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Chronic Subdural Hematomas Associated with Arachnoid Cysts: Significance in Young Patients with Chronic Subdural Hematomas

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Abstract

Although arachnoid cysts (ACs) are associated with chronic subdural hematomas (CSDHs), especially in young patients, the detailed features of CSDHs associated with ACs remain poorly understood. The objective of this study was to clarify the relationship between the location of CSDHs and ACs and the significance of ACs in young patients with CSDHs. We retrospectively assessed 605 consecutive patients 7 years of age and older who were diagnosed with a CSDH between 2002 and 2014. Twelve patients (2%) had ACs, and 10 of the 12 patients were 7–40 years of age. Patients with ACs as a complication of CSDHs were significantly younger than those without ACs (p < 0.05). Three different relationships between the location of CSDHs and ACs were found: a CSDH contacting an AC, an ipsilateral CSDH apart from an AC, and a CSDH contralateral to an AC. In 21 patients with CSDHs who were 7–40 years of age, 10 (47.6%) had ACs (AC group) and 7 (33.3%) had no associated illnesses (non-AC group). All 10 young patients with ACs showed ipsilateral CSDHs including a CSDH apart from an AC. All 17 patients in both the AC and non-AC groups showed headache but no paresis at admission. The pathogenesis of CSDHs associated with ACs may be different among the three types of locations. The clinical characteristics of patients with a combination of a CSDH and an AC including headache as a major symptom may be attributed to young age in the majority of patients with ACs.

Key words: arachnoid cyst, computed tomography, chronic subdural hematoma, magnetic resonance imaging

Introduction

Arachnoid cysts (ACs) are congenital intraarachnoid malformations of the meninges.^{1,2)} The majority of ACs are asymptomatic, but some can become clinically evident as the cyst exerts a mass effect on the surrounding cerebral parenchyma.^{1,2)} ACs are also associated with chronic subdural hematomas (CSDHs), especially in young patients.³⁻¹⁵⁾ In some patients, the AC is adjacent to the CSDH, and in others, the AC is apart from the CSDH.^{4,6,8,11} Various suspected types of pathogenesis have been reported according to the relationship between the location of the AC and CSDH.^{4,6,8,11)} In terms of the clinical characteristics of the patients with a combination of a CSDH and an AC, the most common reported symptom is headache.^{6,15)} On the other hand, headache has been reported to be the most common

symptom in young patients with CSDHs, although paresis is more frequently found in conventional CSDH patients in comparison with headache.^{16–19)} To the best of our knowledge, no previous studies have elucidated whether these characteristics of a CSDH associated with an AC can be attributed to young patients with a small subarachnoid space or to a mass effect of an AC.

In this study, we investigated the relationship between the location of CSDHs and ACs using neuroimaging studies and speculated about the pathogenesis of the association. Then, we compared young patients with CSDHs associated with ACs and other young patients with only CSDHs to clarify the characteristics of CSDHs with ACs.

Methods

I. Patient population

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We retrospectively assessed consecutive patients diagnosed with a CSDH who were admitted to

our hospital between January 2002 and September 2014. Patients who had both a CSDH and an AC were included. Patients 7–40 years of age with a CSDH but not AC were also included. Both patients who underwent surgery for CSDH and conservatively treated patients with CSDH were included. Children 6 years of age or younger were excluded. Outpatients who were not admitted to our hospital were also excluded. All patients were evaluated by neurosurgeons. Our institutional review board (Institutional Review Board for Clinical Research, Tokai University Hospital, IRB No. 13R-069) approved this retrospective study of patients with CSDH who were admitted to our institution.

II. Data collection

Clinical data were obtained from a chart review. The following patient data were recorded: patient's age and sex, laterality of the hematoma, history of trauma, level of consciousness (Glasgow Coma Scale score) at admission, presence of headache and paresis, treatment including surgical methods, and outcomes. Clinical outcomes were assessed with the Glasgow Outcome Scale score at discharge. Young patients were defined as those who were 7-40 years old.

Available computed tomographies (CTs) and/or magnetic resonance images (MRIs) performed at admission were evaluated. For each patient, the maximum diameter of the CSDH was measured on slices caudal to the lateral ventricle body.

III. Statistical analysis

Univariate analyses were performed using Fisher's exact probability test for categorical variables and the Mann-Whitney U test for continuous variables. Numerical data were expressed as the median. Analyses resulting in p values less than 0.05 were considered statistically significant. All statistical analyses were performed with JMP 10 (SAS Institute Inc., Cary, North Carolina, USA).

