Case Report

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Neurocysticercosis presenting as postpartum psychosis: a case report on challenging clinical insight with review of the literature

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Introduction: Postpartum psychosis (PPP) is a psychiatric emergency with symptoms such as mania, depression, hallucinations, and delusions appearing shortly after childbirth. Neurocysticercosis (NCC), a CNS infection from *Taenia solium*, can also manifest psychiatric symptoms, complicating diagnosis. This report discusses a rare case of NCC presenting as PPP, emphasizing diagnostic complexities and the need for a multidisciplinary approach.

Case presentation: A 23-year-old female presented with psychotic symptoms 15 days postpartum, initially treated for PPP with lorazepam, olanzapine, and haloperidol. Following the development of generalized seizures, neuroimaging revealed NCC lesions. Treatment with albendazole and steroids led to symptom resolution.

Discussion: This case highlights NCC's rare presentation as PPP. A thorough evaluation is crucial, as PPP poses risks to both mother and infant. In regions where NCC is endemic, psychiatric presentations due to this infection create diagnostic challenges, especially in limited-resource settings. Neuroimaging is vital for precise diagnosis, and early intervention with a multidisciplinary team enhances outcomes.

Conclusion: The case underscores the complexity of PPP diagnosis and management when coexisting with NCC. Clinicians in endemic regions should consider NCC in postpartum women with atypical psychiatric symptoms, ensuring appropriate investigations. A multidisciplinary approach involving psychiatry, neurology, and radiology is essential for effective management.

Keywords: multidisciplinary approach, neurocysticercosis, postpartum psychosis, psychiatric symptoms, Taenia solium

Introduction

Postpartum psychosis (PPP) is a psychiatric emergency caused by various etiologies[1]. PPP affects between 1 and 2 per 1000 women and presents with maniac and depressive symptoms, cognitive symptoms, symptoms of hallucinations, and delusions during the first few weeks postpartum (typically 3–10 days)[2,3]. Patients may also present with prodromal symptoms like irritability, mood changes, confusion or disorientation, and insomnia[2]. Females with a personal or family history of bipolar disorder, schizoaffective disorder, other psychotic disorders, previous episodes of PPP, etc. are at higher risk of developing PPP[2]. The underlying mechanism behind PPP includes massive estrogen level drop after expulsion of the placenta, environmental factors like psychosocial stressors, sleep disruptions, and genetic factors[4]. Similar presentation like altered mental status/psychiatric symptoms like depression, psychosis, and cognitive impairment is also exhibited by 4.5% of symptomatic patients of neurocysticercosis (NCC)[5,6].

HIGHLIGHTS

- This case report explores the rare presentation of neurocysticercosis (NCC) as postpartum psychosis (PPP), highlighting significant diagnostic challenges.
- NCC can present with psychiatric symptoms similar to PPP, complicating accurate diagnosis, especially in resource-limited settings.
- Effective management of NCC presenting as PPP requires a comprehensive evaluation by psychiatry, neurology, and radiology teams.
- Clinicians in NCC-endemic regions should consider NCC in postpartum women with typical psychiatric symptoms to ensure timely and appropriate treatment.
- The case underscores the need for a multidisciplinary approach and further research to improve diagnostic and treatment strategies for co-occurring NCC and PPP.

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NCC is an infection of the human central nervous system (CNS) caused by the larval stage of pork tapeworm (*Taenia solium*)[7]. Hatching eggs in the digestive tract produces an embryo that penetrates the mucosa, circulates, and develops into cysticerci, commonly in subcutaneous tissue, muscle, eye, and CNS.[8] NCC affects millions of people worldwide especially in underdeveloped countries of Latin America, Asia, and Africa and accounts for about 50 000 deaths per year[8,9]. Clinical presentation of patients with NCC depends upon the number, stage, size, and location of parasite and inflammatory host response. It ranges from asymptomatic to seizures and focal neurological defects in the case of parenchymal lesions while extraparenchymal lesions have different clinical presentations including intracranial hypertension, hydrocephalous, and cranial nerve abnormalities[5,10]. NCC can also present with impaired mental state and psychiatric symptoms[5,6]. NCC is a public health problem in developing countries facing poverty and lack of health care facilities like neuroimaging methods or appropriate immunologic tests makes accurate diagnosis of NCC challenging[9].

