

HIV-Related Toxoplasmosis Infection. A Rare Case of Simultaneous Cardiac and Cerebral Involvement

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Abstract: Toxoplasmosis is a significant opportunistic infection among people living with HIV (PLHIV), primarily manifesting as cerebral disease. Cardiac involvement, while rare, significantly complicates clinical management. This report describes an unusual presentation of concurrent cerebral and cardiac toxoplasmosis in a 50-year-old HIV-positive male initially treated for severe malaria. Despite initial symptomatic relief, the patient rapidly deteriorated neurologically and subsequently succumbed. The autopsy revealed characteristic cerebral and myocardial lesions confirmed by histopathology. The case underscores the diagnostic complexities and the necessity for high clinical suspicion in managing PLHIV with atypical presentations, especially in malaria-endemic areas.

Keywords: toxoplasmosis, HIV/AIDS, cerebral toxoplasmosis, cardiac toxoplasmosis, diagnostic challenges, opportunistic infections

Introduction

Toxoplasmosis, caused by the protozoan parasite *Toxoplasma gondii* [*T. gondii*], is a widespread zoonotic infection affecting approximately one-third of the global population, with varying prevalence based on geographic location, socioeconomic status, and dietary habits.^{1,2} A recent meta-analysis reported an estimated global seroprevalence of *T. gondii* infection at 31%, with Ghana having the highest prevalence.¹ An earlier study published in the Lancet estimated the prevalence of *T. gondii* infection among PLHIV at 35.8%, with roughly 87.1% of cases occurring in sub-Saharan Africa (SSA).² Cats are the primary hosts, and humans are usually infected opportunistically, especially in immunocompromised individuals, when they ingest oocysts in undercooked meat from other infected animals, which serve as intermediate hosts.³ The immune status of the infected host plays a key determinant role in the phenotypic expression of the infection. In immunocompetent hosts, toxoplasmosis is typically asymptomatic or causes mild flu-like symptoms. In immunocompromised patients, particularly those with advanced HIV/AIDS, it poses a significant risk due to its potential for reactivation and severe clinical manifestations, typically with CD4 counts below 100 cells/ μ L. *T. gondii* infection most commonly presents as cerebral toxoplasmosis, characterized by neurological deficits, seizures, and space-occupying brain lesions.^{4,5} Cerebral toxoplasmosis is a leading cause of focal neurological complications and morbidity among PLHIV globally, especially in resource-limited settings where early diagnosis and appropriate prophylaxis remain challenging.⁶ Cardiac toxoplasmosis, on the other hand, is relatively uncommon, and its diagnosis is frequently overlooked or delayed due to nonspecific clinical symptoms, lack of clinical suspicion, and absence of distinct radiological signs.⁷ Cardiac involvement can manifest in several non-specific forms such as myocarditis, pericarditis, potentially leading to severe complications, including heart failure and arrhythmias, further complicating the clinical picture.^{7,8} Simultaneous cerebral and cardiac toxoplasmosis is exceedingly rare, and cases documented in the literature remain limited.^{3,8,9} Other commonly observed complications associated with *T. gondii* infections include focal

neurological deficits, ocular complications, stupor, coma, and death.³ This case report presents a unique clinical scenario wherein a newly diagnosed HIV patient, initially managed for severe malaria, subsequently deteriorated rapidly and died. Autopsy was requested and findings confirmed concurrent cerebral and cardiac toxoplasmosis. This case highlights the diagnostic complexity, the need for heightened clinical suspicion, and the crucial role of timely diagnosis and understanding when to refer, in order to prevent fatal outcomes.

Case Report

30th/April/2025: A 50-year-old male newly diagnosed HIV patient with a CD4+ count of 25 cells/ μ L presented to a peripheral health center with severe frontal and temporal headaches and nausea of unspecified duration. He was also a well-known alcoholic and hypertensive patient with a history of poor adherence to antihypertensive therapy. There was no associated fever or neck stiffness. It was not clear from the notes when he had been diagnosed with HIV.

