

Posterior fossa decompression in acute cerebellar infarction

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Abstract

Objective: There are differences in opinion regarding the optimal treatment for patients with cerebellar infarction. The aim of this study was to determine which patients requiring surgical treatment and which surgical procedure should be preferred when a patient with cerebellar infarction exhibits progressive neurological deterioration.

Patients and methods: A total of 11 patients (7 males and 4 females) were treated surgically at our institution for cerebellar infarction in the past 3 years, of these patients 5 underwent posterior fossa decompression with removal of necrotic tissue as the first treatment and 3 patients underwent external ventricular drainage as the first treatment plus secondary suboccipital craniectomy. In the other 3 patients, ventriculoperitoneal (VP) shunt alone was done.

Results: Of the 11 patients, 7 patients experienced good functional outcome, 2 experienced moderate outcome and 2 patients died. Of the 7 patients who experienced good outcome, 4 patients were treated with suboccipital decompression only, 2 with VP shunt and one with external ventricular drainage followed by decompression.

Conclusion: Cerebellar infarction constitutes an emergency neurosurgical procedure. Decompressive surgery should be the treatment of choice for massive cerebellar infarction causing progressive brainstem compression signs or impairment of consciousness. (p50-54)

Key words: Acute cerebellar infarction, brainstem, suboccipital decompression, and external ventricular drainage

Introduction

Cerebellar infarctions (CIs) without brainstem or cerebral involvement are rare. They constitute 1.5 - 8.1% of cases in clinicopathological series.^{2,3,7,9,12,17,18} Cerebellar infarctions are often fatal. The main determinant of outcome among patients with CIs is the extent of infarction, however, other factors such as haemorrhagic transformation and reflux of blood into damaged vessels may be involved.^{1,9}

Oedematous swelling resulting from ischemia is usually responsible for the neurological deterioration which may be delayed for several days after the onset of CIs.⁵ The choice of treatment for patients with CIs is still controversial. Opinions differ mainly with respect to the best option if

conservative therapy fails.

In this study, we review data for a series of 11 patients who were treated at our institution in the past 3 years. Particular attention was given to the clinical and radiological findings which are important in determining the optimum therapy and in deciding the indication for suboccipital decompressive surgery.

Patients and methods

In the last 3 years 11 patients with cerebellar infarction were presented to our department. There were 7 male and 4 female patients. The patients' age ranged from (29 - 75 years, median 55 years).

The typical presenting symptoms were headache, vertigo, nausea, and vomiting. Cerebellar signs were noted in the 11 patients, 4 patients had potentially embolic diseases (Table 1) and 7 patients were hypertensive. All patients underwent Glasgow Coma Scale (GCS) assessment at the time of admission (Table 2). All patients underwent brain computed tomography (CT) evaluation and 5 patients underwent magnetic resonance imaging (MRI) and magnetic resonance angiography. The cerebellar infarction was attributed to occlusion of the posterior inferior cerebellar artery (PICA)

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in 3 patients, anterior inferior cerebellar artery (AICA) in 2 patients and massive infarction in 6 patients. Neurological assessments with GCS were repeated in the subsequent days, deterioration was considered a decrease in GCS score of 2 points. Fourth ventricle displacement of 3 mm and brainstem deformities were radiological criteria for early suboccipital decompression. A tight posterior fossa was defined according to the criteria proposed by Weisberg as a lack of observation of the basal cisterns in the posterior fossa, an increase in the size of the 3rd ventricle and lateral ventricles and lack of observation of the 4th ventricle.¹⁹ For these patients with tight posterior fossa we preferred to perform surgical decompression.

Four patients with rapidly worsening consciousness level and radiologically evident acute hydrocephalus without transtentorial herniation, underwent external ventricular drainage for 24 - 72 hrs. In 3 of them suboccipital decompression was done due to progression of brainstem compression, in

the other patient ventriculoperitoneal (VP) shunt was done after closure of the external drainage. Five patients with clinical and radiological signs of brainstem compression from the onset of cerebellar infarction and one patient with tight posterior cranial fossa underwent craniectomy, with removal of necrotic tissue as the first treatment and in 4 of them duraplasty with dural patches was done. The other 2 patients whose consciousness remained intact but with radiological evidence of hydrocephalus received VP shunt only. Table 3 shows the summary of the 11 patients.

