Preventive Suboccipital Decompressive Craniectomy for Cerebellar Infarction

A Retrospective-Matched Case–Control Study

Myeong Jin Kim, MD; Sang Kyu Park, MD; Jihye Song, MD; Se-yang Oh, MD; Yong Cheol Lim, MD; Sook Yong Sim, MD, PhD; Yong Sam Shin, MD, PhD; Joonho Chung, MD, PhD

Background and Purpose—No evidence is available on the benefits of preventive suboccipital decompressive craniectomy (SDC) for patients with cerebellar infarction. The purpose of this matched case–control study was to investigate whether preventive SDC was associated with good clinical outcomes in patients with cerebellar infarction and to evaluate its predisposing factors.

Methods—Between March 2007 and September 2015, 28 patients underwent preventive SDC. We performed propensity score matching to establish a proper control group among 721 patients with cerebellar infarction during the same period. Group A (n=28) consists of those who underwent preventive SDC, and group B (n=56) consists of those who did not undergo preventive SDC. We analyzed and compared clinical outcomes between groups.

Results—Clinical outcomes were better in group A than in group B at discharge (P=0.048) and 12-month follow-up (P=0.030). Group B had more deaths within 12 months than group A (log-rank, P<0.05). Logistic regression analysis showed that preventive SDC (odds ratio, 4.815; P=0.009) and the absence of brain stem infarction (odds ratio, 2.862; P=0.033) were independently associated with favorable outcomes (modified Rankin Scale score of 0–2) at 12-month follow-up.

Conclusions—Favorable clinical outcomes including overall survival can be expected after preventive SDC in patients with a volume ratio between 0.25 and 0.33 and the absence of brain stem infarction. Among these patients, preventive SDC might be better than the best medical treatment alone. **(*Stroke*. 2016;47:2565-2573. DOI: 10.1161/STROKEAHA.116.014078.)**

Key Words: brain infarction ■ cerebellar diseases ■ decompressive craniectomy ■ infarction ■ propensity score

Patients with cerebellar infarction should not be neglected because they can experience sudden clinical deterioration from cerebellar swelling. Compared with malignant middle cerebral artery infarctions, malignant cerebellar infarctions can be more urgent because of the smaller space of the posterior fossa, consecutive brain stem compression, and obstructive hydrocephalus. In these situations, suboccipital decompressive craniectomy (SDC) is recommended as a life-saving therapy although no randomized controlled trials on this therapy have been conducted.[1,2] Furthermore, no evidence is available on the benefits of preventive SDC for patients with cerebellar infarction, whereas the benefits of early preventive decompressive hemicraniectomy for malignant middle cerebral artery infarction are supported by randomized controlled trials.[3,4] However, a subset of patients might be expected to benefit in terms of improved survival and functional outcomes from preventive SDC. Determining how to select patients who are good candidates for preventive SDC would be useful. Thus, the purpose of this matched case–control study was to investigate whether preventive SDC was associated with good clinical outcome in patients with cerebellar infarction and to evaluate its predisposing factors.

Materials and Methods

Patient Selection

All research protocols were approved by the institutional review board of our institute, and the need for informed consent was waived. Between March 2007 and September 2015, medical records and radiographic data of 60 patients treated with SDC were reviewed retrospectively in a prospectively collected database from 5 hospitals where 5 neurosurgeons, all alumni of a single institution, treated patients with cerebrovascular diseases using similar medical and surgical methods. Among them, 28 patients underwent preventive SDC with the following criteria: (1) the presence of cerebellar infarction; (2) initial Glasgow Coma Scale (GCS) score ≥9; (3) without clinical

Received May 12, 2016; final revision received July 30, 2016; accepted August 4, 2016.

From the Department of Neurosurgery, Gachon University Gil Medical Center, Incheon, Korea (M.J.K.); Department of Neurosurgery, Incheon St. Mary's Hospital, The Catholic University of Korea (S.K.P.); Department of Neurosurgery, Konyang College of Medicine, Konyang University Hospital, Daejeon, Korea (J.S.); Department of Neurosurgery, Inha University College of Medicine, Incheon, Korea (S.-y.O.); Department of Neurosurgery, Ajou University College of Medicine, Suwon, Korea (Y.C.L.); Department of Neurosurgery, Inje University Seoul Paik Hospital, Korea (S.Y.S.); Department of Neurosurgery, Seoul St. Mary's Hospital, The Catholic University of Korea (Y.S.S.); Department of Neurosurgery, Gangnam Severance Hospital, Seoul, Korea (J.C.); and Severance Institute for Vascular and Metabolic Research, Yonsei University College of Medicine, Seoul, Korea (J.C.).

Correspondence to Joonho Chung, MD, PhD, Department of Neurosurgery, Gangnam Severance Hospital, Severance Institute for Vascular and Metabolic Research, Yonsei University College of Medicine, 211, Eonjuro, Gangnam-gu, Seoul, 135–720, Republic of Korea. E-mail ns.joonho.chung@gmail.com

Stroke **is available at http://stroke.ahajournals.org** **DOI: 10.1161/STROKEAHA.116.014078**

deterioration (no GCS score changes) within 72 hours from onset; and (4) a cerebellar infarction volume ratio between 0.25 and 0.33 on initial or routine follow-up radiographic findings (within 72 hours of onset) generated by computed tomography (CT) or diffusion-weighted imaging. Cerebellar infarction volume ratios were calculated using the equation: volume ratio=cerebellar infarction volume (mm^3)/total cerebellar volume (mm^3). Volumes were estimated using the formula: volume (mm^3)=$(A_1+A_2+A_3+\cdots+A_N)\times H$, where N indicated the number of image sections that showed the cerebellum, A (area, mm^2) was measured manually for each image section, and H (height, mm) was 4 or 5, depending on the thickness of the image section from the CT or diffusion-weighted image. A picture archiving and communication system viewer (INFINITT PiViewSTAR, INFINITT Healthcare Co., Ltd, Seoul, Korea) was used (Figure 1). Total cerebellar volume included brain stem volume. The other 32 patients underwent SDC (not preventive SDC) because of clinical deterioration, GCS score <9, or severe mass effect on radiographic images.

