Case Report

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Neurocysticercosis presenting as a subdural hematoma in the postpartum period: a diagnostic challenge

Doğum sonrası nörosistiserkoz: subdural hematom ile seyreden tanısal zorluk

Gülencan Yumuşak Ergin, Ahmet Gökhan Çakıroğlu, Ramazan Bülbül, Sabri Güngör, Sümeyye Önal Altınkaya

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Abstract

Neurocysticercosis is the most common parasitic infection of the human central nervous system, caused by the larval stage of Taenia solium. Its clinical manifestations vary widely, ranging from asymptomatic cases to seizures, focal neurological deficits, and increased intracranial pressure. The postpartum period involves significant immunological, hormonal, and metabolic changes that may influence the onset, progression, or presentation of infectious diseases. This report presents a rare case of neurocysticercosis presenting as a subdural hematoma during the postpartum period. A twenty-one-year-old woman with no previous medical history was admitted thirty-five days after cesarean section due to severe headache and speech impairment. Cranial computed tomography revealed a left-sided subdural hematoma measuring fifteen millimeters in thickness, associated with a seven-millimeter midline shift. The patient underwent emergency craniotomy to evacuate the hematoma. Histopathological examination of the surgical specimen confirmed the presence of the larval form of Taenia solium, thereby establishing the diagnosis of neurocysticercosis. Following surgery, the patient experienced seizures, which required intensified antiepileptic medication. Antiparasitic treatment consisting of albendazole and corticosteroids was initiated. Further radiological evaluations, including cranial and spinal magnetic resonance imaging, did not identify any additional cystic lesions. The patient responded well to treatment and was discharged on hospital day twenty-three with mild speech impairment. At the one-month follow-up visit, her neurological examination was entirely normal. This case highlights an unusual presentation of neurocysticercosis in the postpartum period and underscores the importance of considering this diagnosis in patients presenting with seizures or subdural hematoma in endemic regions.

Keywords: Neurocysticercosis, subdural hematoma, parasitic infections.

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Öz

Neurocysticercosis, insanlarda merkezi sinir sisteminin en yaygın paraziter enfeksiyonudur ve Taenia solium'un larval formunun neden olduğu bir hastalıktır. Klinik belirtileri oldukça geniş bir yelpazede değişkenlik gösterebilir; asemptomatik olgulardan nöbetlere, fokal nörolojik defisitlere ve artmış kafa içi basınca kadar uzanabilir. Doğum sonrası dönem, enfeksiyon hastalıklarının ortaya çıkışını, seyrini ya da klinik tablosunu etkileyebilecek önemli immünolojik, hormonal ve metabolik değişikliklerle karakterizedir. Bu yazıda, doğum sonrası dönemde subdural hematom şeklinde ortaya çıkan nadir bir neurocysticercosis vakası sunulmaktadır. Önceden bilinen bir hastalık öyküsü olmayan yirmi bir yaşındaki kadın hasta, sezaryen doğumdan otuz beş gün sonra şiddetli baş ağrısı ve konuşma bozukluğu şikayetleri ile başvurdu. Beyin bilgisayarlı tomografisinde sol tarafta on beş milimetre kalınlığında subdural hematom ve yedi milimetre orta hat kayması saptandı. Acil kraniyotomi ile hematom boşaltıldı. Cerrahi örneğin histopatolojik incelemesi, Taenia solium'un larval formunu göstererek neurocysticercosis tanısını doğruladı. Ameliyat sonrası dönemde hastada nöbetler gelişti ve antiepileptik tedavi artırıldı. Albendazol ve kortikosteroid içeren antiparaziter tedavi başlandı. Beyin ve omuriliğin manyetik rezonans görüntülemesinde başka kistik lezyon saptanmadı. Hasta tedaviye olumlu yanıt verdi ve yirmi üçüncü günde hafif konuşma bozukluğu ile taburcu edildi. Bir ay sonraki kontrolde nörolojik muayenesi tamamen normaldi. Bu olgu, doğum sonrası dönemde nadir bir neurocysticercosis sunumu olarak dikkat çekmektedir ve endemik bölgelerde nöbet veya subdural hematomla başvuran kadınlarda ayırıcı tanıda düşünülmelidir.