Results

I. Association between CSDHs and ACs

Between January 2002 and September 2014, we treated 605 consecutive patients diagnosed with CSDHs. The median age of the 605 patients was 76 years (range, 8–96 years). The patients included 442 (73.1%) males and 163 (26.9%) females. Of these 605 patients, 12 (2.0%) had ACs and a median age of 32 years. Eleven patients were male and one was female. The median age of the remaining 593 patients with only a CSDH but no AC was 76 years. Patients with both a CSDH and an AC were

significantly younger than those with only a CSDH but no AC (p < 0.0001).

Table 1 shows the clinical characteristics of the 12 patients with ACs. In one patient (Case 9), an AC had been diagnosed before the onset of CSDH (Fig. 1). Two patients did not show a history of causative trauma. One of them (Case 11), who was 65 years of age, suffered from idiopathic thrombocytopenic purpura with a platelet count of $82,000/\mu m^3$. A history of sports-related trauma was present in four patients (Cases 4, 5, 6, and 8). Regarding symptoms at admission, headache was found in 11 patients and hemiparesis in only a 65-year-old male (Case 11) who did not have headache.

The location of the ACs was the middle fossa in 11 patients and the convexity of the Sylvian fissure in two. Case 3 had bilateral ACs. The 11 ACs located in the middle fossa were Galassi type I, and one was Galassi type II. CSDHs were present on the side ipsilateral to the AC in 11 of the 12 patients (Figs. 2-4). Only one CSDH (Case 12) was located on the side contralateral to the AC (Fig. 5). In 10 patients, CSDHs were close to ACs as seen with neuroimaging studies. In Case 6, a CSDH was present apart from an ipsilateral AC, and the intensity of the AC was low on a fluid-attenuated inversion recovery (FLAIR) image (Fig. 4). During open surgery of Case 6, the CSDH and the AC were confirmed to be disconnected, and the content of the AC was similar to cerebrospinal fluid. ACs appeared to have low density on CTs in 3 of the 11 patients in whom CT was performed at admission, including 1 patient with contiguity between the CSDH and AC (Case 4, Fig. 3). In all five patients including Case 4 in whom contiguity between CSDHs and ACs was shown, the intra-AC intensity was high on FLAIR images (Figs. 2, 3).

Until 2003, a craniotomy with partial removal of the membranes of the hematoma and the AC was performed as the first-choice treatment (Cases 1, 4, and 6). Since 2004, drainage of the hematoma through a burr hole has been used as the first choice. In Case 3, craniotomy was performed 16 days after burr hole surgery because of regrowth of the hematoma as seen on CT. All 12 patients recovered completely. No recurrence of the CSDHs was observed during a follow-up period of 1–92 months (median 16 months).

II. CSDH in young patients 7-40 years old

Of the 605 patients with CSDHs, 21 patients (3.5%) were 7–40 years of age, and the remaining 584 patients (96.5%) were older than 40 years. Of the 21 patients who were 7–40 years old, 10 (47.6%) had ACs, 3 (14.3%) suffered from intracranial hypotension, 1 (4.8%) had hemorrhagic tendency after bone marrow

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Case no.	Age, year/sex	Trauma	Interval	Symptoms	Side/location	*CT density	⁺ MRI/ FLAIR intensity	Side	Maximum diameter (mm)	between cyst and hematoma	Treatment
	8/M	Traffic accident	12 w	Headache vomiting	L/Middle fossa	High	n.a.	Ч	11	Υ	Craniotomy
2	13/M	Bicycle accident	7 w	Headache	L/Middle fossa	n.a.	High	Γ	19	Υ	Bur hole
c S	14/M	Aggression	6 w	Headache	R/Sylvian L/Middle fossa	High	High	К	10	Υ	Bur hole Craniotomy
4	15/M	Soccer-related	8 W	Headache	L/Middle fossa	Low	High	Γ	6	Υ	Craniotomy
5	31/M	Judo-related	$4 \mathrm{w}$	Headache	R/Middle fossa	High	n.a	R	14	Υ	Bur hole
9	32/M	Snowboard-related	20 w	Headache	L/Middle fossa	Low	Low	Γ	20	Z	Craniotomy
4	32/M	I	Ι	Headache	R/Middle fossa	High	High	R	6	Υ	Bur hole
8	35/F	Ski-related	16 w	Headache [‡] GCS 8	L/Middle fossa	High	n.a.	Γ	25	Υ	Bur hole
6	40/M	Fall	20 w	Headache	L/Middle fossa	High	High	Γ	23	Υ	Bur hole
10	40/M	Fall	3 W	Headache nausea	L/Sylvian	High	n.a.	Γ	11	Υ	Bur hole
11	65/M	I	I	Paresis	L/Middle fossa	High	n.a.	Γ	15	Υ	Bur hole
12	71/M	Fall	6 W	Headache	R/Middle fossa	Low	n.a.	Γ	6	N	Conservative

