



## Intracranial toxoplasmosis in retroviral disease: An important differential of many space-occupying lesions in resource-limited settings

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### Abstract

**Background:** Intracranial toxoplasmosis (IT) in HIV could be misdiagnosed for other space-occupying lesions (SOLs).

**Case presentation:** We report of a case of a 52-year-old man who presented with progressive left-sided body weakness of a week and altered sensorium of nine hours. While CT suggested glioma, MRI characterized the lesion better which was suggestive of IT after HIV diagnosis.

The patient was placed on anti-toxoplasmosis and anti-retroviral drugs. Clinical recovery was significant within 12-hours of regimen commencement.

**Conclusion:** A high index of suspicion, MRI and initiating timely therapy are essential to forestall mortality due to this protozoan infection in PLWHA presenting with focal neurological deficits.

Keywords: Toxoplasmosis, Nigeria, Space-occupying lesion, neuroimaging, Protozoon

### Introduction

Intracranial toxoplasmosis (IT) is a leading cause of intracranial mass lesions in persons living with HIV/AIDS (PLWHA) and having significant morbidity and mortality<sup>1</sup>, particularly in low- and middle-income countries. Average prevalence among the immunocompromised ranges from 3-40%<sup>2,3</sup>. Its identification could be challenging despite neuroimaging amidst other relevant diagnostic modalities such as serology and molecular tests while its presentation often overlaps with other space-occupying lesions (SOL), complicating timely diagnosis/management especially in late or missed diagnosis of HIV1. We report a case of a middle-aged man who developed progressive neurological deficit with multiple intracerebral masses suggestive of IT but initially confounded by other SOLs.

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#### Ethical Consideration:

A written informed consent from the patient and an IRB waiver (AMSH/REC/25/60/142) from the health institution were obtained. Adequate explanation was provided to the patient and confidentiality assured.

### Case report:

A 52-year-old man was brought into the emergency room of a private tertiary health institution with complaints of progressive left-sided body weakness of a week and altered sensorium of nine hours prior to presentation. There was associated history of high-grade fever, severe frontal headache, slurred speech and minimal weight loss. Patient's symptoms persisted despite self-medicating with oral anti-malarial. Subsequently, his symptoms worsened, and he developed progressive left-sided body weakness, slurred speech and altered sensorium prompting presentation at our facility. At presentation he was febrile with axillary temperature of 37.8 °C. He was also in altered consciousness and had differential left sided hemiparesis. Blood pressure was 141/84 mmHg and random blood glucose was 115mg/dl. Malaria rapid diagnostic test done was positive for *Plasmodium falciparum* antigen (Abbott diagnostics). Complete blood counts, liver and kidney function tests were essentially normal.

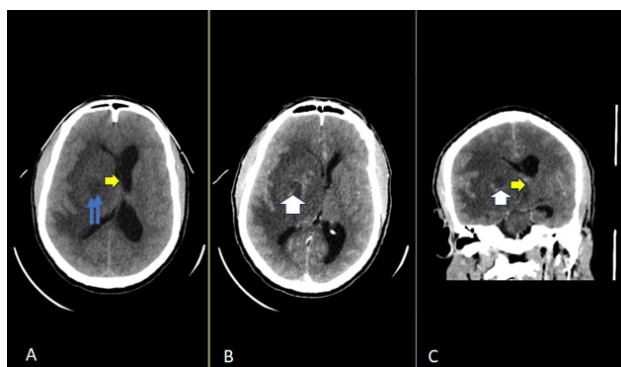


Figure 1: Cranial CT images. Image A is an axial non-contrast, while B and C are post-contrast axial and coronal images respectively. A hypodense lesion is demonstrated in the right basal ganglia (long blue arrows) with extensive perilesional edema, and contralateral midline shift (short yellow arrow); incomplete ring enhancement is seen on post-contrast images (short white arrows in the image B and C).