Gülencan Yumuşak Ergin, Department of Anesthesiology and Reanimation, Aksaray University Training and Research Hospital, Aksaray, Türkiye, e-mail: gulencanyumusak@gmail.com (https://orcid.org/0000-0002-5396-6816) (Corresponding Author)

Ahmet Gökhan Çakıroğlu, Department of Pathology, Aksaray University School of Medicine, Aksaray, Türkiye, e-mail: gulencan_yumusak@hotmail.com (https://orcid.org/0009-0001-6077-9541)

Ramazan Bülbül, Department of Obstetrics and Gynecology, Aksaray University School of Medicine, Aksaray, Türkiye, e-mail: dr.ramazan_bulbul@hotmail.com (https://orcid.org/0000-0003-3321-3718)

Sabri Güngör, Department of Neurosurgery, Aksaray University Training and Research Hospital, Aksaray, Türkiye, e-mail: sabrigngr86@gmail.com (https://orcid.org/0009-0007-0924-5185)

Sümeyye Önal Altınkaya, Department Anesthesiology and Reanimation, Aksaray University School of Medicine, Aksaray, Türkiye, e-mail: drsumeyyeonal@gmail.com (https://orcid.org/0009-0008-4097-5466)

Anahtar kelimeler: Nörosistiserkoz, subdural hematom, parazitik enfeksiyon.

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Introduction

The adult cestode Taenia solium and its larval stage, cysticercus, infect humans and animals, including cattle and pigs. Humans acquire the infection by consuming infected meat. While larvae mainly invade muscle tissue, they can also reach the central nervous system (CNS) via the bloodstream [1]. When cysticerci enter the CNS, they are in a vesicular stage, with the parasite harboring an invaginated scolex surrounded by a translucent vesicular fluid and covered by a transparent membrane. Cysticerci may remain in this state for years or may degenerate after a host immunologic response [1]. Neurocysticercosis (NCC) is the most common parasitic infection of the CNS, caused by the larval form of *Taenia solium*. Within the CNS, NCC can manifest in various locations, including the brain parenchyma, ventricles, basilar cisterns, sulci, gyri, spinal cord, and retina [2]. The clinical spectrum of NCC varies widely, ranging from asymptomatic cases to severe neurological disorders. Among symptomatic cases, seizures are the most common presentation, especially when the larvae are located in the brain parenchyma [3, 4]. NCC is the leading preventable cause of epilepsy worldwide, contributing to approximately 30% of all epilepsy cases in endemic regions, with some communities reporting associations as high as 70% [5]. Neurocysticercosis remains endemic across Latin America, much of sub-Saharan Africa, the Indian subcontinent, and Southeast Asia. Nevertheless, reports from historically non-endemic settings are increasing, a trend largely attributed to population mobility through migration and international travel [6, 7].

Pregnancy and the postpartum period represent a unique physiological phase characterized by significant nutritional, metabolic, hormonal, and immunological changes. These changes can influence the course of various diseases, including NCC, potentially affecting its clinical presentation and progression [8].

Case presentation

A 21-year-old female patient with no known medical history or regular medication use presented to the emergency department (ED) with complaints of tooth pain. On the same day, she returned to the ED with severe headache and speech disturbances. On neurological examination, she was alert, able to follow commands, and exhibited spontaneous eye opening; however, her speech was incoherent. Her obstetric history included a cesarean section 35 days prior under spinal anesthesia. At 34 weeks, a perinatology ultrasound detected 4×3 cm and 2×2 cm cystic formations, difficult to classify as umbilical or placental cysts. At 38 weeks, due to the development of oligohydramnios and breech presentation, a cesarean section (C/S) was performed. The placenta's fetal surface revealed four bulging structures, with the largest measuring 4×2.5×1 cm and the smallest 0.5 cm. These were diagnosed as chorionic cysts (Figure 1). The patient was discharged two days postoperatively without complications.

She reported intermittent headaches after the C/S but did not seek medical attention. On postpartum day 35, she presented again to the ED with tooth pain. Cranial computed tomography (CT) revealed an acute subdural hematoma (SDH) over the left cerebral hemisphere, with a maximal thickness of 15 mm and a midline shift of 7 mm. The brain parenchyma was otherwise unremarkable. The patient's vital signs were stable before surgery. Due to neurological deficits and a frontotemporoparietal subdural hematoma, a craniotomy was performed for hematoma evacuation. Intraoperatively, a subacute,

organized hematoma with membranous and acute and a single flat drain was placed. The dura was closed with interrupted silk sutures, and the bone flap was secured with miniplates and screws. The surgical layers were closed anatomically. The procedure was completed without complications, and the patient was successfully extubated. Postoperatively, her Glasgow Coma Scale score was 15, and she was transferred to the intensive care unit. A postoperative cranial CT was performed (Figure 2).

