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Surgical Treatment of Chronic Subdural Hematoma in 500 Consecutive Cases: Clinical Characteristics, Surgical Outcome, Complications, and Recurrence Rate

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Abstract

Chronic subdural hematoma (CSDH) is one of the most common clinical entities in daily neurosurgical practice. The diagnosis and treatment are well established, but recurrence, complications, and factors related to these problems, especially in the elderly, are not completely understood. This study evaluated the clinical features, radiological findings, and surgical results in a large series of the patients treated at the same institution. 500 consecutive patients (359 men and 141 women) with CSDH were treated by burr hole craniostomy with closed system drainage from January 1987 through February 1999. Causes, clinical and computed tomographic findings, surgical results, re-expansion of brain after surgery, and hematoma recurrence were statistically analyzed to elucidate the potential risks of CSDH. Most patients (89.4%) had good recovery, 8.4% showed no change, and 2.2% worsened. Six patients (1.2%) died, three due to disseminated intravascular coagulation. Recurrence of hematoma was recognized in 49 patients (9.8%), at 1 to 8 weeks (3.5 ± 1.9 weeks) after the first operation. The brain re-expansion rate at one week after operation was 45.0 ± 21.4% in patients with hematoma recurrence and significantly lower than 55.3 ± 19.1% in patients without recurrence ($p < 0.001$). Old age, pre-existing cerebral infarction, and persistence of subdural air after surgery were significantly correlated with poor brain re-expansion ($p < 0.001$). Twenty-seven patients (5.4%) suffered postoperative complications, of which 13 cases were acute subdural hematoma caused by incomplete hemostasis of the scalp wound and four cases were tension pneumocephalus. Careful hemostasis and complete replacement of subdural hematoma by normal saline to prevent influx of air into the subdural space will further improve the surgical outcome for patients with CSDH.

Key words: chronic subdural hematoma, outcome, recurrence, re-expansion, surgical complications

Introduction

Chronic subdural hematoma (CSDH) is one of the most common clinical entities encountered in daily neurosurgical practice. The causes, clinical characteristics, and therapeutic management are well established, and many neurosurgeons pay less attention to the problems posed by CSDH. However, CSDH tends to occur in elderly patients, some of whom suffer clinical recurrence after the first operation, and the postoperative complications are not negligible. Therefore, accurate assessment of the problems, such as complications, recurrence, and related factors, is important. No cumulative data focused on these problems of CSDH has been reported since 1990. Such data is useful to elucidate clinical problems and to better inform the patients and their families.

We describe our series of 500 consecutive surgical cases of CSDH in our institution, and retrospectively analyze the clinical characteristics, causes, surgical results, complications, recurrence rate, and re-expansion of brain. Factors related to hematoma recurrence and brain re-expansion were also statistically analyzed.

Clinical Material and Methods

500 consecutive patients with CSDH were treated by hematoma evacuation and drainage through cranial burr holes in the Department of Neurosurgery, Juntendo University Izunagaoka Hospital from January 1987 to February 1999. Table 1 summarizes the clinical characteristics. There were 359 men and 141

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Table 1 Clinical characteristics of 500 patients with chronic subdural hematoma

Sex	
male	359 cases (71.8%)
female	141 cases (28.2%)
Age	
male	67.3 ± 15.3 yrs
female	71.3 ± 14.2 yrs
Hematoma location	
left	260 cases (52.0%)
right	152 cases (30.4%)
bilateral	88 cases (17.6%)
Hematoma thickness	
left	20.5 ± 5.8 mm
right	20.3 ± 5.2 mm
bilateral (sum)	29.6 ± 9.0 mm
Hematoma density on CT	
low	33.8%
iso	27.0%
high	20.2%
niveau	19.0%

Data are expressed as mean ± standard deviation. CT: computed tomography.

Table 2 Coexisting diseases in 500 patients with chronic subdural hematoma

Coexisting disease	No. of patients (%)
Arterial hypertension	93 (18.6)
Diabetes mellitus	40 (8.0)
Hepatitis	23 (4.6)
Liver cirrhosis	12 (2.4)
Gastric ulcer	4 (0.8)
Renal failure	3 (0.6)
Cardiac arrhythmia	9 (1.8)
Angina pectoris	5 (1.0)
Myocardial infarction	4 (0.8)
Cancer	19 (3.8)
Cerebral infarction	21 (4.2)
Intracerebral hemorrhage	7 (1.4)
Parkinsonism	2 (0.4)

