OCCUPATIONAL, NOSOCOMIAL OR HOSPITAL ACQUIRED TOXOPLASMOSIS

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Abstract

Toxoplasma gondii is protozoan parasite infects wild and domestic animals including birds, cats, sheep, goats, cattle, pigs and poultry. Cats are the definitive host of Toxoplasma and transmitted to other animals or people. There are three forms of T. gondii: the tachyzoite (the rapidly reproducing form), the bradyzoite (a slower reproducing form contained in tissue cysts), and the sporozoite (contained in oocysts). The tachyzoite invade cells in the body where it then multiplies rapidly and can destroy cells. When the cells die, the tachyzoites are released and infect other cells. For this reason, tachyzoites are seen in many tissues and organs throughout the body that are infected during this acute phase of the disease. This is also called the extraintestinal phase of the infection since it can affect all cells outside the intestines in all infected animals. However, only cats have the intestinal phase of the infection. Two or three weeks after the first infection, the Toxoplasma divides more slowly and a protective membrane forms around the parasite cells. The cyst containing the parasites is called a zoitocyst and the cells inside the cyst are called bradyzoites. The tissue cysts are formed primarily in brain, eye, heart muscle, and skeletal muscle. Bradyzoites persist in tissues for many years, possibly for the life of the host.

In cats, *Toxoplasma* infects the small intestine lining where they reproduce asexually. After a few days of rapid reproduction the cells transform into a sexual form, combine, and become enclosed in a cyst called an oocyst. Oocysts contain the sporozoite form of the *Toxoplasma* parasite. Oocysts are found in both wild and domestic cats but not in any other animals or birds.

Key words: Toxoplasma gondii, Occupational Disease, Hospital Acquired infection

Introduction

The parasitic diseases are illnesses caused by infestation (infection) with parasites such as the protozoa (one-celled animals), worms, or insects. They include malaria and schistosomiasis, the world's most common serious infectious diseases. The USA reported that a number of parasitic diseases are known, or suspected to be transmitted by the blood transfusion or needle-stick injury. Of greatest concern are malaria and Chagas' disease, but babesiosis, leishmaniasis and toxoplasmosis also offering the risk in particular locations and/or circumstances? Some of these parasites may be imported into non-endemic areas as a result of population movements and in some cases; the natural range of the parasite is increasing as a result of environmental change. The researches especially on the *Trypanosoma brucei* and *Trypanosoma cruzi* (Herwaldt, 2001) and babesiosis along with measures were done to minimize transmission of these and other parasites by blood transfusion, or needle-stick (CDC, 2014).

Herwaldt and <u>Juranek</u> (1993) in USA reported that because of renewed interest in parasitic diseases, increasing numbers of persons in clinical and research laboratories have the potential for exposure to parasites and therefore are at risk for acquiring parasitic infections. In this review of laboratory-acquired parasitic infections, they concentrated on protozoan diseases that frequently were reported to be laboratory acquired: malaria, leishmaniasis as nosocomial Needlestick, non-intact skin (Knobloch and Dem-

ar, 1997), America and Africa trypanosomiasis, and toxoplasmosis. These diseases can be severe, even fatal, and may be difficult to diagnose. Many laboratorians who have acquired these diseases did not recall having had an accident. Of those with recognized accidents, needle-stick injuries were the commonest. Laboratories should have established protocols for handling specimens that might contain viable infective agents and for responding to laboratory accidents (CDC, 2003).

In USA reported that a nurse developed falciparum malaria after a needle-stick injury from a patient with documented falciparum malaria. Three days prior to her diagnosis, she cared for another patient, who subsequently developed *falciparum* malaria. That patient's parasite isolate genetically matched the nurse's isolate by two independent DNA fingerprinting techniques (Alweis et al, 2004). Plasmodium falciparum molecular genotype was identified in two in 2 American patients who shared a hospital room. The P. falciparum transmitted in a hospital environment from patient to patient by blood inoculum if standard precautions are breached (Jain et al, 2005).

Sewell (1995) in USA stated that the estimated 500,000 laboratory workers were at risk of exposure to infectious agents that cause disease ranging from unapparent to life-threatening infections, but the precise risk to a given worker unknown.. He added that the initial step in a biosafety program is the assessment of risk to the employee. Risk assessment guidelines include the pathogenicity of the infectious agent, the method of transmission, worker-related risk factors, the source and route of infection, and the design of the laboratory facility. Strategies for the prevention and management of laboratory-associated infections are based on the containment of the infectious agent by the physical separation from the laboratory worker and the environment, employee education about the occupational risks, and the availability of an employee health program. Adherence to the biosafety guidelines mandated or proposed by various governmental and accrediting agencies reduces the risk of an occupational exposure to the infectious agents handled in the workplace.

Review, Discussion and Comment

The current study will deal with toxoplasmosis out of four infectious blood protozoan diseases encountered in Egypt, which are risky to Health Care Staff by blood transfusion or even by needle-stick.

Toxolasma gondii is one of the most widely prevalent cysts forming apicomplexans parasites recorded worldwide. Felines serve as definitive hosts, while all non-felines vertebrates, including humans act as intermediate hosts of the parasite with disseminated tissue infections. The parasite is distributed worldwide in the human population and is estimated to affect more than a billion individuals. The parasite has significant impact not only on animal production but also on public health worldwide (Sudan et al, 2013).

Toxoplasmosis is caused by the intracellular protozoan parasite, T. gondii. Immunocompetent persons with primary infection are usually asymptomatic, but latent infection can persist for the life of the host. However, there is a risk of reactivating infection at a later time should the individual become immune-compromised, even if infection was asymptomatic or only mildly symptomatic initially (Weiss and Kim, 2011). The parasite is only known to reproduce in the catfamily. It, however, can infect most types of warm-blooded animals, including humans. Diagnosis is typically by testing the blood for antibodies or by testing the amniotic fluid for the parasite's DN (Flegr et al, 2014). Up to one third of the world's population carried Toxoplasma infection (Montoya and Liesenfeld, 2004). In Egypt, it was serological reported man in 12 governorates (Rifaat et al, 1963), in farm animals ((Rifaat et al, 1968) and congenital mother to child (Wishahy et al, 1971a, b, c). Toxoplasmosis is considered to be a leading cause of death attributed to food borne illness in the United

States. More than 60 million men, women, and children in the U.S. carried *T. gondii*, but very few showed symptoms since their immune system usually kept parasite from causing the illness (Pappas *et al*, 2009).

