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PATHOLOGICAL INVESTIGATIONS ON *TRYPANOSOMA EVANSI* CAUSING ABORTION IN EGYPTIAN SHE- CAMELS

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ABSTRACT

We devote this study to clarify the pathological alterations occurring in naturally infected dromedary she-camels with *T. evansi* causing abortion and stillbirth using parasitological techniques for accurate and rapid detection of *T. evansi* on both fields and experimental levels. Also, to investigate and insure histopathological alterations in the experimentally infected pregnant female mice with *T. evansi* detected in blood of naturally infected she-camels. 220 she- camels (*Camelus dormadarius*) belonged to different localities in Wahat Baharia and Mattroh governorates (suspected cases, clinically diseased cases, aborted cases and healthy animals for comparison) were examined by direct microscopic examination of wet blood smear and Giemsa stained of thin blood smears. Out of them, 92 animals (51.39%) were positive for *T. evansi* infection. The suspected cases of abortions in the infected dams recorded from fifth to tenth month of gestation were 52 (34.78%) cases. The clinical signs of trypanosomiasis in the study areas included intermittent fever (39.5 - 41°C), weakness and emaciation with pale mucous membrane and dry scruffy coat. The eyes appeared dull and half closed with considerable amounts of tears, in addition to the animal stand with his head raised toward the sun. Histopathological examinations revealed degenerative changes and necrosis in placentas and uteri of aborted dams. Congestion and necrosis were detected in liver. Hyperplasia of spleen lymphoid follicles of aborted and stillborn feta was evident.

The experimentally inoculated pregnant female mice with the blood of the naturally infected she-camels were conducted and the uterus, liver and spleen of the aborted dead female mice showed the same lesions as in aborted she-camels, feta and stillborns.

Keywords:

She-camel, *Female mice, Trypanosoma evansi*, abortion, parasitology and histopathology.

INTRODUCTION

T. evansi was the first pathogenic trypanosome recognized by **Evans (1880),** in India from the blood of Indian horses and camels causes a disease known as Trypanosomiasis or surra in all the principal species of domestic animals and it is also highly pathogenic to laboratory animals **(Patel, 1982; Pathak *et al.,* 1993 and Soodan *et al.,* 1995).** The disease is seasonal and the incidence is higher during rainy and post rainy seasons due to preponderance of *Tabanus* flies **(OIE, 2015)**. Other blood-sucking insects, which can transmit the infections include *Stomoxys, Lyperosia, Hematopota* (flies) and *Ornithodorus* (tick) in equine, cattle and buffaloes **(Sumba *et al*. 1998; Sridhar, 1989 and Takle and Abebe, 2001).** *T. evansi* is spindle-shaped, unicellular, extracellular flagella and motile protozoa with rapid twisting motion. Most forms are long and slender belonging to phylum *Sarcomastigophora*, the order of *Kinetoplastidae*, family of *Trypan-somatidae* and the genus of trypanosome, under the salivary group **(Urquhart *et al.,* 1996; Kinne *et al.,* 2001)**. The sub genus Trypanosome includes the pathogenic species *T. evansi*, *T. brucei* and *T. equiperdum* **(Tefera and Gebreab, 2004 and OIE, 2008)**. The parasite multiplies in blood through binary fission and undergoes an acute phase with regular peaks of parasitaemia **(El-Sawalhy, 1999 and Zeleke and Bekele, 2001)**. It is a potential killer of livestock in camels and causes economic losses to the farmers in terms of morbidity, mortality, abortion, infertility, reduced milk yield, progressive loss of appetite, loss in body weight, anemia, edema of lower parts of body, intermittent fever, bilateral enlargement of prescapular lymph nodes, corneal opacity, salivation, lacrimation **(Luckins, 1992; Lohr *et al.,* 2009 and Pourjafar *et al.,* 2012)**. The highest incidence of the disease occurred during high rainfall, which encourages the breeding of vector population **(Juyal, 2017 and Dhami *et al*., 2018)**. It is considered as both blood and tissue parasite, due to its ability to invade the nervous system, not only in horses ,camels, dog ,cat, rabbit, rodents and saw but also in goat ,sheep, cattle and buffaloes **(Uche and Jones, 1992; Juyal, 1996; Mahmoud *et al.,*1999; Singla *et al*.,2003; Hasan *et al*.,2006;Dargantes *et al*., 2009**; **Coura and Borges**, **2010; Siriporn *et al.,* 2011; Arjkumpa *et al.,* 2012 and Sivajothi *et al*.,2015).** In camels, the disease is known as "Tribersa" or El - dabab or El - Gafar **(Pathak and Khanna, 1995 and Pathak *et al.*1997)** and runs in acute and chronic form. In acute form there is intermittent fever reach to 39.5 - 41°C with subcutaneous edematous swelling, lacrimation, and it can be transported via the placenta to the fetus of experimentally infected animal but in chronic form, edema of dependent parts, loss of milk and meat production, abortion and