During the same period, our database contained 1102 patients with cerebellar infarction who did not undergo preventive SDC. We excluded patients who lost follow-up (n=312, including 96 because of death) and patients with modified Rankin Scale (mRS) score ≥1 before new clinical signs because of cerebellar infarction (n=69). Among the remaining 721 patients, we performed propensity score matching to establish a proper control group.

Three independent investigators blinded to the data of the present study retrospectively reviewed the clinical and radiographic data of the included patients using their medical records. Variables evaluated and compared between groups were as follows: age, sex, initial National Institutes Health Stroke Scale (NIHSS) score, initial GCS score, cause of cerebellar infarction, involved hemisphere (unilateral or bilateral), combined brain stem infarction, external ventricular drainage (EVD) for hydrocephalus, atrial fibrillation, hypertension, diabetes mellitus, dyslipidemia, previous stroke, smoking, body mass index, intravenous tissue-type plasminogen activator (tPA), intra-arterial endovascular treatment (EVT), previous myocardial infarction, coronary artery occlusive disease, peripheral artery occlusive disease, and hemorrhagic transformation. Clinical outcomes were assessed with mRS. An mRS of 0 to 2 was defined as favorable; an mRS of 3 to 6 was defined as unfavorable. We compared the overall survival and follow-up functional outcomes between the groups to evaluate effectiveness and safety of preventive SDC. We also evaluated predisposing factors that were associated with good clinical outcomes.

Interventional Managements in Patients With Cerebellar Infarction

All patients were evaluated by a stroke physician (neurologist or neurosurgeon) and assessed by NIHSS score on their admission. All patients underwent an initial imaging study that included a nonenhanced CT scan with or without CT angiography and multimodal magnetic resonance imaging (MRI) with or without magnetic resonance angiography. We performed intra-arterial EVT based on the following criteria: (1) baseline NIHSS score ≥4; (2) no intracerebral hemorrhage detected on CT or MRI; (3) vertebral or basilar artery occlusion detected on CT angiography, magnetic resonance angiography, or conventional cerebral angiography; (4) no bilateral diffuse pontine infarction on diffusion-weighted imaging; and (5) procedure commencement within 12 hours after symptom onset. Intravenous tPA (0.9 mg/kg) was administered to patients within 4.5 hours of symptom onset. Subsequent EVT as a rescue therapy was considered within 1 hour of intravenous tPA in patients with no neurological improvement, defined as an unchanged or worsened NIHSS score relative to baseline.

EVT was performed by means of a femoral approach without general anesthesia. In cases of agitation, an intravenous bolus of midazolam was administered and repeated if necessary. A 5F- or 6F-guiding catheter was placed in the vertebral artery. From March 2010, mechanical thrombectomy with a Solitaire stent was used as a first-line treatment option.[5,6] In the present study, 10 patients (2 in group A and 8 in group B) received mechanical thrombectomy among 14 patients who underwent intra-arterial EVT. The other 4 patients received mechanical thrombolysis without intra-arterial chemical thrombolysis.

In the preset study, all patients underwent MRI on admission, after recanalization procedures (intravenous tPA or intra-arterial EVT), after clinical symptoms became aggravated or when clinical symptoms were not correlated with CT findings. Brain stem infarction was determined via MRI.

Propensity Score Matching

Propensity score matching was performed using multiple logistic regression with preventive SDC versus nonpreventive SDC with respect to age, sex, initial NIHSS score, and initial GCS score. After patient matching was performed by estimated propensity scores via a conditional logistic regression method, multiple logistic regression analysis was performed. Using the logit estimated from the log odds of the propensity score of each patient, we matched a selected case with controls who had the nearest estimated logit value by 1:2 matching. Thus, group A (n=28) consisted of those who underwent preventive SDC and group B (n=56) consisted of those who did not undergo preventive SDC. Balance between the 2 groups for each variable was evaluated by propensity score distributions, and absolute standardized differences before and after matching were calculated (Figure 2). Absolute standardized differences <0.10 implied good balance between the 2 groups. A complete set of baseline data was essential for the development of the propensity model. Thus, we replaced missing data values with the mean for that variable instead of excluding patients with any missing data.

Best Medical Management

The best medical treatment method was used for both groups. All patients were admitted to the stroke unit or neurosurgical intensive care unit during the acute stage. Blood pressure (BP) was monitored routinely every 15 minutes. Continuous arterial line monitoring was performed for patients who underwent surgery or with systolic BP ≥180 mm Hg or diastolic BP ≥100 mm Hg. BP was controlled with continuous intravenous infusion of an antihypertensive agent for BP >180/100 mm Hg at the acute stage, which was later changed to oral medication. Patients with other medical histories such as hypertension or diabetes mellitus, had BP lowered according to recommendations.[1,2,7,8] Arterial oxygen concentration was maintained within normal limits. Oxygen was administered in cases of hypoxemia. In cases of reversible respiratory insufficiency, patients were intubated and assisted with an artificial ventilator. Euvolemia was maintained with normal saline. Cardiac arrhythmias that might reduce cardiac output were corrected in consultation with cardiologists. Glucose level and body temperature were maintained within normal ranges as much as possible. Mobilization with rehabilitation therapy was started as soon as the patients were medically stable. Antiplatelet agents and anticoagulants were used according to the guidelines.[1,2] When follow-up radiological findings indicated a mass effect and swelling or when clinical symptoms revealed increased intracranial pressure, hypertonic agent (mannitol) was administered. Patients did not receive steroids.

