j.Egypt.vet.med.Assoc 78, no 3. 345 – 367 (2018)

PATHOLOGICAL STUDIES ON THE EFFECT OF *TOXOPLASMA GONDII* ON THE GENITALIA OF DROMEDARY CAMELS IN SOME GOVERNORATES OF EGYPT

By

***Gerges, A. A.; **Thanaa K. Hassanien and ***Mary G. Abdel - Malak.**

*Reproductive Diseases Research Department,** Department of Pathology and ***Artificial Insemination and Embryo Transferee Department. Animal Reproduction Research Institute (ARRI), Agricultural Research center (ARC) Egypt.

ABSTRACT

The current study was conducted to evaluate the seroprevalence of Egyptian camel toxoplasmosis in different localities and its pathological alterations in the reproductive organs. Serum samples were collected from 200 dromedary camels and 240 tissue samples of 100 testis, 100 uteri and 40 brains from both sexes. Indirect hemagglutination test (IHAT) was applied for screening all serum samples for Toxplasma antibodyes titers. The isolation of *T. gondii* parasite by laboratory mice inoculation from homogenized parts of testis, uterus and brains of infected camels was conducted to confirm the positive results obtained by IHAT. The epididymal spermatozoa were collected to evaluate the motility, viability, sperm count, total abnormalities and acrosomal defects. According to the obtained results of antibody titer by IHAT the animals were classified into four groups: Group 1 (-ve IgG and - ve IgM , uninfected group), Group 2 (-ve IgG and + ve IgM , recent infection) ,Group 3 (+ve IgG and - ve IgM , old infection) and Group 4 (+ ve IgG and +ve IgM , evolutive toxoplasmosis). The obtained results revealed that 60 % were positive in IHAT and infection rate was 50%, 33.33% and 16.66 in infected male camel respectively. While in infected she-camel, 55% were positive and the infection rate was 36.36 %, 45.45 %, 18.18 % respectively.

The main pathological alterations were neuropathological changes associated with orchitis, endometritis and myometritis. *Toxoplasma gondii* infection resulted in highly significant increase in sperm abnormalities and acrosomal defects as well as decrease in percentages of sperm motility, viability and concentration. The current study concluded that *T. gondii* has direct effect on sperm parameters, which may lead to infertility problems in male camel, and the parasite has the ability to transmit through semen to she-camel.

Keywords:

Dromedary camels, Toxoplasmosis, Indirect hemagglutination test (IHA), Semen picture and Histopathology.

INTRODUCTION

The one humped camel "Camelus dromedaries" plays a very important role in the national income as a source of meat, milk, hair and hides .Camels play an important role in the epidemiology of parasitic diseases under the three aspects of animal health, transmission to other livestock and zoonoses **(Alireza and Anja, 2016)**. Parasitic infections of camels may cause reduced milk and meat production, impaired fertility and decreased calving rates. They may also lower the working efficiency or even result in death and consequently high economic losses **(FAO, 2002)**. Toxoplasmosis is a zoonotic infection of animals caused by the protozoan parasite- *Toxoplasma gondii.* Felidae species are the final hosts, whereas an array of warm blooded animals and human serve as intermediate hosts **(Zhang *et al.,*1999 and Nimri *et al.,* 2004**).Camels acquire *T. gondii* infection through ingestion , drinking of sporulated oocysts that shed by cats in the environment **(Bowie *et al.*1997 and Dubey, 2004)**. Toxoplasma antibodies were detected in Indian camels by **(Gill *et al.* 1990)**, in Saudi Arabia by **(Hussain *et al.* 1988**), in Abu Dhabi, United Arab Emirates by **(Afzal and Sakkir, 1994)**, in Sudan by **(Abdalla *et al*. 2014)** and in Egypt by **(Abu-Zeid , 2002).** In Egypt, the infection rate among slaughtered camels was 54.2 % using IHAT **(Derbala *et al*. 1993).** IHAT considered as a great performance tool, safety and possibilities of standardization for detection of *T.gondii* antibody titer **(Gerges, 1992)**. The prevalence of *T. gondii* infection in camels varied widely depending on the localities of the world **(FAO, 2002)**, ranging from 3.12% in Iran **(Musa, 2008)** to 90.90 % in Red sea state **(Siddig, 2010)**. The presence of *T. gondii* in camel milk **(Musa, 2008)** and edible tissues **(Elfahal *et al.,* 2013**) of carrier camels indicate the possibility of transmission to humans; particularly in pastoral communities in which raw milk, and to some extent raw meat, can be frequently consumed **(Abdel Hafez, 2013**). Recent studies revealed that infection with *T. gondii* not only affect female reproduction, but cause male reproductive impairment **(Dalimi and Abdoli, 2013 and Liu *et al.* 2016**). Infections of the genital tract in male camels may lead to temporary or permanent infertility **(Tibary *et al.* 2006**). In acute infection, of toxoplasmosis, tachyzoites may cause varying degrees of tissue destruction **(Buxton *et al.*2007)**. While, in the chronic

cases the intermediate hosts may harbor tissue cysts in the brain and other tissues and persist for the lifetime of the host **(Dubey, 2010)**. **Dass *et al.* (2011)** reported that *T. gondii* could transmit sexually in rats and *T. gondii* cysts were observed in epididymis and semen of infected male rats eight weeks post-infection. The cysts also observed in vaginal lavage of female rats 12 hours after mating with infected male rats resulting infection in female rats. Also in rabbit **(Liu *et al.* 2016),** in dog **(Arantes *et al*. 2009)**, in cattle **(Scarpelli *et al.* 2009)**, in sheep **(Moraes *et al.* 2010)** and in goat **(Santana *et al*., 2010). Monavari *et al.,* (2013) and Al-Ezzy (2014)** stated that *Toxoplasma gondii* infections impair the male fertility either directly by invading the male genital tract cells or by indirectly causing local inflammatory or immunological responses that could deteriorate reproductive functions. **Welber *et al.* (2011) and Lopes *et al.* (2012)** reported the histopathological changes in reproductive system (Testis, epididymis, seminal vesicles, and prostate) of small male ruminants after *Toxoplasma gondii* infection. The main changes were diffuse testicular degeneration associated with focal interstitial mononuclear inflammatory cells agregation. So, the current study was conducted to evaluate the seroprevalence of toxoplasmosis in Egyptian camel in different localities and its pathological alterations in the reproductive organs.

MATERIAL AND METHODS

Animals:

200-slaughtered dromedary camels (100 she-camels and 100 male camels) aged between 2- 10 years were included in this study.

Blood samples:

Jugular blood samples were collected from all animals using veinpuncture tubes before slaughtering of dromedary camels in the slaughterhouses of Egypt as Nashed and Nahia (Giza governorate);El-basateen (Cairo governorate) and Belbes abattoir (Sharkia governorate). The collected samples were kept in icebox, immediately transported to the lab. Sera were separated by centrifugation at 3200 rpm for 10 minutes. All the sera were kept in microtubes and stored at -20°C until used for serological assay.

1-Serological examination: A-Indirect hemagglutination test (IHAT):

The serum samples were tested for *T. gondii* antibodies (IgM and IgG) using indirect werke AG, W. Germany test kit is according to the manufacture instructions. All samples were serially diluted starting from dilution 1: 8 up to 1:512. All reactive serum samples by the

screening (1:8) were also retested after treatment with 2-mercaptoethanol (2-ME) in order to verify the presence of IgG antibodies **(Camargo *et al.* 1978).**

B-Animal inoculation:

Isolation of *T. gondii* by laboratory mice inoculation**:** Parts from the suspected brain, uteri of she - camels and brain, testis of male camels that were positive for Toxoplasma antibody titer were prepared for inoculation in laboratory albino healthy mice according to the method described by **Dubey (1996).**

2-Semen examination:

A-Epididymal sperm collection:

A total of 100 testis and epididymes (One for each animal) were taken immediately after slaughtering. They transported to the laboratory in sterile normal saline solution supplemented by Gentamycin. At laboratory, testis was washed by saline solution and the cauda epididymes were isolated.5 ml disposable sterile syringe containing S - TALP medium was inserted in the body of epididymis and the medium was pushed gently from the syringe with slight pressure applied all over the epididymis.