premature births **(Tizard, 1985 and Ouma *et al.,* 1997)**. Laboratory animals of choice are rats and mice, which are highly susceptible to experimentally infection **(Gill, 1991and Juyal, 2012)**. The disease has wide distribution in areas of Africa, Middle East and Asia such as Somalia **(Dirie *et al.,* 1989)**, South America **(Radostits *et al*., 2000)**, Kenya **(Njiru *et al.,* 2002)**, Chad **(Delafosse and Doutoum, 2004)** and Pakistan **(Hasan *et al.,* 2006)**. *T. evansi* infections are also responsible for producing a state of severe immune suppression which renders the infected host more susceptible to secondary infections and produce poor immune response to bacterial and viral vaccines **(Holmes, 1980 and Vincendeau *et al.,* 2006)**. The interference due to *T. evans* infection with vaccination program have been postulated and immune suppression has been shown in *T.evansi*-infected mice **(Eyob and Matios, 2013)**, also, their prominent effects on reproductive Performance causing abortion and stillbirth in Egyptian ewes experimentally infected with *T. evansi* **(Dalal *et al.,* 2008)** and in *T. evansi* infected rabbits **(Chandra *et al.,* 2000)**.

Sakr *et al.,* 199 diagnosed *Trypanosoma evansi* infection in 30 out of 100 examined Egyptian dromedary camels. The infected animals showed emaciation, atrophy of the hump, general weakness, edema of the ventral parts of the abdomen and abortion resulted with neonatal premature death. In several studies reported by **(Gutierrez *et al.,* 2000; Pacholek *et al.,* 2001; Njiru *et al*., 2004; Antoine *et al.,* 2007 and Parsani *et al*., 2008)** and the internal organs of infected camels with trypanosomes showed degenerative changes such as fatty changes, congestion of the central vein. Focal leukocytes aggregation in the lung and thickening of most of the alveoli with some areas of emphysema, in addition other changes in spleen and kidneys were observed by **(Gutierrez *et al.,* 2005; OIE, 2008 and Marc *et al.* 2013)**. *T.evansi* infection is described as destructive disease in nature and capable of causing sudden death of the animal. So, the aim of the present investigation is to evaluate the characteristic clinical signs and clarify the pathological alterations occurring in the aborted dromedary she-camels naturally infected with *T. evansi* in different localities and farms of two important governorates for camel husbandry in Egypt and highlight on the prevalence of camel trypanosomiasis in the study areas.

MATERIAL AND METHODS

Sampling and target area:

Blood samples of 220 she-camels (*Camelus dromedarius*) at age of 3-10 years were collected from different localities of Wahat Baharia and Mattroh governorates, Egypt (including apparently healthy, suspected cases, clinically diseased animals with clinical signs of anorexia, intermittent fever ,weight loss, lack of milk production, history of abortion and stillbirth for parasitological investigations to detect the infection with *T. evansi* .

1-Parasitological examinations:

A- Field techniques.

Whole blood samples were collected from she-camels under investigations (n=220) in the field either in the evening or in the early morning by jugular vein puncture in sterilized coated tubes with ethylene EDTA (tetra-acetic acid) as anticoagulant at a concentration of 1.0 mg/1ml of blood for parasitological examination and kept in cooler box and transported to the laboratory.

B- Laboratory techniques.

Diagnosis of the disease was based on the presence of *T. evansi* parasite using wet blood film and by Giemsa stained blood smear. For the wet film technique; a drop of blood was placed on a clean glass slide, cover slip, allowing the blood to spread then examined under microscope as a tentative examination to observe jerky movements of the motile trypanosomes.Thin blood smears were made as air dried smears fixed in absolute methyl alcohol for 2-3 minutes. The slides were immersed in Giemsa's stain for 20-25 minutes and washed with tape water to remove excess stain. After air-drying, the slides were examined under oil immersion objective lens (1000x) for detection and identification of *Trypanosomes* based on their morphological characters as method described by **(Bruits and Ash wood, 1994).** For comparison, blood samples from healthy pregnant dams from the same herd farms were used as control. These camels had normal full term delivery and detected negative for *T. evansi*, by wet blood film and stained blood smears.

C-Laboratory animal inoculation.

The *T. evansi* strain was isolated from the positive blood of the naturally infected she-camel under investigation and has been maintained by serial passage into 5 mice. Positive blood was injected intraperitoneally in mice (0.5 ml each). Serial passages in several mice were carried out. Mice were examined daily by wet film from tail vein until parasitaemia reached more

than 20/high power field. Mice were bled under terminal anesthesia and their bloods were pooled. The strain was maintained in mice, where their infected blood was used for inducing the experimental infection on the pregnant mice. Giemsa-stained blood films were prepared for detection of infection and estimation of parasitemia. 25 white Swiss female pregnant mice (at seven day of gestation) weighting from 23-25 gm obtained from Laboratory Animals House, Research Institute of Ophthalmology, Giza, were used for induction of experimental infection. 20 mice were inoculated intra-peritoneal with the maintained strain in mice with a dose of 1 x 10^4 diluted mouse blood in 2 ml PBS for each one (Infective dose) after counting on heamocytometer per ml of infected blood according to methods of **(Singh *et al.,* 2004).** 5 mice were kept as control. Blood smears of the inoculated mice stained with Giemsa were examined daily to estimate the parasiteamia.

D- Direct microscopic examination of the inoculated mice.