He appeared ill physically but was not in distress. He was oriented to time, place, and person, with mild pallor. His blood pressure was 145/90 mmHg. Initial management included intramuscular diclofenac 75 mg, intravenous (IV) antibiotics (X-pen) 2 million units 6 hourly, and fluid resuscitation with 500 mL of IV normal saline followed by 500 mL of 5% dextrose. He was started on tenofovir/lamivudine/dolutegravir (TLD) and prophylactic co-trimoxazole 960 mg.

1st/May/2025: Laboratory test results: Serum cryptococcal antigen (CrAg) test, and serum and urine Lipoarabinomannan (LAM) tests, were also negative. Malaria rapid diagnostic test (MRDT) was reported as positive. He was started on intravenous artesunate 130 mg, which was given at 0 hours, and at 12 hours. The 24-hour dose was missed. Paracetamol 1g was also administered 8 hourly for symptomatic pain relief. Notably, there was no record of fever and no record of temperature measurement in the patient file.

2nd/May/2025: The patient showed marked improvement per records. He was afebrile, and a repeat MRDT was reported negative, and the patient reported complete resolution of his headaches. He then left the facility without knowledge of the medical team. However, on the morning of 3rd May, the clinical course changed significantly. He was brought back by friends in a severely deteriorated state, unable to walk and was suspected to be intoxicated with a strong alcoholic smell. He was vomiting with tremors in the upper limbs. His pulse rate was 58 beats per minute (bpm), and random blood sugar measured 8 mmol/L. He was started on IV metoclopramide 10 mg to manage the vomiting.

Later that evening at around 6:40 PM, the patient developed several generalized tonic-clonic (GTC) seizures, with persistent unconsciousness between episodes. His Glasgow Coma Scale (GCS) score dropped to 8/15, oxygen saturation on room air was 85%, and pulse rate had risen to 112 bpm. He was given diazepam 10 mg stat, and a nasogastric tube (NGT) was inserted. Plans were made for urgent review by a medical officer (MO/MD).

Despite resuscitative efforts, the patient's condition continued to worsen. By 9:25 PM, he had developed worsening respiratory distress with significant desaturation and subsequently went into cardiac arrest. Cardiopulmonary resuscitation (CPR) was initiated but was unsuccessful, and the patient was pronounced dead. The body was referred for post-mortem at a regional referral hospital, where we first interfaced with it accompanied by a summary of the medical records.

Main Autopsy Findings

The autopsy revealed the following significant findings. The brain was severely edematous with flattened gyri and narrowed sulci. Also noted were round gray-white lesions in the left temporal lobe, the left Thalamus, and just above the right corpus callosum at the grey-white mater interface [See [Figure 1](#)]. The largest lesion was in the left temporal region (about 5 cm in widest diameter) with areas of hemorrhage and liquefactive necrosis [See [Figure 1D](#)]. The heart exhibited multiple distinct oval infarct-like areas in the interventricular septum [See [Figure 1](#)]. No obvious lesions were seen in other body organs. The lesions above were fixed in 10% neutral buffered formalin and processed for microscopy.

Microscopic Examination

H&E-stained sections of the brain demonstrated extensive parenchymal necrosis, inflammation composed of lymphocytes, and macrophages with perivascular inflammatory cell cuffing forming perivascular microglial nodules. There was

