

Unveiling a Giant Hydatid Cyst of the Brain: A Neurosurgical Experience with a Rare Zoonotic Infection

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ABSTRACT

Cerebral hydatid disease is a rare but significant symptom of *Echinococcus granulosus*, accounting for 0.5–2% of all hydatid cases. It typically affects children in endemic areas and is often asymptomatic until the cyst reaches a significant size, making early detection difficult. An 11-year-old kid from rural Punjab, Pakistan, had a four-month history of growing headaches and head enlargement, with no seizures or neurological impairments. A big, well-defined cyst (10.9 × 10.2 × 11.2 cm) in the left parieto-occipital area caused significant mass effect and moderate hydrocephalus, as confirmed by MRI. Despite the cyst's massive size, the child had no neurological abnormalities; serologic testing was negative, and systemic imaging excluded extracranial involvement. The patient underwent a parieto-occipital craniotomy, and the cyst was removed intact using the Dowling-Orlando method. Histopathology confirmed the diagnosis of hydatid illness. Albendazole (10 mg/kg) was given after surgery for 30 days, coupled with anticonvulsants. Recovery was uneventful, and follow-up imaging revealed no recurrences.

This example demonstrates that cerebral hydatid disease should be seriously investigated in children from endemic rural areas who present with increasing macrocephaly or indications of elevated intracranial pressure, even if serology is negative. Early MRI-based detection and precise intact-cyst removal are crucial for avoiding rupture-related morbidity and attaining optimal results.

KEYWORDS: *Cerebral hydatid cyst, Echinococcus, Endemic, Craniotomy.*

INTRODUCTION

Hydatid disease, also called cystic echinococcosis, is a parasitic infection caused by the larvae of *Echinococcus granulosus*. People in endemic areas, including the Middle East, South Asia, North Africa, and portions of South America, are mainly affected. The most frequently involved organs in humans, who serve as unintentional intermediate hosts, are the lungs and liver (in roughly 75% of cases). Yet only 0.5–2% of all documented cases have cerebral hydatid disease, making it an uncommon

manifestation [1,2]. Intracerebral hydatid cysts are rare but, because of their potential severity, warrant a high index of clinical suspicion, particularly in endemic areas.

Symptoms such as headache, seizures, or signs of increased intracranial pressure often appear only after the cyst has enlarged considerably, making the presentation undetectable. The diagnostic difficulty and the significance of imaging in such situations were demonstrated by a noteworthy case involving a 4-year-old child who had a massive 13 cm (about

5.12 in) brain cyst and negative serologic tests [1]. An analysis of 21 pediatric patients with intracranial hydatid disease from a different series at a Moroccan neurosurgical center revealed the infrequency of these cases and their surgical outcomes [2]. According to a global systematic review, recurrence rates can exceed 26.5%, and supratentorial involvement is the most common, indicating that this condition is complex and frequently understudied [3]. For long-term monitoring, surgical planning, and early diagnosis, radiological tools like CT and MRI remain crucial. According to a recent review, imaging is critical in guiding clinicians toward appropriate, life-saving interventions and for detection [4].

Considering these factors, we report a rare case of a giant intracranial hydatid cyst. This case highlights the significance of early detection, radiological assessment, and careful surgical intervention in the treatment of central nervous system hydatid disease.

Case Presentation/Examination

An 11-year-old boy, resident of a rural village in Punjab, Pakistan, presented with complaints of headache and a gradual increase in the size of the head for the past four months; the headache had increased in intensity over the past 1 week, without any associated nausea or vomiting. Although his clinical records did not include objective head circumference measurements, he had obvious macrocephaly with frontal prominence and widened cranial sutures, indicative of elevated intracranial pressure.

There was no history of seizures, altered level of consciousness, abnormal body movements, fever, weakness of limbs, weight loss, trauma, or any surgery. He came from an agricultural family background, but had no cattle at home. Despite this, he lived near stray dogs and local animals, which might have likely exposed him to contaminated food, polluted soil, or dog feces in an endemic rural environment. On examination, he appeared active and well-oriented. His general physical examination

and systemic examinations revealed no abnormalities. His Glasgow Coma Scale score was 15/15, and pupils were bilaterally equal and reactive to light. No neurological deficits were appreciated.