Preventive SDC

SDC was performed with the patient in the park-bench (three quarter) or prone position. The head was fixed with a three-pin head fixator. A unilateral EVD on the parieto-occipital point was usually set up before SDC for hydrocephalus and was performed in 14 (50.0%) patients in this study. Bilateral SDC with the opening of the foramen magnum was performed for all patients, even those with unilateral infarction. The upper and lateral margin of SDC extended as close as possible to the transverse and lateral sinuses. The dura was opened in a large Y incision. As the dura was opened and the infarcted brain began herniating outward, decompressive resection of the infarcted cerebellum was performed in 16 of 28 patients (57.1%). Duroplasty used an artificial dura.

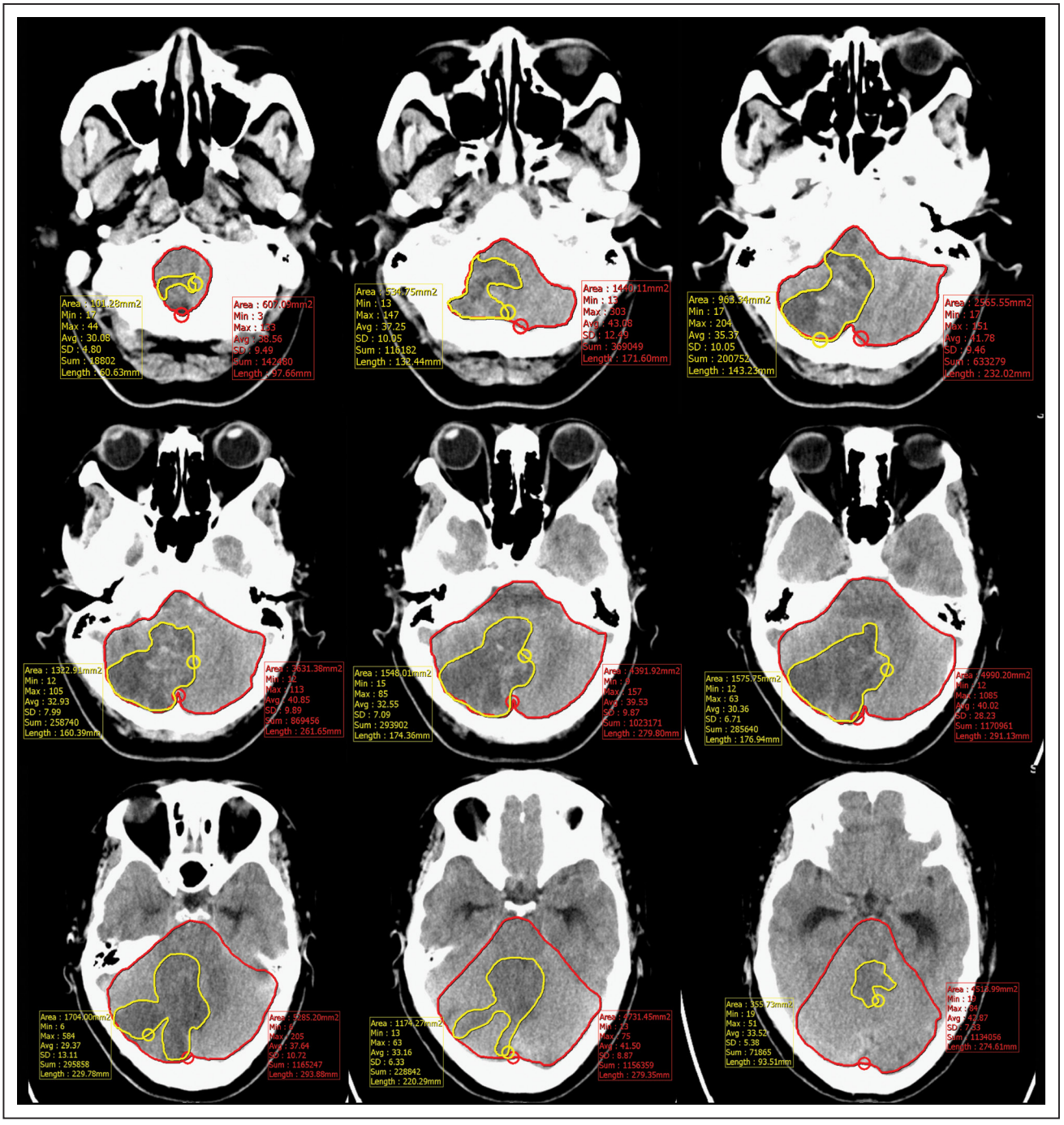

Figure 1. Cerebellar infarction volume and total cerebellar volume including brain stem volume. Measurements used the formula: volume (mm^3)=(A_1+A_2+A_3+⋯+A_N)×H, where N indicated number of image sections that showed the cerebellum, A (area, mm^2) was measured manually on each image section, and H (height, mm) could be 4 or 5 according to the thickness of the image section on a picture archiving and communication system viewer. Yellow lines indicate margin of cerebellar infarction; and red lines indicate margin of total cerebellum including brain stem.

Statistical Analysis

All statistical analyses were consulted with a biostatistician and were performed using R language ver. 3.01 (R Foundation for Statistical Computing, Vienna, Austria). Student *t* tests were used for numeric variables. χ^2 tests were used for nominal variables. The Kaplan–Meier method was used to analyze time to death, with the log-rank test used to compare mortality between groups. Logistic regression analysis was performed to determine independent risk factors for favorable clinical outcomes at the 12-month follow-up. Multiple logistic regression analyses were performed on variables with an unadjusted effect and *P* value <0.10 on simple logistic regression analysis. *P* value <0.05 was considered statistically significant.