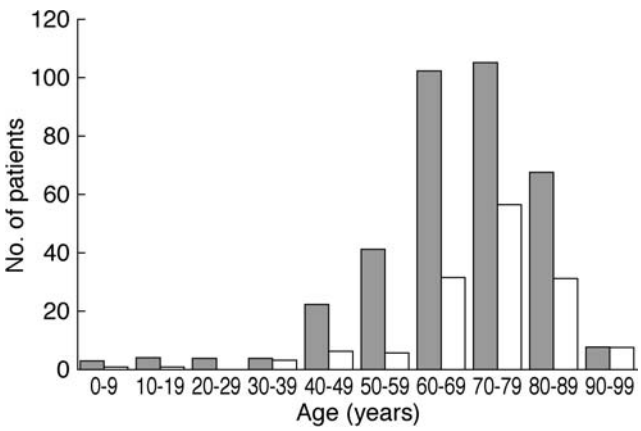

Fig. 1 Age distributions of 359 male (*closed columns*) and 141 female (*open columns*) patients with chronic subdural hematoma, showing a gradual increase in incidence for each decade and the peak in the eighth decade.

women aged from 0 to 99 years. Figure 1 shows the age distribution. The incidence gradually increased in each decade and peaked in the eighth decade (161 cases, 32.2%). The mean age was 67.3 ± 15.3 years for the male patients and significantly higher ($p < 0.01$, t-test) at 71.3 ± 14.2 years for the female patients. There were only seven pediatric patients (younger than 15 years old) with CSDH (1.4% of all cases).

Computed tomography (CT) studies were performed in all patients to confirm the diagnosis and for follow up. CSDHs were located on the left side in 260 cases (52.0%), the right side in 152 cases (30.4%), and bilaterally in 88 cases (17.6%). CT demonstrated CSDHs as hypodense (33.8%) to the cerebral parenchyma, isodense (27.0%), hyperdense (20.2%), and niveau formation (19.0%). The mean preoperative hematoma thickness was 20.5 ± 5.8 mm on the left side, 20.3 ± 5.2 mm on the right side, and was significantly thicker at 29.6 ± 9.0 mm in the bilateral cases ($p < 0.001$, one way analysis of variance [ANOVA]).

Table 2 shows the coexisting diseases in the 500 patients with CSDH. Arterial hypertension (18.6%) was the most common, followed by diabetes mellitus (8.0%) and cerebral apoplexy (cerebral infarction 4.2% and intracerebral hemorrhage 1.4%).

Under general anesthesia, hematomas were evacuated and irrigated with normal saline through two burr holes and closed system subdural drainage was continued for 1 or 2 days after the operation. One case was recognized as calcified hematoma during operation and craniotomy was subsequently performed to remove this organized hematoma.

Routine CT was performed the day after surgery and one week after surgery. The preoperative hematoma thickness and postoperative hematoma thickness (one week after operation) were measured by CT to calculate the re-expansion rate as (preoperative hematoma thickness − postoperative hematoma thickness)/preoperative hematoma thickness × 100 (%). Patients with no complications were discharged on the 8th day postsurgery and followed up for at least 3 months in our clinic.

Neurological assessment was performed one month after the operation to compare the preoperative and postoperative states. Return to the predisease condition was considered good recovery.

Postoperative condition was classified as good recovery, no change, worse, and dead.

Data are given as the mean ± standard deviation. Results were analyzed for statistical significance using the unpaired Student's t-test, Pearson's χ^2-test, and ANOVA with $p < 0.05$ considered significant.

Results

I. Causes

Table 3 shows the origins of CSDH. A definite history of head injury was obtained in 286 patients (57%) and a past history of neurosurgery operation

Table 3 Causes in 500 patients with chronic subdural hematoma

Cause	No. of patients (%)		
Head injury	286 (57)	Severity of head injury	No. of patients (%)
		mild head injury (GCS 15–13)	260 (90.9)
		transient LOC including PTA	9 (3.1)
		moderate head injury (GCS 12–9)	6 (2.1)
		severe head injury (GCS 8–3)	7 (2.4)
		unknown	4 (1.4)
		Total	286
		Intracranial bleeding	No. of patients
		ASH	8
		brain contusion	7
		traumatic SAH	3
		SAH + brain contusion	1
		Total	19
Neurosurgery	42 (8)	Surgical procedure	No. of patients
		aneurysm clipping	23
		VP shunt for idiopathic NPH	4
		arachnoid cyst opening	3
		removal of ICH	3
		craniotomy for head injury	2
		craniectomy for cerebellar infarct	1
		AVM removal	1
		STA-MCA anastomosis	1
		skull base reconstruction	1
		CP shunt	1
		removal of ventricular tumor	1
		unknown	1
		Total	42
Alcoholism	32 (6)		
Anticoagulant drug	26 (5)	Drug administration	No. of patients
		ticlopidine	13
		aspirin	5
		dipyridamole	4
		warfarin	2
		aspirin + cilostazol	1
		aspirin + ticlopidine	1
		Total	26
Coagulopathy	14 (3)		
Arachnoid cyst	9 (2)		
Unknown	144 (29)		

