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A NOTE ON CAMEL TOXOPLASMOSIS IN THE SUDAN

(With 1 Table)

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SUMMARY

Toxoplasmosis is a common parasitic zoonosis and an important cause of abortions, encephalitis, blindness, mental retardation and death worldwide. In this article some findings relevant to *Toxoplasma gondii* infection in camels are discussed. Recorded infections of camel toxoplasmosis, as measured by serological surveys, in various parts of the country are astonishingly high. The prominent importance of toxoplasmosis coupled with rarity and scantiness of data and knowledge concerning *Toxoplasma gondii* status in Sudan seems to justify addressing data relative to causative organism, prevalence, recent diagnostic techniques, chemotherapy, with especial emphasis on its economic impact as well as public health significance.

INTRODUCTION

Toxoplasmosis is one of the most significant animal zoonosis, distributed worldwide and affecting almost all warm-blooded animal species, and especially humans (Tenter et. al., 2000). The causative agent of toxoplasmosis is a coccidian parasite, *Toxoplasma gondii* so named because the organism was first identified in an African rodent called a gondi. The word toxoplasma was derived from the Greek word "toxon" or arc, reflecting the shape of the parasite by light microscopy (Nath and Sinai, 2003). Although cats serve as natural reservoirs of toxoplasma, virtually any animal that ingest material contaminated with oocysts can get infected. The infection can be further transmitted if toxoplasma contaminated tissues are eaten by another warm-blooded host including humans (Nath and Sinai, 2003).

Toxoplasma exists in three developmental forms, sporozoites in oocysts, the slow growing bradyzoites in tissue cysts, and tachyzoites the rapidly growing form of the parasite (reviewed by Dubey, 1998a). Oocysts are found only in cats and are infective only when completely sporulated (Dubey, 1998a). Sporulated oocysts of *Toxoplasma gondii* are very resistant to environmental conditions. They remain infectious in moist soil or sand for up to 18 months (Frenkel, 2000). However, they were killed within 1-2 minutes by heating to 55-60°C (Dubey, 1998b). Sporulated oocysts are highly impermeable and, therefore, are also very resistant to disinfectants (Frenkel, 2000).

Humans and animals acquire infection following oral ingestion of any of the three life stages tachyzoites, bradyzoites (contained in tissue-cysts) in raw or undercooked meat or infected animal tissues or by ingestion of milk contaminated with tachyzoites (Rieman and Meyer, 1975), and sporozoites contained in sporulated oocysts) from the environment, or transplacentally when the host has its primary infection during gestation (Ettinger, 2001), and by blood transfusion or organ transplantation (Dubey, 1993). The life cycle of toxoplasma is reviewed by Dubey (1998a).

In heavy infections, the multiplying tachyzoites may produce areas of necrosis in vital organs such as the myocardium, lungs, liver and brain and during this phase the host can become pyrexic and lymphadenopathy occurs. As the disease progresses bradyzoites are formed, this chronic phase being usually a symptomatic (Uroquhart *et al.*, 1996).

Undoubtedly the most important role of toxoplasmosis in ruminants is its association with abortion in ewes and perinatal mortality in lambs. If the foetus survives in utero, the lamb may be stillborn or, if alive, weak (Uroquhart *et al.*, 1996).

Humans may become infected with toxoplasma at any time during life (Tenter *et al.*, 2001). Seroprevalence rates increase with age and are much higher in populations where ingestion of uncooked meat is common (Tenter *et al.*, 2001). In immune competent patients, the infection is asymptomatic, but some individuals may develop

a mononucleosis-like syndrome. These patients typically develop lymphadenopathy, splenomegaly, a non-specific illness (low grade pyrexia, malaise, myalagia), or ocular manifestations. In the immunocompromised patient, more severe disease may develop (Nath *et al.*, 1987). The most serious manifestation of the disease occurs in congenital foetal involvement in human being. Abortion is a sequel in severe infections acquired early in pregnancy (before 26 weeks of gestation with highest risk at 10-24 weeks) (Nath and Sinai, 2003), and if a child is born alive he may suffer from serious mental retardation within a few weeks after birth (Dureden *et al.*, 1987).

