A Case Report of Intraparenchymal Neurocysticercosis in a Postpartum Female in Saudi Arabia

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Abstract

Neurocysticercosis is a neurologic disease caused by infection with larvae of Taenia solium. It is most commonly transmitted by consumption of pork, water contaminated with T. solium or poor hygiene habits. As only few cases of neurocysticercosis have been documented in Saudi Arabia, the authors report a case of neurocysticercosis in a young Indian female residing in Saudi Arabia who presented with generalized tonic–clonic seizures 6 days after a normal vaginal delivery. Her physical and laboratory investigations as well as chest X-ray and electroencephalogram were all normal. Computed tomography of the head revealed multiple calcified nodular lesions, and magnetic resonance imaging showed ring-enhancing lesion in the left frontoparietal area. Serum enzyme-linked immunosorbent assay (qualitative) was positive for immunoglobulin G antibodies for cysticercosis. A diagnosis of neurocysticercosis was made, and the patient was treated with dexamethasone and levetiracetam for 4 days before discharge. At the 3-month follow-up, the patient’s condition had significantly improved, and her seizures had not recurred. This report recommends considering neurocysticercosis as a differential diagnosis in patients presenting with new-onset seizures, even if the symptoms do not initially indicate neurocysticercosis or if the patient resides in an area where the disease is rare.

Keywords: Generalized tonic–clonic seizure, neurocysticercosis, neuroimaging, Taenia solium

INTRODUCTION

Neurocysticercosis (NCC) is caused by infection with the larvae of Taenia solium and is the leading preventable cause of adult-onset seizure worldwide. The most common route of transmission is by ingestion of T. solium eggs through the fecal–oral route. Common causes of this infection include consuming undercooked food, especially pork, water contaminated with T. solium or uncooked vegetables as well as poor hygiene habits. Notably, there is significant variability among the stages of cyst development in NCC patients who are asymptomatic. Calcified NCC lesions, which usually develop in the final stages of the disease, are more likely to be associated with seizures than any other stage of the larvae. Although the mechanism by which seizures are induced in NCC is poorly understood, it has been speculated that these seizures are related to the inflammatory response involved during cyst degeneration.
NCC is considered a public health concern worldwide and is not confined to endemic areas such as India, Latin America, South East Asia, China and Nepal.\[^1\]\[^2\]\[^3\] Immigration from endemic to nonendemic areas and tourism are likely reasons for such transmissions.\[^1\]\[^2\]\[^3\] In the Arabian Peninsula, NCC is rare; the disease is mostly transmitted through household exposure to individuals from disease-endemic areas.\[^5\]\[^6\]

The authors, herein, report a case of NCC from Saudi Arabia in a young Indian female who presented with generalized tonic–clonic seizures 6 days after normal vaginal delivery. This case argues that NCC should be considered as a differential diagnosis in patients presenting with seizures, even if the patient does not have initial symptoms indicative of NCC and resides in an area where the condition is rarely reported, and especially if the patient is from a disease-endemic country.

**CASE REPORT**

A 36-year-old Indian female who had immigrated to Saudi Arabia 3 years prior was brought to the emergency department after a 3-min episode of generalized tonic–clonic seizure that occurred during sleep 6 days after the patient had a normal vaginal delivery at 37 weeks of gestation. In the postictal state, she was drowsy for about 15 min and did not complain of headache, fever, dizziness, vertigo, visual disturbance, nausea or vomiting. The patient had no history of chronic cough, chronic diarrhea, weight loss or decreased appetite and had no history suggestive of diabetes, hypertension or tuberculosis. The patient's most recent trip was to India 1 year prior. She had a history of epilepsy when she had two episodes of generalized tonic–clonic seizure at the age of 8 years and was treated with carbamazepine for 2 years, after which medication was stopped because there were no seizure episodes.

On physical examination, the patient was alert, conscious and cooperative, with a Glasgow coma scale of 15/15. Her cardiovascular, respiratory, musculoskeletal and gastrointestinal systems were functioning normally. Neurological and fundoscopic examinations were normal, with no signs of meningeal irritation.

The complete blood count, erythrocyte sedimentation rate, electrolytes and blood sugar levels were within the reference range. The results of a purified protein derivative skin test (Mantoux test) were negative. Chest X-ray and electroencephalogram were normal. The patient refused to receive a lumbar puncture. Stool examination was normal.

Computed tomography (CT) scan of the head revealed multiple calcified nodular lesions [Figure 1a and b]. T1-weighted magnetic resonance imaging with contrast showed a ring-enhancing lesion in the left frontoparietal area, which informed suspicions of NCC [Figure 2]. Because the suspected NCC presented during the patient's postpartum period, venous sinuses thrombosis was considered as a possible diagnosis; however, absence of headache or blurred vision ruled this out. While a recurrence of the patient's epilepsy, triggered by the stress of her delivery, was also suspected, the serum (qualitative) enzyme-linked immunosorbent assay (ELISA) results having returned positive for immunoglobulin G (IgG) antibodies for cysticercosis – in addition to the CT findings – excluded this diagnosis, and thus, a diagnosis of intraparenchymal NCC was confirmed.

