

Temporal Fossa Arachnoid Cysts Presenting as Chronic Subdural Hematomas Report of Two Cases and Brief Review of the Literature

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Abstract

Introduction: Intracranial arachnoid cysts are considered to be congenital malformations with a predilection for the temporal fossa. They are often asymptomatic but can sometimes be symptomatic due to enlargement or hemorrhage [1]. Majority of arachnoid cysts, particularly those of smaller sizes, have a benign uneventful lifetime course [2].

Chronic subdural hematoma is one of the commonest diseases encountered by a neurosurgeon in daily practice. It is however rarely seen in young patients. Congenital arachnoid cysts have been implicated in both traumatic and spontaneous chronic subdural hematoma in young individuals. However, because most bleeding after trauma is delayed, it is often overlooked [3].

Arachnoid cyst - associated chronic subdural hematoma differs significantly from its counterparts without AC in epidemiologic, demographic, and clinical characteristics [4].

Optimum treatment strategies to address the chronic subdural hematoma and arachnoid cyst are not very well described [5]. We report two cases of Arachnoid cyst - related chronic subdural hematoma in young individuals, who were treated with evacuation of chronic subdural hematoma with craniotomy, without addressing the cyst itself. Fenestration and cerebrospinal fluid diversion should be reserved only as secondary procedures [5].

Keywords: Chronic Subdural Hematoma, Arachnoid Cyst Rupture.

Abbreviations

AC: Arachnoid cyst

CSDH: Chronic subdural hematoma

MRI: Magnetic Resonance Imaging

Introduction

Arachnoid cyst (AC) is a congenital intracranial lesion often found incidentally on intracranial imaging. Although symptomatic in a small number of patients, AC presents a benign natural history for those presenting without symptoms [6]. In rare circumstances, AC can be complicated by CSDH [7]. Accumulating evidence points to AC as a risk factor for CSDH, especially in children and young adults [8]. However, AC-associated CSDH differs significantly from its counterparts without AC in terms of epidemiologic, demographic, and clinical characteristics, as well as in management and prognosis.

The traumatic CSDH, even though the trauma may be trivial or unrecognized, is uncommon in older children or young adults [9]. Pathogenesis of CSDH associated with the AC is still not exactly known. It is considered to emerge from the vessels surrounding the cyst wall or floating inside the cyst [10, 11]. Several authors

have emphasized the structures between the dura and arachnoid membrane; however, it is nearly impossible to find the bleeding point during operation of CSDH associated with AC because the original anatomical structures are already distorted by the hematoma. Several studies have suggested that the cyst is less compliant than normal brain tissue, and thus traumatic impact can easily spread to the cyst wall, bridging veins, and unsupported vessels around the cyst.

Materials and Methods

We report the case of a 13 years old female who presented with a relatively rapid deterioration of her level of consciousness, with accompanying vomiting and headache as the initial presentation. There was a sport-related traumatic head injury a few days before, but her initial clinical course was uneventful. She was admitted to the emergency department, where an MRI scan was performed. It revealed an extended left -sided chronic subdural hematoma which was causing midline shift and effacement of the ipsilateral sulci. An arachnoid cyst localized in the left temporal pole was observed, in close association with the ipsilateral hematoma, suggesting that its rupture was the underlying cause for the formation of the hematoma (Figure 1).

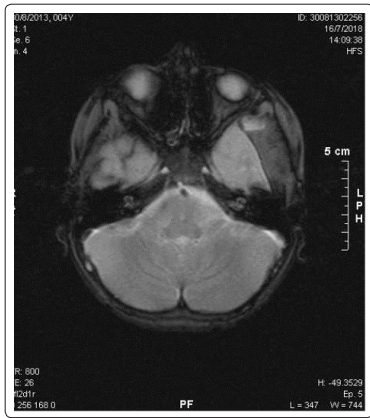


Figure 1

A left fronto-temporal craniotomy was performed for the evacuation of the hematoma, with no effort been attempted to fenestrate the cyst or resect the AC membrane.

The patient post-operative course was uneventful, with postoperative MRI revealing evacuation of the hematoma, no destruction of the cyst components and no reappearance of hemorrhage (Figure 2a & 2b).

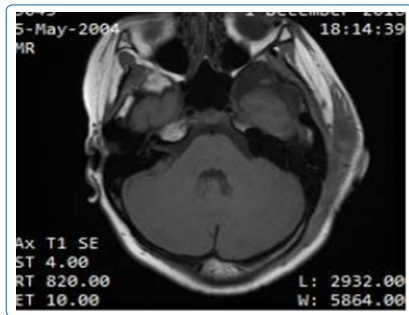


Figure 2a



Figure 2b

We also report the case of a 5 year old male, who presented due to progressively deteriorating episodes of headache, of increasing severity and duration. Vomiting was reported and the level of consciousness was mildly depressed. No profound medical history of head injury was reported. The patient was evaluated with an emergent MRI scan, which revealed an extensive chronic subdural hematoma, extending in the left fronto-temporo-parietal region, causing midline shift and ventricular compression. An associated arachnoid cyst of the temporal pole was identified, in close anatomic

proximity and association with the relevant hematoma (Figure 3a, 3b).

