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Case Report

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Progressively worsening headache due to a giant arachnoid cyst in a teenager: a case report

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ABSTRACT

Headache is one of the most common neurological complaints in the pediatric population and encompasses a broad differential diagnosis. Although primary headache disorders constitute the majority of cases, secondary etiologies—including intracranial space-occupying lesions—must be considered, particularly in patients with persistent, progressive, or treatment-refractory symptoms. Arachnoid cysts are benign, cerebrospinal fluid (CSF)-filled, extra-axial lesions that are typically congenital in origin and arise from developmental abnormalities of the arachnoid membrane. While most arachnoid cysts are asymptomatic and incidentally detected, symptomatic cases may present with chronic headache due to mass effect or impaired CSF circulation. They account for less than 1% of intracranial space-occupying lesions. We report the case of a young female patient with long-standing, treatment-resistant headache without focal neurological deficits. Neuroimaging revealed an intracranial arachnoid cyst exerting mass effect consistent with increased intracranial pressure. Given the persistence of symptoms despite conservative management, surgical intervention was undertaken. The patient underwent cystoperitoneal shunt placement. Postoperative follow-up demonstrated marked clinical improvement, with near-complete resolution of headache severity and frequency. Radiological evaluation confirmed a significant decrease in cyst size and alleviation of mass effect. This case underscores the importance of comprehensive evaluation for secondary causes in pediatric patients presenting with refractory headache. Although arachnoid cysts are frequently incidental findings, symptomatic lesions may require surgical management. Cystoperitoneal shunting can provide favorable clinical and radiological outcomes in appropriately selected patients. Early recognition and timely intervention are essential to prevent prolonged morbidity.

Keywords: Arachnoid cyst, headache, shunt

INTRODUCTION

Arachnoid cysts are benign, cerebrospinal fluid (CSF)-filled, extra-axial cystic lesions lined by flattened arachnoid cells and a collagenous membrane.[1] They are thought to arise from developmental splitting or duplication of the arachnoid membrane during embryogenesis. These lesions are typically located between the cortical surface and the skull base or within the arachnoid membrane itself, one of the three meningeal layers enveloping the central nervous system.[2]

The clinical presentation of arachnoid cysts is heterogeneous and largely dependent on cyst size, anatomical location, and their effect on adjacent neural structures and CSF circulation. Although the majority remain clinically silent and are incidentally identified on neuroimaging, symptomatic lesions may result from progressive cyst enlargement, mass effect, distortion of surrounding parenchyma, or obstruction of CSF pathways leading to elevated intracranial pressure.[3] Furthermore, secondary complications such as intracystic or subdural hemorrhage may precipitate acute neurological deterioration.[4]

Reported clinical manifestations include chronic headache, epileptic seizures, dizziness, gait ataxia, nausea and vomiting, tinnitus, diplopia, visual disturbances, and other signs consistent with increased intracranial pressure.[5]

In this report, we describe a young female patient presenting with long-standing, refractory headaches who was subsequently diagnosed with an intracranial arachnoid cyst. Given the persistence of symptoms and radiological evidence of mass effect, a cystoperitoneal shunt was placed to achieve cyst decompression and restoration of CSF dynamics. The patient demonstrated complete resolution of symptoms in the early postoperative period, accompanied by radiological

evidence of cyst volume reduction and alleviation of mass effect.

CASE

A 13-year-old female patient was referred to our neurosurgery clinic with complaints of persistent headache from the pediatric clinic. The patient had experienced recurrent pain attacks for approximately four years, which had progressively worsened over the preceding two months. The pain was predominantly right-sided, severe enough to awaken her from sleep, and was particularly pronounced during the morning and evening hours. The headaches occurred in recurrent attacks, each lasting at least 30 minutes. Over time, both the frequency and duration of the attacks increased. During headache attacks, pain intensity varied between 8 and 10 points on the Visual Analog Scale (VAS). According to the ICHD-3 criteria, the patient's symptoms were consistent with cluster headache. Initially, partial relief was achieved with 500 mg of paracetamol; however, as the symptoms progressed, the patient began taking an additional 100 mg of flurbiprofen. Despite this escalation in analgesic therapy, adequate pain control could not be achieved. Furthermore, the patient's academic performance gradually declined over the course of the illness. The patient did not receive any preventive or advanced medical therapy or other medical treatment at our clinic. Information regarding the medications used and their dosages was obtained from the patient's medical history and review of the medical records.

