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CASE REPORT: A RARE CASE OF TOXOPLASMA ENCEPHALITIS

Internal Medicine

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ABSTRACT

The rise in HIV cases is leading to a higher occurrence of neurological problems. These problems can result from either the HIV virus directly affecting the central nervous system or from other opportunistic infections. Although cerebral toxoplasmosis is a frequently seen opportunistic infection in HIV-positive individuals, it is seldom encountered before the HIV diagnosis is confirmed. We present a case where a change in mental status was caused by acute Toxoplasma gondii encephalitis.

KEYWORDS

CASE REPORT:

A 42-year-old man was brought to the emergency department due to a two-day progression of fever and altered mental state. He initially complained of a mild headache seven days before, which then evolved into increasing confusion, vomiting, and eventually a generalized tonic-clonic seizure. The patient's wife denied any significant past illnesses, substance abuse, or prior hospitalizations. However, she did mention one suspicious seizure episode about 11 months ago that had not been investigated.

Upon physical examination, the patient appeared lethargic and unwell. His vital signs at admission were as follows: body temperature of 38.5 ºC, blood pressure 130/80 mmHg, heart rate 102 beats/minute, respiratory rate 19/minute, and oxygen saturation of 96% on room air. A bedside serum glucose test showed a level of 128 mg/dL. During a focused neurological examination, the patient exhibited drowsiness and disorientation regarding time, place, and person, but no specific focal deficits were observed. His cranial nerves were functioning normally, and he had full mobility in all four limbs without limitations. Deep tendon reflexes were within the expected range, and there was no ataxia or neck stiffness. Pupils were of moderate size and reacted appropriately. Examination of the limbs did not reveal any skin rashes or injection marks. Examination of other body systems, including the heart, lungs, abdomen, and genitourinary system, did not reveal any abnormalities.

A CT scan of the brain showed hydrocephalus and a hypodense lesion in the midbrain. Initial laboratory tests showed a normal complete blood count, normal electrolyte levels, a negative urine drug screen, and a slightly elevated erythrocyte sedimentation rate (38 mm/hour). Analysis of cerebrospinal fluid (CSF) revealed clear fluid with a protein concentration of 1.28 g/L and a glucose concentration of 5.7 mmol/L. No bacterial growth was observed after 48 hours of incubation.

He was initiated on Phenytoin to prevent seizures and was given Metronidazole and Cefotaxime empirically for a potential brain abscess. However, there was no improvement in his fever and altered mental state. As a result, the medical team explored other potential causes of brain lesions with ring enhancement. They also assessed the patient's immune system and conducted a rapid HIV antibody test, which returned a positive result. Subsequent testing confirmed the presence of HIV infection, although the anti-toxoplasma IgG and IgM tests were negative.

Further evaluation included an MRI, which revealed a ring-enhancing lesion with edema in the left tectal plate of the midbrain. Despite this finding, following consultation with an infectious disease specialist, the patient was treated for cerebral toxoplasmosis using Pyrimethamine, Sulfadiazine, Folic acid, and Dexamethasone. After six days of anti-toxoplasmosis therapy, he showed significant improvement with a positive neurological response. He was discharged two weeks later with an oral anti-toxoplasmosis regimen and was referred to his primary care physician. Plans were made to initiate antiretroviral therapy on an outpatient basis.

Upon the patient's complete mental recovery, he disclosed a history of intravenous drug use, which he had discontinued two years prior. A subsequent MRI conducted three weeks later displayed a reduction in the size of the lesion and brain edema. Consequently, a repeat test for toxoplasma-specific IgG yielded a positive outcome.

DISCUSSION

Neurological manifestations associated with HIV can manifest at any point from the time of viral acquisition to the advanced stages of AIDS. These manifestations are diverse and have the potential to impact various parts of the nervous system, including the brain, spinal cord, autonomic nervous system, and peripheral nerves. In fact, HIV affects the nervous system in a significant majority, ranging from 70% to 80%, of individuals who are infected. This impact can be attributed to the direct influence of the virus itself, opportunistic infections, and/or the development of malignancies.

Among neurological disorders occurring in HIV/AIDS patients, Toxoplasma encephalitis is the most commonly observed. Brain imaging using CT scans typically reveals multiple, bilateral, hypodense lesions that enhance with contrast in the majority of patients with Toxoplasma encephalitis. However, it is worth noting that this condition may occasionally present with a single lesion or even no lesions on a CT scan. MRI, being more sensitive than CT scans, is the preferred imaging technique for detecting these lesions.

MRI studies have shown that approximately 70% of cerebral toxoplasmosis cases exhibit multiple lesions. Although relatively rare, around 14% to 17% of AIDS patients with this condition may have a single lesion. Notably, patients with toxoplasma encephalitis typically show rapid improvement once appropriate treatment is initiated. Neurological improvement can be observed in 51% of patients by the third day of treatment and in 91% of patients by the fourteenth day. Additionally, most patients experience radiological improvement within the third week of treatment. Therefore, it is advisable to conduct follow-up neuroradiologic assessments 2 to 4 weeks after the initiation of therapy.

CONCLUSION

Patients with Toxoplasma encephalitis may display only a single lesion or even no evident lesions on a CT scan.While a negative IgG test result makes the diagnosis highly unlikely, it does not completely rule it out. In AIDS patients, Toxoplasma-specific IgG antibody levels are often low to moderate, and in some cases, no specific IgG antibodies may be detectable. IgM antibody tests typically yield negative results. If a patient exhibits clinical symptoms consistent with toxoplasmosis, but the IgG antibody levels are low, a follow-up test conducted two to three weeks later should show an increase in antibody levels if the illness is indeed caused by acute toxoplasmosis, assuming the patient's immune system is not severely compromised. Toxoplasma gondii has the

potential to affect any part of the brain. The presentation of CNS toxoplasmosis with hydrocephalus is an exceptionally rare occurrence. Neurological disorders can be the initial indicators of AIDS in 7% to 20% of patients. Consequently, a clear history of AIDS may not always be evident. Therefore, clinicians should consider this possibility when evaluating patients with ring-enhancing cerebral lesions, even in cases where there is no prior history of immunosuppression or HIV infection. This proactive approach is crucial for achieving an early diagnosis and administering appropriate treatment.

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