Results

The clinical characteristics and outcomes of propensity score–matched subjects in both groups are shown in Table 1. Mean age was 59.0±11.6 years in group A and 59.4±10.9 years in group

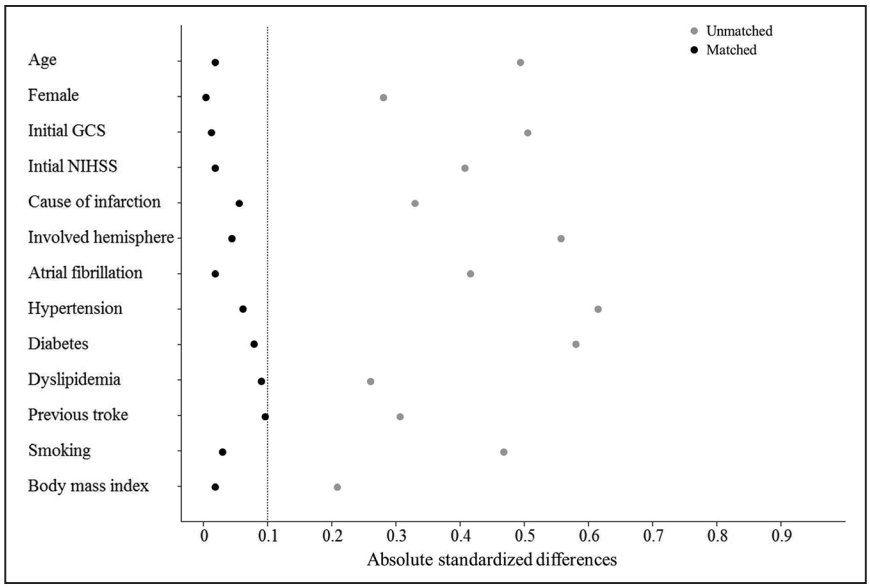

Figure 2. Absolute standardized difference (ASD) before and after propensity score matching. ASD <0.10 implied good balance between the 2 groups. GCS indicates Glasgow Coma Scale score; and NIHSS, National Institutes Health Stroke Scale score.

B (P=0.913). Initial GCS score was 12.1±4.1 in group A and 12.0±3.8 in group B (P=0.950). Initial NIHSS score was 5.2±2.1 in group A and 5.3±1.9 in group B (P=0.863). Procedure-related complications in group A occurred in 2 (7.1%) patients who underwent additional surgery for dura repair for cerebrospinal fluid leakage. In group B, 8 patients underwent SDC for clinical deterioration (decreased GCS score) combined with aggravated radiographic findings 72 hours from onset.

Clinical outcomes at discharge and after 12 months of follow-up were significantly different between groups. At discharge, 18 (64.3%) of the 28 patients in group A showed favorable outcomes with 1 death, whereas 27 (48.2%) of 56 patients in group B showed favorable outcomes with 5 deaths (P=0.048). The reason for death was brain stem compression caused by uncontrolled progressive cerebellar swelling. One patient in group A and 3 patients in group B died in spite of SDC. At the 12-month follow-up, 18 (66.7%) of the 27 patients in group A showed favorable outcomes, whereas 26 (51.0%) of the 51 patients in group B showed favorable outcomes (P=0.030; Figure 3). Deaths occurring within 12 months and the numbers of patients at risk per month are shown in Figure 4. More deaths occurred in group B than in group A (log-rank, P<0.05), which seemed to be consistent over time. The reasons for death were pneumonia sepsis (1 in group A and 1 in group B), sudden death (possibly because of heart problems; 2 patients in group B), and chronic illness after infarction (1 in group A and 1 in group B).

Logistic regression analysis with adjustment for age and sex showed that preventive SDC (odds ratio, 4.815; 95% confidence interval, 1.522–24.325; P=0.009) and the absence of brain stem infarction (odds ratio, 2.862; 95% confidence interval, 1.225–9.146; P=0.033) were independently associated with favorable outcomes (mRS, 0–2) at the 12-month follow-up (Table 2). Initial NIHSS score <8 had a P value of 0.048 in simple logistic regression analysis, but did not reach statistical significance after adjustment (P=0.068).

Discussion

The results of this study indicated that patients who underwent preventive SDC (group A) showed better clinical outcomes and survival than patients who did not receive preventive SDC (group B). The absence of brain stem infarction could affect favorable outcomes at the 12-month follow-up. We also found that patients with cerebellar infarction volume ratio between 0.25 and 0.33 could be expected to have favorable outcomes including overall survival after preventive SDC.

Patients with a small cerebellar infarction volume without brain stem damage tend to have a benign course and used to be managed conservatively.[9–11] However, large and malignant swellings after cerebellar infarction combined with clinical deterioration should be treated by SDC.[1,2,10,12–14] In the absence of these extreme or absolute indications, we need to know which patients could achieve better clinical outcomes with preventive SDC. Delayed swelling commonly follows a large infarction of the cerebellum. Clinical warning signs of severe edema formation such as progressive deterioration of consciousness and brain stem signs, may occur late when the patient is already regarded stable usually within 2 to 4 days. Additionally, they are unspecific and often misinterpreted. Although early symptoms may be limited to impaired cerebellum function, edema can cause brain stem compression and can rapidly progress to the loss of brain stem function. Thus, we performed preventive SDC, mainly according to the infarct volume ratio, in patients who were clinically stable without clinical deterioration within 72 hours. With reference to initial GCS score, patients with initial GCS score <9 should be considered for SDC because it might indicate rapid progression of cerebellar edema and loss of brain stem function. This fact made us to include patients with GCS score ≥9 for preventive SDC in the present study. Because we lack evidence of our indications and the benefits of preventive SDC, we tried to evaluate a subset of patients who might be expected to benefit from preventive SDC.

