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Neurocysticercosis presenting with visual and unilateral auditory hallucinations and behavioral change: a case report

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Abstract

Introduction

Neurocysticercosis (NCC) is the most common parasitic infection of the central nervous system (CNS)(1)(2). It is caused by larvae stages of Taenia Solium and is the major cause of acquired epilepsy worldwide(3). Seizure disorders are common neurological manifestation(4); however, neuropsychiatric manifestations have also been reported(5,6). Humans acquire infection through ingestion of eggs of Taenia Solium from contaminated food(7). The brain immunological response towards the cysts may be responsible for the pleomorphic manifestations(8), including psychiatric symptoms that may take years before the onset of seizures(6). As a result, diagnostic confusion is common, and this often affects management and outcome.

Case report

We report a case of 30-year-old black African male who presented with a history of auditory hallucinations and behavioral changes without focal neurologic signs or symptoms. The onset of generalized tonic-clonic seizures prompted him to seek medical attention although prodromal psychiatric symptoms started about a year before the onset of seizures. MRI of the brain revealed multiple cysts involving mainly the cerebral cortex.

Conclusion

This case highlights the need to exclude organic cortical pathology should in rare forms of psychiatric manifestations such unilateral auditory hallucinations and acute behavioral disturbances. Correct diagnosis and prompt management will prevent complications and usually have a favorable prognosis.

Keywords: hallucinations, psychiatric manifestation, neurocysticercosis

Key clinical message

Pleomorphic manifestations of NCC pose a challenge in diagnosis. We present a case of a 30-year old male who presented with prodromal psychiatric symptoms before the onset of generalized tonic-clonic seizures. This case highlights challenges in diagnosis though prompt management promises a favorable outcome.

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Case report

We present a case of a 30-year old black African medical doctor who came for care after an episode of generalized tonic-clonic convulsions which was observed by his wife while he was asleep. The episode started with a loud grunting noise, rolling of eyes sideways and was followed by generalized tonic-clonic convulsions which lasted for about two minutes and took him about five minutes to regain his consciousness. One month before the seizure episode, he had what he called “strange experience, which was characterized by visualization of "a very shiny transparent film accompanied by stars like moving objects and eventual loss of vision.” Then he later found himself lying on the ground approximately 10 meters away from his previous position. He felt somnolent with generalized body aches and also had nausea, profuse sweating, and a generalized headache. He recalled only the “strange experience” and then waking up, with no memory of what had occurred in between.

For about a year before the onset of generalized seizures, he had been experiencing intermittent episodes of hearing voices in the left ear, which he described as if someone was "playing a recorded tape of voices of people arguing in the marketplace.” The voices were mostly confined to one ear and were usually characterized by voices of people arguing or discussing although occasionally incomprehensible sounds were also heard. The voices were neither commenting on nor commanding his action and never had he experienced his thoughts to be spoken aloud. He acknowledged the voices as hallucinations. However, as they were transient, he did not attribute them to any serious medical condition.

The patient recalled an incident which happened four months prior to the first convulsive seizure, whereby he suddenly became aggressive, pacing around aimlessly and talking incoherently. This behavior prompted his wife to hide until he was calm enough to converse rationally a few minutes later.

Further interviewing revealed that the above symptoms were also accompanied by behavior changes about which his wife and close family members voiced their disapproval. The patient described engaging in more pleasurable activities such as overspending beyond his means, commonly spending nights out drinking and at night clubs and engaging in casual sex due to an increase in libido. He acknowledged that he was mostly in a “jovial mood,” more hyperactive and had clear direction about what he wanted to accomplish. As a result, he had a decreased need for sleep. Meanwhile attributed the “transient hallucinations” as being due to a lack of sleep.

He denied experiencing a depressed mood, feelings of hopelessness or guilt, thoughts of self-harm or suicidal ideation.

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Over the course of these seizure episodes and psychiatric disturbances, there was no accompanied history of fever, headache or progressive impairment of motor function.

Review of other systems was unremarkable.

Upon mental status examination, the patient was apprehensive about the seizures. Otherwise, no other remarkable findings were noticed.

Neurological examination was negative for signs of raised intracranial pressure or focal neurological deficits. All cranial nerves were intact; however, there was a presence of hyperreflexia with bilateral plantar flexor.

Audiogram was negative for hearing impairment.

Laboratory investigations including complete blood count and ESR, sugar, serum electrolytes, renal function, liver function, lipid profile and creatinine phosphokinase were within the normal limit. The full blood count revealed slight eosinophilia. Tuberculin test, P24+ markers for HIV-1 and 2 were all tested negative. In our setting, serological analysis such as Enzyme-Linked Immunosorbent Assay and Electroimmunotransfer Blot Assay were not available.

At this point, an impression of complex partial seizure with secondary generalization was made.

EEG and brain MRI scan were ordered.