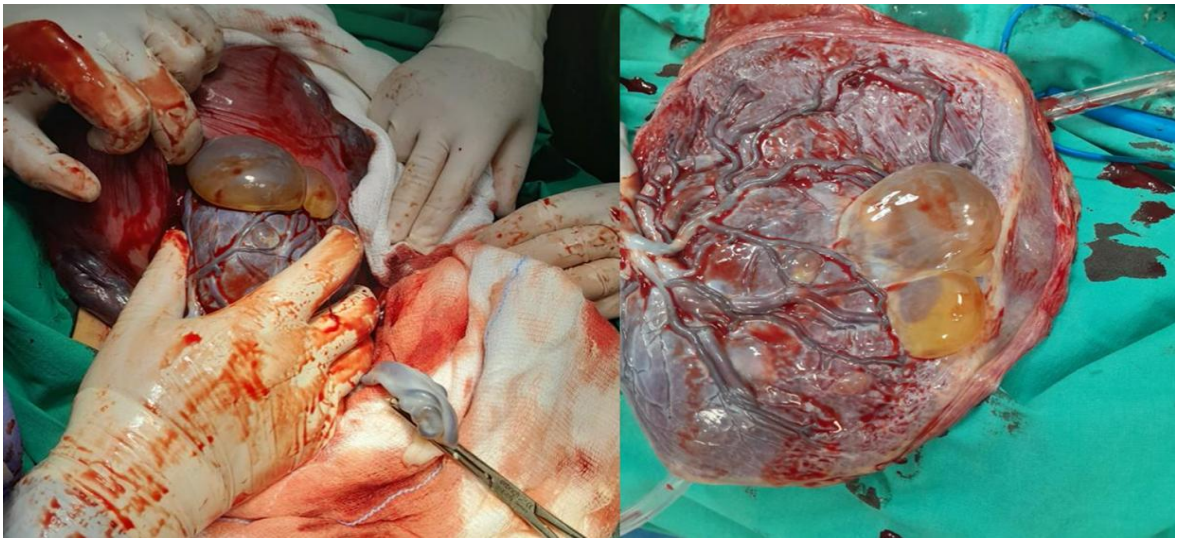

Figure 1. Gross view of the fetal surface of the placenta demonstrating multiple chorionic cysts, with the largest cyst measuring 4 × 2.5 × 1 cm