Previously, some authors recommended preventive SDC before signs of severe brain stem compression or hydrocephalus were present clinically or visible on CT or MRI because the value of clinical signs and neuroradiological parameters was uncertain or might be detected too late.[2,10] In addition, they insisted that the risk of SDC or EVD was fairly small

Table 1. Clinical Characteristics and Outcomes of Propensity Score–Matched Subjects

	Group A (n=28)	Group B (n=56)	*P* Value
Age, mean±SD	59.0±11.6	59.4±10.9	0.913
Female, n (%)	11 (39.3)	21 (37.5)	1.000
Initial GCS score, mean±SD	12.1±4.1	12.0±3.8	0.950
Initial NIHSS score, mean±SD	5.2±2.1	5.3±1.9	0.863
Cause of infarction, n (%)			0.621
Cardiac embolism	10 (35.7)	22 (39.3)	…
Arterial atherosclerosis	4 (14.3)	11 (19.6)	…
Dissection	8 (28.6)	15 (26.8)	…
Other or undetermined	6 (21.4)	8 (14.3)	…
Involved hemisphere, n (%)			0.734
Unilateral	17 (60.7)	31 (55.4)	…
Bilateral	11 (39.3)	25 (44.6)	…
Atrial fibrillation, n (%)	11 (39.3)	24 (42.9)	0.872
Hypertension, n (%)	12 (42.9)	22 (39.3)	0.674
Diabetes mellitus, n (%)	9 (32.1)	20 (35.7)	0.533
Dyslipidemia, n (%)	6 (21.4)	15 (26.8)	0.474
Previous stroke, n (%)	3 (10.7)	8 (14.3)	0.423
Smoking, n (%)	13 (46.4)	25 (44.6)	0.776
Body mass index, mean±SD	23.4±2.4	22.8±3.4	0.886
Volume ratio, mean±SD	0.46±0.08	0.49±0.10	0.365
Brain stem infarction, n (%)	10 (35.7)	22 (39.3)	0.272
Intravenous tPA, n (%)	3 (10.7)	14 (25.0)	0.059
Intra-arterial EVT, n (%)	3 (10.7)	11 (19.6)	0.094
Previous MI, n (%)	1 (3.6)	2 (3.6)	0.784
CAOD, n (%)	1 (3.6)	2 (3.6)	0.784
PAOD, n (%)	1 (3.6)	1 (1.8)	0.567
Hemorrhagic transformation	8 (28.6)	15 (26.8)	0.517
mRS at discharge			0.048
Favorable (0–2)	18 (64.3)	27 (48.2)	…
Unfavorable (3–6)	10 (35.7)	29 (51.8)	…
mRS at follow-up	n=27	n=51	0.030
Favorable (0–2)	18 (66.7)	26 (51.0)	…
Unfavorable (3–6)	9 (33.3)	25 (49.0)	…

CAOD indicates coronary artery occlusive disease; EVT, endovascular treatment; GCS, Glasgow Coma Scale; MI, myocardial infarction; mRS, modified Rankin Scale; NIHSS, National Institutes Health Stroke Scale; PAOD, peripheral artery occlusive disease; and tPA, tissue-type plasminogen activator.

and patients undergoing preventive SDC as a treatment option preceding hydrocephalus or brain stem compression tended to recover better than those undergoing emergency SDC after deterioration.[2] However, Koh et al[15] reported that infarct volume and territorial distribution of infarcts are not different in patients who deteriorate and those who remain stable. They found that only hydrocephalus, brain stem deformity, and basal cistern compression on CT or MRI seemed to correlate with clinical deterioration, suggesting some difficulties to perform preventive SDC. From the analysis of long-term clinical outcomes after SDC, Jüttler et al[16] concluded that the value of different treatment strategies for space-occupying cerebellar infarction and prognostic factors for patient selection remained unclear. They also suggested that randomized controlled trials for the issue of preventive SDC were needed. We agreed with their suggestion. However, randomized controlled trials for preventive SDC may be difficult to perform because of the slow enrollment of patients and various clinical

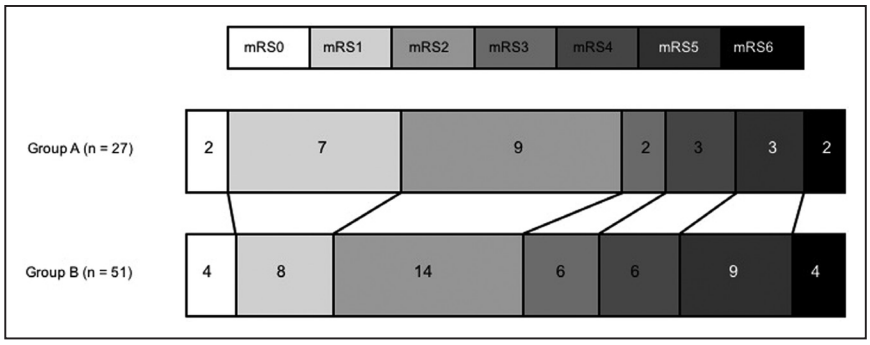

Figure 3. Clinical outcomes (modified Rankin Scale [mRS]) for 2 groups at the 12-month follow-up.

parameters. Thus, our findings, compared with the matched control group after balancing the baseline differences between the 2 groups, might be a good evidence to support the association of preventive SDC with favorable outcomes.

In most patients with space-occupying cerebellar infarction, obstructive hydrocephalus usually causes early sign of clinical deterioration.[11,17] Raco et al[11] recommended EVD as the first choice, and they considered SDC only when there was further clinical deterioration. However, EVD alone could be dangerous in those patients because it not only had the risk to induce upward herniation but also could not release brain stem compression, especially when mass effect was increasing.[12,18–21] Currently, SDC combined with EVD is considered as a proper option. In this study, however, the frequency of EVD placement (50%; 14 of the 28 patients in group A) was lower compared with previous reports.[10–14,16,22] Jüttler et al[16] performed EVD in 48 (85.7%) of 56 patients. Among them, 9 patients were treated by EVD alone. We usually performed EVD in all patients with space-occupying cerebellar infarction because they usually had significant hydrocephalus. In the 8 patients who underwent SDC in group B because of clinical deterioration combined with aggravated radiographic findings, all (100%) received EVD as a combined procedure with SDC. Furthermore, among the remaining 24 patients in our SDC series who were excluded from this study, 21 patients (87.5%) received EVD. In our SDC series of 60 patients, therefore, the frequency of EVD placement was 50% (14/28) in patients who underwent preventive SDC (group A), whereas the frequency was 90.6% (8 in group B and 21 in excluded group out of 32 patients) in patients who underwent emergent SDC.