NCC and the postpartum period are independent risk factors for psychiatric disorders. However, neuroinflammatory processes or disruptions in neural circuits can be implicated in mood and psychosis as possible pathophysiological links between NCC and PPP[5]. There is a 50%–80% chance of repeated psychiatric episodes after experiencing PPP. Inadequate treatment of PPP increases the risk of accidents, suicide, homicide, neglect, and even child harm and infanticide. PPP accounts for 38% of maternal suicides in the UK[2]. According to a study conducted by Gilden *et al*[11], 17.8% of women with PPP had impaired mother-infant bonding. The above facts establish the importance of early intervention and support for a patient with PPP. As NCC can also present with psychiatric presentations, the coexistence of PPP and NCC can bring dilemmas in diagnostic, especially in endemic areas facing poverty and poor health care facilities, and therapeutic approaches compromising the good management outcome[6].

The description of NCC in the postpartum period is sparse in the literature and even in endemic regions of NCC, its presentations as PPP is rare and less reported.[5] This case illustrates a rare but significant instance of NCC presenting as PPP and will add value to the literature.

In our case, we present a 23-year-old female presenting with psychosis 15 days postdelivery which was ultimately diagnosed as NCC through radiological modality. This case has been reported in line with the SCARE guidelines[12].

Case presentation

A 23-year-old female was brought to the emergency department after multiple episodes of unresponsive spells 15 days after normal vaginal delivery. She was not responding to the call of family members and was frothing from mouth which lasted for about 15 minutes. For a few days, she was observed as self-smiling and self-crying, irritable, verbally abusive, decreased appetite, disturbed sleep, and unconcerned towards her neonate. She had a history of similar episodes for 5 years and no history of seeing things not seen by others, substance abuse, epilepsy, and no history suggestive of diabetes, hypertension, or tuberculosis. She had no similar psychiatric illness in the family members.

On examination, her vitals were normal. Her cardiovascular, respiratory, and gastrointestinal systems were functioning normally. Neurological examinations were grossly intact. On mental state examination, the patient was nonresponsive to greetings, exhibited a blunted affect, and had a narrow range of motion. Her reactivity was decreased, her speech could not be assessed, and she could not establish rapport. The blood glucose, electrolyte, urea, creatinine, complete blood count, erythrocyte sedimentation rate, and liver function tests were found to be normal which is shown in Table 1.

The patient was then diagnosed with PPP with unresponsive spells and was managed with lorazepam (1 mg per oral once daily for 2 days and twice daily for 1 day continue), olanzapine (10 mg per oral at bedtime continue), haloperidol (10 mg deep intramuscular as needed), phenargon (50 mg deep intramuscular as needed) and with normal saline (intravenous over 24 hours). After 2 days, the patient developed generalized tonic colic seizure with headache and dizziness in the pre-ictal phase, abnormal body movement and loss of consciousness in the ictal phase, and confusion for 15–20 minutes in the post-ictal phase. For this, the patient was managed with a single dose of olanzapine (15 mg per oral) and two doses of lorazepam (1 mg in the morning and 2 mg in the evening). Neuromedicine consultation was done following which the patient was then advised for non-contrast computed tomography (NCCT) and ophthalmology was consulted for an eye examination.

Eye examination was normal whereas NCCT showed some undefined lesions. So contrast-enhanced computed tomography (CECT) was done which revealed a few rounds to oval thin smooth peripherally enhanced lesions with the eccentrically placed focus of calcification with diffuse vasogenic edema in grey white matter junction in the right frontoparietal lobe, a few minimally enhancing hyperdense lesion with the eccentric focus of calcification in left frontoparietal lobe with surrounding vasogenic edema as well as the focus of calcification with no surrounding edema in left parietal lobe (as shown in Fig. 1) Findings were indicative of NCC in various stages.

