

Treatment of Cerebellar Infarction by Decompressive Suboccipital Craniectomy

Han-Jung Chen, MD; Tao-Chen Lee, MD; and Chi-Peng Wei, MD

Background and Purpose: We present an anecdotal series of 11 patients without past history of stroke with progressive neurological deterioration while on medical therapy for large cerebellar infarctions. Clinical signs of brain stem compression developed in these patients. Computerized tomography of the head confirmed mass effect from brain edema. It was the clinical judgment of the neurologists and neurosurgeons that each of these 11 patients would expire without surgical intervention.

Methods: All 11 patients (seven men, four women; mean age, 54 years) were treated with suboccipital craniectomy for decompression and temporary ventriculostomy for cerebrospinal fluid pressure monitoring and drainage.

Results: Seven patients demonstrated neurological improvement on the first postoperative day. Two patients returned to their previous jobs 3 months after surgery. The Barthel Index indicated that six individuals were functioning with minimal assistance within a follow-up period of 16–60 months. The remaining three were functionally dependent. No mortality was noted in this series.

Conclusions: These results suggest that decompressive suboccipital craniectomy may be an effective, lifesaving procedure for malignant cerebellar edema after a large infarction. (*Stroke* 1992;23:957–961)

KEY WORDS • cerebellar infarction • hydrocephalus • surgery

Stroke is the most prevalent disease involving the central nervous system. During the acute period after a cerebellar infarction, current management is primarily supportive to prevent extension of the infarct or the development of pulmonary complication. Neurological deterioration is attributed to the surrounding edema during the acute period. Fortunately, the patients usually improve as the edema subsides. However, cerebellar edema can result in marked hydrocephalus and brain stem compression by upward transtentorial or tonsillar herniation.^{1–3} Contemporary medical modalities include steroids, mannitol, barbiturates, and hyperventilation.¹ Since antiedemic agents are sometimes ineffective for the acute edema following infarction, surgical decompression by suboccipital craniectomy may be an effective alternative when medical modalities fail. In this study we present an anecdotal series that describes the results of suboccipital craniectomy on 11 patients presenting with progressive neurological deterioration while on medical therapy for large cerebellar infarctions.

Subjects and Methods

Patients experiencing spontaneous acute cerebellar infarction were admitted to the neurological or neurosurgical services. These patients were followed by a stroke service team composed of neurologists, neuro-

surgeons, and respiratory care professionals. Stroke was diagnosed by clinical history, physical examination, and head computed tomography (CT). Patients diagnosed with stroke were treated with adequate medical supportive methods. When necessary they were observed in neurological intensive care units. Usually 300 mg aspirin was given daily. If the victim began demonstrating neurological deterioration, antiedemic agents, such as mannitol, steroids, and hyperventilation, were implemented. Emergency head CT was used for clinical evaluation. When the clinical judgment of the physicians was that medical therapy was ineffective and a fatal outcome was imminent, then emergency surgical decompression was performed.

From January 1986 to September 1989, 11 cerebellar infarction victims without stroke and trauma histories (seven men, four women; age range, 36–73 years) deteriorated while on medical therapy (Table 1). Five of these patients had deteriorated at local hospitals and were transferred to our hospital for treatment. In all patients, clinical signs of brain stem compression accompanied neurological deterioration. Computed tomography demonstrated unilateral or bilateral cerebellar infarction with hydrocephalus. The range of time that elapsed between onset of symptoms and further neurological deterioration was 24–168 hours (median time, 72 hours). All 11 patients were thought to be responding poorly to aggressive medical treatment. Each of these stroke victims was treated with temporary ventricular drainage and suboccipital craniectomy with opening of the foramen magnum for decompression.

Patients were placed in the lateral decubitus position to avoid the possibility of air embolism. The head was turned slightly to the opposite site on a Mayfield pin headholder. The neck was kept in slight flexion, and a

From the Division of Neurosurgery, Department of Surgery, Chang Gung Medical School and Hospital at Kaohsiung, Kaohsiung, Taiwan, Republic of China.

Address for reprints: Han-Jung Chen, MD, Division of Neurosurgery, Department of Surgery, Chang Gung Memorial Hospital at Kaohsiung, Niao-Sung Hsiang, Kaohsiung Hsien, Taiwan, Republic of China.

