



Surgical treatment of cerebellar infarction: long-term outcomes and prognostic factors

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Objective: Patients with cerebellar infarction showing neurological deterioration require surgical treatment for posterior fossa decompression. Although decompressive suboccipital craniectomy (DSC) with or without ventriculostomy is recommended, long-term follow-up data on the outcomes of DSC are insufficient, and some prognostic factors have yet to be elucidated. We analyzed the long-term clinical outcomes and prognostic factors of decompressive surgery in patients with severe cerebellar infarction.

Methods: In this retrospective review of patients with cerebellar infarction with severe swelling who underwent surgical treatment between 2008 and 2018, 33 eligible patients were investigated to determine the neurological, surgical, and radiological parameters that could affect long-term outcomes. In assessments based on modified Rankin scale (mRS) scores, patients were considered to show a poor outcome if their score was 4 points or higher and a good prognosis if their score was less than 4 points.

Results: The initial neurological assessment was based on the Glasgow coma scale score. All patients underwent DSC, and necrosectomy was performed in 10 patients. Nine patients underwent external ventricular drainage to prevent upward transtentorial herniation and hydrocephalus progression. In long-term prognostic assessments conducted using the mRS score 2 years after surgery, 21 of the 30 surviving patients had scores of less than 4, while the other 9 had mRS scores of 4 or higher. In particular, 4 out of 5 patients with brain stem infarction remained severely disabled.

Conclusion: We analyzed several factors and found that the presence of brainstem infarction influenced long-term prognoses after DSC.

Keywords: Cerebellum; Infarction; Decompressive craniectomy; Outcomes assessment; Prognosis

Introduction

Cerebellar infarcts are medically curable diseases, and the majority of patients show mild symptoms, such as dizziness, vertigo, and ataxia [1]. However, a subgroup of patients with severe cerebellar swelling show clinical or radiological deterioration within several days after the onset of cerebellar stroke. Large cerebellar infarctions can result in a space-occupying mass effect and increase posterior fossa pressure. This pathophysiologic phenomenon can cause crit-

ical complications such as obstructive hydrocephalus, brain stem compression, and upward transtentorial herniation or downward transforaminal herniation, ultimately resulting in life-threatening conditions [2-5].

Surgical treatment is essential in patients who show severe neurological deterioration despite receiving maximal medical therapy. External ventricular drainage (EVD) is considered to be the first step in surgical management and is recommended for the treatment of obstructive hydrocephalus after cerebellar infarction [6]. In patients with cerebellar infarction showing clinical deterioration due to severe swelling, decompressive suboccipital craniectomy (DSC) with dural expansion is recommended as a life-saving procedure [6-9]. In each case, necrosectomy and/or resection of the posterior arch of C1 was performed additionally, and some previous studies have described their therapeutic effects [4,10,11].

However, long-term follow-up data of DSC are insufficient, with

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some debates on the optimal surgical technique, timing of surgery, and prognostic factors [7,8,12]. In our retrospective study, we surgically treated 33 patients with cerebellar infarction accompanying massive cerebellar swelling. We also analyzed the long-term outcomes and prognostic factors of decompressive surgery in patients with severe cerebellar infarction.

Material and Method

Patient selection

The study was designed as a retrospective, single-center trial. We included consecutive patients with cerebellar infarction accompanied by severe cerebellar swelling treated by DSC from January 2008 to December 2018. Additional procedures such as EVD and necrosectomy were performed if deemed appropriate. Data pertaining to the patients' medical history, neurological evaluation, radiological examinations, and surgical treatment were obtained from computerized patient charts. Patients with insufficient follow-up data were excluded from the study. A total of 33 patients who underwent DSC for cerebellar infarction were included in the study. The investigative protocol was approved by Inje University's Institutional Review Board (no. 2021-05-009).