The droplets that appeared from the incisions were received in 60 mm petri dishes and placed on warm stage **(Hassanien *et al.*, 2013).**

B-Evaluation of epididymal spermatozoa:

Percentages of forward progressive motile freshly harvested epididymal spermatozoa were assessed as soon as possible through examination under (40-x) objective of phase-contrast microscope **(Plasson 1975)**. Percentages of live spermatozoa and sperm abnormalities were determined using the Eosin-Nigrosin stain **(Barth and Oko 1989).** Microscopic examination was carried out using objective (100x) oil immersion. Percentages of acrosome defects were determined in smears stained by fast green FCF according to **Wells and Awa (1970)**. Sperm cell concentration ($x10^6$/ml) was recorded using improved Neubaur heamocytometer according to **Khan (1971).** Statistical analysis was done using a computerized statistical analysis system **(Costat, 1986)**.

3-Histo-pathological examination:

A-Tissue samples:

240 tissue samples were prepared for this study, the samples were taken from 100 testis, 100 uteri and 40 brains of dromedary male and she-camels which collected from the slaughtered houses from different governorates in Egypt. Samples were fixed in 10% formal saline,

washed, dehydrated and embedded in paraffin wax. The tissues were sectioned at 3 - 4 micron thicknesses and stained with Haematoxylin and Eosin for histopathological examination according to **Bancroft *et al.*, (1996**).

B-Direct microscopic examination:

Impression smears were done from cerebral portion of the brains of the slaughtered camels which recorded positively for *T. gondii* antibody titres and also from the inoculated albino mices as well as one half of the brain of infected slaughtered camels and mice were homogenized with a manual glass homogenizer in 0.5 ml of normal saline. A small portion of each previously mentioned homogenate was spread on three slides and allowed to dry by air. All slides were stained with Giemsa stain and examined under the microscope for *T. gondii* tissue cysts according to **Dubey (1996)**.

RESULTS

1-A- Serological examination for male camel:

According to results of Indirect hemagglutination test camels of both sexes were classified into four groups: Group 1 (-ve IgG and - ve IgM ,uninfected group), Group 2 (- ve IgG and +ve IgM ,recent infection) ,Group 3 (+ve IgG and - ve IgM ,old infection) and Group 4 (+ve IgG and + ve IgM ,evolutive toxoplasmosis). From 100 male camel serum samples, were positive in IHA test with the following distribution: in the second group, in the third group and in the fourth group compared by in the first group, which represented as control group (negative in the IHA). The infection rates were 30/60 (50%), 20/60 (33.33%) and 10/60 (16.66%) in infected groups respectively.

Table (1): IgG and IgM Toxoplasma antibodies in the 4 groups of male camels.

Groups / IHAT	Group 1 -ve IgM -ve IgG	Group 2 + ve IgM -ve IgG	Group 3 -ve IgM +ve IgG	Group 4 + ve IgM + ve IgG
Total (%)	40 (40%)	30 (30%)	20 (20%)	10 (10%)
Infection rate	0	50%	33.33%	16.66%

B- Serological examination for she- camel:

Moreover, the percentage of she-camel in the 4 group according to *T. gondii* antibodies IgG and IgM by IHAT are shown in (Table 2).

From a total of 100 she- camel serum samples, were positive in the IHA test with the following distribution: in the 2nd group, in the 3rd group and in the 4th group compared by in the 1st group which represented as control group (negative in the IHA). The infection rates were 36.36% (20/55), 45.45% (25/55) and 18.18% (10/55) in infected groups respectively.

Table (2): IgG and IgM Toxoplasma antibodies in the 4 groups of she- camels.

Groups / IHAT	Group 1 -ve IgM –ve IgG	Group 2 + ve IgM -ve IgG	Group 3 -ve IgM +ve IgG	Group 4 + ve IgM + ve IgG
Total (%)	45 (45%)	20 (20%)	25 (25%)	10 (10%)
Infection rate	0	36.36%	45.45%	18.18%

C-Results of animal inoculation:

The stained slides with Giemsa stain were examined under the optical microscope revealed that tachyzoites successfully recovered from peritoneal cavity of the inoculated 4 mice after sacrified at 6th weeks post infection and freshly demonstrated as oval or slightly crescent-shaped Fig. (1). Brain cysts (Mature brain cysts and immature cysts) using brain print method and stained with Giemsa were also detected from sacrified mice at 7th weeks post infection as ovoid shaped cyst enclosed numerous numbers of bradyzoites Fig. (.2).

2- Evaluation of epididymal spermatozoa:

Table (3) outlines the effect of Toxoplasmosis represented as Igs assembly on spermiogram of male camel. The freshly collected epididymal sperm from 100 male camels were subjected to evaluation with respect to individual motility, live spermatozoa, sperm concentration and sperm abnormalities as well as acrosomal defects. Then, they divided into 4 groups based on Igs assembly as mentioned before. The mean percentages of progressive motile spermatozoa live spermatozoa, sperm abnormalities, and acrosomal defects as well as sperm concentration in the first three groups only while the fourth one was azoospermia. Analysis of variance

revealed a highly significant (P<0.01) effect of toxoplasmosis on all semen parameters in infected groups (2nd, 3rd, and 4th p) group than normal one (first group). The mean of individual motility (39.33± 1.21 and 33.25± 1.71% vs49.25± 1.09 respectively), live spermatozoa (47.83± 1.13 and 38.00± 0.83% vs, 60.25± 0.86 respectively) and sperm concentration (117.00± 2.81 and12.75± 0.49 vs 170.00± 5.43x 10^6 sperm/ ml respectively) were significantly decreased (p≤ 0.01) in infected groups compared with normal camels. On the other hand, the total sperm abnormalities(50.50 ± 1.14 and 26.00 ± 0.59 vs 16.37 ± 0.24 % respectively) and acrosomal defects (27.75 ± 0.49 and 13.50 ± 0.43 vs 6.75 ± 0.17 % respectively) were significantly increased (p≤ 0.01) in infected groups than normal one.

Table (3): Effect of Toxoplasmosis represented as Igs assembly on spermiogram of male camel (Means ±SE).

Semen parameters / Groups	Individual motility (%)	Live spermatozoa (%)	Acrosomal defects (%)	Total sperm abnormalities (%)	Epidydimal sperm conc. (x 10^6 sperm/ ml)
Group 1 IgM (- ve) IgG (-ve)	**49.25 A ± 1.09**	**60.25 A ± 0.86**	**6.75 C ± 0.17**	**16.37 C ± 0.24**	**170.00 A ± 5.43**
Group 2 IgM (+ve) IgG (- ve)	**39.33 B ± 1.21**	**47.83 B ± 1.13**	**13.50 B ± 0.43**	**26.00 B ± 0.59**	**117.00 B ± 2.81**
Group 3 IgM (-ve) IgG (+ ve)	**33.25 C ± 1.71**	**38.00 C ± 1.83**	**27.75 A ± 1.81**	**50.50 A ± 1.14**	**12.75 C ± 0.49**
Group 4 IgM (+ ve) IgG (+ve)	**Azoospermia**				

Means with different superscript A, B and C are significantly different at (P≤ 0.01).

