

Effect of continuous cisternal cerebrospinal fluid drainage for patients with thin subarachnoid hemorrhage

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Abstract: External cerebrospinal fluid (CSF) drainage is an effective method to remove massive subarachnoid hemorrhage (SAH), but carries the risk of meningitis and shunt-dependent hydrocephalus. This study investigated whether postoperative cisternal CSF drainage affects the incidence of cerebral vasospasm and clinical outcome in patients with thin SAH. Seventy-eight patients with thin SAH, 22 men and 56 women aged from 17 to 73 years (mean 51.2 years), underwent surgical repair for ruptured anterior circulation aneurysm. Patients were divided into groups with (38 patients) and without (40 patients) postoperative cisternal CSF drainage, and the incidences of angiographical and symptomatic vasospasm, shunt-dependent hydrocephalus, meningitis, and the clinical outcome were compared. The incidences of angiographical vasospasm (31.6% vs 50.0%), symptomatic vasospasm (7.9% vs 12.5%), shunt-dependent hydrocephalus (5.3% vs 0%), and meningitis (2.6% vs 0%) did not differ between patients with and without cisternal CSF drainage. All patients in both groups resulted in good recovery. Postoperative cisternal CSF drainage does not affect the incidence of cerebral vasospasm or the clinical outcome in patients with thin SAH.

Keywords: subarachnoid hemorrhage; cerebrospinal fluid drainage; cerebral vasospasm; meningitis; hydrocephalus; ruptured intracranial aneurysm

Introduction

Cerebral vasospasm following aneurysmal subarachnoid hemorrhage (SAH) is still one of the major causes of mortality and morbidity. Early surgery for ruptured intracranial aneurysms is recommended to avoid rebleeding and to remove subarachnoid clot, which will reduce the incidence of cerebral vasospasm (Kassell et al 1990). As surgery cannot remove all of the subarachnoid hematoma, external cerebrospinal fluid (CSF) drainage with or without fibrinolytic treatment is a promising method to remove residual SAH (Kasuya et al 1991; Kodama et al 2000). The incidence of cerebral vasospasm after surgery is reported to be lower in patients with thick SAH who underwent external CSF drainage than in patients who did not (Klimo et al 2004). However, external CSF drainage carries the risk of meningitis and neuronal injury (Kodama et al 2000; Klimo et al 2004), and increases the incidence of shunt-dependent hydrocephalus (Kasuya et al 1991; Hirashima et al 2005). Cerebral vasospasm also develops after surgery in 13% to 24% of patients with thin SAH, although the incidence is lower than that in patients with thick SAH (Claassen et al 2001; Smith et al 2005).

The present study investigated whether postoperative cisternal CSF drainage affects the incidence of cerebral vasospasm and clinical outcome in patients with thin SAH.

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Methods and materials

Patient recruitment

This study was based on a series of 567 consecutive patients with aneurysmal SAH diagnosed at our institution between January 1996 and April 2004. The inclusion criteria for this study were: thin SAH without intracerebral hematoma by computed tomography (CT) on admission, defined as the Fisher group 1 and 2 (Fisher et al 1980); ruptured aneurysm in the anterior circulation; surgical obliteration of the ruptured aneurysm within 72 hours after the onset of SAH; and age less than 75 years old. Ethical approval was granted by the institution.

Diagnosis and treatment procedures

The diagnosis of SAH was established based on the findings of CT or xanthochromia of the CSF if the CT was negative. The neurological condition of the patients on admission was evaluated by two certified neurosurgeons (YO, KO) and classified using the Hunt and Hess system (Hunt et al 1968). Ruptured aneurysm was confirmed by digital subtraction angiography and was treated with surgical clipping.

A standard pterional approach using the operating microscope was performed after widely opening the Sylvian fissure. Self retractors were intermittently used on the frontal and temporal lobes. Cerebral veins were carefully dissected and preserved. All operations were performed or supervised by a senior author (AO).