The patient was administered dexamethasone (4.5 mg/kg intravenously every 6 h) for 4 days to alleviate the intraparenchymal inflammation and perilesional edema in addition to levetiracetam 500 mg orally twice daily. This treatment resulted in successful control of her seizures during the hospitalization period. Ophthalmology consultation confirmed the absence of any orbital involvement. At the 3-month follow-up, the patient's condition had significantly improved, and no seizure recurrence was observed.

**DISCUSSION**

The religious dietary laws in Saudi Arabia help in markedly reducing the risk of NCC. However, based on this case report and similar findings, the authors suggest that NCC should be considered as a differential diagnosis in patients presenting with seizures, particularly if they are from an NCC-endemic area, have had household exposure to individuals from endemic areas...
or traveled to endemic areas. Most NCC cases are asymptomatic; however, clinical presentations can vary drastically based on the number, site and size of calcified cysts. The immunological status of the patient also plays a role in the clinical manifestation of NCC. Seizures are considered the most common presentation, followed by severe and protracted headaches.

Seizure occurrence is most significantly associated with the presence of parenchymal cysts. Other clinical manifestations of NCC include focal neurological deficits, decline in cognitive functions and intracranial hypertension. The parenchymal form of NCC has a favorable prognosis, with 60%–90% of the cases spontaneously resolving, irrespective of intervention.

Both generalized and partial seizures with secondary generalization are the most common form of seizures. The proposed mechanism by which parenchymal NCC induces seizures is through degeneration of the cyst, which attracts the host immune response. The degradation leads larva to undergo hyaline degradation and eventually results into granular phase and then the calcification stage. Furthermore, the calcification of cysts, producing perilesional edema, may exert a strong effect over prolonged periods of infestation.

Neuroimaging helps distinguish the various types of NCC and to rule out or rule in other differential diagnosis such as tuberculosis and the less likely central nervous system toxoplasmosis, abscess, metastasis and glioblastoma. In our case, because NCC presented during the postpartum period, the other possible differential diagnosis was venous sinuses thrombosis. However, the patient did not have any history of headache and blurred vision, and thus, the diagnosis of venous sinuses thrombosis was excluded. The patient had a history of epilepsy during her childhood, and the current episode could have been a recurrence, particularly likely triggered by the stress of her delivery and subsequent sleep deprivation. However, recurrence was ruled out when her serum ELISA results were found to be positive for IgG antibodies for cysticercosis in addition to findings of calcification with perilesional edema on CT, thereby confirming parenchymal NCC. Based on the revised diagnostic criteria of NCC, our patient reached a definitive diagnosis by achieving two major neuroimaging criteria and one clinical criteria.

Using monoclonal antibody-based ELISA in detecting cysticercal antigen in serum or cerebrospinal fluid has demonstrated remarkable utility for the detection of viable parasites, particularly when following up the patient. However, its sensitivity has not been studied yet.

Tissue biopsy is not routinely performed in clinical practice, but it provides one of the elements of the revised absolute criteria: if there is histological evidence of the parasite, this will confirm the diagnosis. Identifying the type of NCC is essential for the initiation of optimal treatment and a good prognosis. It should be noted that intraventricular NCC cysts have a tendency to cause obstructive hydrocephalus as a result of chronic arachnoiditis or manifest as meningitis. In terms of treatment, the conservative management of intraparenchymal NCC is often sufficient; by contrast, intraventricular (extraparenchymal) NCC may require neurosurgical endoscopy. The treatment of active parenchymal NCC includes a combination of an anthelmintic such as albendazole and anticonvulsants. Baird et al. recommend cysticidal therapy for patients with active intraparenchymal NCC, but the role of corticosteroids as adjuvants remains debatable. As our patient had a perilesional edema around calcified nodular lesions, we treated her with antiepileptics and a short course of steroids. Because our patient had a calcified larva, she was deemed unlikely to benefit from cysticidal agents; cysticidal therapy is most effective for viable cysts. Further, given that most patients with degenerating cysts resolve spontaneously and symptomatic control often suffices, the use of cysticidal drugs in such patients would likely not provide any additional benefits.

**CONCLUSION**

NCC is rarely reported in Saudi Arabia. However, the present case report suggests that after excluding eclampsia, infectious, metabolic and structural diseases, NCC should
be considered as a differential diagnosis in patients presenting with seizures, especially if they are from a disease-endemic area.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that her name and initial would not be published and due efforts will be made to conceal her identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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