

Toxoplasmosis in HIV and non HIV prisoners in Malaysia

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Abstract. This is the first Malaysian study to determine the trend and risk factors of *Toxoplasma gondii* infection in HIV and non-HIV among prisoners in terms of socio-demographic and behavioural characteristics, clinical presentations and haematological distributions. Blood samples from 303 participants, comprising 133 HIV positive and 170 HIV negative inmates were collected in EDTA and plain tubes. Two mls of each blood sample in plain tubes were centrifuged at 1500 rpm for 10 minutes and the sera obtained were subjected to ELISA for detection of *Toxoplasma* IgM and IgG antibody towards *Toxoplasma* antigen. Seropositive samples for *Toxoplasma* IgM or both *Toxoplasma* IgM and IgG were further tested with Novalisa *Toxoplasma gondii* IgG avidity test to rule out acute from latent infections. Blood in EDTA tubes were sent to Clinical Diagnostic Lab (CDL), University Malaya Medical Centre (UMMC), Kuala Lumpur for complete blood count and differential count analysis. Overall seroprevalence of anti-*T. gondii* antibodies was detected in 41.9% (127 out of 303) of the participants. Anti-*T. gondii* antibodies was detected in 63.2% (84 out of 133) of HIV positive subjects and in 25.3% (43 out of 170) of HIV negative subjects. Seroprevalence of anti-*T. gondii* antibodies was significantly higher in HIV positive than in HIV negative subjects (OR = 5.06; 95% CI = 3.09-8.30; $p < 0.001$). The rate of *T. gondii* seropositivity increased significantly in those aged 40 years and above, HIV positive individuals and those with history of drug abuse. White blood cells (WBCs), neutrophils and basophils counts decreased significantly in those infected with *Toxoplasma*. Creating awareness about *T. gondii* infection and follow-up of their status is recommended. Moreover, screening of *T. gondii* infection in HIV-infected individuals should be considered for better treatment and management, including control and prevention.

INTRODUCTION

Toxoplasmosis is a zoonotic disease caused by an ubiquitous, intracellular, protozoan parasite known as *Toxoplasma gondii*. It is estimated one third of the world population were infected with *T. gondii* but most are asymptomatic (Hill *et al.*, 2005). *Toxoplasma* infection is usually acquired through ingestion of contaminated raw meats containing tissue cysts of *Toxoplasma*, ingestion of vegetables or water contaminated with oocyst from cat faeces or via transplacental. *Toxoplasma* infection can also be acquired by accidental inoculation of zoite in the laboratory, blood or leukocyte transfusion, or from a transplanted organ,

however these incidents are rare (Nissapatorn & Abdullah, 2004; Ngui *et al.*, 2011). Toxoplasmosis in healthy individuals is clinically unapparent (presenting with flu-like symptoms) or self-limited. Whilst, infected immunocompromised (i.e. HIV-infected) individuals may experience severe symptoms due to reactivation of latent toxoplasmosis which leads to development of toxoplasmic encephalitis (TE). They may present with fever, confusion, headache, seizures, nausea, and poor coordination (CDC, 2014). TE is one of the most frequent opportunistic infections (OIs), particularly in patients with full-blown acquired immunodeficiency syndrome (AIDS). TE complicates AIDS (Nissapatorn *et al.*, 2004).