Before 1970, the tachyzoite and the bradyzoite were the only stages of *toxoplasma* known to man. These two stages constitute the asexual life cycle of the parasite present in all intermediate warm-blooded hosts including land and water living mammals, birds, and humans (Carer, 2013).

Research on *Toxoplasma* is spurred for essentially three reasons. First, *Toxoplasma* can cause life-threatening disease, e.g. encephalitis, retinitis, myocarditis and pneumonia. Second, *Toxoplasma* is used as a model-system of Apicomplexan parasites which include important disease causing pathogens such as *plasmodium* (the causative agent of malaria), *Eimeria* and *Cryptosporidium*. Family, *Toxoplasma* is an important veterinary pathogen with high estimated costs owing to disease, abortion or vaccination in animal farming (Butler *et al*, 2012).

Transmission: Felines are the only animals in which *T. gondii* can complete its reproductive cycle. Following feline ingestion of any of the forms of *T. gondii*, the parasite infects the gut epithelial cells and reproduces. The feline then excretes infectious oocysts in feces. When non-felines, including humans, ingest *T. gondii* oocysts, the organisms invade intestinal epithelium and disseminate throughout the body. They then encyst in any type of nucleated cell and can lie dormant within tissues for the life of the host (Jones *et al*, 2014).

There are seven means of acquiring toxoplasmosis in humans (Tenter *et al*, 2000): 1-Through vertical transmission from an infected mother to her fetus congenital infection (Congenital). 2- Ingestion of infectious oocysts from the environment (usually from soil contaminated with feline feces), 3- Cleaning cat litter boxes. 4- Drinking unpasteurized goat milk or equine milk. 5- Ingestion of tissue cysts in meat from an infected ani-

mal, 6-Eating unwashed raw vegetables or fruits. 7- Via blood transfusion or organ transplantation from an infected donor, (Acquired infection). 8- Also, accidentally by needle-stick injuries as well as handling specimens that may contain viable organisms to the laboratory for examination (Herwaldt and Juranek, 1993).

Toxoplassmosis in blood donors: History of immune weakness was found to be a significant risk factor for T. gondii seropositivity by logistic regression analysis. So, can OS associated with T. gondii infection contribute to the immunosuppression in seropositive blood donors? Toxoplasmosis, particularly in those with encephalitis, proved to be a major cause of morbidity and mortality in immuno-compromised patients (Dubey, 1998). Those patients usually experience chronic OS, and concurrent infection with the opportunistic intracellular parasite would be expected to further exacerbate this condition. Despite evidence that T. gondii can alter immune response, a mechanism of T. gondii-induced immunosuppression has not been completely elucidated. ROS have a general immune suppressive effect. OS favors a Th2-polarizing condition (King et al, 2006), by reducing INF-gamma production of activated Th1 clones and potentiating IL-4 secretion of activated Th2 clones and upregulating antibody production (Frossi et al, 2008). On the other hand, infection with T. gondii causes a switch from a Th2 response to a Th1 immune response and induces inflammatory cytokines as well as enhances macrophage capacity to secrete ROS that promotes OS and further immunosuppression (Santiago et al, 1999). Since data suggested that T. gondii exposure resulted in OS, which might profoundly affect the immune function (Baier-Bitterlich et al, 1997), it is logical to hypothesize that OS plays a significant mediator role in the T. gondiiinduced immunosuppression

In general, the anti-*Toxoplasma* antibodies were reported in the healthy blood donors worldwide. This was true as in Kenya (Grif-

fin and Williams, 1983), western, southwestern and eastern Saudi Arabia (Sarwat et al, 1993; Al-Amari, 1994; Yanaza and Kumari, 1994), the Czech Republic (Svobodova and Literak, 1998), Northeast Thailand (Pinlaor et al, 2000), Bamako (Maiga et al, 2001), Malaysia (Nissapatorn et al, 2002), Kuwait (Iqbal et al, 2003), Northeast Brazil (Coelho et al, 2003), Turkey (Yazar et al, 2006), Mexico (Alvarado et al, 2007, Alvarado-Esquivel et al, 2016), India (Sundar et al, 2007), Egypt (Elsheikha et al, 2009), Iran (Karimi et al, 2016) and Scotland (Burrells et al, 2016). Moreover, Zghair et al. (2015) reported that in acute and chronic toxoplasmosis in males blood donors recorded higher significant (P < 0.05) mean concentration for total and free testosterone hormone, but the mean concentration of follicle stimulating hormone revealed non-significant (P < 0.05) differences in both disease activities.

Besides blood, transmission of *T. gondii* was reported by the whole or white blood cell transfusions (Siegel *et al*, 1971; Shulman, 1994; Galvan *et al*, 2005) or the renal transplantation in cat and dog (Bernstein *et al*, 1999), or CMV in renal transplant recipients (Iqbal *et al*, 2003; Barsoum, 2006; Campbell *et al*, 2006; Nissapatorn *et al*, 2011; Hamza *et al*, 2015) from positive donors to recipients.

The pathogenesis of disease varies with the strain of the parasite, duration of infection, the organs attacked, age of host and immune status of patient. Individual response to *toxoplasma* infection is determined by immune status, timing of infection, and the genetic composition of the host and organism (Suzuki, 2002).

Montaya and Remington (2000) referred to the asymptomatic toxoplasmosis in immunocompetent individuals, however, acute toxoplasmosis often manifests with influenza-like: swollen lymph nodes or muscle aches and pain that last for month or more. The symptoms of young children and immune-compromised patients such as those with HIV/AIDs, chemotherapy, and organ

transplantation, may develop sever toxoplasmosis and this can cause damage to the brain or the eye.

