



## Case Study

# Myocardial and Cerebral Toxoplasmosis with Cerebral Tuberculosis in a patient with Acquired Immunodeficiency Syndrome – Autopsy Case Report

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## ABSTRACT

### Keywords

Toxoplasmosis,  
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Toxoplasmosis is a parasitic infection cause by *Toxoplasma gondii*. This parasite is associated with congenital infection and can cause abortion, encephalitis or systemic infection in immune- compromised patients. In this study, one autopsy case of Myocardial and central nervous system toxoplasmosis with cerebral tuberculosis in immunocompromised patient is reported. 35-year-old female presented with Fever, headache, nausea and vomiting, chest pain, palpitation, fatigue since one weeks. Disseminated toxoplasmosis is a major health problem in immune- compromised patients

## Introduction

*Toxoplasma gondii* is known as one of the most common infectious protozoan parasites that has a worldwide distribution (Boothroyd John and Grigg Michael, 2002; Dubey, 2008; Petersen, 2007). Cats are recognized as the only definitive host of *T. gondii*, but humans and other warmed-blooded animals and birds are as intermediate hosts. Humans can be infected by ingestion of raw or uncooked meat containing tissue cysts, unwashed vegetables, contaminated water or soil. Congenital toxoplasmosis occurs via the trans-placental route (Kasper *et al.*, 2004). *Toxoplasma* infection is largely asymptomatic, but in those individuals who are immune-compromised with AIDS, malignant patients under chemotherapy or organ transplant recipients can become

disseminated and cause severe toxoplasmosis and/or encephalitis (Gallino *et al.*, 1996; Velimirovic, 1984; Wanke *et al.*, 1987).

In the present article a case of Myocardial and cerebral toxoplasmosis with cerebral tuberculosis in acquired immunodeficiency syndrome patients from west Maharashtra India is reported.

## Case Report

A 35-year-old female was admitted with Fever, headache, nausea and vomiting, chest pain, palpitation, fatigue since one week. And one episode of convulsion followed by coma. Husband died 1 year back due to acquired immunodeficiency syndrome.

## **Examination**

General condition was poor, pulse 110/min (tachycardia), Respiratory rate 27/min, CNS no response to stimuli, generalized hypotonia.

On investigation – Hb 9 gm/dl, TLC-1500/cumm, Platelet count – 50,000/cumm, ESR – 80 at the end of 1 hour, ELISA for HIV test was positive which was confirmed by Western blot test. CD 4 count was 50 /cumm. CSF examination showed 270 lymphocytes/microlitre and tubercle bacilli were present on ZN Staining.

## **Gross examination**

Subarachnoid space showed thick exudate at the base of brain, meninges were congested and slightly opaque. All other organs showed congestion.

## **Microscopy**

Section from both atria and both ventricles showed focal areas of necrosis, oedema along with mononuclear cell infiltrate consisting of lymphocytes and plasma cells, Pseudocysts containing bradyzoites of *Toxoplasma gondii* within and outside muscle fibre were seen. Sections from brain showed inflammatory infiltrate along with pseudocysts containing bradyzoites of *Toxoplasma gondii*. Section from meninges and brain showed scattered epithelioid cells at places forming ill defined granulomas. Rest of organs showed congestion.

## **Discussion**

*T. gondii* infections can cause acute infection in immunosuppressed patients and congenital toxoplasmosis. It has been known that 15- 58% of humans are infected with *T. gondii*, but the rate of infection varies due to

many factors (Walzer and Genta Robert, 1989).

Disease in immune-compromised individuals (i.e. persons with AIDS, transplant recipients, immuno-suppressive drug users) usually due to reactivation of latent infection and can lead to lethal meningoencephalitis, focal lesion of CNS and less commonly, myocarditis or pneumonitis.

Diagnosis of toxoplasmosis is made clinically, radiologically and by serology, histology or by molecular methods. Toxoplasmosis associated with HIV infection manifest primarily as toxoplasmic encephalitis and is a frequent cause of focal CNS lesions. Characteristically, toxoplasmic encephalitis presents with headache, altered mental status and fever.

Most common focal neurological signs are motor weakness, speech disturbance. Patients can present with seizures, cranial nerve abnormalities, visual field defects, sensory disturbances, cerebellar dysfunction, meningismus, movement disorders and neuropsychiatric manifestations (Rathore, 2005). Ocular and pulmonary diseases are the most common presentations in patients with cerebral toxoplasmosis (Rabaud *et al.*, 1994). Toxoplasmosis rarely presents as a rapidly fatal form of diffuse encephalitis.