All patients underwent normovolemia and correction of hyponatremia as a prophylactic therapy for cerebral vasospasm. Patients treated between January 1996 and December 1999 underwent placement of cisternal CSF drainage at the prepontine cistern and intrathecal administration of 4 mg of nicardipine hydrochloride every 12 hours between 3 and 14 days after the onset of SAH (Suzuki et al 2001). Patients treated between January 2000 and April 2004 underwent neither cisternal CSF drainage nor intrathecal administration of nicardipine.

Evaluation of outcome

Angiographical vasospasm was evaluated by digital subtraction angiography and/or CT angiography performed 7 days after the onset of SAH (Otagawa et al 2002). Angiographical vasospasm was defined as arterial narrowing of more than 60% reduction in lumen diameter on the second digital subtraction angiography and/or CT angiography (Schneck et al 1964). The symptomatic vasospasm was evaluated by two certified neurosurgeons (YO, KO). The diagnosis of

symptomatic vasospasm was made based on the following clinical criteria at our institute: 1) the symptoms based on deterioration of consciousness and/or focal neurological deficits that emerge between day 4 and day 14 after SAH; 2) a head CT scan excluding other causes of neurological worsening, such as rebleeding or hydrocephalus; 3) no other identifiable cause of neurological worsening, such as electrolyte disturbance, hypoxia, or seizure; and 4) confirmation of vasospasm by DSA and/or CTA.

Postoperative meningitis was defined as one or more of the clinical signs of meningitis and no evidence of infection other than in the central nervous system, such as pneumonia and urinary tract infection. Clinical signs of meningitis include high fever, altered mental status (irritability, confusion, lethargy, coma), severe headache, vomiting, meningeal signs (stiffness of neck, Kernig/Brudzinski sign, or back pain), and rash (Greenberg 1997). If any of these signs were observed, increased cell count in CSF and positive CSF culture confirmed the diagnosis of meningitis.

The clinical outcome at 2 months after SAH was assessed with the Glasgow Outcome Scale (Jennett et al 1975).

Statistical analysis

The incidences of angiographical and symptomatic vasospasm, shunt-dependent hydrocephalus, meningitis, and the clinical outcome were compared. We also performed an individual analysis from patients having a CSF drainage over 10 days. Categorical data were analyzed using the chi-square test and Fisher's exact probability test. Continuous data were analyzed by the Mann-Whitney *U* test. $p < 0.05$ was considered statistically significant.

Results

Seventy-eight patients, 22 men and 56 women aged from 17 to 73 years (mean 51.2 years), satisfied the criteria described above. The number of patients with and without cisternal CSF drainage was 38 and 40, respectively. Ruptured aneurysm was located on the anterior cerebral artery in 30 patients, the internal carotid artery in 31 patients, and the middle cerebral artery in 17 patients. There were no significant differences in patient characteristics (age, sex, Hunt & Hess grade, and location of ruptured cerebral aneurysm) between the groups (Table 1).

Digital subtraction angiography and/or CT angiography 7 days after the onset of SAH identified angiographical vasospasm in 32 patients (41.0%). Symptomatic vasospasm was identified in 8 patients (10.3%). The symptom due to the vasospasm was transient in all patients. There was no

Table 1 Patient characteristics

	Patients with CSF drainage (n = 38)	Patients without CSF drainage (n = 40)	
Age	48.4 ± 14.8	54.0 ± 8.1	ns
Sex (M/F)	13/25	9/31	ns
Hunt & Hess grade			ns
1	5	11	
2	30	25	
3	3	4	
Location of ruptured aneurysm			ns
ACA	12	18	
ICA	18	13	
MCA	8	9	

Abbreviations: ACA, anterior cerebral artery; CSF, cerebrospinal fluid; F, female; ICA internal carotid artery; M, male; MCA, middle cerebral artery; ns, not significant.

statistical difference in the incidence of angiographical and symptomatic vasospasm between the two groups (Table 2). Two patients underwent CSF shunt for hydrocephalus 47 to 58 days after surgical repair of cerebral aneurysms. The incidence of shunt-dependent hydrocephalus did not differ between the two groups (Table 2). Only one patient with postoperative cisternal CSF drainage suffered meningitis, which was cured by antibiotic administration. The incidence of development of meningitis did not differ between the two groups (Table 2). All patients in both groups resulted in good recovery 2 months after the onset of SAH (Table 2).