Wet smear of 5µ of fresh peripheral blood collected from female mice tails were placed on a clean glass slide and covered with cover slide then examined directly using a light microscope to detect the jerky movement of trypanosomes according to **(Carmona *et al*., 2006).**

E-Giemsa stained blood smears.

A drop of blood was placed on one end of a clean glass slide and a smear was drawn out. The blood smears were air-dried, fixed in methyl alcohol for 2 minutes and allowed to dry, then stained by Giemsa stain 10%. Trypanosomes were detected by microscopic examination at oil immersion-according to **(Abdel-Rady, 2008).**

2-Histopathological examination:

Thirty four uteri of aborted she-camels, internal organs (liver and spleen) of twenty-six aborted feta with their placentas and twelve stillbirth calves were collected.

The uteri, spleens and livers of the experimentally inoculated female mice were immediately collected and grossly examined.

Tissue samples from all these organs were fixed in 10% neutral buffered formalin, routinely processed in automated tissue processor, embedded in paraffin, sectioned at 3-5 µm, stained with hematoxylin and eosin (H&E) and examined by light microscope according to **(Bancroft and stevens,1990).**

RESULTS

1-Parasitological results:

A- She-camels.

Wet blood Films were taken from total number of she-camels in the target areas (n=220) and out of them; there were 179 suspected infected cases and 41were normal healthy cases.

Out of 179 the suspected ones; 92 (51.39%) were *T. evansi*-positive by blood film examination with high parasitaemia. The infected pregnant dams showed intermittent fever and 52 (34.78%) cases of abortion were recorded in (Table 1). Light microscope examination revealed that *T. evansi* parasites were observed in Giemsa stained smear of blood from an aborted she-camel as shown in Fig. (1).

B- Female mice.

Infected mice also showed dullness and delivery problems, such as stillbirth, and fetal death that also led to female death. Mice from the control group had normal delivery of healthy off springs. The present study revealed that, trypanosomes observed in the experimentally infected pregnant female's mice using wet blood films and Giemsa stained blood film at three days after infection and there was high parasitaemia on approximately seventh day post inoculation, and the abnormal forms and hypochromic of RBCs were clearly noticed as shown in Fig. (2).

2-Pathological results:

A-Clinical manifestations.

In the present study, infected she-camels showed clinical signs of weakness and loss of appetite and weight with hard scruffy coat. The animal appeared restlessness and raise his head towards the sun. Edema of the front limbs was clearly noticed as shown in Fig. (3). A specific odor of the urine is detected by camel owners, which is efficient for diagnosing the disease and loss of body condition. Affected camels also may exhibit a characteristic sweet odor due to an increase of urinary ketones. Intermittent fever (39.5 - 41°C), sudden abortion of several cases of the pregnant she-camels at different stages of gestation (from 5 th month till 10th month) also, death of complete delivered calves with weak bodies after one or 2 days (stillbirth). Number and percentages of all examined she-camels, their aborted feta and stillbirth for detection of *T. evansi* in study target area were recorded in (Table 1).

Table (1) : Number and percentages of all examined she-camels, their aborted feta and stillbirth for detection of *T. evansi* in the study target area.

Target area of she - camels	Suspected infected she- camels	Clinically diseased she-camels of suspected	Aborted she camels of clinically diseased	Uteri of aborted slaughtered she camels	Aborted feta (placenta, liver and spleen)	Stillbirth after death
Mattroh governorate (n=95)	82	38	20	12	10	4
Wahat Baharia (n=125)	97	54	32	22	16	8
Total number (n=220)	179	92 (51.39%)*	52 (34.78%) **	34	26 (50%) ***	12 (23.07%) ****

***The percentage of positive samples from diseased she-camels calculated according to the total no. of suspected she-camels in target areas (n=179).**

**** The percentage of positive samples from aborted she-camel calculated according to the total no. of suspected she-camels in target areas (n=179).**

***** The percentage of positive samples from aborted feta calculated according to the total no. of aborted she-camels in target areas (n=52).**

****** The percentage of positive samples from stillbirth calves calculated according to the total no. of aborted she-camels in target areas (n=52).**

- Healthy she-camels in the target areas which were examined: 220-179 = 41animals.

B-Pathological findings in aborted dams:

Uterus and placenta:

Grossly: the uterus of aborted she-camel appeared enlarged in size with mucoid exudates in its lumen. On cut section, the endometrium was hyperemic, edematous and showed necrotic spots Fig. (4).

Microscopical examination of uteri of aborted she-camel infested with *T. evansi* revealed focal hemorrhage and edema with aggregation of inflammatory cells as well as periglandular

fibroblastic proliferation in the endometrium. The lumen of the uterine glands contained cellular debris Fig. (5). Severe congestion of large blood vessels with perivascular and mononuclear cell infiltrations accompanied with fibroblastic proliferation were noticed Fig. (6). In addition to most of the uteri of infected she-camels suffered from chronic endometritis characterized by degeneration and atrophy in the endometrial glands Fig. (7). Also, the microscopic examination of the placentas of aborted dams revealed necrosis and desquamation of the epithelium cell covering with cellular debris and inflammatory cells aggregation Fig. (8).

B-Pathological findings in aborted feta and still birth.