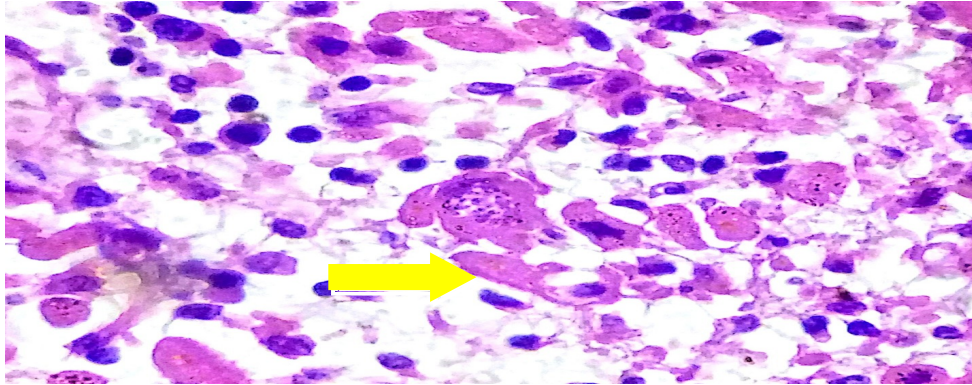
The most commonly used serologic tests to detect the presence of anti-*T. gondii* IgG and IgM. IgG antibodies can be detected with the Sabin Feldman dye test (considered to be the gold standard). CSF from patients with toxoplasmic encephalitis may reveal mild pleocytic mononuclear predominance and protein elevation.6 Polymerase chain reaction (PCR) based detection of *T. gondii* DNA has sensitivity of 12-70 % and specificity of 100% in patients with

toxoplasmic encephalitis (Parmley *et al.*, 1992).

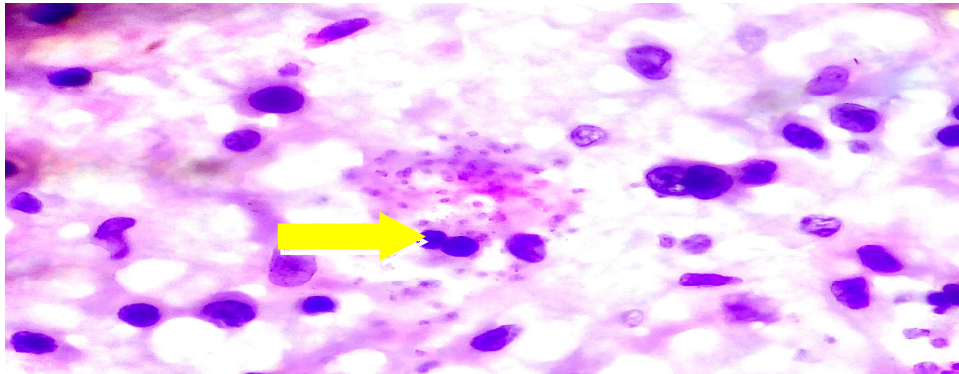
isolation of *T.gondii* from culture of body fluids (Blood, CSF, and bronchoalveolar lavage fluids) or tissue biopsy.

Toxoplasmosis can be diagnosed by

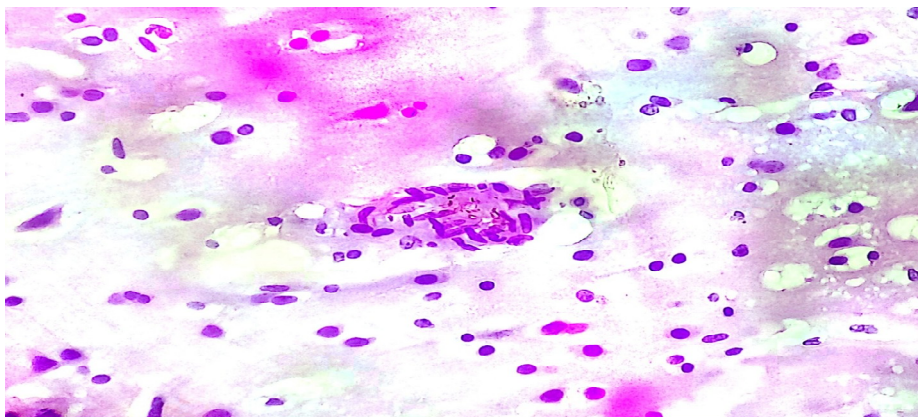
**Figure.1** Photomicrograph of myocardial toxoplasmosis (Bradyzoites within myocardial fiber)



**Figure.2** Photomicrograph of Cerebral toxoplasmosis (Bradyzoites)



**Figure.3** Photomicrograph of Cerebral tuberculosis



Brain biopsy showing tachyzoites or cyst provides a definitive diagnosis for toxoplasmosis encephalitis. Conservative approach is particularly helpful when the lesion is surgically inaccessible. Combination of Pyrimethamine/Sulfadiazine and folinic acid is considered the standard regime for the treatment of toxoplasmosis encephalitis.

## Conclusion

We present an autopsy case of cerebral and cardiac toxoplasmosis with tuberculous Meningitis. Broad differential should be kept in mind when seropositive patient present with symptoms of meningitis and appropriate testing should be performed. When the diagnosis is made, treatment is required for symptomatic patients who are immunocompromised. Proper education and counseling regarding risk factors can reduce the incidence and risk of acquiring the infection.

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