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The Feasibility of Early Shunting for Hydrocephalus after Subarachnoid Hemorrhage

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Abstract

The feasibility of early shunting for hydrocephalus after the occurrence of subarachnoid hemorrhage has not yet been explored. We investigated factors associated with the development of hydrocephalus and the risk of shunt obstruction or infection in patients undergoing early shunt surgery. All cases of hydrocephalus after subarachnoid hemorrhage managed at our institution between January 2010 and December 2020 were included. Patients were classified based on the timing of shunt implantation after hemorrhage onset into either the early shunt group (≤28 days) or the late shunt group (>28 days). Of 138 subarachnoid hemorrhage patients managed during the recruitment period, 53 underwent shunt surgery, with 15 in the early shunt group and 38 in the late shunt group. The severity of subarachnoid hemorrhage, presence of Sylvian hematoma, and placement of an external ventricular and/or cisternal drain were significantly associated with the development of hydrocephalus. There was no significant difference between the early and late groups in terms of the rate of shunt obstruction or infection. In the early group, preoperative cerebrospinal fluid cell count was significantly higher in those who developed obstruction than those who did not (307.3 ± 238.2/3 μL vs. 73.8 ± 95.7/3 μL; p = 0.0364). This retrospective study showed no significant difference between early and late shunt implantation in the rate of shunt obstruction and infection. These findings suggests that planning shunt surgery in the early phase after subarachnoid hemorrhage might be feasible, depending on cerebrospinal fluid test results.

Keywords: hydrocephalus, subarachnoid hemorrhage, shunt surgery in early phase, shunt obstruction

Introduction

Reported incidence rates of hydrocephalus after subarachnoid (HAS) hemorrhage vary significantly from 6% to 67%.[1-4] HAS risk factors include inflammation, apoptosis, autophagy, and oxidative stress, but associated underlying mechanisms have not yet been determined.[2] The placement of a ventriculoperitoneal shunt (VPS) or lumboperitoneal shunt (LPS) plays an important role in treatment.[1,2] The rate of shunt obstruction is high when implantation takes place soon after subarachnoid hemorrhage (SAH), because of the presence of blood components in the cerebrospinal fluid (CSF). However, delaying shunt placement is associated with complications related to persistent disturbance of consciousness, which include aspiration pneumonia and prolonged hospitalization.[5,6] Data regarding the feasibility of early shunting for HAS is limited. Analysis and clarification of risk factors for HAS would allow planning for shunt surgery without delay based on imaging and neurological findings. This study aims to investigate factors associated with the development of hydrocephalus and shunt obstruction or infection to determine the feasibility of early shunt surgery and inform the selection of suitable patients.

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Materials and Methods

This study protocol was approved by the ethics committee of Osaka National Hospital and was performed in accordance with committee guidelines (approval number: 21035). All eligible patients were informed about the study through an opt-out notice on the ethics committee's website. All patients treated for SAH in our institution from January 2010 to December 2020 were reviewed. The following data were recorded: age; sex; Fisher grade; Hunt and Kosnik grade; World Federation of Neurological Surgeons (WFNS) grade; presence and location of intracranial hematoma; intraventricular hemorrhage (IVH) score;[7] type and location of the aneurysm; Glasgow coma scale (GCS) score at admission; treatment method (surgical clipping vs. endovascular coiling); placement of external ventricular or lumbar drain for CSF diversion; CSF analysis results; antiplatelet medication use; HAS development; placement of VPS or LPS; type of shunt bulb; timing of shunt placement; symptoms of suspected HAS; decompressive craniectomy; cranioplasty; vasospasm; infection at the time of diagnosis; the number of days to hydrocephalus; development of shunt obstruction; the presence of shunt infection; length of hospital stay; and modified Rankin scale (mRS) score at discharge. All patients diagnosed with HAS were included in the analysis. HAS was initially diagnosed by the presence of one or more symptoms including intolerable headache, nausea/vomiting, a decline in GCS, disorientation, and inability to walk, and was confirmed by the identification of ventricular enlargement on imaging. In the absence of ventricular enlargement, a CSF tap test was performed. After the test, improvement of symptoms allowed diagnosis of HAS. CSF testing was performed approximately once per week during CSF drainage and immediately before shunt procedures to exclude meningitis. The CSF cell count included the total number of leukocytes, erythrocytes, and dysplastic cells measured in the CSF. Patients with a diagnosis of HAS underwent either VPS or LPS implantation, with the decision made at the discretion of the treating neurosurgeon due to the lack of departmental guidelines. Shunt procedures were performed only after confirming the absence of any infection. In VPS procedures, Certas Plus (Codman, Raynham, MA, USA), Hakim (Codman, Raynham), or proGAV2.0 (Braun, Bethlehem, PA, USA) shunts were used; in LPS procedures, Certas Plus, Strata (Medtronic, Dublin, Ireland), Hakim, or proGAV2.0 shunts were used. In VPS procedures, a ventricular catheter was placed via an anterior horn puncture of the lateral ventricle. In LPS procedures, a lumbar catheter was placed via a lumbar interspinous puncture between L2 and L5. The initial pressure setting was based on the spinal fluid pressure observed during CSF drainage or preoperative lumbar puncture.

