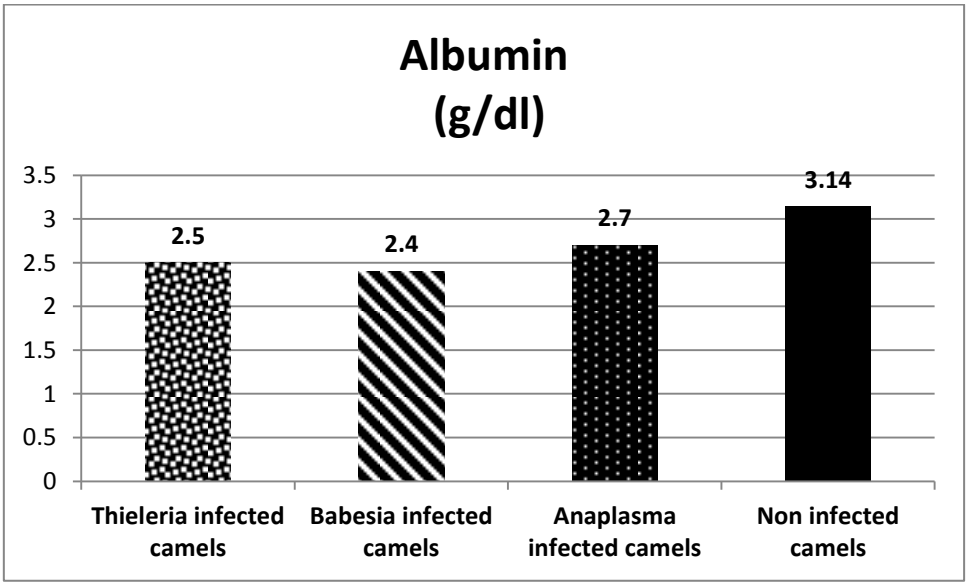


Fig. (28): Level of albumin (mg/dl) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

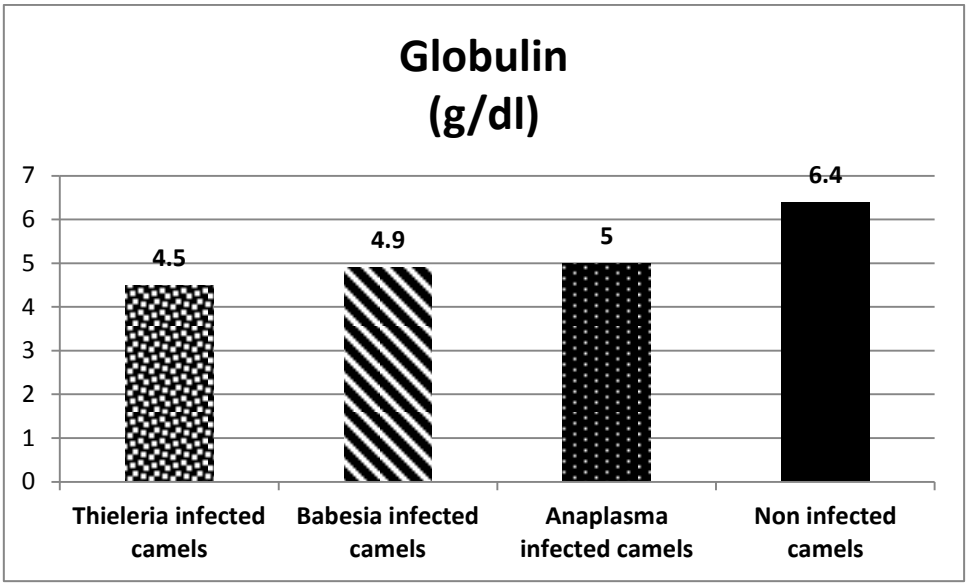


Fig. (29): Level of globulin (mg/dl) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

2- Kidney Function Tests:

Level of urea was significant increased ($P<0.05$) in the Thieleria infected camels, Babesia infected camels and Anaplasma infected camels when compared to non-infected camels. Levels of the creatinine exhibited non-significant changes in all infected camels, when compared to non-infected camels **(Table 6, Figs. 30 & 31)**

Table (6): Level of kidney function tests of Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

Groups	Kidney Function Tests	
	Urea (gm/dl)	**Creatinine (gm/dl)**
Thieleria infected camels	63.4±2.6*	1±0.2
Babesia infected camels	49.3±2.0 *	1.3±0.2
Anaplasma infected camels	46.4 ±1.0*	0.98±0.2
Non infected camels	25±1.5	1.1±0.0

*→ is referring to significant changes in comparison with non-infected camels when P<0.05 %.

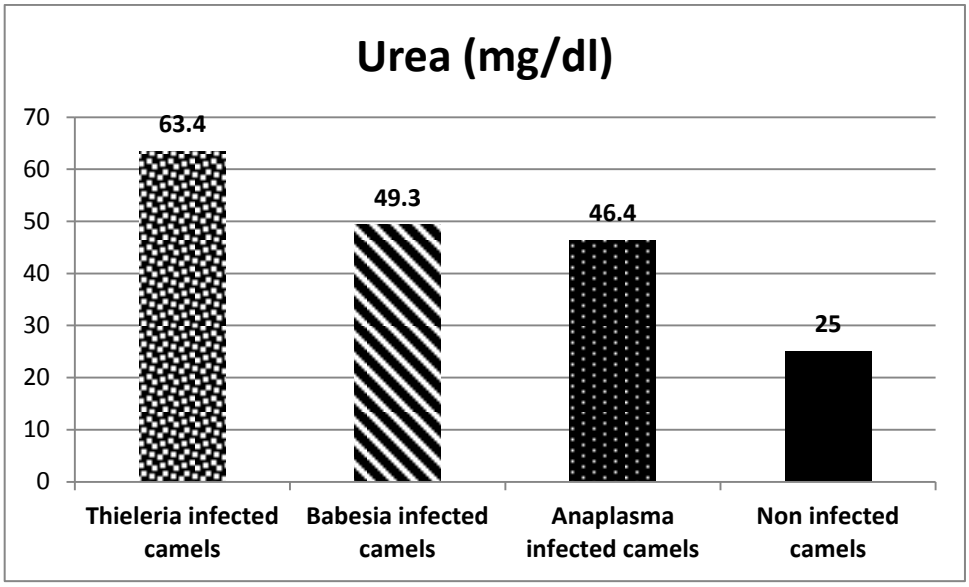


Fig. (30): Level of Urea (gm/dl) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

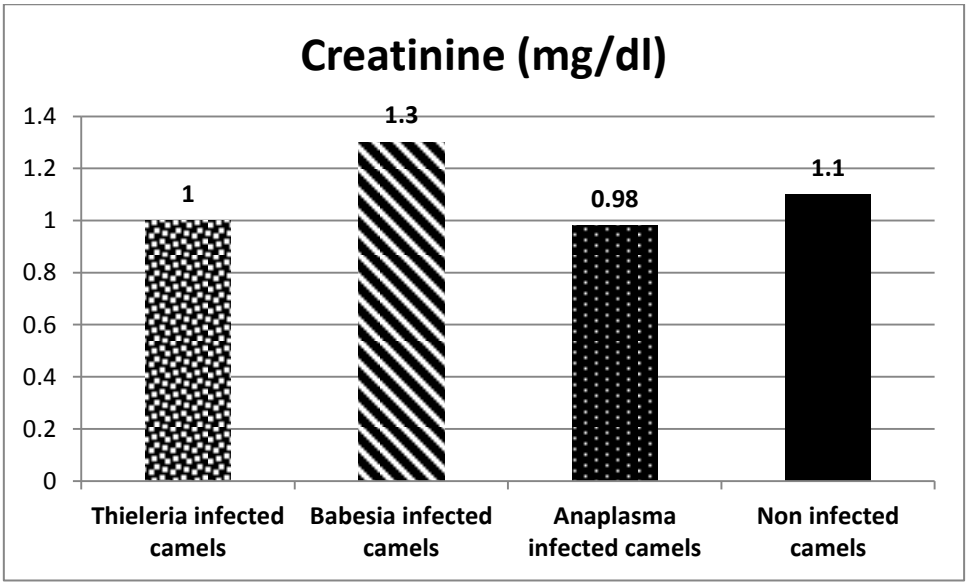


Fig. (31): Level of Creatinine (gm/dl) of the Thieleria infected camels, Babesia infected camels, Anaplasma infected camels and non-infected camels.

Histopathological results:

Pathological examination of liver of Theileria infected camel revealed diffuse vacuolar degeneration at hepatic parenchyma **(Figs. 32&33).** The Lymph nodes showed necrosis at the lymphoid follicle **(Figs.34, 35).** There was multinucleated giant target cell with multiple prominent nucleoli. Some lymphocytes contain basophilic parasitic oraganisms (koches blue bodies) **(Figs. 36, 37, 38)**.

In Anaplasma infected camel; the liver showed vacoular degeneration and portal fibrosis **(Figs. 39, 40, 41)**. In the lymph nodes showed capsular fibrosis as well as necrosis in lymphoid tissues **(Figs. 42, 43)**.

In Babesia infected camel, the liver showed cellular degenerationan portal cirrhosis **(Figs.44, 45, 46)**. The lymph nodes revealed hyperplasia in lymphoid follicle and lymph adenitis in some cases **(Figs. 47), while** depletion, edema, necrosis and heamorrhge were reported in other cases **(Figs. 48, 49)**, besides severe necrosis **(Figs. 50&51)**.

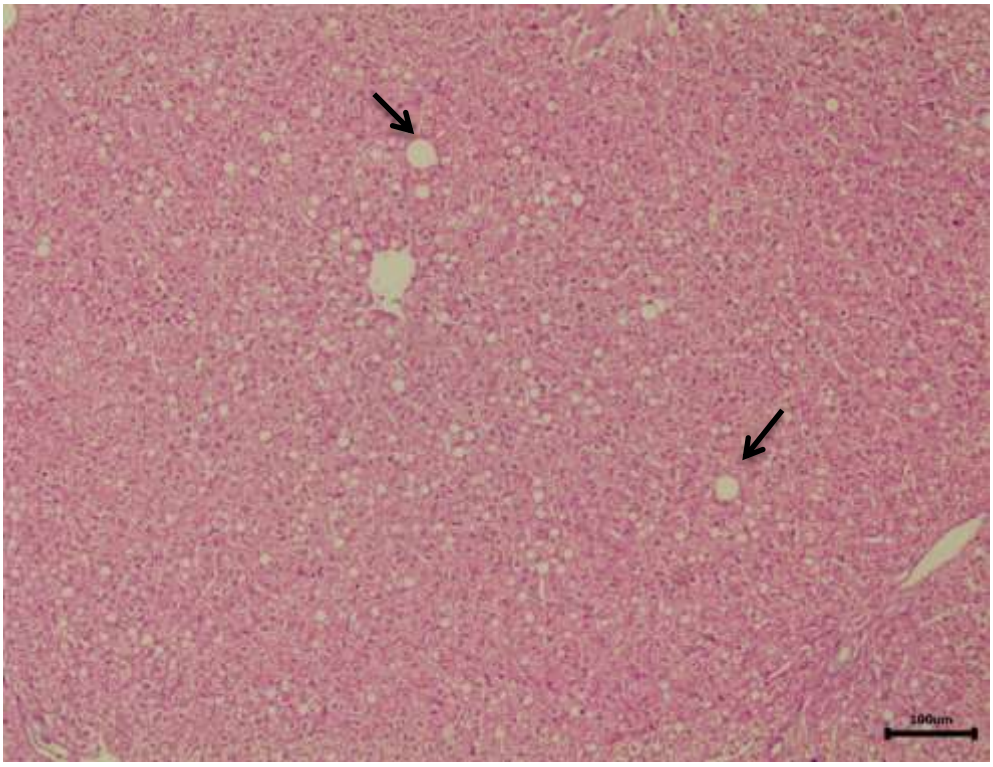


Figure (32): Photomicrographe from liver of Thieleria infected camels showing diffuse vacuolar degeneration at hepatocytes. **H&E stain. (bar=100 µm)**.