The patient was further advised for magnetic resonance imaging (MRI) of the head but due to the poor financial status, the patient could not afford it. The patient was then admitted to the ward where the antipsychotic treatment (tablet olanzapine 15 mg per oral once daily for 7 days) along with tablet albendazole (400 mg per oral twice daily for 8 days) and steroid (tablet dexamethasone 6 mg per oral once daily for 8 days) was given for NCC. The patient's condition alleviated during the stay and then she was discharged with oral antipsychotics (tablet olanzapine 10 mg).

Discussion

NCC presenting as PPP is rare and requires prompt diagnosis and management due to its potentially life-threatening consequences to both mother and child. In this case report, we describe a 23-year-old female who presented with psychotic symptoms 15 days postdelivery which was initially diagnosed as PPP but neuroimaging ultimately established a diagnosis of NCC. This case study discusses the clinical presentation, diagnostic evaluation, therapeutic interventions, and the management of this challenging case.

Table 1

Laboratory investigations and findings.

Test	Result	Unit	Ref. range
Complete blood count (CBC)			
Haemoglobin	15.0	gm/dL	11–16
PCV	46.2	%	37–48
Total leucocyte count	12650	cell/mm^3	4000–11000
Difference leucocyte count			
Neutrophil	66	%	40–75
Monocyte	06	%	2–10
Lymphocyte	26	%	20–45
Eosinophil	02	%	1–6
Basophil	00	%	0–1
Platelet count	497000	cell/mm^3	150000–400000
Mean platelet volume (MPV)	5.72	mill/mL	1.5–4.5
RBC	80.8	fL	80–96
MCV	26.2	Pg	27–34
MCH			
MCHC	32.5	gm/dL	32–36
MPV	7.2	fL	6.5–12.0
Glucose (random)	76.0	mg/dL	<140
Urea	41.0	mg/dL	10–50
Creatinine	0.59	mg/dL	0.3–1.2
Total protein	7.7	mg/dL	6.0–8.3
Albumin	4.4	g/dL	3.5–5.0
Total bilirubin	0.83	mg/dL	0.2–1.2
Conj. bilirubin	0.14	mg/dL	0.0–0.2
ALT (SGPT)	29.0	U/L	10–35
AST (SGOT)	123.0	U/L	35–130
ALP (ALK.PHOS.)	3.7	mmol/L	3.5–5.0
Sodium	139.1	mmol/L	136–145
Potassium	3.7	mmol/L	3.5–5.0

PPP is a psychiatric emergency with a prevalence of 1–2 per 1000 women[1]. It is primarily associated with manic and depressive symptoms, cognitive impairment, hallucinations, and delusions occurring typically within the first few weeks postpartum. Known risk factors include a history of bipolar disorder or previous episodes of PPP[2,3]. Significant drops in estrogen levels, sleep disruption, and psychosocial stressors may trigger PPP[4].

Atypical presentation, lack of diagnostic criteria in the diagnostic and statistical manual of mental disorders, and absence of consensus guidelines for management make the diagnosis and management of PPP challenging[1]. A comprehensive evaluation, including detailed medical, psychiatric, and neurological assessments, is necessary to explore the various potential causes of new psychotic symptoms during the postpartum period, as the differential diagnosis is extensive which includes infection, thyroid disorders, parathyroid disease, blood loss, tumor, autoimmune disorder, substance-related symptoms, anoxia, obstetric complications like eclampsia, hemorrhage, endometritis and psychiatric conditions like generalized anxiety disorder, obsessive-compulsive disorder, and acute stress reaction due to a traumatic birth[1,2].

Complete blood counts and comprehensive metabolic panels should be included in the laboratory testing which were found normal in our case and hence differential diagnosis was narrowed. A lumbar puncture and brain imaging, such as CT and MRI, may also be necessary, especially in cases with newly developing psychotic symptoms or co-occurring neurologic symptoms which initially was not done in our case due to financial constraints[1].