Received August 9, 1991; accepted March 2, 1992.

TABLE 1. Profiles of 11 Patients With Cerebellar Infarction Treated by Decompressive Suboccipital Craniectomy

Patient	Age/Sex	Associated illness	GCS on admission	GCS before surgery	Infarct location	Time from ictus to deterioration (hours)
1	51/M	None	13	7	L PICA	30
2	48/M	Polycythemia	14	8	L PICA	168
3	56/F	None	12	7	L SCA	70
4	56/F	*	9	7	BL SCA	48
5	58/M	None	13	4T	R PICA	24
6	36/M	†	14	7	BL SCA	30
7	60/F	Hypertension	12	8	R AICA	48
8	50/F	‡	13	5T	L PICA	96
9	50/M	BL ureteral stones, hypertension	13	4T	BL PICA	34
10	73/M	Hypertension	14	5T	R PICA	144
11	57/M	None	15	7	R PICA	96

GCS, Glasgow Coma Scale score; M, male; F, female; T, intubated; L, left; BL, bilateral; R, right; PICA, posterior inferior cerebellar artery; SCA, superior cerebellar artery; AICA, anterior inferior cerebellar artery.

*Laparotomy for ovarian papillary cystadenocarcinoma 6 months after craniectomy.

†Sigmoid cancer operated 2 months before craniectomy.

‡Rheumatic heart disease with atrial fibrillation.

good airway was maintained. If bilateral lesions were present patients were placed in the prone position. A unilateral occipital horn ventricular draining catheter was usually set up before craniectomy for marked hydrocephalus. It was used for monitoring intraventricular pressure and postoperative cerebrospinal fluid drainage. A paramedian or median skin incision was made, depending on the location of the lesions. Unilateral or bilateral suboccipital craniectomy with opening of the foramen magnum was performed. The upper and lateral margin of craniectomy extended to the transverse and lateral sinuses. The dura was opened in a large cruciate incision. As the dura was opened, the infarcted brain began herniating outward. Decompressive resection of the herniated infarcted brain was done in nine cases because of tightness of the posterior fossa on closure of the wound. The ventricular draining catheter was usually removed in 72 hours.

Patients were evaluated by the Glasgow Coma Scale (GCS) and by neurological examination before and after surgery (Tables 1 and 2). They were transferred

for rehabilitation if the clinical conditions became stable. After discharge from the hospital, patients were also evaluated for functional independence by the Barthel Index (BI) scale (Table 3).⁴ A patient scoring 100 by this scale is able to perform daily activities without assistance. A score of 61–95 indicates a patient who

TABLE 3. Barthel Index

Index item/level of functioning	Score	
	Independent	With help
1. Feeding (food needs to be cut up=help)	10	5
2. Personal toilet (wash face, comb hair, brush teeth, etc.)	5	0
3. Bathing	5	0
4. Dressing	10	5
5. Bowel control (occasional accident or needs enema or suppository=help)	10	5
6. Bladder control (occasional accident or needs help with collecting device=help)	10	5
7. Toilet transfers	10	5
8. Chair/bed transfers (minimal assistance=10; able to sit but needs maximum assistance to transfer=5)	15	5–10
9. Ambulation (unable to walk but able to propel wheelchair=5)	15	5–10
10. Stair climbing (independent with assistive devices=10)	10	5
Level of functioning	Total score	
Totally dependent	0–20	
Severely dependent	21–60	
Moderately dependent	61–90	
Slightly dependent	91–99	
Independent	100	

TABLE 2. Glasgow Coma Scale Score on Seventh Postoperative Day and Barthel Index Score After Follow-up

Patient	GCS on seventh postoperative day	Length of follow-up (months)	BI
1	15	60	100
2	15	58	100
3	13	56	75
4	14	55	75
5	7T	52	45
6	14	49	50
7	15	39	90
8	15	36	75
9	12	33	75
10	10T	18	50
11	15	16	90

GCS, Glasgow Coma Scale score; BI, Barthel Index score; T, intubated.