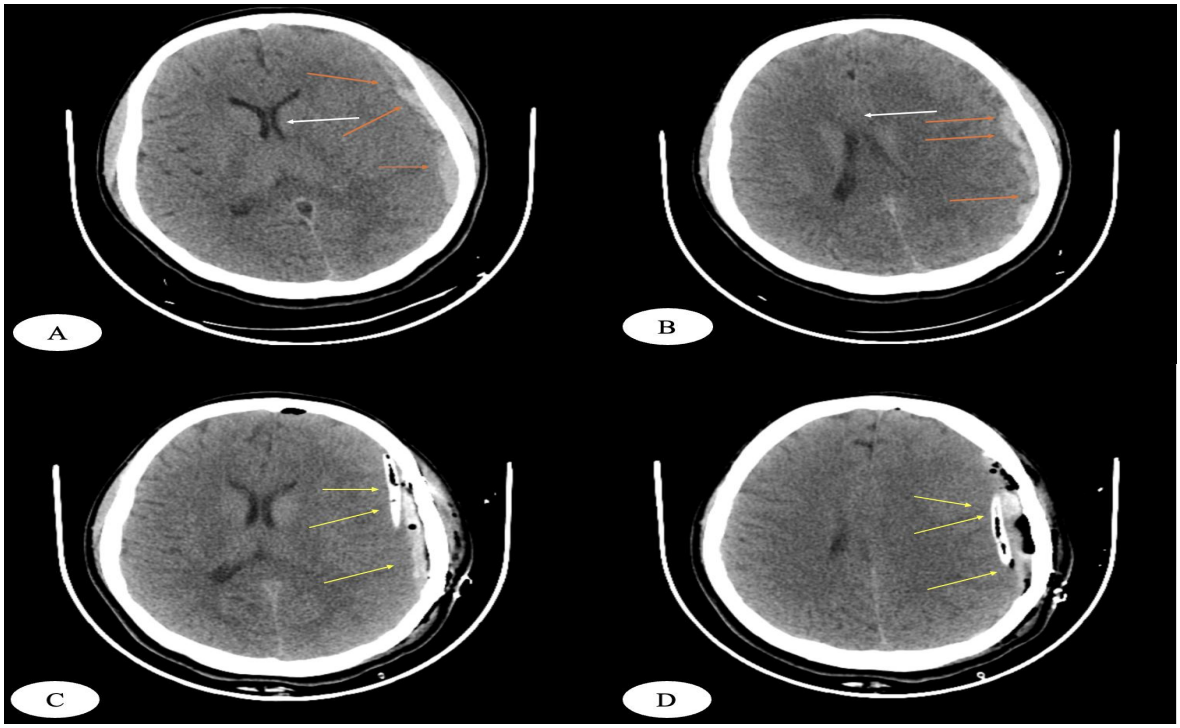

Figure 2. Preoperative cranial CT images

(A, B) revealing a large left frontoparietal subdural hematoma with mass effect and midline shift (A, B: orange arrows indicate the subdural hematoma; white arrow indicates the midline shift). Postoperative cranial CT images (C, D) demonstrating successful evacuation with resolution of midline shift (C, D: yellow arrows indicate the postoperative drain catheter and expected postoperative changes)

Antiepileptic therapy with levetiracetam (1000 mg twice daily) was initiated. On postoperative day 2, she developed speech disturbance and facial paralysis, but no additional pathology was identified on imaging. On postoperative day 3, despite antiepileptic treatment, she had a seizure, requiring phenytoin. With increased seizure activity, she was intubated and sedated with midazolam and thiopental. Empiric broad-spectrum antibiotics were initiated for fever and elevated C-reactive protein, consisting of meropenem 1 g every 8 hours and vancomycin 1 g every 12 hours. On hospital day 4, pathology confirmed NCC. Histological analysis of the hematoma-containing soft tissue (4×3×0.4 cm) revealed fibrotic cystic structures, eosinophilic abscesses, necrosis, and cuticle fragments, consistent with cysticercosis (Figure 3). Following diagnosis, dexamethasone (0.4 mg/kg/day) and albendazole (15 mg/kg/day) in two divided doses were initiated. An electroencephalogram was normal (Figure 4A). The patient was extubated on hospital day 8, and seizure activity ceased. Preoperative imaging was reassessed, with repeat cranial CT, brain magnetic resonance imaging (MRI), and spinal MRI showing no additional cysts or pathologies. Ophthalmic examination ruled out parasitic involvement. Agglutination and PCR studies were unavailable due to hospital limitations. Based on the recommendation of the infectious diseases team, broad-spectrum antibiotic therapy was administered for 21 days as part of perioperative management. Antiparasitic treatment was continued for 20 days and subsequently discontinued after favorable clinical and radiological improvement during close follow-up. Dexamethasone tapering was initiated on day 20, and the drug was gradually discontinued thereafter. The patient was discharged on day 23 with mild dysarthria but no additional neurological deficits. At the one-month follow-up, her neurological examination and EEG were entirely normal (Figure 4B).