Investigations

On investigations, his hemoglobin was 12.0 g/dL (14.0-17.4 g/dL), leukocyte count was $8.56 \times 10^9/L$ ($5.0-10.0 \times 10^9/L$), and platelet count was $296 \times 10^9/L$ ($140-400 \times 10^9/L$). His renal function tests, liver function tests, serological tests for HIV, Hepatitis B, and Hepatitis C, chest x-ray, echocardiogram, and urine analysis were normal.

MRI of the brain showed an abnormal fluid signal intensity, a large, well-defined cyst in the left parieto-occipital region extending to the high parietal region. The signals appeared near isointense to cerebrospinal fluid on all T1WI, T2WI, and FLAIR sequences. The cyst measured 10.9 x 10.2 x 11.2 cm in transverse, craniocaudal, and anteroposterior dimensions, respectively (shown in Fig. 1). The cystic lesion resulted in significant mass effect on the regional cerebral parenchyma, brainstem, cerebral aqueduct, and ipsilateral lateral ventricle (shown in Fig. 2). The ventricular chain, including the lateral and 3rd ventricles, was moderately dilated, with the 4th ventricle being normal.

Figure 1: a Axial T2-weighted MRI showing a large hyperintense cyst in the left parieto-occipital region with ventricular displacement.

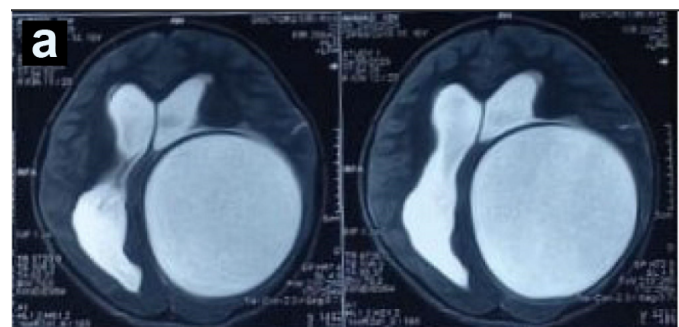
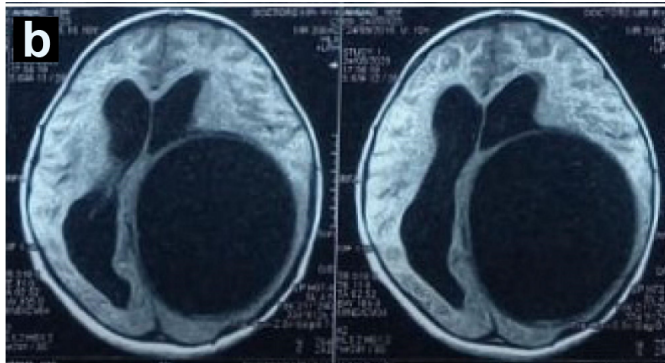


Figure 2: b T1-weighted MRI showing a hypointense cyst consistent with CSF; mass effect on the ipsilateral ventricle is visible.



The differential diagnosis included a neuroglial/neurenteric cyst or a hydatid cyst based on the imaging findings. However, such cases are difficult to differentiate from other cystic lesions, such as cystic gliomas, abscesses, epidermal cysts, large granulomas, and arachnoid cysts. In the thorax and abdominal CT scans, there was no cystic lesion. Based on these imaging findings, a diagnosis of cerebral hydatid was made, and he was admitted to the neurosurgical ward for surgical removal of the cyst.