Diagnosis of toxoplasmosis on clinical ground is usually difficult, and recourse must be made to the demonstration of either the organism or antibodies against it. The most convincing diagnosis is the isolation of the parasite by inoculation of suspect material into mice (Solusby, 1982). It has the disadvantage that unless the strain of toxoplasma is highly virulent, it requires three weeks before examination of the mice will yield recognizable Toxoplasma cysts (Uroquhart *et al.*, 1996). In the main, diagnosis is based on a correlation of clinical and serological findings (Manal, 2003). The most useful and widely studied methods for serodiagnosis are: dye test (Sabin and Feldman, 1948), indirect immunofluorescence antibody test (Remington *et al.*, 1968), direct and indirect haemagglutination test (Jacobs and Lunde, 1957). More recently, ELISA test has been developed which is capable of detecting a recent infection by the estimation of IgM, as compared to IgG, antibody (Uroquhart *et al.*, 1996). Zhang and Wei (2001) reported that Modified Agglutination Test (MAT) and Latex Agglutination Test (LAT) could alternatively be used for the diagnosis of toxoplasmosis. Zhang *et al.* (1999), suggested that Immunosorbent Agglutination Assay (IgM, ISAGA) is a sensitive, specific, easy to perform, and is useful for mass screening and diagnosing recent toxoplasmosis infection or reactivation. Polymerase chain reaction (PCR)-based testing has become the preferred method for diagnosis, occasionally replacing tissue biopsy (Lewis *et al.*, 2002).

The choice of drugs for treating cerebral toxoplasmosis in humans is currently limited. There are only three drugs available, and of these, pyrimethamine and sulphonamide are invariably used in combination. Clindamycin is an alternative choice. Another drug, spiramycin, has poor central nervous system penetration but achieves high concentrations in the placenta and is useful for treatment of toxoplasmosis during pregnancy (Nath and Sinai, 2003). In cats and dogs as presented in Table (1), Clindamycin hydrochloride or trimethoprim-sulphonamide combination administered for four weeks can be utilized. Other anti Toxoplasma drugs include doxycycline, minocycline, azithromycin, and clarithromycin (Ettinger, 2001).

The purpose of this study was to review the prevalence of Toxoplasma antibodies in camels in the Sudan and to address some data related to its impact on reproductive performance of camel, as well as public health significance, especially among the nomads who consume cameline milk and raw liver.

Camel toxoplasmosis:

Camels constitute one of the most useful domestic animals particularly for nomads. Besides their social and economical status in the Sudan they are used for food, transport and sport. In the Sudan, the one humped camel "Camelus dromedarius" plays a very important role in the national income and is a source of meat, milk, hair and hides, and constitutes a major item in the livestock foreign trade list. Estimations of the camel population in the Sudan are about 3.1 million head (Schwartz and Dioli, 1992) to 3.3 million head (Comprehensive National Strategy Statistics, 2001). The main camel zone in the Sudan extends between latitude 10° and 20°N. it is bounded by the Ethiopian mountains and the Red Sea hills on the East and Ingasana mountains and Bahr-Elarab in the South (Babiker, 1984). Camel milk is an important staple for the pastoralists, but there appears to be little, if any, marketing of this commodity.

In Sudan camels have multifacet economical impacts. Slaughtered-camels export, mainly young camels, is the mainstay of Sudan's trade with neighbouring countries (Egypt, Libya and Saudi Arabia). Racing camel breed is highly valued in Saudi Arabia and the Gulf States. Local markets on the other hand, absorb meat of old unproductive animals, hide for manufacture of leather goods and limited amount of hair.

Toxoplasma antibodies were detected in Indian camels by Gill and Prakash (1969), in Saudi Arabia by Hussain *et al.* (1988), in Abu Dhabi by Afzal and Sakkir (1994), and in Egypt by Abu-zeid (2002), Fahmy *et al.* (1979).

Numerous reports revealed widespread prevalence of toxoplasmosis among Sudanese camels (El Din *et al.*, 1985, Abbas *et al.*, 1987; Bornstein and Musa, 1987; Elamin *et al.*, 1992;

and Manal, 2003). Variable seropositivity tests were reported among camels in the Sudan. Using the indirect haemagglutination tests (IHA), seropositivity rates of 12% (Abbas *et al.*, 1987); 22.5% (Bornstein and Musa, 1987); and 54% (El Din *et al.*, 1985).