Radiological evaluations

Brain computed tomography (CT) was initially performed for diagnosis, and brain magnetic resonance imaging (MRI) was conducted to confirm cerebellar infarction. In some cases, digital subtraction angiography was performed to confirm the arteries involved and to exclude abnormal vascular lesions. A neuroradiologist who was not involved in the treatment rated the mass effect on brain swelling caused by cerebellar infarction by assessing preoper-

ative brain CT images according to the CT score published by Jauss et al. [13]. This scoring system evaluates the ventricle, quadrigeminal cisterns, and lateral ventricle, and each item is graded from 0 to 3 depending on the severity. The total score was calculated by adding the scores for each item. A total score of 0 to 3 indicated no or slight mass effect, 4 to 6 indicated a moderate mass effect, and 7 to 9 indicated a severe mass effect (Fig 1).

Surgical procedure

We performed decompressive surgery and maximal duraplasty in patients with moderate to severe cerebellar swelling who showed acute neurologic deterioration despite maximal medical therapy. The CT scores were used to recognize infarct size and degree of brainstem compression, but the swelling and mass effects may deteriorate quickly in the acute period, so we gave more importance to the immediate symptoms of patients in determining surgery. Removal of necrotic tissue and resection of the posterior arch of C1 could be determined by CT findings prior to surgery, but were mostly determined by the degree of brain swelling in the surgical findings. EVD was performed in the right frontal horn if compression of the fourth ventricle with obstructive hydrocephalus was apparent before or after DSC.

Examination protocol

All patients were treated in the neurological intensive care unit (NICU), examined using a standardized examination protocol, and evaluated in serial neurological examinations conducted daily after hospitalization and immediately before and after surgery. The level of consciousness was assessed using the Glasgow coma scale (GCS). We also assessed cranial nerve abnormalities and focal neurological signs, including motor abnormalities, coordination



Fig. 1. Examples of computed tomography scores based on the method reported by Jauss et al. [13], which represents the degree of swelling as follows: (A) mild, (B) moderate, and (C) severe.

disturbances, sensory disturbances, and reflex status. Repeat CT was performed at least 3 times during the pre- and postoperative periods. Functional deficits were assessed using the modified Rankin scale (mRS): 1 = no symptoms; 2 = no significant disability despite mild symptoms; 3 = moderate disability, requires some help, but is able to walk without assistance; 4 = moderate severe disability, unable to attend to own bodily needs without assistance and unable to walk without assistance; 5 = severe disability, requiring constant nursing care, being bedridden; and 6 = death. The mRS score was examined at each outpatient visit after discharge.

Analysis and statistical methods

Demographic data, clinical features, level of consciousness evaluated using the GCS, neurological and radiological examination data, and surgical results were obtained from the patients' electronic medical records. We divided the patients into 2 groups according to the mRS scores measured 2 years after surgery to determine the long-term prognostic factors. Based on previous studies, a mRS score of 4 or more was considered to indicate a poor outcome [14–17]. Age, GCS score before surgery, bilateral infarction, brainstem infarction, surgical methods, and time between stroke onset and DSC were investigated for the analysis of prognostic factors. To compare categorical data between the groups, cross-tables were produced using Fisher exact test to determine statistical significance. The Mann-Whitney U test was used for continuous data. Statistical analysis was performed using IBM SPSS ver. 22.0 (IBM Corp., Armonk, NY, USA), and a P-value < 0.05 was considered to indicate significance.

Results

Patient characteristics

Data were collected from 33 patients who underwent DSC for acute cerebellar infarction. Twenty-two patients (66.7%) were male, and the average age was 63 years (range, 43–82 years) (Table 1). Twenty-seven of 33 patients had at least one cardiovascular risk factor, such as atrial fibrillation (2, 6.1%), hypertension (25, 75.8%), diabetes mellitus (10, 30.3%), hypercholesterolemia (6, 18.2%), smoking (10, 30.3%), or chronic kidney disease (1, 3.0%). Before cerebellar infarction, none of the patients suffered brain damage, including cerebral hemorrhage, infarction, or tumors.