ASH: acute subdural hematoma, AVM: arteriovenous malformation, CP: cystoperitoneal, GCS: Glasgow Coma Scale, ICH: intracerebral hemorrhage, LOC: loss of consciousness, NPH: normal pressure hydrocephalus, PTA: post-traumatic amnesia, SAH: subarachnoid hemorrhage, STA-MCA: superficial temporal artery-middle cerebral artery, VP: ventriculoperitoneal.

in 42 patients (8%). The causes of head injury were falling during walking (148 cases), traffic accident (66 cases), falling from a height (38 cases), struck by another person (17 cases), and others (17 cases). Most head injuries were mild without consciousness disturbance. Mild head injury (Glasgow Coma Scale [GCS] 15–13 points) occurred in 260 patients (90.9%), moderate head injury (GCS 12–9 points) in six patients (2.1%), and severe head injury (GCS 8–3 points) in seven patients (2.5%). Transient loss of consciousness or posttraumatic amnesia was recognized in nine patients (3.1%). Nineteen of 286 patients had intracranial hemorrhage: acute subdural hematoma in eight, brain contusion in seven, traumatic subarachnoid hemorrhage in three, and traumatic subarachnoid hemorrhage with brain contusion in one. The mean interval from head injury to operation was 8.3 ± 4.8 weeks.

Most of the 42 patients (8%) with a past history of neurosurgery underwent aneurysm clipping (23 cases) including additional ventriculoperitoneal (VP) shunting to control hydrocephalus (17 cases). The mean interval from the neurosurgery operation to burr hole craniostomy was 10.5 ± 5.6 weeks. The interval for neurosurgical cases is significantly longer than that for head injuries ($p < 0.05$, t-test).

History of alcoholism was recognized in 32 patients (6%). Twenty-six patients (5%) had received anticoagulant therapy (ticlopidine in 13 cases, aspirin in 5 cases, dipyridamole in 4 cases, warfarin in 2 cases, aspirin + cilostazol in 1 case, aspirin + ticlopidine in 1 case). Coagulopathy caused by malignant tumor and/or disseminated intravascular coagulopathy (DIC) was noted in 14 patients (3%).

Arachnoid cyst was identified by CT in nine patients, four of whom were pediatric patients (<15 years old). Four of the seven pediatric patients with CSDH also had arachnoid cysts. The incidence of association with arachnoid cyst in pediatric patients (57.1%) is significantly higher ($p < 0.001$) than in adult patients (1.0%). The arachnoid cysts were located in the middle fossa (7 cases), frontal convexity (1 case), and posterior fossa (1 case). Interestingly, seven of these nine patients with arachnoid cyst suffered CSDH in the same side.

Some patients had several causative factors at once. 144 patients (29%) did not have any definite causes of CSDH but these include patients who could not remember any episode of head injury.

II. Clinical findings

The clinical presentations are summarized in Table 4. The most frequent symptom was gait disturbance (315 cases, 63.0%) followed by hemiparesis (293 cases, 58.6%) and headache (191 cases, 38.2%).

Table 4 Presenting symptoms in 500 patients with chronic subdural hematoma

Symptom	No. of patients (%)
Single symptom	
gait disturbance	315 (63.0)
hemiparesis	293 (58.6)
headache	191 (38.2)
dementia	123 (24.6)
incontinence	87 (17.4)
consciousness disturbance	83 (16.6)
vomiting	15 (3.0)
convulsion	12 (2.4)
anisocoria (brain herniation)	10 (2.0)
motor aphasia	9 (1.8)
Combined symptoms (2)	
gait disturbance + hemiparesis	240 (48.0)
gait disturbance + dementia	103 (20.6)
headache + hemiparesis	83 (16.6)
gait disturbance + incontinence	80 (16.0)
Combined symptoms (3)	
gait disturbance + hemiparesis + headache	45 (9.0)
gait disturbance + hemiparesis + dementia	33 (6.6)
gait disturbance + hemiparesis + consciousness disturbance	10 (2.0)

Headache was recognized in 71.3% of patients less than 60 years old and 30.5% of patients more than 60 years old, significantly more frequent in the younger age group ($p < 0.001$, χ^2-test). Dementia (123 cases, 24.6%) and urinary incontinence (87 cases, 17.4%) were also frequently recognized. Dementia was significantly frequently recognized in bilateral cases (33.0%) compared with the left cases (25.4%) and right cases (18.4%) ($p < 0.05$, χ^2-test). Consciousness disturbance was present in 83 patients (16.6%). Brain herniation with pupillary dilatation was recognized in 10 patients (2.0%). CSDH may cause parkinsonism (tremor at rest, rigidity, and bradykinesia) but only two patients (0.4%) showed parkinsonism.[43]

CSDH was associated with several symptoms. The most frequent were gait disturbance and hemiparesis (240 cases, 48.0%) followed by gait disturbance and dementia (103 cases, 20.6%). CT follow up after neurosurgery or head injury in 73 patients detected CSDH without symptoms in 30 patients.