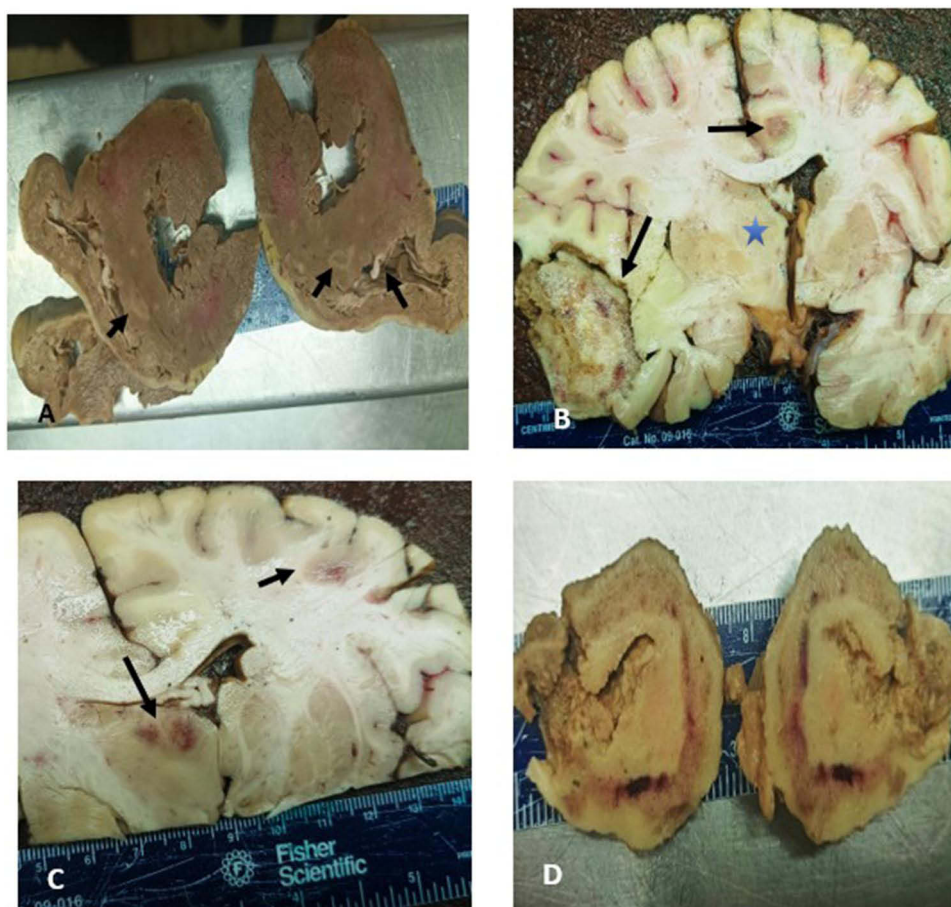


Figure 1 Gross images of the heart and the brain showing the lesions. Gross pictograms of the heart and the brain after fixation in formalin. (A) Cross section of the heart showing multiple intramural oval to round grey-colored lesions (black arrows) of *Toxoplasma gondii* in the heart. (B) Cut section of the brain shows round lesions in the right temporal lobe and above the corpus callosum on the left (see black arrows). The right lateral ventricle is compressed by a lesion in the thalamus shown in panel. (C) (see blue asterisk). (D) A closer look at the biggest lesion from the temporal lobe cut cross-sectionally, showing necrosis and areas of hemorrhage.

also evidence of meningitis with inflammatory cells in the pia mater. Several bradyzoites of *Toxoplasma gondii* were scattered at the edges of the necrotic foci mixed with gliotic tissue [See Figure 2]. Sections from the heart showed multiple focal areas of myocardial necrosis accompanied by similar inflammation, and rare bradyzoites were seen within the myocytes [See Figure 3].

Discussion

Toxoplasmosis remains a pivotal concern among immunocompromised populations, notably those with HIV/AIDS.^{3,10} Typically presenting as cerebral toxoplasmosis, clinical symptoms such as headaches, seizures, and altered consciousness dominate. In the case above, the patient presented with headaches, and *T. gondii* infection should have been suspected by the clinician who was managing this patient at his earliest presentation, given the fact that he was a newly diagnosed HIV patient with such reduced CD4+ cell count. On the other hand, the clinician suspected cryptococcal meningitis and he/she requested for serum cryptococcal antigen test, which returned negative. More often than not, such patients who present at peripheral centers are treated for malaria, as was the case here; however, it can be suspected that the initial MRDT was a false positive. It is therefore not surprising that a day later, MRDT was repeated, and it was reported as negative despite the observed clinical improvement with the antimalarial therapy.