Clinical findings and preoperative course

The median GCS score was 13, and 28 patients had a GCS score \geq 8 at admission. The most common symptoms of cerebellar infarction onset were vertigo (75.8%) and nausea (39.4%). The most common focal neurological abnormalities at admission were

Table 1. Demographic data of the patients treated using decompressive suboccipital craniectomy (n=33)

| Basic characteristic | Value |
|--|------------|
| Age (yr) | 63 (43–82) |
| Male sex | 22 (66.7) |
| Vascular risk factors | |
| Hypertension | 25 (75.8) |
| Diabetes mellitus | 10 (30.3) |
| Smoking | 10 (30.3) |
| Hypercholesterolemia | 6 (18.2) |
| Atrial fibrillation | 2 (6.1) |
| Chronic kidney disease | 1 (3.0) |
| Initial symptom | |
| Vertigo | 25 (75.8) |
| Nausea | 13 (39.4) |
| Headache | 10 (30.3) |
| Vomiting | 8 (24.2) |
| Dysarthria | 2 (6.1) |
| Mental deterioration | 1 (3.0) |
| Neurological signs at admission | |
| Ataxia | 24 (72.7) |
| Reduced consciousness | 23 (69.7) |
| Dysarthria | 14 (42.4) |
| Motor weakness | 7 (21.2) |
| Oculomotor dysfunction | 2 (6.1) |
| Dysphagia | 1 (3.0) |
| Level of consciousness | |
| GCS score at admission | 13 (6–15) |
| Preoperative GCS | 8 (6–10) |
| Postoperative GCS | 13 (6–15) |
| Infarction location | |
| Posterior inferior cerebellar artery | 29 (87.9) |
| Superior cerebellar artery | 10 (30.3) |
| Anterior inferior cerebellar artery | 3 (9.1) |
| Posterior cerebral artery | 5 (15.2) |
| Unilateral infarction | 25 (75.8) |
| Bilateral infarction | 8 (24.2) |
| Brainstem infarction | 7 (21.2) |
| Etiology | |
| Large vessel disease | 14 (42.4) |
| Cardiac embolism | 9 (27.3) |
| Undetermined | 10 (30.3) |
| Documented arterial occlusion/stenosis | |
| Basilar artery occlusion | 4 (12.1) |
| Vertebral artery occlusion | 4 (12.1) |
| Vertebrobasilar stenosis | 6 (18.2) |
| Hydrocephalus | 16 (48.5) |
| Surgical procedure | |
| External ventricular drainage | 8 (24.2) |
| Removal of necrotic tissue | 10 (30.3) |

Values are presented as median (range) or number (%). GCS, Glasgow coma scale.

ataxia (72.7%) and decreased consciousness (69.7%). In most patients (30, 90.9%), consciousness deteriorated between admission

and surgery, with a median GCS score reduction from 13 to 8. Mean time from stroke onset to surgery was 15.8 ± 17.2 hours (range, 4–72 hours). Twenty-four patients underwent DSC within 24 hours after admission, 7 underwent surgery between 24 and 48 hours after admission, and 2 underwent surgery between 48 and 72 hours after admission.

Neuroradiological findings

Brain CT was performed in all patients, and additional examinations, including CT angiography, MRI, and transfemoral cerebral angiography, were performed to better delineate infarct extension in some patients. Cerebellar infarction was bilateral in 8 patients (24.2%). The territory of the posterior inferior cerebellar artery (PICA) was the most commonly affected (87.9%), followed by the superior cerebellar artery (SCA). Additional brainstem infarctions were found in 7 patients (21.2%) (Table 1). Fourth ventricle compression was found in most patients (28, 84.8%), and hydrocephalus was found in 16 patients (48.5%).

