Arachnoid Cyst-associated Chronic Subdural Hematoma: A Case Report

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ABSTRACT

Chronic subdural hematomas (cSDH), a common condition tends to develop several weeks after head trauma in patients with atrophic brains, such as the elderly. Arachnoid cysts (AC) are congenital intracranial lesions filled with cerebrospinal fluid (CSF). They have a benign natural history in most patients, and are often found incidentally on intracranial images performed after head trauma. On rare occasions, ACs are complicated by cSDHs. Herein, we present a case involving a patient with AC-associated cSDH.


Key words: Arachnoid cyst, Chronic subdural hematoma, Burr hole drainage, Subdural effusion, Head trauma

Introduction

Chronic subdural hematomas (cSDH) often develop several weeks subsequent to head trauma in patients with atrophic brains (e.g. the elderly).[1] They can evolve from acute subdural hematomas or subdural effusions (SDE). Currently, cSDHs are treated using burr hole drainage with placement of a subdural drain kept in place for 24 to 48 hours. Arachnoid cysts (AC) are congenital intracranial lesions filled with cerebrospinal fluid (CSF), arising from splitting of arachnoid membrane.[2] Most ACs that become symptomatic do so in early childhood. Presenting symptoms include those of intracranial hypertension, seizure, hemorrhage, focal skull protrusion, and endocrine with visual dysfunction in suprasellar cysts. In most adolescent and adult patients, ACs have a benign natural history, and they are often found incidentally on intracranial images performed to evaluate patients with headache or head trauma. AC is rarely complicated by cSDH. In this study, we report a

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case of AC-associated cSDH developing weeks later after brain concussion. We describe and discuss the clinical course of this patient who we successfully treated using burr hole drainage.

Case Report

In this study, a 64 year-old unconscious male traffic accident victim was brought to our hospital’s emergency department (ED). Initial computed tomography (CT) scan of the head showed no intracranial hematoma. However, a hypodense cyst with a maximal diameter of 3.7cm was found incidentally in the right parietal lobe (Figure 1). The patient denied any past history of brain injury or stroke. The patient was found to have no focal neurological deficit after regaining consciousness at the ED. Brain concussion and an incidentally-found AC was diagnosed. After 12-hour observation, the patient was prescribed medications for symptomatic treatment upon discharge and his family members were given instructions for close observation at home.

In the first week, the patient complained of headache and dizziness, which improved after medical treatment in an outpatient clinic. He remained neurologically intact. However, left side numbness and clumsiness began to develop one month later. He visited the ED again the 6th week after his initial event. During his second visit, he had clear consciousness and no decrease in muscle power or change in pupil size. CT scan showed a 1-cm-thick, isodense right parietal extra-axial collection, extending to frontal and temporal regions. The lesion, which caused a 0.3-cm midline shift, compressed the underlying brain and cyst (Figure 2). It was better visualized after enhancement of the brain region using intravenous contrast agent (Figure 3), no enhancing membrane was observed.

Under the impression of cSDH, the patient was admitted. Burr hole drainage was arranged on the next day. After dural opening, typical motor oil-like fluid was found and removed, confirming our diagnosis. We did not find outer membrane, inner membrane, or cyst wall. The subdural space was irrigated via a drain tube, which was left in place for external drainage for 24 hours postoperatively. The tube was removed when the output was negligible. Throughout the course, there was no CSF-like fluid observed. The patient was discharged 2 weeks later with complete resolution of preoperative neurological deficits. Another brain CT follow-up was performed three months after surgery for recurrent headache, revealing total removal of cSDH and remarkable reduction in cyst size (Figure 4).

Discussion

The reported incidence of intracranial AC in general population is about 1%.\[^{3-5}\] Common locations of ACs include middle fossa (MF), cerebropontine angle, cerebral convexity, suprasellar region and posterior fossa.\[^{1,5}\] Based on size, mass effect and communication with subarachnoid space on CT cisternogram, the Galassi classification system has been used for classifying ACs.\[^{3}\] For adult patients with incidentally discovered ACs, a single follow-up imaging study in 6-8 months is usually adequate.\[^{13}\] There is no need for any treatment if there is no change in size.
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Figure 1. Axial non-contrast CT image showing a 3.7 x 2.4 cm cyst in the right parietal lobe. The hypodense cyst content has the same density as cerebrospinal fluid within lateral ventricles.