She denied associated symptoms such as nausea, vomiting, visual disturbances, or seizures. There was no history of head trauma, central nervous system infection, or previous neurosurgical intervention. Her personal and family medical histories were unremarkable, with no known chronic systemic diseases.

On admission, the patient was in good general condition and fully conscious, with a Glasgow Coma Scale (GCS) score of 15. Pupillary examination revealed bilaterally reactive pupils with intact direct and consensual light reflexes. Cranial nerve examination was normal. Motor and sensory examinations were within normal limits, and cerebellar function tests demonstrated no abnormalities. No focal neurological deficits were identified. Ophthalmological evaluation, including fundoscopy, revealed no abnormality despite radiological evidence of significant mass effect.

Non-contrast brain magnetic resonance imaging (MRI) demonstrated a large extra-axial cystic lesion exhibiting signal characteristics identical to CSF on all sequences. The lesion extended along the right fronto-parieto-temporal convexity and reached the Sylvian fissure. The maximal axial dimensions measured approximately 108×66 mm. Imaging findings were consistent with a giant arachnoid cyst. Significant mass effect was observed, including compression of the adjacent right fronto-parieto-temporal lobes and a 10-mm leftward midline shift. Additionally, a retrocerebellar cystic dilatation measuring approximately 26 mm was noted (**Figure 1**). The lesion was classified as Galassi type III.

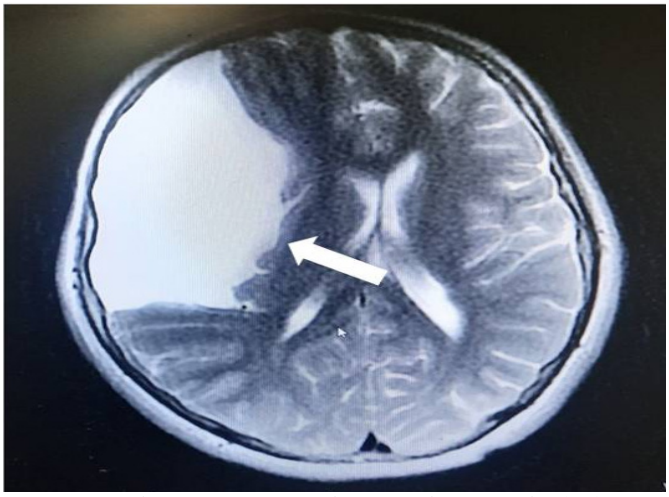

Figure 1. Axial non-contrast cranial computed tomography (CT) image obtained with a 2.5-mm slice thickness demonstrates a right-sided, well-defined extra-axial hypodense lesion with attenuation characteristics consistent with cerebrospinal fluid, compatible with an arachnoid cyst (white arrow). The lesion produces significant mass effect, resulting in a leftward midline shift and compression with effacement of the ipsilateral lateral ventricle.

Subsequently, a non-contrast cranial computed tomography (CT) scan was obtained, revealing a well-demarcated hypodense lesion with attenuation values consistent with CSF in the right frontal region, measuring up to 10 cm in maximal diameter (**Figure 2**). These findings further supported the diagnosis of an arachnoid cyst.

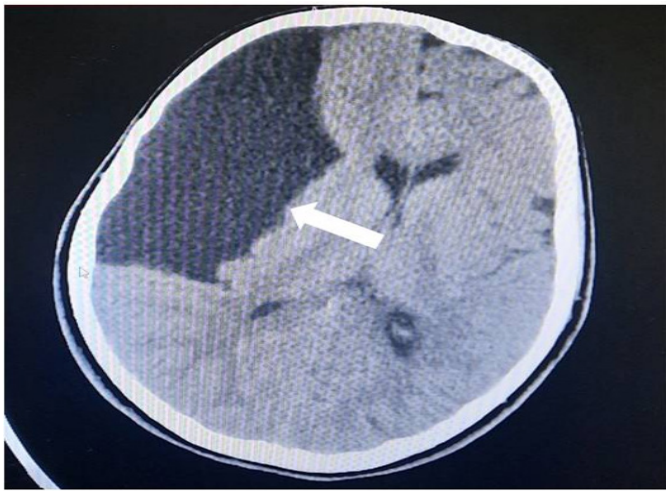

Figure 2. Axial T2-weighted magnetic resonance imaging (MRI) obtained with a 3-mm slice thickness demonstrates a right-sided, well-circumscribed extra-axial lesion exhibiting cerebrospinal fluid–like signal intensity, consistent with an arachnoid cyst (white arrow). The lesion exerts significant mass effect, resulting in a leftward midline shift and effacement of the ipsilateral lateral ventricle, with associated compression of the adjacent cortical and subcortical structures.