He was prescribed Carbamazepine 200 mg orally twice daily with the target of reaching the daily dose of 800 mg within two weeks. However, the patient did not take the Carbamazepine. He returned two months later with brain MRI scans that showed multiple well defined focal lesions in the left parietal lobe, left occipital lobe, right temporal region, and right periventricular area. See figures 1 through 5 which are consistent with the diagnosis of neurocysticercosis.

At this point, Albendazole 400 mg twice daily for one month and intravenous Dexamethasone 8 mg daily for five days were added to 800 mg of carbamazepine. He was compliant with the Albendazole and Dexamethasone, but his compliance to Carbamazepine improved only after he experienced two other “abnormal experiences” both of which did not escalate into tonic-clonic convulsions. The first occurred on the 4th day of Albendazole and Dexamethasone treatment. The first episode was characterized by seeing flashes of light and figures in the air. The second episode occurred a few days later when he was about to start his car about which he noticed vision impairment, palpitations and a feeling of lightheadedness, but he managed to lie flat in his car seat, and the symptoms eventually resolved. He did not experience loss of consciousness nor any tonic-clonic convulsions.

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He stopped the Carbamazepine within six months, describing that he felt cured though he agreed to be watchful of early warning signs and symptoms. Apart from minor body twitches which are progressively improving, it has been more than a year since the last episode of tonic-clonic seizure. He no longer experiences hallucinations, and both the patient and his close family members have noted that his mood has remained relatively stable.

Discussion

Though seizure disorders are the most common presentation for NCC, early neuropsychiatric presentation as in our case may have posed challenges in diagnosis; however, the onset of sudden onset generalized tonic-clonic convulsions supported by radiological findings was consistent with the diagnosis of cerebral neurocysticercosis. Though MRI findings suggestive of NCC may be difficult to distinguish from other space-occupying lesions such as a tumor or CNS tuberculoma(9); absence of low-grade fever, Normal ESR, negative tuberculin test, the presence of eosinophilia, good response to Albendazole, Dexamethazone and Carbamazepine further excluded CNS tuberculoma but supported the diagnosis of neurocysticercosis.

The clinical history suggests a diagnosis of complex partial seizure with onset from the occipital lobe migrating to the temporal and parietal lobe and eventual involvement of all the brain hemispheres. The auras in the form of elementary visual hallucinations and pattern of visual loss all point to an involvement of the occipital cortex at the onset of seizures.

Few have reported unilateral auditory hallucination, but this phenomenon may pose a challenge to whether the problem is secondary to brain pathology or merely a manifestation of conductive hearing problems(10) as has been reported in elderly people with hearing impairment. However, our case was a young man without hearing impairment and the brain MRI showed multiple cortical cysts, including that of the right temporal lobe lesion something that may suggest this pattern of auditory hallucination which is contralateral to the side of the lesion to be of organic pathology involving the auditory pathway(11).

Mood symptoms are common in neurocysticercosis (NCC), often in the form of depression(12) although cases of mania are not uncommon(6). The pathogenic process leading up to the development of mania or hypomania symptoms (as in this case) is not clear. Although current evidence points for changes in neurotransmission level, this needs further scientific probing(6).

Good response for neuropsychiatric symptoms and seizures on carbamazepine, albendazole, and dexamethasone is consistent with the American Academy of Neurology's recommendation for

the treatment of NCC(13). Common practice for the management of psychotic symptoms in NCC includes the use of antipsychotics. The remission of psychotic symptoms without the use of antipsychotics may question the role of antipsychotics in the management of epileptic psychosis. However, the transient nature of psychotic symptoms, in this case, may suggest a favorable outcome for these symptoms without antipsychotics provided that the primary organic pathology is well managed.

Conclusion

This case highlights that NCC is a treatable disease with a good response to pharmacologic treatment provided that early diagnosis prompt management are appropriately done. Clinicians should have a high index of suspicion for organic brain pathology in adult onset epileptic seizures with or without prodromal neuropsychiatric symptoms. With such patients, NCC should be considered as a possible etiology, regardless of whether the patient resides in an NCC endemic area.

Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Conflict of interest

The authors have no conflict of interest to declare

Authors' contribution

AAN managed the patient; AAN and HR did the literature search and wrote the manuscript. All authors have read and approved the final manuscript.