3- Histopathological results:

The brain:

Macroscopically: the brain of infected dromedary male camels showed congestion with focal area of inflammation Fig. (3), the brain of infected dromedary she-camels showed severe congestion with focal whitish areas on the surface Fig. (4). microscopically: the examined brain of male camels infected with *T. gondii* showed focal gliosis associated with astrocytic edema Fig. (5), also degeneration of neurons associated with perivascular and pricellular edema and congestion of cerebral blood vessels and by higher magnification, it showed *T.gondii* cyst containing numerous numbers of bradyzoites Fig. (6). While the examined brain of she- camels infected with *T.gondii* showed meningitis with dilated meningial blood vessels, edema and moderate mononuclear inflammatory cells infiltration Fig. (7), also**,** encephalitis with focal degeneration of neural cells and brain cysts of *T.gondii* appeared near the degenerated neurons, by higher magnification, it showed *T.gondii* cysts containing numbers bradyzoites Fig. (8).

The testis:

Macroscopically: the testis of infected dromedary male camels showed edema and adhesion of tunica at the area of body and tail of testicle Fig. (9). microscopically: regarding the histopathological lesions of infected testis in the classified into 4groups, it was shown that, the testis of first group did not show any pathological alterations (uninfected cases). The testis of second group, group 2 (recent infection) suffered from moderate orchitis. The lesions showed vacuolar degeneration of seminiferous tubules, interstitial edema and congestion as noticed in Fig. (10), fewer numbers of spermatozoa in the lumen of seminiferous tubules. While the lesions in the epididymis of the same group showed vacuolar degeneration of tubules with interstitial edema, fibroblastic proliferation and degenerated spermatozoa in the lumen of the epididymal tubules Fig. (11). the histopathological lesions of infected testis of group 3 (old infection) suffered from chronic orchitis demonstrated that some seminiferous tubules showed vacuolar degeneration in the germinal lining while others were completely degenerated,edema and excessive interstitial infiltrates of mononuclear inflammatory cells mainly lymphocytes with less number of spermatozoa in the lumen of the tubules. Other cases showed degeneration of most of seminiferous tubules with interstitial edema. Congestion and interstitial tissue proliferation were showed Fig. (12). the lesions in the epididymis of the same group showed degeneration of epididymal tubules with degenerated spermatozoa and

tissue debris in the lumen. Diffuse peritubular and interstitial fibroblastic proliferation were showed Fig. (13), degenerated spermatozoa with spermatid giant cells in the lumen of the epididymal tubule were noticed Fig. (14). histopathological lesions in group 4 (evolutive toxoplasmosis**)** showed severe chronic orchitis; degeneration of most of seminiferous tubules and necrobiotic changes of sertoli cells, as well as excessive proliferations of the interstitial Leydig cells with peritubular edema and mononuclear cell aggregations mainly lymphocytes were noticed Fig. (15). desquamation of the germinal lining of most of the seminiferous tubules and disapearance of spermatozoa Fig. (16). degeneration of seminiferous tubules with interstitial edema and *T.gondii* (thin, ovoid) cysts containing numerous bradyzoites appeared in the interstitial tissue Fig. (17). the lesions in the epididymis of the same group showed the epididymal tubules with narrowed lumens which surrounded by thick layer of fibrous connective tissue proliferations and complete absence of spermatozoa in the lumen of the tubules were noticed Fig. (18).

The uterus:

Macroscopically: Uteri of dromedary she-camel appeared flaccid with corrigated endometium and grayish area with dark black spot in the body of the uterus was seen Fig. (19). microscopically: the uteri of she-camels infected with *T. gondii* classified into 4groups according to the serological examination. The uteri of first group could not be showed any pathological alterations (uninfected cases). The uteri of group 2 (recent infection) were suffering from moderate endometritis showed desquamation of the surface lining epithelial of sub mucosa. Sub epithelial mononuclear inflammatory cell infiltrations with edema and congestion were noticed Fig. (20). the uteri of group 3 (old infection) were suffering from chronic endometritis and showed degenerated uterine glands. Congestion with moderate infiltrations of round inflammatory cells and *T.gondii* cysts appeared between the proliferated fibrous connective tissue were noticed Fig. (21). the uteri of group 4 (evolutive *toxoplasmosis*) were suffering from severe chronic endmetritis showed degenerated and atrophied uterine glands with periglandular edema were noticed. Perivascular inflammatory cell aggregations and fibroblastic proliferations were shown in Fig. (22). the myometrium showed interstitial edema and fibroblastic proliferation with mononuclear inflammatory cells infiltration mainly lymphocytes, plasma cells and macrophages. *T.gondii* cyst containing numerous bradyzoites between muscles fibers of the myometrium was noticed Fig. (23).

DISCUSION

Animal toxoplasmosis is recognized as a cause of abortion, stillbirth, infertility and neonatal mortality in sheep and other domestic animals **(Wiss and Kim, 2009; Ortega-Mora *et al.*, 2009 and Giadinis *et al.*, 2011)**. Among farm animals, sheep and goat are more widely infected with *T. gondii* than other animals **(Soulsby, 1986, Dubey, 1994 and Santana *et al.*, 2010**). In the present study, the seroprevalence of *T. gondii* infection in dromedary male camels was 60% and in she-, camel was 55%. These results come in agreement with **Derbala *et al.* (1993)** who stated that, the infection rates among slaughtered camels in Egypt were 54.2% using IHAT. In the present study the main histopathological alteration of tissues infected with *T.gondii* revealed degenerative changes mainly vacuolar type, in addition to focal accumulations of mononuclear inflammatory cells mainly lymphocytes causing encephalitis, endometritis, orchities and epididymitis. These results come in parallel with **Beverley and Waston (1971)** in ovine, **Dubey *et al.* (1992) and Desouky *et al.* (2005)** in rabbit, **Arantes *et al.* (2009)** in male dogs and **Lopes *et al.* (2011**) in ram. This may be due to hypogonadotrophic hypogonadism resulting in hypothalamic dysfunction that caused due to *T. gondii* infection in the brain **(Topley and Wilson, 1998).** In addition to releasing of proteolytic enzymes which causing local disruption of the host cell plasmalemma as reported by **Mc-Cauley *et al.* (2014)**. The desquamation of the endometrial epithelium and atrophy of uterine glands seen in the uteri infected with *T. gondii,* these results agree with the observations of **Zare *et al.*** (**2006)** in rats**.** These uterine changes might be due to failure in ovarian activity **(Tibary *et al.*, 2001 and 2006**). In this respect**, Terpsidis *et al.* (2009)** stated that chronically infected mice for three months with *T. gondii* developed ovarian and uterine atrophy. The author attributed this condition to pituitary gonadotrophin insufficiency resulting from the inhibition of gonadotrophin releasing hormones (GnRH) from hypothalamus. In this study, the alterations due to *toxoplasmosis* in dromedary camels ranged from moderate to chronic and severe chronic cases according to the severity of exposure to the field strain infection. In addition, due to the excessive fibroblastic proliferations the uterine glandular atrophy was resulted. These investigations were resembled to that conducted by **Abdoli *et al.* (2012). Adiga and Jagetia (1999)** discussed that this condition of tissue damages may be due to the elevation of LDH in serum and plasma occurred so provide a quantitative basis for the loss of cell viability and its application in assessing the cytotoxicity of the cell**.**