Given the progressive nature of the patient's symptoms (which were consistent with increased intracranial pressure), the substantial mass effect with midline shift, and the lack of satisfactory response to conservative treatment, surgical intervention was recommended. After evaluating available treatment options, cystoperitoneal shunting was selected because it was considered the most appropriate strategy to achieve continuous cyst decompression and rapid reduction of intracranial mass effect. The patient underwent cystoperitoneal shunt placement. A burr hole was created in the right frontoparietal region overlying the cyst. The catheter

was connected to a medium-pressure valve system equipped with an automatic antisiphon device to minimize the risk of overdrainage. No intraoperative complications occurred.

Postoperative neuroimaging demonstrated a marked reduction in cyst volume and significant improvement in the midline shift (**Figure 3, 4**). Compared with preoperative imaging, the midline deviation was substantially decreased. Clinically, the patient experienced significant relief of headache symptoms even in the early postoperative period. She remains under regular clinical and radiological follow-up for 3 years.

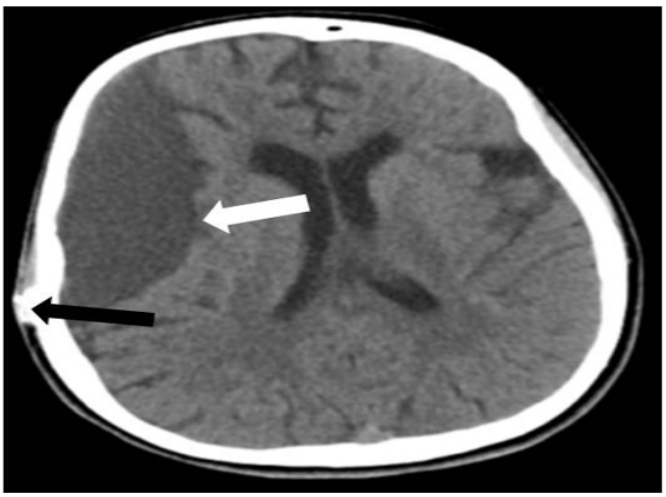

Figure 3. Postoperative axial computed tomography (CT) image obtained three days after surgery with a 2.5-mm slice thickness demonstrates partial reduction in the size of the lesion and improvement of the midline shift. The white arrow indicates the residual cyst, while the black arrow points to the shunt pump.

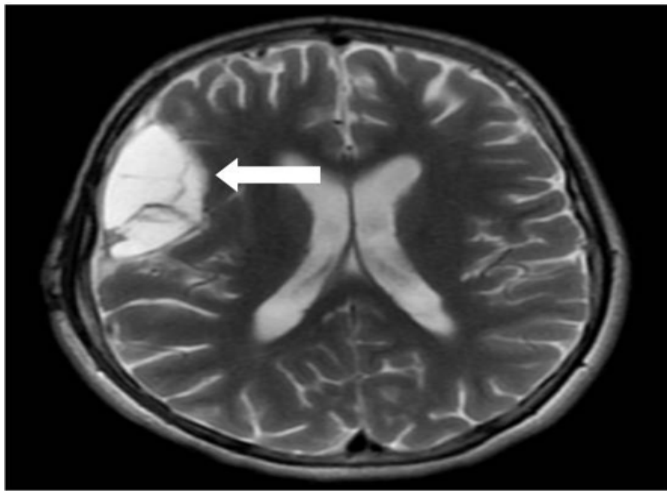

Figure 4. Axial T2-weighted magnetic resonance imaging (MRI) obtained three months postoperatively with a 3-mm slice thickness demonstrates marked reduction in the volume of the arachnoid cyst (white arrow). There is resolution of mass effect, with re-expansion of the previously compressed ipsilateral lateral ventricle and restoration of normal cortical and subcortical architecture.