A total of 482 serum samples from pastoral camels in the Butana plains, mid-Eastern Sudan, were tested for Toxoplasma antibodies by latex agglutination test (LAT) by Elamin and his colleagues (1992). Sixty-seven percent 67% of the camels were seroreactive. Manal (2003), detected *Toxoplasma gondii* antibodies in different locations of camels in the Sudan, using latex agglutination test. The overall prevalence was 61.7%; 64% in Butana; 59% North Kordofan; and 51% in River Nile. No sex-linked difference in seroreactivity among camels (Elamin *et al.*, 1992, Fahmy *et al.*, 1979; Manal, 2003). A positive correlation between seroreactivity and age among camels reported by Elamin *et al.*, (1992) and Manal (2003), that agree with findings of Fahmy *et al.* (1979) in Egypt and Hussain *et al.*, (1988) in Saudi Arabia. It is conceivable that the longer an animal lives, the greater the chance of its being exposed to *Toxoplasma gondii* (Elamin *et al.*, 1992). Acquisition of Toxoplasma infection by camels is thought to occur through ingestion or inhalation of sporulated oocysts that are shed by cats in the environment (Elamin *et al.*, 1992).

Toxoplasma gondii oocysts were isolated from kittens that were fed raw cameline meat. Toxoplasma tachyzoites and cysts were detected in the brain of suckling calf-camels and mice inoculated with milk of three experimentally infected lactating she-camels (Manal, 2003). The outcome of *Toxoplasma gondii* infection varies according to the time of infection during pregnancy and the number of parasites inoculated, into pregnant she-camels. In general congenital toxoplasmosis in camels result in delivering weak (unable to stand), and had diarrhoea, refused suckling calves that may die soon after birth (Manal, 2003).

Conclusion: In this report the high prevalence of *Toxoplasma gondii* reported in pastoral camels in the Sudan may be of public health significance, since nomads consume milk of camels raw. Although tachyzoites are sensitive to proteolytic enzymes and are destroyed by gastric digestion, a recent study showed that tachyzoites survived for up to two hours in acid pepsin solutions, and that oral application of tachyzoites might have caused an infection (Dubey, 1998c). Rieman and Meyer (1975), Sacks *et al.*, (1982), suggested that tachyzoites may enter the host by penetration of mucosal tissue and thereby gain access to the host's circulation or lymphatic system before reaching stomach. Recognition of transplacental toxoplasmosis in camels is important from economic and public health points of view as cats may eat infected placentae, and hence shed millions of oocysts in its faeces.

REFERENCES

Abbas, B.; ElZubeir, A.E.A.; Yassin, T.T.M., (1987). Survey for certain zoonotic diseases in camels in Sudan. Rev. Elev. Med. Vet. Pays Trop. 40(3), 531-533.

Abu-zeid, Y.A., (2002). Protein G ELISA for detection of antibodies against Toxoplasma SAG 1 in dromedaries. J. Egypt. Soc. Parasitol. 32: 247-257.

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.

Babiker, M.M., (1984). Abundance and economic potential of camels in the Sudan. J. Arid. Environ. 7 : 377-394.

Bornstein, S.; Musa, B.E., (1987). Prevalence of antibodies to some viral pathogens, Brucella abortus, and *Toxoplasma gondii* in serum from camels (Camelus dromedaries) in Sudan. J. Vet. Med. B, 34: 364-370.

Dubey, J.P., (1993). Toxoplasma, Neospora, Sarcocystis and other tissue-cyst forming cocidia of humans and animals. In: Kreier JP, editor. Parasitic Protozoa 2nd ed. Parasitic Protozoa, vol. 6. pp 1-158.

Dubey, J.P., (1998a). Advances in the life cycle of *Toxoplasma gondii*. Int. J. Parasitol. 28 : 1019-1024.

Dubey, J.P., (1998b). *Toxoplasma gondii* oocyst survival under defined temperature. J. Parasitol. 84: 862-865.

Dubey, J.P., (1998c). Re-examination of resistance of *Toxoplasma gondii* tachyzoites and bradyzoites to pepsin and trypsin digestion. Parasitology. 116: 43-50.

Dureden, B.I.; Reid, T.M.S.; Jewsbury, J.M.; Turk, D.C., (1987). A new short textbook of Microbial and Parasitic infection. Hodder and Stoughton Educational. pp.419.

El Din, E.A.Z.; El Khawad, S.E.; Kheir, H.S.M., (1985). A serological survey for Toxoplasma antibodies in camels (Camelus

dromedaries) in the Sudan. Rev. Elev. Med. Vet. Pays Trop. 38 :247-249.

Elamin, E.A., Elias, S.; Daugschies, A.; Rommel, M., (1992). Prevalence of *Toxoplasma gondii* antibodies in pastoral camels (Camelus dromedaries) in the Butana plains, mid-Eastern Sudan. Vet. Parasitol., 43: 171-175.

Ettinger, S.J., (2001). Toxoplasmosis, In: Pocket companion to textbook of veterinary internal medicine. W. B. Saunders Company. London. 207-211.