Grossly: the body parts of aborted feta and stillborn calves were fully developed but they had poor body condition. The necropsy revealed subcutaneous edema and congestion Fig. (9). The liver was enlarged with moderate to severe congestion along with pale patches of necrosis and the spleen was of normal size but showed moderate to severe congestion.

Microscopical findings of aborted feta and stillbirth:

The liver of aborted feta showed necrobiotic changes in the hepatocytes with hyperplasia of bile duct associated with fibroblastic proliferations and mononuclear cell aggregations in the portal area Fig. (10). the liver of stillborn calves showed focal inflammatory cell aggregations with necrosis of hepatocytes Fig. (11). the microscopical examination of spleen of stillborn calves revealed hyperplastic lymphoid follicles with wide germinal center Fig. (12) as well as the spleen of aborted feta revealed the same lesion.

C-Pathological examination of experimentally infected pregnant female mice.

Histopathological examination of the non-infected control group did not showed any pathological changes in all examined tissues. In experimentally infected groups, variable prominent pathological changes were seen through the course of infection.

During parasiteamia (7days post inoculation), the gross examination resemble that of aborted dams and the histopathological lesions of the uterus showed partial desquamation and sometimes stratification in the endometrial epithelium and moderate infiltration of inflammatory cells mostly sub epithelial. The lamina propria appeared edematous with degeneration of the uterine glands Fig. (13). the spleen showed hyperplastic lymphoid follicles Fig. (14), while liver showed degenerative changes varying from granular to vacuolar and necrobiotic changes of the hepatocytes. The Parasite appeared in lumen of the central vein Fig. (15).

DISCUSSION

In the present work, the percentage of *T. evansi* infection was (51.39 %) and this result is different than that reported by **(Amyl, 2014)** who recorded the prevalence of *T. evansi* in dromedary camels was (32%) at Darwa quarantine, Aswan, Egypt. Our higher incidence of the infected she- camels with *T. evansi* was due to some environmental conditions resulting from their habitat with other animals such as sheep and goats which considered as carrier to the disease and there is a possibility that she - camels of the present study have been infected while grazing in the areas having large number of vector population. These opinion come in agreement with (**Pathak and Khanna, 1995; Atarhouch *et al.,* 2003 and Gutierrez *et al.* 2005)**. Evaluation of the results of the current study in naturally infected she-camels and experimentally infected mice come in parallel to that recorded by **(Sakr *et al.,* 1991).**

Inoculation of the causative agent of the disease in laboratory female mice is a highly sensitive method for the diagnosis of *T. evansi* infection because the clinical signs of *T. evansi* infection are not sufficiently patho gnomonic for diagnosis; therefore, laboratory tests are required for detecting the parasite. In early stages or acute cases, when parasite counts are high, examination of wet blood films, stained blood smears might reveal the trypanosomes. In chronic cases when the parasitemia is low, the examination of thick blood smears with the inoculation of laboratory mice is important **(Oleo *et al.,* 1996)**.So, mouse inoculation test is generally regarded and accepted as the most sensitive method to detect the animal trypanosomiasis and this test considered as an impractical field tool **(Reid *et al.,* 2001)**. *T. evansi* is also highly pathogenic to laboratory animals (rat, mice and rabbit) **(Patel *et al.,* 1982; Uche and Jones, 1992; Biswas *et al.,* 2001; Singla *et al*., 2003)**.

Clinical signs such as dullness, pale mucus membrane with edema in the dependant parts of the body during the chronic stage could be due to a significant decrease in the albumin levels, resulting in alterations in osmotic pressure of the blood, the same symptoms were reported by **(Zelleke *et al.,* 1989)**. Fever characterized by high temperature might be due to the effects of toxic metabolites produced by dying trypanosomes. This opinion agrees with **(Wellde *et al.,* 1989)**. Emaciation and lacrimation of the eyes observed in our study were also noticed by **Sakr *et al.,* 1991 and AL-Rawashdeh *et al*., 2000** in *T. evansi* infected *dromedary camels* in Egypt. *T. evansi* would be expected to release large quantities of cytoplasmic and mitochondrial enzymes into the serum, thereby causing further tissue damage in addition, the

Parasite utilizes glucose and oxygen for its growth and multiplication resulting in depletion of these metabolites leading to degenerative changes in the host. Further changes develop in the organs either due to cellular damage caused by toxicants released by the parasite, or due to immunological reactions such as reported by **(Holmes *et al.,* 2017)** in bovine and camels.