III. Outcome

One month after burr hole craniostomy, 447 of 500 patients (89.4%) showed good recovery, 42 patients (8.4%) showed no change, 11 patients (2.2%) worsened, and six (1.2%) of these latter 11 patients died. Causes of death were DIC (3 cases), cerebral infarction (2 cases), and multiple organ failure (1 case).

Patients who showed no change or worsened were considered to have poor outcome (53 patients). The correlations between clinical, etiological, and symptomatic factors were examined in this poor outcome group (Table 5). Age had no influence as the

Table 5 Summary of variables related to poor outcome in 53 patients with chronic subdural hematoma

Variable	Percentage	p Value (χ^2-test)
Sex		NS
male	10.6%	
female	10.6%	
Age		NS
≧80 yrs	10.7%	
<80 yrs	10.6%	
Causes		
head injury		NS
yes	9.4%	
no	12.1%	
neurosurgery		<0.0001
yes	42.9%	
no	7.6%	
alcoholism		NS
yes	9.4%	
no	10.7%	
anticoagulant drug		NS
yes	18.5%	
no	10.1%	
coagulopathy		<0.0001
yes	50.0%	
no	9.5%	
arachnoid cyst		NS
yes	11.1%	
no	10.6%	
Symptoms		
gait disturbance		<0.001
yes	6.3%	
no	17.8%	
hemiparesis		<0.001
yes	4.1%	
no	19.8%	
headache		<0.001
yes	1.6%	
no	16.2%	
dementia		NS
yes	10.6%	
no	10.6%	
incontinence		NS
yes	9.2%	
no	10.9%	
consciousness disturbance		NS
yes	12.0%	
no	10.3%	
convulsion		<0.01
yes	41.7%	
no	9.8%	
anisocoria (brain herniation)		NS
yes	30.0%	
no	10.2%	

χ^2-test: Pearson's chi-square test, NS: not significant.

Table 6 Summary of variables related to re-expansion rate in 500 patients with chronic subdural hematoma

Variable	Re-expansion rate (%)	p Value (t-test)
Sex		NS
male	55.5 ± 19.3	
female	51.1 ± 19.7	
Age		<0.001
<70 yrs	58.9 ± 20.2	
≧70 yrs	50.4 ± 18.1	
Coexisting disease		
arterial hypertension		NS
yes	55.6 ± 17.1	
no	54.0 ± 20.0	
diabetes mellitus		NS
yes	56.5 ± 15.7	
no	54.1 ± 19.8	
cerebral infarction		<0.001
yes	40.2 ± 19.5	
no	54.9 ± 19.3	
Subdural air accumulation after operation		<0.001
yes	45.3 ± 18.3	
no	56.6 ± 19.2	
Causes		
head injury		NS
yes	53.8 ± 19.7	
no	54.9 ± 19.3	
neurosurgery		NS
yes	55.2 ± 24.7	
no	54.2 ± 19.0	
alcoholism		NS
yes	49.9 ± 20.9	
no	54.6 ± 19.4	
anticoagulant drug		<0.005
yes	39.6 ± 25.1	
no	54.7 ± 19.2	
arachnoid cyst		<0.001
yes	77.2 ± 14.6	
no	53.9 ± 19.4	

Data are expressed as mean ± standard deviation. NS: not significant, t-test: Student's non-paired t-test.

outcome of patients 80 years old or more did not differ from that of patients less than 80 years old. Patients with a past history of neurosurgery had a significantly higher rate of poor outcome ($p < 0.0001$). However, 22 of the 42 patients who underwent neurosurgery had CSDH detected by CT after neurosurgery and 12 had CSDH incidentally found without symptoms. Therefore, most outcomes were defined as no change. Seven of 14 patients who had coagulopathy showed significantly poor outcome. Three of the 14 patients already had brain herniation before surgery because of large subdural hematoma, one was in extremely poor condition, and three died of DIC after the surgery. The outcome for patients with gait disturbance, hemiparesis, and headache was significantly better ($p < 0.001$). Patients with convulsion had a significantly poor outcome ($p < 0.01$) and three of these 12 patients also had coagulopathy. Indeed, coagulopathy is an ominous indicator of poor prognosis in patients with CSDH.