Statistical analysis

Statistical analyses were performed using JMP 16 software (SAS Institute Inc., Cary, NC, USA). Patients who underwent shunt surgery were classified based on the timing of implantation after SAH onset into an early shunt group (ESG; ≤ 28 days) and a late shunt group (LSG; > 28 days). Shunted patients were then categorized according to the development of shunt obstruction and the presence of shunt infection. Continuous variables are expressed as means with standard deviation and were compared using the 2-tailed Student's *t*-test or one-way analysis of variance with post-hoc Tukey-Kramer analysis. Categorical variables are expressed as numbers with percentages and were compared using the chi-squared or Fisher's exact test. Receiver operating characteristic curve analysis was performed to determine the optimal CSF cell count cut-off value for shunt obstruction. Values of $p < 0.05$ were considered significant.

Results

During the study period, 138 SAH patients were treated, including 84 (60.9%) women. The mean age was 62.4 years (range 26-93). WFNS classification was grade I in 48 patients, grade II in 22, grade III in 8, grade IV in 23, and grade V in 37. The number of patients classified as Fisher grade I, II, III, and IV was 4, 11, 121, and 30, respectively. The cause of SAH was saccular aneurysm in 133 patients, dissecting aneurysm in 4, and unknown in one (Fig. 1).

Risk factors for developing HAS

Shunts were implanted in 53 patients. WFNS grade ($p < 0.0001$), Hunt and Kosnik grade ($p = 0.001$), presence of Sylvian hematoma ($p = 0.0016$), and insertion of a ventricular or cisternal drain ($p = 0.0004$) were each significantly associated with the development of HAS. Sex, age, Fisher grade, presence of intraventricular hematoma, aneurysm type, aneurysm treatment method, presence of a lumbar drain, and the use of antiplatelet medication were not associated with HAS (Supplementary Table 1).

Risk factors for shunt obstruction

Shunt obstruction occurred in 6 patients (11.3%); all were women. Female sex was significantly associated with shunt obstruction ($p = 0.028$). The mean age of patients who developed shunt obstruction was significantly higher than that of those who did not (77.3 ± 9.9 years vs. 62.8 ± 12.1 years; $p = 0.010$). The mean preoperative CSF cell count was significantly higher in the shunt obstruction group (209.4 ± 215.3/3 μL vs. 67.1 ± 112.4/3 μL; $p = 0.011$). Aneurysm treatment method, WFNS grade, Hunt and Kosnik grade, aneurysm location, presence of intraventricular hematoma, IVH score, type and presence of CSF drainage, type of shunt procedure, type of shunt bulb, duration from SAH onset to shunting, CSF protein concentration, and use