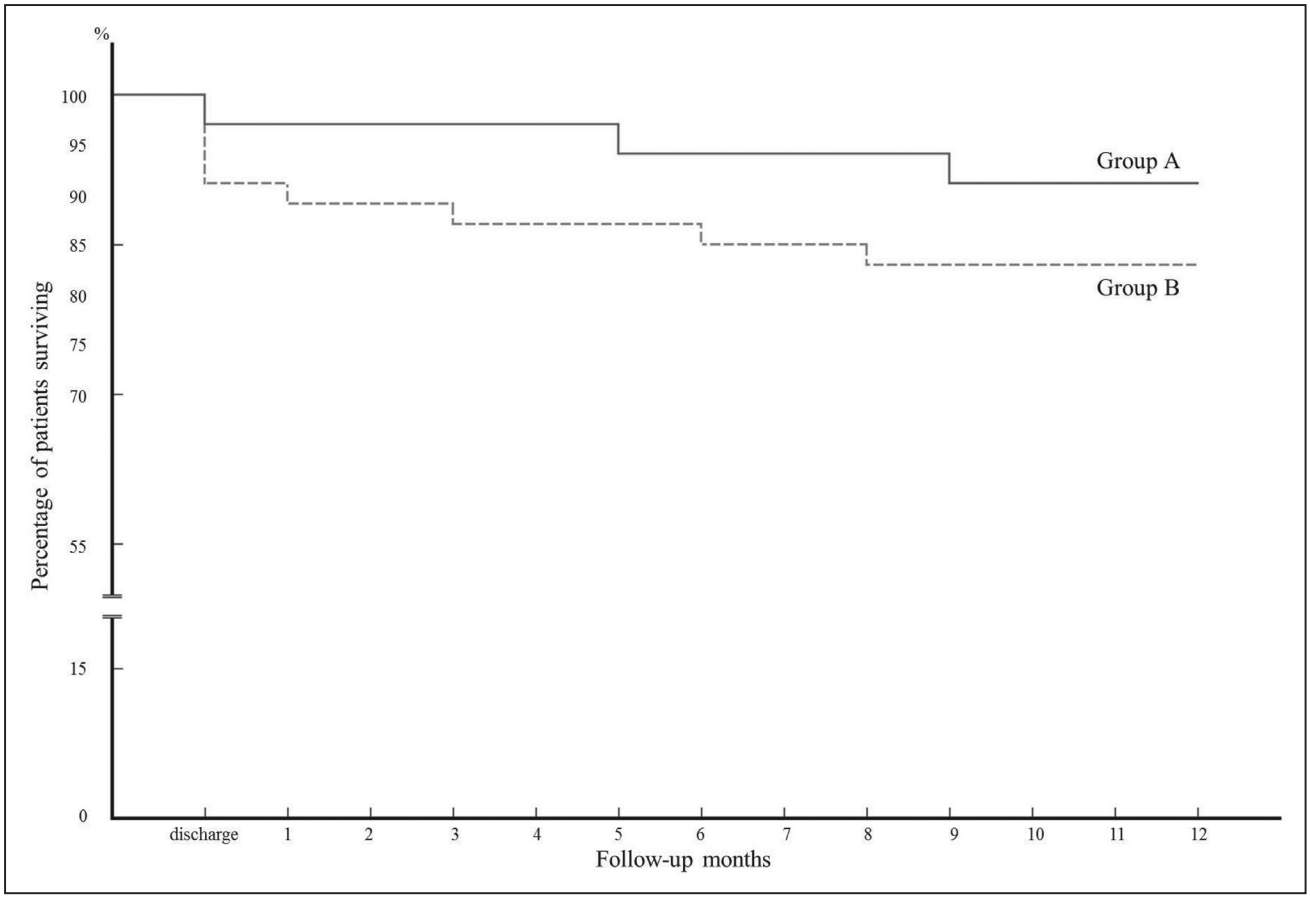

Figure 4. Kaplan–Meier cumulative mortality to 12 mo. Group B had more deaths than group A (log-rank, $P<0.05$).

Table 2. Predisposing Factors for Favorable Outcomes (Modified Rankin Scale, 0–2) at the 12-Month Follow-Up

	Unadjusted		Adjusted	
	OR (95% CI)	*P* Value	OR (95% CI)	*P* Value
Age, y				
<60	1	…	1	…
≥60	0.926 (0.903–1.026)	0.293	0.844 (0.682–1.223)	0.804
Sex				
Female	1	…	1	…
Male	0.644 (0.277–1.271)	0.188	0.528 (0.064–1.781)	0.726
Initial NIHSS				
≥8	1	…	1	…
<8	3.374 (1.017–8.373)	0.048	4.311 (0.892–10.475)	0.068
Cause of infarction				
Cardiac embolism	1	…	…	…
Arterial atherosclerosis	0.935 (0.479–2.753)	0.634	…	…
Dissection	1.172 (0.743–1.734)	0.514	…	…
Other or undetermined	1.018 (0.871–1.416)	0.439	…	…
Involved hemisphere				
Unilateral	1	…	…	…
Bilateral	0.927 (0.470–3.124)	0.668	…	…
Preventive SDC				
Not performed	1	…	1	…
Performed	3.711 (2.650–17.214)	0.007	4.815 (1.522–24.325)	0.009
Brain stem infarction				
Yes	1	…	1	…
No	2.123 (1.258–6.852)	0.042	2.862 (1.225–9.146)	0.033
EVD				
Yes	1	…	…	…
No	1.202 (0.519–2.660)	0.397	…	…
Atrial fibrillation				
Yes	1	…	…	…
No	0.832 (0.376–1.928)	0.365	…	…
Hypertension				
Yes	1	…	1	…
No	2.028 (0.869–6.484)	0.094	2.119 (0.973–8.682)	0.073
Diabetes mellitus				
Yes	1	…	…	…
No	1.077 (0.641–4.806)	0.552	…	…
Dyslipidemia				
Yes	1	…	…	…
No	1.164 (0.424–3.197)	0.769	…	…
Smoking				
Yes	1	…	…	…
No	0.724 (0.438–3.447)	0.728	…	…