Our study revealed abortion in different stages of pregnancy and neonatal death among the infected she-camels also stillbirth calves. These results come in agreement with **(Dial, 1993 and Tibary, 2006)**. It has been speculated that an interaction between the trypanosomes and the host may cause placental tissue damage and facilitate parasitic crossing of placental barrier in donkey by **(Kumar *et al*. 2015)**.The damage to the placenta caused by the parasite promote insufficient placental leading to interruption of pregnancy so placental insufficiency may be the one of the factors responsible for abortion and still birth in infected camels. These results agree with **(Silva *et al.,* 2013 and Shirish *et al*., 2016)**. The gross pathological findings such as severe congestion of aborted feta due to *T. evansi* infection, similar observation reported in ewes by **(Silva *et al.* 2013).** The microscopically findings of hemorrhages and hepatocellular necrosis with lymphocytic infiltrations and fatty degeneration, also, hyperplastic changes in spleen of aborted feta and stillborn calves.These results were recorded by **(Losos, 1980**) and similar to those reported in a study of experimentally infected donkey foal by **(Kumar *et al.* 2015)**. In this study, the pathological results observed in the uteri and placentas were similar to those obtained by **Chandra *et al.,* 2000** in rabbits; **Chaudary and Iqpal, 2000** in racing dromedary camel.Edema of the uteri of aborted dams and the mononuclear cell infiltration in the stroma of the uterus and the engorged blood vessels appeared to be the part of the immune proliferative response of the host against trypanosome infection and these pathological alterations could be considered as a good defense of the uterine tissue against *T. evansi* infection. This suggestion was supported by **(Coura and Borges, 2010)** who recorded that these histopathological findings play a great role in the occurrence of abortion in pregnant camels and death of calves during or after parturition. Uterine fibroblastic proliferation, inflammatory cells infiltration and glandular damage were responsible for infertility and low productivity of the animals as reported by **Gutierrez *et al.,* 2005** who believed that uterine damage alters protein synthesis. In addition, **Amer, 1992**; **Dalal *et al.,* 2008 and Marc *et al*., 2013** stated that periglandular fibroblastic proliferation plays an important role in the reduction of uterine milk and consequently early embryonic death. Moreover, necrosis was considered as a main cause of abortion and

stillbirth. These investigations come in agreement with **(Löhr *et al.,* 1986**; **and Radostits *et al.,* 2000)** in buffaloes infected with *T. evansi*. They reported that in trypanosome-infected East African Zebu cows resulted in abortions and still births by ratio from 7 to 10%. In the present study, the reason of abortion would be due to a progression of stress on animals as pregnancy developed and the immunity was suppressed as a result of infection **(Elhassan *et al*., 1995)**, reduction in dry matter intake **(Audu *et al.,* 1999)**, reduced nitrogen and energy balance **(Zwart *et al.,* 1991),** increased basal metabolic rate **(Stephen, 1986),** increased catabolism of tissue reserve **(Akinbamijo *et al.,* 1992)** and possibly uptake of host nutrient by the parasites. Destructive effect and cellular lesions associated with *T. evansi* infection are described as complication of the immune interaction between the host immune system and the parasite mechanism to evade the immune response of the host. Immune complexes, cytokines (interferon, interleukins and tissue necrotic factors) are considered the products resulted from that interaction and played the major role in the cellular damage in infected host **(Coles, 1980 and Shah *et al.,* 2004)**.Trypanosome enzymes such as phospholipases **(Tizard *et al.,* 1978),** neuraminidases and proteases **(Enwezor and Sackey, 2005)** have all been implicated in membrane fluidity and cellular damage **(Fung *et al*., 2007)**. Moreover, the phospholipases generate free fatty acids which lead to excessive accumulation of fluid in tissue spaces caused by a disturbance in the mechanism of fluid interchange between capillaries, the tissue spaces and the lymphatic vessels. All these possibilities indicate great liver damage which were irreversible pathological changes and lead to death of the infected animals as occurred in this study especially in female mice. These results agree with that reported by **(Biswas *et al.,* 2001 and Derakhshanfar *et al.,* 2010).** In addition to the inflammatory cells infiltrations have been reported in *T. evansi* infections of she-camels were parallel to that reported by **(Abd-Elmaleck *et al.,* 2014)**. Finally, the significant histopathological lesions in aborted and stillborn fetuses of the present study suggested the involvement of the parasite in the etiopathogenesis of reproductive failure in camels.

CONCLUSION

This study concluded that Surra is one of the most important diseases of camels and *T. evansi* induces destructive irreversible damage with reproductive disorder in she-camels and female mice which lead to death of the animals with progression of the disease and has a relatively high prevalence in the study area. Also, the disease causes a significant impact on the camel

production and economic losses in the growth of the study area by reduction in reproductive capacity like abortion, neonatal death, stillbirth and cost of treatment, so the control of surra become difficult as there is no vector specificity and a wide range of hosts.