FIGURE 1. Computed tomogram showing tightness in posterior fossa with absence of fourth ventricle in case 8 on first day of admission.

requires some assistance with daily activities. A score <60 implies that a patient is functionally dependent (Tables 2 and 3). Patients were also regularly followed up in other departments for underlying diseases.

Results

Preoperative GCS scores and GCS scores 1 week after surgery are listed in Tables 1 and 2. Seven patients improved on the first postoperative day. Ten patients demonstrated neurological improvement on the seventh postoperative day, and all 11 patients survived the perioperative period. The BI score evaluation was performed 16–60 months after surgery. Two patients (cases 1 and 2) returned to previous jobs 3 months after surgery. Six patients (cases 3, 4, 7, 8, 9, and 11) were functioning at a level of assisted independence. The remaining three patients required assistance from family members to accomplish their activities of daily living. Finally, subjective assessment of surgical procedure and its outcome by the family was positive for all victims except case 5.

Of the 11 patients who underwent suboccipital craniectomy, one (case 8) was a 50-year-old woman who had had rheumatic mitral stenosis for >10 years. She suffered from a sudden onset of vertigo, nausea, and vomiting the day before admission. At the time of admission she was lethargic, with a GCS score of 13. Dysarthria, nystagmus, and a positive Babinski sign were noted. Electrocardiography showed atrial fibrillation. Admission head CT demonstrated tightness in the posterior fossa (Figure 1). She was observed in the neurological intensive care unit and treated with supportive medical modalities. Her consciousness remained clear for 2 days but deteriorated with obtundation. After intubation she was hyperventilated, and an emergency head CT was performed (Figure 2). This demon-



FIGURE 2. Computed tomogram showing low attenuation and mass effect in distribution of posterior inferior cerebellar artery in case 8 on third day of admission.

strated a definite infarction in the left posterior inferior cerebellar artery territory with marked hydrocephalus. Because there was no improvement after medical treatment, a left suboccipital craniectomy with ventricular drainage was performed. Immediately after surgery she was extubated but remained on intraventricular pressure monitoring. The ventricular draining catheter was removed on the third postoperative day. She made marked improvement and was discharged 10 days after craniectomy. Two months after surgery, she could walk smoothly with the help of a cane.

Another patient (case 9) was a 50-year-old man who suffered from bilateral ureteral stones and hydronephrosis. He also had poorly controlled hypertension for >1 year. The day before admission he presented with headache, repeated vomiting, and drowsiness. Physical examination demonstrated a somnolent but arousable consciousness. The GCS score was 13 with a blood pressure of 180/100 mm Hg. Head CT revealed relative tightness in the posterior fossa. In the early morning of the second admission day, the GCS score dropped to 8. He was intubated, hyperventilated, and placed on a mannitol intravenous infusion in the intensive care unit. A follow-up head CT revealed cerebellar infarction in the bilateral posterior inferior cerebellar artery territory (Figure 3). The GCS score was only 4 with pinpoint pupils just before emergency suboccipital craniectomy. His neurological condition gradually improved, with a GCS score of 12 on the fifth postoperative day. The patient could walk with support 2 weeks after surgery. Three months after decompressive craniectomy the patient underwent ureterolithotomy.

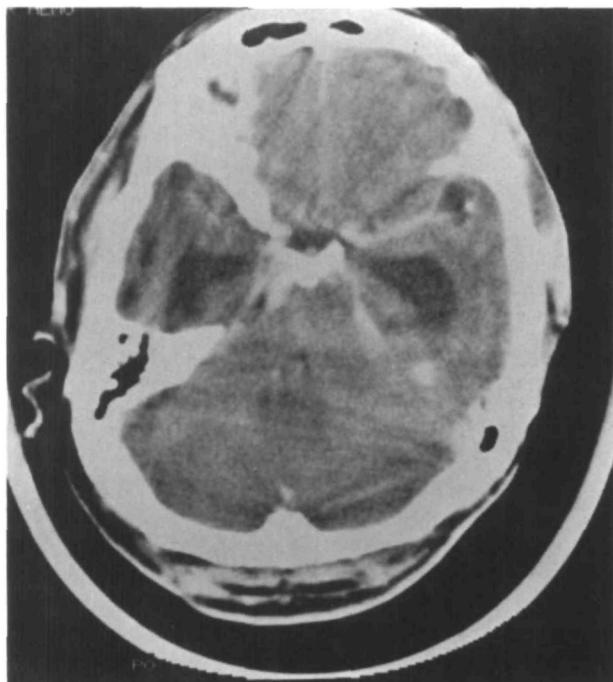


FIGURE 3. Computed tomogram showing low attenuation in bilateral cerebellar hemispheres with compression of fourth ventricle and hydrocephalus in case 9 on second day of admission.