Results

Results and treatment in the current series are shown in Table 4. Outcomes at the time of discharge from the hospital were classified according to Glasgow Outcome Scale (GOS): i.e., good recovery, moderate disability (disabled but independent), severe disability (conscious but disabled), persistent vegetative state (unconscious, unable to follow commands) or death.¹⁰

Table 1 - Potentially embolic diseases among patients in the present series

Potentially embolic diseases	No. of patients
Cardiac infarction	2
Atrial flutter	1
Recent delivery	1
Total	4

Table 2 - GCS score at the time of admission in the present series

GCS score	No. of patients
6	2
9-12	4
13	3
14	2
Total	11

Table 3 - Summary of the 11 patients

No.	Sex	Age/yr	GCSs	Timing of surgery	CT or MRI	Procedures	Results
1	M	64	13	3 rd day	Infarction on PICA distribution with 4 th ventricular effacement	-SDC -removal of the necrotic tissue -duraplasty using dural patch	Good recovery
2	F	61	14	6 th day	Infarction on PICA distribution with 4 th ventricular effacement	-SDC -removal of the necrotic tissue	Good recovery
3	M	70	6	1 st day EVD and SDC on 3 rd day	Massive cerebellar infarction, hydrocephalus	-EVD -SDC	Died on 10 th postop day due to respiratory problems
4	F	29	9	1 st day	Infarction on PICA distribution	-SDC	Moderate disability
5	F	55	11	3 rd day EVD and SDC, then VP shunt on 10 th day	Massive cerebellar infarction, hydrocephalus	-EVD -SDC and removal of necrotic tissue & duraplasty -VP shunt	Good recovery
6	M	60	12	EVD on 1 st day and SDC on 3 rd day	Infarction on AICA distribution with hydrocephalus	-EVD -SDC	Good recovery
7	M	58	13	4 th day	Infarction on AICA distribution with hydrocephalus	-VP shunt	Good recovery
8	M	75	14	1 st day	Massive cerebellar infarction, hydrocephalus	-VP shunt	Good recovery
9	F	60	6	3 rd day	Massive cerebellar infarction	-SDC and removal of necrotic tissue & duraplasty	Died due to pulmonary embolism
10	M	63	13	2 nd day	Massive cerebellar infarction	-SDC and removal of necrotic tissue & duraplasty	Good recovery
11	M	67	10	EVD 2 nd day and VP shunt on 7 th day	Massive cerebellar infarction, hydrocephalus	-EVD -VP shunt	Moderate disability

Abbreviations: SDC = suboccipital decompressive craniectomy, EVD = external ventricular drainage, VP shunt = ventriculoperitoneal shunt, PICA = posterior inferior cerebellar artery, AICA = anterior inferior cerebellar artery

Table 4 - Treatment and outcomes according to GOS

Treatment	No. of patients			
	Total	GR	MD	Death
SDC	5	4	-	1
EVD+SDC	3	1	1	1
Shunt	3	2	1	-
Total	11	7	2	2

Abbreviation: SDC = suboccipital decompressive craniectomy, EVD = external ventricular drainage, GR = good recovery, MD = moderate disability

Seven of our patients experienced good recovery, of them 4 patients were operated by posterior fossa decompression, 2 by VP shunt only and one patient treated by external ventricular drainage followed by suboccipital craniectomy. One patient treated by VP shunt only survived with moderate disability and also one patient treated by external drainage followed by craniectomy, 2 other patients died.



Figure 1a - Preoperative MRI showing right cerebellar infarction with brain stem compression

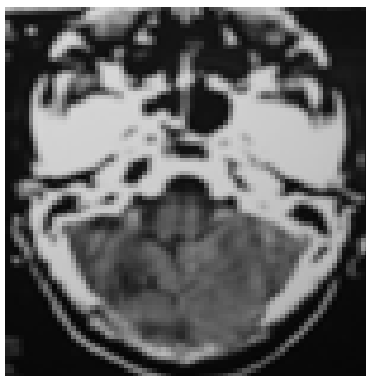


Figure 1b - Postoperative CT after decompression and no brain-stem compression

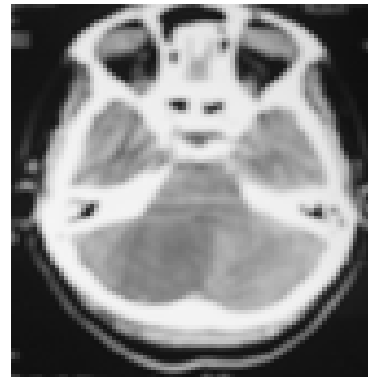


Figure 2a - Preoperative CT showing right cerebellar infarction with brainstem compression and hydrocephalus