Table

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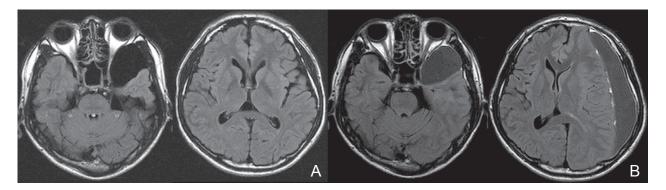


Fig. 1 Magnetic resonance image/fluid-attenuated inversion recovery (FLAIR) images in Case 9 of an arachnoid cyst diagnosed before the onset of chronic subdural hematoma. A: A FLAIR image at a medical check-up 2 years before the onset of chronic subdural hematomas (CSDH) showing an arachnoid cyst in the left middle fossa. B: A FLAIR image at admission demonstrating a left CSDH. Intensity of the arachnoid cyst becomes high.

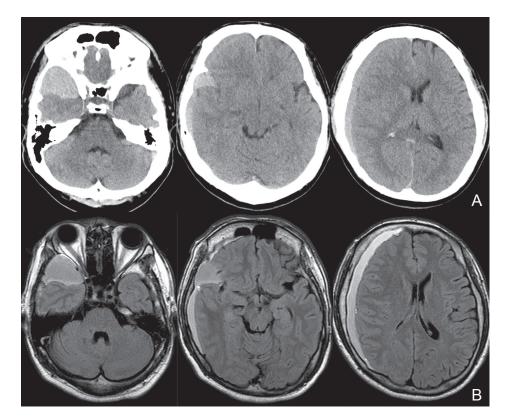


Fig. 2 Images of Case 7 in which a chronic subdural hematoma is close to an arachnoid cyst. A: A computed tomography showing a high-density arachnoid cyst in the right middle fossa. B: A magnetic resonance image/fluid-attenuated inversion recovery image demonstrating high intensity in the arachnoid cyst.

transplantation for fanconi anemia, and 7 showed no other associated illnesses (33.3%). On the other hand, of the 584 patients who were older than 40 years, only 2 patients (0.3%) had ACs. In terms of a relationship between the location of a CSDH and an AC, all 10 young patients with a combination of a CSDH and an AC showed that a CSDH was located on the ipsilateral side to an AC. In 9 of the 10 patients, a CSDH was close to an AC, and the remaining 1 had a CSDH apart from an AC.

Table 2 shows a comparison between the 10 patients with ACs and the 7 patients without any

other diseases in patients who were 7–40 years old with CSDHs. All four patients with CSDHs who were younger than 20 years of age showed ACs. Two patients without ACs had a history of sports-related trauma, which occurred during American football training and snowboarding. All 17 patients had headache but no paresis. All 17 patients underwent surgery, and their outcomes at discharge were good. Between the two groups, no significant difference was found in age, sex, frequency of trauma history, frequency of sports-related trauma, consciousness level at admission, hematoma thickness, or outcome.

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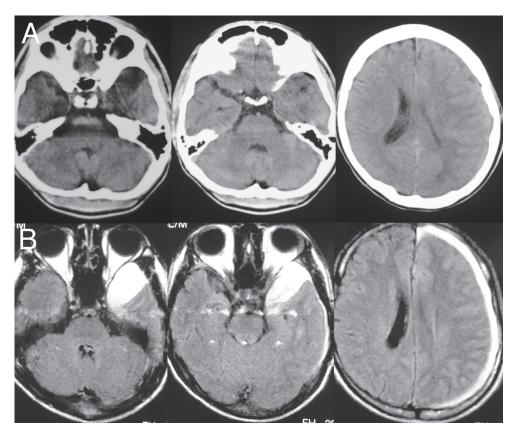


Fig. 3 Images of Case 4 in which a chronic subdural hematoma is close to an arachnoid cyst. A: A computed tomography showing a lowdensity arachnoid cyst in the left middle fossa. B: A magnetic resonance image/ fluid-attenuated inversion recovery image demonstrating high intensity in the arachnoid cyst.