A provisional diagnosis of right hemispheric cerebrovascular accident was made to exclude intracranial space occupying lesions with poorly treated malaria fever. On contrast-enhanced computed tomography (CT) scan of the brain, a

large, irregular, heterogeneously enhancing right basal ganglia mass with central necrosis, extensive vasogenic oedema, and midline shift were observed. These findings were in keeping with high-grade glioma (Figure 1). Concurrently, immunochromatography for HIV 1 & 2 antibodies was conducted on account of history of weight loss which came out reactive. Subsequently, other SOLs in PLWHA were considered such as intracranial toxoplasmosis (IT), intracranial cryptococcoma and primary central nervous system lymphoma (PCNSL). Magnetic resonance imaging (MRI) was immediately scheduled to redefine the mass intrinsically but was delayed by two weeks due to financial constraints. MRI gave a better characterization of the lesion; revealing bilateral basal ganglia involvement with the right-sided nodular mass showing irregular peripheral/nodular enhancement with central necrosis and patchy areas of diffusion restriction, and the left-sided lesion demonstrating a thick hypointense ring on T2 image and a central hyperintense nidus with the ring demonstrating avid post-contrast enhancement and moderate perilesional oedema (Figure 2). The multiplicity of lesions, location in the basal ganglia, irregular peripheral enhancement and nodular enhancement of the right and left basal ganglia lesions respectively were strongly suggestive of IT.

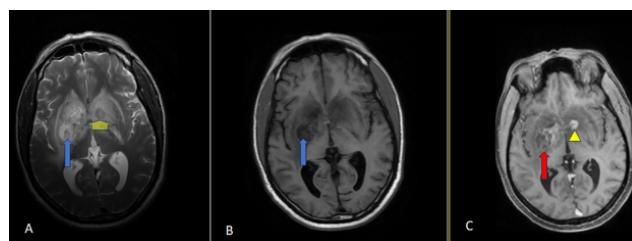


Figure 2: Axial sections of Cranial MRI in T2W, T1W and Post contrast sequences respectively, demonstrating a heterogeneous mass in the region of the right basal ganglia (blue long arrows in image A and B) with irregular and nodular post-contrast enhancement (red long arrow the image C). A rounded nodule is also seen in the left basal ganglia with a thick hypointense ring and central hyperintense nidus (short yellow arrow in image A), It demonstrates an avid post-contrast enhancement (yellow arrow head in image C).

Blood Visitect CD4+ cell test was less than 200cells/ $\mu$ l and serum cryptococcal antigen was negative. Serum *Toxoplasma gondii* serology assay was not affordable at the time.

### Management and Outcomes

Upon the diagnosis of the HIV and with the tentative diagnosis of IT, the patient was commenced immediately on anti-retroviral therapy (ART) comprising tenofovir, lamivudine and dolutegravir. In addition, he was commenced on oral sulfamethoxazole/trimethoprim (Co-trimoxazole) 960mg twice daily. He was also given intravenous dexamethasone 8mg stat, then 4mg 8 hourly and another course of antimalarial treatment.

Patient made progressive clinical improvement within 24-hours of the commencement of ART and twice daily co-trimoxazole as evidenced by improved consciousness and resolving hemiparesis. He was subsequently discharged on the fifth day and continued treatment as outpatient for 6 weeks. A maintenance daily dose of 960mg co-trimoxazole was subsequently continued for sixth months while sustaining ART regimen. After three months of treatment, serum *Toxoplasma gondii* Ig G and IgM assays were found to be positive and negative respectively.

### Discussion

Intracranial toxoplasmosis, caused by obligate protozoan, *Toxoplasma gondii* represents the leading cause of focal central nervous system (CNS) lesion in patients with advanced HIV infection<sup>1</sup>. It remains a major opportunistic infection in regions with high HIV prevalence. Humans become primarily infected by ingesting raw or undercooked meat containing viable tissue cysts, or by ingesting water or food contaminated with oocysts from infected cat feces. Many immunocompetent persons become asymptomatic while the immunocompromised such as our index patient risk re-activation from latent brain cysts due to impaired immune response<sup>4</sup>. Global exposure to over the parasite is about one-third of the world's population<sup>5</sup> while seroprevalence in Nigeria ranges from 26.8%-54.2% especially in PLWHA<sup>6,7</sup>.