In this case, comprehensive laboratory investigation, history, and assessment of the patient ruled out autoimmune disorder, thyroid disorder, tumor, substance use, other psychiatric conditions, and obstetric complication, and the patient was diagnosed with PPP. Being a psychiatric emergency, PPP requires inpatient hospitalization that should be done with a safe plan for the infant which was followed in this case presented. Suicide and infanticide risk should also be considered during treatment plans[2]. Mood stabilizers, particularly lithium are the first line for prophylaxis and treatment of acute PPP. Benzodiazepines and antipsychotics may be used if there is concern for catatonia. Safety in breastfeeding should be considered. Electroconvulsive therapy should be considered in cases that are refractory to mediation. Psychoeducation of the patient and family should be done[1,2]. In the presented case, the patient was managed with benzodiazepines like lorazepam, antipsychotics like olanzapine and haloperidol, and anti-histaminic like phenargon. Other medications were preferred over lithium due to its transfer in the breast milk leading to elevations of thyroid-stimulating hormone, blood urea nitrogen, and creatinine in a few reported cases and required monitoring[13].

Primary prevention includes considering the established risk factors, including the history of bipolar disorder or PPP. Women with risk factors for PPP should be seen by a psychiatrist during pregnancy and have pre-birth planning meetings, strategies to ensure sleep, and pharmacologic plans. Secondary prevention includes early detection. Finally, tertiary prevention includes the management of PPP[2]. However, after 2 days of management of

