Case Report

Arachnoid Cyst Complicated with Subdural Hematoma: Case Report and Review of the Literature

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ABSTRACT

Arachnoid cysts are constitutional anomalies with an asymptomatic course. There is no consensus on their etiology. They may lead to increased intracranial pressure accompanied by acute or subacute hemorrhage fenestrating into subdural space in young adults. A case of arachnoid cyst that experienced loss of consciousness one week after a head trauma and was complicated by subdural hematoma is presented. Chronic subdural hematomas are rare in young adults. An arachnoid cyst should be considered in these cases and the operation planned accordingly.

Key Words: Arachnoid Cyst, Subdural Hematoma, Surgery

INTRODUCTION

Arachnoid cyst is a congenital malformation characterized by cerebrospinal fluid (CSF) accumulation between the two layers of arachnoid membrane (3,6). It is most frequently located in the Sylvian fissure in middle fossa (3,4,6,10,13-15). It may present with epileptic seizure, headache, increased intracranial pressure, and focal neurological findings as well as with macrocrania, cranial asymmetry, and mental retardation in children (3,6). The detection of incidence of arachnoid cysts has risen as high as 54% through advanced radiological imaging methods (7,10,11,15).

Chronic subdural hematoma (CSDH) is more common in elderly due to cerebral atrophy development. Patients usually have a history of minor head trauma. In such cases with enlarged subdural space, hemorrhaging into the subdural space associated with laceration of bridging veins occurs (1,16). In adolescents and adult populations, subdural space is narrow, as a result of which CSDHs are rare. In these cases, as well as systemic diseases such as coagulopathy, vasculopathy, infection, tumor, and alcoholism, constitutional anomalies like arachnoid cysts (AC) may be detected (10,11,13-15,17,18). An 11-yearold male patient operated due to subacute SDH diagnosis has been presented. Arachnoid cysts in children may be suggestive of subacute or chronic SDH, which is rarely seen in this age group.

CASE REPORT

A 11-year-old boy had struck his head against a wall after a fall and started having headaches 2 days before admission to our hospital. His headache was unresponsive to analgesics and became gradually more severe. He was brought to the emergency outpatient clinic because of apathy and sleepiness by his family. On neurological examination, he 2nd Neurosurgery Clinic, Ankara Numune Education and Research Hospital, Ankara, Turkey

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Serdar Sokak No.77/3 O6170 Yenimahalle, Ankara, TURKEY Phone :+90 312 343 08 66 Fax :+90 312 310 34 60 E-mail : alidalgic@yahoo.com was responsive to verbal stimuli by eye-opening, non-conforming to orders, and moaning. The pupils were asymmetric (right 2mm/left 3mm), but responsive to light. He also had mild right hemiparesis. CT scan revealed a left fronto-temporoparietal iso-hypodense subdural hematoma that was 35 mm at the widest area, compressing the gyri and sulci, and producing 8 mm midline shift (Figure 1A). CT scan also revealed a hypodense lesion associated with arachnoid cyst located at the left temporal pole distal to Sylvian fissure (Figure 1B). Coagulation tests such as bleeding time, coagulation time, activated thromboplastin time (APTT), partial thromboplastin time (PTT), and international normalized ratio (INR) were normal.

The patient was operated on urgently, and two burr holes were opened to the left frontal and parietal region. Despite drainage and irrigation, the cortex could not be elevated adequately because of the septas located in the subdural space and a fronto-temporo-parietal craniotomy was performed. The temporal lobe was hypoplasic, and the Sylvian fissure was enlarged. Multiple pseudomembranes of subdural hematoma were excised. The arachnoid cyst mounted partially on the subarachnoid space. The neurological examination was normal on the postoperative first day, and in the first month. MRI performed 3 months after the operation revealed a hygroma that was isointense with CSF, causing enlargement in the left temporal region and Sylvian fissure without any signs of compression. No recurrence of subdural hematoma was observed (Figure 2).

DISCUSSION

Arachnoid cysts make up 1 % of all intracranial space occupying lesions. However, the ratio of asymptomatic arachnoid cysts diagnosed incidentally has increased to 54% with the aid of the advanced neuroradiological tools such as CT scan and MRI. (3,6,7,11). They are more frequent in children (6) and known as congenital intraarachnoidal - leptomeningeal malformations. An arachnoid cyst is characterized by the collection of CSF between the two layers of arachnoid membrane and is located in the CSF cisterns, which causes their enlargement (3, 6, 10, 13 - 15). Although the etiopathogenesis of the disease has not been clearly defined, it is believed by some to be secondary to head trauma and infections (13).

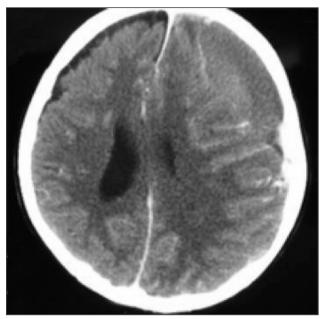


Figure 1A: CT scan revealed a left fronto-temporo-parietal iso-hypodense subdural hematoma



Figure 1B: CT scan revealed an arachnoid cyst located at the left temporal pole distal to Sylvian fissure

Three-fourths of all arachnoid cysts are located supratentorially, while one fourth are infratentorial. The most frequent site is the Sylvian fissure (34.6% - 59.6 %). Cerebral convexity (10.1% - 12.9%), sellar region (6.7% - 11.3%) and interhemispheric fissure (6.5%) are the other frequent locations. Cerebellomedullary cistern is the most frequent

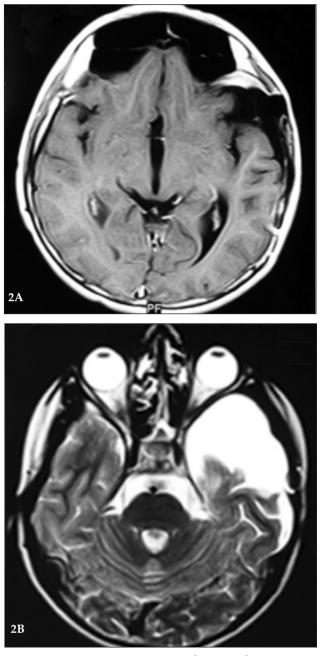


Figure 2: MRI performed 3 months after the operation revealed an arachnoid cyst in the left temporal region and Sylvian fissure without any signs of compression in the T1 scan (**2A**) and T2 scan (**2B**).

infratentorial site for arachnoid cysts (15%) (3-7,10,15).

