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Case Report

Spontaneous resorption of a convexity arachnoid cyst associated with intracystic hemorrhage and subdural hematoma: A case report

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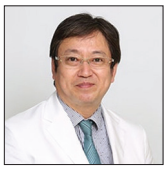

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ABSTRACT

Background: Intracranial arachnoid cysts (ACs) are developmental anomalies usually filled with cerebrospinal fluid (CSF), rarely resolving throughout life. Here, we present a case of an AC with intracystic hemorrhage and subdural hematoma (SDH) that developed after a minor head injury before gradually disappearing. Neuroimaging demonstrated specific changes from hematoma formation to AC disappearance over time. The mechanisms of this condition are discussed based on imaging data.

Case Description: An 18-year-old man was admitted to our hospital with a head injury caused by a traffic accident. On arrival, he was conscious with a mild headache. Computed tomography (CT) revealed no intracranial hemorrhages or skull fractures but an AC was seen in the left convexity. One month later, follow-up CT scans showed an intracystic hemorrhage. Subsequently, an SDH appeared then both the intracystic hemorrhage and SDH gradually shrank, with the AC disappearing spontaneously. The AC was considered to have disappeared, along with the spontaneous SDH resorption.

Conclusion: We present a rare case where neuroimaging demonstrated spontaneous resorption of an AC combined with intracystic hemorrhage and SDH over time, which may provide new insights into the nature of adult ACs.

Keywords: Arachnoid cyst, Spontaneous resorption, Subdural hematoma

INTRODUCTION

Intracranial arachnoid cysts (ACs) are benign congenital entities filled with cerebrospinal fluid (CSF) but may also contribute to risks of subdural hematoma (SDH) development after head trauma.[14] They occur at a prevalence of 2.6% in pediatric patients and 1.4% in adults, favoring males. Natural AC pathogenesis is poorly understood and, in 95% of cases, remains asymptomatic throughout life.[11] However, Al-Holou *et al.* reported that, in pediatric patients, 11.7% of ACs spontaneously decreased in size with a complete resolution of 3.6%, in contrast to spontaneous decreases in 0.9% of adult patients.[1,2] This report describes a case in which a convexity AC, incidentally discovered after head trauma, was accompanied by intracystic hemorrhage and SDH formation before disappearing spontaneously. We present neuroimaging changes, showing

intracystic and SDH development (plus AC resorption over time), and discuss the related mechanisms.

CASE DESCRIPTION

An 18-year-old man experienced a high-speed automobile crash and was transported to our hospital by ambulance. On admission, the patient was alert, with no neurologic deficits, but reported a mild headache and dizziness. He had no remarkable medical or family histories.

Computed tomography (CT) revealed a low-density cystic lesion with bone thinning on the left convexity [Figure 1]. No intracranial hemorrhages or skull fractures were observed but, the next day, persistent headaches led to magnetic resonance imaging to assess for cerebral contusions and examine the cystic lesion. After assessment by experienced neurologists (no AI assistance), the absence of contusions and hemorrhaging was confirmed and the cyst (isointensive with the CSF) was diagnosed as an incidentally discovered AC on the convexity [Figure 2]. The patient was diagnosed with a cerebral concussion and admitted to the hospital for follow-up. The next day, there were no new neurological symptoms, the headache disappeared, and the patient was discharged.

Approximately 1 month post-injury, the patient returned to the outpatient clinic with a mild headache. At that time, the AC appeared as a high-density lesion on CT images, suggesting intracystic hemorrhaging [Figure 3a]. The patient underwent routine imaging follow-up without surgical treatment because the symptoms were mild, with little mass effect on the brain. Furthermore, a CT scan 6 weeks after the injury revealed a denser hemorrhage and the presence of an SDH [Figure 3b], but AC size appeared slightly reduced. CT 8 weeks after injury showed further reductions in SDH and cyst size [Figure 3c], while CT 10 weeks after injury showed that both had nearly disappeared [Figure 3d]. At 6 months post-injury, follow-up outpatient visits were completed with no symptoms or complications.