The re-expansion rate of the brain was evaluated at one week after burr hole craniostomy and related variables were analyzed (Table 6). The re-expansion rate in patients who were 70 years old or more was significantly lower than that of patients who were less than 70 years old ($p < 0.001$). Figure 2 shows the correlation between re-expansion rate and age (coefficient of correlation 0.247, $p < 0.001$). Clearly, younger patients showed better re-expansion of the brain after the surgery. Coexisting diseases such as arterial hypertension and diabetes mellitus did not correlate with the re-expansion of brain, but the re-

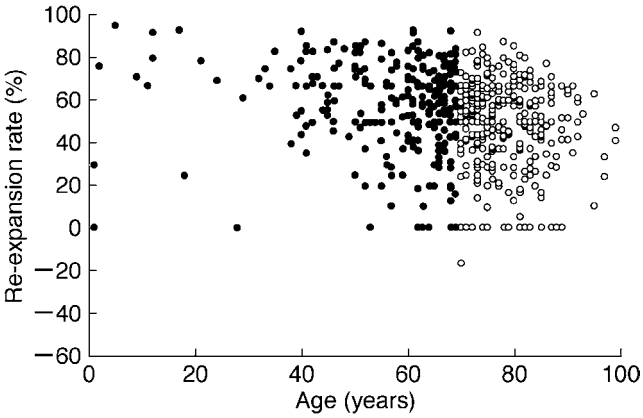

Fig. 2 Correlation between age and re-expansion rates of 500 patients with chronic subdural hematoma at 1 week after burr hole craniostomy. *Closed circles* indicate patients aged less than 70 years and *open circles* indicate patients aged 70 years or older.

expansion rate of 40.2 ± 19.5% in patients with pre-existing cerebral infarction was significantly lower than 54.9 ± 19.3% in patients without cerebral infarction ($p < 0.001$). The re-expansion rate of 45.3 ± 18.3% in patients with subdural air accumulations was significantly lower than 56.6 ± 19.2% in patients without subdural air accumulations ($p < 0.001$). Air in the subdural space physically blocked re-expansion of the brain after evacuation of CSDH. The re-expansion rate of 39.6 ± 25.1% in patients with anticoagulant therapy was significantly lower than 54.7 ± 19.2% in patients without anticoagulant therapy ($p < 0.005$). Eleven of these 26 patients with anticoagulant therapy had a past history of cerebral infarction. The presence of old cerebral infarction might affect re-expansion. The re-expansion rate in patients with arachnoid cyst was 77.2 ± 14.6% and significantly high ($p < 0.001$) because most were pediatric patients.

Age, presence of air in the subdural space after surgery, and pre-existing cerebral infarction were thought to be the factors affecting re-expansion of the brain after surgery. In the present study, age was correlated to poor re-expansion of the brain after surgery, but age (even 80 years old or more) was not correlated to poor outcome (Tables 5 and 6).

IV. Recurrence of CSDH

Recurrence of CSDH after first burr hole craniostomy was recognized in 49 patients (9.8%). The interval from the first operation to the reoperation ranged from 1 to 8 weeks (mean 3.5 ± 1.9 weeks). Recurrence was on the same side in 31 cases and on the contralateral side in 18 cases. Retrospective re-examination of the CT scans of the 18 contralateral cases found 14 cases already showed the presence of thin subdural hematoma or effusion in the contralateral side at the first operation.

Correlations between clinical, etiological, and radiological factors and recurrence were examined (Table 7). Age, etiology, and CT findings had no correlation with recurrence. Patients receiving anticoagulant drugs had a high recurrence rate (18.5%) but not statistically significant. The interval from trauma to first operation was 7.2 ± 3.8 weeks in patients with recurrence and 8.5 ± 5.0 weeks in patients without recurrence but this difference was not statistically significant. The only factor correlated with recurrence was the re-expansion rate at one week after the surgery. The re-expansion rate of 45.0 ± 21.4% in patients with recurrence was significantly lower than 55.3 ± 19.1% in patients without recurrence ($p < 0.001$).

The presence of thin subdural hematoma or effusion on the contralateral side and poor re-expansion rate of the brain after burr hole craniostomy are the causative factors of hematoma recurrence.

V. Postoperative complications

Postoperative complications developed in 27 patients (5.40%) as in Table 8. Central nervous system (CNS) complications occurred in 23 patients. The most common complication was acute subdural hematoma, which occurred in 13 patients (2.6%). One of these 13 patients developed acute subdural hematoma due to pre-existing DIC and died. The other 12 cases of acute subdural hematoma were caused by fresh bleeding from the scalp wound. Eight acute subdural hematomas were evacuated by craniotomy and five cases were evacuated through burr holes. All 12 survivors had no permanent neurological deficits. Tension pneumocephalus occurred in four patients, three with bilateral CSDHs and one with unilateral CSDH. All patients with tension pneumocephalus showed consciousness disturbance and two suffered brain herniation. All of these four patients were immediately treated by reopening the operative wound to evacuate the air in the subdural space. Two patients had cerebral infarction, in the posterior cerebral artery area and in the internal carotid artery area. Both patients had permanent neurological deficits (visual field deficit and hemiparesis). The other CNS complications were putaminal hemorrhage (1 case), acute epidural hematoma (1 case), subdural empyema (1 case), and scalp wound infection (1 case).