Cardiac involvement is notably rare, often missed clinically, and typically diagnosed post-mortem.^{7,10} In this case, it was next to impossible to even suspect it. Myocarditis or pericarditis due to toxoplasmosis can result in severe morbidity and mortality. Histopathological confirmation, through visualization of tachyzoites and bradyzoites, remains definitive.^{7,8} Several other issues

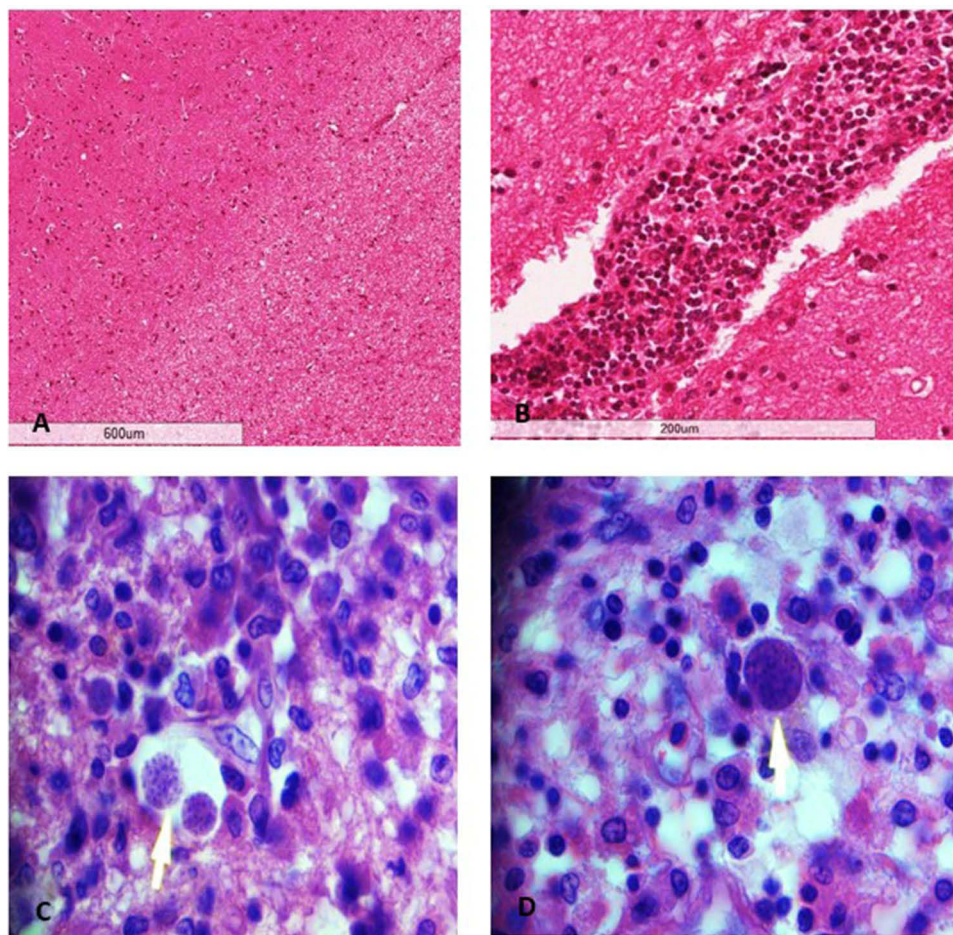


Figure 2 H&E sections of the brain lesions. H&E-stained sections of the brain. **(A)** A low-power Image showing the interface between the normal brain (upper right corner) and the affected necrotic brain (lower left corner). **(B)** A high-power image of the arachnoid with evidence of meningitis. **(C and D)** show bradyzoites (yellow arrows) of *T. gondii* containing multiple tachyzoites at an optimized magnification in the background of a gliotic brain with inflammation.

Note: A and B were images taken from whole-slide images scanned with an Aperio scanner, while C and D are high-power images of the same brain taken using an infinity capture to optimize magnification for a clear view.

made management of this patient difficult. While we acknowledge that the disease was advanced, the patient also lacked compliance to treatment that he escaped from the facility and was only brought back the next day in a much worse state than he had been in. This escape could have been caused by several factors, and key among the many could be a craving for alcohol, as the patient returned when he was intoxicated.

What Could Have Been Done Better?