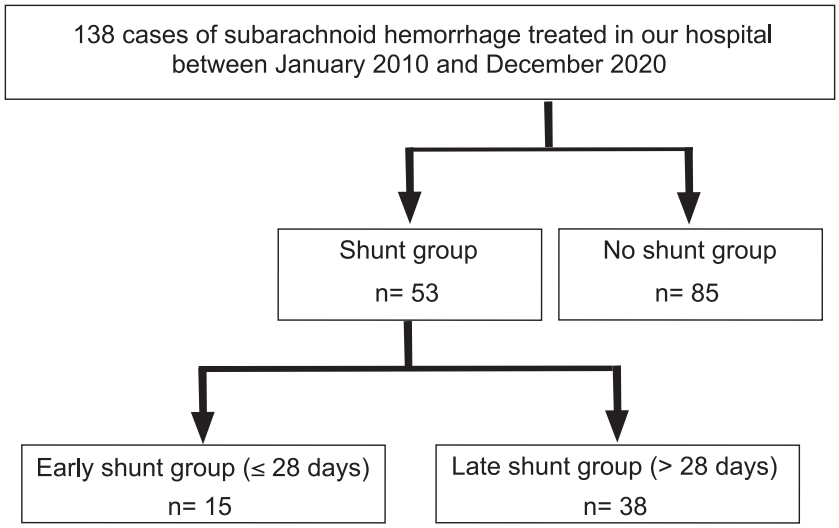

Fig. 1 Study flowchart.

of antiplatelet medication were not significantly associated with shunt obstruction (Supplementary Table 2). In the LPS group, mean age was significantly higher in patients who developed shunt obstruction than those who did not (78.0 ± 6.9 years vs. 64.1 ± 13.0 years; p = 0.049; Supplementary Table 3). In the VPS group, no variables were significantly associated with shunt obstruction (Supplementary Table 4).

Risk factors for shunt infection

Shunt infection occurred in 3 patients (5.7%). Absence of intraventricular hematoma was significantly associated with shunt infection (p = 0.036). Sex, age, aneurysm treatment method, WFNS grade, Hunt and Kosnik grade, aneurysm location, IVH score, type and presence of CSF drainage, type of shunt procedure, type of shunt bulb, duration from SAH onset to shunting, CSF cell count, CSF protein concentration, and use of antiplatelet medication were not significantly associated with shunt infection (Supplementary Table 5).

Comparison of ESG and LSG

Shunting was performed early (within 28 days of SAH onset) in 15 patients and late (after 28 days) in 38. Shunt timing was significantly associated with the number of days until hydrocephalus diagnosis (p < 0.001), but not with infection at the time of diagnosis (Table 1). Three shunt obstructions occurred in each group. Two shunt infections occurred in the ESG and one in the LSG. There was no significant difference in the rate of shunt obstruction or infection between the 2 groups. The mean length of hospital stay was significantly shorter in the ESG (65.9 ± 23.5 days vs. 118.2 ± 128.1 days; p = 0.0084). However, multiple regression analysis showed that shunt timing (p = 0.145), vasospasm (p = 0.616), and decompressive craniectomy (p = 0.979) were not independently associated with the length of hospital stay (Table 2). The proportion of patients with an mRS score of 0-2 at discharge was not significantly different, with 33.3% in ESG and 15.8% in LSG (p = 0.156; Table 1).

Risk factors for shunt obstruction in the ESG and LSG

In the ESG, preoperative CSF cell count was significantly higher in those who developed obstruction than those who did not (307.3 ± 238.2/3 μL vs. 73.8 ± 95.7/3 μL; p = 0.0364). The mean duration of CSF drainage placement was slightly shorter in the shunt obstruction group (13.0 ± 2.0 days vs. 17.0 ± 3.5 days), but the difference was not significant. Sex, age, aneurysm treatment method, WFNS grade, Hunt and Kosnik grade, aneurysm location, presence of intraventricular hematoma, IVH score, type and presence of CSF drainage, type of shunt procedure, duration from SAH onset to shunting, mean preoperative CSF protein concentration, and use of antiplatelet medication were not significantly associated with shunt obstruction (Table 3). In the LSG, those who developed obstruction were significantly older than those who did not (79.3 ± 10.0 years vs. 63.4 ± 11.6 years; p = 0.035) (Table 4).