Clinical manifestations: The clinical pictures of toxoplasmosis vary, depending on on the parasite characteristics such as virulence of the strain and inoculum size, as well as host factors such as the genetic background and the immune status (Montoya and Liesenfeld, 2004). Toxoplasmosis in immunocompetent individuals is typically mild or asymptomatic and usually results in lifelong immunity. Symptoms most commonly include lymphadenopathy that might be accompanied by headache, fever, fatigue, muscular or abdominal pain as well as myocarditis, hepatitis and pulmonary necrosis. However, there were cases of toxoplasmosis where overtly serious clinical symptoms might occur; these include ocular toxoplasmosis, congenital toxoplasmosis, and reactivation of a latent infection in the immunocompromised individual (Bhopale, 2003).

Early diagnosis of toxoplasmosis in pregnant women can be of great help in the early intervention and prevention of congenital disorders that usually led to fetal death (Porteela *et al*, 2004). Saleh *et al*. (2014) reported anti-*Toxoplasma* antibodies among Egyptian child-bearing aged females.

When toxoplasmic infection is acquired for the first time during pregnancy, infection can be transmitted to the fetus, resulting in the congenital toxoplasmosis and associated neurological, ocular manifestations, the maternal infection and its effect on the fetus (Paquet and Yudin, 2013).

The maternal toxoplasmosis infection is acquired orally. Fetal infection results from transmission of parasites via the placenta following primary maternal infection. It is likely that transmission occurs in most cases during the parasitemic phase in the days after infection and before the development of a serologic response (Elsheikha and Morsy, 2009). To survive and multiply, the tachyzoite invades host cells, especially in the brain and muscle, where it forms tissue cysts

which can remain dormant for years. In immune competent animal models, tissue cysts can be formed within a week of infection. It is not known how long this process takes in relatively immunologically immature fetus. The transition from acute infective tachyzoite form, which is responsible for cell damage, to the dormant bradyzoite form contained in tissue cysts impenetrable to antibiotics, has important implications for the therapeutic window of opportunity (de-Moura et al, 2006). Maternal infection during pregnancy is most accurately diagnosed when based on a minimum of two blood samples at least two weeks apart showing seroconversion from negative to positive toxoplasma-specific IgM or IgG (Chemla et al, 2002) such serial testing of susceptible women is usually feasible only as part of a prenatal screening program. The more frequently a woman is retested, the greater the chance of detecting infection early on, and when treatment is more likely to be effective. However, costs of frequent testing and the chances of false positive results increase as the frequency of retesting increases. Furthermore, the benefit of routine screening is debatable, given there is no high quality evidence that treatment improves clinically relevant outcomes (Amendoeria and Camillo-Coura, 2010).

PCR for *T. gondii* DNA in amniotic fluid is the best method for diagnosing fetal infection, but accuracy varies between laboratories and techniques (Binquet *et al*, 2003). Real time PCR appears to be the most sensitive test, and sensitivity of PCR increased with the gestational age at maternal seroconversion (Tan *et al*, 2007).

Toxoplasmosis is a significant cause of congenital disease. Congenital toxoplasmosis occurs in between 1 and 10/10000 live births in Europe (Montoya and Liesenfeld, 2004). The tranplacental transmission occurs when an immune-competent woman acquires a primary infection during pregnancy, or may also be due to a reactivated infection in immune-compromised women. Primary

infections acquired four to six months before conception usually result in no transplacental transmission to the fetus (Dubey and Jones, 2008). Gajurel et al. (2015) in USA stated that toxoplasmosis in the hematopoietic cell transplant (HCT) recipients is associated with high morbidity and mortality rates. Prophylaxis following HCT is recommended for high-risk pre-HCT Toxoplasmasero-positive (pre-HCTSP) recipients. However, there is no agreement or consistency among programmers on whether to adopt the prophylaxis or not, or if used, on the chosen the anti-Toxoplasma prophylactic regimen. Kwofie et al. (2016) stated that the presence of anti-T. gondii-IgG antibodies only, and T. gondii DNA in placental tissues indicate the women might have been infected early during the pregnancy, placing about 39.8 % of the babies at risk. These results strongly influenced the policy to screen and treat the pregnant women for T. gondii infection.

Kaye (2011) stated that persons with toxoplasmosis in the United States are asymptomatic, but if a woman is infected during pregnancy, the parasite can cross the placenta and cause congenital toxoplasmosis in the fetus. The severity of congenital toxoplasmosis depends on when in the pregnancy the mother is exposed, but it can cause ocular and central nervous system disease as well as lead to growth failure and hearing and vision abnormalities. It is important for pediatric nurse practitioners to be aware of the clinical presentation and the treatment of congenital toxoplasmosis.

The risk of intrauterine infection of the fetus, the risk of congenital toxoplasmosis, and the severity of the disease depend on the time of maternal infection during pregnancy, the immunological competence of the mother during parasitaemia, the number and virulence of the parasites transmitted to the fetus, and the age of fetus at the time of transmission (Pinon *et al*, 2001).

Primary infection during pregnancy may result in severe damage or death of the fetus and long-term sequelae in the child. The risk of congenital infection increases from the first trimester (10-25%) to the third trimester (60-65%) with the development of a good blood flow (Dubey and Jones, 2008). The toxoplasmosis severity, however, reached its pick in the first trimester and the lowest in the third one (Hegab and Al-Mutawa, 2003).

Prevention: To prevent food-borne horizontal transmission of *T. gondii* to humans, all meat should be well cooked to minimum temperature of 67°C before consuming so as to kill tissue cysts. Tissue cysts can also be killed by cooling to -13C. Pregnant women should be especially careful, and should limit contact with cats, cat litter, soil and raw meat (Hill and Dubey, 2002). In addition, meat should not be tasted during seasoning or cooking. Washing kitchen knives infrequently after preparation of raw meat was independently associated with an increased risk of primary infection during pregnancy (Flegr, 2006).

Definition of the needle sticks injury: The accidental puncture of the skin by a needle during a medical intervention, percutaneous piercing wound caused by a sharp instrument. Commonly encountered by people handling needles in the medical setting, such injuries are an occupational hazard for health care professionals. Law enforcement personnel are also at high risk for needle stick injuries at work. Despite their seriousness, it is estimated that half of all needle stick injuries go unreported. On the other hand, as needle sticks have been recognized as occupational hazards, their prevention has become the subject of regulations (NOSH, 2012).