It was probably too late to manage the patient for a better outcome to be achieved. This patient needed to be referred to a higher-level facility at the very earliest presentation where diagnostic imaging like computed tomographic (CT) scans could be done to aid diagnosis of cerebral toxoplasmosis. Also, such facilities have infectious disease specialists and multidisciplinary teams, which would better manage such complex cases.

Although Highly Active Antiretroviral Therapy (HAART) was started together with cotrimoxazole, it was too late for it to have any benefit. The first diagnosis of malaria was done based on MRDT, and then treatment was initiated. It was probably better to first confirm with a blood smear to avoid misdiagnosis. The patient's escape from the health facility to go and drink alcohol worsened his condition and prognosis further.

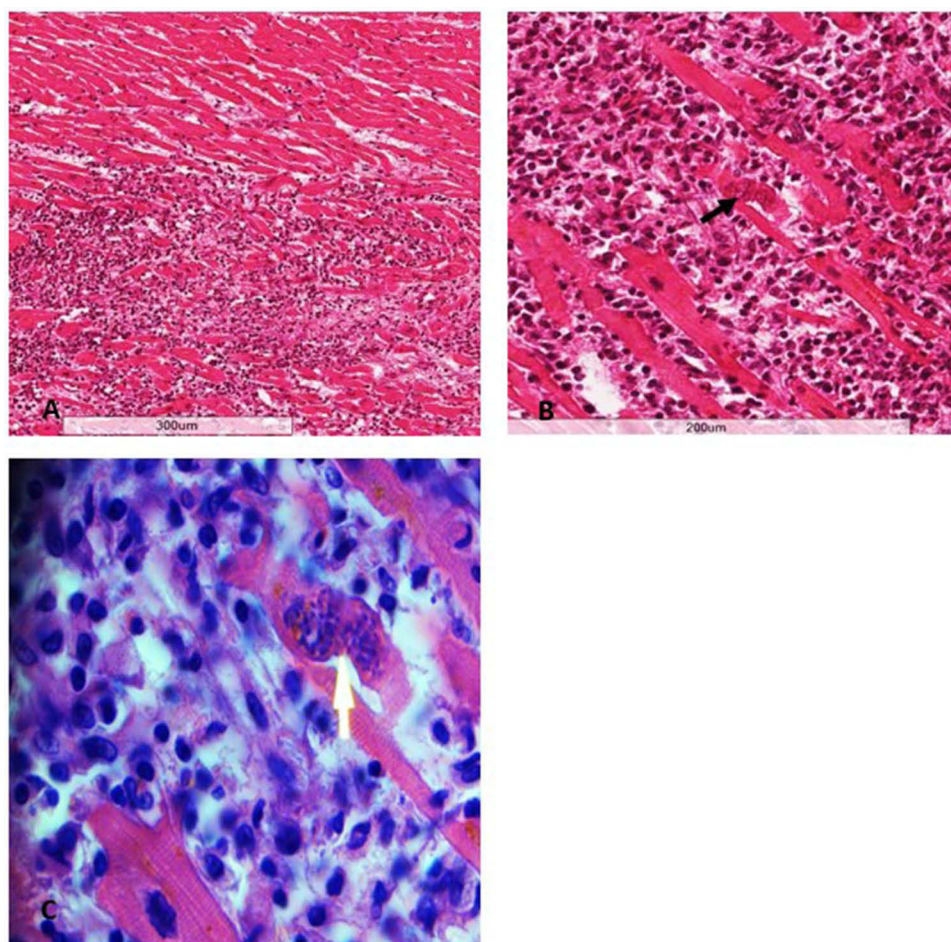


Figure 3 H&E sections of the heart at low and high power. H&E-stained sections of the heart showing evidence of myocarditis. At low-power (**A**) and high-power in (**B**), there is an intense monocytoid inflammatory cell infiltrate. In (**B** and **C**), we can appreciate the loss of myocyte fibers and intense infiltration by mononuclear cells. Also shown here are bradyzoites within the muscle fiber in (**B** and **C**) (black and yellow arrows respectively). (**C**) is an image taken with an infinity capture to optimize magnification while (**A** and **B**) were taken from VSI scanned by an Aperio scanner.