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Images

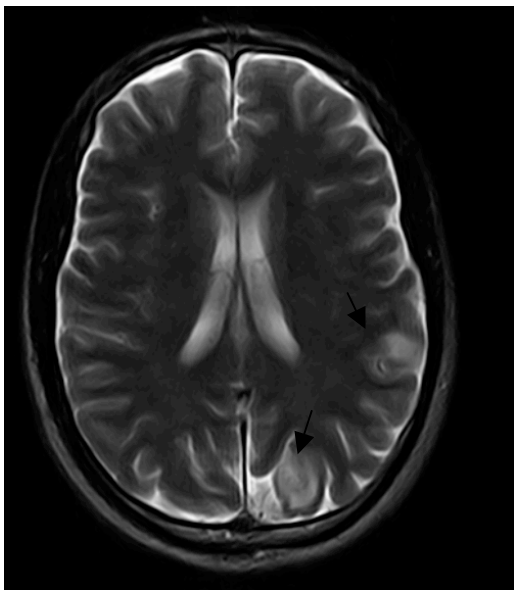
Figure 1 

Figure 1. Axial T2W image showing hyperintense lesions on the left parietal and left occipital lobes

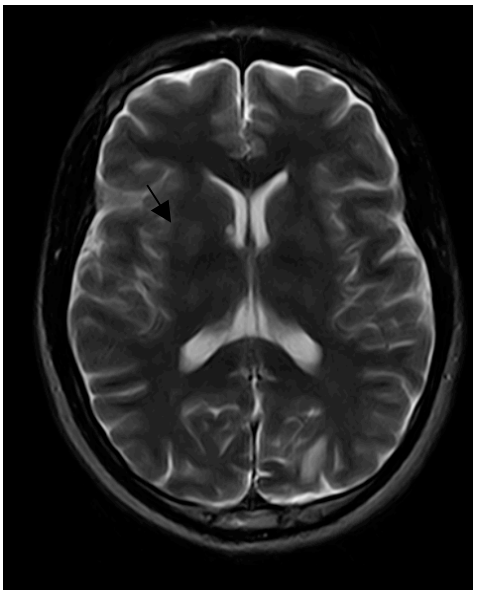
Figure 2 

Figure 2. Axial T2W image showing hyperintense lesion on the right periventricular area.

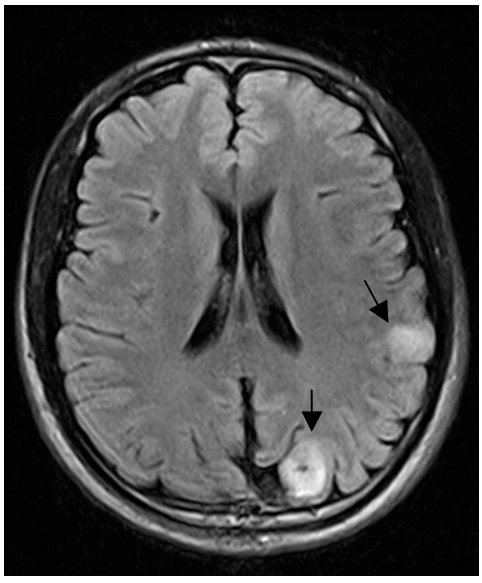
Figure 3 

Figure 3. Axial FLAIR image showing reasonable perilesional edema surrounding lesions on the left parietal and left occipital lobes.

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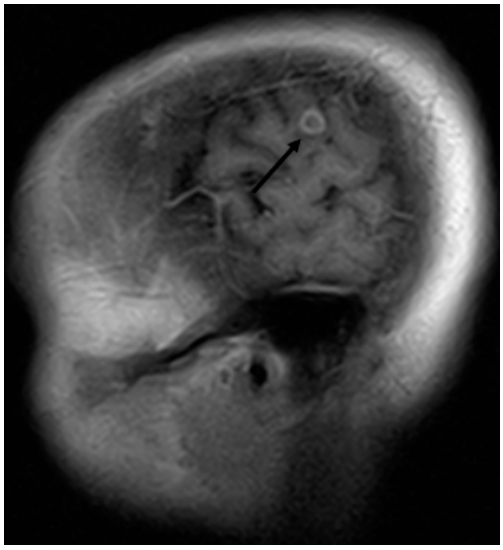
Figure 4 

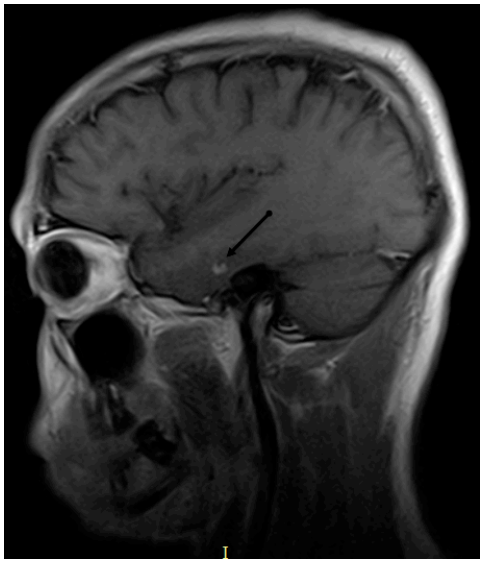
Figure 5 

Figure 4 and Figure 5. Sagittal, Post-contrast T1W images showing ring enhancing lesions on the right parietal and right temporal lobes.