In this study, the isolation of *T. gondii* from testicle, epididymis, and brain fragments by

laboratory mice inoculation carried out after histopathological examination come in line with **Haziroglu *et al.* (2003)** in rabbit, **Arantes *et al.* (2009)** in male dog, **Lopes *et al.* (2009)** in rams and **Dvorakova *et al.* (2014)** in male mice. **Dass *et al.* 2011** revealed that *T. gondii* could transmit sexually in rats. In this study, *T. gondii* cysts were observed in testicular and epididymal tissues of infected male camel. In addition, the parasite cysts were detected in endometrial and myometrial tissues of she-camels. These observations confirm the sexually transmission of *T. gondii*. Therefore, *T. gondii* gained greater opportunities for venereal transmission. Infection of the genital tract in male camel may lead to temporary or permanent infertility **(Al-Qarawi 2005; Tibary *et al.,* 2006)**. According to WHO criteria, sperm number, motility, viability and morphology are used to assess the function of spermatozoa **(WHO, 2010)**. According to histopathological and serological examination several pattern of inflammation were noticed in the present study, moderate (group 2), chronic (group 3) and severe chronic (group 4) cases of orchitis as well as epididymities. In the present study *T. gondii* infection in male camel resulted in highly significant increase in sperm abnormalities and acrosomal defects as well as decrease in percentages of sperm motility, viability and sperm concentration. These findings were agreement with **Khaki *et al.* (2011) and Al-Ghezy *et al.* (2016)** who reported the same pattern in male rat infected with *T. gondii*. Morover, **Topsides *et al.* (2009) and Abdoli *et al.* (2012**) suggested that toxoplasmosis could cause impairment on the reproductive parameters of male rats. The effect of *T. gondii* on spermatogenesis come in line with clinical studies which proved the presence of *T. gondii* in seminal fluid and tissue of infected human **(Flegr *et al.,* 2014)** which affect directly on decreasing sperm count **(Dvorakova *et al.,* 2014)** probably due to increase in apoptosis of germ cells that is triggered by a decreased gonadotropin level **(Yang *et al.*, 2006).** In the current study *T. gondii* infection in male camel resulted in highly significant increase in sperm abnormalities and acrosomal defects. These come in line with **Terpsidis *et al.* (2009) and Abdoli *et al.* (2012)** who stated that sperm abnormalities increased throughout 70 days after infection. In addition, pro- inflammatory cytokines and reactive oxygen species (ROS) may play an important role in infertility **(Agarwal *et al.,* 2008).** ROS may damage fertility by decreasing polyunsaturated fatty acids on sperm membrane including DNA damage and impairing acrosomal reaction **(Monavari *et al.,* 2013).** Regarding to group 4, the histopathological observation revealed sever chronic orchitis accompanied by testicular

dysfunction due to degeneration of seminiferous tubules which effect on fertility resulting azoospermia as recorded by semen picture analysis, these results come in parallel with **Antonios *et al.* (2000) and Derar *et al*.(2017).** It has been postulated that, the indirect effect of *T. gondii* on fertility may be mediated through altered Sertoli cell function due to the formation of immune complexes or the marked decline in testicular concentrations of both estradiol and testosterone **(Al-Qarawi 2005 and Al-Kennany, 2010).** It is known to suppress the secretion of testicular testosterone and augment the release of testicular histamine, thus apparently causing the quantitative reduction of advanced spermatogenic cells in infertile dromedary male camel **(Tibary *et al.* 2001; Al-Qarawi 2005; Tibary *et al.* 2006).**

CONCLUSION

The current study concluded that *T. gondii* infection have direct effects on sperm parameters that subsequently lead to infertility problems in male camels as well as the parasite is able to transmit with semen to she-camel. Moreover, this study suggested that, the brain, uterus and testis of infected slaughtered dromedary camels with *T. gondii* can be an important source for infection to consumers and slaughterhouse workers.

REFERENCES

Abdalla, M .I; Ahmed, A.I.; Tamador, E. E. A. and Osman, M. O. (2014): Area-wide Detection of Anti-*Toxoplasma gondii* Antibodies in Dairy Animals from the Khartoum State, Sudan. J. Life Sci.; 8, (9): 723-730**.**

Abdel-Hafez, A. A. (2013): Epidemiological Study on *Toxoplasma gondii* Infection in Beja Sheep in the Red Sea State, Sudan." Ph. D. Thesis, Sudan academy of Science.

Abdoli, A.; Dalimi, A. and Movahedin, M. (2012): Impaired reproductive function of male rats infected with *Toxoplasma gondii* Andrologia (44): 679 - 687.

Abu-zeid, Y. A. (2002): Protein G ELISA for detection of antibodies against Toxoplasma SAG I in' dromedaries. J. Egypt. Soc. Parasito.; 32: 247-257.

Adiga, S. K. and Jagetia, G.C. (1999): Effect of teniposide (Vm-26) on the cell survival, micronuclei induction and lactate dehydrogenase activity on V79 cells. Toxicology 138:29-41.

Afzal, M.; Sakkir, M. (1994): Survey of antibodies against various infectious disease agents in racing camels in Abu Dhabi, United Arab Emirates. Rev. Sc. Tech. 13 (3): 787-793.

Agarwal, A.; Makker, K. and Sharma, R. (2008): Clinical relevance of oxidative stress in male factor infertility: An update. Am J. Reprod Immunol; 59 (1):2-11.

Ahmed E, B.; Van Rijbroek, A.D.; Kal, H.B.; Sadri-Ardekani, H. and Mizrak, S.C. (2009): Proliferative activity in vitro and DNA repair indicate that adult mouse and human Sertoli cells are not terminally differentiated, quiescent cells. Biol Reprod 80: 1084 -1091.

Ahmed, A. M.; Mohammed, J. Q. and Nuhal, N.J. (2013): Affliction with toxoplasmosis among infertile men In Kamal Al-Samarae Hospital -Baghdad City. AL Taqani .26 (3):56 - 62.

Al-Ezzy, A.I. (2014): Detection of Chlamydia trachomatis immunoglobulins in seminal plasma by micro immunofluorescence test and their effect on semen parameters of infertile males in Iraq. J. Biol. Agric. Healthc; 4:48-53

Al-Ezzy, A.I. (2016): Effect of TORCH agents and Chlamydia trachomatis on reproductive parameters and fertility hormones of Iraqi infertile males.Asian J. Pharm.Clin. Res.; 9 (3): 47-56.

Al-Ghezy, Z. S.; Al-Abady, F. A. and Al-Snafi, A.S. (2016): Effect of toxoplasmosis and its treatment on male rat reproductive function. Asian J. Pharm. Sci. and Tech. 6 (2) 82 - 88.

Alireza, S. and Anja, J. (2016): parasitic diseases of camels in Iran – a literature review. Parasite. J.; 24: 21.

Al-Kennany, E.R. (2010): Pathological study on the capability of *Toxoplasma gondii* to induce oxidative stress and initation a primary lesion of atherosclerosis experimentally in broiler chickens. J Anim Vet Adv, 6, 938-942.

Al-Qarawi, A. A.; Omar, H. M.; Abdel- Rahman, H. A.; El- mougy, S. A. and El-belaly, M. S. (2005): infertility in the dromedary bull: Areview of causes, relation and implication. Anim. Rep. Sci (87) 73- 92.

Antonios, S.; Ismail, H. and Essa, T. (2000): Hypothalamic origin of reproductive failure in chronic experimental toxoplasmosis. J. Egypt Soc. Parasitol. 30 (2):593-599.

Arantes, T.P.; Lopes, Z. W. D.;Ferreira, R. M.; Pinto Pieroni, J. S.; Pinto, V. M. R.; Sakamoto, C. A.; da Costa, A. J. (2009): *Toxoplasma gondii:* evidence for the transmission by semen in dogs. Exp. Parasitol. 123:190 -194.

Bancrof, J. D.; Stevens, A. and Turner, D. R. (1996): Theory and practice of histological technique, 4th Ed., Churchill Livingstone Co., New York, USA.

Beverley, J.K. A.;Waston, W. A.(1971): Prevention of experimental and of naturally occurring ovine abortion due to toxoplasmosis. Vet. Rec. 88:39 - 44.

Boothroyd, J.; Black, M.; Bonnefoy, S.; Hehl, A.; Manger, I. and Tomavo, S. (1997): Population biology of *Toxoplasma gondii* and its relevance to human infection. Curr Opin Microbiol; 5: 438 - 442.

Bowie, W.R.; King, A.S.; Werker, D.H.; Isaac-Renton, J.L.; Bell, A.; Eng, S.B.V and Marion, S.A. (1997): Outbreak of toxoplasmosis associated with municipal drinking water. Lancet, 350, 173-177.

