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Nontraumatic subdural hemorrhage due to arachnoid cyst rupture

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Arachnoid cysts (ACs) are benign congenital lesions that are often detected incidentally during imaging studies performed for various etiological reasons. These cysts are typically asymptomatic, and in most cases do not require surgical intervention. Although ACs are commonly encountered, they are rarely complicated by intracystic hemorrhage or subdural hematoma. In this report, we present a case of a nontraumatic rupture of an AC, which remained undiagnosed until the age of 52 years.

Keywords:

Arachnoid cyst, headache, rupture, subdural hemorrhage

Introduction

Arachnoid cysts (ACs) are cerebrospinal fluid (CSF)-filled cystic structures located between the layers of the arachnoid membrane. They are congenital lesions resulting from a developmental defect in the formation of the arachnoid membrane during embryogenesis. ACs are most commonly located in the anterior and middle cranial fossa or the retrocerebellar region. They are reported to occur in approximately 2.6% of the pediatric population and 1.4% of the adult population, with a higher prevalence in males.^[1,2]

Although rare, ACs may rupture, leading to complications such as subdural hematoma or subdural hygroma. Here, we present the case of a patient who was admitted to our emergency department with a non-traumatic headache and was diagnosed with a subacute subdural hematoma and acute intracystic hemorrhage.

A 52-year-old man with no known systemic diseases presented to the emergency department with a headache that had started 4 days prior. The patient denied any history of head trauma, as well as any Valsalva maneuvers, constipation, or head shaking that could increase intracranial pressure. On examination, his blood pressure was 138/91 mmHg, pulse rate was 66 beats per minute, oxygen saturation was 99% on room air, and body temperature was 36°C. He was conscious, cooperative, and fully oriented, with a Glasgow Coma Scale (GCS) score of 15. The patient's neurological examination was completely normal; no cranial nerve palsy, hemiparesis, hypoesthesia, dysarthria, ataxia, or neck stiffness was detected. The physical examination findings of the other systems were also normal. The patient was not taking any medication and supplements and had no history of prior surgical operations or allergies. The patient had no genetic diseases and stated that none of their family members had an intracranial AC. The patient reported that he had never experienced such a

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prolonged and severe headache before, and that the pain was unresponsive to analgesics. A noncontrast brain computed tomography (CT) scan was performed.

CT imaging revealed a large AC located anterior to both temporal lobes, extending around the brainstem and pons. Within the cyst located in the left temporal region, a hyperdense area suggestive of acute intracystic hemorrhage was noted, along with signs of subacute hemorrhage [Figure 1a]. The hemorrhage had extended into the subdural space of the left temporal region, resulting in a 1 cm midline shift [Figure 1b].

Laboratory findings, including renal functions, liver functions, hemoglobin, electrolytes, and coagulation parameters, were within normal limits (INR was 0.97 [reference range: 0.8–1.2], creatinine was 1.15 mg/dL [reference range: 0.7–1.3], and platelet count was 247 K/µL [reference range: 140–400]). A neurosurgery consultation was requested for the patient. The neurosurgeon recommended surgical drainage. However, since our hospital is a secondary-level facility and was undergoing extensive renovations at that time, the patient was referred to a tertiary-level hospital. The patient underwent successful surgical drainage. After being monitored in the intensive care unit for 1 day, he was followed in the neurosurgery ward for 4 days and was discharged without any neurological sequelae. Written informed consent approval was obtained from the patient.

Discussion

Although ACs are typically detected during childhood, they may remain undiagnosed into adulthood in asymptomatic individuals. Because they are often silent, ACs are usually discovered incidentally during cross-sectional neuroimaging performed for unrelated etiological investigations.^[1,3] The prevalence of ACs has been reported as approximately 1.4% in adults and

0.4% in children.^[4,5] They may become symptomatic depending on their size and anatomical location. Symptoms can include visual impairment, nausea and vomiting, macrocephaly, cranial nerve palsies (including the vagus, oculomotor, trochlear, and abducens nerves), facial nerve palsy, trigeminal neuropathy, hemifacial spasm, sensorineural hearing loss, vertigo, and eighth cranial nerve neuropathy. While asymptomatic ACs generally do not require intervention, surgical treatment may be necessary in cases involving mass effect, focal neurological deficits, seizures, or intractable headache.^[1,3,6]