Clinical features of IT are non-specific and ranges from headaches, disorientation, drowsiness, hemiparesis, reflex changes, to convulsions. Fever

is a notable symptom observed among patients identified in Nigeria as was observed in our patient<sup>8</sup>. These features also mimic other intracranial SOLs especially PCNSL, tuberculomas, cryptococcoma or metastases<sup>1</sup>. MRI is more sensitive than CT scan in characterizing IT<sup>8</sup> but is less available in low-and middle-income countries. CT done earlier in this patient was at first misleading however, the diagnosis of HIV in the patient averted an immediate surgical intervention. HIV screening coupled with MRI should be advised in SOLs presenting with lateralising signs when in doubt of immune competence<sup>9</sup>. Since infective SOLs are more common in PLWHA than non-infectious SOLs in low-and middle-income countries<sup>1</sup>, it is imperative that a common presentation of a rare disease is less likely than a rare presentation of a common disease<sup>10</sup>. Moreover, the rapid response to anti-toxoplasmosis therapy in any case of suspected IT is a good diagnostic tool<sup>1</sup>. In resource-limited settings IT diagnosis is usually based on a combination of clinical suspicion, radiological findings, and response to empiric anti-toxoplasma therapy<sup>1</sup>. Further diagnostic modality explored in our patient was *Toxoplasma gondii* serology test (Enzyme-linked immunosorbent assay). This was conducted after three months of treatment and the presence of IgG in the absence of IgM connotes a resolution of acute toxoplasmosis<sup>11</sup>. Molecular method such as polymerase chain reaction (PCR) is another diagnostic method suitable for toxoplasmosis but was inaccessible as at management of our patient. PCR is now used to complement serological diagnosis in toxoplasmosis<sup>12</sup>. Moreover, genetic typing of this protozoan using more specific PCR modifications such as with restriction fragment length polymorphism could identify genotypes which are useful in outbreak settings of toxoplasmosis<sup>12,13</sup>. However, aforementioned neuroimaging techniques as used by our patient are important to facilitate diagnosis and monitor therapeutic effect of IT<sup>2,13</sup>.

Co-trimoxazole (sulphamethoxazole-trimethoprim) being the more available and inexpensive anti-toxoplasmosis medication in our setting was aptly commenced on the awareness of the HIV status even before the MRI results suggested IT. The immediate clinical response in the

patient was supportive of the provisional diagnosis<sup>1</sup>. Co-trimoxazole has been widely used for the management of IT with great success and low relapse rate<sup>14</sup>. Other drugs useful against *Toxoplasma gondii* include pyrimethamine and sulphadiazine or spiramycin<sup>15</sup>, however, the accessibility and success rate of co-trimoxazole has enhanced its use. These drugs have various modes of action against the protozoan, while some inhibit folate synthetic pathway as in co-trimoxazole others directly block protein synthesis subunits thereby hindering the genetic building blocks of the parasite<sup>15</sup>. Independently, some of these drugs cannot achieve cure unless in combination as used in our patient<sup>15</sup>. Adjunctive corticosteroid was also administered on account of the significant cerebral oedema observed in the neuroimaging. This was in combination with the ART administered concomitantly. This therapy was truly lifesaving considering the morbidity and mortality associated with this protozoan infection.

## Conclusion

Early recognition of IT in PLWHA with focal neurological deficits is crucial. Clinicians practicing in resource-limited settings must maintain a high index of suspicion, prioritize neuroimaging (MRI) in SOLs and initiate timely empiric anti-toxoplasma therapy when findings are suggestive.

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