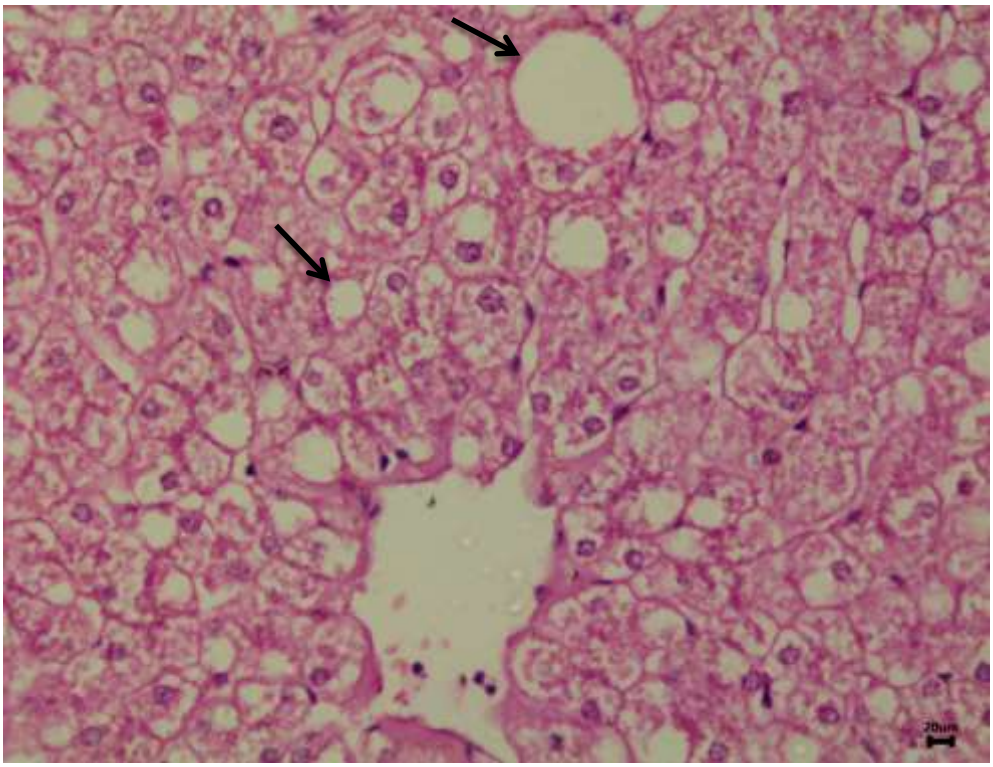


Figure (33) High power from previous figure showing distinct small and large vacoules at the hepatocyte **H&E stain.** (**bar=20 µm)**.

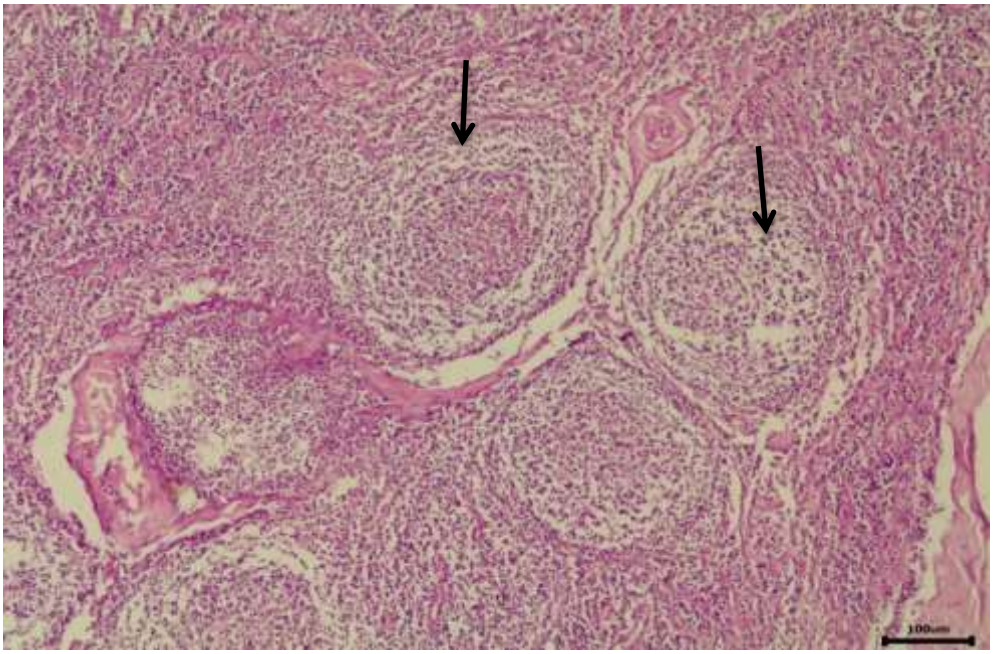


Figure (34) Photo micrographe from lymph node of Thieleria infected camel shawing depletation of the lymphoid elements at the lymphoid follicle **H&E stain.** (**bar=100 µm)**.

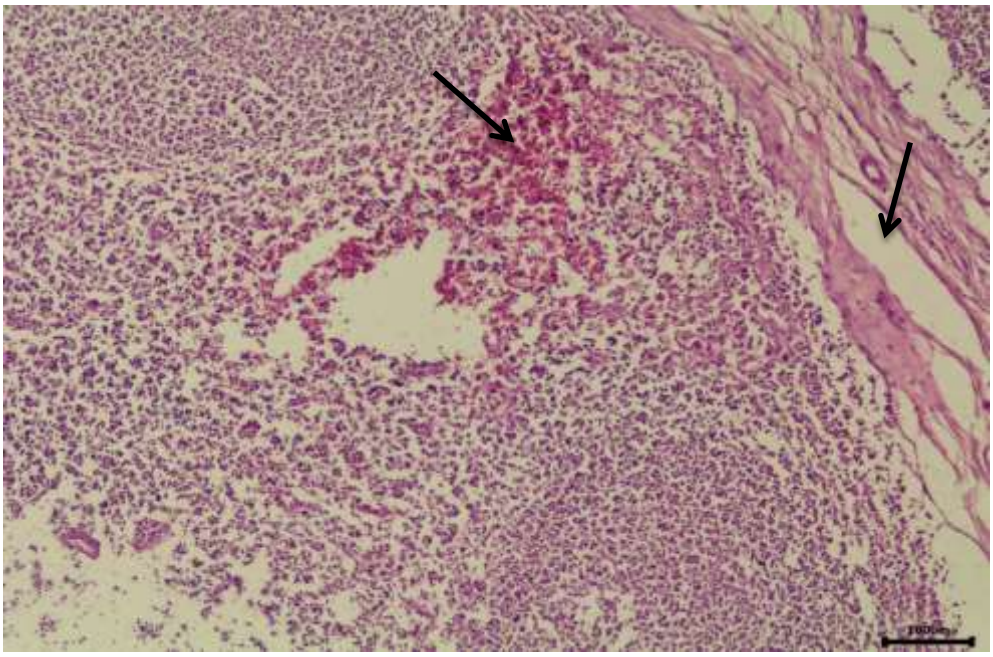


Figure (35) Photo micrographe from lymph node of Thieleria infected camel shawing sever necrosis at the lymphoid follicle and edema at the capsule. **H&E stain.** (**bar=100 µm)**.

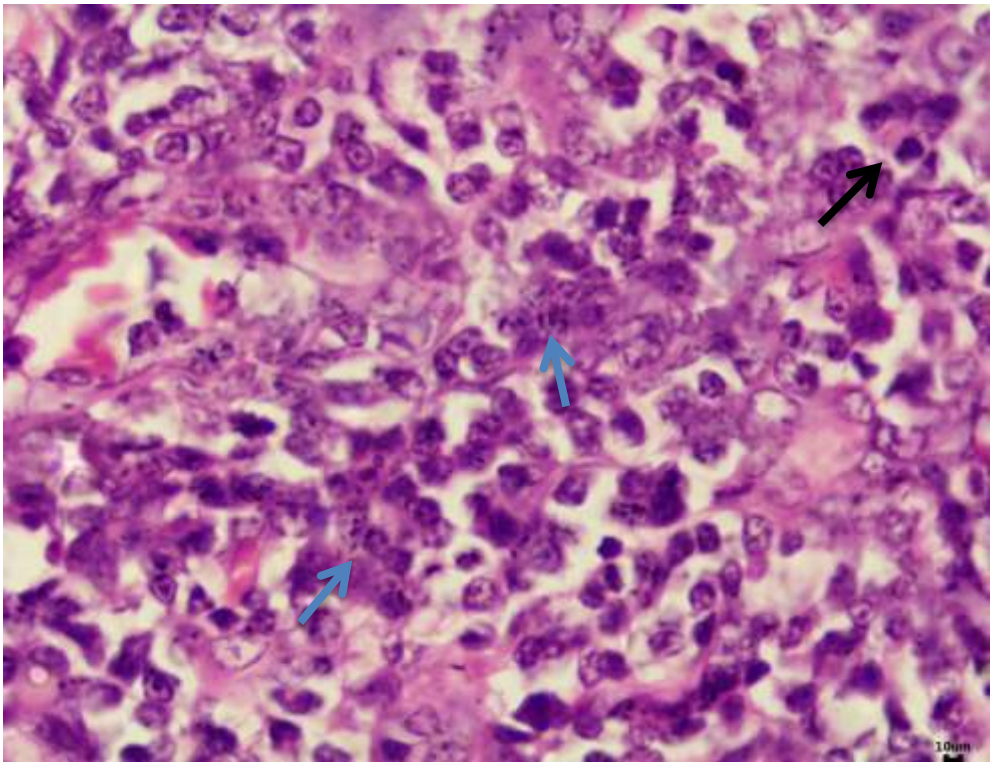


Figure (36) Photo micrographe from lymph node of Thieleria infected camel showing multinucleated giant target cell with multiple prominent nucleoli (black arrow). Some lymphocytes contain basophilic parasitic oraganisms (koches blue bodies) (blue arrow) **H&E stain (bar=10 µm)**.

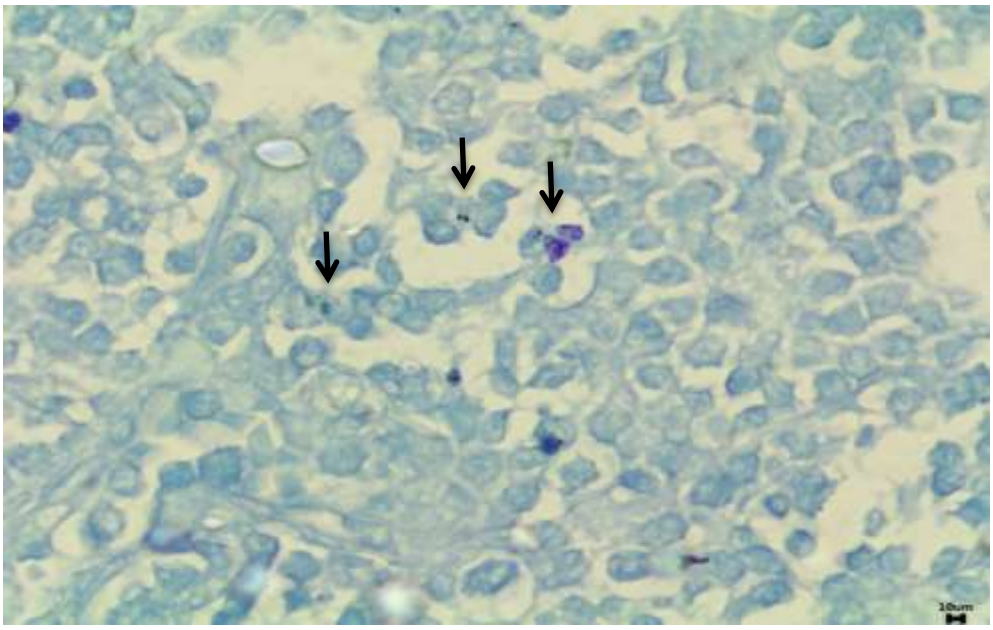


Figure (37) Giemsa-stained section from lymph node of Thieleria infected camel showing koches blue bodies at lymphocyte **(X1000, bar 10 µm).**

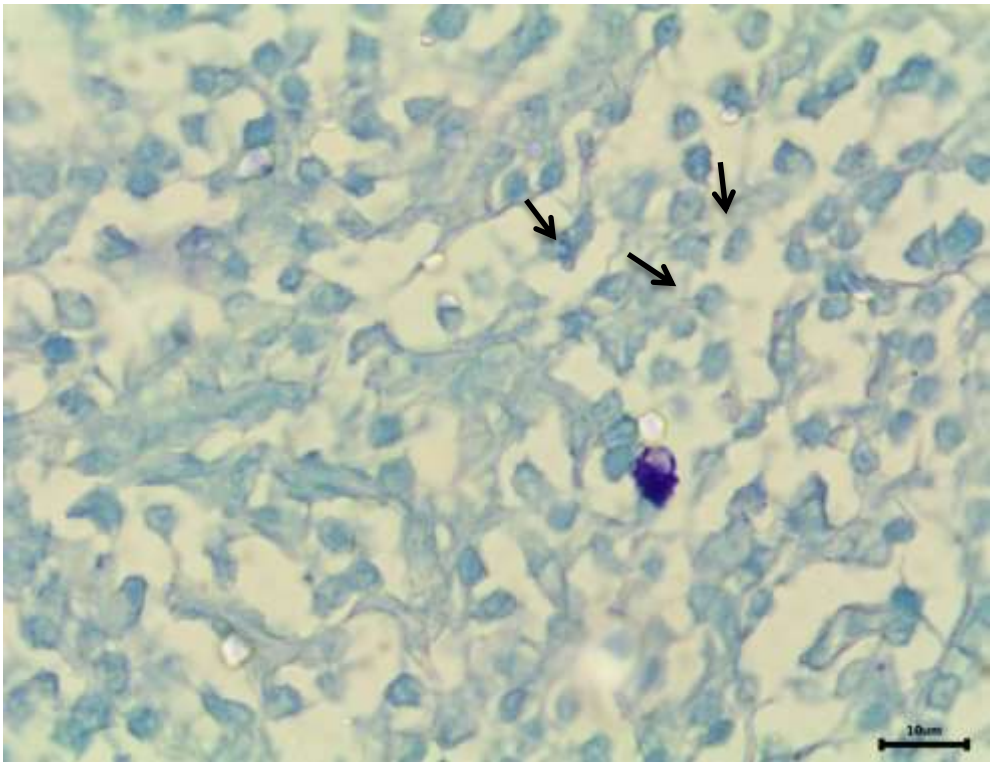


Figure (38): Giemsa-stained section from lymph node of thieleria infected camel showing koches blue bodies at lymphocyte **(X1000, bar 10 µm).**