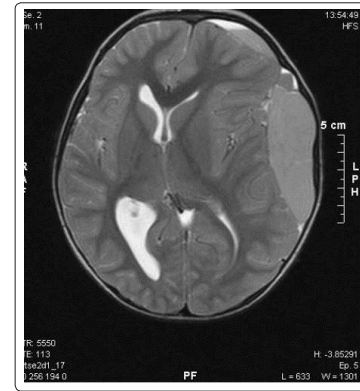


Figure 3a

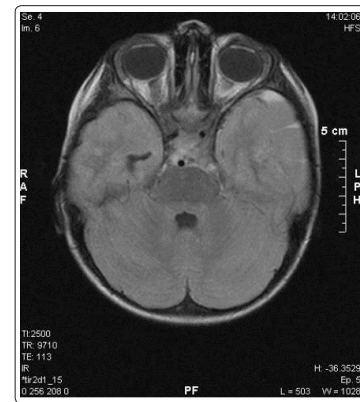


Figure 3b

An extensive left fronto-temporo-parietal craniotomy was executed in order to evacuate the underlying hematoma with a drain left in the relevant subdural space. A post-operative CT scan was executed, which revealed the absence of any remnants of the hematoma and the close anatomic relationship of the underlying cyst with the hematoma (Figure 4a, 4b). No surgical intervention on the cyst was executed. The patient's postoperative clinical course was uneventful and his recovery was excellent.



Figure 4a



Figure 4b

Results

AC is a benign intracranial lesion that is always detected incidentally by imaging modalities. The reported incidence of intracranial AC in the general population ranges from 0.3% to 2.6% [12-15]; however, there are no data on the development of CSDH in patients with incidentally identified intracranial AC at the long-term follow-up.

The most common location of ACs is middle cranial fossa followed by retro cerebellar and convexity locations. Middle cranial fossa ACs is more frequently seen in the vicinity of sylvian fissure where they cause scalloping and thinning of the overlying bone.

Wester and Helland found no difference in hematoma frequency among small, medium, and large temporal ACs [16]. In that study, temporal location of AC was a statistically significant risk factor for CSDH; however, middle fossa is the most frequent location of AC reported in epidemiologic studies [12, 14].

One of the common complications of AC is the development of CSDH or subdural hygroma (7). The incidence of subdural fluid collection as a complication of the middle fossa AC is reported to be from 6.6 to 17.5 % [17, 18].

Page et al. proposed two theories to explain the origin of CSDH in AC [19]. The first theory involved the rupture of bridging veins or vessels in the cyst wall due to the easy transfer of pressure through the cyst. The second theory indicated that AC is less compliant than the normal brain resulting in reduced intracranial cushioning following trauma. The underlying cyst cavity provides fluctuating movement of the cystic fluid, transferring the shearing force to the outer membrane thus tearing the small vessels between the outer arachnoid membrane and dura resulting in small bleeding which progresses the condition to the symptomatic chronic stage. According to Haines et al [20]. The subdural space is not a potential space contrary to pleural cavity, but the dura is continuous and tenuously attached to the arachnoid by a few cell junctions. There is a continuity of cellular layers and no intervening space. Collection of hematoma or fluid is usually not subdural in location but is rather created by a shearing open of tissue damage.

There is no consensus as to the optimal treatment of AC associated CSDH. Some authors have advocated burr hole drainage as sufficient for CSDH drainage, (21) whereas others have argued that fenestration or partial/subtotal removal of AC membrane through craniotomy is necessary [6, 21].

The most current strategy is to follow the stratified and personalized strategies in the management of AC-associated CSDH [22]. Vigilant surveillance and expectant therapy are feasible for patients with no or mild symptoms and only a slight space-occupying effect. Burr whole drainage without manipulation of the AC membrane should be the first-choice surgical procedure in symptomatic patients. In the event of early recurrence after burr whole drainage, repeated burr whole drainage is still effective in some patients. Craniotomy combined with fenestration or removal of the AC membrane is another option for patients with recurrence. Both of our cases were managed with extensive craniotomy due to the midline shift and the large amount of subdural blood collection, which was questionable if it could be drained effectively with burr hole evacuation. Burr whole drainage of the clot without manipulation of the cyst, craniotomy/craniotomy with drainage of the clot and excision/fenestration of the cyst has all yielded similar results [5].

However, there are several limitations to the proposed guidelines for the treatment of that entity. AC-associated CSDH is a rare entity, and most of the information published to date has been in the form of case reports. Statistical analysis is inappropriate at present.

CSDH is a rare complication in patients with intracranial AC. Male children, juveniles, and young adults with recent head trauma or sport-related injury are most commonly affected [4]. Young patients with arachnoid cyst are at a high risk of developing a chronic subdural hematoma following trivial head trauma and should be kept under close observation. Because most SDHs or ICHs associated with ACs are sub-acute or chronic, children with ACs should be regularly followed up within 3 months after minor head injury [3]. However, large numbers are required to evolve a standard treatment protocol.

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