Buxton, D.; Maley, S.W.; Wrlght, S.E.; Rodger, S.; Bartley, P. and Innes, E.A. (2007): *Toxoplasma gondii* and ovine toxoplasmosis: New aspects of an old story. Vet. Parasitol, 149: 25-28.

Camargo, M.E.; Ferreira, A.W.; Mineo, J.R.; Takiguti, C.K.; Nakahara, O.S (1978): Immunoglobulin G and immunoglobulin M enzyme-linked immunosorbent assays and defined toxoplasmosis serological patterns. Infect Immun 21: 35-38.

Costat Computer Program Copyright (1986): Version 3:03 copyright software.

Dalimi, A. and Abdoli, A. (2013): *Toxoplasma gondii* and male reproduction impairment: A new aspect of Toxoplasmosis research. Jundishapur J. Microbiol.; 6 (8): e7184.

Dass, S. A.; Vasudevan, A.; Dutta, D.; Soh, L. J.; Sapolsky, R, M. and Vyas, A. (2011): Pro tozoan parasite *Toxoplasma gondii* manipulates mate choice in rats by enhancing attractiveness of males. *PLoS One.*; 6 (11).

Derar R, D. R; Ali, A.; Osman, S, A.; Al-Sobayil, S. A.; Elhassan Saeed, E. M. A; Hassanein, K. and Al-Hawas, A. (2017): Potential pathogens in infertile male dromedary camels and their association with the spermiogram and clinical findings. Comp. Clin. Pathol. 26: 965.

Derbala, A. A., Zayed, A. A. and Tawfik, M. A. (1993): Prevalence of Toxoplasma antibodies in serum of cattle and camels. J. Egyptian Vet. Med. Assoc. (53): 319 -324.

Desouky, H.M., Barakat, A.M. and Abd El-Razik, K.A. (2005):"*Toxoplasma gondii* in rabbit-does: serological, hormonal, pathological and molecular aspect" Egypt.J. Comp.Path. And Clinic. Path. 18:145 - 167.

Dubey, J. P. (2004): Toxoplasmosis-a waterborne zoonosis. Vet Parasitol. 126:57-72.

Dubey, J. P. (2010): Toxoplasmosis of Animals and Humans, 2nd edn. CRC Press, Boca Raton, FL, USA.

Dubey, J.P. (1994): Parasitemia and tissue infection in sheep fed *Toxoplasma gondii* oocysts. J Parasitol 66:111-119.

Dubey, J.P. (1996): Pathogenicity and infectivity of *Toxoplasma gondii* oocysts for rats. J. Parasitol., 82, 951-956.

Dubey, J.P.; Brown, C.A.; Carpenter, J.L.; Brandelti, E. and Moore, J.J. (1992): Fatal toxoplasmosis in domestic rabbits in the USA. Vet Parasitol; 44:305-309.

Duncanson, P.; Terry, R. S.; Smith, J .E. and Hide, G. (2001): High levels of congenital transmission of *Toxoplasma gondii* in a commercial sheep flock. Int. J.Parasitol., 31, 1699-1703.

Dvorakova, K.; Sidlova, A.; Ded, L.; Hladovcova, D.; Vieweg, M.; Weidner, W., Steger, K.; Stopka, P. and Dogan, A. P. (2014): *Toxoplasma gondii* decreases the reproductive fitness in mice. *PLoS ONE*, 9 (6): 69-70.

Elamin, E.A. and Elias, S. (1992): Prevalence of *Toxoplasma gondii* antibodies in pastoral camels (*Camelus dromedarius*) in the Butana plains, mid-Eastern Sudan Veterinary Parasitology, pp 171-175.

Elfahal, A. M., Amira, M. E., Hussien, M. O., Enan, K.A., Musa, A. B., and El Hussein, A. M. (2013): Seroprevalence of *Toxoplasma gondii* in dairy cattle with reproductive problems in Sudan." Veterinary Sci. 19 (5) 220 -222.

Fahmy, A. A.; Mandour, A. M.; Arafa, M. S.; Abdel Rahman, B. M., (1979): Toxoplasmosis of camels in Assiut governorate. J. Egypt. Vet. Med. Assoc. 3 (2):180 - 4.

Flegr, J.; Klapilov, K. and Ka, K. (2014): Toxoplasmosis can be a sexually transmitted infection with serious clinical consequences. Not all routes of infection are created equal. Med. Hypotheses. 83:286-9.

Food and Agriculture Organization (2002): Manual for laboratory diagnosis of infectious abortion in small ruminants. FAO, pp. 97-109.

Gerges, A. A. (1992): Serological investigation on ovine Toxoplasmosis in Egypt. M. V. Sc. Thesis, Parasitology, Fac. Vet. Med. Cairo Univ.

Giadinis, N.D., Terpsidis, K., Diakou, A., Siarkou, V., Loukopoulos, P., (2011): Massive toxoplasma abortions in a dairy sheep flock and therapeutic approach with different doses of sulfadimidine. Turk. J. Vet. Anim. Sci., 35(3), 207-211.

Gill, H.S.; Prakash, O.L.; Sheehan, D.C. and Hrapchak, B.B. (1990): Toxoplasmosis in India: prevalence of antibodies in camels. Trop. Med. Parasitol. 63: 265-267.

Hassanien, T. K.; Rewash, M. Z. and Abdelaziz, E. E. (2013): Pathological studies on the effect of mycoplasma infection in male dromedary camel. Egypt. J. Comp. Path and Clinic. Path.; 26 (1): 187- 208.

Haziroglu, R.; Altintas, K.; Atasever, A.; Gulbahar, M.Y. and Tunca, O.R. (2003): Pathological and immunohistochemically studies in rabbits experimentally infected with *Toxoplasma gondii*. Turk. J .Vet. Anim .Sci; 27: 285-293.

Hussain, M. F.; Bakkar, N.; Basmaeil, S. M.; Gar EINabi, A. R. (1988): Prevalence of toxoplasmosis in Saudi Arabian camels (Camelus dromedaries). Vet. Parasitol. 28: 175-178.

Khaki, A.; Farzadi, L.; Ahmadi, S.; Ghadamkheir, E.; Khaki, A. A.; Saedeh, S. and Sahizadeh, R. (2011): Recovery of spermatogenesis by Allium cepa in *Toxoplasma gondii* infected rats. Afri. J. Pharm. and Pharmacol. 5 (7): 903 - 907.

Khan, A. A. (1971): Sexual behavior of the male camel (Camelus dromedaries) and studies on semen. M. V.Sc. Thesis, Bikaner Univ., Udaipur, India.

Kijlstra, J. and Jongert, N. F. (2008): Studies on the significane of sexual intercourse for the transmission of *Toxoplasma gondii.* Zent. Bl. bakteriol .231:323 - 332.

Liu, S. G.; Qin, C.; Yao, Z. J. and Wang, D. (2016): Study on the transmission of Toxoplasma *gondii* by semen in rabbits. Zhongguo Ji Sheng Chong Xue Yu Ji Sheng Chong Bing Za Zhi.; 24 (3):166 -70.

Lopes, W. C.; da Costa, A. J.; Santana, L. F.; Dos Santos, R. S. and Rossanese. W. (2009): Aspects of toxoplasma infection on the reproductive system of experimentally infected rams (Ovis Aries). J Parasitol Res., 111 (2 - 4): 312- 9.

Lopes, W. D.; Santos, T. R.; Luvizotto, M. C.; Sakamoto, C. A.; Oliveria, G. P. and Costa, A. J. (2011): Histopathology of the reproductive system of male sheep experimentally infected with *toxoplasma gondii.* Parasitol Res., 109 (2): 405 - 9.