Discussion

This study investigated risk factors associated with the development of HAS and shunt obstruction and infection, providing indications for identifying suitable cases for early shunt surgery. SAH activates the fibrinolytic, kinin, and complement systems in the subarachnoid space, which causes inflammation within the space and in the arachnoid granulations. These mechanisms, along with mechanical obstruction of CSF pathways caused by hematoma, result in HAS.[8] Oreskovic and Klarica[9] hypothesized that CSF production and absorption occur in brain capillaries. CSF moves from the cerebral arteries to the plasma

Table 1 Comparison between early and late shunt procedure groups

		ESG (n=15)	LSG (n=38)	P value
Symptoms of suspected HAS	Headache	1 (6.7)	1 (6.7)	0.201
	Nausea/Vomiting	1 (6.7)	2 (13.3)	
	Decline in GCS	12 (80.0)	30 (78.9)	
	Disorientation	1 (6.7)	2 (13.3)	
	Disability to walk	0 (0.0)	8 (21.0)	
	Improvement after a CSF tap test	0 (0.0)	2 (13.3)	
Decompressive craniectomy: n (%)		2 (13.3)	13 (34.2)	0.290
Vasospasm: n (%)		4 (26.7)	10 (26.3)	0.979
Infection at the time of diagnosis: n (%)		3 (20.0)	8 (21.1)	0.932
Number of days to hydrocephalus (days)	Mean ± SD	17.6±4.1	68.8±163.1	<0.001
Number of days from hydrocephalus diagnosis to shunt (days)	Mean ± SD	5.26±1.9	10.3±8.6	0.111
Cell count (/3μL)	Mean ± SD	120.5±157.0	718.3±3135.0	0.390
Type of shunt: n (%)	LP	10 (66.7)	29 (76.3)	0.47
	VP	5 (33.3)	9 (23.7)	
Infection: n (%)		2 (13.3)	1 (2.6)	0.154
Obstruction: n (%)		3 (20.0)	3 (7.9)	0.21
Hospitalization (days)	Mean ± SD	65.9±23.5	118.2±128.1	0.0084
mRS at discharge: n (%)	0 - 2	5 (33.3)	6 (15.8)	0.156

ESG, early shunt group; LSG, late shunt group; HAS, hydrocephalus after subarachnoid hemorrhage; GCS, Glasgow Coma Scale; CSF, cerebrospinal fluid; LP, lumbaroperitoneal shunt; VP, ventriculoperitoneal shunt; mRS, modified Rankin Scale

Table 2 Related factors for length of hospitalization

		Hospitalization (Mean±SD, days)	Univariate	Multivariate
Decompressive craniectomy	(+)	108.9±37.2	P=0.011	P=0.979
	(-)	100.4±125.3		
Vasospasm	(+)	89.6±40.1	P=0.896	P=0.616
	(-)	107.1±126.2		
Shunt timing	ESG	65.9±23.5	P=0.0084	P=0.145
	LSG	116.9±126.7		

ESG, early shunt group; LSG, late shunt group

and brain parenchyma via the perivascular space around capillaries and cerebral veins.[10] After SAH, hematoma occludes the perivascular space, obstructing CSF flow.[3,10] In our study, SAH severity was significantly associated with HAS risk. This suggests that a greater degree of hemorrhage within the subarachnoid space results in greater disruption of CSF flow around the brain capillaries, leading to hydrocephalus.

Vassilouthis et al.[11] reported that cerebral infarction caused by vasospasm occurs in 45% of patients with HAS. Cerebral vessel wall damage caused by infarction impairs the movement of CSF in the brain from the perivascular to vascular space and impairs CSF absorption, both of which contribute to hydrocephalus.[12] The presence of a Sylvian hematoma is a risk factor for vasospasm after SAH.[13] Consistent with this, Sylvian hematoma was significantly associated with the development of HAS in our cohort. These findings suggest interrelations between subarachnoid hematoma, vasospasm, and HAS.