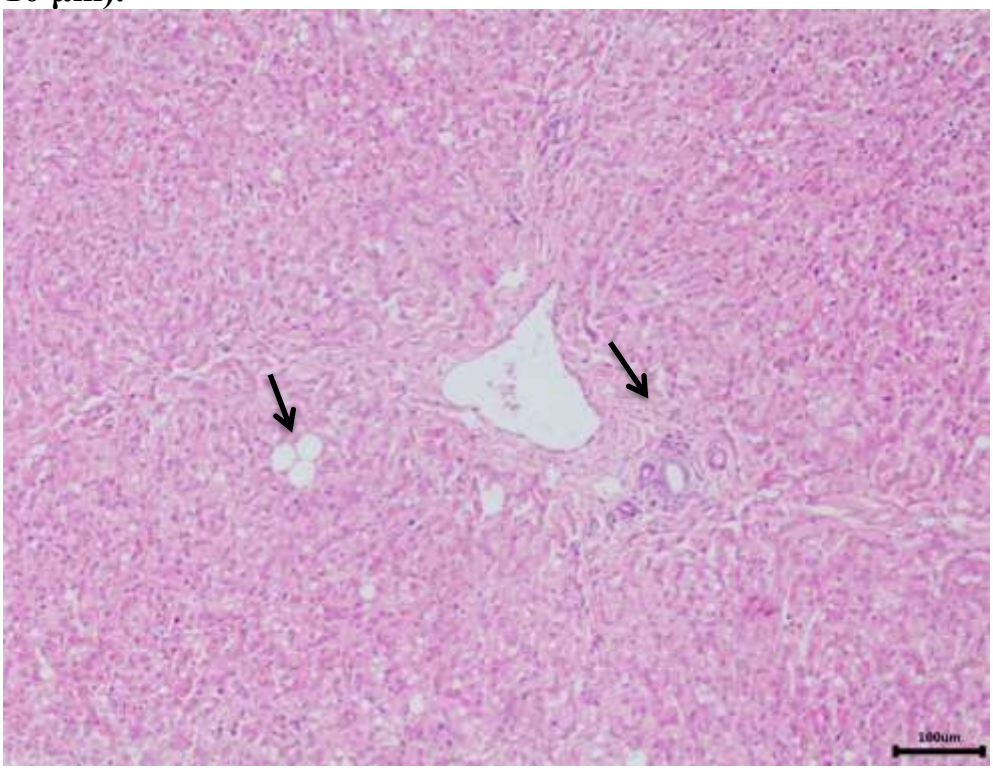


Figure (39): Photo micrographe from liver of Anaplasma infected camel showing vacuolar degeneration and portal fibrosis in liver **H&E stain. (bar=100 µm)**.

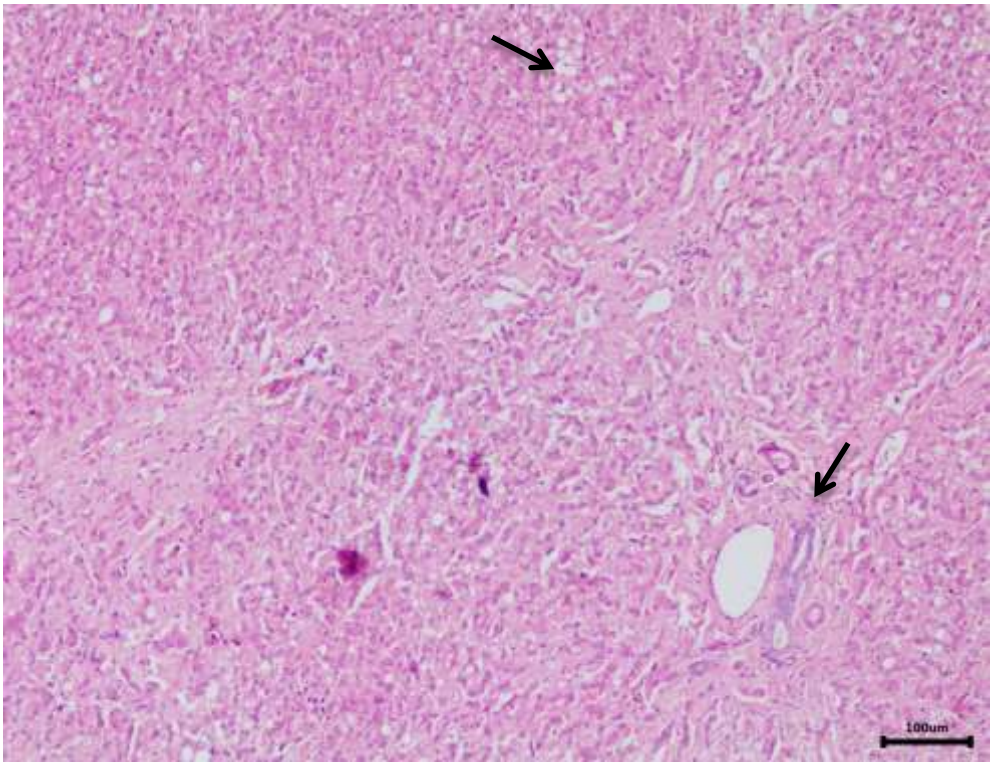


Figure (40): Photo micrographe from liver of Anaplasma infected camel showing vacuolar degeneration and massive portal fibrosis in liver **H&E stain. (bar=100 µm)**.

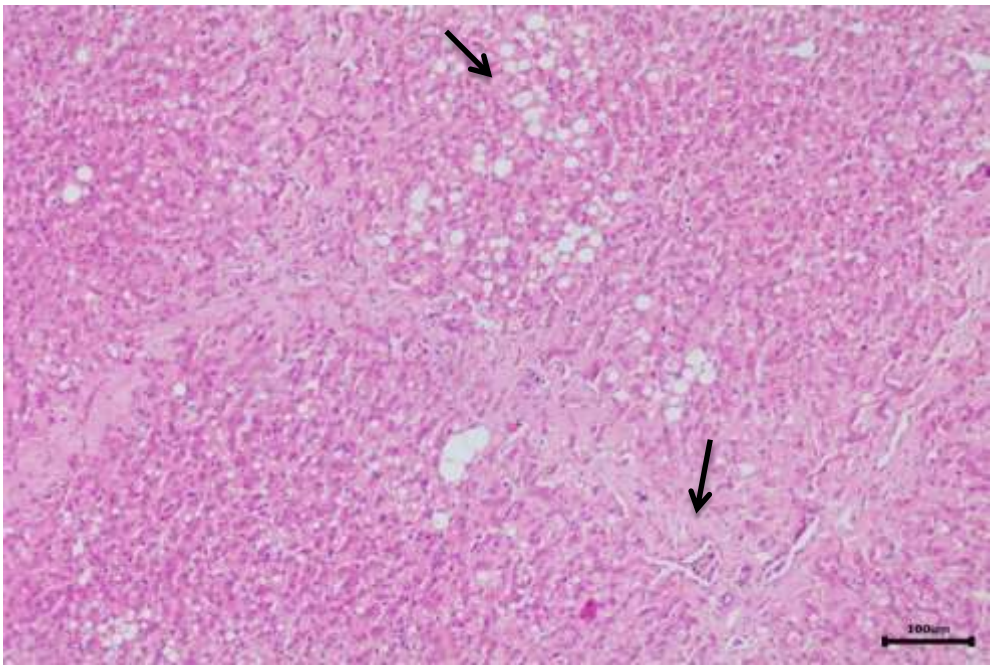


Figure (41) Photo micrographe from liver of Anaplasma infected camel showing vacuolar degeneration and portal cirrhosis in liver **H&E stain. (bar=100 µm)**.

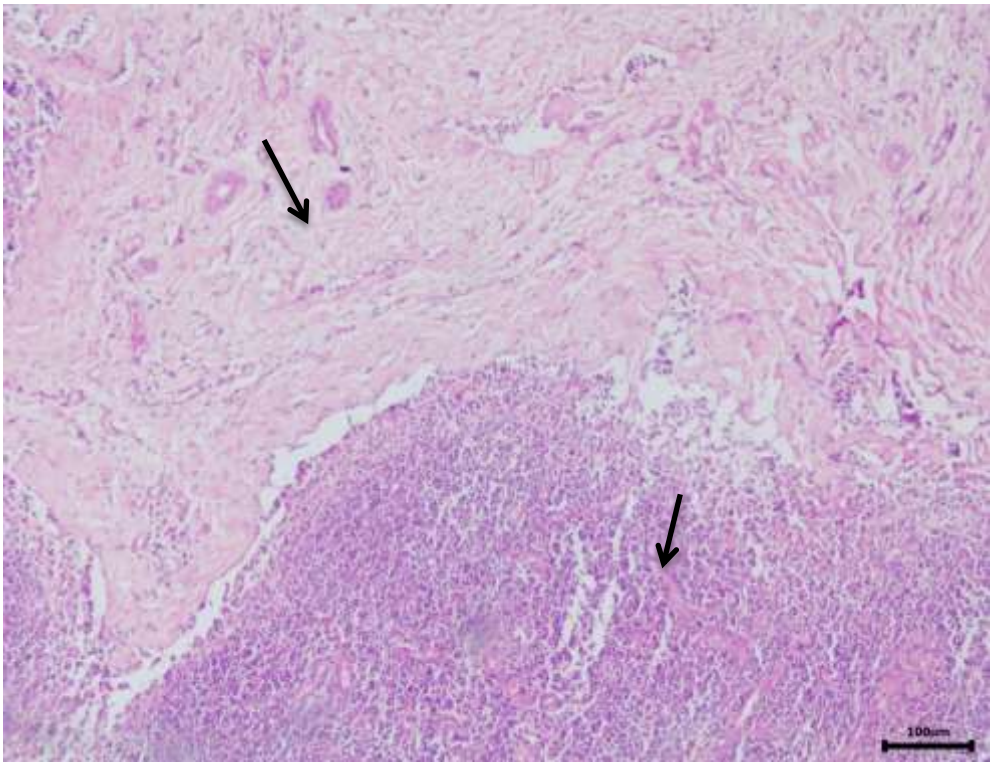


Figure (42): Photo micrographe from lymph node of Anaplasma infected camel shawing sever necrosis at the lymphoid follicle and thick fibrosis at the capsule. **H&E stain.** (**bar=100 µm)**.

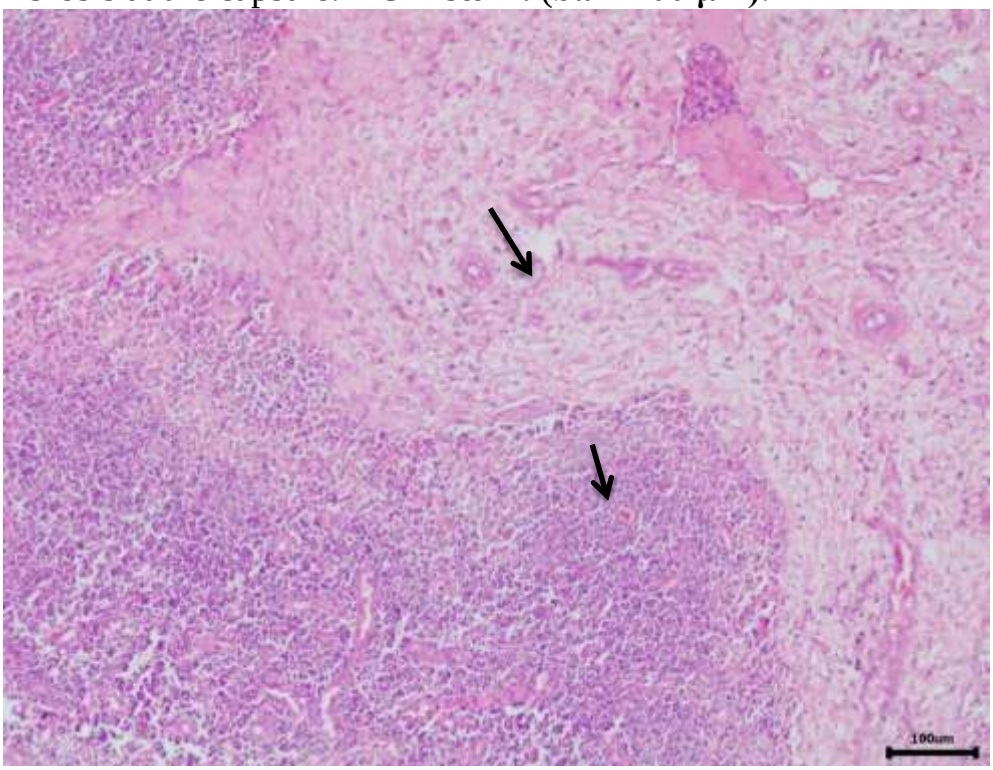


Figure (43): Photo micrographe from lymph node of Anaplasma infected camel shawing sever necrosis at the lymphoid follicle and thick fibrosis at the capsule. **H&E stain.** (**bar=100 µm)**.