Lopes, W. D.; Santos, T. R.; Luvizotto, M. C.; Sakamoto, C. A.; Oliveira, G.P. and Costa, A. J. (2012): Histopathology of the reproductive system of male sheep experimentally infected with *Toxoplasma gondii.* Parasitol Res.; 109 (2):405**.**

McCauley, J.B.; Jones, J.L. and Singh, K. (2014): Manual of Clinical Microbiology. Toxoplasma. 11th ed. Washington DC: ASM Press.

Monavari, S. H.; Vaziri, M. S.; Khalili, M.; Shamsi-Shahrabadi, M.; Keyvani, H.; Mollaei, H. *et al.* (2013): Asymptomatic seminal infection of herpes simplex virus: Impact on male infertility. J Biomed Res.27 (1):56 -61.

Moraes, E. P.; Freitas, A. C.; Gomes-Filho, M. A.; Guerra, M. M.; Silva, M. A.; Moraes, E. P. B. X. ;Freitas, A. C. and Gomes, M. A. (2010): Characterization of reproductive disorders in ewes given an intrauterine dose of *Toxoplasma gondii* tachyzoites during the intrauterine insemination Anim. Reprod. Sci.; 122 (1-2): 36 - 4.

Musa, H. O. (2008): Detection of *Toxoplasma Gondii* Antibodies in Females in Port Sudan City." B.Sc. thesis, University of Ahlia, ortSudan.

Nimri, L.; Pelloux, H. and Elkhatib, L. (2004): Detection of *Toxoplasma gondii* DNA and specific antibodies in high-risk pregnant women. Am. J. Trop. Med. Hyg., 71,831-835.

Nourian, A.(1992): Serological study of toxoplasmosis in camels of Fars Province. DVM Dissertation, Shiraz University Parasitology Research, 98 (6), 600 - 601.

Ortega-Mora, L. M., Gottstein, B., Conraths, F. J., and Buxton, D. (2009): Protozoal abortion in farm ruminants, Guide lines for diagnosis and control. London: CAB International.

Plasson, S.J. (1975): Veterinary Obstetries and Gynecology. William and Wilson Company, 4th (Ed.), New York, USA, pp. 415 - 486.

Safarpoor Dehkordi, F.; Haghighi Borujeni, M. R.; Rahimi, E. and Abdizadeh R. (2013): Detection of *Toxoplasma gondii* in raw caprine, ovine, buffalo, bovine, and camel milk using cell cultivation, cat bioassay, capture ELISA, and PCR methods in Iran. Foodborne Pathogens and Disease, 10 (2), 120-125.

Santana, L. F.; da Costa, A. J.; Pieroni, J.; Lopes, W. D. Z.; Santos, R. S.; de Oliveira, G. P.; de Mendonc¸a, R. P. and Sakamoto, C. A. M. (2010): Detection of *Toxoplasma gondii* in the reproductive system of male goats. Rev. Bras. Parasitol. Vet. (19):179 -182.

Sazmand, A.; Tavassoli, M.; Ebrahimnejad, B.; Kazemnia, A. and Asadollahi, Z. (2014): PCR assays for detection of *Toxoplasma gondii* infection in Iranian camels (Camelus dromedarius) of Yazd Province. Journal of Veterinary Clinical Pathology, 8 (29), 383 -389.

Scarpelli, L.; Lopes, W. D. Z.; Migani, M.; Bresciani, K. D. S. and Costa, A. J. (2009): *Toxoplasma gondii* in experimentally infected Bos tau- rus and Bos indicus semen and tissues. Pesq. Vet. Bras. (29):59 - 64.

Siddig, A. B. (2010): Study on Toxoplasmosis in Humans and Cats in the Red Sea State." M.Sc. Thesis, SUST.

Slifko, T. R.; Smith, H. V. and Rose, J. B. (2000): Emerging Parasite Zoonoses Associated with Water and Food." Int. J. Parasitol. 30: 1379 -1393.

Soulsby, E.J.L. (1986): Helminths, Arthopods, and protozoa of domesticated animals. 7th edition. Bailliere, Tindall, London, 680p.

Terpsidis, K.I.; Papazahariadou, M. G.; Taitzoglou, I. A.; Papaioannou, N. G.; Georgiadis, M. P. and Theodoridis, I.T.(2009): *Toxoplasma gondii*: Reproductive parameters in experimentally infected male rats. Exp Parasitol, 121:238 - 41.

Tibary, A. C. F.; Anouassi, A.; and Sghiri, A. (2006): Infectious cause's loss in camelids. Establishment of PCR assay. Theriogenology, 66: 663- 647.

Tibary, A.; Anouassi, A. and Memon, M. A. (2001): An approach to the diagnosis of infertility in camelids: retrospective study in alpaca, llamas and

Topley and Wilson (1998): "Microbiology and microbial infections" 9th Ed. Vol.5 (Parasitology) Eds: Francis E.G. Cox, Julius P. kreir and Derek Wakelin. Arnold. Oxford University. Press, New York.

Topsides K.; Papazahariadou, M.; Taitzoglou, I.; Papaioannou, N.Georgiadis, M. and Theodoridis, I. (2009): Reproductive parameters in experimentally infected male rats with *Toxoplasma gondii*. Exp. Parasitol. 121:238 -241.

Welber ,D. Z. L.; Thaís, R. S.; Luvizotto, M. C. R.; Sakamoto, C. A. M. Oliveira, G. P. and Costa, A. J. (2011): Histopathology of the reproductive system of male sheep experimentally infected with *Toxoplasma gondii* .Parasitology Research, 109 (2): 405 - 409.

Wells, M. E. and Awa, O. A. (1970): New technique for assessing acrosomal characteristics of spermatozoa. J. Dairy Sci., 53: 227- 232.

Williams, R.H., Morley, E.K., Hughes, J.M., Duncanson, P., Terry, R.S., Smith, J.E. and Hide, G. (2005): High levels of congenital transmission of *Toxoplasma gondii* in longitudinal and cross-sectional studies on sheep farms provides evidence of vertical transmission in ovine hosts. Parasitol, 130, 301-307.

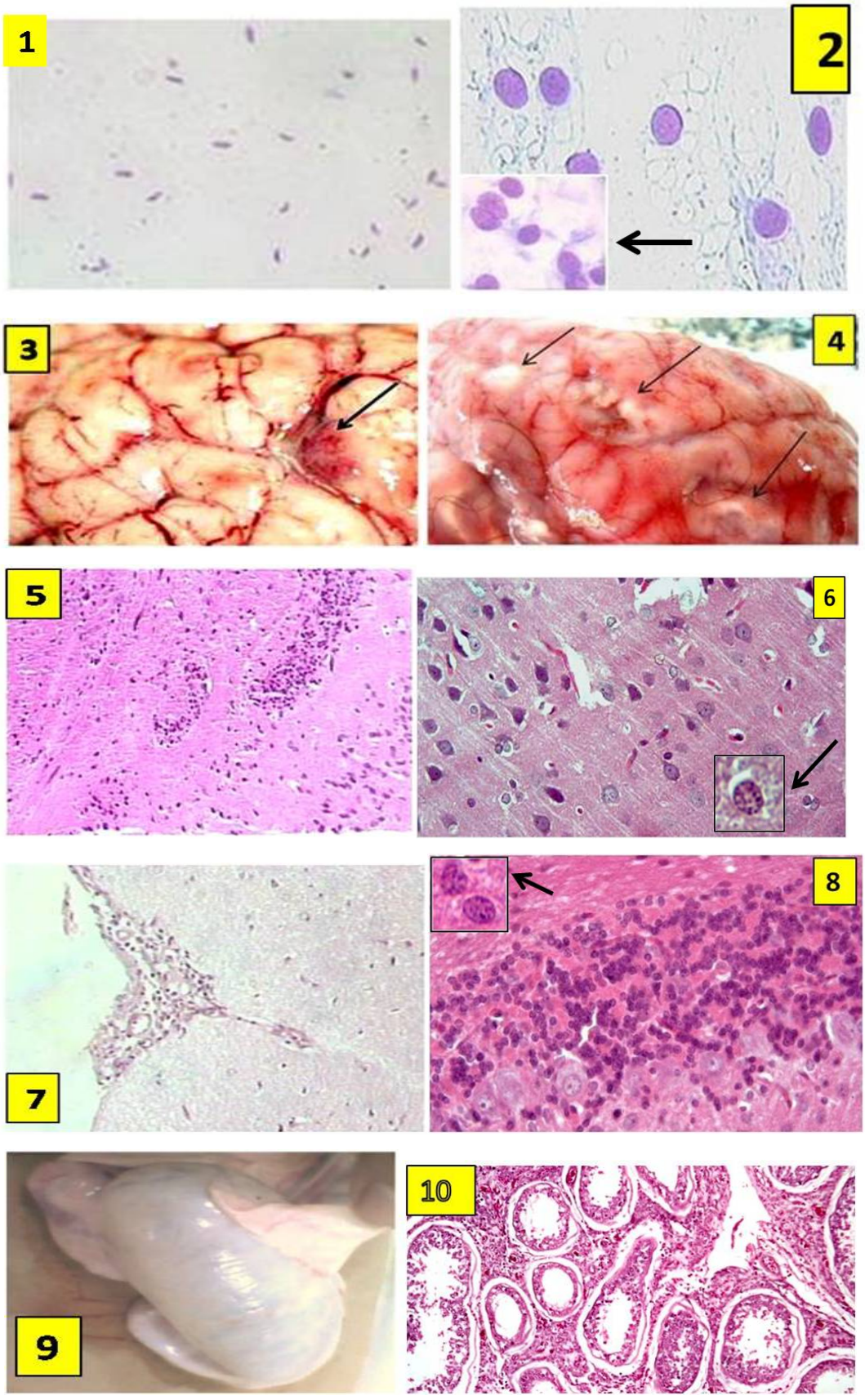
Weiss, L.M.; Kim, K. (2009): Toxoplasmosis: A history of clinical observations. *Int J Parasitol.*; 39 (8):895 -901.