CSF production increases in the acute phase of SAH. As

Table 3 Risk factor of shunt obstruction in ESG

		Obstruction (n=3)	No obstruction (n=12)	P value
Man: n (%)		0 (0.0)	3 (25.0)	0.332
Age	Mean ± SD	75±11.5	61.3±14.0	0.129
Operation: n (%)	Clip	1 (33.3)	4 (33.3)	0.922
	Coil	2 (66.7)	7 (58.3)	
	FD	0 (0.0)	0 (0.0)	
Hunt & Kosnik grade: n (%)	I	1 (33.3)	2 (16.7)	0.796
	II	0 (0.0)	2 (16.7)	
	III	0 (0.0)	2 (16.7)	
	IV	1 (33.3)	2 (16.7)	
	V	1 (33.3)	4 (33.3)	
WFNS grade: n (%)	I	1 (33.3)	2 (16.7)	0.691
	II	0 (0.0)	3 (25.0)	
	III	0 (0.0)	0 (0.0)	
	IV	0 (0.0)	1 (8.3)	
	V	2 (66.7)	6 (50.0)	
Fisher group: n (%)	I	0 (0.0)	0 (0.0)	0.495
	II	1 (33.3)	1 (8.3)	
	III	1 (33.3)	7 (58.3)	
	IV	1 (33.3)	4 (33.3)	
Location of aneurysm: n (%)	ACA	0 (0.0)	1 (8.3)	0.259
	AcomA	1 (33.3)	4 (33.3)	
	BA-SCA	0 (0.0)	0 (0.0)	
	BA tip	0 (0.0)	0 (0.0)	
	ICA	0 (0.0)	0 (0.0)	
	ICA-PcomA	0 (0.0)	4 (33.3)	
	MCA	2 (66.7)	1 (8.3)	
	PCA	0 (0.0)	0 (0.0)	
	PICA	0 (0.0)	0 (0.0)	
	VA	0 (0.0)	1 (8.3)	
	unknown	0 (0.0)	0 (0.0)	
Posterior circulation: n (%)		0 (0.0)	1 (8.3)	0.268
Intraventricular hematoma: n (%)		1 (33.3)	10 (83.3)	0.417
IVH score	Mean±SD	10±7	10±7.3	1
Type of drainage: n (%)	No	0 (0.0)	0 (0.0)	0.604
	Spinal	2 (66.7)	6 (50.0)	
	Ventricular	0 (0.0)	0 (0.0)	
	Ventricular and spinal	1 (33.3)	6 (50.0)	
	Cisternal and spinal	0 (0.0)	0 (0.0)	
	Cisternal, ventricular and spinal	0 (0.0)	0 (0.0)	
Duration of drainage	Mean±SD	13±2	17±3.5	0.0659
Type of shunt: n (%)	LP	2 (66.7)	8 (66.7)	1
	VP	1 (33.3)	4 (33.3)	

Table 3 Risk factor of shunt obstruction in ESG (continued)

		Obstruction (n=3)	No obstruction (n=12)	P value
Duration from onset to shunt	Mean±SD	24±6.9	22.3±4.3	0.276
Cell count (/3μL)	Mean±SD	307.3±238.2	73.8±95.7	0.0364
Total Protein (mg/dL)	Mean±SD	111.3±104.9	44.8±26.7	0.219
Antiplatelet agents: n (%)		3 (100.0)	3 (25.0)	0.086

WFNS, World Federation of Neurological Surgeons; IVH, intraventricular hemorrhage; ACA, anterior cerebral artery; AcomA, anterior communicating artery; BA, basilar artery; SCA, superior cerebellar artery; ICA, internal carotid artery; PcomA, posterior communicating artery; MCA, middle cerebral artery; PCA, posterior cerebral artery; PICA, posterior inferior cerebellar artery; VA, vertebral artery; Total Protein, total protein; LP, lumbaroperitoneal shunt; VP, ventriculoperitoneal shunt

a result, intracranial pressure is elevated, and hematoma and inflammatory products are cleared from the subarachnoid space. Drainage of large amounts of CSF via an external ventricular or lumbar drain presumably prevents CSF inflow to the subarachnoid space, which causes subsequent collapse of the space and adhesions that further obstruct CSF pathways.[8] In our study, placement of an external ventricular or lumbar drain was significantly associated with HAS. Excessive CSF drainage or meningitis caused by external drainage may cause HAS. In contrast, ambulation may decrease the incidence of HAS through related changes in subarachnoid CSF flow and the shifting of intracranial subarachnoid hematoma to the spinal subarachnoid space.[14] Therefore, patients with high-grade SAH, who typically cannot ambulate in the early stages and are more likely to have an external ventricular or lumbar drain applied, are at greater risk for HAS.