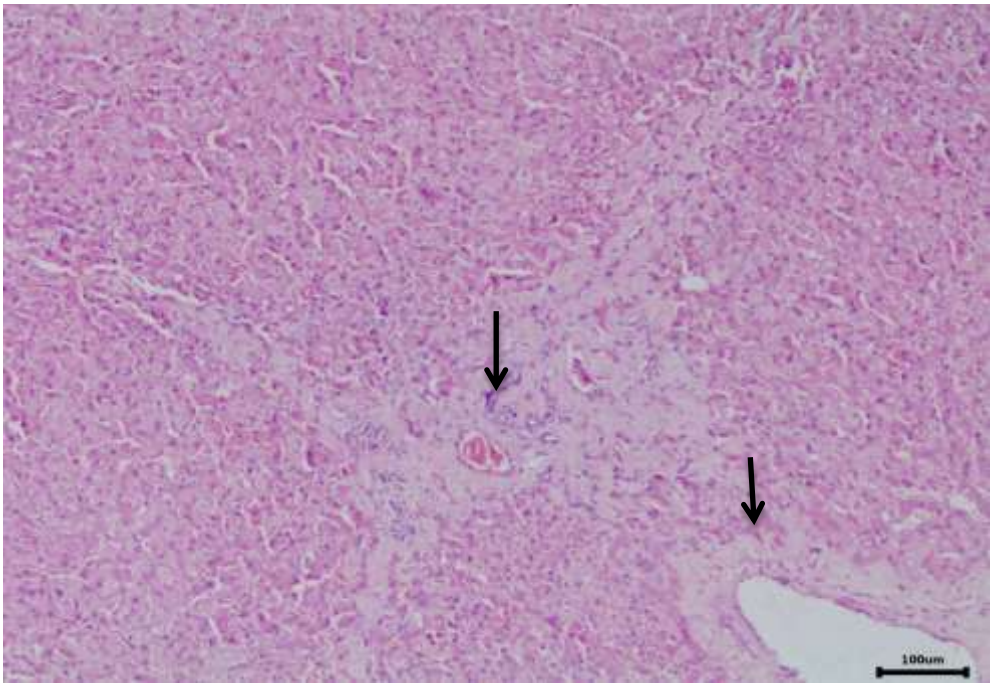


Figure (44): Photomicrographe from liver of Babesia infected camel showing portal cirrhosis in liver **H&E stain. (bar=100 µm)**.

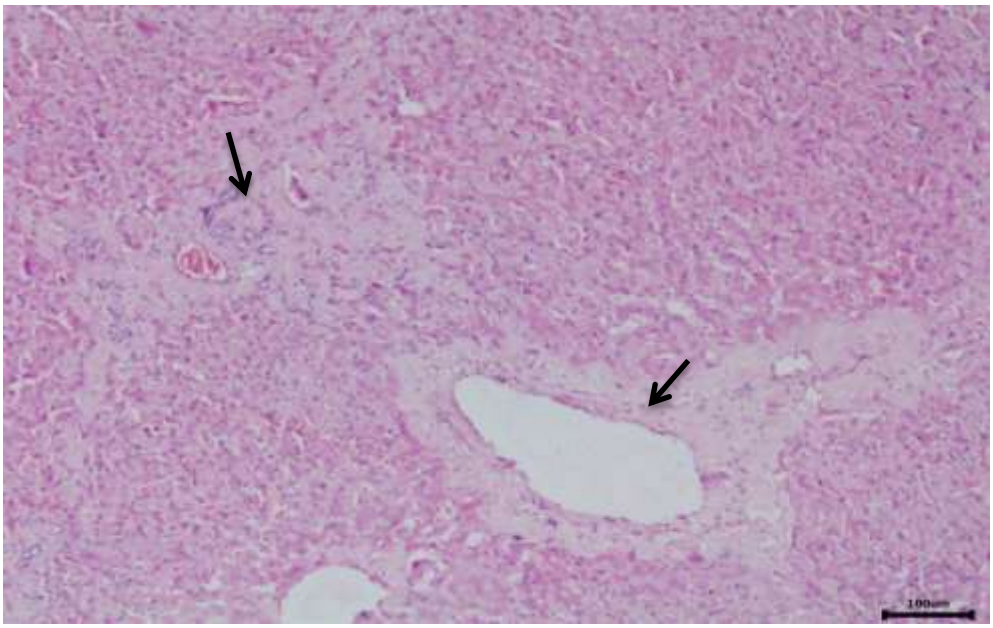


Figure (45): Photomicrographe from liver of Babesia infected camel showing severe portal cirrhosis in liver **H&E stain. (bar=100 µm)**.

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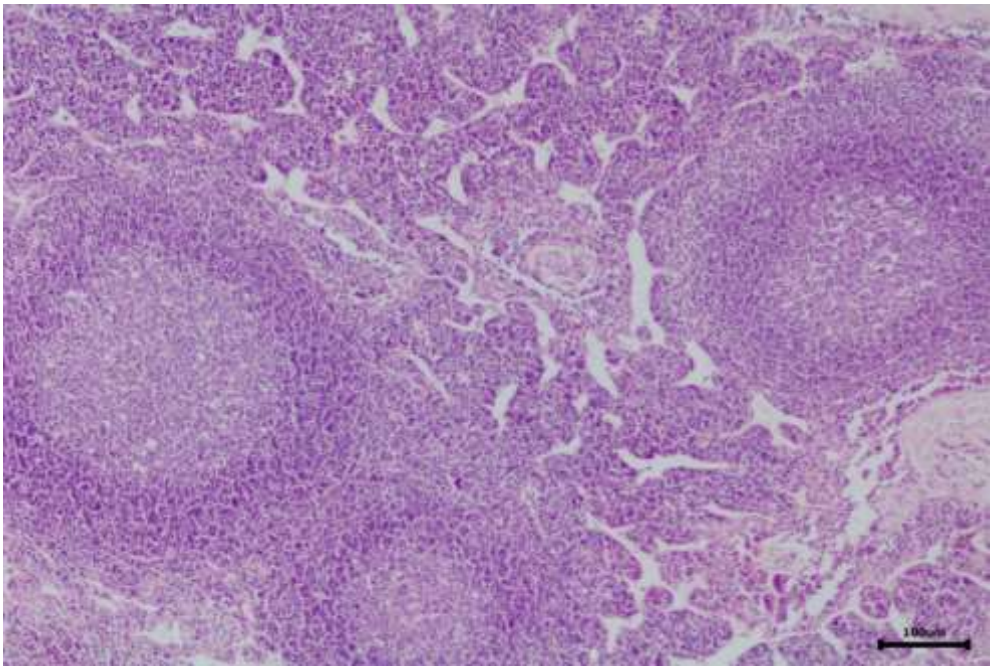


Figure (46): Photo micrograph from lymph node of Babesia infected camel showing hyperplasia in lymphoid follicle **stain H&E. (bar=100 µm)**

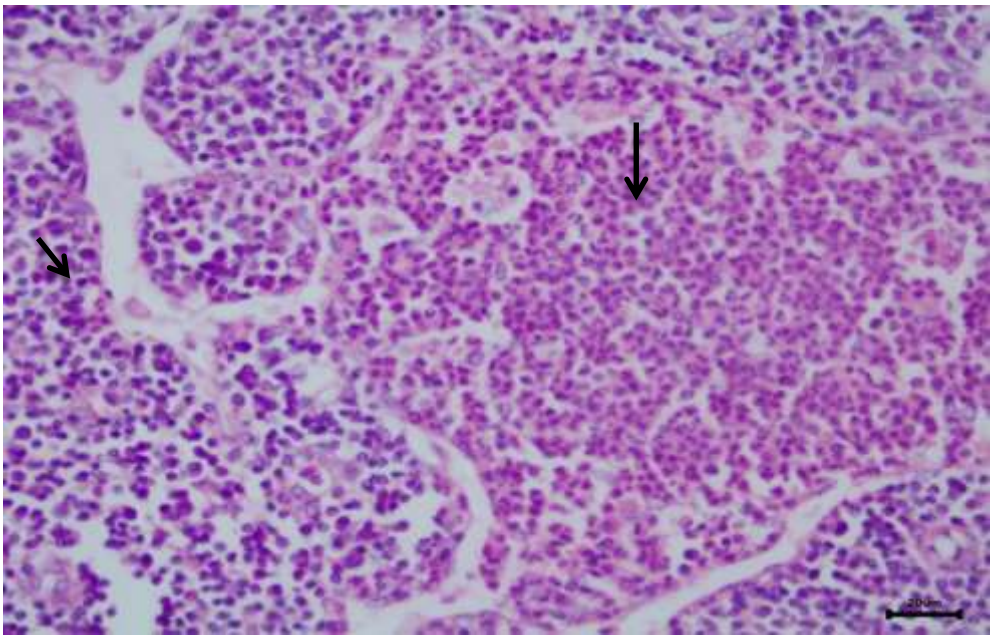
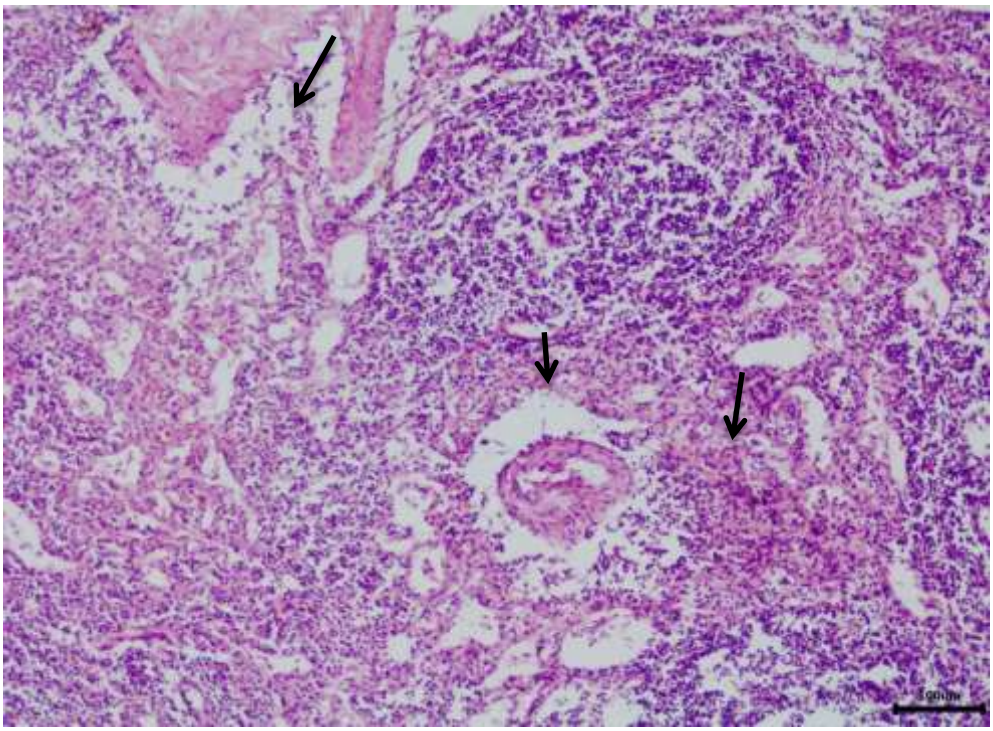
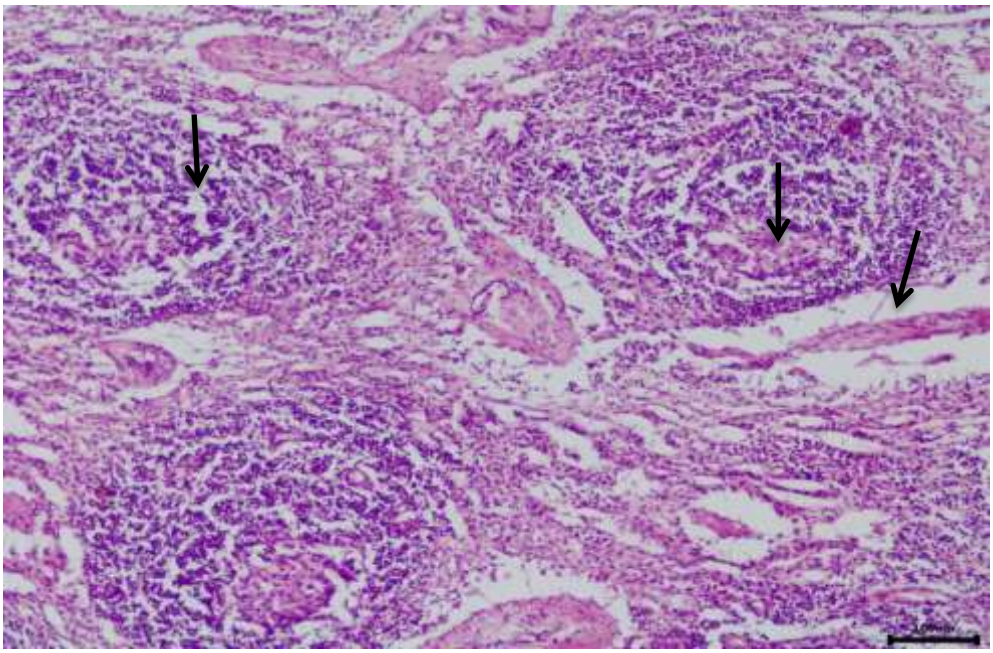


Figure (47): Photo micrograph from lymph node of Babesia infected camel showing lymphadenitis with neutrophelic cellular infilteration **H&E stain (bar=20 µm)**.