Treatment, Outcome, and Follow-up

Parietal-occipital craniotomy with dissection of the dura mater was done, thin adhesions were separated, and using the Dowling-Orlando technique, a translucent cyst of 14 x 13 x 8 cm in size was delivered (shown in Fig. 2). The intraoperative dimensions (14 × 13 × 8 cm) differ from the radiologic measurements (10.9 × 10.2 × 11.2 cm) due to variations in measurement planes, expansion following dural opening due to decompression, and the inclusion of adherent tissue and distorted cortical margins during surgical assessment. The gross specimen appeared as a large, intact, fluid-filled cyst with smooth outer walls, consistent with hydatid morphology (shown in Fig. 3). Faint vascular markings and adherent blood-stained fibrinous material were visible on the surface. Surgical retraction was being applied to assess the dissection

plane. Gauze pads were placed circumferentially to isolate the field and minimize the risk of contamination in the event of rupture. The cyst was removed en bloc without rupture, a critical step to prevent intraoperative spillage, anaphylaxis, and recurrence. Histopathological examination was compatible with a hydatid cyst. The postoperative CT scan revealed ample space with no evidence of residual disease (shown in Fig. 4a, Fig. 4b b).

Figure 3: Intraoperative image showing an exposed cerebral hydatid cyst following dura opening with a smooth, glistening surface consistent with an intact laminated membrane and retraction for dissection plane assessment.



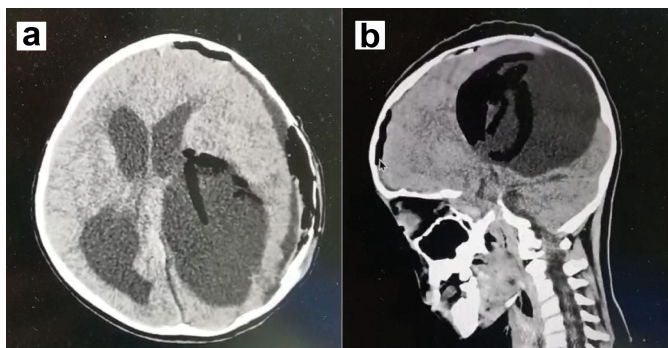
After the neurosurgical intervention, anti-seizure medication (Levetiracetam 500 mg twice a day) was prescribed for the prevention of seizures. In addition, treatment with Albendazole (10 mg/kg) was initiated and continued for 30 days to prevent recurrence of the hydatid cyst, along with routine monitoring of blood counts and liver function tests.

Figure 4: Gross specimen of the intact cerebral hydatid cyst following successful surgical excision.



Figure 5: **a** Axial non-contrast postoperative CT showing a hypodense cavity in the left parieto-occipital region with a small air pocket consistent with pneumocephalus.

b Sagittal CT showing the vertical extent of the hypodense resection cavity with a superior air-fluid level and decompressed brain structures indicating successful cyst removal.



The patient showed significant improvement in headache with no neurological deficits. Later, he was discharged after five days of post-operative hospital stay and followed up monthly for three months,

during which he showed significant improvement. The postoperative anticonvulsant therapy with levetiracetam will last six months, with a screening EEG scheduled at three months and repeat MRI brain imaging planned annually for at least two years to monitor for recurrences.

DISCUSSION

Intracranial hydatid disease results from hematogenous dissemination of *Echinococcus granulosus* larvae after translocation from the gastrointestinal tract, bypassing hepatic and pulmonary filters via the middle cerebral artery distribution. Cyst growth is slow (~1–10 cm/year) and often remains asymptomatic until a substantial mass effect develops [5]. Generally, hydatid cysts larger than 10 cm are categorized as "giant," with reported ranges in the literature often between 10 and 15 cm; our patient's lesion, which measured about 11 cm on MRI, falls within this range.

In pediatric patients, such as our 11-year-old case, the brain's plasticity may mask neurological deficits until the cyst becomes very large, which likely explains the absence of neurological deficits in our patient despite marked mass effect and moderate hydrocephalus. MRI and CT are essential diagnostic tools, as hydatid cysts typically appear as well-defined, spherical lesions with signal intensity similar to CSF and no enhancement or perilesional edema unless complicated [6]. However, neuroimaging alone cannot rule out other cystic lesions. Serologic testing, including ELISA or indirect hemagglutination, has low sensitivity in isolated cerebral cases and often yields false negatives [1]. In pediatric cases reported in the literature, serologic positivity usually correlates with extracranial involvement. Histopathological confirmation remains the definitive diagnostic standard.