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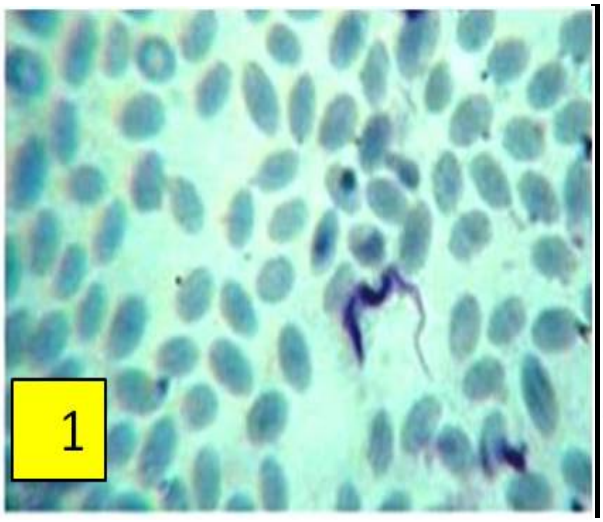
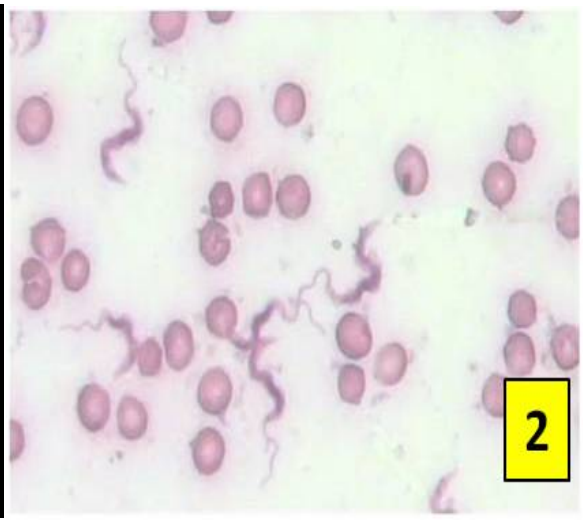
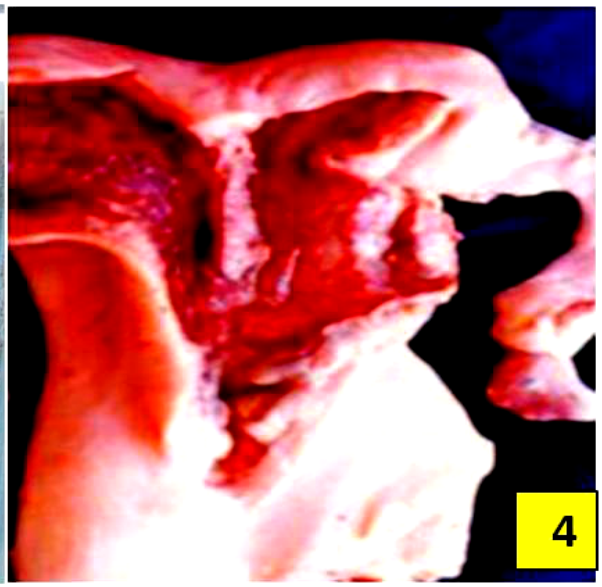
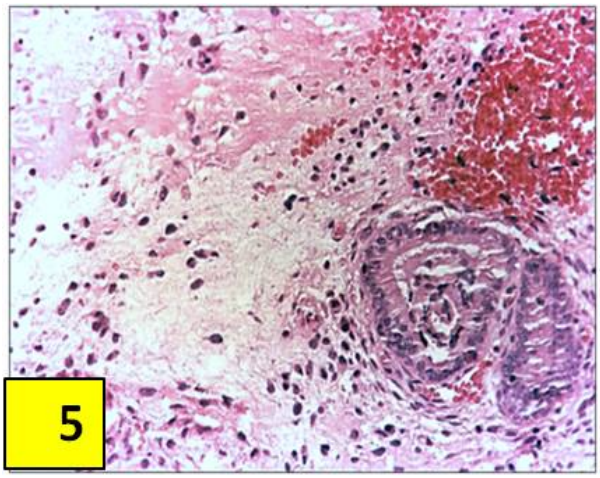
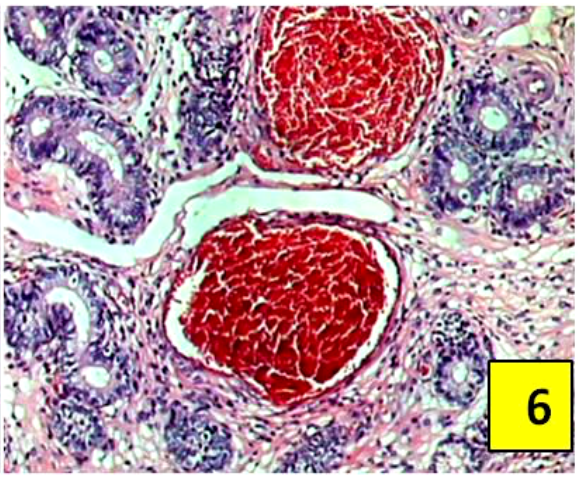
FIGURES