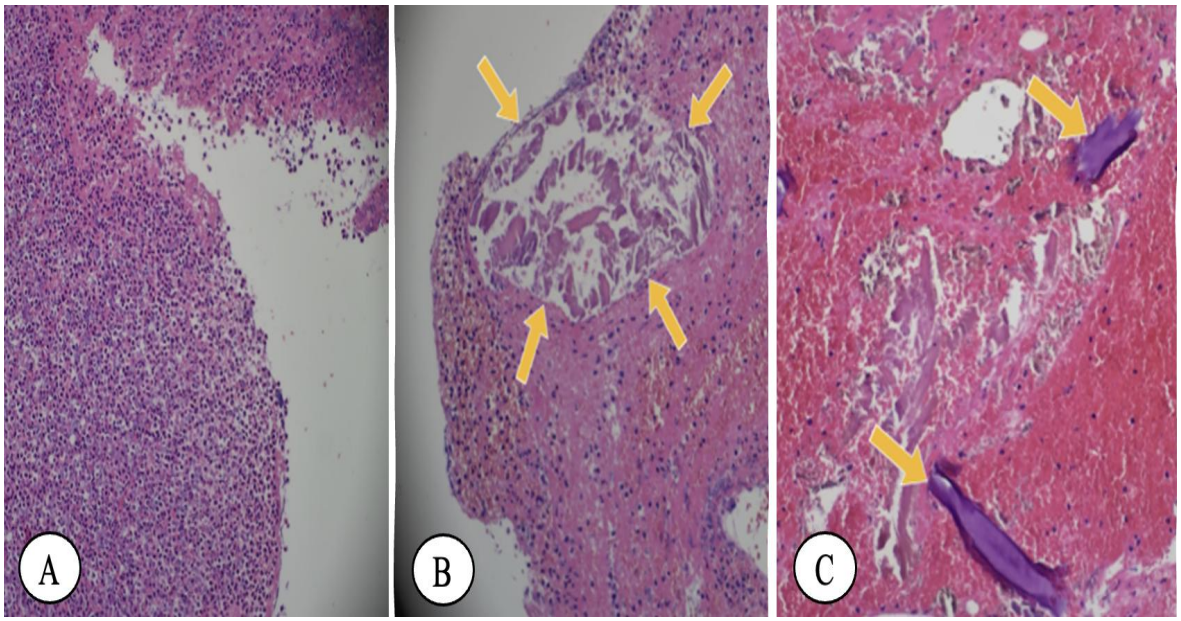

Figure 3. Histopathological examination of the lesion

A) Intense eosinophilic inflammation with microabscess formation surrounding parasite cuticle fragments (Hematoxylin and eosin, H&E; 10×20), B) Taenia solium larva (cysticercus) enclosed within a fibrous cyst wall containing cuticle fragments (H&E; 10×20; yellow arrows indicate the cysticercus and the fibrous wall), C) Degenerated cuticle fragments without an intact cystic wall of the cysticercus (H&E; 10×20; yellow arrows indicate cuticle fragments)

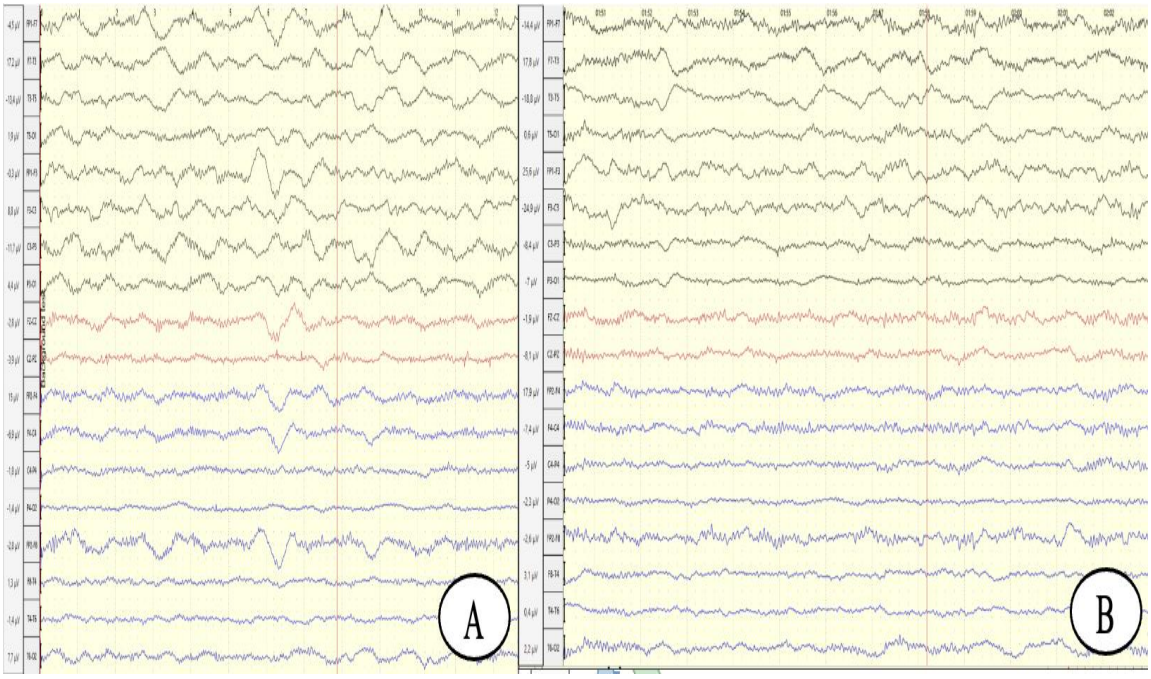

Figure 4. A) Patient's initial EEG at the time of diagnosis, B) The follow-up EEG obtained at the 1-month control visit