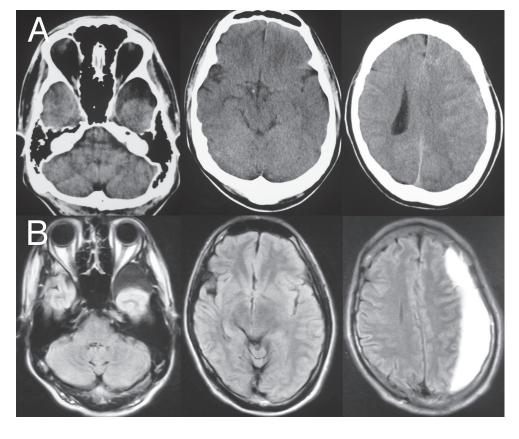


Fig. 4 Images of Case 6 in which a chronic subdural hematoma is apart from an arachnoid cyst on the ipsilateral side. A: A computed tomography showing a lowdensity arachnoid cyst in the left middle fossa. B: A magnetic resonance image/ fluid-attenuated inversion recovery image demonstrating low intensity in the arachnoid cyst.

Fig. 5. A computed to reproduce of the sharing a sharing

Fig. 5 A computed tomography showing a chronic subdural hematoma located on the side contralateral to an arachnoid cyst (Case 12). Enlargement of the bilateral subarachnoid space indicates brain atrophy.

Table 2Comparison between patients with and without
arachnoid cysts in 17 patients, 7-40 years of age with
chronic subdural hematomas

Variable	Patients with arachnoid cyst (n = 10)	Patients without arachnoid cyst (n = 7)	p value
Age (median, years)	31.5	34	0.434
Age < 20 years	4 (40%)	0 (0%)	0.056
Sex, male	9 (90%)	6 (85.7%)	1
Trauma history	9 (90%)	6 (85.7%)	1
Sports-related	4 (40%)	2 (42.9%)	1
Consciousness disturbance at admission	1 (10%)	0 (0%)	1
Headache	10 (100%)	7 (100%)	1
Paresis	0 (0%)	0 (0%)	1
Hematoma thickness (median, mm)	12.5	11	0.301
Good recovery at discharge	8 (100%)	9 (100%)	1

Discussion

The present study showed 12 patients (2.0%) with ACs among 605 patients with CSDHs. The frequency of ACs in those with CSDHs, which was 2.0%, was comparable to the previously reported prevalence of 2-2.5%.^{8,13,15} The patients with ACs were significantly younger than the patients without ACs, consistent with previous studies.^{8,15}

I. Imaging studies and the mechanism of CSDHs associated with ACs

Three types of relationships were found between the location of ACs and CSDHs on imaging studies: a CSDH close to an AC, a CSDH apart from an AC on the ipsilateral side, and a CSDH on the side contralateral to an AC. In the case of a CSDH close to an AC, the following mechanisms have been suggested. Observation of small bridging vessels between the dura and outer membrane of the AC during surgery for AC without CSDH in the middle fossa has been reported, and detachment of the AC membrane from the dura by traumatic forces was suspected to cause bleeding from the vessels.^{7,15)} Wester et al. reported the intraoperative observation that the bridging Sylvian veins traversed an AC membrane and entered the dural venous sinus. They suggested that the bridging veins could induce leakage of blood into the subdural space from the entry point of these vessels.¹⁵⁾ Tearing of the outer wall of an AC after head injury is also speculated to induce subdural effusion, which could develop into a CSDH.²⁰⁻²²⁾ Page et al. reported that ACs are less pliable than the normal brain, resulting in reduced intracalvarial cushioning following trauma. Consequently, hemorrhage may occur in the ipsilateral hemispheric subdural space apart from the AC.^{11,12}) This mechanism explains the occurrence of the CSDH apart from the AC on the ipsilateral side in Case 6. The patient of Case 6, who was 32 years old, was equal to the median age of this patient series of CSDHs with an AC as a complication. We also considered that the association of a CSDH and an AC in Case 6 was not coincidental. In Case 12, the occurrence of the CSDH on the side contralateral to the AC may be incidental, because this patient was 71 years old, which was close to the average age of the 605 CSDH patients in this study and because brain atrophy was shown on CT (Fig. 5).