DISCUSSION

Although headache is a common complaint during childhood, it is frequently underestimated by caregivers; nevertheless, it may occasionally represent an underlying serious intracranial pathology.[6] Headaches occurring at an early age, those that awaken the child from sleep or are most pronounced in the early morning hours, the "worst headache ever experienced," or headaches accompanied by seizures, visual disturbances, focal motor deficits, or balance impairment should prompt careful evaluation and comprehensive neuroimaging.[7] In the present case, none of these red-flag features were observed. The neurological examination was entirely normal, and the patient's sole complaint was progressively worsening headache.

The etiology of pediatric headaches is broadly classified into primary and secondary categories.[8] Primary headache disorders include migraine, tension-type headache, and cluster headache syndromes.[8] In contrast, secondary headaches arise from structural, vascular, infectious, inflammatory, or neoplastic processes involving the central nervous system.[8] Among structural causes, arachnoid cysts represent a rare but clinically relevant entity.[9] Arachnoid cysts account for approximately 1% of all intracranial space-occupying lesions and are most commonly located in the middle cranial fossa, followed by the suprasellar and posterior fossa regions.[9] Although frequently asymptomatic and incidentally detected, these lesions may become symptomatic depending on their size, anatomical location, and associated mass effect or disturbance of CSF circulation.[9]

The optimal management of arachnoid cysts remains a matter of debate. Surgical intervention is generally reserved for patients with signs of elevated intracranial pressure, progressive cyst enlargement, or radiological evidence of significant compression of adjacent neural structures.[10] Microsurgical total excision or fenestration offers the theoretical advantage of definitive treatment without shunt dependency.[11] However, complete resection may not be feasible in all cases due to anatomical constraints, risk of recurrence, and the potential for injury to adjacent eloquent brain tissue.[11]

Minimally invasive approaches, including endoscopic ventriculocystostomy or cyst fenestration, have gained popularity due to reduced operative morbidity and shorter hospitalization periods.[12,13] Nevertheless, procedure-related complications—such as intraventricular or intracerebral hemorrhage—have been reported.[12] Endoscopic techniques are generally considered safe and effective particularly when cyst anatomy permits adequate fenestration and CSF diversion without the need for permanent shunting.[12] However, recurrence and the need for reoperation remain potential limitations with this procedure.[12] In addition, as is the case in our institution, this technique may not be available in all hospitals.

In the present case, cystoperitoneal shunt placement was preferred due to the large size of the cyst, the presence of a 10-mm midline shift, and the need for reliable long-term decompression. Considering these radiological findings, shunt placement was considered the most appropriate treatment strategy. The early postoperative course was uneventful, with marked radiological and clinical improvement. The use of a medium-pressure shunt system equipped with an automatic antisiphon device allowed controlled drainage, thereby minimizing the risk of overdrainage and subsequent subdural hematoma formation.

CONCLUSION

Persistent or treatment-resistant headache in the pediatric population warrants thorough evaluation, even in the absence of focal neurological deficits. Significant intracranial mass lesions may be present despite a normal neurological examination. Although the optimal management of symptomatic arachnoid cysts remains controversial and should be individualized according to cyst characteristics and patient factors, cystoperitoneal shunting represents a technically straightforward, widely applicable, and effective surgical option. In appropriately selected patients, it can provide rapid symptom relief, significant reduction of cyst volume, and favorable radiological outcomes.

ETHICAL DECLARATIONS

Informed Consent

Informed consent was obtained from the legal guardians of the pediatric patient described in this report. Where developmentally appropriate, assent was also sought from the child. The inclusion of vulnerable populations in this study adhered to national and international ethical guidelines. Extra care was taken to ensure voluntary participation, understanding, and protection of participant dignity and autonomy.

Peer Review Process

This report underwent external peer review.

Conflict of Interest

The authors declare no conflicts of interest.

Financial Disclosure

This case report did not receive any financial support.

Author Contributions

Concept: HA, EC, SÖ; Design: HA, EC, SÖ; Control: HA; Resources: EC, SÖ, RNT; Materials: EC, SÖ, RNT; Data Collection and/or Processing: EC, SÖ, RNT; Analysis and/or Interpretation: EC, RNT, SÖ; Literature Review: HA, EC, RNT; Writing the Article: HA, EC, RNT; Critical Review: SÖ.

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