Figure 2. Axial non-contrast CT image showing an isodense extra-axial collection over right parietal lobe and compression of the right lateral ventricle.

Figure 3. Axial contrast-enhanced CT image showing the interface (black dots) between the enhancing brain surface and the non-enhancing chronic subdural hematoma.

Figure 4. Postoperative axial non-contrast CT image showing total removal of chronic subdural hematoma and remarkable reduction in arachnoid cyst size.
The reported incidence of cSDH as a complication of asymptomatic AC varied widely. In contrast to “usual” cSDH which tends to occur in the aged population, patients at their second decade of life had the highest risk of AC-associated cSDH. The symptoms of cSDH are similar in adults without or with ACs. In pediatric patients (<15 years old), the most frequent symptoms are signs of intracranial hypertension, including headache and vomiting. A history of trauma, including surgical trauma on the cyst (shunting or fenestration), was noted in most patients. The locations of AC-associated cSDHs are mostly located ipsilateral to AC and intracystic hemorrhage is common, especially in hematomas associated with MF cysts. In addition, MF cysts usually remain visible after cSDHs are removed. In our case, the convexity cyst shrank significantly after removal of the compressing cSDH, indicating that convexity ACs may behave differently from MF ones.

There is no consensus regarding the optimal treatment of AC-associated cSDH. Selection of treatment strategy depends on patient age and cyst size. Concomitant treatment of cSDH and AC, either by fenestration or resection, is more commonly performed in pediatric patients, though a more conservative approach is advocated by some authors. For adult patients, burr hole drainage is the first-line treatment. Similar to those without AC, recurrence is also common in patients with AC-associated cSDH. Using a more aggressive approach, Wu et al. experienced no recurrence among their 14 patients. Two of the nine pediatric patients treated by Muslumen at al. developed subdural fluid collection after burr hole drainage. Both had large type III cysts and were treated by subduroperitoneal shunt placement with good recoveries. They recommended shunting as the second-line treatment for recurrent cSDHs associated with large MF cysts.

In addition to AC, there are other types of intracranial CSF collection that are known to be associated with cSDH. Subdural effusion, also known as subdural hygroma or subdural hydroma, has been associated with development of cSDH after trauma. Before “conversion” into cSDH, the chemical and cellular compositions of SDE are similar to those of CSF. In adults, brain atrophy with increased sulcal CSF volume has also been associated with cSDH in both younger and older subgroups. Similarly, AC can be regarded as a loculated CSF collection whose lining can be destroyed by trauma, causing its contents to be discharged into the subdural space forming SDE. Subsequently, the pathophysiology of AC-associated cSDH resembles that of SDE-associated cSDH. Our patient might also have had SDE prior to accumulation of cSDH.

In conclusion, although asymptomatic AC is mostly benign, patients with AC detected incidentally during head trauma evaluation deserve special attention and frequent follow up because there is risk of AC-associated cSDH. Regardless of whether there is AC or not, burr hole drainage is the treatment of choice in adult patients with symptomatic cSDH.

Conflicts of Interest Statement

The authors declare that there are no conflicts of interest regarding the publication of this paper.
References

與蜘蛛網膜囊腫相關的慢性硬腦膜下出血：
病例報告

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摘要

慢性硬腦膜下出血是老人或是腦萎縮病人在頭部外傷數星期後常見的病症。蜘蛛網膜囊腫是一種內容物為腦脊髓液的先天性病灶，大多數病人沒有症狀，通常是因為頭部外傷後進行腦部影像檢查時意外發現。少數蜘蛛網膜囊腫病人會在外傷後併發慢性硬腦膜下出血，如本文提出的病例。

關鍵詞：蜘蛛網膜囊腫、慢性硬腦膜下出血、鑽洞引流手術、硬腦膜下積液、頭部外傷