Arachnoid Cyst is it a Risk Factor for Chronic Subdural Hematoma in Young Adults? (A Report of Four Cases and Review of Literature)

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INTRODUCTION

Chronic subdural hematoma (CSDH) is a common type of intracranial hemorrhage and is predominantly seen in the elderly. It typically consists of darkish red liquefied blood breakdown products with an associated neomembrane and located in the otherwise potential space between the arachnoid and the dura.

The space that the CSDH occupies is technically the intradural space. It is a disruption of the dural border cell layer from the deep pachymeninges.

Blood in this space provokes an inflammatory reaction, which results in an enveloping membrane surrounding the blood. If this follows clearly dated trauma, a CSDH may become symptomatic days to weeks after the event. Most of the time, there is no clear traumatic history that precedes the discovery of a CSDH. In the cases in which there is no associated trauma, other etiologies have to be considered.

Arachnoid cysts (AC) are the most frequent congenital cystic intracranial abnormality. They represent extra cerebral, intra-arachnoidal cerebrospinal fluid collections comprising 1% of intracranial space-occupying lesions. Subdural hematomas are infrequently an encountered complication of AC.

This study was designed to describe four cases with AC associated with CSDH and in light of a literature review; we discuss arachnoid cysts as a possible risk factor for subdural hematoma, especially in young adults.

PATIENTS & METHODS

Four cases of CSDH related to AC underwent surgery during the period between October 2011 and January 2013 in the Department of Neurosurgery, Benha University. Before coming to our attention, none of the patients complained of neurological disorders or received a diagnosis of AC. All patients had reported minor head trauma from 30 to 45 days with a mean of 37 days before hospital admission. There were three males and one female aged between 10 to 41 years with a mean of 26.75 years (Table 1). All four patients underwent cerebral CT scanning in the emergency department, and assessment of their coagulation status by screening with complete blood count, liver function tests, prothrombin time (PT), international normalization ratio (INR), and activated partial prothrombin time (PTT).
Table 1: Summary of data in four patients with CSDH in association with ACs

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (years), Sex</th>
<th>Minor Head Injury</th>
<th>Presenting symptom</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>10, Male</td>
<td>Bicycle fall</td>
<td>Headache</td>
</tr>
<tr>
<td>2</td>
<td>22, Male</td>
<td>Soccer related</td>
<td>Headache</td>
</tr>
<tr>
<td>3</td>
<td>41, Female</td>
<td>Fall</td>
<td>Headache</td>
</tr>
<tr>
<td>4</td>
<td>34, Male</td>
<td>Aggression</td>
<td>Headache, and Right Hemiparesis</td>
</tr>
</tbody>
</table>

**RESULTS**

It was obvious that these patients in whom a CSDH was associated with an AC were young with mean age of 26.75 years, and as in cases involving an isolated CSDH, their CSDH manifested typically within few weeks after a minor head injury with a mean duration of 37 days and their neurological symptoms were due to intracranial hypertension.

**Illustrative Cases**

**Case 1**

A 10 years old boy presented with a 14 days history of gradually worsening headache accompanied by one attack of vomiting. His recent history was remarkable for head injury caused by falling from his bicycle, one month before. His level of consciousness was normal without any visual disturbances and examination revealed no neurologic deficit. Of note, the patient did not receive any drug that could have implicated with hemorrhagic phenomena, and laboratory assessment of his coagulation status was within normal. CT Brain demonstrated a left frontotemproparietal CSDH of mixed hypo and isodenisty (Fig. 1 a&b).

The patient underwent emergency surgery to evacuate the CSDH through a burr hole under general anesthesia. During the postoperative course the patient recovered smoothly and his headache disappeared.

Early postoperative CT brain revealed satisfactory drainage of CSDH and demonstrated an arachnoid cyst over the ipsilateral Sylvian fissure (Fig. 2). The patient was completely free of neurological symptoms at regular follow-up visits.

**Case 2**

A 22 years old healthy man presented with severe headache accompanied by blurring of vision. His recent history was remarkable only for insignificant head trauma five weeks ago, during a football game. CT brain demonstrated right frontotemporoparietal isodense CSDH with intracerebral edema and midline shift. The aforementioned findings were inconsistent with a mild head trauma. Therefore, a contrast enhanced CT brain was ordered, and no other space-occupying lesions were identified (Fig. 3 a&b). CSDH was evacuated through a burr hole. The postoperative course of the patient was uneventful, postoperative CT brain revealed right Sylvian AC (Fig. 4 a) and satisfactory drainage of the hematoma with insignificant residual (Fig. 4 b).