Needle stick injuries are a common event in the healthcare environment. When drawing blood, administering an intramuscular or intravenous drug, or performing other procedures involving sharps, the needle can slip and injure the healthcare worker (CDC, 2006). This allows for transmission of pathogens. These injuries also commonly occur during needle recapping and as a result of failure to place used needles in approved

sharps containers. Lack of access to or failure to use appropriate personal protective equipment can cause needle stick injuries. Night shifts also put practitioners at risk for needle stick injuries (Lavoi *et al*, 2014).

During surgery, a surgical needle or other sharp instrument may inadvertently penetrate the glove and skin of the surgeon or other operating room personnel. Injuries may occur when needles are passed between personnel, when personnel load needles into a needle driver, when personnel place needles in an overfilled or poorly located sharps container, or tie off sutures while still connected to the needle (CDC, 2008a). Generally needle stick injuries cause only minor bleeding or visible trauma, however, even in the absence of bleeding the risk of viral infection remains. Scalpel injuries tend to be larger than a needle stick. In turn, a needle stick injury may also pose a risk for a patient if the injured health professional has a blood borne illness (Ker et al, 2010).

Some parasites can be blood borne: This means; the parasite can be found in the bloodstream of infected people; and the parasite might be spread to other people via exposure to an infected person's blood (for example, by the blood transfusion or by sharing needles or syringes contaminated with blood), Examples of parasitic diseases that can be blood-borne included the following: babesiosis (Saleh et al, 2015), leishmaniasis, malaria (Tarantola et al, 2004; Saleh et al, 2016), and toxoplasmosis (Field et al, 1972). . In nature, many blood-borne parasites are spread by insects (vectors), so they are also referred to as vector-borne diseases. But, Toxoplasma gondii is not transmitted by an insect-vector (CDC, 2014).

In general, there are three major of routes of entry for diseases agents, these include parasites, viruses, bacteria and fungi into the human body, i.e. Transmission through contact with body fluids of the infected or contact with contaminated objects (WHO, 2010)

The nurses thought a little needle pricking might be a carrier of one of many transferred

diseases. It increases among nurses who are too busy to take a routine blood samples to make sure her blood is clean. In addition, the visual information ensured more awareness between the working nurses and interactive workshops. Particular steps, from handling needle, recapping needle, pulling needle to air discharging, are all necessary to be the cautiously treated by every nurse staff (Thomas and Murray, 2009).

The behavior of recapping needles persists despite the well documented dangers and international recommendations against this practice. In addition, there are deficits in knowledge, and practice of simple protective measures such as wearing gloves, not removing needles with hand before disposal, and using disposal containers (CDC, 2008b).

Nursing personnel account for more than 40% of the needle stick injuries (NSIs) even in developed countries. The circumstances in which most NSIs occur involve manipulating a needle in a patient; 26%, sharp disposal 21%, collision with a worker or sharp; 10%, clean-up; 9%, and recapping needles; 5% (CDC, 2011)

However, this exposure can have a further influence on the quality of life of the injured nurses, and can cause great worry, anxiety, and fear for nurse staff and her family and colleagues, as well as feelings of stigma and low self-confidence (Gonzalez-Medina and Le, 2011).

Needle-stick and other sharps injuries are a well-known risk within health-care settings and reflect the necessity of using such equipment to deliver health care. Puncture wounds arising from contaminated needles or other sharps infected by a patient's blood can transmit diseases (Health and Safety Executive, 2013).

The practice among nurses about post occupational exposure to blood and body fluids is inadequate. Knowing the infectious-disease status of the source patient, as well as understanding the risks of transmission, might make nurses more adherent to infectious-diseases prescriptions (Baudu, 2011).

The nurses must allow the wound to bleed freely. Immediately reporting blood exposure is very important. Like several studies, many exposures were not reported (Shiva *et al.* 2011).

Definition of accidental exposure to blood: The unintended contact with blood and or with body fluids mixed with blood during a medical intervention. The accidental contact with blood occurs especially in following situations: 1-while re-capping, 2- during surgery, especially during wound closure, 3while taking biopsy, 4- when an uncapped needle has ended up in bed linen, surgery clothing...etc., 5- while taking an unsheathed used needle to the waste container, 6while cleaning up and transporting of waste material, 7- while using more complex collection & injection techniques, 8- in a & e (accident and emergency) departments and 9- under high-stress interventions (diagnostic or therapeutic endoscopy in patients with gastrointestinal bleeding), Accidental exposure to blood following a needle-stick injury is probably one of the most common occupational health accidents in medical care.

Definitions Medical Sharp Injury: Stated that sharps injuries pose a serious threat to health professionals, patients, and downstream workers "Medical Sharp" means object or instrument which is used for carrying out activities to healthcare and which is able to cause injury by means of cutting or piercing the skin; Injury includes infection (Adams *et al*, 2013).

The safer sharp means the medical sharp that is designed and constructed to incorporate a feature or mechanism which prevents or minimizes the risk of accidental injury from cutting or piercing skin (Statutory Instrument, 2013)

Work-related blood borne pathogen exposure: Every percutaneous needle stick and sharps injury carries a risk of infective blood borne pathogens. Yet, these exposures often have been considered "part of the job." Health Care workers primarily are exposed to these pathogens via contaminated needle-

stick & sharps injuries. One probably knew one colleague who had sustained an injury, or perhaps you had stuck yourself. It is important that you and your colleagues fully understand these risks (Adams *et al*, 2013).

