



Arachnoid Cyst Complicated by Contralateral Chronic Subdural Hematoma

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Simple hematoma evacuation is often performed in cases of chronic subdural hematoma (CSDH) associated with arachnoid cysts (ACs) on the same side after head injury. However, it is not clear which procedure is appropriate when ACs and CSDH are located on different sides. Here, we present a case of a 3-year-old boy with right frontal-temporal subdural hematoma and an AC over the left frontal-temporal region. Treatment involved craniotomy with hematoma evacuation, but no drainage of contralateral AC was applied. The patient was discharged 7 days after surgery without any neurologic deficits and no symptoms of headache or poor appetite. These results suggest that craniotomy and hematoma evacuation without intervention for the AC is adequate in patients with contralateral CSDH with a large AC.

Key words: Arachnoid cyst, chronic subdural hemorrhage, Children

INTRODUCTION

Chronic subdural hematoma (CSDH) is encountered in neurosurgical practice. Most affected patients are male and aged >70 years.¹ Arachnoid cysts (ACs) are considered as congenital deformities, rarely cause symptoms, and are usually found accidentally. However, they are risk factors for chronic subdural hemorrhage, especially in children and young adults.² These individuals are also vulnerable to minor head injuries and may develop symptoms when hemorrhage is present.³ Contrastingly, ipsilateral subdural hemorrhage is commonly detected by imaging and treated by draining the hematoma either leaving the ACs intact or removing them concurrently.^{4,5} Mostly, both the procedures have good prognoses postsurgery. However, the causes and treatment strategies for patients with contralateral CSDH with AC are debatable. Here, we describe a patient with this profile.

CASE REPORT

A 3-year-old Taiwanese boy experienced headache after an injury caused by falling while running. His family denied

loss of consciousness at the time. The pain persisted despite medication, and symptoms such as nausea, vomiting, and drowsy consciousness developed on the day of hospital visit. His parents denied the presence of any congenital disease and confirmed that he went through normal developmental milestones. On admission, there were no external head injuries; he had drowsy consciousness with the Glasgow Coma Scale E4M6V4. Bilateral pupils responded normally to light and isocoria. No papilledema was noted. He showed a normal response to pain, light touch, and pinprick; full strength in his arms and legs; and normal deep tendon reflex. Laboratory testing revealed no abnormalities. Axial computed tomography showed right frontal-temporal subacute subdural hematoma and a large AC, Galassi type III, without intracystic hemorrhage at the contralateral middle fossa region. Skinning at the adjacent skull was also noted [Figure 1]. There was no midline shift. A neurosurgeon was consulted, and subdural hematoma in the right frontal-temporal area was evacuated via a small craniotomy. The outer membrane was identified underneath the dura mater; the hematoma pressure was high. About 40 ml of motor-oil-like hematoma was evacuated. The left AC was

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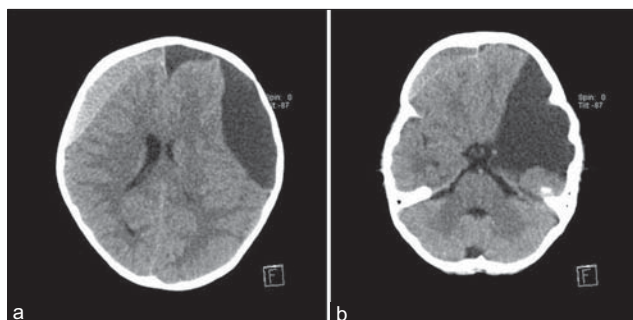


Figure 1: The axial view of noncontrast computed tomography on presentation. (a) Subdural hemorrhage at right frontal–temporal region. (b) Arachnoid cyst, Galassi type III without intracystic hemorrhage

not operated on. After the surgery, he was discharged without any signs of neurologic deficits and no symptoms of headache, poor appetite, or drowsy consciousness.

DISCUSSION

ACs comprise about 1% of intracranial masses, and most ACs (49%) are located at the Sylvian fissure.⁶ With the advances in neuroimaging, the incidence of diagnosed ACs is increasing. Most ACs are asymptomatic but may become symptomatic in the presence of hemorrhage, including intracystic or subdural hemorrhages. The etiology of AC is not well understood. It was hypothesized that AC etiology may be explained by meningeal maldevelopment.⁷ Traumatic chronic subdural hemorrhage is usually found in elderly and infant populations but seldom in young adults.⁸ About 2% of young adults with CSDH have coexisting ACs.⁹ Therefore, coexisting ACs in young adults with CSDH should be expected after minor head traumas.

The literature mainly focuses on the pathogenesis and treatment of ACs with ipsilateral subdural hematoma (SDH).^{5,10,11} However, the pathogenesis of AC with contralateral SDH is unclear. Different etiologies have been proposed. One study proposed that loose attachment between the arachnoid membrane and dura may cause easy vascular bleeding in the dura.⁷ Page *et al.*¹⁰ proposed two theories: (1) flow changes in the AC after mild head trauma caused rupture of bridging veins and (2) brains with ACs are less compliant to head trauma than brains with no ACs, causing rupture of bridging veins.¹⁰ A study on 12 patients with ACs without hemorrhage found small vessels between the outer membrane and dura in four patients, suggesting this as the bleeding source in patients with SDH with ACs.⁵ Burr hole drainage has been suggested for subdural hemorrhage without fenestration; resection has been suggested if the patient was previously asymptomatic for ACs.^{4,5,11}

However, ACs with contralateral CSDH are rare, and the potential treatment is still debatable. Two cases of bilateral

subdural hemorrhage with temporal ACs following head trauma were reported; both patients underwent bilateral craniotomy with further removal of the blood in the hematoma and recovered uneventfully.¹² Furthermore, a 10-year-old boy with an exclusively spontaneous subdural hemorrhage contralateral to temporal AC recovered gradually without undergoing surgery.¹³

In our case, the patient was asymptomatic before trauma and showed CSDH after a falling injury. He underwent craniotomy with hematoma evacuation without the treatment of AC and showed no symptoms postsurgery. Therefore, the possible mechanism of contralateral CSDH can involve ACs that are less compliant than brain parenchyma, resulting in contralateral bridging vein tear after a minor head trauma. Connecting small vessels have been reported between the arachnoid membrane and dura in a patient with ACs.⁵ Another possible mechanism is that the whole brain abnormality vasculature between the ACs and dura is vulnerable to minor head trauma, causing hemorrhage. Patients with intracranial hypotension may develop subdural hemorrhage or effusions.^{14,15} Therefore, because ACs are chronic intracranial space-occupying lesions, evacuation of ACs may suddenly decrease intracranial pressure and cause stretching of the contralateral bridging vein, increasing the subdural hemorrhage incidence. These results reinforce our treatment strategy of CSDH with contralateral ACs. Thus, we should perform the operation on CSDH without surgical approach of ACs if the patient is asymptomatic for ACs before trauma. However, ACs with contralateral CSDH are rare, and this is, to our knowledge, the second reported case. Thus, statistical analysis of current literature is not appropriate. Optimal treatment needs to be further investigated.

Most ACs are asymptomatic; ipsilateral subdural hemorrhage is more common than contralateral subdural hemorrhage. Less compliant ACs and contralateral bridging vein tears may lead to the development of CSDH after head trauma. Thus, we suggest no surgical approach for treating ACs if there are no symptoms related to ACs prior to the trauma.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient has given his consent for his images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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