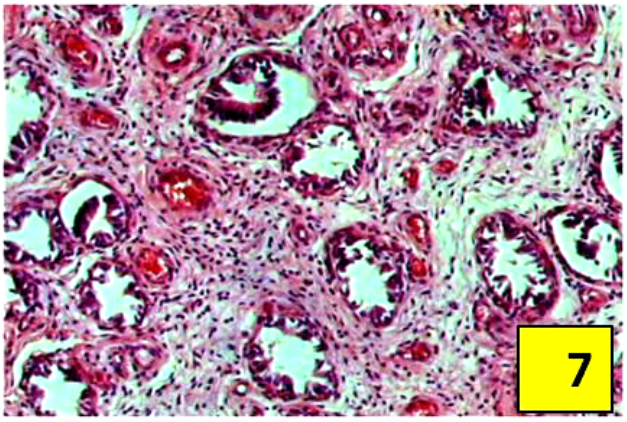








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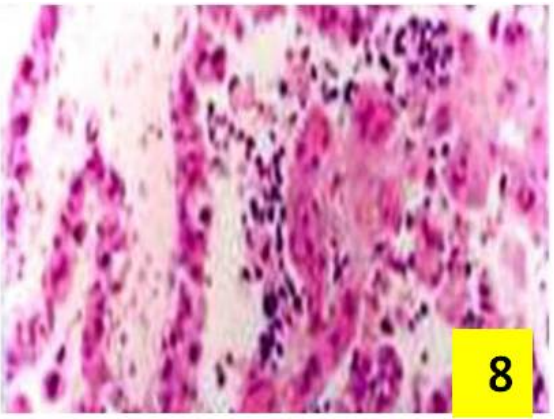
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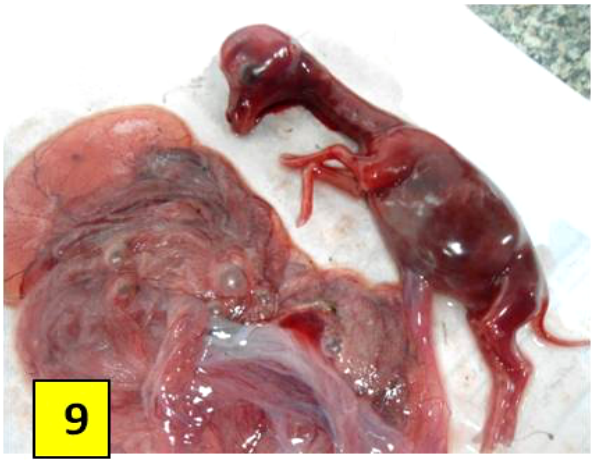
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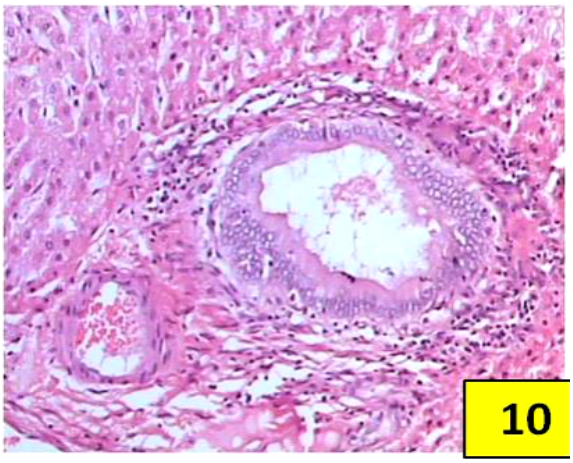
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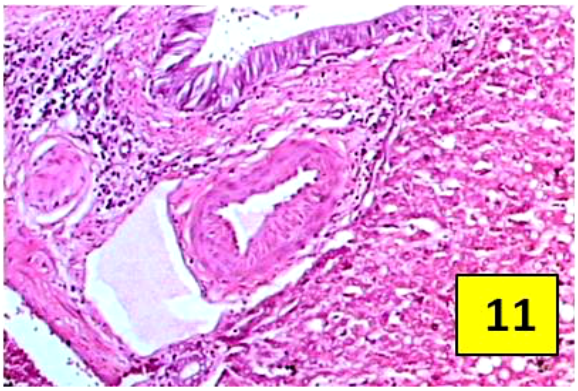
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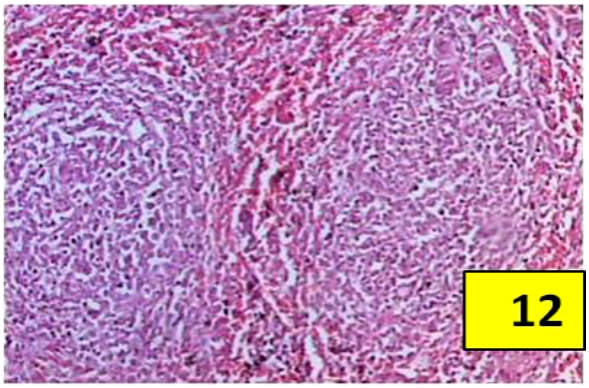
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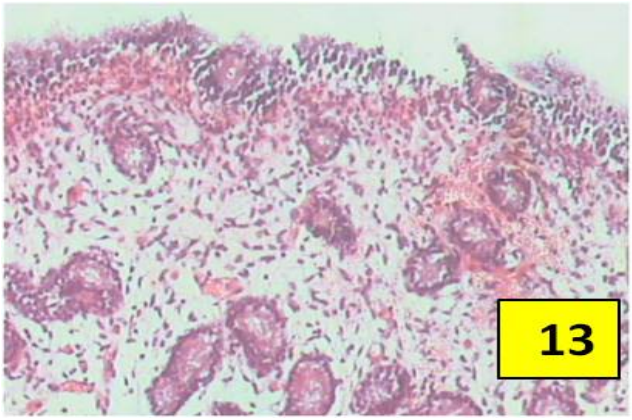
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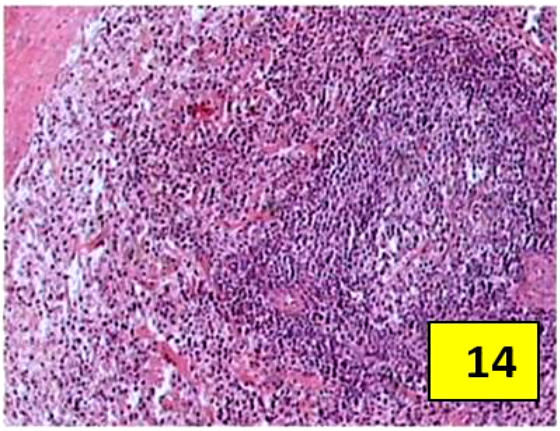
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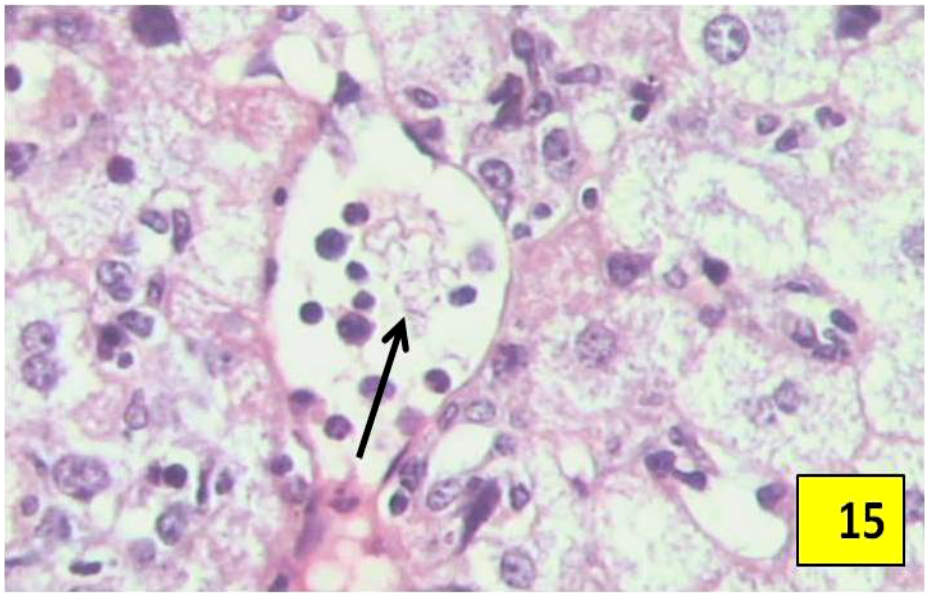