Several risk factors for shunt obstruction have been reported in previous studies. Khan et al.[15] reported that older patients were more likely to experience shunt obstruction. They suggest that brain tissue is more fragile in the older people, and is therefore more likely to enter and occlude the shunt tubing during the procedure. Constipation has also been reported as a risk factor.[16] Constipation is more prevalent in women and the older people[17] and might explain our finding that shunt obstruction was more common in these patient populations. Shunt obstruction in the LSG was also significantly associated with age in our study. Elevated numbers of erythrocytes and leukocytes in the CSF are other risk factors for shunt obstruction. Sun et al.[6] reported in their study of 76 patients with HAS that the CSF erythrocyte count was significantly higher in patients who developed shunt obstruction than in those who did not (258/μL vs. 6/μL). Brydon et al.[5] suggest that red blood cells affect shunt function and that patients with bloody CSF should not undergo shunting; they reported that a large number of erythrocytes in the CSF can obstruct the valve. In a study of 274 patients with HAS, Kaestner et al.[18] reported that the leukocyte count was significantly higher in patients who developed shunt obstruction than in those who did not (51/μL vs. 13/μL). They suggested that inflammation might be associated with shunt obstruction.[18] CSF cell count was significantly associated with shunt obstruction in our ESG but not LSG patients. CSF cell counts should therefore be taken into consideration when performing early shunting. Red blood cell counts are elevated immediately after SAH and gradually decrease.[19] CSF findings after onset are reported to be xanthochromia for about 2 weeks.[20] As red blood cell counts have been reported to be related to obstruction,[5] a lack of association with CSF cell count in patients who undergo later shunting may be due to low red blood cell counts at that stage. However, a limitation of this study is that the proportions of cells and erythrocyte count in the CSF were not known.

In this study, the absence of intraventricular hematoma was significantly associated with shunt infection; however, the possible mechanism underlying this association is not clear.

Data regarding the feasibility of early shunting for HAS is limited. Jost et al.[21] divided patients of HAS into 2 groups based on the shunt timing cut-off of 21 days after SAH and compared the 2 groups. They reported no adverse effects resulting from early shunt implantation.[21] In our cohort, patients were classified based on a shunt timing of 28 days, as the first quartile for our surgery timing was 27.5 days. Consistent with the previous report, we observed no significant difference between ESG and LSG in the rates of shunt obstruction or infection. Jost et al.[21] also reported no significant difference between their early and LSGs in terms of infection and necessary revision. In patients with SAH, decompression and vasospasm have previously been reported to be related to the length of hospital stay.[22,23] Therefore, we performed a multivariate analysis for length of hospital stay according to decompression, vasospasm, and shunt timing. Shunt timing was significantly associated with length of hospital stay in univariate, but not in multivariate analysis. Shunt timing could therefore reduce the length of hospital stay, but other factors also contributed. In this study, shunt timing was significantly