General postoperative complications were recog-

Table 7 Factors related to recurrence in 500 patients with chronic subdural hematoma

Factor	Recurrence (N = 49)	No recurrence (N = 451)	p Value
Sex			NS (χ^2-test)
male	10.3%	89.7%	
female	8.5%	91.5%	
Age			NS (χ^2-test)
<70 yrs	9.7%	90.3%	
$\geqq 70$ yrs	9.9%	90.1%	
Interval from trauma to first operation	7.2 ± 3.8 wks	8.5 ± 5.0 wks	NS (t-test)
Re-expansion rate	45.0 ± 21.4%	55.3 ± 19.1%	<0.001 (t-test)
Subdural air accumulation after operation	30.6%	19.5%	NS (χ^2-test)
Causes of chronic subdural hematoma			NS (χ^2-test)
head injury	9.1%	10.7%	
neurosurgery	11.9%	9.6%	
alcoholism	15.6%	9.4%	
anticoagulant drug	18.5%	9.3%	
coagulopathy	0.0%	10.1%	
arachnoid cyst	11.1%	9.8%	
CT findings			NS (χ^2-test)
low density	10.1%	89.9%	
iso density	13.3%	86.7%	
high density	5.0%	95.0%	
niveau formation	9.5%	90.5%	

Data are expressed as mean ± standard deviation. CT: computed tomography, χ^2-test: Pearson's chi-square test, NS: not significant, t-test: Student's non-paired t-test.

Table 8 Summary of postoperative complications in 27 patients with chronic subdural hematoma

Complication	No. of patients
Central nervous system complications	23
acute subdural hematoma	13
tension pneumocephalus	4
cerebral infarction	2
putaminal hemorrhage	1
acute epidural hematoma	1
subdural empyema	1
wound opening	1
General complications	4
pneumonia	2
ileus	1
DIC	1

DIC: disseminated intravascular coagulation.

nized in four patients (0.8%). Two patients suffered from pneumonia and one of them died. The other general complications were ileus (1 case) and DIC (1 case).

Overall, four patients died of postoperative complications and four patients suffered worsening of neurological signs because of complications.

Discussion

The etiology of CSDH is not completely understood. Traumatic subdural effusion is widely accepted as a preliminary stage in the development of CSDH.[28,30] Traumatic subdural effusion is a result of arachnoid tearing caused by head injury or neurosurgery and this fluid with or without blood in the subdural space facilitates the formation of so-called ''outer membrane.''[4,11,36] This outer membrane then forms internal capillaries or sinusoids. These fenestrated blood vessels allow plasma fluid leakage into and resultant enlargement of the subdural space.[2,25–27] Bleeding then occurs repeatedly from capillaries with degenerating endothelium, and is accompanied by local hyperfibrinolysis, which is one of the causes of the growth of effusions into CSDH.[8,13,14] Several experimental studies of CSDH have shown that contact of cerebrospinal fluid (CSF) and/or fibrin in the blood with the dura mater is an essential precursor to outer membrane formation.[1,42]

Some of our clinical data also suggest factors in the etiology of CSDH. The present series included 42 patients with a past history of neurosurgery and 21 patients with VP shunts (4 cases for normal pressure hydrocephalus, 17 cases after clipping surgery) to control hydrocephalus. Twenty-three of these 42 patients underwent aneurysm clipping surgery, which requires opening of the arachnoid membrane

and CSF leaks into the subdural space through this arachnoid tear. Interestingly, only one patient underwent removal of brain tumor, an intraventricular meningioma, by the paramedian superior parietal transcortical transventricular approach. These facts suggest that neurosurgical procedures which open the CSF space and intracranial hypotension caused by VP shunt procedures might facilitate the formation of postoperative CSDH.[6,39] Our series also included seven pediatric patients with CSDH, four of whom had associated arachnoid cysts. This high incidence of association with arachnoid cyst in pediatric cases was statistically significant. Tearing of the arachnoid cyst wall with or without bleeding after head injury may be a causative factor of CSDH in children.[31,33,37] Indeed, our present data support the importance of arachnoid tearing as an initial factor in the formation of CSDH. A previous study of 256 patients with CSDH to elucidate the pathogenesis of CSDH revealed that the majority of young patients (less than 40 years old) had some promotive factors such as arachnoid cyst, VP shunt, and cerebral atrophy or severe head injuries with loss of consciousness.[45]