Surgical treatment

All 33 patients were treated with DSC and duraplasty. Removal of necrotic tissue was performed in 10 patients (30.3%). Preoperative EVD was performed in 3 patients to prevent upward transtentorial herniation. Postoperative EVD was performed in 5 patients who showed persistent hydrocephalus after DSC and in one patient with postoperative intraventricular hemorrhage. Seven patients

with mild hydrocephalus did not require EVD. Minimal intracerebral hemorrhage was found on postoperative CT in 2 patients, but no additional surgery was required. The EVD could be removed within 2 weeks in all patients, and none of the patients underwent ventriculoperitoneal shunting. None of the patients required resection of the posterior arch of C1.

Early course and mortality

All patients were monitored and treated in the NICU. The mean duration of NICU treatment was 21.3 ± 11.2 days (range, 7–72 days). During this phase, 2 patients died: 1 patient died from acute myocardial infarction and one patient died from sepsis. The surviving patients were referred to the rehabilitation department. One month after discharge, one patient died of myocardial infarction.

Long-term outcome and prognostic factors

Over a follow-up period of approximately 2 years, no other patients died. Twenty-one of the 30 patients had a favorable functional outcome (mRS, 0 to 3), while the remaining 9 patients showed persistent major disability (mRS, 4 to 5) at the 2-year follow-up assessment. Fig. 1 shows the distribution of mRS scores. In univariate analysis, radiological findings of brainstem infarction were associated with poor outcomes (mRS ≥ 4) at the 2-year follow-up (Table 2). Only 1 of the 21 patients in the favorable functional outcome group showed brain stem infarction, while 4 of the 9 in the poor functional outcome group had brain stem infarction.

Table 2. Univariate analysis of long-term outcomes

| Patient factor | mRS ≤ 3 (n = 21) | mRS ≥ 4 (n = 9) | P-value |
|---|-----------------------|----------------------|---------|
| Age (yr) | 60.29 \pm 11.34 | 64.89 \pm 12.18 | 0.327 |
| Age ≥ 60 yr | 13 (61.9) | 5 (55.6) | 0.745 |
| GCS score at admission | 13 (8–15) | 13 (6–15) | 0.47 |
| GCS score ≤ 8 | 1 (4.8) | 3 (33.3) | 0.069 |
| GCS before surgery | 8 (7–10) | 8 (6–9) | 0.145 |
| GCS score ≤ 8 | 12 (57.1) | 6 (66.7) | 0.704 |
| Bilateral infarction | 4 (19.0) | 3 (33.3) | 0.64 |
| Brainstem infarction | 1 (4.8) | 4 (44.4) | 0.019 |
| Treatment factors | | | |
| EVD as initial treatment attempt before DSC | 1 (4.8) | 2 (22.2) | 0.207 |
| Infarction evacuation (necrosectomy) | 7 (33.3) | 3 (33.3) | 0.669 |
| Interval between ictus and operation | 17.38 \pm 19.74 | 11.89 \pm 9.28 | 0.435 |
| Hydrocephalus | 11 (52.4) | 5 (55.6) | 0.596 |
| Computed tomography score | | | |
| Mild | 3 (14.3) | 2 (22.2) | |
| Moderate | 13 (61.9) | 4 (44.4) | |
| Severe | 5 (23.8) | 3 (33.3) | |

Values are presented as mean \pm standard deviation, number (%), or median (range).

mRS, modified Rankin scale; GCS, Glasgow coma scale; EVD, external ventricular drainage; DSC, decompressive suboccipital craniectomy.

Patients in the favorable outcome group were younger than those in the poor outcome group on average (60.29 vs. 64.89 years), but the difference was not statistically significant ($P = 0.33$). Other factors such as sex, GCS score, vascular risk factors, etiology, symptoms, surgical factors, CT scores, neurological signs, and the laterality of cerebellar infarction showed no significant association with poor outcomes.

