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Case Report

Post partum seizures without hypertension: a case report of neurocysticercosis mimicking eclampsia

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ABSTRACT

Postpartum seizures are most commonly attributed to eclampsia; however, alternative etiologies must be considered when classical features such as hypertension and proteinuria are absent. Neurocysticercosis (NCC) is the leading cause of acquired epilepsy in endemic regions, including India, accounting for nearly 30% of epilepsy cases in such populations. We report a case of a 23-year-old primigravida at 33 weeks and 6 days of gestation who underwent emergency lower segment cesarean section for placenta previa with active vaginal bleeding. The postoperative period was uneventful initially. On postoperative day 3, she developed a focal seizure without hypertension or proteinuria, for which magnesium sulfate was empirically initiated for suspected atypical eclampsia. A recurrent focal seizure with preserved awareness occurred on postoperative day 5, prompting neurology consultation. Magnetic resonance imaging of the brain revealed multiple ring-enhancing lesions consistent with NCC. The patient was managed with antiepileptic drugs, anti-parasitic therapy, and corticosteroids, with clinical improvement and no further seizure episodes during hospital stay. This case underscores the importance of considering NCC in the differential diagnosis of postpartum seizures in endemic regions, particularly when classical eclamptic features are absent, and highlights the indispensable role of early neuroimaging in guiding management.

Keywords: Neurocysticercosis, Postpartum seizures, Eclampsia, Placenta previa, Ring-enhancing lesions, Antiepileptic therapy

INTRODUCTION

Seizures arising in the postpartum period represent a clinical emergency requiring prompt diagnosis and management. Eclampsia-defined as new-onset tonic-clonic seizures in a woman with preeclampsia-remains the most frequently implicated etiology and is classically associated with hypertension and/or proteinuria occurring at or beyond 20 weeks of gestation or in the postpartum period. Despite its prevalence, clinicians must recognize that eclampsia is not sole cause of peripartum convulsions. Approximately 10-15% of peripartum seizures may be attributed to non-eclamptic etiologies, including cerebrovascular accidents, metabolic disturbances, intracranial space-occupying lesions, and parasitic infections of the central nervous system; this proportion is likely underestimated in regions where neurological etiologies are endemic.[1,2]

NCC, caused by intracerebral infestation with the larval stage (cysticerci) of the pork tapeworm Taenia solium, is the single most prevalent cause of acquired epilepsy worldwide and a significant public health burden in low- and middle-income countries.[3] It is disproportionately prevalent in developing nations, particularly in the Indian subcontinent, Sub-Saharan Africa, and Latin America, where it is estimated to account for 30% of epilepsy cases in endemic regions and up to 50% of new-onset seizures in certain populations.[4] The organism reaches the central nervous system haematogenously following ingestion of T. solium eggs shed in the faeces of a tapeworm carrier, most commonly through consumption of contaminated

food or water. Poor sanitation, open defecation practices, and close contact with pigs are the principal risk factors for transmission in endemic communities.[3,5]

The diagnosis of NCC in postpartum period is particularly challenging due to its clinical overlap with eclampsia and complex immunological alterations associated with pregnancy and its resolution. The Th2-dominant immune milieu of pregnancy is thought to suppress the host inflammatory response against cysticerci, potentially allowing silent lesions to persist asymptomatically throughout gestation; postpartum immune reconstitution may then precipitate inflammatory degeneration of cysts, resulting in new-onset seizures in women who were previously undiagnosed carriers.[6] Neuroimaging-particularly MRI of brain-is central to definitive diagnosis, with the modified Del Brutto diagnostic criteria providing a widely accepted framework for classification of the NCC.[3]

We present a case of postpartum seizures initially presumed to represent atypical eclampsia in a primigravida who had undergone emergency cesarean section for placenta previa, in whom MRI subsequently revealed the diagnosis of NCC. This case highlights the importance of broadening the differential diagnosis of postpartum seizures and the indispensable role of early neuroimaging in guiding management in endemic regions.

CASE REPORT

A 23-year-old primigravida at 33 weeks and 6 days of gestation presented to our institution with complaints of active vaginal bleeding. She was a known case of placenta previa diagnosed on routine antenatal ultrasonography. In view of significant maternal haemorrhage and imminent fetal compromise, an emergency lower segment cesarean section (LSCS) was performed under the regional anaesthesia.

The intraoperative course was unremarkable. A live preterm male neonate was delivered with Apgar scores of 7 and 9 at 1 and 5 minutes respectively. Uterine haemostasis was achieved satisfactorily. The immediate postoperative period was uneventful, with the patient haemodynamically stable and afebrile.