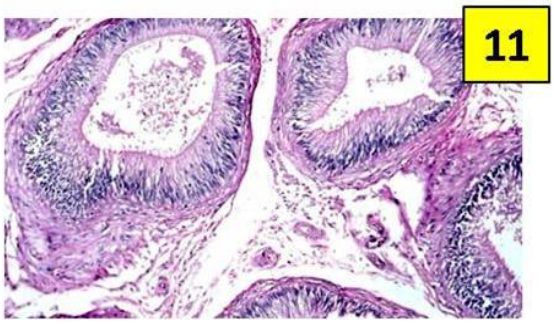
World Health Organization (2010): Laboratory Manual for the Examination and Processing of Human Semen, 5th edn. WHO Press, Geneva, Switzerland.

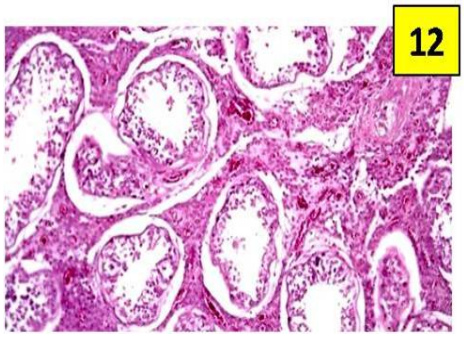
Yang, H. S.; Han, D. K.; Kim, J. R. and Sim, J. C. (2006): Effects of alpha- tocopherol on cadmium-induced toxicity in rat testis and spermatogenesis. J. Korean. Med. Sci. (21): 445 - 451.

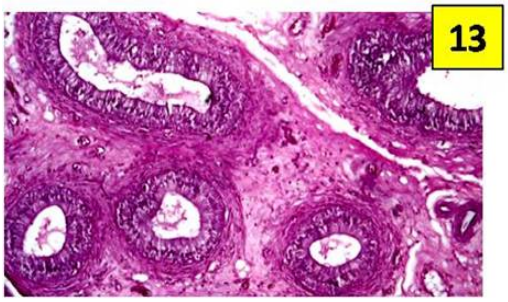
Zare, F.; Dalimi, A. and Ghaffarifar, F. (2006): Detection of active *Toxoplasma gondii* (RH strain) in the different body tissues of experimentally infected rats. *Modares J Med Sci*; 9 (1):19 -23.

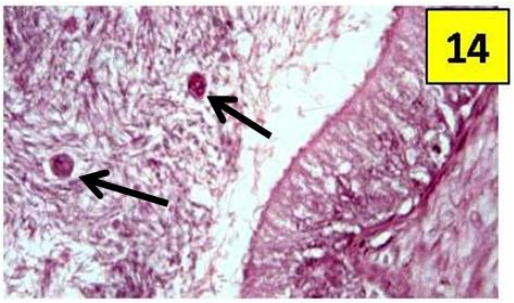
Zhang, G. N.; Ibragimova, N. M.; Simoni P.S. and Lopatinkiova, V. N. E. (1999): Epidemiological study on Toxoplasma infection in human beings and animals in Shandong Province. Zhonghua Liu Xing Bing Xue Za.; 10: 30-33.