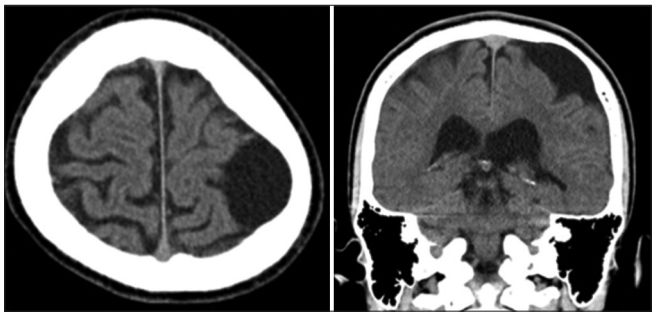

Figure 1: Computed tomography on admission, showing a hypodense, cystic lesion with bone thinning in the right convexity.

DISCUSSION

ACs are often found incidentally on neuroimaging and their spontaneous enlargement or disappearance is rare.[1,2] However, in our case, the incidentally-found AC developed both intracystic hemorrhaging and SDH before spontaneously disappearing. We thus considered the AC resorption as a result of head trauma, a result seen by Haddad *et al*. who reported that 16 of 59 resorption cases were spontaneous after head trauma. Meanwhile, 33 of those cases had no clear mechanism for resorption.[4] As there are no treatment guidelines for incidentally discovered AC in adults, understanding the mechanisms of morphologic changes in AC after head trauma would be essential in establishing an appropriate treatment strategy.

It is generally thought that spontaneous resorption may occur due to ruptures of the cyst wall and drainage of cyst components into the subdural space.[4] Since this wall is multiple layers of collagen-anchored arachnoid cells, minor head trauma, excessive breathing, sports activities, or even coughing may cause the cyst wall to rupture.[4,11] Such minor events are often overlooked and the AC may spontaneously disappear.[6,9] It has also been suggested that the size change mechanism involves the influx of CSF through a one-way valve, as seen in a case of cyst shrinkage after fibrin glue injection for a sacral AC.[16] Thus, inflammation caused by

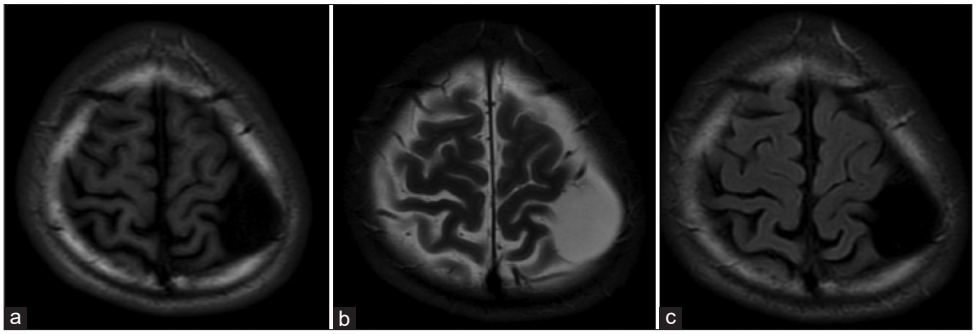

Figure 2: T1- (a), T2- (b), and fluid-attenuated (c) magnetic resonance images on admission, showing an extra-axial cystic lesion in the left convexity. No cerebral contusions or hemorrhages are visible.