Discussion

Cerebellar infarction, like cerebellar hemorrhage, is a neurological emergency. The clinical pictures of cerebellar infarction have been studied in detail.^{1-3,5,6} In addition to cerebrovascular disease, the etiology of cerebellar infarction includes dehydration, polycythemia, diabetes mellitus, rheumatic heart disease with atrial fibrillation, and vertebral artery dissection, either occurring spontaneously or as a result of chiropractic manipulation or neck injuries.^{1,7,8} Vertebral artery dissection may be one of the most common and important causes of cerebellar infarction.² There were no trauma histories in this series.

The brain swelling following infarction results from cytotoxic and vasogenic edema.⁹ Severe brain ischemia initially produces cytotoxic edema without apparent disruption of the blood-brain barrier. Early ischemia disturbs the regulatory mechanism within the cell membrane and results in accumulation of intracellular fluid. During this acute period, the infarction is not visible on CT. However, as a result of the infarction process, protein-bound fluid is able to diffuse across a damaged blood-brain barrier and produce vasogenic edema.⁹

The most common symptom at onset is an inability to stand or walk. Vomiting, dizziness, and headache are also very common. The pupils are generally small but reactive. Although conjugate deviation to the contralateral side with nystagmus is not as common, at times diplopia with more severe weakness of the abducting eye, a frank ipsilateral sixth nerve paralysis, subtle facial palsy, and dysarthria are present at onset. Papilledema has also been noted, although rarely.^{1,2}

Large cerebellar infarcts produce a progressive syndrome by gradually enlarging to produce mass effect.

Once significant swelling develops a vicious cycle begins. The swollen cerebellar hemisphere compresses the fourth ventricle, leading to hydrocephalus. The lower brain stem tegmentum is compressed directly, and the cerebellar tonsils are forced into the foramen magnum, thus sealing the inferior outlet of the posterior fossa. In addition, upward transtentorial herniation can develop, with distortion of the midbrain aqueduct and buckling of the quadrigeminal plate.^{1,6,10} Pressure on the midbrain or oculomotor nerve is responsible for the ipsilateral dilated pupil late in the course of cerebellar infarction. Many patients with cerebellar infarction exhibit these symptoms and signs, which are identical to those of cerebellar hemorrhage. If untreated the patients will become comatose, and finally apnea from medullary compression will occur.^{1,2,6} Patients may rapidly lapse into a moribund state. The clinical symptoms and signs in other patients with cerebellar infarction demonstrate coexistent lateral medullary infarction or intrinsic brain stem deficits. The management of these patients is different. In some cases it may be difficult to clinically separate pressure signs produced by a swollen cerebellum from more extensive brain stem ischemia, in which case arteriography could better define the extent of the vascular lesion.^{3,5,11}

Surgical decompression has previously been shown effective in selected cases of cerebellar infarction.^{2,3,5} Although this is a small anecdotal series, decompressive suboccipital craniectomy appears to be an effective, lifesaving method of treating cerebellar edema secondary to stroke. The surgical decompressive procedure should be considered an additional mode of therapy when contemporary medical therapy fails to curtail edema in stroke victims. All patients treated by decompressive suboccipital craniectomy survived. Patients demonstrating neurological decline while on medical therapy may derive more benefit from surgical decompression.

We favor decompressive suboccipital craniectomy with temporary ventriculostomy instead of only simple ventriculostomy for several reasons. The potential complications of simple ventriculostomy include upward herniation of the cerebellum through the tentorial notch with compression of the brain stem.¹² Also, in our experience resolution of the infarction will not be complete for about 6 days without decompressive craniectomy.⁸ If the drainage catheters stay in place for >6 days the chance of infection will increase,¹³ and patients will need to undergo another risky surgery if a permanent shunting procedure is necessary. However, the temporary ventricular draining catheter can usually be removed within 72 hours after decompressive craniectomy.