Facts about occupational infection: Every year, health care workers experience between 600,000 and 800,000 exposures to blood (United States Department of Labor-Occupational Safety and Health Administration 2001). In USA stated that at least 1,000 health care workers are estimated to contract serious infections annually from needle stick and sharps injuries (Jagger and Perry, 2013). Alvarado-Esquivel et al. (2011) in Mexico reported serologic and contributing factors for toxoplasmosis in workers occupationally exposed to unwashed raw fruits and vegetables, and the results may help in the design of optimal preventive measures against Toxoplasma infection especially in reproductive age female workers. Flegr (2013) Czech Republic reported that within the past 10 years, however, many independent studies have shown that *Toxoplasma* disease, with a worldwide prevalence of about 30%, could be indirectly responsible for hundreds of thousands of deaths due to its effects on the rate of traffic and workplace accidents, and also suicides. Moreover, latent toxoplasmosis is probably one of the most important risk factors for schizophrenia. At least some of these effects, possibly mediated by increased dopamine and decreased tryptophan, are products of manipulation activity by Toxoplasma aiming to increase the probability of transmission from intermediate to definitive host through predation

In Kuwait, Omar *et al.* (2015) reported that the needle stick injuries were the most common exposure among Health care personnel, and nurses were the most frequently involved HCP category. They added that the majority were nurses: 166 (66.7%), followed by doctors: 35 (14.1%), technicians: 26 (10.4%) and housekeeping personnel: 22 (8.8%). Needle stick injury was the most common type of exposure, in 189 (75.9%),

followed by sharp-object injury, mucousmembrane exposure and contact with nonintact skin. The majority of needle stick exposures, i.e. 177 (93.7%), were caused by hollow-bore needles. Exposure to blood represented 96.8%, mostly during drawing of blood and the insertion or removal of needles from patients (88 or 35.4%) and when doing surgical interventions (56 or 22.6%). A good proportion of exposures could be easily prevented. HBV vaccination coverage was incomplete. Easily preventable exposures such as injuries related to 2-handed recapping of needles (24 or 9.6%) and collection of garbage (21 or 8.4%) were reported. The exposures mainly occurred in the patient wards (75 or 30.1%) and the operating theaters (56 or 22.6%).

Conclusion

No doubt, toxoplasmosis is an occupational blood-protozoan disease. Generally speaking, toxoplasmosis is a worldwide distributed disease, characterized by a complex epidemiology. The risk of infection for humans depends on their contact with infective oocysts in a contaminated environment and on the amount of tissue cysts located within consumed meat. Unfortunately, the prevalence of tissue cysts is largely unknown for game species. Cat feces and meat are the most important sources of infection. On the other hand, T. gondii is a serious threat particularly to female especially pregnant ones, non-immune women and their babies and congenital toxoplasmosis is another risk to man and animal. More researches are ongoing to evaluate prevalence of toxoplasmosis among the nurses and laboratory technicians and also to focus on the specific impact of training intervention on improving their knowledge, attitude, and compliance. What occupations are at risk?

Sources of occupational infection include contact with infected raw meat, infected animals, contaminated soil or water, or contact with contaminated cat feces. Laboratory personnel who handled contaminated needles or glassware also contracted toxoplasmosis. It is an occupational risk for: animal care workers including breeders, keepers, zoo attendants, veterinarians or slaughterhouse workers, meat inspectors, line processors, butchers or cooks, agricultural workers, landscapers and gardener, laboratory workers and health care workers. Protocols should be provided for handling specimens that could contain viable organisms, using protective clothing and equipment, dealing with spills of infectious organisms, and responding to accidents. Special care should be exercised when using needle-stick, sharp objects and blood transfusion.

References

Adams, D, Down, S, Hicks, D, 2013: FIT4 Safety: recommendations in the diabetes care setting. Br. J. Nurs. 22, 17:997-1000.

Al-Amari, OM, 1994: Prevalence of antibodies to *Toxoplasma gondii* among blood donors in Abha, Asir Region, southwestern Saudi Arabia. J. Egypt. Pub. Hlth. Ass. 69:77-88.

Alvarado, C, Suarez, MF, Rodríguez, A, Torres, L, et al, 2007: Seroepidemiology of infection with *Toxoplasma gondii* in healthy blood donors of Durango, Mexico. B.M.C. Infect. Dis. 7:75-80.

Alvarado-Esquivel, C, Estrada-Martínez, S, Liesenfeld, O, 2011: *Toxoplasma gondii* infection in workers occupationally exposed to unwashed raw fruits and vegetables: a case control seroprevalence study. Parasit. Vectors 4:235-9

Alvarado-Esquivel, C, Rascón-Careaga, A, Hernández-Tinoco, J, Corella-Madueño, MA, Sánchez, LF, *et al*, 2016: Seroprevalence and associated risk factors for *Toxoplasma gondii* infection in healthy blood donors: A cross-sectional study in Sonora, Mexico. Biomed. Res. Int. 2016:9597276. doi:10.1155/2016/9597276.

Alweis, RL, DiRosario, K, Conidi, G, Kain, K C, Olans, R, et al, 2004: Serial nosocomial transmission of *Plasmodium falciparum* malaria from patient to nurse to patient. Infect Control Hosp. Epidemiol. 25:55–9.

Amendoeria MR, Camillo-Coura, LE, 2010: A brief review on toxoplasmosis in pregnancy. Sci. Medic. 21, 1:113-9

Baier-Bitterlich, G, Fuchs, D, Wachter, H, 1997: Chronic immune stimulation, oxidative stress, and apoptosis in HIV infection. Biochem. Pharmacol. 53:755-63.

Barsoum, R, 2006: Parasitic infections in transplant recipients. Nat. Clin. Pract. Nephrol. 2: 490-503.

Baudu, A, lot, F, Abiteboul, D, Iheriteau, F, Touche, S, G *et al*, 2011: Pour le comite de pilotage national de la surveillance aes-raisingeres suivi des accidents exposant au sang chez les professionnels de santé non immunizes et exposes au vhp,2005-2007 (France). Bull. Epidemiol. Hebdomad. 35, 36:388-91.

Bernstein, L, Gregory, C, Aronson, L, Lirtzman, A, Brummer, D, 1999: Acute toxoplasmosis following renal transplantation in three cats and a dog. J. Am. Vet. Med. Assoc. 215: 1123-6.