Figure(48) Photo micrograph from lymph node of Babesia infected camel showing lymphoid depletion, odema and necrosis lymphoid follicle **stain H&E., bar=100 µm)**.



Figure(49) Photo micrograph from lymph node of Babesia infected camel showing lymphoid depletation, odema and necrosis lymphoid follicle **stain H&E., bar=100 µm)**.

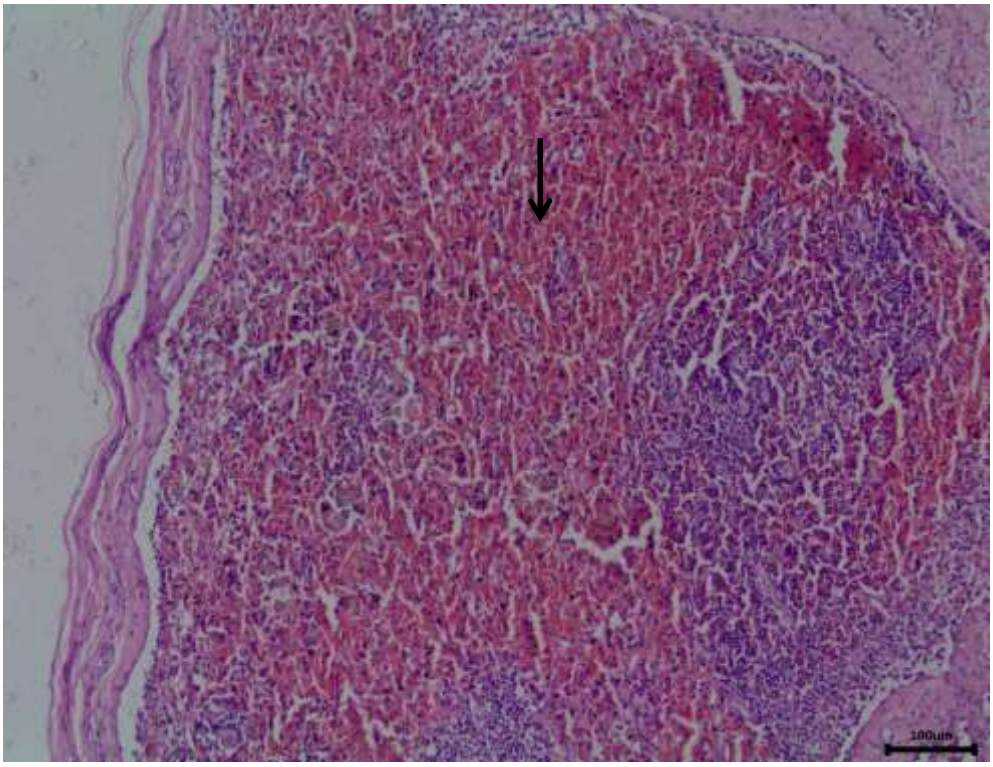
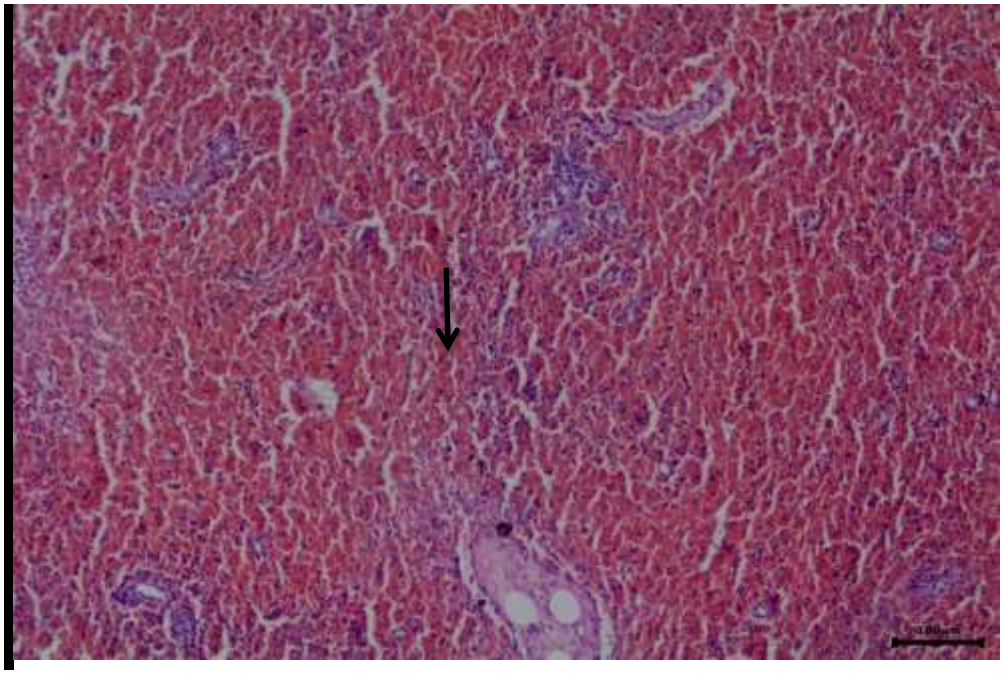


Figure (50) Photo micrograph from lymph node of babesia infected camel showing sever necrosis and hemorrhage in lymphoid tissues.**stain H&E., bar=100 µm)**.



Figure(51) Photo micrograph from lymph node of babesia infected camel showing showing sever heamorrhage in lymphe node **stain H&E., bar=100 µm)**.

DISCUSSION

Camels are an important source of milk, wool and meat; however, they are also used for transport and might also be considered as draught animals (**Kamani et al., 2008).** Camels are hardy animals and can tolerate the harsh conditions of arid regions; these animals face a wide variety of diseases. Large numbers of ticks are often found on camels **(Hamed et al., 2011)**.

In camels infected with haemoparasites suggested the presence of anaemia due to decrease in haemoglobin concentration and total erythrocytes count, which occurred because of lysis of erythrocytes and elimination of infected red cells by the reticuloendothelial system (**Maharan, 2004**). Furthermore, irregular heartbeats might be indicative of the first step of cardiac problems. Arrhythmia is defined as an irregular heartbeat, when the heart beats too fast, too slowly, or irregularly, and when the electrical signals to the heart that coordinate heartbeats are not functioning normally (**Tharwat et al., 2013**). The presence of ticks infesting different parts of the body on infected camels identified them as a significant biological transmitter of the disease (**Wernery and Kaaden, 2002**). The vital signs of infected camels were observed to change, since increased body temperature might suggest the liberation of pyrogens due to lysis of body cells followed by stimulation of thermoregulatory centres for fever crises. Further, anemia will decrease blood perfusion, therefore tissue hypoxia will occur, thereby resulting in anaerobic metabolism of decreased perfusion, and respiratory rate will be increased (**Maharan, 2004**). Higher heart rate in infected camels, which could also be the result of hypoxia (anaemic hypoxia), caused by the decreased erythrocyte count and haemoglobin concentration which affects oxygen transport to body tissues, leading to tissue deprivation of adequate oxygen supply (**Constable et al., 2017**).

Hematology has been widely used in attempts to provide information about disease states, performance problems and fitness in animals. A deviation

of certain blood parameters from their normal limits might serve a guide for diagnosis or for the differential diagnosis of a disease condition **(Mal et al., 2001).**

The present study was carried on 100 of camels obtained from Daraw and Aswan slaughter houses, belonging to Aswan Governorate during period from October 2019 to September 2020. The survey camel appeared fit subclinically without any symptoms. Our Study revealed presence of three different haemoparasites includes,*Thieleria*, *Anaplasma* and *Babesia* .

Theileriosis is an important hemoparasitic disease of animals inducing a variety of clinical manifestations ranging from a subclinical presentation to a fatal disease **(Mukhebi et al., 1992)**.The genus *Theileria* is distinguished by infection of leukocytes by sporozoites, maturation of schizonts into merozoites and subsequent infection of red blood cells to form piroplasms **(Uilenberg, 2006)**.

In our study prevalence of theileriosis displayed 15% in asymptomatic carrier camels , this result similar to **Hekmatimoghaddam et al.,(2012**) examined 114 apparently healthy camels and found that 15.79% were infected with Theileria spp In Iran, In contrast, in Egypt**, Nassar, (1992**) examined 200 apparently healthy camels and found that 30% were infected with Theileria spp. In another study in Upper Egypt, **Hamed et al., (2011)** reported that only 6.75% of the examined camels were harboring the erythrocytic forms of Theileria camelensis. There was Varied prevalence of Theileria infection in camels in Egypt was previously reported; as **Salem and El Olemy, (2017)** who found that 32.47% of the studied camels were infected with T. annulata (25 out of 77)**,** also **El-Refaii et al., (1998)** recorded that 33.3% of studed camels infected with Theileria in Cairo, while **El-Fayoumy et al., (2005**) mention that

44.8%of surveyed camels infected with thieleria in North Coast, incontraste**,** **Hamed et al., (2011)** recorded 6.75% of Thieleria infected camels in Upper Egypt, also **Abd-Elmaleck et al., (2014)** reported 9.18% in Assiut, moreover, **Youssef et al., (2015)** said that 30.86% of studded camels were infected with thieleria in Giza, Additionally **Osman et al., (2015)** reported that, 9% of examined camels infected with thieleria in NewValley, and **Abou El-Naga and Barghash, (2016)** recorded that 50.8% from studded camels were infected with thieleria in Matrouh **.**

In the present study, there were no clinical signs observed in all examined camels. this result agree with **Salem and El Olemy, (2017)** who examined a total of seventy seven apparently healthy dromedary camels (Camelus dromedarius), of both sexes, introduced to El-Bassateen slaughterhouse in Cairo, Egypt and revealed that microscopic and molecular prevalence of T. annulata were 32.47% and 40.26 %, respectively. Also, **Ibrahim et al., (2017**) showed about 75% of in the investigated camels were found to be positive for piroplasmosis .however, no pathognomic clinical signs of piroplasmosis. Additionlly **Hamed et al., (2011)** recorded that 15 camels infected with Theileria did not show any clinical signs**.** This means that we face problematic to cure these infected animals as they become carriers of the parasite and serve as reservoirs for transmission **(Abou El-Naga and Barghash, 2016).** The carrier-state for piroplasmida may be defined as the long term persistence of a parasite in its host, with the ability of transmission to other hosts via vector infection, to maintain a transmission cycle. Long-term persistence implies that the parasite can maintain itself and propagate in the vertebrate host while escaping the immune system. In most Theileria, it is accepted that the piroplasm stage is maintained in the host via asexual division and re-infection of red blood cells **(Norval et al., 1992).** These results may be attributed to the most benign Theileria species generally have higher parasitaemia ranges in the carrier state

that impacts on the ability to infect the tick vector and subsequent transmission to a new host. Parasitaemia is generally expressed as percentage infected red blood cells. During clinical reactions, piroplasm parasitaemia may be as high as 1–45% depending on the species **Uilenberg and Schreuder, 1976; Young et al., 1978; Waal, 1992; Latif et al., 2002; Schetters et al., 2010**, but this generally drops well below 1% for the asymptomatic carrier state (**Uilenberg and Schreuder, 1976; Young et al., 1986; Grootenhuis et al., 1987; Zaeemi et al., 2011; Ueti et al., 2012**). These ranges will be determined by the specific biology of the parasite in the carrier state, i.e. ability to maintain and propagate in the vertebrate host **(Mans et al., 2015).** However, it has been proposed that a reservoir of schizonts that maintains parasitaemia levels exists that escapes the immune system, especially since schizonts sampled 657 days after initial infection could be propagated in cell culture **(Grootenhuis et al., 1987)**. Maintenance of parasitaemia levels is necessary, since red blood cells have a lifespan that range from 70 to 160 days in large mammals before being destroyed (**Adili and Melizi, 2014**). This would imply that parasites need to replicate and reinfect new red blood cells at least once in this period to maintain their parasitaemia levels. In this regard, remarkable stability in piroplasm parasitaemia levels has been observed in T. parva carrier buffalo kept under tick-free conditions where animals maintained parasitaemia levels for up to 20 years (**Pienaar et al., 2014**).