**Fig. 1 a&b:** Preoperative CT brain showing left frontotemporoparietal CSDH

**Fig. 2 a&b:** a. Early postoperative CT brain demonstrating Galassi type 2 arachnoid cyst over the left Sylvian fissure, and b. adequate drainage of the hematoma with drain in place

**Fig. 3 a&b:** CT brain with contrast showing isodense CSDH hematoma with midline shift.

**Fig. 4 a&b:** CT brain showing right Sylvian AC with complete resolution of CSDH
DISCUSSION

In all these four cases, as in reported cases3-11 the CSDH was ipsilateral to the AC and the AC caused no symptoms until the CSDH manifested neurologically.

True arachnoid cysts are congenital. The lesion could arise from a minor developmental aberration of the subarachnoid space, owing to changes in mesenchymal condensation or CSF flow into the pia-arachnoid space. Ultrastructural studies that involved transmission electron microscopy have specified that the cyst lining consists of single or multiple layers of cells and that ACs are wholly situated within the arachnoid membrane9,10.

They can develop anywhere within the subarachnoid space, intimately related to the cisterns. In the general population, nearly half of cysts occur within the Sylvian fissure. Arachnoid cysts show a slight predilection for the male sex. A majority of the arachnoid cysts are recognized during the first two decades of life9.

The coexistence of an intracranial AC with subdural haematoma is quite rare, and the first description of such case was described in 193814.

Current histological knowledge also leaves the pathogenesis of CSDH unclear. In an electronic microscopic study, researchers described a dural microscopic space, researchers described the dural border cells that extend from the dura and adhere tightly to the arachnoid membrane, delimiting a “potential space”—namely the “subdural compartment”5. This space remains virtual until it is created and opened by a traumatic event. In an earlier study of patients with post-traumatic acute SDH investigators found that neuroimaging studies showed the hematoma in the “intradural” compartment. From anatomical findings and studies involving transmission electron microscopy it seems that the membrane separating an AC from a CSDH forms when the membranous structures of the AC and CSDH overlap and adhere tightly to each other. The presence of a congenital arachnoid malformation such as an AC weakens the subdural compartment at the level of the junction of the dural border cells and favors subsequent bleeding; causing a minimal and asymptomatic acute SDH that ultimately evolves into a symptomatic CSDH2.

Some investigators found small bridging vessels between the dura and outer membrane of the AC, and suggested that these small vessels are the source of initial bleeding leading to CSDH in AC. Moreover, the fragile supporting stroma also predisposes to rupture even after a minor injury. In some occasions, subsequent re-bleed or osmotic influx of fluid could explain the gradual increase in the subdural fluid collection7.

Preoperative CT brain, obtained in all patients demonstrated the AC and CSDH in 2 cases (case 3 & case 4), while in case 1 & case 2 AC was completely concealed by the ipsilateral CSDH. CSDHs differed from the brain parenchyma in density, appearing hypodense, isodense, or slightly hyperdense according to whether bleeding was longstanding or recent13.

In all our four patients the AC appeared similar to cerebrospinal fluid in signal density. After the CSDH was removed the AC reexpanded in case 3 & case 4, and became apparent in case 1 & 2. AC in case 1&3 was type II according to Galassi classification, while the remaining cases demonstrated AC of type I according to the same classification.

The satisfactory results obtained by performing burr hole irrigation of the CSDH and by leaving the AC for follow-up, agree with some previous reports10,12.

All patients’ neurological symptoms regressed within days of surgery, and at long-term follow-up (3–18 months) none of them had recurrent lesions. The patients’ postoperative clinical course resembled that of patients with the more usual CSDH uncomplicated by an AC.

Other investigators have evacuated the CSDH and inserted a cysto-peritoneal shunt7. Some have proposed using craniectomy or craniotomy with evacuation of the CSDH and fenestration of the AC13. Endoscopic membrane excision has also been used6.

There are only a few published cases in which authors described a CSDH associated with an AC that were treated conservatively. Most of these patients harboring these lesions had only mild neurological symptoms and the CSDH resolved spontaneously13.

CONCLUSION

As also reflected in a literature review, patients with AC, especially when located in Sylvian fissure, appear to harbour a life-long risk of contracting subdural hematoma. Data suggest that when a CSDH and AC are diagnosed in the same patient, the neurological symptoms arise exclusively from the mass effect of the CSDH, and can be successfully treated by undergoing CSDH drainage alone while the AC is left intact for follow-up. Neurosurgeon should consider the possibility of CSDH in young patients with prior history of minor head injury, as AC is not uncommon.
REFERENCES