Bhopale, GM, 2003: Development of a vaccine for toxoplasmosis: current status. Microbes Infect. 5:457-62

Binquet C, Wallon M, Metral P, 2003: Toxoplasmosis sero-conversion in pregnant women: The differing attitudes in France. Presse. Med. 33:775-86

Burrells, A, Opsteegh, M, Pollock, KG, Alexander, CL, Chatterton, J, et al, 2016: The prevalence and genotypic analysis of *Toxoplasma gondii* from individuals in Scotland, 2006-2012. Parasit. Vectors 9, 1:324-30.

Butler, NJ, Furtado, JM, Winthrop, KL, Smith, JR, 2012: Ocular tox-oplasmosis II: Clinical features, pathology and management. Clin. Exp. Ophthalmol. 41:95-108

Campbell, AL, Goldberg, CL, Magid, S, et al, 2006: First case of toxoplasmosis following small bowel transplantation and systematic review of tissue-invasive infection following non-cardiac solid organ transplantation. Transplantation 81:408-417.

CDC, 2003: Guideline for Hand Hygiene in Health-Care Settings: Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task 51, 16:1-44

CDC, **2006**: Definition of Nosocomial Infections. Infect. Cont. J. 7, 3:1-120

CDC, **2008a**: Health Information for International Travel. DHHS, Atlanta, GA, USA.

CDC, 2008b: Workbook for Designing, Implementing, and Evaluating Sharps Injury Prevention. Program. http://www.cdc.gov/sharpssafeyy/pdf/sharpsworkbook.

CDC, 2011: Division of Healthcare Quality Promotion, Basic Infection Control and Prevention Plan for Outpatient Oncology Settings, page 2

- **CDC**, **2014:** Prevention 1600 Clifton Rd. Atlanta, GA. 30329-4027. USA. 800-CDC-Info (800-232-4636) *TTY*: (888) 232-9348.
- Chemla, C, Villena, I, Aubert, D, 2002: Preconception seroconversion and maternal seronegativity at delivery do not rule out the risk of congenital toxoplasmosis. Clin. Diagn. Lab. Immunol. 9:489-98
- Chen, JC, Silverthorne, C, 2008: The impact of locus of control on job stress, job performance and job satisfaction in Taiwan. Leadership Organ. Develop. J. 29, 572-582
- **Coelho, R, Kobayashi, M, Carvallo, LBJr, 2003:** Prevalence of IgG antibodies specific to *T. gondii* among blood donors in Recife, Northeast Brazil. Rev. Inst. Med. Trop. Sao Paulo, 45: 229-31.
- **De Moura, L, Bahia-Oliveira, LM, Wada, M Y, 2006:** Waterborne toxoplasmosis, Brazil, from field to gene. Emerg. Infect. Dis. 12:326-32 **Dubey, JP, 1998:** Toxoplasmosis. In: Topley and Wilson's Microbiology and Microbial Infection, 5th edition, by Collier, L. *et al,* Oxford University Press Inc., New York.
- **Dubey, JP, Frenkel, JK, 1998:** Toxoplasmosis of rats: A review, with considerations of their value as an animal model and their possible role in epidemiology. Vet. Parasitol. 77:1-32
- **Dubey, JP, Jones, JL, 2008:** *Toxoplasma gondii* infection in humans and animals in the United States. Int. J. Parasitol; 38, 11:1257-78
- **Dubey, JP, Passos, IM, Rajendran, C, Ferreira, LR, Gennari, SM, etal, 2011:** Isolation of viable *Toxoplasma gondii* from feral guinea fowl (*Numida meleagris*) and domestic rabbits (*Oryctolagus cuniculus*) from Brazil. J. Parasitol. 97, 5:842-5
- **Elsheikha, HM, 2008:** Congenital toxoplasmosis: priorities for further health promotion action. Publ. Hlth. 122:335-53.
- Elsheikha, HM, Aboul-Dahab, MA,Abdel Maboud, AI, El-Sherbini, ET, 2009: Prevalence and risk factors of *Toxoplasma gondii* antibodies in asymptomatic Egyptian blood donors. J. Egypt .Soc. Parasitol. 39, 1:S351-61
- **Elsheikha, HM, Morsy, TA, 2009:** Role of immune response in *Toxo-plasma gondii* tachyzoite-bradyzoite stage interconversion: A Janus in determining disease outcome. J. Egypt. Soc. Parasitol. 39, 2:595-8
- Ferreira da Silva Mda, F, Barbosa, HS, Gross, U, Luder, CG, 2008: Stress related and spo-

- ntaneous stage differentiation of *T. gondii*. Mol. Biosyst. 4:824-34.
- **Field, PR, Moyle, GG, Parnell, PM, 1972:** The accidental infection of a laboratory worker with *Toxoplasma gondii*. Med. J. Aust. 2:196-8
- Filice, GA, Clabots, CR, Riciputi, PE, Goñi, O, Pomeroy, C, 1999: Changes in cytokine levels during reactivation of *Toxoplasma-gondii* infection in lungs. Infect. Immun. 67:2082-9.
- **Flegr, J, 2006:** Increased risk of traffic accidents in subjects with latent toxoplasmosis a retrospective case-control study. BMC Infect. Dis. 2:11-8.
- Flegr, J, 2013: How and why *Toxoplasma* makes us crazy. Trends Parasitol. 29, 4:156-63.
- Frossi, B, De Carli, M, Piemonte, M, Pucillo, C, 2008: Oxidative micro-environment exerts an opposite regulatory effect on cytokine production by Th1 & Th2 cells. Mol. Immunol., 45:58-64.
- **Gajurel, K, Dhakal, R, Montoya, JG, 2015:** *Toxoplasma* prophylaxis in haematopoietic cell transplant recipients: a review of the literature and recommendations. Curr. Opin. Infect. Dis. 28, 4:283-92
- Galvan, RM, Covarrubias, X, Rodriguez, R, Troyo, R, Alfaro, N, Correa, D, 2005: *T. gondii* antibodies in Mexican blood donors. Transfusion, 45:281-282.
- Gonzalez-Medina, D, le, QV, 2011: infectious diseases and interpersonal trust: international evidence. Health, 3,206-210.Doi:4236/34037
- **Griffin, L, Williams KA, 1983:** Serological and parasitological survey of blood donors in Kenya for toxoplasmosis. Trans. Roy. Soc. Trop. Med. Hyg. 6:143-5.
- Hamza, H, El-Taweel, H, Abou-Holw, S, Khalil, S, Wagdy, E, 2015: *Toxoplasma gondii* seropositivity in renal patients: rate, pattern, predictors and related morbidity. J. Egypt. Soc. Parasitol. 45, 1:7-15.
- **Health and Safety Executive, 2013:** Health and safety (sharps instruments in healthcare) regulations: Guidance for employers and employees. www//Hse.gov.uk/ pubns/ hsis7.
- **Hegab, SM, Al-Mutawa, SA, 2003:** Immunopathogenesis of toxoplasmasis. Clin. Exp. Med. 3, 2:84-105.
- **Herwaldt, BL, 2001:** Laboratory-acquired parasitic infections from accidental exposures. Clin. Microbiol. Rev. 14:659-88.
- Herwaldt, BL, de Bruyn, G, Pieniazek, NJ, et