One of the most comprehensive reviews on AC rupture is by Massimi *et al.*^[7] In addition to analyzing 16 cases from their own clinic, the authors conducted a literature review encompassing 430 patients. Most cases in this series occurred in pediatric and adolescent populations, with an annual rupture risk estimated at 0.04%. Approximately 10% of children with an AC may experience cyst rupture. Notably, 52% of cases had a documented history of head trauma. Although the precise mechanism of AC rupture remains unclear, proposed contributing factors include direct trauma to the temporal bone, sudden head movements, or rupture of bridging veins. Minor, unreported trauma, Valsalva maneuvers, or physical exertion has also been suggested as potential triggers. In the reviewed cases, subdural collections (acute/chronic subdural hematoma or hygroma) were reported in 80.6%, intracystic hemorrhage in 6.5%, and epidural hemorrhage in 2.5%.

In our case, although there was no history of obvious trauma, the patient noted that the headache began following a long walk. Headache and vomiting are the most commonly reported symptoms of AC rupture. Some cases may present with papilledema or vision loss, while focal neurological deficits are less frequent. Among the 430 cases in Massimi *et al.*'s review, 10% presented with focal deficits and 6% with seizures.^[7]

Hall *et al.* conducted a 12-year retrospective review of 550 patients diagnosed with ACs.^[2] Rupture occurred in 14 cases (2.5%), all of whom had a GCS score of 15 on admission. Most presented with headache and nausea. A history of trauma was reported in 64% of the cases. Subdural hematoma was observed in eight patients, hygroma in four, and intracystic hemorrhage in two. Notably, only one of these patients had a prior diagnosis of AC; the remaining 13 were unaware of their cyst until rupture occurred. Similarly, our patient had no prior knowledge of the AC.

CT is sufficient to visualize fluid-filled ACs. ACs appear as thin-walled, well-circumscribed lesions with a density similar to CSF and do not enhance with contrast. Lesions located near the cortical surface may exhibit a biconvex

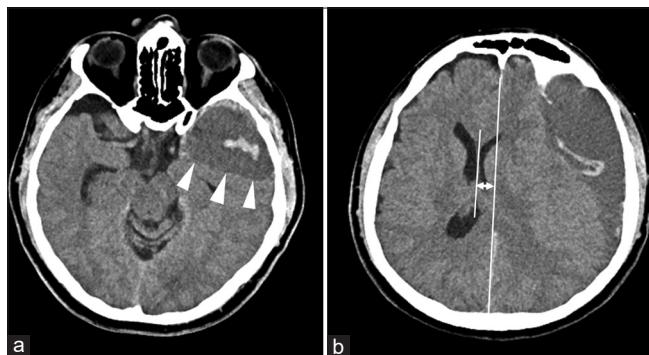


Figure 1: (a) Hyperdense areas consistent with acute hemorrhage, along with regions of subacute bleeding, are observed within the arachnoid cyst located in the left temporal region. (b) A subacute subdural hematoma containing foci of acute bleeding is seen, causing compression of the left lateral ventricle and resulting in a midline shift

or semicircular shape with a characteristic straight inner margin. Hemorrhages occurring within the cyst or the subdural space may appear isodense or hyperdense relative to brain parenchyma, depending on the age of the bleed.^[1,6]

Conclusion

In patients with ACs presenting to the emergency department with headache, intracranial hemorrhages requiring surgical intervention may be observed, even in the absence of focal neurological deficits.

Author contribution statement

HKE: Conceptualization, Investigation, Methodology, Resources, Supervision, Writing – original draft, Writing – review and editing
EA: Conceptualization, Investigation, Methodology, Resources, Supervision, Writing – original draft, Writing – review and editing
MCG: Conceptualization, Investigation, Writing – original draft, Writing – review and editing. SÖ: Conceptualization, Investigation, Writing – original draft, Writing – review and editing.

Conflicts of interest

None Declared.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal.

The patient understand that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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