

CASE REPORT

Neurocysticercosis in pregnancy: maternal and fetal outcomes

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Abstract

Neurocysticercosis (NCC) is a parasitic infection with the larvae of *Taenia solium* from contaminated pork. It is a leading cause of seizures in the developing world. Symptoms may be secondary to live or degenerating cysts, or previous infection causing calcification or gliosis. Diagnosis is based on clinical presentation, radiological confirmation of intracranial lesions and immunological testing. Management involves symptom control with antiepileptics and antiparasitic agents. Few cases have been described of maternal NCC during pregnancy. We describe a 25-year-old female presenting to a London hospital with secondary generalized seizures. MRI of the brain confirmed a calcified lesion in the right parietal lobe, and she gave a corroborative history of NCC during her childhood in India. She was stabilized initially on antiepileptics, but during her pregnancy presented with breakthrough seizures and radiological evidence of NCC reactivation. She was managed symptomatically with antiepileptics and completed the pregnancy to term with no fetal complications.

INTRODUCTION

Neurocysticercosis (NCC) is the major cause of adult-onset epilepsy in the developing world and is caused by infection of the central nervous system (CNS) by the larvae of *Taenia solium*. It is rare in the UK; however, it is endemic in developing regions, including South America, sub-Saharan Africa and areas of Asia, specifically India, China and regions of South-East Asia. The prevalence rate of NCC in the UK remains low and cases can largely be attributed to immigration from endemic areas rather than local transmission [1].

Cysticercosis develops when humans become intermediate hosts through ingesting embryonated eggs of tapeworm which release oncospheres entering the bloodstream, developing cystercerci in tissues. *Taenia solium* larvae may remain dormant in the CNS for several years before degenerating into granulomas and developing calcified lesions. At this stage, the infection is no longer considered active [2]. However, transient perilesional

edema around calcified foci is common and associated with episodic seizure activity in patients with calcified NCC [3].

The clinical manifestations of NCC are variable and depend on the number, size and location of cysts and the immune response of the host. The most common site affected is the brain parenchyma and these can precipitate seizures. Calcified cysts and their associated perilesional edema cause higher seizure recurrence rates than simple larvae cysts. There is generally a good response to antiepileptic drugs. Prognosis is worse in patients with a higher burden of calcified cysts, which can cause recurrent and potentially refractory seizures [1]. Other clinical presentations include focal neurological deficits, general cognitive decline and intracranial hypertension.

Diagnosis is based on clinical presentation, cranial imaging (CT or MRI) and immunological testing. The enzyme-linked immunoelectrotransfer blot (EITB) assay detects serum antibodies to *T. solium*. Its sensitivity is as high as 98% in cases with more

[†] The authors wish it to be known that, in their opinion, the first two authors should be regarded as joint first authors.

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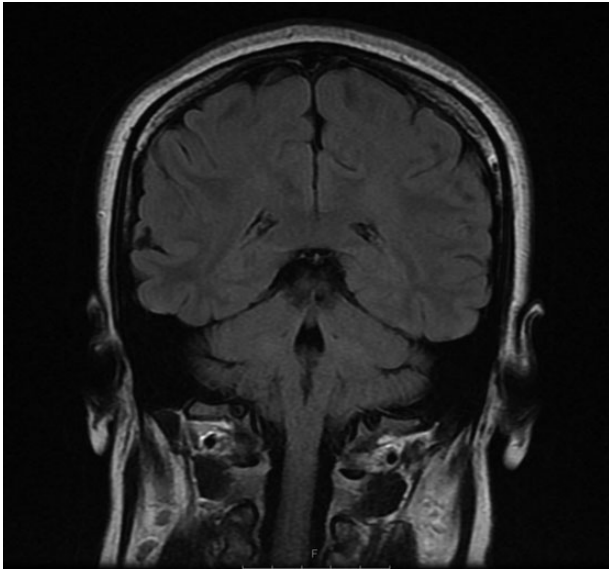


Figure 1: MRI T₂ FLAIR sequence (coronal section) shows a small focus of low signal intensity in the cortex of the right posterior parietal lobe measuring 5.2 mm in diameter. MR imaging was performed at 1.5 T [Repetition time (TR)/echo time (TE): 8002/126].

than one parenchymal cyst or subarachnoid disease; however, it falls to 50–60% in cases with only one cyst and is also poor in cases with calcified cysts. If EITB is unavailable, cerebrospinal fluid can be tested using ELISA to detect anticysticercal antibodies [1].

Management is primarily directed at symptom control with first-line antiepileptic therapy. Antiparasitic agents, albendazole or praziquantel, should be initiated only after seizures have been controlled since the acute inflammation caused by degenerating cysts can exacerbate symptoms [4]. Neurosurgical intervention may be required in cases of obstructive hydrocephalus, ventricular cysticercosis and drug-refractory seizures.

The management of active NCC in pregnancy can be challenging. Treatment with antihelminthic therapies can precipitate or increase inflammation and cerebral edema and thus, is deferred until the seizure is controlled [5]. During pregnancy, treatment with praziquantel is the preferred choice over albendazole, as the latter is felt to have a worse teratogenic profile. Few cases of NCC in pregnancy have been reported [6].

