

Spontaneous disappearance of middle fossa arachnoid cyst after head injury

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Abstract. A case of middle fossa arachnoid cyst is presented, which disappeared after head injury. Five days after the trauma, CT scan revealed subdural fluid collection in addition to an arachnoid cyst in the middle fossa on the same side. As the subdural fluid resorbed, the cyst became smaller and disappeared on the follow-up CT scans without surgical intervention.

Key words: Arachnoid cyst – Middle fossa – Head injury.

Arachnoid cysts account for 1%-4.8% of all atraumatic intracranial space-occupying lesions [8, 19] and the middle cranial fossa is the most common site. Although the incidence has increased since the introduction of computed tomography (CT) [8], the natural course of these cysts has not been fully elucidated [1, 6]. The necessity of surgical treatment, especially in asymptomatic cases, remains controversial [1, 5, 8–10, 12, 19–22].

We report a case of a middle fossa arachnoid cyst which gradually absorbed after head injury. This interesting case suggests the possible mechanism of cyst absorption.

Case report

A 9-year-old boy was referred to Uwajima Hospital on 26 September 1983 because of headache, nausea and vomiting after head trauma. Past history, including the prenatal period, was not significant. No prior episodes of intracranial inflammation or head injury were reported. On 16 September 1983, he was involved in a traffic accident and struck his frontal and occipital areas. After an unconscious period of 5 min or so, he continued to be drowsy for several hours with episodes of vomiting. No fracture was demonstrated on skull films. On the following day, he was transferred to another hospital for a CT scan, which revealed a circumscribed, low-density area located at the tip of the left temporal lobe (Fig. 1). The lesion caused a questionable left-toright shift. He became completely alert and free of complaint. On the 5th day after trauma, his frontal headache and vomiting recurred, and the CT scan was repeated. In addition to a middle fossa cyst, a low-density zone was found subjacent to the skull on the left side, with a slight midline shift.

Ten days after his head injury, the patient was referred to our clinic for further evaluation. His physical and mental development was normal. His head was neither enlarged nor grossly

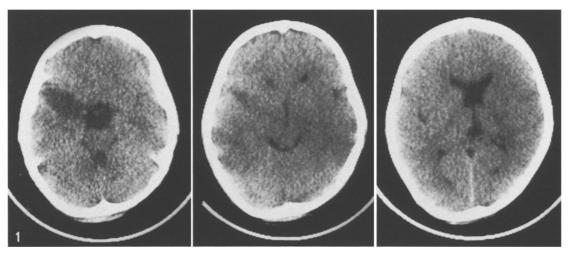


Fig. 1. CT scan performed on the day after head injury shows low-density area in the anterior portion of the left middle fossa. Cavum septi pellucidi and questionable left-to-right shift are noted

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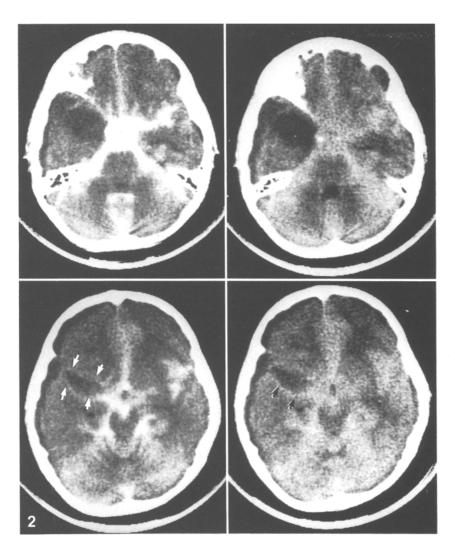


Fig. 2. Metrizamide CT cisternography 3 h (left) and 7 h (right) after intrathecal administration. There is no free communication between cerebrospinal fluid pathway and both low-density lesions. A halo of metrizamide indicates the presence of subarachnoid space around the middle fossa cyst (arrows)

deformed. He was neurologically intact and the fundi were normal. Skull radiographs showed slight elevation of the lesser wing of the left sphenoid bone, but no bulging of the squamous portion of the temporal bone. A CT scan showed a persistent left subdural fluid collection and a cystic lesion in the left middle fossa (Fig. 3, left). Metrizamide CT cisternography demonstrated that the cyst and the subdural fluid collection did not freely communicate with the cerebrospinal fluid (CSF) pathways. By 3 and 7 h after the intrathecal administration of metrizamide, the contrast filled the subarachnoid space, giving rise to a halo of contrast around the cyst, demonstrating the presence of a CSF space around it. The lesion was diagnosed as an arachnoid cyst, associated with a subdural fluid collection (Fig. 2).

Because the patient was asymptomatic at the time of admission, no surgical treatment was undertaken. A CT scan was repeated 18 days following the trauma and a slight increase in the volume of the subdural fluid was noted. Surprisingly, the arachnoid cyst seemed to have decreased in size (Fig. 3, center). Subsequently, the volume of subdural fluid and the arachnoid cyst became smaller, and the cyst lost its smooth round contour (Fig. 3, right). By 6 months both lesions had disappeared (Fig. 4).