Diagnosis of *T. Gondii* Infection

Definitive diagnosis of *T. gondii* infection involves clinical evaluation complemented by laboratory investigations and histopathological examination. Serological testing, particularly enzyme-linked immunosorbent assays (ELISA) and immunofluorescence antibody tests (IFAT), is essential for the detection of anti-toxoplasma IgG and IgM antibodies, aiding differentiation between acute and chronic or latent infections.¹¹ Molecular techniques, notably polymerase chain reaction (PCR) targeting the highly sensitive and specific B1 gene or the repetitive 529 bp DNA fragment, are invaluable—especially in immunocompromised patients where antibody responses may be impaired or ambiguous.¹¹ Definitive confirmation often requires histopathological identification of bradyzoites or tachyzoites within tissue biopsies, which is diagnostic and confirms active disease.¹² There is also a role of imaging in the diagnosis of toxoplasmosis, though imaging alone is not enough. The imaging techniques used involve CT scan (Computed Tomography), MRI (Magnetic Resonance Imaging), ultrasonography, and nuclear imaging.¹³ *T. gondii* can also be identified by cytology of Cerebrospinal fluid (CSF) through both lumbar and ventricular taps.^{14,15}

Treatment for *T. Gondii*

Standard therapy for cerebral and systemic toxoplasmosis typically includes combination treatment with pyrimethamine, sulfadiazine, and leucovorin (folinic acid) to mitigate pyrimethamine-associated hematological toxicity.¹⁶ Treatment duration commonly spans at least 4–6 weeks, or until clinical and radiological improvement is achieved.¹⁶ Alternative

therapies—such as pyrimethamine combined with clindamycin or atovaquone—are used in cases of intolerance or hypersensitivity to sulfadiazine.¹⁷ In severe cases with cerebral involvement, adjunctive corticosteroids may be used cautiously to control cerebral edema, with careful monitoring due to the potential risk of exacerbating latent infection.^{16,17} For HIV-positive patients, initiation or optimization of antiretroviral therapy (ART) remains a cornerstone to restoring immune function. Following acute therapy, secondary prophylaxis using pyrimethamine, sulfadiazine, and leucovorin is indicated until sustained immune recovery (CD4 counts consistently above 200 cells/ μ L) is achieved.^{17,18} In the presented case, the initial misdiagnosis as severe malaria highlights the critical need for heightened suspicion in HIV patients presenting with neurological and atypical systemic signs, particularly in resource-constrained settings.^{1,8} Appropriate early intervention, including anti-toxoplasma therapy and supportive measures, could significantly alter outcomes.

Limitations

In our setting, the immunohistochemical stain for *T. gondii* was unavailable and therefore was not done in this case; however, the bradyzoites were clear enough on H&E to confirm the diagnosis. Diagnostic imaging and blood tests were also not provided alongside the hospital record which could have added value to the case report.

Conclusion

This case outlined the rarity and clinical significance of concurrent cerebral and cardiac toxoplasmosis infection among PLHIV and underscored the great importance of autopsy in patients with HIV infection. Early and accurate diagnosis remains challenging yet critical for favorable outcomes. Clinicians must maintain a high index of suspicion and ensure comprehensive diagnostic evaluations in immunocompromised patients presenting with complex clinical syndromes. Future clinical protocols should integrate broader differential considerations in PLHIV presenting acutely ill, regardless of common regional diseases.

Data Sharing Statement

The data and materials of this case report are available from the corresponding author upon request after approval from the Pathology Department and Mbarara Regional Referral Hospital.

Consent and Ethical Considerations

A written informed consent for autopsy was obtained from the next of kin (NOK). After the autopsy, we provided the family with a provisional report stating the probable cause of death pending histological examination for confirmation. Two weeks after burial, the NOK came back to pick the final cause of death report and a death certificate. He briefed about the findings and their significance, and we sought a written consent from him for the use of photographic images of the body parts of the deceased for an academic publication. Institutional approval was not required for this case report.

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Author Contributions

All authors made a significant contribution to the work reported, whether in the conception, study design, execution, acquisition of data, analysis and interpretation. They all took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

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