- *al*, **2004:** *Babesia divergens*-like infection, Washington State. Emerg. Infect. Dis. 10:622-9.
- **Herwaldt, BL, Juranek, DD, 1993:** Laboratory-acquired malaria, leishmaniasis, trypanosomiasis, and toxoplasmosis. Am. J. Trop. Med. Hyg. 48, 3:313-23.
- Hill, D, Dubey, JP, 2002: *Toxoplasma gondii*: Transmission, diagnosis and prevention. Clin. Microbial. Infect. 8:634-40
- **Iqbal, J, Nampoory, MR, John, K, Khalid, N, Al-Mousawi, M, 2003:** Determination of antibodies to *T. gondii* and CMV in renal transplant recipients. Transplant. Proc. 35, 7:2703-5.
- **Jagger, J, Perry, J, 2013: Safety-**engineered devices in **2012**: the critical role of healthcare workers in device selection. Infect Control Hosp. Epidemiol. 34, 6:615-8.
- **Jagger, J, Perry, J, Gomaa, A, Phillips, E, 2008:** The impact of U.S. policies to protect healthcare workers from blood borne pathogens: the critical role of safety-engineered devices. J. Infect. Pub. Hlth. 1, 2:62-71.
- Jain, SK, Persaud, D, Perl, TM, Pass, M, Murphy, K, *et al*, 2005: Nosocomial malaria and saline flush. Emerg. Infect. Dis. 11, 7: 1097-9.
- **Karimi, G, Mardani, A, Zadsar, M, 2016:** Prevalence of *Toxoplasma gondii* among Iranian Blood Donors: A Narrative Review Article. Iran J. Parasitol. 11, 1:10-8.
- **Kaye, A, 2011:** Toxoplasmosis: diagnosis, treatment, and prevention in congenitally exposed infants. J. Pediatr. Hlth. Care 25, 6:355-64.
- Ker, K, Edwards, PJ, Felix, LM, Blackhall, K, Roberts, I, 2010: Caffeine for the prevention of injuries and errors in shift workers. The Cochrane Database of Systematic Reviews 5: CD008508.
- **King, MR, Ismail, AS, Davis, LS, Karp, DR, 2006:** Oxidative stress promotes polarization of human T cell differentiation toward a T helper 2 phenotype. J. Immunol. 176:2765-72.
- **Knobloch, J, Demar, M, 1997:** Accidental *Leishmania mexicana* infection in an immunosuppressed laboratory technician. Trop. Med. Int. Hlth. 2:1152-5
- Kwofie, KD, Ghansah, A, Osei, JH, Frempong, KK, Obed, S, et al, 2016: Indication of risk of mother-to-child *Toxoplasma gondii* transmission in the Greater Accra Region of Ghana. Matern. Child Health J. 2016 Jul 27. [Epub ahead of print]
- Maiga, I, Kiemtore, P, Tounkara, A, 2001: Prevalence of anti-*Toxoplasma* antibodies in

- patients with acquired immunodeficiency syndrome & blood donors in Bamako. Bull. Soc. Path. Exot. 94:268-70.
- Montoya, JG, Liesenfeld, O, 2004: Toxoplasmosis. Lancet; 363, 9425: 1965-76
- Nissapatorn, V, Kamarulzaman, A, Init, I, Tan, L, Rohela, M. *et al*, 2002: Seroepidemiology of toxoplasmosis among HIV-infected patients and healthy blood donors. Med. J. Malaysia 57:304-10.
- Omar, AA, Abdou, NM, Salama, MF, Al-Mousa, HH, 2015: Occupational injuries prone to infectious risks amongst healthcare personnel in Kuwait: a retrospective study. Med. Princ. Pract. 24, 2:123-8.
- **Pappas, G, Roussos, N, Falagas, ME, 2009:** Toxoplasmosis snapshots: Global status of *Toxoplasma gondii* sero-prevalence and implications for pregnancy and congenital toxoplasmopsis. Int. J. Parasitol; 39, 12:1385-94
- **Paquet, C, Yudin, MH, 2013:** Infectious Diseases Committee: Toxoplasmosis in pregnancy: Prevention, screening, and treatment. J. Obst. Gynecol. Canada 35, 1:S1-7
- **Perry, C, 2007:** Infection Prevention and Control. Blackwell, Oxford.
- Pinlaor, S, Ieamviteevanich, K, Pinlaor, P, et al, 2000: Sero-prevalence of specific total immunoglobulin, IgG and IgM antibodies to *T. gondii* in blood donors from Loei Province, Northeast Thailand. Southeast Asian J. Trop. Med. Publ. Hlth. 31:123-7.
- **Pinon, J, Dumon, H, Chemla, C, 2001:** Strategy for diagnosis of congenital toxoplasmosis: Evaluation of methods comparing mothers and newborns and Standard methods for post-natal detection of IgG, IgM and IgG antibodies. J. Clin. Microbiol. 39:2267-71.
- Rifaat, MA, Morsy, TA, Salem, SA, Khalil, H M, Garnham, PC, 1963: Toxoplasmosis in U. A.R.: A survey of toxoplasmosis by the skin test, the agglutination test and the Sabin Feldman dye test among the population of 12 Governorates. Egypt. J. Soc. Med. 2, 3:151-6.
- **Rifaat, MA, Michael SA, Morsy, TA, 1968:** Toxoplasmin skin test survey among buffaloes and cattle in U.A.R. (Preliminary report). J. Trop. Med. Hyg. 72:297-8.
- Santiago, H, Oliveira, M, Bambirra, E, Faria, A, *et al*, 1999: Co-infection with *T. gondii* inhibits antigen-specific Th2 immune responses, limiting tissue inflammation and parasitism in BALB/c mice infected with *Leishmania major*.