Galassi has classified the fossa arachnoid cysts located in the Sylvian fissure into three types: Type I is located in the temporal apex and communicates with CSF in the cisternogram; Type II enlarges the proximal and middle segments of the Sylvian fissure and has partial communication with CSF in the cisternogram; Type III enlarges the whole Sylvian fissure, and there is a shift of midline structures, but there is no communication with CSF in cisternogram. Type II is the most frequent type (4).

Arachnoid cysts are usually asymptomatic. They may present either by non-specific headache, seizure, focal neurological findings or by increased intracranial pressure due to secretory activity and enlargement of the cyst. (8). In addition, asymmetry of the cranium, macrocrania, and mental retardation may accompany in children (6).

On the other hand, arachnoid cysts may cause subdural hematoma to fenestrate into AC leading to acute clinical conditions presenting with SDH and increased intracranial pressure and associated loss of consciousness (10,18). Rare subdural hygroma cases developing due to cyst rupture have also been reported (2, 5). As a result of hemorrhage or rupture, the clinical picture in children and young adults progresses primarily with acute/subacute complaints and findings associated with increased intracranial pressure, which leads to changes in consciousness (1, 16).

Subdural hematoma (SDH) is a rare occurrence in children and young adults as the brain parenchyma fills up the entire subdural space. However, dynamic spaces such as AC may set a potential area for SDH development and be complicated by SDH following a head trauma. With rupturing of bridging veins, the external wall of AC is also lacerated; thus, AC fenestrates into the subdural space and is complicated by subdural hemorrhage. It has also been reported that the veins in AC and/or on AC wall are more sensitive to mechanical effects and the laceration of these veins may result in chronic subdural hematoma (CSDH) (10,13-15,17). Similarly, the AC membrane may be lacerated without any damage to any of the vascular structures, leading to subdural hygromas (5).

Parsch et al. reported 12 cases of a 16-case CSDH series that were complicated by AC to be under 34 years of age (15). Page et al., on the other hand, reported 7 cases, one of whom was 57 years old while the rest were 23 years old or younger (13). In a series of 529 cases that were operated due to CSDH, Mori et al. found the mean age as 69.5 years, while 12 of these cases which were AC complicated had a mean age of 27.8 years, with the a statistically significant difference (10).

In the first 24 hours, a thin membrane comprised of fibrin and fibroblasts appears on the surface of hematoma that fenestrated into the subdural space and sustains its development until the 4th day. The membrane is organized later on and microvascular circulation and capillary structures form. In the second week and afterwards, the hematoma mass is liquefied by phagocytes (1,16). Mathew et al. found that the SDHs smaller than 1 cm were reabsorbed before being organized; however, those that were larger became chronic (9).

The most important diagnostic tools are CT and MRI. On CT, there is a hyperdense image in the acute phase, an iso-hyperdense image in the subacute phase, and a hypodense image in the chronic phase. Nevertheless, in the chronic phase, there may be mixed appearances associated with fresh hemorrhages (11,12). On MRI, however, both T1 and T2 are hypointense in the acute phase; both T1 and T2 are hyperintense in the subacute phase, while T1 is iso-hypodense and T2 is iso-hypointense in the chronic phase (7,12). In addition, fresh hemorrhages and associated membrane development may aid in the detection of septa. Mori et al. detected highdensity hematoma in 58% of the AC cases complicated by SDH and in 19.5% of the cases with no AC (10). Despite admitting the association of SDH mass with the age, Munk et al. claimed the possibility of isodense CT findings in AC complicated cases, and thus, they emphasized the importance of through evaluation of the cases with SDH located in the middle fossa, where AC is most frequently located (11).

In SDH cases, the conventional therapy has been drainage and irrigation through a burr-hole. However, there is no consensus yet as to the most effective therapy in AC complicated cases. Mori et al. performed drainage and irrigation through a burrhole on 12 CSDH complicated AC cases and reported satisfactory results (10). Similarly, Parsch et al. performed burr-hole drainage and irrigation on the 12 cases of the 16-case series and craniotomy and membranectomy on the 2 cases, while 2 cases were followed up with conservative therapy. The two cases that underwent craniotomy had been performed burr-hole drainage, after which they had recurrent SDH (15). Both groups of authors reported that they did not perform a second operation for AC on cases that had undergone burr-hole drainage and irrigation (10,15). Page et al. recommended

craniotomy, membranectomy, and hematoma drainage for middle fossa ACs complicated by CSDH (14).

In this case, it was presumed that there was a hemorrhage into the AC due to a head trauma a week earlier and that this had opened out into the subdural space as a complication and gradually led to the development of a SDH.

In cases with CSDH, detected in children and young adults in particular, AC should be kept in mind together with systemic disorders of blood coagulation. In such cases, the potential space formed by AC predisposes to recurrent hemorrhages; thus, dense membranous structures with septations due to multiple hemorrhages may be observed. When adequate drainage cannot be achieved by burr-hole drainage, SDH can be drained by membranectomy and AC can be partially treated.

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