CASE REPORT

A 25-year-old female patient of South Asian origin presented to the neurology department with two self-terminating, secondary generalized tonic-clonic seizures. She had a history of NCC in childhood, diagnosed aged 8 while she was living in India. She received antiepileptics (stopped after 3 years without medical advice) and antiparasitic agents at the time. She remained seizure-free for 10 years and had an uneventful first pregnancy, but re-presented with seizures 3 months postpartum. There was no significant travel history in the last 5 years. She underwent an MRI brain scan, which demonstrated a 5.2-mm focus of calcification in the cortex of the right posterior parietal lobe with no enhancement, consistent with previous NCC (Fig. 1). At this time, the patient was started on lamotrigine and remained asymptomatic on lamotrigine 50 mg BD.

The patient remained stable on lamotrigine until 21 weeks gestation in her second pregnancy, when she presented to the

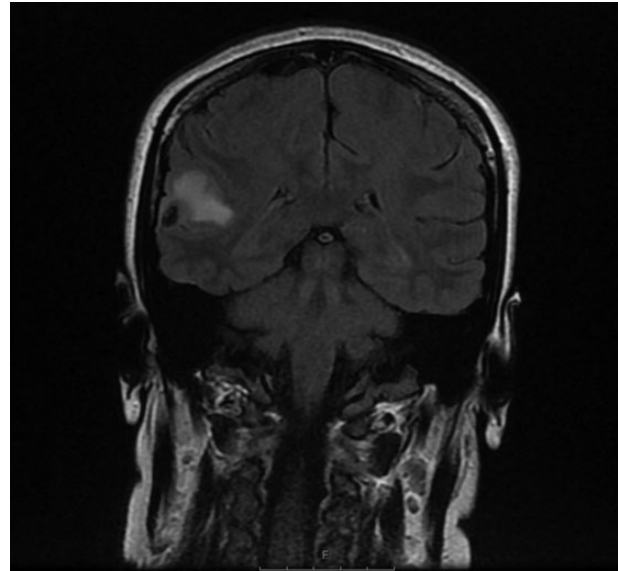


Figure 2: MRI T₂ FLAIR sequence (coronal section) performed during pregnancy, showing the small focus of calcification in the right parietal lobe with moderate surrounding edema. MR imaging performed at 1.5 T (TR/TE: 8000/130).

neurology clinic with a 2-day history of headaches, vomiting, change in behavior and a generalized tonic-clonic seizure. She was normotensive on admission and a repeat MRI brain performed demonstrated moderate edema surrounding the previously known calcified lesion in the right parietal lobe (Fig. 2).

The dose of lamotrigine was increased to 100 mg BD and levetiracetam 250 mg BD was introduced to control the seizure activity. No antihelminths were initiated at this point. The patient’s headache resolved as an inpatient and she experienced no further seizures. An MRI brain 6 weeks post-discharge confirmed resolution of the edema surrounding the calcified cyst and her *Taenia solium* serology came back negative. Labor was induced early for obstetric indications and she delivered a healthy male infant.

DISCUSSION

We describe a case of NCC reactivation during pregnancy, where symptoms were successfully controlled with antiepileptic agents. Cyst eradication therapy was avoided due to potential worsening of symptoms which would pose a threat to both mother and fetus. Guidelines released in 2002 by Garcia *et al.* [7] suggested that patients with exclusively calcified lesions should not be administered antiparasitic treatment as such lesions represent dead cysticerci. At the time of publication, no controlled data existed regarding the role of anti-inflammatory medication in such patients.

Calcified *T. solium* cysts are the most common radiological finding in NCC and present in 10–20% of the endemic population [8]. These calcified cysts are commonly foci of seizure activity. A prospective study in Peru showed that 50% of patients with calcified cysts and a positive cysticercosis serology presenting with recurring seizures showed perilesional edema [3]. This suggests the potential use of immunosuppressive or anti-inflammatory medications, alongside antiepileptic drugs in treating recurring episodes in these patients.

The pathophysiology of perilesional edema remains debated. One hypothesis is that intermittent release or recognition of

parasite antigen by the host or periodic loss of immune suppression results in an inflammatory reaction [9]. Another proposition is that selective calcified lesions have a propensity to cause perilesional edema through disruption of the blood–brain barrier [10, 11].

Due to the paucity of reported cases, it is uncertain if pregnancy is associated with an increased risk of reactivation of NCC. During pregnancy, it is possible that the shift toward a type 2 immune response and manipulation of host hormones by helminthes increase parasite density and survival [12]. However, more studies are required to look into this further as the literature on NCC reactivation in pregnancy is sparse and poorly understood.

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CONFLICT OF INTEREST STATEMENT

None declared.

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ETHICAL APPROVAL

None required.

CONSENT

Written informed consent was obtained from the patient for publication of this case report and the accompanying images.

GUARANTOR

P.D. is the guarantor of this study.

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