Surgical removal is the primary treatment and employs techniques such as Dowling–Orlando or Arana–Iniguez via hydrodissection to preserve cyst integrity [2]. Cyst rupture during surgery poses

serious risks, including intracranial dissemination, anaphylaxis, and recurrence. An extensive multicenter study (76 cases over 22 years) found that intact cyst removal was successful in approximately 74% of cases, while recurrence occurred in 25%. Notably, rupture was associated with a 5.3% mortality rate due to anaphylaxis [2]. Although postoperative mood disturbances have been reported in some cases, none were observed in our patient during follow-up.

For medical therapy, albendazole should be administered for at least three months preoperatively and continued for at least one month postoperatively if viable scoleces are identified, to reduce the risk of residual cysts and recurrence [8]. It should be noted that the best course of action for solitary intracranial cysts remains under debate, and the majority of evidence supporting the use of albendazole in cerebral hydatid disease derives from hepatic hydatid disease.

Our case is consistent with others from developing countries, where intracranial cysts often reach substantial sizes (10–15 cm) before diagnosis, commonly presenting with symptoms such as headache or macrocephaly, yet with minimal neurological deficits. These findings align with other cases in the literature, including a 17-year-old with bilateral giant cysts and a 9-year-old with intraventricular expansion [5], emphasizing the silent but aggressive nature of these lesions and the effectiveness of complete surgical removal coupled with medical management.

Preventive strategies remain crucial and include controlling stray dog populations, routine deworming, and meat inspection, all supported by data from endemic regions [1,9]. The prognosis is generally excellent if surgery is completed without cyst rupture, with most patients achieving full recovery and no recurrence during follow-up [9].

The relatively short follow-up time of three months, which is insufficient to accurately assess long-term

recurrence risk, is a limitation of this report. As a result, ongoing surveillance is necessary.

CONCLUSION

This case emphasizes several vital lessons: clinicians must maintain high suspicion for hydatid disease in children from endemic areas who present with progressive headaches or macrocephaly; detailed preoperative imaging and careful surgical planning are critical for patient outcomes; multidisciplinary collaboration among neurosurgeons, radiologists, and infectious disease specialists enhances surgical safety and postoperative care; and finally, attention to psychosocial health is necessary in the recovery phase, as postoperative mood changes may indicate underlying neuroinflammation or stress responses.

Key Clinical Message:

1. Cerebral hydatid disease, though rare, should be suspected in children from endemic regions presenting with progressive neurological symptoms.
2. Successful management requires early recognition, intact cyst excision with meticulous technique, and vigilant postoperative follow-up to prevent recurrence and complications.

Acknowledgement

None.

Study Approval Statement

Not required.

Statement of Ethics

This study complies with all relevant ethical standards.

Consent to Participate

Written informed consent was obtained from the patient's parent for the publication of this case

report. A copy of the written consent is available for review by the Editor-in-Chief of this journal upon request.

Consent to Publish Statement

The authors affirm that the patient's parent provided informed consent for publication of the images included in this case report.

Conflict of Interest Statement

The authors have no conflicts of interests to declare.

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Data availability Statement

All data generated or analyzed during this study are included in this published article. The data supporting the findings of this study have been de-identified to ensure the participant's confidentiality and privacy.

Author Contributions

C. S. and S.S. contributed to the conception, data gathering, literature review, and writing the first draft of the manuscript; A. B., R. T., A. A. and E. S. played a role in writing the draft, optimizing the grammar, literature review, and English language editing, extensively reviewing the first draft, and substantially contributing to revising it critically for important intellectual content in the final manuscript. A. A. and A. B. prepared the images. All authors accepted the accountability of the research work authenticity and agreed to publish the final version.

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