On postoperative day (POD) 3, the patient developed a focal seizure lasting approximately 60 seconds, characterized by clonic movements of the right upper limb with secondary generalization. There was no associated loss of awareness during the ictal episode. Vital sign assessment at the time of the event revealed a blood pressure of 110/70 mmHg; urine dipstick was negative for protein. Given the postpartum clinical context and the possibility of atypical eclampsia, intravenous magnesium sulfate was commenced as per institutional protocol.

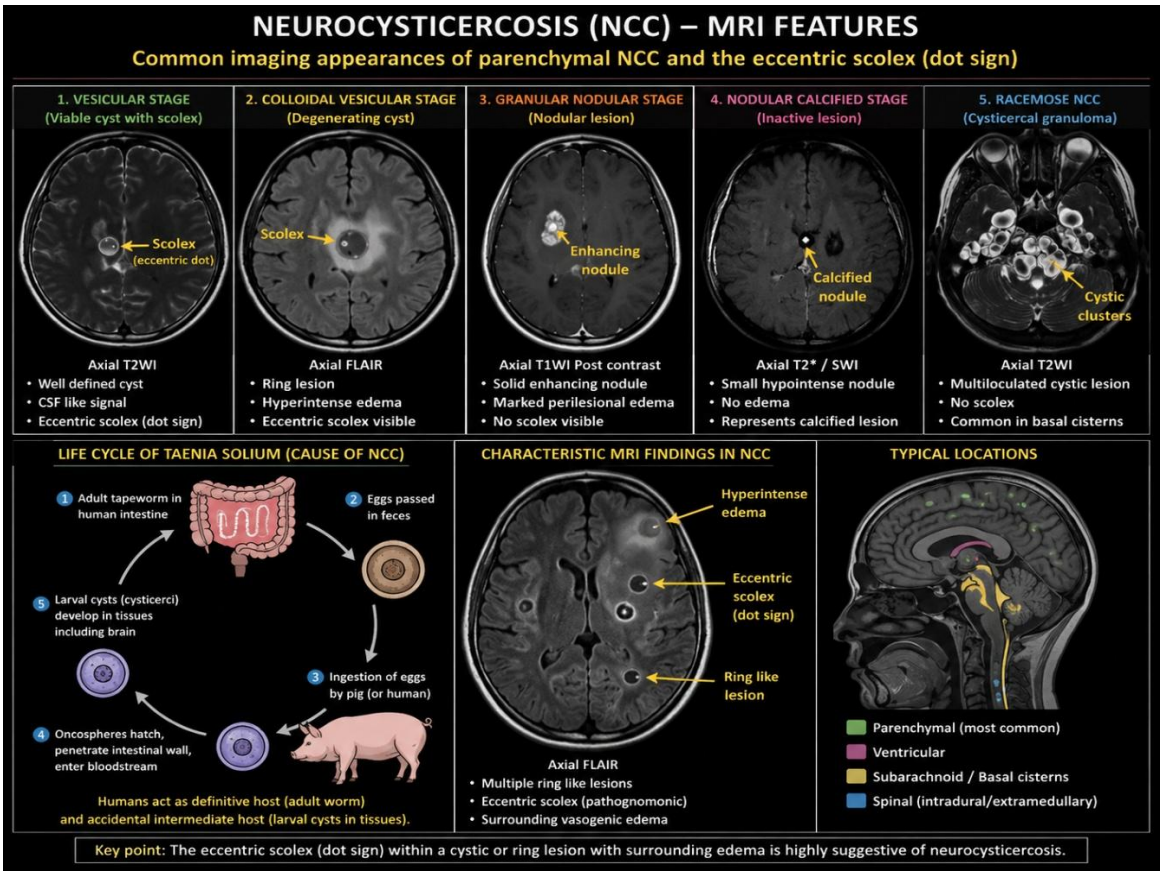

Figure 1: MRI characteristics and life cycle representation of parenchymal NCC showing vesicular, colloidal vesicular, and granular nodular stages with characteristic ring-enhancing lesions and eccentric scolex.

On POD 5, the patient experienced a second focal seizure, again with preserved awareness and right-sided motor features, despite ongoing magnesium sulfate therapy. The atypical semiology-focal onset, preserved awareness, and absence of hypertension or proteinuria-raised concern for a non-eclamptic aetiology. A neurology consultation was obtained, and MRI of the brain was performed urgently.