In all patients with a CSDH in contact with an AC, MRI/FLAIR imaging showed high intensity in the AC even though a CT showed low density in the AC. When an AC is in contact with a CSDH, blood breakdown products may filter through the dividing anatomical membrane and alter the cerebrospinal fluid signal of the AC on MRI/FLAIR images, but they are insufficient to alter the density of the signal on a CT image.⁶⁾ On the other hand, an MRI/FLAIR image showed a low-intensity AC indicating cerebrospinal fluid in the AC that was apart from the CSDH. This was confirmed during craniotomy in Case 6.

II. Significance of ACs in young patients with CSDHs

In the present series of 605 CSDH patients, 21 patients (3.5%) were 40 years of age or younger.

Previously reported rates of patients who were 40 years of age or younger in an entire population of CSDH patients in the CT era were 2.4-8.8%, which was compatible to the rate of this study.^{16,17} A combination of ACs and CSDHs was found in 10 of 12 patients who were 40 years of age or younger. To exclude an effect of age on characteristics of CSDHs associated with ACs, among those who were 7-40 years old and who had CSDHs, we compared 10 patients with ACs and 7 patients with no other diseases. In this study, young patients were defined as 40 years or younger according to the previous studies.^{16–19)} Patients under 7 years of age were excluded because a major cause of CSDH in these patients is brain atrophy after cerebral infarction induced mainly by child abuse.²³⁾ In the young patients with CSDHs, no significant difference was demonstrated in age, sex, trauma history, consciousness level, occurrence of headache, occurrence of paresis, and outcomes between patients with and without ACs. Clinical characteristics may not differ between young CSDH patients with and without ACs, though no definitive conclusion was drawn from the results because of the small number of patients who were 7-40 years of age in this study.

In the present series, approximately half of the patients 7–40 years of age with CSDHs had ACs. Considering the relatively low reported AC prevalence, which is 0.2-1.7%,^{1,24–27)} the presence of an AC is the most influential factor for occurrence of a CSDH in young patients. In terms of symptoms, headache was found in all patients who were 7–40 years of age with and without ACs, and no patients showed paresis. Several studies reported that headache is more common in young patients with CSDHs as a manifestation of increased intracranial pressure.^{16–19)} Young patients may be more affected by increased intracranial pressure if the CSDH enlarges, because the subarachnoid space is smaller in comparison with older individuals.¹⁶⁾

A sports-related CSDH was found in four young patients with ACs and two young patients without ACs. Several cases of CSDHs associated with ACs after sports injuries have been reported.^{28–31)} Sport is an important cause of CSDHs in young patients.¹⁷⁾ If a patient with an AC shows refractory headache after sport, falling, or even no apparent previous injury, neuroimaging studies might be recommended to differentiate the complication of a CSDH.

III. Treatment for a CSDH associated with an AC

In the present series, craniotomy with partial removal of the membranes of the hematoma and the AC was performed in the early period, and drainage of the hematoma through a burr hole was performed in the latter period. In one patient, craniotomy was performed after burr hole surgery because of hematoma regrowth. The treatment of a CSDH associated with an AC is still controversial regarding whether removal of the membrane through a craniotomy or a simple burr hole craniostomy is selected as the first choice.^{6,7,10} Recently, most studies have suggested that burr hole irrigation should be chosen as the first treatment. In case of recurrence after burr hole surgery, removal of the membranes of an AC through a craniotomy is necessary.^{6–8,10,14}

Conclusion

In 605 patients with CSDHs, the 12 patients with ACs were significantly younger than the remaining 593 patients without ACs. Three types of relationships regarding the location between CSDHs and ACs were recognized on neuroimaging studies, and the types of pathogenesis were likely different from each other. Approximately half of the patients 7–40 years of age with CSDHs had an AC as a complication. Headache, which was a characteristic symptom in patients with ACs, was also the most frequent symptom in young patients without ACs who were 7–40 years of age.

Conflicts of Interest Disclosure

All authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this article. All authors have registered Self-reported COI Disclosure Statement Forms online through the website for The Japan Neurosurgical Society members.

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