Discussion

DSC is considered the treatment of choice in patients with space-occupying cerebral infarctions [5,7,9,18–21]. However, the surgical procedures for cerebellar infarction are decided on the basis of clinical experience and previous retrospective studies because randomized controlled trials comparing surgery to medical treatment for severe cerebellar infarction are difficult to perform; thus, retrospective studies of long-term prognostic indicators after DSC can be performed to improve the validity and reliability of surgery. Our findings confirm that poor functional outcomes at approximately 2 years after DSC were more related to brain stem infarction than any of the other factors.

Although surgical indications for space-occupying middle cerebral artery infarction have been standardized through randomized controlled trials, the surgical treatment for cerebellar infarction is still not standardized [22–24]. This has led to some debates on the type of surgery, including DSC, EVD, additional necrosectomy, and resection of the posterior arch of C1. This study included no cases of resection of the posterior arch of the C1. EVD was considered the initial treatment for patients showing decreased consciousness due to obstructive hydrocephalus and was performed in patients with persistent hydrocephalus at any time before or after surgery. Additional evacuation of necrotic tissue was performed during DSC in patients who required an increase in decompression volume and decreased cytotoxic edema. Although the use of various surgical methods caused great heterogeneity in the prediction of prognostic factors, the analysis is meaningful because all surgical methods achieved the primary objective of ensuring decompression of the cerebellum to minimize damage to brain tissue.

In this study, the mortality rate was 9.1% (3/33), and 30.0% (9/30) of the patients had poor functional outcomes ($mRS \geq 4$). Previous studies on long-term outcomes have reported mortality rates ranging from 20% to 32%, with 40% to 49% of the patients showing poor outcomes [7–9,18,25,26]. In a systematic review and meta-analysis by Ayling et al. [26] (11 studies with 283 patients), moderate to severe disability was observed in 28% of patients, and the mortality rate at a median follow-up period of 9 years after DSC for cerebellar infarction was 20%. In a study by

Lindeskog et al. [18], poor outcomes ($mRS \geq 4$) were seen in 46% of 22 patients at the yearly follow-up after DSC, and brainstem infarction and bilateral cerebellar infarction were associated with poor outcomes. The mortality rate and percentage of patients showing poor outcomes in the present study were lower than those in previous studies. This may be attributed to the short follow-up period (2 years). In addition, the selective bias, in which patients who can visit the hospital often would have affected it.

Decreased level of consciousness with or without brain stem compression signs is a strong indication for decompressive surgery [8,13,27]. Neurological deterioration usually occurs within 3 to 4 days (range, 0–9) after onset [5,8,13,28,29]. Due to the non-specific symptoms of cerebellar infarction and the low sensitivity of CT imaging in evaluating the posterior fossa, edema development is not recognized until it becomes severe. Therefore, deterioration of consciousness is an important warning sign of severe cerebellar swelling [8,13,27]. For immediate treatment, including surgery, patients should be kept under observation in stroke or intensive care units for the first week after onset. Although some studies have shown that preoperative GCS scores are predictors of long-term outcomes [8,16,30], they were not statistically significant in our study. This is presumed to be due to the fact that a number of factors were not controlled, including the time taken to arrive at the hospital and the medication content.

Many authors report that the most frequently affected area in cerebellar infarction is the PICA territory, followed by the SCA [7,31–33]. Some reports have demonstrated that patients with PICA infarction are especially at risk for malignant cerebellar edema, and severe swelling in isolated SCA and anterior inferior cerebellar artery (AICA) infarction is rare [34]. The most commonly affected artery in our study was the PICA, followed by the SCA, PCA, and AICA. A comparison of swelling severity was not possible because data regarding arteries other than the PICA were scarce in our study.