CSDH tends to occur in elderly people because brain atrophy causes enlargement of subarachnoid space and stretching of the bridging veins, and these pre-existing conditions facilitate tearing of the arachnoid membrane and leakage of bloody CSF into the subdural space after mild head injury.[16,17] Our present study found that the incidence peaked in the eighth decade and more than 90% of previous head injuries were mild. Acute subdural hematoma is still believed to be a precursor of CSDH,[44] but the present study included only eight cases of acute subdural hematoma (1.6%). The present study showed male dominance (71.8%) and 69.6% of hematomas occurred on the left side including bilateral cases. These are unlikely to be coincidences, as men are more likely to suffer head injury than women. Spontaneous absorption of CSDH can occur.[12,32] We think that right-sided CSDH is sometimes asymptomatic and can be absorbed spontaneously without symptoms.

In the present study, the most common symptom was gait disturbance (63.0%) followed by hemiparesis (58.6%). Headache was also common, especially in patients less than 60 years old (71.3%). Headache is more common in younger patients because they tend to have increased intracranial pressure caused by CSDH.[7] Dementia was recognized in 24.6% of patients and significantly tended to occur in the bilateral cases. Urinary incontinence was also well recognized (17.4%). These facts suggest that CSDH should be differentiated from normal pressure hydrocephalus, multiple cerebral infarction, and senile dementia, because dementia caused by CSDH in elderly patients is treatable.

Functional results have been satisfactory in 72% to 95% of recent cases.[7,21,38] Our patients showed good recovery in 89%. Mortality varies in recent series from 2% to 4.3%,[7,21,22,38] and our result was 1.2%. Age, systemic complications such as cardiovascular disease in elderly patients, coagulopathy, and poor preoperative neurological state are contributory causes of postoperative death.[7,15,21,35] In the present study, 50% of deaths were due to DIC. Statistical analysis also showed that coagulation disturbance was significantly correlated to poor outcome. In contrast, age was not related to poor outcome in the present study. Therefore, even patients aged 80 years or more can expect a good recovery after burr hole craniostomy.[9]

Persistence of an enlarged subdural space, or poor re-expansion of the brain, in patients undergoing evacuation of CSDH creates the potential for reaccumulation of the hematoma.[18,34,38,41] Therefore, re-expansion of the brain after hematoma evacuation is an important factor for good outcome. Fibrous organization of the subdural neomembrane, impairment of cerebral blood flow, and increased brain surface elastance are possible explanations for poor brain re-expansion after surgery.[10,17] Examination of the relationship between brain surface elastance and brain re-expansion after evacuation of CSDH showed that patients with persistent enlarged subdural space had higher elastance and there was a correlation between high elastance and high age.[10] The present study analyzed variables related to brain re-expansion rate after burr hole craniostomy and found that age, pre-existing cerebral infarction, presence of subdural air, and administration of anticoagulant agent were significantly correlated with poor re-expansion. Pre-existing cerebral infarction in patients receiving anticoagulant agents might be a cause of poor brain re-expansion. Brain surface compliance and re-expansion tend to correlate during ischemia.[20] Patients with old cerebral infarction are likely to have high brain surface elastance and poor brain re-expansion after hematoma removal. Therefore, we think that the preoperative brain characteristics such as brain surface elastance and factors obstructing brain expansion such as subdural air are important determinants of brain re-expansion after burr hole craniostomy.

Recurrence of CSDH after first burr hole craniostomy is not rare, and the reported incidence is 7% to 18%.[7,19,21,40] Our series had a recurrence rate of 9.8%. Recurrence and reoperation are not trivial problems because most patients are elderly.

Many factors such as age, sex, anticoagulant therapy, brain re-expansion, pneumocephalus, intracranial hypotension, surgical techniques, and preoperative CT and magnetic resonance imaging findings have been proposed as causative factors for hematoma recurrence.[3,19,38,40] Multilobular hematoma was also reported as a causative factor of recurrent CSDH.[23] We found that the presence of thin subdural hematoma or effusion in the side contralateral to the first operation carried a high risk of recurrence. The present study revealed that poor brain re-expansion was correlated with recurrence. Poor brain re-expansion after hematoma removal is thought to create the potential for reaccumulation of the hematoma because of the absence of a tamponading effect.[34] We also showed the presence of subdural air after surgery prevented good re-expansion of the brain. Therefore, maneuvers to prevent air influx into subdural space during surgery will reduce the recurrence rate of CSDH. In the present study, recurrence of CSDH was found at 1 to 8 weeks (mean 3.5 ± 1.9 weeks) after the first operation. This result suggests that patients with CSDH should be followed up for at least 2 months after surgery to check for recurrence.