(*Continued*)

Table 2. Continued

	Unadjusted		Adjusted	
	OR (95% CI)	*P* Value	OR (95% CI)	*P* Value
Intravenous tPA				
Yes	1	…	…	…
No	1.865 (0.675–12.680)	0.131	…	…
Intra-arterial EVT				
Yes	1	…	…	…
No	2.093 (0.699–5.623)	0.154	…	…
Hemorrhagic transformation				
Yes	1	…	…	…
No	0.728 (0.336–3.273)	0.687	…	…

CI indicates confidence interval; EVD, external ventricular drainage; EVT, endovascular treatment; NIHSS, National Institutes Health Stroke Scale; OR, odds ratio; SDC, suboccipital decompressive craniectomy; and tPA, tissue-type plasminogen activator.

It could be interesting to speculate whether preventive SDC reduces the frequency of EVD placement. And, we did speculate that it might be possible because early decompression of the aqueduct and ventricle before obstruction could help cerebrospinal fluid circulation.

It is well known that there is a strong correlation between recanalization and clinical outcome in acute ischemic stroke. However, this relationship might not be applicable to the present study because of the subset of selected patients who underwent preventive SDC. Without statistical significance, intravenous tPA (P=0.059) and intra-arterial EVT (P=0.094) tended to be more frequent in group B than in group A (Table 1). In group A, 3 patients who received intravenous tPA underwent intra-arterial EVT. In group B, 5 patients experienced recanalization of occluded vessels after receiving intravenous tPA alone. Additionally, 9 patients who received intravenous tPA underwent intra-arterial EVT. The other 2 patients underwent intra-arterial EVT without receiving intravenous tPA. The success rates of recanalization were 66.7% (2/3) in group A and 75.0% (12/16) in group B. There were no complications related to recanalization procedures except for hemorrhagic transformation after recanalization in 5 patients (2 in group A and 3 in group B). However, the clinical outcome was significantly better in group A than in group B at the 12-month follow-up (P=0.030).

This study had several limitations. First, this was a retrospective study without randomization and therefore had potential for selection bias even though we performed propensity score matching. As we mentioned it above, however, randomized controlled trials might be difficult to perform because patient recruitment would be slow. Our data from 5 hospitals over 10 years had only 60 cases of SDC. This number would be reduced by the inclusion and exclusion criteria of a randomized trial. In the present study, we used specific criteria to determine when to perform preventive SDC. However, several patients did not receive preventive SDC, as each institution had different time points for applying the criteria. Additionally, certain patients and their families, because of personal beliefs, were unwilling to receive any kind of brain surgery even if clinical symptoms became aggravated. Similarly, other patients and their families did not agree to receive preventive SDC as they felt that the condition was not clinically serious enough to warrant such treatment. These situations prevented us from performing preventive SDC in more patients. Second, the method for measuring volumes gave only approximate numbers measured by manual drawing (Figure 1). Therefore, the volume ratio could differ for patients, depending on the operator doing the measurements. However, we measured each image section that showed the cerebellum to obtain real volumes for infarction and cerebellum. Finally, preventive SDCs were performed by 5 neurosurgeons, all alumni of a single institution. This might have caused surgical techniques to vary in some certain ways and could have influenced clinical outcomes. However, our technique of SDC did not differ greatly from the currently generalized techniques.[1,13,16] Additionally, we performed bilateral SDC in all patients, and resection of infarcted cerebellum was performed in 57.1% of patients, while an EVD was inserted in 50.0% of patients. No patients were treated via EVD alone. Although the best neurosurgical approach (SDC only, SDC plus EVD, range of SDC, or removal of infarcted cerebellum) remains unknown, we think that our SDC techniques did not likely deviate from the general techniques and did not influence clinical outcomes. Although we performed preventive SDC using almost the same technique, we lack a standard SDC technique that can be easily, effectively, and safely performed by any neurosurgeon, whereas a newly suggested 5-keyhole method of hemicraniectomy could be performed easily for patients with malignant middle cerebral artery infarction.[23]

Conclusions

Favorable clinical outcomes including overall survival can be expected after preventive SDC in patients with a volume ratio between 0.25 and 0.33 and with the absence of brain stem infarction. Among these patients, preventive SDC might be better than the best medical treatment alone. A well-designed, large, randomized controlled multicenter trial should be conducted to evaluate the safety and efficacy of preventive SDC.

Acknowledgments

We heartily thank our partners, K.Y. Lee (Department of Neurology, Gangnam Severance Hospital, Yonsei University College of Medicine, Seoul, Korea), J.H. Rha (Department of Neurology, Inha University College of Medicine, Incheon, Korea), J. Koo (Department of Neurology, Seoul St. Mary's Hospital, Catholic University of Korea), J.M. Hong and J.S. Lee (Department of Neurology, Ajou University School of Medicine, Suwon, Korea), and D.H. Shin (Department of Neurology, Gachon University Gil Medical Center, Incheon, Korea) for their contribution to the present study by offering the best clinical management of the patients.