In this study, a high CT score indicating severe compression was not associated with patient prognosis. Rather, the presence of brainstem infarction showed the greatest effect on prognosis. Decompressive surgery can release the brainstem pressure caused by brain swelling; however, the effectiveness of decompression surgery is reduced if the brainstem itself is infarcted. Moreover, this study only focused on patients with severe cerebellar infarction who underwent surgery, so comparison of CT scores with those obtained for patients who received only medical treatment would produce meaningful results. In addition, we recommend preoperative MRI examinations to distinguish brainstem infarction and compression. Since the evaluations and CT scoring were performed just before surgery, moderate grade accounted for the larg-

est percentage of cases (Table 2).

Limitations

This study had several limitations. First, this was a retrospective study that only compared patients who underwent surgery. The exclusion of patients with cerebellar infarction who only received medical treatment caused a selection bias. Second, additional EVD or necrosectomy was performed at the discretion of the 4 surgeons without a standardized protocol. Therefore, this study only reflects the postoperative prognosis of a selected group patients, and the findings have limited generalizability. For this reason, a randomized controlled study with a wider patient population should be conducted to improve the overall quality of the findings, allowing appropriate comparison of the prognosis of different surgical methods.

Conclusion

Surgical treatment is an essential procedure that can save the lives of patients with severe cerebellar infarction. However, surgical protocols are not well-established, and there is a lack of research on the long-term prognostic factors after surgery. Our study showed that the presence of brainstem infarction influenced the postoperative functional outcomes. This finding can facilitate the long-term management of these patients, including rehabilitation for functional recovery. Future research should focus on identifying prognostic factors that can predict functional outcomes by standardizing treatment protocols for severe cerebellar infarction.

Conflicts of interest

No potential conflict of interest relevant to this article was reported.

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REFERENCES

1. Cioffi FA, Bernini FP, Punzo A, D'Avanzo R. Surgical management of acute cerebellar infarction. *Acta Neurochir (Wien)* 1985;74:105–12.
2. Jensen MB, St Louis EK. Management of acute cerebellar stroke. *Arch Neurol* 2005;62:537–44.

3. Koh MG, Phan TG, Atkinson JL, Wijidicks EF. Neuroimaging in deteriorating patients with cerebellar infarcts and mass effect. *Stroke* 2000;31:2062–7.
4. Raco A, Caroli E, Isidori A, Salvati M. Management of acute cerebellar infarction: one institution's experience. *Neurosurgery* 2003;53:1061–5.
5. Hornig CR, Rust DS, Busse O, Jauss M, Laun A. Space-occupying cerebellar infarction. Clinical course and prognosis. *Stroke* 1994;25:372–4.
6. Powers WJ, Rabinstein AA, Ackerson T, et al. Guidelines for the early management of patients with acute ischemic stroke: 2019 update to the 2018 guidelines for the early management of acute ischemic stroke. A guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 2019;50:e344–418.
7. Pfefferkorn T, Eppinger U, Linn J, et al. Long-term outcome after suboccipital decompressive craniectomy for malignant cerebellar infarction. *Stroke* 2009;40:3045–50.
8. 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;40:3060–6.
9. 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; 153:2075–83.
10. Kim MJ, Park SK, Song J, et al. Preventive suboccipital decompressive craniectomy for cerebellar infarction: a retrospective-matched case-control study. *Stroke* 2016;47:2565–73.
11. Tartara F, Bongetta D, Colombo EV, et al. Strokectomy and extensive cerebrospinal fluid drainage for the treatment of space-occupying cerebellar ischemic stroke. *World Neurosurg* 2018;115:e80–4.
12. 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;16:259–62.
13. Jauss M, Krieger D, Hornig C, Schramm J, Busse O. Surgical and medical management of patients with massive cerebellar infarctions: results of the German-Austrian Cerebellar Infarction Study. *J Neurol* 1999;246:257–64.
14. Wilson JT, Hareendran A, Grant M, et al. Improving the assessment of outcomes in stroke: use of a structured interview to assign grades on the modified Rankin Scale. *Stroke* 2002;33: 2243–6.
15. Chen HJ, Lee TC, Wei CP. Treatment of cerebellar infarction by decompressive suboccipital craniectomy. *Stroke* 1992;23:957–61.