Anaplasmosis is an arthropod borne disease of ruminants caused by species of the genus Anaplasma (Rickettsiales: Anaplasmataceae) (**Kocan et al., 2004**). Of the known Anaplasma spp., A. marginale is the most virulent, characterized by a progressive hemolytic anemia, and is responsible for extensive economic losses in tropical and subtropical areas (**Wernery et al., 2002; Hairgrove etal., 2015**).On the other hand, A. centrale is capable of

producing a moderate degree of anemia, but clinical outbreaks in the field are extremely rare **(Carelli et al., 2008)**.

In our study prevalence of Anaplasmosis displayed 10% in carrier camels. Anaplasma was detected in two forms belonging to A. marginale and A. central these results were lower than those reported by **Sudan et al., (2014**) as he recorded that 83.8% of studded camels infected with anaplasma sp, and also **Rabana et al., (2011)** who mentioned that, 95.5% of examined camels infected with anaplasmosis. Moreover, **Abdelrahim et al., (2009)** showed that, the prevalence of hemoparasites in camels was 21.5%, *Anaplasma* sp. was the common hemoparasites seen in examined camels in Nigeria, while **Abou El-Naga and Barghash, (2016**) recorded that, 47.4% of examined camels were harbored anaplasmosis in Egypt. In our study asymptomatic anaplasma infected camels was considered as acarrier animals. This result agree with **Sudan et al., (2014**) who recorded sub clinical Anaplasma marginalis infection , with a clinical history of dullness, progressive loss of condition. This result may due to A. marginale develops persistant infections in mammalian and tick hosts ,both of wich serve as reservoirs for infection of susceptible hosts. The only known site of replication of A. marginale in cattle is bovine erythrocytes **(Richey, 1981)**.within these cells membrane –bound inclusion bodies contain from 4-8 rickettsiae, and as many as70 or more of the erythrocytes may become infected cells by the bovine reticuloendothelial system results in mild to severe anemia and icterus **(Richey, 1981)**. Once cattle become infected with A. marginale, they remain persistently infected carriers for life, whether or not they develop clinical disease **(Richey, 1991).** Throughout the remainder of the persistently infected carrier's life, there are relatively uniform cycles over a 10- to 14-day period of increasing and decreasing numbers of circulating erythrocytes infected with A. marginale **(Kieser et al., 1990; Viseshakul et al., 2000).** Moreover, Persistent infection is characterized by cyclic rickettsemia ranging from 102 to 107

infected erythrocytes per mL of blood that occur at approximately five-week intervals **(Kuttler and Simpson, 1978; Stewart et al., 1979; Eriks et al., 1989;).** Although deaths may still occur, persistent infections usually confer resistance to clinical anaplasmosis **(Kocan et al., 2010)**. Persistently infected cattle exposed to mechanical and/or biological vectors serve as reservoirs of infection to introduce Anaplasma sp into populations of native cattle thereby leading to endemic disease stability **(Reeves and Swift, 1977; Echaide et al., 1998; Futse et al., 2003;)**. Furthermore, native cattle in non-endemic areas may become infected with A. marginale following the introduction of a carrier animal from an endemic area **(Smith et al., 1989)**. A. marginale infection associated with contaminated surgical equipment or hypodermic needles may give rise to clinical cases occurring outside the normal vector season **(Reeves and Swift, 1977 and Smith et al., 1989).**

Babesiosis, caused by infection with intraerythrocytic parasites of the genus *Babesia*, is one of the most common infections of free-living animals worldwide and is gaining increasing interest as an emerging zoonosis in humans. Although capable of infecting a wide range of vertebrates, Babesial parasites require both a competent vertebrate and non-vertebrate host to maintain transmission cycles. All Babesial parasites described to date are transmitted by ticks to their vertebrate hosts. The parasites replicate in the vertebrate hosts' red blood cells and are called piroplasms due to their pear-shaped appearance within the infected host cells (**Kakoma and Mehlhorn, 1993 and Telford et al., 1993)**. All mammalian hosts examined have been able to develop immunity to *Babesia species*, either after an episode of infection and recovery or after prophylactic immunization. Both humoral and cellular factors are involved in immunity to Babesiosis. -Almost any mammal that serves as a host for a Babesia-infected tick is a potential reservoir (**Telford et al., 1993**).

In the present study, we recorded 5% of camels infected with Babesia. Few papers have reported *Babesia sp.* in camels; *B. caballi* was recorded for the first time in Sudanese camel **Abdelrahim et al., (2009)** and the infection of Camelus dromedaries by *Babesia* was recorded in Egypt **(Abd-Elmaleck et al., 2014).** Our result is lower than **Abou El-Naga and Barghash, (2016**), who recorded the prevalence of *babesia* in 11.8% from examined camel. Varied prevalence of babesia infection in camels was previously reported by **Swelum et al., (2014**) who recorded that *Babesia* infection rate of 13.2% in camels in Saudi Arabia. On the other hand, more recent study in Iran indicated 6.56% of camels were positive for *Babesia spp.* **(Khamesipour et al., 2015)**. While in Pakistan **Chaudhry et al., (2010**) recorded that 29% of studded cattle infected with *Babesia.*

In our study asymptomatic babesia infected camels was considered as carrier. This result agree with **Homer et al., (2000)** recorded that many *Babesia species* infect many vertebrates without any apparent disease manifestations. This means that we face problematic to cure these infected animals as they become carriers of the parasite and serve as reservoirs for transmission; in particular B. bovis is more dangerous than B. bigemina because it is less sensitive to some babesiacidal compounds **(Chaudhry et al .,2010).** also agree with **Kirupananthan et al.,(2016)** they record 30 blood samples were collected from apparently healthy cattle suspected to be carrier cattle and analyzed using light microscopy indicated that 47% of the samples to be positive babesiosis . Moreover, immune animals display an already developed antibabesial immune response upon encountering a new infectious challenge; these animals do not show the stage of rising parasitemia, and often no parasites can be detected in circulating blood. The existence of the chronic asymptomatic carrier state in Babesial infections of domestic and wild animals has been recognized for many

years (**Lykins etal., 1975; Ruebush et al., 1981; Conrad et al 1991; Figueroa et al., 1992; Telford et al., 1993).**

In our study These varied prevalence rates of heamoparasite (*Thieleria* (15**%**), *Anaplasma* (10**%**), and *Babesia* (5**%**) in addition to negative *Trypanosoma* results in the examined camels might be due to the different localities, environments, tick control programs, methods of selecting the studied animals, endemicity as well as the chronicity of infections.

In our study the prevalence rate of heamoparasite (*Thieleria, Anaplasma*, and *Babesia*) infection to be the most frequently in autumn season (16**%)**, followed by summer (8**%**), then winter (5**%**) and at the lowest rate during spring (1**%)**. This result agree with **Lempereur et al., 2012; Asmaa et al., 2014** who mention that, *Theileria spp., Babesia spp.,* and *Anaplasma spp.* infections are increased with the increase of tick infestation during the rainy season. Also agree with **Hussam et al., (2022)** who showed that, the infection with *Theileria spp., Babesia spp.* increased during autumn .Additionally, **Farhan and hamed, (2017)** found that *Babesia* and *Theileria* infection were more frequently in rainy season than dry with significant variation and this due to abundance of arthropod vector (tick) that play active role in transmission of these protozoa, especially during the rainy season which serves as favorable breeding season of arthropod**.** Moreover, Seasonality of blood parasite infection in animals is evident as a function of the seasonal prevalence of the vectors **(Constable et al., 2017)**. On the other hand, our result disagrees with **Lempereur et al., 2012; Asmaa et al., 2014** recorded that *Theileria spp., Babesia spp.,* and *Anaplasma spp.* infections are been the lowest rate during winter**.** Also **Hussam et al., (2022)** showed that the infections with Theileria spp., Babesia spp. were increased during spring.

In our study Giemsa stain blood films showing thieleria infected Camels with the intra-erythrocytic trophozoit signet ring and cocci, shaped piroplasms

as well as the intra lymphocytic schizogonies "Koch's blue bodies" with presence of dacrocyte cell, macrocytic and hypochromic erythrocytes. Similar results with those were obtained by **(Youssef et al., 2015 and Salem and El Olemy 2017)**. Howelljolly bodies and macrocytes were detected also in camels infected with Theileria in Assuit, Egypt **(Abd-Elmaleck et al., 2014)**. Also it is agree with **Razavi et al., (2012)** who recorded macrocytic hypochromic anemia in camels naturally infected with T. annulata. It is disagree with **Ismael et al., 2014**; **Osman et al., 2015; Ayadi et al., 2017** who recorded that Theileria annulata infection induced microcytic hypochromic. Similar results also obtained by **Durrani et al., (2006)** in Friesian cattle infected with T. annulata.

In our study Giemsa stain blood films revealed Anaplasma within erythrocytes of camel and presence of hawell jolly body and macrocytic hypochromic erythrocytes. This result is agrees with **Alsaad et al., (2009)** show anaplasma marginale appear as spherical in periphery of infected red cells with macrocytic RBCS.

In our study Giemsa stain blood films Showing presence of *Babesia* in RBCS of camels and presence of eccentric hypochromic erythrocyte .Similar result was obtained by **(Swelum et al ., 2014).**

In our study, the morphological erythrocyte characteristics of blood films from *Thieleria, Anaplasma* and *Babesia* infected camel revealed macrocytosis, poikilocytosis, anisocytosis, hypochromacia. This attributed to a regenerative bone marrow response to anemia in these camels **(Razavi et al., 2012).** Moreover, Hemolytic anemia that occurs during acute stage is normocytic form and then become macrocytic with adequate reticulocytes **(Sulaiman et al., 2010).**

In our study there was significant decrease (P<0.05) in RBCs count, Hb. Concentration, and PCV % in Thieleria infected camels when compared with non-infected camels. This agree with the results were previously reported by**; Ismael et al., 2014; Osman et al., 2015; Youssef et al., 2015; Salem and El Olemy 2017** who recorded significant decrease (P<0.05) in RBCs count, Hb. Concentration, and PCV %. This result could be attributed to the extensive removal of parasite-damaged erythrocytes by phagocytosis in the reticulendothelial system **(Osman et al., 2015**). Morever **Boulter and Hall, (2000**) who mentioned that, the suppressing effect of tumor necrosis factor-α on the hematopoietic progenitors subsequently decreased erythrocyte production and survival

In our study There was significant increase in lymphocytes % (P<0.05) in Thieleria infected camels when compared with non-infected camels. This may attributed to hemoparasite-activated macrophages secreted proinflamatory cytokines such as interlukin-1 (IL-1), interlukin-12 (IL-12) and tumor necrosis factor (TNF) **(Hemmer et al., 2000)**. Theses proinflamatory components are considered important factors for activation of Lymphocytosis **(Shoda et al., 2000)**. This result dis agree with the results were previously reported by **Salem and El Olemy, (2017)** who showed insignificantly changed lymphocyte count, and **Youssef et al., (2015**) who recorded lymphopenia.

Also, in our study,there were significant increase (P<0.05) in eosinophil % in Theleria infected camels when compared with non-infected camels. The eosinophilia attributed to response to the antigen antibody reactions of parasitic infection **(Feldman et al., 2000)**. This agree with the results were previously reported by **Youssef et al., 2015; Salem and El Olemy 2017** who recorded eosinophilia in Theleria infected camels. In our study other parameters including neutrophil, basophil values showed non-significant changes when compared

with non-infected camels. This result agree with the observations were noted by **Osman et al., (2015)** who reported insignificant change in TLC, neutrophil, monocyte and, and also agree with **Youssef et al., (2015)** who recorded insignificant change in monocytes counts in Theleria infected camels when compared with non-infected camels. These results disagree with the results previously reported by **Youssef et al., 2015; Salem and Elolemy 2017** in the leukogram of T. annulata infected camels indicated significant leukocytosis, neutrophilia.