The rate of cure of CSDH after burr hole craniostomy is high, but neurological deterioration occasionally complicates the postoperative course of this treatable disease. Acute subdural hematoma, hypertensive intracerebral hemorrhage, tension pneumocephalus, and other cerebrovascular diseases may occur as postoperative complications after burr hole craniostomy.[5,7,21,22,24] The overall rate of postoperative complications has not been clearly reported. In the present study, the overall rate of postoperative complications was 5.4%. Eleven cases of fresh bleeding into CSDH were recently reported after 104 burr hole craniostomies (11%).[7] All 11 patients had received anticoagulant therapy preoperatively. In the present study, the most frequent complication was acute subdural hematoma (13 cases, 2.6%) caused by bleeding from the scalp wound and fresh blood flowed directly into the evacuated subdural space. The importance of careful hemostasis of the scalp wound during operation and correction of eventual coagulation disturbance must be emphasized to prevent this postoperative bleeding. We also began to pack the burr hole with Gelfoam to prevent fresh blood flow into the subdural space. CSDH fluid does not coagulate in the drainage tube because of associated hyperfibrinolysis unless fresh bleeding occurs. Therefore, recognition of coagulated fresh blood in the subdural drainage tube after surgery indicates the presence of acute subdural hematoma. In the present study, tension pneumocephalus was the second most common complication (4 cases, 0.8%). Three of these cases were bilateral CSDHs. We think that incomplete replacement of subdural blood with normal saline was the cause of tension pneumocephalus. Tension pneumocephalus is a fatal complication causing brain herniation. We replace subdural hematoma by normal saline to prevent air accumulation in the evacuated subdural space to avoid this complication. We routinely perform craniography immediately after the operation to check for the presence of intracranial air to prevent this dangerous complication soon after surgery.

Postoperative hypertensive intracerebral hemorrhage may also occur as a postoperative complication after burr hole craniostomy.[5,24] In the present study, one case of hypertensive putaminal hemorrhage occurred after the operation. Transient hyperemia immediately after rapid decompression of CSDH is suspected to be a mechanism of intracerebral hemorrhage.[29] The most common coexisting disease was arterial hypertension (18.6%) in this study. To prevent this fatal complication, careful blood pressure management is needed after burr hole surgery. Recognition and attention to prevent such postoperative complications associated with poor prognosis will further improve the postoperative outcome for patients with CSDH.

Most patients with CSDH (89%) recover after burr hole craniostomy with closed system drainage. However, some patients (15%) suffer recurrence of hematoma or postoperative complications, mostly those aged 70 years or more. The outcome for patients with coagulopathy is poor. Old age, persistence of subdural air, and pre-existing cerebral infarction are causative factors for poor brain re-expansion after surgery. Poor re-expansion of brain is correlated to hematoma recurrence. Therefore, air influx into the subdural space should be prevented during surgery.

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Commentary

The value of this publication lies in the remarkably large series of patients treated at a single institution and in the fact that all pertinent aspects of chronic subdural hematoma treatment have been precisely analyzed. Interestingly, hematoma evacuation was undertaken under general anesthesia, and closed subdural drainage was kept for only 1 or 2 days postoperatively. At our institution, the majority of patients undergo surgery awake with only local anesthesia being applied; also, subdural drainage with closed system is usually continued for 5 to 7 days after the initial operation. While the overall recurrence rate of 9.8% in the present series is quite low, I wonder whether there was no secondary recurrence among the 500 patients, since we have occasionally experienced such cases as well. I wish to congratulate the authors not only for the good treatment results and low complication rate, but also for offering the reader a number of important statistic data related to the treatment of chronic subdural hematoma with burr hole craniostomy and closed drainage system.

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Drs. Mori and Maeda present a large series of chronic subdural hematoma (CSDH) in 500 consecutive cases. Most of patients in this group were elderly. Hematomas were evacuated through two burr holes, the hematoma cavity was irrigated with normal saline and closed system subdural drainage was continued for 1 to 2 days. After one month, 89.4% of patients showed good recovery, 2.2% worsened and 1.2% died. Recurrence of CSDH was recognized after first operation in 9.8% of patients. It is important to know that the factor correlated with the recurrence of CSDH was re-expansion rate of the brain at one week after evacuation of the hematoma. The significant factors influencing the re-expansion rate of brain are thought to be patient older than 70 years, pre-existing cerebral infarction, subdural air accumulation after CSDH evacuation and anticoagulant therapy. This is a well-performed study of CSDH. We are glad to share the authors' experience to improve the outcome of surgical management on CSDH.

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