Cerebral Toxoplasmosis in an Immunocompetent Individual Presenting as a Solitary Space-Occupying Lesion: A Case Report

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Cerebral toxoplasmosis · Immunocompetent individual · Space-occupying lesion · Basal ganglia

Abstract
We report a case of an apparently healthy, immunocompetent individual who presented with headache and symptoms suggestive of raised intracranial pressure. Neurological imaging revealed marked edema in the basal ganglia. His history was remarkable for sustained contact with domestic felines. He was started on trimethoprim-sulphamethoxazole based on history, strongly positive serology, and radiological findings. He showed a marked response to treatment. The characteristic radiological findings, serology, and dramatic response to treatment confirmed the diagnosis in this case. A tissue diagnosis was not required in our case. This case highlights that immunocompetent individuals can develop neurological manifestations. Though cerebral toxoplasmosis is classically associated with multiple lesions, our case highlights that presentation as a solitary space-occupying lesion is also possible. Further research is needed to ascertain if prolonged exposure or extensive exposure is associated with severe manifestations.

Introduction
Toxoplasmosis is caused by the organism Toxoplasma gondii. This disease usually manifests as a subclinical infection or with nonspecific flu-like symptoms. Neurological disease is usually linked with reactivation of latent infection in individuals with impaired immunity. The treatment is mainly with pyrimethamine-sulfadiazine or trimethoprim-sulphamethoxazole if the first-line agent is not available.

Case Presentation
A 35-year-old, previously healthy male presented to the emergency with complaints of headache and increased drowsiness of 1-week duration. The headache had come suddenly. It was predominantly over his right temporal region, was throbbing, nonradiating, continuous with nocturnal worsening, associated with watering from the ipsilateral eye, and mild left-sided facial deviation. He also reported nonspecific dizziness upon standing. The headache was moderate to severe, was temporarily relieved with analgesics, and was aggravated by coughing and straining.

There was no reported history of loss of consciousness, fever, visual disturbance, difficulty in speech, difficulty in swallowing, weakness or numbness of limbs, jerky movements of face or limbs, or urinary or fecal incontinence. The systemic review was unre-
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Table 1. Patient’s investigations

| Test                  | Value     | Test                | Value    |
|-----------------------|-----------|---------------------|----------|
| Total leukocyte count | 15,700/microl | LDH                | 293 U/L  |
| Hemoglobin            | 15.8 gm/dL | Procalcitonin      | 0.056    |
| Platelet count        | 226,000/microl | CRP                | 1.2 mg/L |
| Bilirubin             | 0.74 mg/dL | HbA1C              | 6%       |
| AST                   | 31 U/L    | HIV serology       | Negative |
| ALT                   | 77 U/L    | Toxoplasma serology| IgG 57 U/L IgM negative |
| Albumin               | 4.8 gm/dL | USG abdomen        | Normal apart from grade 2 fatty liver |
| Globulin              | 2.7 gm/dL | ECG                | T-wave inversion leads V2, V3, V4, V5, and borderline QT interval |
| GGT                   | 56 U/L    | ECHO               | Concentric left ventricular hypertrophy with no regional wall motion abnormalities. The ejection fraction was 58%, and slight jerky motion of the septum was noted |
| ALP                   | 118 U/L   | Ophthalmoscopy     | Normal fundus |
| Urea                  | 27 gm/dL  |                     |          |
| Cr                    | 0.68 mg/dL|                     |          |
| Ca                    | 9.09 mg/dL|                     |          |
| Phosphorus            | 4.16 mg/dL|                     |          |
| Sodium                | 135 mmol/L|                     |          |
| Potassium             | 3.6 mmol/L|                     |          |
| Bicarbonate           | 25 mmol/L |                     |          |
| Chloride              | 95 mmol/L |                     |          |

Fig. 1. a–d Nonenhanced MRI brain showing the swollen right putamen, caudate, and internal capsule along with T2 and FLAIR hyperintensity and focal areas of restricted diffusion.
The patient was presently employed as an assistant and had to house sit for 7 cats; his duties included changing cat litter. The patient was a nonsmoker and consumed alcohol only in moderation. There was no recreational drug use, and the patient was not sexually active.

Past medical and surgical history was not significant, and the patient was in good health before the onset of his current complaints. A plain MRI brain revealed swollen right putamen, internal capsule, and caudate with T2 and FLAIR hyperintensity along with multiple small focal areas of restricted diffusion. These findings were suggestive of inflammatory etiology (Fig. 1).

He was advised admission but self-discharged from the emergency room. He represented 5 days later with increasing drowsiness along with multiple episodes of vomiting. His headache had been persistent during this period. He reported no new neurological symptoms apart from increasing drowsiness.

On examination, his blood pressure was 130/70 mm Hg, pulse was 75 beats per minute regular and good volume, the respiratory rate was 18 breaths per minute, and saturation was 98% on room air. He was drowsy but arousable and oriented in time, place, and person. His neck was supple, and cranial nerves were intact apart from a mild flattening of the left nasolabial fold. The power was 5/5 in all limbs tested, and plantars were downgoing. Sensations were grossly intact. The cardiovascular, respiratory, and gastrointestinal

Fig. 2. a–f Repeat MRI 5 days after the first presentation showing T2, FLAIR hyperintense, and T1 hypointense lesion in the right basal ganglia. Multiple 3–5-mm round to ovoid discrete and confluent foci within. There is diffuse vasogenic edema in the right fronto-temporo-parietal deep white matter with midline shift toward left along with mild uncal herniation and effaced left-side ambient cistern.
examinations were unremarkable. In view of his previous imaging, the differentials at this stage were primary neurological malignancy, infiltrative diseases such as sarcoidosis, and inflammatory disorders such as tuberculosis or toxoplasmosis.