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.,

Frenkel, J.K., (2000). Biology of *Toxoplasma gondii*. In: Ambroise-Thomas P, Peterse E, editors. Congenital toxoplasmosis: Scientific background, clinical management and control. Paris: Springer- Verlag. Pp9-25.

Gill, H.S.; Prakash, O., (1969). Toxoplasmosis in India: prevalence of antibodies in camels. Trop. Med. Parasitol. 63: 265-267.

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

Jacobs, L.; Lunde, M.N., (1957). Haemagglutination test for toxoplasmosis. Science. 125: 1035.

Lewis, J.S.Jr; Khoury, H.; Storch, G.A.; DiPersio, J. (2002). PCR for the diagnosis of toxoplasmosis after hematopoietic stem cell transplantation. Expert. Rev. Mol. Diagn. 2(6): 616-624.

Manal, Y.I., (2003). Studies on Toxoplasma and Sarcocystis from camels (Camelus dromedarius) in the Sudan. Ph. D. thesis. University of Khartoum. Sudan. Pp108.

Nath, A.; Jankovic, J.; Pettigrew, L.C., (1987). Movement disorders and AIDS. Neurology. 37: 37-41.

Nath, A.; Sinai, A.P., (2003). Cerebral toxoplasmosis. Curr. Treat. Options Neurol. 5(1): 3-12.

Remington, J. S.; Miller, M.J.; Brownlee, I., (1968). Serological diagnosis of *Toxoplasma gondii* infection. Pediatrics. 41: 1082.

Rieman, H.P.; Meyer, M., (1975). Toxoplasmosis in an infant fed unpasteurized goat milk. J. of pediatrics. 87: 573-576.

Sabin, A.B.; Feldman, H.A. (1948). Dyes as microchemical indicators of anew immunity phenomenon affecting a protozoan parasite (Toxoplasma). Science. 108: 660-663.

Sacks, J.J.; Roberto, R.R.; Brooks, N.F., (1982). Toxoplasmosis infection associated with raw goat's milk. J. Am. Med. Assoc. 248: 1728-1732.

Schwartz, H.J.; Dioli, M., (1992). The one-humped camel (Camelus dromedaries) in Eastern Africa. A pictorial guide to disease, health care and management. Verlag Josef Margraf Scientific Books. D-6992 Weikersheim FR Germany. Pp 282.

Smyth, J.D., (1996). *Toxoplasma gondii*. In: Animal Parasitology. Cambridge University Press. 3rd edition. 99-104.

Solusby, E.J.L. (1982). Toxoplasma. In: Helminths, Arthropods and Protozoa of domestic animals. 7th edition. Bailliere Tindall. London. 670-682.

Tenter, A.M.; Heckeroth, A.R.; Weiss, L.M., (2000). *Toxoplasma gondii*: from animals to humans. Int. J. Parasitol. 30 : 1217-1258.

Uroquhart, G.M; Armour, J.; Duncan, J.L.; Jennings, F.W. (1996). Toxoplasma. In: Veterinary Parasitology. 2nd edition. Blackwell Science Ltd. London. 234-238.

Zhang, S.; Wei, M.; Zhao, H.; Shi, G., (1999). Establishment of immunoglobulin M (IgM)-immunosorbent agglutination assay (ISAGA) for diagnosis of toxoplasmosis. Zhonggu Ji Sheng Chong Xue Yu Ji Sheng Chong Bing Za Zhi. 17(4): 225-227.

Zhang, S.Y.; Wei, M.X., (2001). Quantitative and qualitative comparison of three agglutination tests for detecting *Toxoplasma gondii* antibodies. Zhonggu Ji Sheng Chong Xue Yu Ji Sheng Chong Bing Za Zhi. 18(1): 46-48.

Table (1). Drugs used in the mangement of *Toxoplasma gondi* in dogs and cats.

Generic drug name	Common canine dosage	Common feline dosage
Clindamycin hydrochloride	12.5 mg/kg, q12 h, for 28 days, PO, IM	12.5 mg/kg, q12 h, for 28 days, PO, IM
Pyrimethamine	0.25-0.5 mg/kg, q 24 h, for 28 days, PO	Usually not used owing to toxicity
Trimethprim-sulphonamide	15 mg/kg, q 12 h, for 28 days, PO	15 mg/kg, q 12 h, for 28 days, PO
Doxycycline	5-10 mg/kg, q 12 h, for 4 weeks, PO	5-10 mg/kg, q 12 h, for 4 weeks, PO

IM = intramuscular

PO = oral

* Source Ettinger (2001).