In our study there was significant decrease ($P<0.05$) in RBCs count, Hb. Concentration, and PCV % in Anaplasma infected camels when compared with non-infected camels. This result agree with **Al-Saad., (2009) and Azeem et al., (2019)** who reported significant reduction ($P<0.05$) in the mean of total RBC counts, hemoglobin concentration, and PCV in *Anaplasma*-positive in camel. This result also agreee with **Cruz et al., (2019)**who recorded significant reduction ($P<0.05$) in the mean of total RBC counts, hemoglobin concentration, and PCV in *Anaplasma*-positive in sheep .Such reduction attributed to intravascular erythrocytes hemolysis, erythrocyte phagocytosis by the reticuloendothelial system, and restricted erythropoiesis in the bone marrow. The decrease in red blood series elements is attributed to the short life span of red blood cells in the blood circulation due to extravascular erythrophagocytosis due to the presence of *Anaplasma* rickettsia in the circulation **(Massard et al., 1998)**. While other parameters in our study including WBC, count, MCV, MCH, and MCHC values showed non-significant changes in Anaplasma infected camels when compared with non-infected camels. This result dis agrees with **Azeem et al., (2019**) who showed a rise in TLC in Anaplasma infected camels. In our study there was significant increase ($P<0.05$) in eosinophil % in Anaplasma infected camels when compared with non-infected camels. While,

other parameters including Neutrophil, basophil values showed non-significant changes in compare with non-infected camels.

In our study There was significant decrease (P<0.05) in RBCs count, Hb. Concentration, and PCV % in Babesia infected camels when compared with non-infected camels. This result agree with **Ismael *et al*., (2014); Abd-Elmaleck *et al*., (2015) and Azeem et al., 2019** who recorded that Babesiosis positive camel samples show decline in PCV, RBCs, Hb.). Also, agree with the study was done by **Esmaeilnejad *et al*., (2012)** on haematological and biochemical parameters in small ruminants naturally infected with *Babesia ovis* also they mentioned same changes in PCV, RBCs, Hb due to bebesiosis in small ruminants. High pathogenicity with Babesia occurs when the RBCs, packed cell volume (PCV) and hemoglobin concentration of animals was decreased resulted in anemia (**Rubino et al., 2006)**.This changes was attributed to immunomediated phenomena by autoantibodies directed against component of membrane of infected and uninfected erythrocytes **(Rubino et al., 2006)**.Also production of toxic hemolytic factors of the parasite inducing erythrophagocytosis and release of kallikrein as vasoactive molecules **(Brockus and Andreasen, 2003).** B. bovis initiates cytoadhesion of infected RBC to capillaries, consequently destruction of RBC can adversely affect host organs **(Schnittger and Rodriguez, 2012).** Also anemia occurs due to mechanical damage and destruction of RBCs by the binary fission of trophozoites **(Zobba et al., 2008).** Babesia and Theileria parasites invade erythrocytes of infected animals subsequently induced destruction of parasitized erythrocytes **(Otsuka et al., 2002)**. While other parameters in our study including WBC, count, MCV, MCH, and MCHC values showed non-significant changes in Babesia infected camels when compared with non-infected camels .There was significant increase in lymphocytes % (P<0.05) at Babesia infected camels when compared with

non-infected camels. this result agree with **(Esmaeilnejad et al., 2012)**.This result attributed to hemoparasite-activated macrophages secreted proinflamatory cytokines such as interlukin-1 (IL-1), interlukin-12 (IL-12) and tumor necrosis factor (TNF) **(Hemmer et al., 2000)**. Theses proinflamatory components are considered important factors for activation of lymphocytosis (**Shoda et al., 2000)**.Also, In our study There was significant increase (P<0.05) in eosinophil % in Babesia infected camels when compared with non-infected camels The eosinophilia attributed to response to the antigen antibody reactions of parasitic infection **(Feldman et al., 2000)**.While, other parameters including Neutrophil, basophil values showed non-significant changes compared with non-infected camels **.**

In our study the level of AST revealed significant increases (P<0.05) in Thieleria, Babesia, and Anaplasma infected camels in compare with non-infected camels at P<0.05. While, ALT revealed significant increase (P<0.05) only in Anaplasma infected camels in comparison with non-infected camels.This result agrees with **Youssef et al., (2015)** who reported significant increase in serum AST activity in camels infected with theileriosis. Also, agree with **Ismael et al., (2014)** who reported highly significant rises in AST and ALT activities in thieleria infected camels and this result also agrees with **Swelum et al., (2014)** who recorded significant increase in AST level in Babesia-infected camels. In addition to**, Yfruham, et al., (1998)** who recorded that, hemoprotozoans caused relative significant increase (P<0.05) in the AST and ALT levels which was indicated to a possible damage to the liver, myocardium and kidney**,** and distraction of RBCs. The liver plays a central locale for babesiosis: it is known as the site where the preerythrocytic stages of *Babesia* and *Theileria* parasites

asexually multiply and where host immune mechanisms develop to get these pre-erythrocytic stages **(Cohen and Lambert, 1982)**.

Our results in *Anaplasma* infected camels agree with **Azeem et al., (2019)** recorded significant increase in ALT, AST in *Anaplasma* infected camels and decrease in albumin and total protein in case of Anaplasmosis. **Al-Saad, (2009)** also demonstrated increase in AST, ALT and decrease in total protein due to Anaplasmosis. **Allen et al., 1981; Coskun et al., 2012** also reported that Anaplasmosis induced elevation in serum AST and alkaline phosphatase (ALP) in cattle. While, This result disagree with **Salem and El Olemy, (2017)** record insignificant changes in ALT, AST in T. annulata-infected camels . In the present study, an increase in AST, ALP concentration was is attributed to hepatic dysfunction.The rise in activity of AST caused by muscular trauma as result from recumbency due to anaplasmosis **(Sandhu et al., 1998)**. After infection, parasitemia increases until the hemolytic crisis occurred in more than 50% of infected RBCs **(Allison and Meınkoth, 2010)**. The number of infected erythrocytes was basically increased; in consequence phagocytosis by reticulo-endothelial cells of parasitized erythrocytes leads to development of hemolytic anemia and icterus. Serum AST, ALT concentrations are common indicators of hepatic function. Hematological and biochemical modifications are the index of severity of disease (**Turgut, 2000)**. The biochemical adjustment reflected pathological changes in the liver and muscles infected with anaplasmosis in cattle **(Hornok et al., 2007)**. Also hemolytic anemia and hepatic dysfunction were occurred due to anaplasmosis **(Khan et al., 2011)**.

In our study Level of total protein ,albumen and globulin exhibited non-significant decrease in T. annulata, Babesia, and Anaplasma infected camels in comparison with non-infected camels This result agree with **Salem and El Olemy, (2017)** recorded insignificant changes in the hepatic and renal

biomarkers between T. annulata-infected and non-infected camels, which could exclude the hepatic or renal damage. While this result disagrees with **Youssef et al., (2015**) reported significant hypoproteinemia, hypoalbuminemia, hyperglobulinemia in camels infected with theileriosis. Also, this result disagree with **swelum et al., (2014)** recorded significant increase in total protein, albumen, and globlin level in Babesia-infected camels.

In our study the Level of urea was significantly increased ($P<0.05$) in the Thieleria, Babesia and Anaplasma infected camels when compared to non-infected camels, while Levels of the creatinine exhibited non-significant changes in all infected camels when compared to non-infected camels. Noticeable elevation in urea was attributed to the disturbance in kidney, muscle exhaustion, and colonization of *B. ovis* inside the renal blood circulation (**Uilenberg and Babesia, 2006)**. It was supposed that ovine babesiosis was associated with impairment of the renal function attributed to remarkable histological changes as leading acute tubular necrosis, proliferative glomerulitis, glomerular hemorrhage, renal thrombosis, congestion and dilatation in the blood vessels (**Habella et al., 1991)**. This results agree with (**Bhatia et al., 2014)** who reported that, anaplasmosis evidently caused renal upsets as nephrosis, ischemia, dehydration, and heart problems, which due to increase in BUN levels.

This result agrees with **Swelum et al., (2014)** who recorded significant increase in urea level in Babesia-infected camels. Also, this result agrees with **Salem and El Olemy, (2017)** who recorded insignificant changes in creatinine T. annulata-infected camels. On another hand, This result disagree with **Coskun et al., (2012)** who mentioned that *Anaplasma marginale*-associated parasitemia induced non-significant blood urea levels in infected cattle. However **Salem and El Olemy, (2017)** recorded insignificant changes in urea level in *T. annulata*-infected camels. While, **Youssef et al., (2015**) reported significant increased creatinine concentration, in camels infected with theileriosis.

In our study pathological findings in *Theileria* infected camel showed diffuse fatty degeneration at hepatic parenchyma and fibrosis in liver. Lymph nodes displayed depletion and necrosis at the lymphoid follicle. There was detection of Koch's blue bodies at lymphocytes of the lymph nodes.

Singh, et al., (2001) also reported that, infected camels with blood parasites were leading to distinct histological deviations in the target organs involving liver and lymph nodes. It was distinguished with fatty degeneration of the hepatocytes with necrosis and depletion of the lymphoid follicles, besides infiltration of Koch's blue bodies at lymphocytes. Histological changes were attributed to progressive destruction of erythrocytes via the reticulo-endothelial pahagocytosis resulted in hypoxic tissues and destructive damage to the related organs

This result agrees with **Oryan et al., (2013)** record that T. annulata piroplasms cause lymphadenopathy. histopathologically, it induced proliferation of lymphocytes in the lymph nodes ,portal tracts of the liver, and interstitial tissue of the kidneys showed numerous multifocal areas of necrosis

Our result also agree with **El-Refaii et al., 1998** who detected large numbers of macro and microschizonts inside lymphocytes of the lymph nodes of T. annulata infected animals. Also, there was hyperplasia of lymphoblasts, besides lymphocytic depletion of the lymphoid follicles. Liver was intensely infiltrated with lymphocytes associated with intensive vacuolar degeneration of hepatocytes. Moreover, it is also agree with **Mohammed and Elshahawy, (2018)** who reported the lesions of thieleria in cattle, it induced renal infarction, hepatic degeneration. Simillary **Sandhu, (1996)** noticed that T. annulata infections induced noticeable necropsy lesions such as hepatomegaly. It was

indicated severe damages to the hepatobiliary system due to hypoxia that resulted from hemolytic anemia and jaundice .

Concerning to anaplasma infected camel in our study. It exhibited fatty degeneration and portal fibrosis in liver. The Lymph nodes showed capsular fibrosis and necrosis in lymphoid cells.

These results agree with **Das et al., (2021)** reported that The major histopathological changes noticed included degenerative changes in hepatocytes and renal tubular epithelial cells. While **Lima et al., (2019)** mentioned that cattle infected with anaplasma revealed focal infiltration of macrophages and lymphocytes in the liver and kidney, in addition hepatic sinusoidal dilatation.

Also **Coetzee et al., (2005)** recorded inflammatory process characterized by mononuclears infiltration into liver. Moreover**, Jaswal et al., (2015)** mentioned that, in anaplasma infected cattle, liver fatty changes were observed in the hepatocytes along with retention of bile which may be dueto cholestasis along with mild infiltration of mononuclear cells in portal triad and marked thickening of glission capsule or perihepatitis. Coagulative necrosis of the heaptocytes and the bile ducts hyperplasia has been reported earlier **(EgbeNwiyi et al., 1997).** Hepatic necrosis is afeature of this disease. **Anderson and Hurtado, (1989)** also reported centrilobular hypoxic hepatic necrosis which may be due to erythrocyte destruction .**DeVos et al., (2006)** also reported biliary retention in most cases of anaplasmosis as there is accumulation of haemosiderin in cells of mono-nuclear phagocytic system.

Camel infected with Babesia in our study showed portal cirrhosis in liver. The examined lymph nodes revealed hyperplasia in lymphoid follicle associated with lymph adenitis in some cases, furhermore, there was depletion, edema and

necrosis lymphoid follicle, besides severe necrosis in other cases. This result agrees with **Mohammed and Elshahawy, (2018)** reported that, Babesia bovis, induced renal infarction, hepatic degeneration.

Our pathological results in the examined lymph node from infected camels are agree with **Levine, (1961)** who recorded that, blood Babesiosis induced hyperplasia of the reticuloendothelial system mainly in lymph nodes with lymphadenopathy, swelling, mass or enlarged lymph nodes. Also, **Wozniak et al., (1997)** mentioned that *Babesia gibsoni* infection was manifested by reactive lymphadenopathy resulting in activation and expansion of T and B lymphocyte populations, macrophage recruitment and activation. Simillary, **Sudhakara Reddy et al., (2016)** noted that clinical examination of Babesia infection in dogs reveals lymphadenopathy characterized by swollen lymph nodes and icterus.

The histolopathogical changes represented in our results could be attributed to progressive destruction of erythrocytes via the reticulo-endothelial pahagocytosis resulted in hypoxic tissues and destructive damage to the related organs **(Singh, et al., 2001)**.

SUMMARY

Blood parasites are known to affect the health of camels. They have a high economic impact in several parts of the world, including tropical and temperate countries. There is paucity of information on haemoparasites of camels and their significance on health and productivity in Egypt. This study was undertaken to determine the prevalence of haemoparasites of camels slaughtered in Daraw and Aswan slaughter houses, belonging to Aswan Governorate, Egypt, and Evaluation the effect of these blood parasite infections of camels *(camelus dromedarius*) on biochemical and hematological parameters , in addition to histopathological examination.