A repeat MRI with contrast showed enlarged T2, FLAIR hyperintense, and T1 hypointense right basal ganglia. There were multiple small 3–5 mm round to ovoid discrete and confluent enhancing foci within along with significant mass effects, no diffusion restriction, or blooming. These features favored an inflammatory origin of the lesion; possibilities are toxoplasmosis or granulomatous as tuberculosis; differential of glioma was considered unlikely (Fig. 2). His investigations were as follows Table 1.

In view of his history, radiological imaging, and serology, he was diagnosed as a case of cerebral toxoplasmosis. His CSF examination was not performed. He was started on mannitol 150 mL Q 8 hourly, Septran DS 3 tab twice daily, furosemide, levetiracetam, and pantoprazole.

His clinical condition improved over the course of the next few days. His drowsiness improved, and his facial deviation got better. His repeat MRI 1 week later showed a reduction in edema and mass effect (Fig. 3).

The symptoms improved, and the patient was discharged from the hospital. He was reviewed 1 month later in the outpatient department. His symptoms had improved, and his repeat MRI brain showed a significant reduction in the lesion (Fig. 4).

Discussion

Toxoplasmosis is caused by the ubiquitous, intracellular protozoan T. gondii. This obligate intracellular apicomplexan infects nearly a third of the world’s population [1]. Transmission of this protozoan to humans is usually via the following means: consumption of food or water contaminated with infectious oocysts, eating undercooked meat containing tissue cysts, transplacental transmission from the infected mother to the fetus, and transmission through organ transplant from an infected donor [2]. Felines are the only animals in which T. gondii can complete its reproductive cycle [2].

The seroprevalence of this organism among individuals aged 6–49 years in the USA is around 11% [3]; this figure may be as high as 78% in some parts of Brazil [4]. Middle Eastern countries also demonstrate high seroprevalence, with rates approaching 30–50% [5, 6].

Immunocompetent individuals with primary infection are usually asymptomatic. However, acute toxoplasmosis with headache, myalgias, lymph node enlargement,
weight loss, and ocular involvement may also be seen in patients with intact host immune response [7].

Central nervous system involvement is usually related to the reactivation of latent infection in individuals with a suppressed immune system. Indeed, it is the most common cause of encephalitis and focal intracerebral lesions in people living with HIV/AIDS [8]. The usual manifestations of cerebral toxoplasmosis include headache, altered sensorium, pyrexia, focal deficit, seizures, cranial nerve palsies, behavioral abnormalities, or visual abnormalities [9–12].

The occurrence of cerebral toxoplasmosis in immunocompetent cases is exceedingly rare; moreover, the neuroradiological features are different when compared to immunocompromized hosts. The typical nodular or ring-enhancing lesions are usually absent, with linear radiating enhancement in the cortical and subcortical regions being prominent [13]. Three main patterns of CNS involvement have been reported; diffuse encephalopathy, meningoencephalitis, or solitary/multiple space-occupying lesions [14].

Fig. 4. a–f Small foci to T2/FLAIR hyperintensities in right gangliocapsular without mass effect or midline shift. The scan shows significant reduction in size of the lesion compared to previous scans.
Serologic diagnosis (enzyme-linked assay) for anti-Toxoplasma IgG and IgM. IgM antibodies are usually present in acute infection with IgG positivity showing previous infection or reactivation. PCR testing and histopathological analysis showing tachyzoites are confirmatory tests.

The patient in our case report presented with headache and space-occupying lesion on radiological investigations. We had initially considered the differential of neurological malignancy, tuberculosis, sarcoidosis, and toxoplasmosis. The absence of exposure to mycobacterium and normal chest radiograph were against tuberculosis as a possibility. No hilar lymphadenopathy and normal calcium levels reduced the possibility of sarcoidosis contributing to his presentation. His imaging was suggestive of inflammatory process rather than a neoplastic disorder. He had a strong history of exposure to several cats and positive serology which favored cerebral toxoplasmosis. One case report suggests that close contact with multiple cats increases the likelihood of CNS disease in immunocompetent individuals [15].

The treatment of choice is pyrimethamine plus sulfadiazine along with leucovorin. However, trimethoprim-sulfamethoxazole may be used as an alternative if pyrimethamine is not available.

In this case, we started our patient on a trimethoprim (5 mg/kg dosing) base regimen. His clinical and radiological improvement with treatment substantiated the diagnosis, and invasive testing was not performed.

Cerebral toxoplasmosis in immunocompetent individuals is rare; furthermore, presentation as a solitary space-occupying lesion can further confound the diagnosis. The strong history, characteristic basal ganglia involvement, and positive serology helped us reach our diagnosis.

Conclusion

This case report shows that cerebral manifestation of toxoplasmosis can occur in immunocompetent individuals. A detailed history and strong radiological and serological features made it possible to reach the diagnosis in our case.

Statement of Ethics

Written informed consent was obtained from the patient for publication of this case report and any accompanied images. Ethical approval was not required for this case report in accordance with the Dubai Scientific Research Ethics Committee policies.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Khawar Tariq Mehmood was involved in study concept, manuscript preparation, and writing the body text along with editing and proof reading. Amina Shahid was involved in writing the discussion and editing the manuscript. All the authors approved the final manuscript.

Data Availability Statement

All imaged and data analyzed during this case report are included in this article. Further inquiries can be directed toward the corresponding author.

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