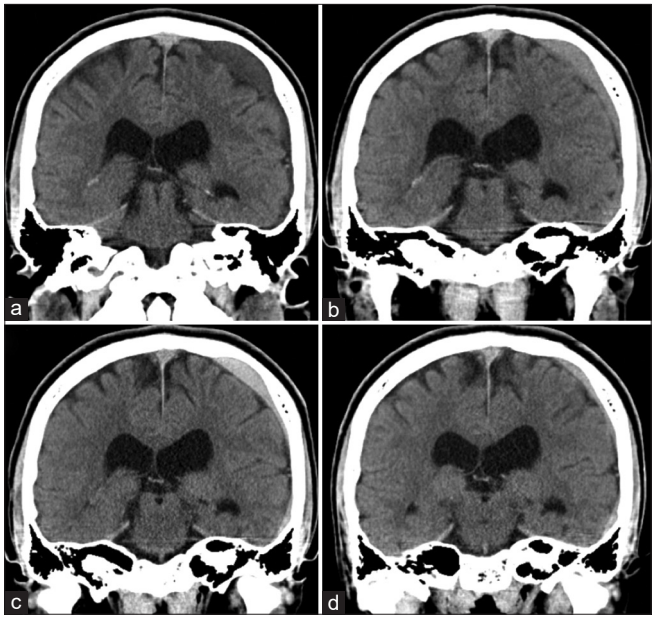

Figure 3: Computed tomograms, showing changes in arachnoid cysts (ACs), the intracystic hemorrhage, and subdural hematoma (SDH) over time (a: 4 weeks, b: 6 weeks, c: 8 weeks, and d: 10 weeks after injury). The intracystic hemorrhage occurred first and the cystic component spread into the subdural space forming an SDH, where it was gradually absorbed while the AC disappeared simultaneously.

meningitis, hemorrhage, or tumors may close this one-way valve and shrink the AC.[6] Another theory is that the AC shrinks due to intra- and extra-cystic changes in the osmotic gradient.[4] However, since the present case was hemorrhagic, the mechanism of disappearance may differ from such simple cyst wall ruptures, one-way valve closure, or loss of osmotic difference.

It is well known that AC is sometimes complicated by SDH or subdural hygroma, with Wester and Helland reporting that 4.6% of ACs lead to the development of subdural or intracystic hematomas.[15] Although the pathophysiology associated with intracystic hemorrhage or SDH is unknown, two theories regarding hematomas exist. First, reports about small vessels between the dura mater and the AC suggest that these vessels may rupture, causing intracystic bleeds or SDH, and, because the AC is less compliant than the normal brain, bridging veins or unsupported vessels over the wall are therefore prone to hemorrhage and rupture.[8,10,13] According to a second theory, the wall secretes fluid, which gradually elevates intracystic pressure, causing that wall to rupture in areas without vessels, and resulting in subdural hygroma and SDH.[12]

When an AC is accompanied by hemorrhaging, the hemorrhage can be intracystic only or with SDH; however, the mechanisms of such difference are unknown. Iaconetta *et al*. reviewed 37 ACs with intracystic hemorrhages, most of which (21/37 cases; 57%) were accompanied by SDHs.[5] Intracystic hemorrhages may have occurred due to vascular damage after trauma, with mixed blood and CSF from the damaged AC wall flowing into the subdural space and forming an SDH. There are two possibilities for such blood flow into the subdural space: either the wall could have been damaged simultaneously with the blood vessels at the time of injury or the AC could have ruptured due to increased intracystic pressure associated with the hemorrhage without wall damage at the time of injury. In our case, we speculate that minor trauma to the AC wall blood vessels caused an intracystic hemorrhage. As a result of this localized bleeding, pressure increases in the AC caused an eventual rupture, resulting in SDH formation. This theory is supported by the disappearance of the AC after subsequent, spontaneous resorption of the SDH.

Once formed, SDHs often increase in size and become symptomatic. Patients with severe brain atrophy, liver and blood diseases, and those taking antiplatelet drugs are most likely to develop symptoms and usually require surgery. In our case, the patient was young and without brain atrophy; the SDH and AC resolved without surgical intervention. Bristol *et al*. reported that rupture of the middle fossa AC is more likely to be symptomatic, while such ruptures in other areas tend to be asymptomatic and resolve spontaneously.[3] This may be related to the small volume of the middle fossa, where even small hemorrhages tend to have a mass effect. On the other hand, in the asymptomatic present case, the blood and CSF mix draining from the AC spread thinly over a wide area of the cranial convexity, resulting in only a minor mass effect on the brain.

Most ACs are stable throughout life and do not require surgical treatment but, in some cases, they can be associated with hematomas and require surgical intervention.[7] As the mechanism of asymptomatic to symptomatic transition (and surgical indication) remains unclear, careful vigilance in patients after head trauma is paramount.

This report is a case report, so more cases are needed to estimate the guideline for treating the cases like our case. Our case may help the future treatment of ACs.

CONCLUSION

We presented a rare case of AC with intracystic hemorrhage and SDH formation that disappeared spontaneously over time, demonstrated by neuroimaging. This case may provide new insight into the nature of hematoma development associated with ACs.

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Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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