MRI of the brain demonstrated multiple ring-enhancing lesions with surrounding oedema, distributed in the parenchyma bilaterally, most prominent in the right parietal and frontal lobes. The lesions were consistent with the colloidal vesicular stage of NCC. No hydrocephalus or midline shift was identified. The MRI findings were reviewed in conjunction with the clinical history, and a diagnosis of active parenchymal NCC was established.

The patient was commenced on oral albendazole 400 mg twice daily for 28 days as anti-parasitic therapy, levetiracetam 500 mg twice daily as an antiepileptic drug, and oral dexamethasone to attenuate the inflammatory response associated with cyst degeneration. Magnesium sulfate was subsequently tapered and discontinued. The patient demonstrated progressive clinical improvement, with no further seizure episodes recorded during her hospital stay. Neonatal care was managed concurrently by the paediatric team. The patient was discharged on POD 14 with outpatient neurology follow-up arranged.

DISCUSSION

This case illustrates a diagnostically challenging scenario in which postpartum seizures, the most common and well-recognised cause of which is eclampsia, were in fact attributable to an entirely different pathology-NCC. The clinical overlap between these two conditions, particularly in the early postpartum period, demands that clinicians maintain a broad differential diagnosis and respond promptly to atypical or refractory presentations.

Eclampsia is classically characterised by generalised tonic-clonic seizures in the setting of preeclampsia (new-onset hypertension with or without proteinuria at ≥20 weeks of gestation or in the postpartum period). In the present case, the absence of hypertension, proteinuria, and other features of the systemic preeclamptic syndrome should have prompted earlier consideration of an alternative diagnosis. Importantly, in resource-constrained settings, the empirical initiation of magnesium sulfate for atypical postpartum seizures is not unreasonable as an initial measure; however, non-response to therapy-as observed here-should trigger urgent neuroimaging without delay.[7,8]

NCC is caused by the larval stage of Taenia solium, which reaches the central nervous system through haematogenous dissemination following ingestion of T. solium eggs from contaminated food or water. The clinical presentation of NCC is highly variable and depends on the number, location, size, and developmental stage of the cysticerci, as well as the intensity of the host inflammatory response.[3] Seizures are the most common presentation, occurring in 70-90% of symptomatic cases.[4] The MRI appearance of ring-enhancing lesions with perilesional oedema-as seen in our patient-is characteristic of the colloidal vesicular or early granular nodular stage of cyst degeneration, at which point the host inflammatory response is most intense and seizure threshold is lowest.[3,4]

The relationship between pregnancy and NCC is of particular interest. The immunological changes of pregnancy-characterised by a shift toward a Th2-dominant, relatively immune-tolerant state-may paradoxically result in accelerated degeneration of cysticerci in the postpartum period, when immune reconstitution occurs. This inflammatory reactivation may unmask previously silent cysticercal infection and precipitate new-onset seizures.[3,6] The 23-year-old primigravida in this case may well have harboured asymptomatic cysticercal cysts during gestation, with postpartum immune reconstitution triggering the inflammatory cascade responsible for her seizures.

Management of NCC-associated seizures in the postpartum period encompasses three parallel therapeutic axes: antiepileptic drugs to suppress seizure activity; anti-parasitic therapy (albendazole or praziquantel) to reduce the cyst burden; and corticosteroids to mitigate the inflammatory response precipitated by cyst destruction.[3,7,8] The concurrent breastfeeding status of the patient must be considered when selecting agents, given the potential for drug transfer in breast milk; however, given the clinical urgency in this case, standard therapeutic protocols were implemented with appropriate neonatal monitoring.

This case adds to the existing body of literature documenting NCC as a significant but underdiagnosed cause of postpartum seizures in endemic regions.[9,10] Clinicians practising in areas where T. solium is prevalent should maintain a high index of suspicion for NCC in any postpartum woman with seizures that are focal in onset, associated with preserved awareness, refractory to magnesium sulfate, or occurring in the absence of hypertension and proteinuria. Early neuroimaging is the cornerstone of diagnosis and should not be delayed by diagnostic uncertainty.

CONCLUSION

Not all postpartum seizures are attributable to eclampsia. NCC is an important differential diagnosis, particularly in endemic regions such as India, and should be actively considered when classical preeclamptic features are absent or when seizures are focal and refractory to magnesium sulfate therapy. Early neuroimaging- preferably MRI-is essential for timely and accurate diagnosis. A multidisciplinary approach integrating obstetrics, neurology, and neonatology is required for optimal outcomes in both mother and neonate.

The key take way point from this case is that: "Early consideration of NCC in atypical postpartum seizures may prevent delay in diagnosis and inappropriate prolonged treatment for presumed eclampsia."

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