The physicians' objectives in the acute management of stroke patients include the preservation of life, prevention of extending cerebellar injury, and avoidance of systemic complications. The patient's age, family support, and potential neurological outcome must also be considered before aggressive management is undertaken.¹⁴ Suboccipital craniectomy for cerebellar infarction with marked brain edema may preserve life and prevent extension of brain stem injury. Although surgical decompression is an aggressive procedure, most patients can recover with a relatively good quality of life. The variability in outcome is dependent on the

location and extent of the infarction, the age of the patient, and possibly the timing of surgery. Younger stroke victims with a supportive family and a potential for neurological recovery are more likely to have a favorable outcome. Therefore, when medical modalities fail to curtail brain edema in a young patient, emergency suboccipital craniectomy should be considered.

In conclusion, 11 patients with cerebellar infarction who faced imminent death from brain swelling underwent decompressive suboccipital craniectomy, and all improved. The neurological functional outcome was considered reasonable by most patients and families. This surgical procedure can be a lifesaving treatment for malignant brain swelling after cerebellar infarction.

Acknowledgments

The authors are greatly indebted to Dr. Robert F. Heimburger for the subject matter and Miss Judith L. Perry for criticism of the English usage.

References

1. Heros RC: Cerebellar hemorrhage and infarction. *Stroke* 1982;13:106-109
2. Sypert GW, Alvord E: Cerebellar infarction, a clinicopathological study. *Arch Neurol* 1975;32:357-363
3. Lehrich JR, Winkler GF, Ojemann RG: Cerebellar infarction with brainstem compression. *Arch Neurol* 1970;22:490-498
4. Mahoney FI, Barthel DW: Functional evaluation: The Barthel index. *Md Med J* 1965;14:61-65
5. Duncan GW, Parker SW, Fisher M: Acute cerebellar infarction in the PICA territory. *Arch Neurol* 1975;32:364-368
6. Feely MP: Cerebellar infarction. *Neurosurgery* 1979;4:7-10
7. Delangre T, Mihout B, Borh JY, Samson M: Primary thrombocythemia in a patient with cerebellar infarction. *Stroke* 1985;16:524-526
8. Rousseaux M, Devos P, Lesoin F, Petit H: "Pseudotumoral" cystic cerebellar infarction with slow evolution. *Neurosurgery* 1985;16:61-63
9. Katzman R, Clasen R, Klatzo I, Meyer JS, Pappius HM, Waltz AG: Brain edema in stroke: Study group on brain edema in stroke. *Stroke* 1977;8:512-540
10. Tomaszek DE, Rosner MJ: Cerebellar infarction: An analysis of twenty-one cases. *Surg Neurol* 1985;24:223-226
11. Scotti G, Spinnler H, Sterzi R, Vallar G: Cerebellar softening. *Ann Neurol* 1980;8:133-140
12. Cunes RA, Caronna TJ, Winestock PP: Upward transtentorial herniation. *Arch Neurol* 1979;36:618-623
13. Rosner MJ, Becker DP: ICP monitoring: Complications and associated factors. *Clin Neurosurg* 1976;23:494-519
14. Kondziolka D, Fazl M: Functional recovery after decompressive craniectomy for cerebral infarction. *Neurosurgery* 1988;23:143-147

Stroke

JOURNAL OF THE AMERICAN HEART ASSOCIATION



American Heart Association | American Stroke Association®

Treatment of cerebellar infarction by decompressive suboccipital craniectomy.

H J Chen, T C Lee and C P Wei

Stroke. 1992;23:957-961

doi: 10.1161/01.STR.23.7.957

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Copyright © 1992 American Heart Association, Inc. All rights reserved.

Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:

<http://stroke.ahajournals.org/content/23/7/957>

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in *Stroke* can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the [Permissions and Rights Question and Answer](#) document.

Reprints: Information about reprints can be found online at:
<http://www.lww.com/reprints>

Subscriptions: Information about subscribing to *Stroke* is online at:
<http://stroke.ahajournals.org//subscriptions/>