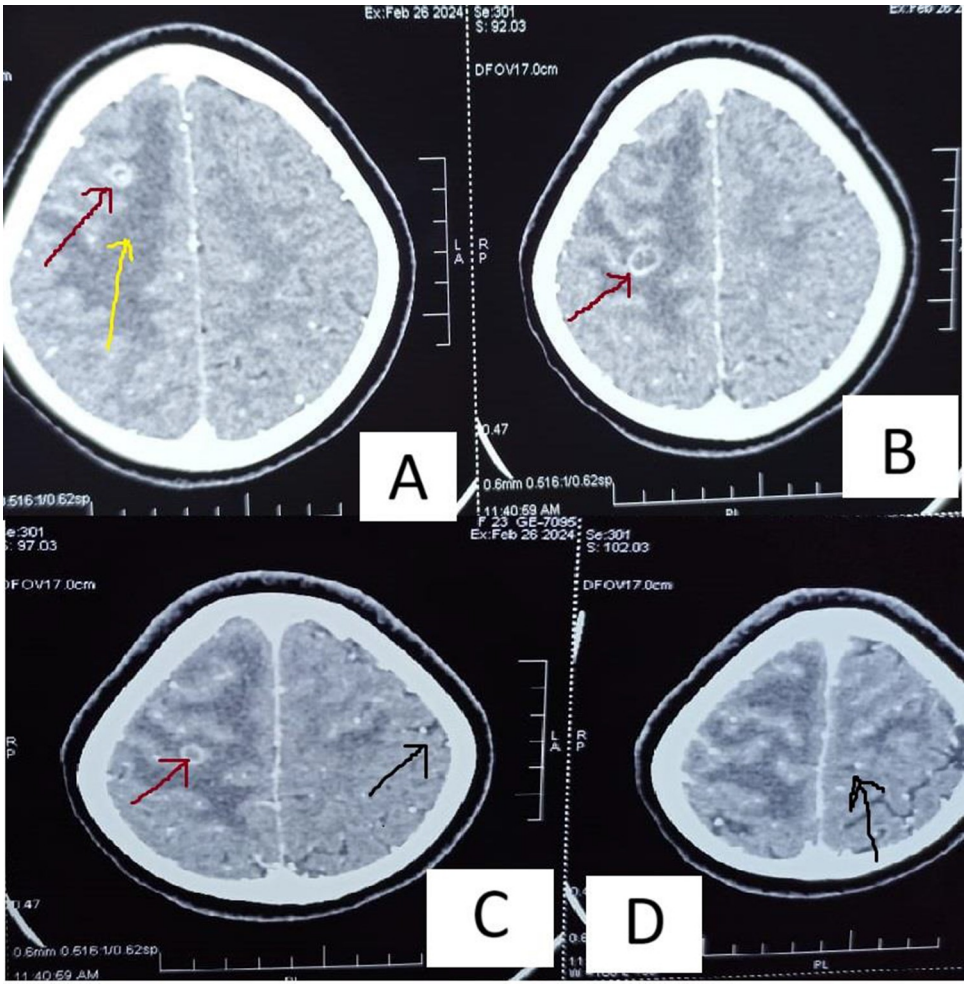

Figure 1. Contras-enhanced computed tomography (CECT) revealed a few round to oval thin smooth peripherally enhanced lesions with the eccentrically placed focus of calcification with diffuse vasogenic edema in grey white matter junction in the right frontoparietal lobe (A, B, C, red arrow; A, yellow arrow). Few minimally enhancing hyperdense lesions with the eccentric focus of calcification in the left frontoparietal lobe with surrounding vasogenic edema as well as the focus of calcification with no surrounding edema in the left parietal lobe (C, D, black arrow).

PPP, the patient developed generalized tonic colic seizure with headache and dizziness in the pre-ictal phase, abnormal body movement and loss of consciousness in the ictal phase, and confusion for 15–20 minutes in the post-ictal phase was managed with a single dose of olanzapine (15 mg per oral) and two doses of lorazepam (1 mg at morning and 2 mg at evening) following which neuro medicine consultation was done and the patient was advised for NCCT and eye examination. Eye Examinations were normal but NCCT showed some undefined lesions. CECT was performed which led to a diagnosis of NCC.

NCC, caused by the larval stage of *T. solium*, is a major public health concern in developing countries[9]. It predominantly affects the CNS and can present with a wide range of neurological symptoms, including seizures, intracranial hypertension, and psychiatric manifestations[5,6].

Diagnosis of NCC depends on clinical history, exposure history, neuroimaging, and immunological tests[7,9]. Del Brutto's diagnostic criteria for NCC are mentioned in Table 2[9]. Diagnosis of NCC can be challenging, especially in endemic and resource-limited countries where laboratory and imaging techniques are scarce. The method of choice for diagnosis of NCC is Neuroimaging which includes cranial computed tomography (CT) and/or MRI, which should be combined with serological tests[9]. In the presented case, CECT revealed a few round to oval thin smooth peripherally enhanced lesions with the eccentrically placed focus of calcification with diffuse vasogenic edema in grey white matter junction in the right frontoparietal lobe, a few minimally enhancing hyperdense lesions with eccentric focus of calcification in the left frontoparietal lobe with surrounding vasogenic edema along with focus of calcification with no surrounding edema in left parietal lobe which were indicative of NCC.

Most psychiatrists believe that postpartum mental illnesses, like psychosis, are not separate conditions but forms of existing disorders, such as depression or schizophrenia. Mental and behavioral disorder associated with the puerperium (F53) is included in the International Classification of Diseases-10 classification and is recommended to use it only as a last resort when it's not possible to classify the condition as another disorder, either because insufficient information is available or because special additional features make their classification elsewhere inappropriate. With the purpose of

Table 2

Diagnostic criteria for neurocysticercosis[9].

Diagnostic criteria
Absolute criteria
• Histologic demonstration of the parasite from biopsy of a brain or spinal cord lesion
• Evidence of cystic lesions showing the scolex on neuroimaging studies
• Direct visualization of subretinal parasites by fundoscopic examination
Major criteria
• Evidence of lesions highly suggestive of neurocysticercosis on neuroimaging studies
• Positive serum immunoblot for the detection of anticysticercal antibodies
• Resolution of intracranial cystic lesions after therapy with albendazole or praziquantel
• Spontaneous resolution of small single-enhancing lesions
Minor criteria
• Evidence of lesions suggestive of neurocysticercosis on neuroimaging studies
• Presence of clinical manifestations suggestive of neurocysticercosis
• Positive CSF ELISA for detection of anticysticercal antibodies or cysticercal antigens
• Evidence of cysticercosis outside the CNS
Epidemiologic criteria
• Individuals coming from or living in an area where cysticercosis is endemic
• History of frequent travel to disease-endemic areas
• Evidence of a household contact with *Taenia solium* infection
Degrees of diagnostic certainty
Definitive diagnosis
• Presence of one absolute criterion
• Presence of two major plus one minor or one epidemiologic criteria Probable diagnosis
• Presence of one major plus two minor criteria
• Presence of one major plus one minor and one epidemiologic criteria
• Presence of three minor plus one epidemiologic criteria