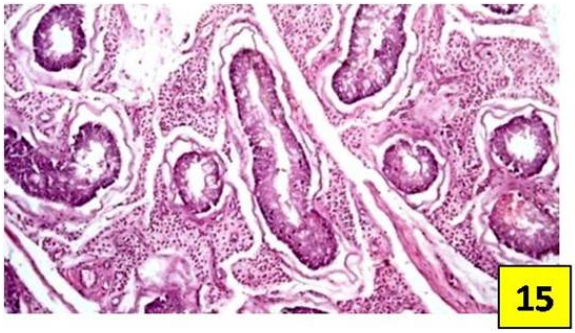

1
2
3
4
5
6
7
8
9
10

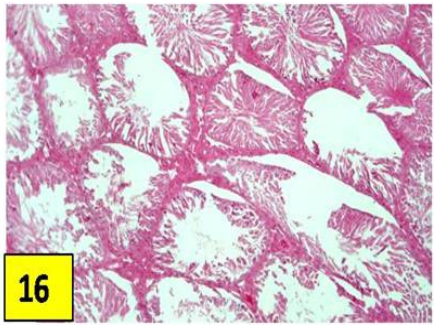

11

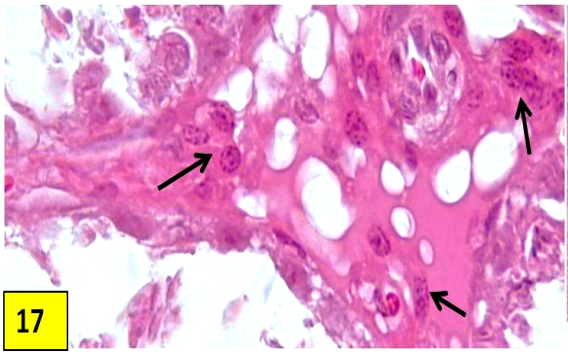

12

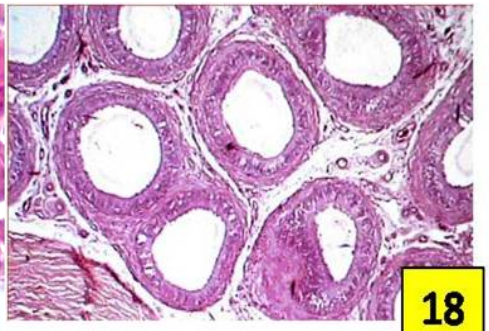

13

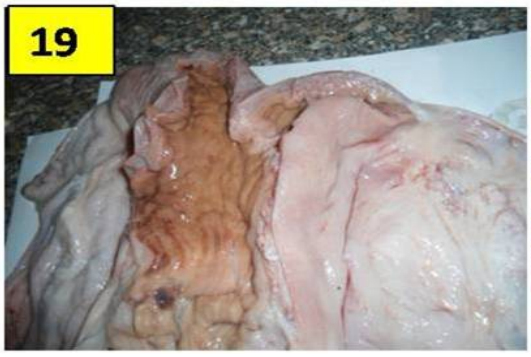

14

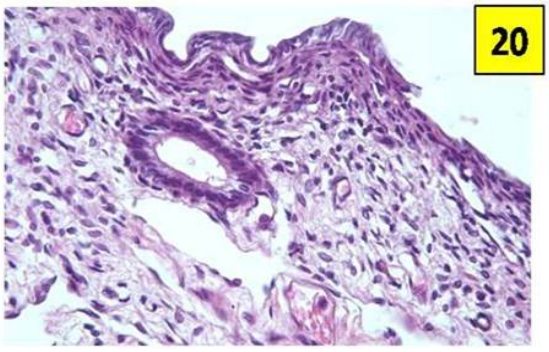

15

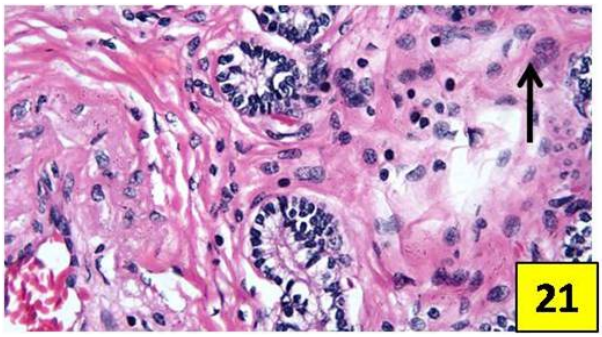

16

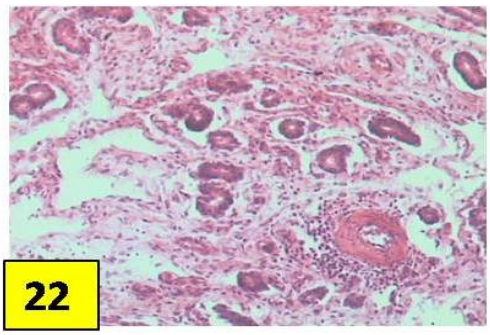

17


18


19


20





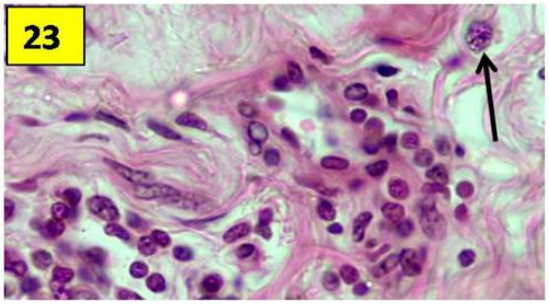


Fig. (1): Peritoneal fluid smear obtained from peritoneal cavity of experimentally infected mice by *T. gondii* .The tachyzoites appeared slightly crescent or oval in shape, stained with Giemsa stain.**X400.**

Fig. (2): Brain of mice (by brain print method) showed mature brain cysts of *T.gondii* and the immature cysts (arrow) stained with Giemsa stain. The cysts appeared ovoid in shape and enclosed high numbers of bradyzoites. **X1000.**

Fig. (3): Brain of infected dromedary male camel showed congestion with focal area of inflammation (arrow).

Fig.(4): Brain of infected dromedary she-camel showed congestion with focal whitish areas (arrows).

Fig. (5): Brain of male camel infected with *T. gondii* showed focal gliosis associated with astrocytic edema. **H&E. X100.**

Fig. (6): Brain of male camel infected with *T. gondii* showed degeneration of neurons associated with perivascular and pricellular edema and congestion of cerebral blood vessels.**H&E.X400.** Higher magnification showing *T.gondii* cyst containing numerous bradyzoites (arrow) **X1000.**

Fig. (7): Brain of she- camel infected with *T.gondii* showed meningitis with dilated meningial blood vessels. Edema and moderate infiltration of mononuclear inflammatory cells was noticed**. H&E.X100.**

Fig. (8): Brain of she-camel infected with T.gondii showed encephalitis with focal degeneration of neural cells. The brain cysts of *T.gondii* near the degenerated neurons. **H&E. X400**. Higher magnification showed *T.gondii* cysts containing numbers of bradyzoites (arrow) **X1000.**

Fig. (9): Testicle of infected dromedary male camel showed edema and adhesion of tunics at the area of body and tail of testicle.

Fig. (10): Testis of camel infected with *T.*gondii, group (2) showing vacuolar degeneration of seminiferous tubules. Interstitial edema and congestion was noticed. **H&E. X100.**

Fig. (11): Epididymis of camel infected with *T.gondii, group* (2) showing vacuolar degeneration of tubules with interstitial edema and fibroblastic proliferation .Degenerated spermatids in the lumen of the epididymal tubules was noticed.**H&E.X100.**

Fig. (12): Testis of camel infected with *T.gondii, group* (3) showing degeneration of most of seminiferous tubules with interstitial edema. Congestion and interstitial tissue proliferation was shown. **H&E.X100.**

Fig. (13): Epididymis of camel infected with *T.gondii*, *group* (3) showing degeneration of epididymal tubules with degenerated spermatozoa and tissue debris in the lumen. Diffuse peritubular and interstitial fibroblastic proliferation was noticed. **H&E. X100.**

Fig. (14): Epididymis of camel infected with *T.gondii* ,*group* (3) showing the degenerated spermatozoa and spermatid giant cells in the lumen of the epididymal tubules (arrow).**H&E.X400.**

Fig.(15): Testis of camel infected with *T.gondii*, *group* (4) show the degeneration of most of seminiferous tubules and necrobiotic changes of sertoli cells, Excess proliferations of the interstitial Leydig cells with peritubular edema and mononuclear cell aggregations was shown. Complete absence of spermatozoa in the lumen of the tubules. **H&E.X100.**

Fig.(16): Testis of camel infected with *T.gondii*, *group* (4) showed desquamation of the germinal lining of most of the seminiferous tubules and disapearance of spermatozoa. **H&E.x100.**

Fig.(17): Higher magnification of Fig. (16) showing degeneration of seminiferous tubules with interstitial edema and *T.gondii* (thin, ovoid) cysts containing numerous bradyzoites appeared in the interstitial tissue (arrows). **H&E.x400.**

Fig.(18): Epididymis of camel infected with *T.gondii, group* (4) show the epididymal tubules with narrowed lumen surrounded with thick layer of fibrous connective tissue proliferations. Complete absence of spermatozoa in the lumen of the tubules.**H&E.X100.**

Fig. (19): Uterus of dromedary she-camel appeared flaccid with corrigated endometium. Grayish area with dark black spot in the body of the uterus was noticed.

Fig. (20): Uterus of she-camel infected with *T.gondii*, group (2) showed desquamation of the surface lining epithelial of sub mucosa .Sub epithelial mononuclear inflammatory cells infiltration with edema and congestion were noticed.**H&E.X100.**

Fig. (21) Uterus of she-camel infected with *T.gondii*, group (3) showed degenerated uterine glands and congestion with moderate infiltrations of round inflammatory cells and *T.gondii* cyst appeared between the proliferated fibrous connective tissue (arrow).**H&E.X400.**

Fig. (22): Uterus of she-camel infected with *T.gondii*, group (4) showed degenerated and atrophied uterine glands with periglandular edema. Perivascular inflammatory cell aggregations and fibroblastic proliferations were shown. **H&E. X100.**

Fig. (23): Uterus of she-camel infected with *T.gondii*, group (4) showed interstitial edema with fibroblastic proliferations and inflammatory round cells infiltration. *T.gondii* cyst containing numerous bradyzoites between the muscle fibers of the myometrium (arrow).**H&E.X400.**