Fig. (1): Giemsa staining smear of blood film from an infected aborted she-camel with *T. evansi* **(X1000) (oil).**

Fig. (2): Giemsa staining blood smear of experimentally infected pregnant female mice after 7 days (post inoculation) with whole blood of infected she-camel infested. Not to abnormal forms and hypo chromic of RBCs **(X1000) (oil).**

Fig.(3): She-camel infected with *Trypanosoma evansi* showing dry scruffy coat, edema in the front limb, atrophy of the hump and raise the head towards the sun.

Fig. (4): Uterus of aborted she camel showing enlarged size with mucoid exudates in the lumen, hyperemic, edematous and necrotic spots.

Fig. (5): Uterus of she-camel infested with *Trypanosome evansi* showing focal hemorrhage, edema, focal aggregation of inflammatory cells and periglandular fibroblastic proliferation in the endometrium. **(H&E X 100).**

Fig. (6): Uterus of aborted she-camel infested with *T.evansi* showing severe congestion of large blood vessels and perivascular and periglandular mononuclear cell infilteration and fibroblastic proliferation. **(H&E.X100).**

Fig. (7): Uterus of aborted she-camel infested with *T.evansi* suffered chronic endometritis showing degeneration and atrophy in the endometrial glands, perivascular and periglandular mononuclear cell infiltrations and fibroblastic proliferation **(H&E.X100).**

Fig. (8): placenta of aborted she-camel showing necrosis and desquamation of the covering epithelial cell with cellular debris and inflammatory cells aggregation. **(H&E X100).**

Fig. (9): Aborted camel fetus at the fifth month of gestation with its placenta showing severe congestion of body surface and edema.

Fig. (10): Liver of aborted camel fetus showing necrobiotic changes in the hepatocytes with hyperplasia of bile duct. Fibroblastic proliferations and mononuclear cell aggregations in the portal area **(H&EX100).**

Fig. (11): Liver of stillborn camel calf showing edema with necrosis of hepatocytes and focal area of inflammatory cell aggregations **(H&E X100).**

Fig. (12): Spleen of stillborn fetus showing hyperplasia of lymphoid follicles with wide germinal center. **(H&E X100).**

Fig. (13): Uterus of experimentally infected female mice with *T.evansi* showing partial desquamation and sometimes stratification in the endometrial epithelium and moderate infiltration of inflammatory cells in sub epithelial while lamina propria appeared edematous with degeneration of the uterine glands. **(H&E X200).**

Fig. (14): Spleen of experimentally infected female mice with *T.evansi* showed hyperplastic lymphoid follicles **(H&E X100).**

Fig. (15): Liver of experimentally infected female mice (day7after inoculation) with *T.evansi* showing degenerative and necrobiotic changes of the hepatocytes with Parasite in the central vein **(H&E X400).**