Disclosures

None.

References

1. Jauch EC, Saver JL, Adams HP Jr, Bruno A, Connors JJ, Demaerschalk BM, et al; American Heart Association Stroke Council; Council on Cardiovascular Nursing; Council on Peripheral Vascular Disease; Council on Clinical Cardiology. Guidelines for the early management of patients with acute ischemic stroke: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*. 2013;44:870–947. doi: 10.1161/STR.0b013e318284056a.
2. The European Stroke Organisation (ESO) Executive Committee and the ESO Writing Committee. Guidelines for management of ischaemic stroke and transient ischaemic attack 2008. *Cerebrovasc Dis*. 2008;25:457–506.
3. Hofmeijer J, Kappelle LJ, Algra A, Amelink GJ, van Gijn J, van der Worp HB; HAMLET Investigators. Surgical decompression for space-occupying cerebral infarction (the Hemicraniectomy After Middle Cerebral Artery Infarction With Life-Threatening Edema Trial [HAMLET]): a multicentre, open, randomised trial. *Lancet Neurol*. 2009;8:326–333. doi: 10.1016/S1474-4422(09)70047-X.
4. Vahedi K, Hofmeijer J, Juettler E, Vicaut E, George B, Algra A, et al; DECIMAL, DESTINY, and HAMLET Investigators. Early decompressive surgery in malignant infarction of the middle cerebral artery: a pooled analysis of three randomised controlled trials. *Lancet Neurol*. 2007;6:215–222. doi: 10.1016/S1474-4422(07)70036-4.
5. Song D, Cho AH. Previous and recent evidence of endovascular therapy in acute ischemic stroke. *Neurointervention*. 2015;10:51–59. doi: 10.5469/neuroint.2015.10.2.51.
6. Siu KL, Lee DG, Shim JH, Suh DC, Lee DH. Mechanical thrombectomy using the solitaire fr system for occlusion of the top of the basilar artery: intentional detachment of the device after partial retrieval. *Neurointervention*. 2014;9:26–31. doi: 10.5469/neuroint.2014.9.1.26.
7. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al; National Heart, Lung, and Blood Institute Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; National High Blood Pressure Education Program Coordinating Committee. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA*. 2003;289:2560–2572. doi: 10.1001/jama.289.19.2560.
8. James PA, Oparil S, Carter BL, Cushman WC, Dennison-Himmelfarb C, Handler J, et al. 2014 evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC 8). *JAMA*. 2014;311:507–520. doi: 10.1001/jama.2013.284427.
9. Khan M, Polyzoidis KS, Adegbite AB, McQueen JD. Massive cerebellar infarction: "conservative" management. *Stroke*. 1983;14:745–751.
10. Mathew P, Teasdale G, Bannan A, Oluoch-Olunya D. Neurosurgical management of cerebellar haematoma and infarct. *J Neurol Neurosurg Psychiatry*. 1995;59:287–292.
11. Raco A, Caroli E, Isidori A, Salvati M. Management of acute cerebellar infarction: one institution's experience. *Neurosurgery*. 2003;53:1061–1065, discussion 1065.
12. Chen HJ, Lee TC, Wei CP. Treatment of cerebellar infarction by decompressive suboccipital craniectomy. *Stroke*. 1992;23:957–961.
13. Pfefferkorn T, Eppinger U, Linn J, Birnbaum T, Herzog J, Straube A, et al. Long-term outcome after suboccipital decompressive craniectomy for malignant cerebellar infarction. *Stroke*. 2009;40:3045–3050. doi: 10.1161/STROKEAHA.109.550871.
14. Tsitsopoulos PP, Tobieson L, Enblad P, Marklund N. Surgical treatment of patients with unilateral cerebellar infarcts: clinical outcome and prognostic factors. *Acta Neurochir (Wien)*. 2011;153:2075–2083. doi: 10.1007/s00701-011-1120-4.
15. Koh MG, Phan TG, Atkinson JL, Wijdicks EF. Neuroimaging in deteriorating patients with cerebellar infarcts and mass effect. *Stroke*. 2000;31:2062–2067.
16. Jüttler E, Schweickert S, Ringleb PA, Huttner HB, Köhrmann M, Aschoff A. Long-term outcome after surgical treatment for space-occupying cerebellar infarction: experience in 56 patients. *Stroke*. 2009;40:3060–3066. doi: 10.1161/STROKEAHA.109.550913.
17. Mohsenipour I, Gabl M, Schutzhard E, Twerdy K. Suboccipital decompressive surgery in cerebellar infarction. *Zentralbl Neurochir*. 1999;60:68–73.
18. Hornig CR, Rust DS, Busse O, Jauss M, Laun A. Space-occupying cerebellar infarction. Clinical course and prognosis. *Stroke*. 1994;25:372–374.
19. Heros RC. Surgical treatment of cerebellar infarction. *Stroke*. 1992;23:937–938.
20. Kase CS, Wolf PA. Cerebellar infarction: upward transtentorial herniation after ventriculostomy. *Stroke*. 1993;24:1096–1098.
21. Tulyapronchote R, Malkoff MD, Selhorst JB, Gomez CR. Treatment of cerebellar infarction by decompression suboccipital craniectomy. *Stroke*. 1993;24:478–480.
22. Kudo H, Kawaguchi T, Minami H, Kuwamura K, Miyata M, Kohmura E. Controversy of surgical treatment for severe cerebellar infarction. *J Stroke Cerebrovasc Dis*. 2007;16:259–262. doi: 10.1016/j.jstrokecerebrovasdis.2007.09.001.
23. Chung J, Bang OY, Lim YC, Park SK, Shin YS. Newly suggested surgical method of decompressive craniectomy for patients with middle cerebral artery infarction. *Neurologist*. 2011;17:11–15. doi: 10.1097/NRL.0b013e3181f4ec88.