Discussion

Arachnoid cysts have generally been thought to be slowly expanding lesions and various theories have been proposed

for the mechanism of cyst expansion [7, 9–11, 25]. However, it has not always been apparent that the cyst causes neurological deficits and/or intracranial hypertension. For this reason, surgical treatment of this lesion, often found incidentally, is still controversial. Some authors limit the surgical indication to complicated or symptomatic cases [1, 5, 12, 19, 21, 22]. Conversely, others think all arachnoid cysts should be treated surgically because they expand slowly and pose potential risks, including acute enlargement, subdural fluid collections, and subdural or intracystic hematoma [2, 8–10, 20].

Head injury is a well-known precipitating factor in clinical deterioration of individuals with this malformation [16, 23]. Several mechanisms for an acute increase in intracranial pressure have been postulated. One is a tear in the deeper layer of the cyst wall, which causes a one-way valvular arrangement facilitating the inflow of CSF and blocking its egress [23]. The other is hemorrhage within the cyst and the subdural space [3, 8, 14, 16, 23]. The source of bleeding is thought to be the poorly supported vessels in the cyst wall or arachnoid membrane [7, 8, 23]. Chronic subdural hematomas [13, 17, 23] have been found contralateral to the cyst [18, 26]. Subdural hygromas are

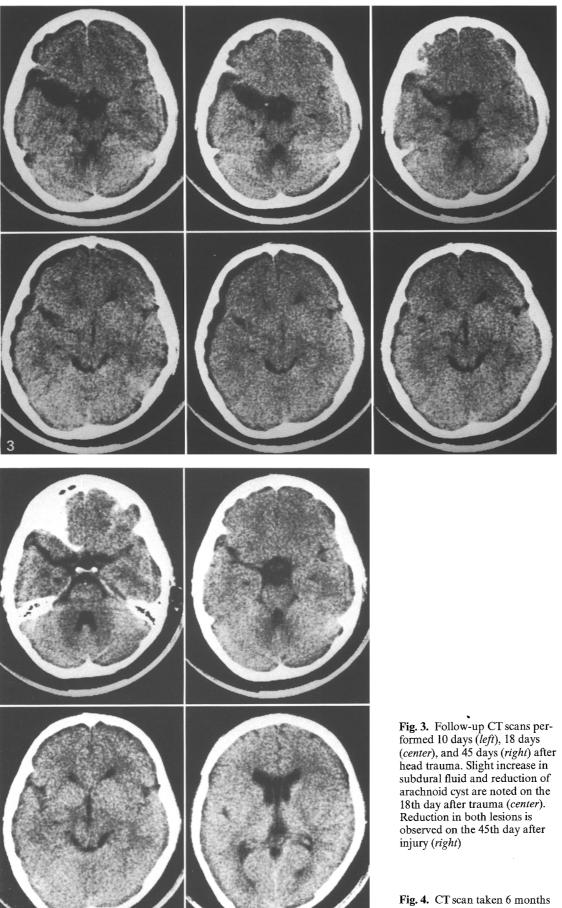


Fig. 4. CT scan taken 6 months after head injury. Arachnoid cyst and subdural fluid collection have disappeared completely

also a major cause of symptoms [4, 8, 9, 15, 24]. A tear in the outer membrane of the cyst resulting in a fluid collection has been confirmed at operation [8].

However, there has been no report that in a patient with an arachnoid cyst, a head injury had led to shrinkage of the cyst. Moreover, the literature does not contain any cases where the arachnoid cyst spontaneously decreased in size and disappeared ultimately without surgical intervention.

As to the possible mechanisms involved in the present case, two possibilities seem likely: a tear of an outer membrane caused by the trauma gave rise to the formation of a subdural hygroma, and as absorption of the subdural fluid occurred, the cyst fluid leaked into the subdural space to be absorbed much as it would through continuous drainage by a cyst-peritoneal shunt. Another possibility would be the presence of the subdural hematoma. The first CT scan showed a questionable midline shift, which might have been due to an isodense thin subdural hematoma that could not be identified on CT scan because of movement artifact. Differences in osmotic pressure between the subdural hematoma and the cyst could have facilitated the outflow of cyst fluid into the subdural space and disappeared with absorption of the subdural hematoma. The latter is less likely because once the subdural hematoma becomes iso-osmotic or disappears, the cyst should recur. This single case cannot be used to generalize about therapy.

At present, there is no definite evidence that surgical treatment would change and improve the situation for children with asymptomatic arachnoid cysts. Obviously, spontaneous reduction and disappearance of the cyst can occur. Whether or not surgical communication of the cyst with the subdural or subarachnoid spaces is indicated remains to be proven. We must know more about the natural history of this disease before the proper surgical indications can be defined.

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