The duration of external CSF drainage in patients with cisternal CSF drain was 11.7 (5 to 14) days. The number of patients having CSF drainage over 10 days was 28. We performed an individual analysis from patients having an

external CSF drainage over 10 days and the rests, which proved no significant difference in variables between the groups (Table 3).

Discussion

The present study demonstrated that postoperative cisternal CSF drainage did not affect the incidence of cerebral vasospasm or the clinical outcome in patients with thin SAH.

The advantages of external CSF drainage after SAH are continuous removal of spasmogenic substances in the subarachnoid space, restoration of CSF circulation, and control of intracranial pressure (Kawakami et al 1987). Symptomatic vasospasm occurred in 17% and 51% of patients with and without lumbar CSF drainage, respectively, indicating that external clearance of spasmogenic substances reduces vasospasm in patients with thick SAH (Klimo et al 2004). However, the relationship between the incidence of vasospasm and the use of postoperative external CSF drainage in patients with thin SAH has not been reported so far. The present study found no statistical difference in the incidence of vasospasm between patients with thin SAH who were and were not treated with cisternal CSF drainage.

External CSF drainage carries the risk of meningitis (Ogura et al 1998; Kodama et al 2000). The incidence of meningitis was 7% to 15% in a large series of neurosurgical patients with external CSF drainage, which occasionally leads to life-threatening sequelae (Schade et al 2005). In our series, one patient with cisternal CSF drainage suffered meningitis, although no significant difference was observed between patients with and without cisternal CSF drainage. Continuous CSF drainage after SAH can also lead to shunt-dependent hydrocephalus (Kasuya et al 1991). Aggravated subarachnoid fibrinosis and impaired CSF absorption after external CSF drainage are the main causes of the high incidence of hydrocephalus (Yamamoto et al 1983). Furthermore, a surgical tear in the arachnoid membrane and/or decompressive surgery itself could alter CSF hydrodynamics adversely, which may

Table 2 Patients with and without CSF drainage

	Patients with CSF drainage (n = 38)	Patients without CSF drainage (n = 40)	
Angiographical vasospasm	12/38 (31.6%)	20/40 (50.0%)	ns
Symptomatic vasospasm	3/38 (7.9%)	5/40 (12.5%)	ns
Shunt-dependent hydrocephalus	2/38 (5.3%)	0/40 (0%)	ns
Meningitis	1/38 (2.6%)	0/40 (0%)	ns
GR 2 months after the onset	38/38 (100%)	40/40 (100%)	ns

Abbreviations: CSF, cerebrospinal fluid; GR, good recovery in the Glasgow Outcome Scale.

Table 3 Patients with drainage over 10 days

	Drainage over 10 days		
	Yes (n = 28)	No (n = 10)	
AVS (yes/no)	7/21	5/5	ns
SVA (yes/no)	2/26	1/9	ns
H-K	1.96 ± 0.46	1.83 ± 0.41	ns
VPS (yes/no)	2/26	0/10	ns
CSF drained/day	147.1 ± 49.3	177.1 ± 92.6	ns

Abbreviations: AVS, ; CSF, cerebrospinal fluid; H-K, ; SVA, ; VPS, .

lead to internal or external hydrocephalus (Yoshimoto et al 1998; Kan et al 2006). VP shunt operation resolves both types of hydrocephalus after SAH. In our series, 2 patients with cisternal CSF drainage suffered shunt-dependent hydrocephalus, but no significant difference was observed between patients with and without cisternal CSF drainage.

Conclusion

Although our patient cohort was limited and the present study was not a randomized trial, we demonstrated that postoperative cisternal CSF drainage does not affect the incidence of vasospasm or the clinical outcome in patients with thin SAH defined as Fisher group 1 and 2. Therefore, the setting of the external CSF drainage may not be necessary in such patients.

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