Table 4 Risk factor of shunt obstruction in LSG

		Obstruction (n=3)	No obstruction (n=35)	P value
Man: n (%)		0 (0.0)	3 (7.89)	0.071
Age	Mean ± SD	79.3±10.0	63.4±11.6	0.035
Operation: n (%)	Clip	0 (0.0)	17 (48.6)	0.367
	Coil	2 (100.0)	17 (48.6)	
	FD	0 (0.0)	1 (2.86)	
Hunt & Kosnik grade: n (%)	I	1 (33.3)	2 (5.71)	0.369
	II	0 (0.0)	5 (14.2)	
	III	1 (33.3)	5 (14.2)	
	IV	0 (0.0)	8 (22.9)	
	V	1 (33.3)	15 (42.9)	
WFNS grade: n (%)	I	1 (33.3)	5 (14.3)	0.206
	II	0 (0.0)	3 (8.57)	
	III	0 (0.0)	2 (5.71)	
	IV	2 (66.7)	6 (17.1)	
	V	0 (0.0)	19 (54.3)	
Fisher group: n (%)	I	0 (0.0)	1 (2.86)	0.817
	II	0 (0.0)	2 (5.71)	
	III	2 (66.7)	27 (77.1)	
	IV	1 (33.3)	5 (14.3)	
Location of aneurysm: n (%)	ACA	0 (0.0)	3 (8.3)	0.133
	AcomA	0 (0.0)	5 (33.3)	
	BA-SCA	0 (0.0)	1 (0.0)	
	BA tip	0 (0.0)	1 (0.0)	
	ICA	0 (0.0)	2 (0.0)	
	ICA-PcomA	1 (33.3)	7 (33.3)	
	MCA	0 (0.0)	12 (0.0)	
	PCA	1 (33.3)	0 (0.0)	
	PICA	0 (0.0)	1 (0.0)	
	VA	0 (0.0)	3 (0.0)	
	unknown	1 (33.3)	0 (0.0)	
Posterior circulation: n (%)		1 (33.3)	6 (17.1)	0.468
Intraventricular hematoma: n (%)		2 (66.7)	31 (88.6)	0.281
IVH score	Mean±SD	9±3	11.2±1.6	0.764
Type of drainage: n (%)	No	1 (33.3)	0 (0.0)	0.101
	Spinal	0 (0.0)	11 (31.4)	
	Ventricular	0 (0.0)	2 (5.71)	
	Ventricular and spinal	2 (66.7)	13 (37.1)	
	Cisternal and spinal	0 (0.0)	3 (8.57)	
	Cisternal, ventricular and spinal	0 (0.0)	6 (17.1)	
Duration of drainage	Mean±SD	12.3±6.1	20.6±1.7	0.464
Type of shunt: n (%)	LP	1 (33.3)	28 (80.0)	0.068
	VP	2 (66.7)	7 (20.0)	

Table 4 Risk factor of shunt obstruction in LSG (continued)

		Obstruction (n=3)	No obstruction (n=35)	P value
Duration from onset to shunt (days)	Mean±SD	45±10.5	82.0±169.8	0.684
Cell count (/3μL)	Mean±SD	62.5±7.78	758.0±3227.0	0.499
Total Protein (mg/dL)	Mean±SD	120±86.7	78.3±44.0	0.374
Antiplatelet agents: n (%)		1 (33.3)	15 (42.9)	0.086

WFNS, World Federation of Neurological Surgeons; IVH, intraventricular hemorrhage; ACA, anterior cerebral artery; AcomA, anterior communicating artery; BA, basilar artery; SCA, superior cerebellar artery; ICA, internal carotid artery; PcomA, posterior communicating artery; MCA, middle cerebral artery; PCA, posterior cerebral artery; PICA, posterior inferior cerebellar artery; VA, vertebral artery; Total Protein, total protein; LP, lumbaroperitoneal shunt; VP, ventriculoperitoneal shunt

associated with the number of days to hydrocephalus diagnosis; the number of days from hydrocephalus diagnosis to shunt was not significantly different between ESG and LSG. Therefore, we believe the rate of hydrocephalus progression to be a major factor in shunt timing. These results suggest that shunt surgery within 28 days of SAH onset is feasible, under the condition of a CSF test to confirm that cell counts have decreased.

This retrospective study showed that there was no significant difference in the rates of shunt obstruction and infection between early and late implantation. Planning shunt surgery in the early phase after SAH may be a feasible strategy when implemented with periodic imaging and CSF tests.

Limitations

This study has several limitations. Selection bias may be present due to its retrospective design; our findings therefore may not be generalizable. The timing of shunt surgery was planned based on the appearance of hydrocephalus symptoms, such as decreased level of consciousness, ventricular enlargement on imaging, and the tap test; however, there may have been variation in these decision-making between surgeons. Shunt timing was significantly associated with length of hospital stay in univariate analysis, but not in multivariate analysis. A randomized control trial or study with propensity matching and a greater number of patients is necessary to confirm our findings.

Supplementary Material

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Conflicts of Interest Disclosure

All authors have no conflict of interest.

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