Discussion

The incidence of NCC varies across regions, with higher rates observed in populations residing in or migrating from endemic areas. In these settings, cysticercosis remains a major public health concern. According to the WHO, current maps of endemicity are limited because they reflect entire countries rather than subnational areas, which may obscure important geographic and cultural differences. This is particularly relevant for large countries or those with heterogeneous populations, where the disease may be restricted to specific communities. Endemic regions include sub-Saharan Africa, Latin America, and East, South, and Southeast Asia, where public health interventions remain essential. In contrast, countries such as Türkiye are considered non-endemic, and reported cases are rare, typically associated with migration or sporadic exposure in rural areas. This epidemiological context underscores the rarity and clinical significance of our presented case [6, 7].

During pregnancy, it is rare but often presents with seizures, headaches, or focal deficits. Immunological and hormonal changes may influence disease progression, complicating diagnosis and management [8]. In pregnant patients with new-onset seizures, differential diagnoses should include preeclampsia and eclampsia, which share symptoms like altered consciousness and visual disturbances with NCC. A thorough history and examination are essential for diagnosis. Persistent seizures also risk fetal hypoxia. Managing NCC during pregnancy and the postpartum period is particularly challenging due to these overlapping clinical features and the complexities of treatment in this population [8].

The patient had oligohydramnios and placental cysts, with no known link to Taenia infection. These changes may stem from pregnancy-related immunological shifts, which could also have influenced the infection. NCC was undetected during pregnancy and diagnosed postpartum after a subdural hematoma.

Subdural NCC cysts are extremely rare. Beyond the classic parenchymal form, atypical subdural presentations have been reported. An early report described a patient with a history of cerebral cysticercosis who presented with signs of raised intracranial pressure. Although CT suggested a subdural

hematoma, intraoperatively the hematoma proved to be a subdural collection of multiple cysticerci, representing the first description of this complication at the time of publication [9]. In one case, multiple free cysticerci in the subdural space mimicked a chronic subdural hematoma, and the diagnosis was confirmed intraoperatively and histologically in an 85-year-old patient managed with burr-hole drainage and albendazole [10]. A prior report documented a solitary cerebral granuloma accompanied by a subdural effusion**,** with regression of both the effusion and the granuloma following albendazole therapy [11]. To contextualize our subdural presentation within the atypical spectrum of NCC, we compiled a focused summary of previously reported subdural/hematoma-like cases (Table 1).

Table 1. Atypical subdural presentations of neurocysticercosis reported in the literature

Report	Age/ Sex	Clinical presentation	Imaging studies	Surgery	Medication	Outcome
Feinberg and Valdivia, 1984 [9]	33/F	Grand mal seizures, occipital headaches	Left calsified frontal cystic lesion, chronic subdural hematoma	Frontotemporal craniotomy	None	Improved
Rajshekhar, 2001 [11]	24/F	Focal seizures	Lesion in the right posterior frontal region, subdural effusion	None	Albendazole (400 mg twice a day, three weeks)	Improved
Im et al., 2005 [10]	85/M	Motor and sensory seizures, epileptic aphasia	Subdural collection divided by multiple septa	Burr hole	Albendazole (duration unknown)	Improved

Radiological imaging plays a crucial role in the diagnosis and staging of NCC. CT and MRI allow for the assessment of cyst morphology, localization, and disease burden, as well as the identification of surrounding inflammation [12]. There was no radiological imaging performed before the development of the subdural hematoma in this patient. Additionally, subsequent CT and MRI evaluations did not reveal any additional cyst formation.

Most hemorrhagic presentations reported in the literature are subarachnoid. In neurocysticercosis, subarachnoid hemorrhage has been described in both aneurysmal and non-aneurysmal forms. Although a unifying mechanism has not been established, NCC-related inflammatory vasculopathy is thought to precipitate cerebrovascular events, contributing to both ischemic and hemorrhagic complications [13]. The mechanism leading to SDH in NCC is not fully understood but may involve inflammatory processes, vascular compromise, or direct cystic expansion causing subdural hemorrhage [14].