tracking the workload and planning for healthcare services, the doctors can classify the conditions as mild (F53.0) or severe (F53.1) even if everything about the case is not known[14].

American College of Obstetrics and Gynaecology recommends that clinicians provide immediate medical attention for PPP. Given the rarity of PPP and majority of cases being new diagnosis, universal screening is not recommended. However, a thorough past medical and obstetric history is indicated at time of obstetric visit to verify if risk factors like bipolar disorder or history of PPP exist in the mother[15]. Comparative analysis of the presented case with the relevant case studies has been shown in Table 3.

Treatment of NCC should be individualized and initially focused on symptomatic management and definitive therapy which includes medical or surgical treatment for cyst should be considered when appropriate[9,10]. Symptomatic treatment involves antiepileptic drugs, anti-inflammatories, analgesics, and management of intracranial hypertension when present. Anti-parasitic therapy is initiated when the patient is stable for which albendazole and praziquantel are commonly used along with corticosteroids to control the host immune response[9]. In this case, the patient was managed with albendazole and steroids after which the patient's condition was alleviated. Surgical intervention may be needed in cases of obstructive hydrocephalus caused by intraventricular cysts or cysts in the basal cisterns or large cysts or cyst clusters[16]. However, this was not necessary in this case as per neuro medicine consultation. In case neuroimaging is not available, the patient should be managed symptomatically only and anti-parasitic drugs should be avoided due to the potential risk of aggravating raised intracranial pressure and brain herniation[9].

The presentation of NCC as PPP necessitates a multidisciplinary approach for accurate diagnosis and effective management. This includes detailed medical, psychiatric, and neurological assessments, along with appropriate neuroimaging and laboratory investigations. Treatment must address both the psychiatric and neurological components, with antipsychotics for psychosis and antiparasitic therapy combined with corticosteroids for NCC.

The management strategy, in this case, involved in-patient care, the use of antipsychotics (olanzapine, haloperidol), benzodiazepines (lorazepam), and antiparasitic therapy (albendazole) along with steroids, which led to significant improvement in the patient's condition after which the patient was discharged with oral antipsychotics and antiepileptic medications. Psychoeducation of the patient and family was also done. The patient did not report further for follow-up.

The primary limitation of this case was the inability to perform MRI due to the patient's financial constraints, which may have provided more detailed insights into the extent of NCC. Future cases should aim for comprehensive neuroimaging when possible. The literature documents cases of NCC with psychiatric presentations, but descriptions of its presentations as PPP are sparse. This case contributes valuable insights into the intersection of these two conditions, reinforcing the need for heightened clinical awareness and comprehensive diagnostic approaches in similar clinical scenarios. It is recommended that clinicians in endemic areas for NCC maintain a high index of suspicion for this condition in postpartum women presenting with atypical psychiatric symptoms. Enhanced awareness, timely diagnosis, and a multidisciplinary approach are critical for improving patient outcomes. Further research and documentation of similar cases are needed to develop robust diagnostic and treatment guidelines.