16. Tohgi H, Takahashi S, Chiba K, Hirata Y. Cerebellar infarction. Clinical and neuroimaging analysis in 293 patients. The Tohoku Cerebellar Infarction Study Group. *Stroke* 1993;24:1697–701.
17. Lee MS, Lee YS, Lee JH, Ryu KY, Kang DG. Clinical outcomes of decompressive craniectomy for malignant brain edema after acute cerebral infarction. *J Korean Soc Geriatr Neurosurg* 2011;7:132–7.
18. Lindeskog D, Lilja-Cyron A, Kelsen J, Juhler M. Long-term functional outcome after decompressive suboccipital craniectomy for space-occupying cerebellar infarction. *Clin Neurol Neurosurg* 2019;176:47–52.
19. Gurol ME, St Louis EK. Treatment of cerebellar masses. *Curr Treat Options Neurol* 2008;10:138–50.
20. Heros RC. Surgical treatment of cerebellar infarction. *Stroke* 1992;23:937–8.
21. Khaja AM, Grotta JC. Established treatments for acute ischaemic stroke. *Lancet* 2007;369:319–30.
22. Hofmeijer J, Kappelle LJ, Algra A, et al. 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;8:326–33.
23. Vahedi K, Vicaut E, Mateo J, et al. Sequential-design, multicenter, randomized, controlled trial of early decompressive craniectomy in malignant middle cerebral artery infarction (DECIMAL Trial). *Stroke* 2007;38:2506–17.
24. Han H, Koh EJ, Choi H, Kim BC, Yang SY, Cho KT. The effect of decompressive craniectomy in elderly patients aged 80 and over with middle cerebral artery infarction. *J Korean Soc Geriatr Neurosurg* 2016;12:206–11.
25. Tsitsopoulos PP, Tobieson L, Enblad P, Marklund N. Clinical outcome following surgical treatment for bilateral cerebellar infarction. *Acta Neurol Scand* 2011;123:345–51.
26. Ayling OGS, Alotaibi NM, Wang JZ, et al. Suboccipital decompressive craniectomy for cerebellar infarction: a systematic review and meta-analysis. *World Neurosurg* 2018;110:450–9.
27. Langmayr JJ, Buchberger W, Reindl H. Cerebellar hemorrhage and cerebellar infarct: retrospective study of 125 cases. *Wien Med Wochenschr* 1993;143:131–3.
28. Cuneo RA, Caronna JJ, Pitts L, Townsend J, Winestock DP. Upward transtentorial herniation: seven cases and a literature review. *Arch Neurol* 1979;36:618–23.
29. Khan M, Polyzoidis KS, Adegbite AB, McQueen JD. Massive cerebellar infarction: "conservative" management. *Stroke* 1983;14:745–51.
30. Puffer RC, Graffeo C, Rabinstein A, Van Gompel JJ. Mortality rates after emergent posterior fossa decompression for ischemic or hemorrhagic stroke in older patients. *World Neurosurg* 2016;92:166–70.
31. Hong JM, Bang OY, Chung CS, Joo IS, Huh K. Frequency and clinical significance of acute bilateral cerebellar infarcts. *Cerebrovasc Dis* 2008;26:541–8.
32. Kang DW, Lee SH, Bae HJ, Han MH, Yoon BW, Roh JK. Acute bilateral cerebellar infarcts in the territory of posterior inferior cerebellar artery. *Neurology* 2000;55:582–4.
33. Gurer G, Sahin G, Cekirge S, Tan E, Saribas O. Acute bilateral cerebellar infarction in the territory of the medial branches of posterior inferior cerebellar arteries. *Clin Neurol Neurosurg* 2001;103:194–6.
34. Kase CS, Norrving B, Levine SR, et al. Cerebellar infarction. Clinical and anatomic observations in 66 cases. *Stroke* 1993;24:76–83.