In this patient, a probable cyst located in the subdural region may have led to the development of a subdural hematoma. The presence of intense eosinophilic inflammation and parasite cuticle fragments on histopathology strongly supports a parasitic etiology. Clinicians should suspect subdural NCC cysts in atypical subdural fluid collections, especially in endemic areas. Early diagnosis and antiparasitic treatment are crucial.

Following the improvement in the patient's postoperative clinical condition, seizure activity persisted. However, after the administration of antiparasitic agents and dexamethasone, the seizure activity decreased. Although the 2017 IDSA/ASTMH guidelines often advise prolonged antiparasitic therapy for subarachnoid NCC, sometimes extending for months until radiologic resolution is achieved, the infectious diseases team recommended a shorter course in this patient given clinical stability and a favorable postoperative course [15].

Spinal anesthesia can contribute to subdural hematoma formation, though rarely. Dural puncture may cause cerebrospinal fluid (CSF) loss, leading to brain displacement, vein rupture, and hemorrhage. Risk increases with pregnancy, dehydration, repeated punctures, large dural defects, anticoagulant use, and vascular abnormalities. Pregnant patients may have a higher risk of SDH after dural puncture due to the frequent use of neuraxial analgesia. This risk is especially increased in those experiencing headaches following dural puncture [16, 17].

In our patient, a post-dural puncture hematoma was initially considered until the pathological diagnosis was confirmed. It remains unclear whether the subdural cyst resulted from dural traction during spinal anesthesia or if pre-existing NCC cysts were affected by postpartum hormonal and immunological shifts. The patient also reported postpartum headaches, which made it challenging to differentiate between post-spinal headache and NCC-related symptoms. Although spinal anesthesia-related SDH is an important differential consideration, the localization of the lesion, the absence of the typical clinical course of post–dural puncture complications, and the histopathological confirmation of cysticercus tissue strongly favor NCC as the primary cause. Therefore, while alternative explanations were carefully considered, the convergence of pathological and clinical findings points toward NCC as the underlying mechanism of SDH in this case. Taken together, NCC is the most plausible primary cause of the SDH in our patient. Nevertheless, a contributory mechanism related to spinal anesthesia, specifically cerebrospinal fluid loss causing dural traction, may have facilitated the development or progression of subdural bleeding. Published reports show wide variability in the timing of diagnosis; in one case, a presumptive diagnosis of subdural hematoma was made as late as 20 weeeks after the index event [18]. Recognized risk factors for post–dural puncture subdural hematoma include pregnancy, repeated dural punctures, exposure to anticoagulants, intracranial vascular abnormalities, and cerebral atrophy [19]. Such factors may promote vascular fragility and intravascular dysfunction, thereby predisposing susceptible patients to subdural bleeding. Although NCC is the most likely primary cause, a contributing effect of CSF loss–related dural traction in a vulnerable vascular environment cannot be ruled out.

Pathological evaluation is crucial for a definitive diagnosis. In CNS specimens (brain or subarachnoid space), small parasite fragments are typically observed. However, the most important initial clue is an intense eosinophilic tissue reaction, such as eosinophilic microabscesses, surrounding the parasitic tissue fragments (Figure 3A).

Pathologists must be familiar with the detailed structures of the larval stage of Taenia (Cysticercus) to diagnose neurocysticercosis. Small parasite fragments are often overlooked. In the vesicular stage, Cysticercus consists of a vesicular fibrous wall and an invaginated scolex (parasite head). Later, the parasite evaginates, maturing into Taenia. However, due to intense eosinophilic immune reactions, parasites are usually observed in cyst form. The cuticle, a multilayered eosinophilic collagenous structure, is enclosed within a fibrous cyst wall containing cuticle fragments (Figure 3B). Due to the strong host immune response, the cysticercus fibrous wall often disrupts, leaving only cuticle fragments surrounded by an intense eosinophilic reaction (Figure 3C) [20]. Combining serological, molecular, and imaging methods enhances diagnostic accuracy, especially in endemic regions [12].

Due to the limitations of our hospital's laboratory tests, an immunological examination could not be performed. The pathological diagnosis confirmed the disease with certainty, while the clinical improvement following antiparasitic treatment further supported our diagnosis [21].

This case highlights a rare presentation of NCC as a subdural hematoma in the postpartum period. Early multidisciplinary management that combines neuroimaging, operative and, when indicated, histopathological confirmation, and antiparasitic and anti-inflammatory therapy is essential. NCC should be considered in the differential diagnosis of postpartum seizures and subdural lesions, especially in patients with epidemiologic risk factors.

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