Conclusion

This case illustrates a rare but significant instance of NCC presenting as PPP and therefore, highlights the complex interplay between NCC and PPP, emphasizing the diagnostic challenges and the necessity for a comprehensive approach to management. NCC, although a rare cause of PPP, should be considered in endemic regions or patients presenting with atypical psychiatric symptoms following childbirth. In this case, the diagnosis was confirmed through radiological findings, which were pivotal in guiding the treatment strategy. The patient's condition improved with a combination of antipsychotic medications and specific treatment for NCC, including albendazole and corticosteroids. This underscores the importance of a multidisciplinary approach involving psychiatry, neurology, and radiology for optimal patient outcomes. Future research and case studies are essential to better understand the co-occurrence of these conditions and to develop standardized guidelines for their management. The patient's recovery and discharge on appropriate medications demonstrate the potential for positive outcomes with timely and accurate diagnosis and treatment. Early and accurate diagnosis, coupled with appropriate treatment, can significantly improve patient outcomes, even in resource-limited settings.

Table 3

Comparative analysis of the presented case with the relevant case studies.

Parameters	Thapa *et al*[17]	Present case	Ursini *et al*[5]	Ursini *et al*[5]
Age (years)	51	23	28	32
Gender	Male	Female	Female	Female
Clinical presentation	Abnormal behavior, irrelevant talk, irritability, episodes of anger, poor eye contact, continuous speech at an increased rate, rhythm, and intensity	Unresponsive spells, frothing from the mouth, self-smiling and self-crying, irritability, verbal abuse, decreased appetite, disturbed sleep, and unconcerned toward neonate in the postpartum period	Loss of consciousness and five focal-to-bilateral tonic-clonic seizures 15 days postpartum Previous history of NCC as well	One simple visual focal seizure and two focal-to-bilateral tonic-clonic seizures 5 days postpartum Previous history of NCC as well
Laboratory findings	CT brain: multiple calcified lesions suggestive of NCC	Complete blood count: normal	Post-ictal EEG: diffuse delta activity on the left and spikes and waves in bilateral temporal regions	Interictal EEG: sporadic theta activity in the occipital region
	MRI brain: multiple ring-enhancing lesions suggestive of NCC	Liver function test: normal	Interictal EEG: spikes-and-waves activity in the left temporal-occipitoparietal region	MRI brain: multiple calcified lesions with perilesional vasogenic edema
	EITB (enzyme-linked Immunotranser blot) test: positive for NCC	CT brain: lesions suggestive of NCC	MRI and CT: multiple calcified lesions with perilesional vasogenic edema	
Management	Oral antipsychotic medications intravenous sedation as needed steroids and albendazole (400 mg twice a day for 4 weeks)	Lorazepam (1 mg PO OD for 2 days, then BD for 1 day), olanzapine (10 mg po HS), haloperidol (10 mg deep I/M SOS), phenargon (50 mg deep I/M SOS) and normal saline (I/V over 24 hours) for PPP	Carbamazepine (400 mg three times a day), dexamethasone (8 mg total daily dose) for 2 months.	Antiepileptic drug (levetiracetam, 500 mg three times a day), corticosteroids (dexamethasone, 7.5 mg total daily dose) for 2 months.
	Sodium valproate for seizure during stay	Albendazole and steroids for NCC	The corticosteroid drug was tapered off over 6.5 months by reducing the dose by 0.75 mg every 4 weeks	Corticosteroid drug was tapered off over 6.5 months by reducing the dose by 0.75 mg every 4 weeks
Outcome	Recovered completely and was discharged with oral antipsychotic and antiepileptic medications	Significantly improved and discharged with oral antipsychotic and antiepileptic medications	Postpartum recurrence was managed successfully. The patient remains on carbamazepine, with discontinuation to be evaluated 2 years after the last seizure occurrence	Postpartum recurrence was managed successfully. The patient remains on levetiracetam, with corticosteroids tapered off, and no relapse

Ethical approval

The study is exempt/waived from ethical approval in our institution as it poses minimal risk to the patient and the study is for educational purposes/activities.

Consent

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

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Author's contribution

T.N.Y., K.R., A.B.: literature review, follow-up the patient, writing the manuscript, and final approval of the manuscript. B.K.H., A.A.: Writing the manuscript, follow-up the patient, final approval of the manuscript. A.Y., B.K.S., P.K.B., P.D., S. P.: final approval of the manuscript. S.S.: follow-up the patient, writing the manuscript. R.K.: writing the manuscript..

Conflicts of interest disclosure

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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