The present study was carried out on (100) number of camels obtained from Daraw and Aswan slaughter houses, belonging to Aswan Governorate during period from 10/2019 to 10/2020. The survey camel appeared sub clinically without any symptoms. Two separate blood samples were collected from jugular vein of camels from Aswan slaughter houses. One sample was taken in vacuum tube anticoagulated with EDTA for hematological studies. . Thick and thin blood smears were made for morphological examination of some protozoan blood parasites. While, the second blood sample was taken in plain tubes, centrifuged at 3000 rpm for 10 min and the clear serum was separated carefully and stored in Eppendorf tubes at -20 °C until serum biochemistry analysis. Samples from lymph node and liver infected camels with blood parasite were collected and fixed in 10 % neutral-buffered formalin for histopathological examinations.

In our study, microscopic examination of the Giemsa stained blood smears revealed varied prevalence rates of three heamoparasite (Thieleria sp

(15%), Anaplasma(10%), and Babesia(5%)) with total heamoparasitic prevalence (30%) in out of the 100 asymptomatic studied camels, camels examined throughout four different seasons (From 10/2019 till 10/2020).

In our study prevalence rate of heamoparasite (Thieleria, Anaplasma, and Babesia) infection to be the most frequently in autumn season (16**%)**, followed by summer (8**%**), then winter (5**%**) and at the lowest rate during spring (1**%)**.

In our study, **hematological results** revealed a significant decrease ($P<0.05$) in RBCs count, Hb. concentration, and PCV % in Thieleria, Anaplasma, and Babesia infected camels when compared with non-infected camels. While other parameters including WBC, count, MCV, MCH, and MCHC values showed non-significant changes in Thieleria, Anaplasma, and Babesia infected camels when compared with non-infected camels.

Significant increase ($P<0.05$) in lymphocytes % Thieleria, Anaplasma, and Babesia infected camels was noticed when compared with non-infected camels. Also, significant increase in eosinophil % in Thieleria, Anaplasma, and Babesia infected camels when compared with non-infected camels. Monocytes showed increase in Anaplasma infected camels. While, other parameters including Neutrophil, basophil values showed non-significant changes with non-infected camels

Biochemical results Level AST revealed significant increases ($P<0.05$) in Thieleria, Anaplasma, and Babesia infected camels in compare with non-infected camels. While, ALT revealed significant changes ($P<0.05$) only in Anaplasma infected camels in comparison with non-infected camels. The mean levels of the total protein and globulin exhibited non-significant decrease in T.

annulata, Babesia, and Anaplasma infected camels in comparison with non-infected camels.

The levels of the urea was significant increases ($P<0.05$) in the Thieleria, Anaplasma, and Babesia infected camels when compared to non-infected camels. Levels of the creatinine exhibited non-significant changes in all infected camels, when compared to non-infected camels.

Histopathology

Pathological findings of liver of Theileria infected camel showed diffuse fatty degeneration at hepatic parenchyma**.** Lymph nodes displayed necrosis at the lymphoid follicle**.** There was detection of Koch's blue bodies at lymphocytes of the lymph nodes.

While infection by Babesia, it showed fibrosis in liver. Lymph nodes revealed hyperplasia in lymphoid follicle and lymph adenitis**. Furhermore,** there was depletion, edema and necrosis lymphoid follicle, besides severe necrosis.

Concerning to Anaplasma infected camel; it exhibited fatty degeneration and portal fibrosis in liver. Lymph nodes detected fibrosis and necrosis in lymphoid cells.

CONCLUSION

From the obtained results, it could be concluded that:
- Blood parasites infections in camels at Aswan Governorate includes *Thieleria, Babesia,* and *Anaplasma.*

-Most of positive cases are clinically health, this means that we face problematic to cure these infected animals as they become carriers of parasite and serve as reservoirs for transmission of infection to other animals.

-Even in these carrier animals, there were clinicopathological deteriorations involving hematological and biochemical alterations as well as histolopathogical changes on the target organs of the examined camels.

Recommendation

Screening and treatment of infected and carrier animals as well as control program of the vectors must be done specially during the seasons of vector-borne disease.

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الملخص العربى

من المعروف أن طفيليات الدم تؤثر سلبيا على صحة الإبل. مما يجعل لها بعد اقتصادي كبير في أجزاء عديدة من العالم، بما في ذلك البلدان الاستوائية والمعتدلة. هناك ندرة في المعلومات عن طفيليات الدم للإبل وأهميتها على الصحة الانتاجية في مصر. اجريت هذه الدراسة لتحديد مدى انتشار طفيليات الدم في الإبل المذبوحة في مجازر دراو واسوان التابعة لمحافظة اسوان ، مصر. وتقييم تأثير عدوى طفيليات الدم على المعايير البيو كيميائية وأمراض الدم بالإضافة الى فحص الأنسجة المرضية لهذه الابل(الجمل الجمل) .

ولقد أجريت الدراسة الحالية على عدد ١٠٠ من الإبل تم الحصول عليها من مجازر دراو وأسوان التابعة لمحافظة أسوان خلال الفترة من أكتوبر ٢٠١٩ إلى سبتمبر ٢٠٢٠. و قد كانت الجمال تبدو دون أية أعراض إكلينكية. وتم جمع عينتين منفصلتين من الدم من الوريد الوداجي للإبل من مسالخ أسوان. حيث تم أخذ عينة واحدة في أنبوب مفرغ مضاد للتخثر مع الإديتا لعمل فحوصات الدم. وعمل مسحات دم سميكة ورقيقة للفحص المورفولوجي لبعض طفيليات الدم الأولية. بينما ، تم أخذ عينة الدم الثانية في أنابيب عادية ، وطردها عند ٣٠٠٠ دورة في الدقيقة لمدة ١٠ دقائق وفصل المصل الصافي بعناية وتخزينه في أنابيب إيبندورف عند -٢٠ درجة مئوية حتى تحليل الكيمياء الحيوية في الدم. ومن الناحية الباثولوجية فقد تم جمع عينات من العقدة الليمفاوية والكبد المصابة بطفيليات الدم وتثبيتها في ١٠٪ من الفورمالين المحايد المعزول للفحوصات التشريحية المرضية.

من حيث نسبة انتشار طفيليات الدم بالثاليريا، و الأنابلازما والبابيزيا، فقد كانت ١٥٪ ، ١٠٪ و ٥٪ على التوالي حيث نسبة انتشار الكلى لطفيليات الدم ٣٠ ٪ مع سلبية جميع الابل لطفيل الدم التريبانوسوما خلال الفترة من أكتوبر ٢٠١٩ حتى سبتمبر ٢٠٢٠.

فى دراستنا معدل انتشار طفيليات الدم (الثاليريا، و الأنابلازما والبابيزيا) هى الاكثر

شيوعا فى فصل الخريف(١٦٪) ويليه الصيف (٨٪)، ثم الشتاء (٥٪) وباقل معدل خلال الربيع (١٪).

أظهر الفحص المجهري لمورفولوجيا كريات الدم الحمراء لفيلم الدم المصبوغ بجيمسا تشوهات مورفولوجية متنوعة في كريات الدم الحمراء في الجمال المصابة بطفيلي الدم. وتشمل زيادة عدد الكريات الغير بالغة ، تغيرات فى شكل وحجم الكرات ، مع نقص الصباغ.

ولقد أظهرت نتائج الدم انخفاضًا معنويًا (عند مستوى أقل من 0.05 %) في عدد كرات الدم الحمراء ، ونسب الهيموجلوبين ، حجم الخلية الحزمى ٪ في الإبل المصابة بالثاليريا، والبابيزيا و الأنابلازما عند مقارنتها بالإبل غير المصابة. بينما أظهرت المعاملات الآخرى والتى تشمل عدد كرات الدم البيضاء ، متوسط حجم الخلايا ، متوسط الهيموجلوبين الخلوي ومتوسط تركيز الهيموجلوبين الخلوي تغيرات غير معنوية في الإبل المصابة عند مقارنتها بالإبل غير المصابة.

كما لوحظ زيادة معنوية في نسبة الخلايا الليمفاوية في الإبل المصابة بالثاليريا، والبابيزيا مقارنة بالإبل غير المصابة. أيضا ، هناك زيادة معنوية في نسبة الحمضيات في الإبل المصابة بالثاليريا، و الأنابلازما والبابيزيا عند مقارنتها بالإبل غير المصابة. وأوضحت الخلايا الأحادية زيادة في الإبل المصابة بأنابلازما. بينما أظهرت معاملات أخرى متضمنة العدلات والقعدات تغيرات غير معنوية عند مقارنتها بالإبل غير المصابة.

وبينت النتائج البيوكيميائية أن هناك زيادات معنوية فى مستوى الاسبرتيت أمينو ترانسفيريز في الإبل المصابة بالثاليريا، و الأنابلازما والبابيزيا عند مقارنتها بالإبل غير المصابة. بينما أظهر نشاط الأنيين أمينو ترانسفيريز تغيرات معنوية فقط في الإبل المصابة بالانابلازما مقارنة بالإبل غير المصابة. أظهر متوسط مستويات البروتين الكلي والجلوبيولين تغيرات غير معنوية في الإبل المصابة بالثاليريا، و الأنابلازما والبابيزيا عند مقارنتها بالإبل غير المصابة.

كـان متوسـط مسـتويات اليوريـا فـى زيـادة معنويـة فـي الإبـل المصـابة بالثاليريـا، و الأنابلازمـا والبابيزيـا مقارنـة بالإبـل غيـر المصـابة. علـى العكـس فـى مستويات الكريـاتينين ، حيث أظهر تغيرات غير معنوية في جميع الإبل المصابة مقارنة بالإبل غير المصابة.

من ناحية التشريح المرضي ، كانت أكثر الآفات المرضية شيوعا بين طفيليات الدم في الإبل فـى صـورة التليف والتنكس الدهني فـي حمـة الكبد. أمـا الغدد الليمفاويـة فقد كانت تعانى من نضوب وذمة ونخر في الجريب اللمفاوي.

الخلاصة

من خلال دراستنا نستنتج

١- اصابات الجمال بطفيليات الدم فى محافظة اسوان تشمل الثيليريا والبابيزيا والانابلازما .

٢- معظم الاصابات فى الجمال بطفيليات الدم دون أي اعراض اكلينيكية مما يعنى اننا نواجه مشكلة حيث ان هذه الجمال ناقلة للمرض و تعمل كمخزن لنقل العدوى للحيوانات الاخرى.

٣- الجمال الناقلة لطفيليات الدم تعانى من تدهورات باثولوجيه اكلينيكية تشمل تدهور في نتائج الدم وتدهور فى النتائج البيوكيميائية لهذه الابل بالإضافة الى تغيرات باثولوجيه في الانسجة.

التوصية

يجب تشخيص وعلاج الحيوانات المصابة والناقلة للمرض وعمل برنامج لسيطرة والقضاء على القراد وخاصة في الفصول الاكثر شيوعا لحدوث المرض .

بسم الله الرحمن الرحيم

﴿ رَبِّ اشْرَحْ لِي صَدْرِي ﴿٢٥﴾ وَيَسِّرْ لِي أَمْرِي ﴿٢٦﴾
وَاحْلُلْ عُقْدَةً مِنْ لِسَانِي ﴿٢٧﴾ يَفْقَهُوا قَوْلِي ﴿٢٨﴾

صدق الله العظيم

سورة طه

قسم الباثولوجيا والباثولوجيا الإكلينيكية

دراسات باثولوجية اكلينيكية على الجمال المصابة ببعض طفيليات الدم بمحافظة اسوان

مقدمة من:

أحلام أحمد أبوزيد

ماجستير العلوم الطبية البيطرية

كلية الطب البيطري - جامعة جنوب الوادي – قنا - ٢٠١٦

تحت إشراف

الأستاذ الدكتور / ساري خليل عبد الغفار

أستاذ الباثولوجيا والباثولوجيا الإكلينيكية

كلية الطب البيطري

جامعة أسيوط

الأستاذ الدكتور/ محمد كرمي حسين

أستاذ الرقابة الصحية علي الاغذية

عميد كلية الطب البيطرى

جامعة أسوان

الدكتورة /مروة أحمد أحمد

أستاذ مساعد الباثولوجيا

كلية الطب البيطري

جامعة أسوان

الاستاذ الدكتور/ عصام محمد إبراهيم

رئيس بحوث الباثولوجيا اكلينيكية

وكيل المعهد لشئون التشخيص وصحة البيئة

معهد بحوث الصحة الحيوانية

الدقى- الجيزة

رسالة مقدمة إلى

كلية الطب البيطري- جامعة أسوان

للحصول على درجة دكتوراه الفلسفة في العلوم الطبية البيطرية

(الباثولوجيا الإكلينيكية)

٢٠٢٢-١٤٤٣