- Infect. Immun. 67:4939-44.
- Sarwat, MA, Ahmed, AB, Zamzami, OM, Fawzy, AFA, Morsy, TA, 1993: *Toxoplasma gondii* in Saudi blood donors: Serological study using 3 tests. J. Egypt. Soc. Parasitol. 23, 3:751-7.
- Saleh, AMA, Ali, HA, Ahmed, SAM, Hosny, SM, Morsy, TA, 2014: Screening of *Toxoplasma gondii* infection among childbearing age females and assessment of nurses' role in prevention and control of toxoplasmosis. J. Egypt. Soc. Parasitol. 44, 2:329-42
- Saleh, AMA, Adam, SM, Abdel-Motagaly, A ME, Ibrahim, AMA, Morsy, TA, 2015: Human Babesiosis: A general review with special reference to Egypt. J. Egypt. Soc. Parasitol. 45, 3: 493-510.
- Saleh, AMA, Adam, SM, Ibrahim, AMA, Morsy, T.A, 2016: Malaria: A general minireview with reference to Egypt. J. Egypt. Soc. Parasitol. 46, 1:35-48
- **Sewell. DL, 1995:** Laboratory-associated infections and biosafety. Clin. Microbiol. Rev. 8, 3: 389-405
- Shiva, F, Sanaei, A, Shamshiri, AR, Ghotbi, F, 2011: Survey of need stick injuries in pediatric health personnel of 5 university hospitals in Tehran. J. Pakistan Med. Assoc. 61,:127-131
- **Shulman, IA, 1994:** Parasitic infections and their impact on blood donor selection and testing. Arch. Pathol. Lab. Med. 118:366-70.
- Siegel, J, Rhinehart E, Jackson M, Chiarello, L, Health Care Infection Control Practices Advi-sory Committee, 2007: Guideline for isolation precautions: Preventing transmission of infectious agent in health care settings. Am. J. Infect. Control 35, 10:S65-164.
- **Siegel, S. Lunde, M, et al, 1971:** Transmission of toxoplasmosis by leukocyte transfusion. Blood 37:388-94.
- **Statutory Instrument, 2013:** The Health and Safety (Sharp Instruments in Healthcare) Regulations 2013 No. 645. Printed and published in the UK by the Stationery Office Limited under the authority and superintendence of Carol Tullo, Controller of Her Majesty's Stationery Office and Queen's Printer of Acts of Parliament.
- **Sudan, V, Jaiswal AK, Shanker, D, 2013:** Recent trends in the diagnosis of toxoplasmosis. Clin. Rev. Opinions 5, 2:11-7
- **Sundar, P, Mahadevan, A, Jayshree, R, et al, 2007:** *Toxoplasma* sero-prevalence in healthy voluntary blood donors from urban Karnataka. Indian J. Med. Res. 126: 50-5.

- **Suzuki, Y, 2002:** Host resistance in the brain against *Toxoplasma gondii.* J. Infect. Dis; 185: 58-65.
- **Svobodova, V, Literak, I, 1998:** Prevalence of IgM & IgG antibodies to *T. gondii* in blood donors in the Czech Republic. Eur. J. Epidemiol. 14:803-5.
- **Tan, HK, Schmidt, D, Stanford, M, 2007:** Risk of visual impairment in children with congenital toxoplasmic retinochoroiditis. Am. J. Opthalmol. 144:648-58
- **Tarantola, A, Rachline, AC, Konto, C, Houzé, S, Lariven, S, et al, 2004:** Occupational malaria following needle-stick injury. Emerg. Infect. Dis. 10:1878-80.
- **Tenter, A, Heckeroth, A, Weiss, L, 2000:** *Toxoplasma gondii* from animals to humans. Int. J. Parasitol. 30:1217-58.
- **Thomas WJ, Murray JR, 2009:** The incidence and reporting rates of needle-stick injury amongst UK surgeons. Ann. Roy. Coll. Surge. England 91, 1:12-17
- Ustun, S, Aksoy, U, Dagci, H, Ersoz, G, 2004: Frequency of toxoplasmosis in patients with cirrhosis. Wd. J. Gastroenterol. 10, 3:452-4.
- Weiss, LM, Wittner, M, Tanowitz, HB, 2011: The treatment of babesiosis. N. Engl. J. Med. 344:773-9.
- **Yanaza, A, Kumari, P, 1994:** Prevalence of *Toxoplasma* anti-bodies in blood donors in Al-Hassa. Ann. Saudi Med. J. 14:23-30.
- **Yazar, S, Eser, B, Yay, M, 2006:** Prevalence of anti-*T.gondii* antibodies in Turkish blood donors. Ethiop. Med. J. 44:257-61.
- **Zghair, KH, Al-Qadhi, BN, Mahmood, SH, 2015:** The effect of toxoplasmosis on the level of some sex hormones in males' blood donors in Baghdad. J. Parasit. Dis. 39, 3:393-400.
- Wishahy, AO, Rifaat, MA, Morsy, TA, El Naggar, BA, 1971a: Toxoplasmosis in an Egyptian child with mediastinal lymphadenopathy. J. Trop. Med. Hyg. 74:82.
- Wishahy, AO, Rifaat, MA, Morsy, TA, El Naggar, BA, 1971b: Toxoplasmosis in a case of neonatal jaundice. Gaz. Egypt. Paediatr. Assoc. 19, 4:307-9.
- Wishahy, AO, Rifaat, MA, Morsy, TA, El Naggar, BA, 1971c: Some observations on toxoplasmosis in Egyptian children. J. Egypt. Soc. Parasitol. 2/3:149-50.