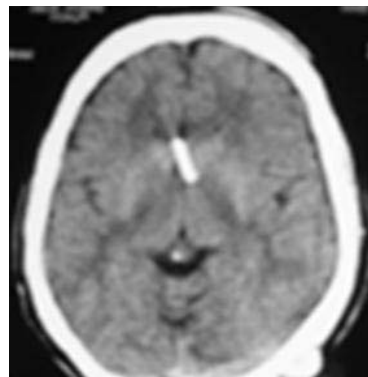
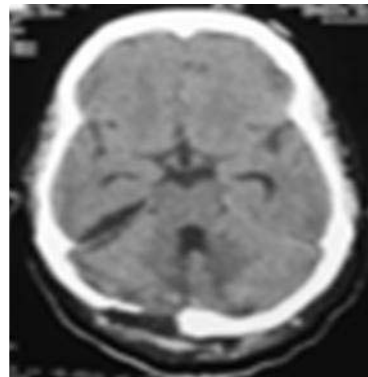


Figure 2b - Postoperative CT after decompression and VP shunt

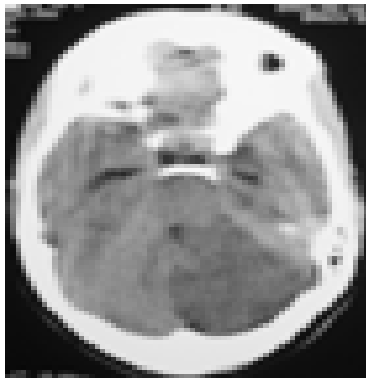


Figure 3a - Preoperative CT showing left cerebellar infarction

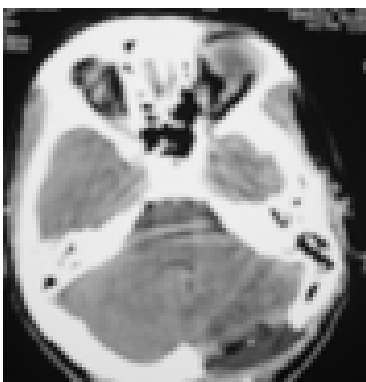


Figure 3b - Postoperative CT after decompression

Discussion

There are differences in opinion regarding the optimal treatment for a patient with cerebellar infarction. Although, conservative therapy is the preferred treatment for patients who are alert and in a clinically stable condition, the optimal treatment for patients with impaired consciousness remains controversial.^{4,6,11}

Surgical interventions (decompressive craniectomy and/or ventricular drainage) may be necessary in patients with cerebellar infarction if mass effect develops. However, patient selection and timing of surgery remain controversial and there is little data on clinical signs in the early course that are predictive of outcome. The precise clinical features that lead to deterioration in cerebellar infarction are not completely understood. Patients with mass effect demonstrated on CT scan commonly deteriorate when obstructive hydrocephalus ensues. A number of radiological features have been shown to be predictive of neurological deterioration, including 4th ventricle distortion and shift, obstructive hydrocephalus, brainstem deformity and basal cistern compression.

Our data suggest that external drainage may be effective in the treatment of CI that becomes life threatening. The rationale for this is that consciousness deterioration is not always caused by secondary brainstem compression; by the swollen cerebellar tissues, it may result from hydrocephalus. In such cases, drainage of hydrocephalus may interrupt the cascade of events that would otherwise increase the subtentorial mass effect.^{8,16} This was observed in 3 of our patients, of whom drainage led to complete recovery of neurological function in 2 and the other one had moderate disability. Raco, et al found the same results in 8 of their patients.¹⁵

A number of authors have described the use of CSF drainage in their treatment if patients present with hydrocephalus and cerebellar infarction. We disagree with authors who suggest that placement of external ventricular drains increase the risk of upward transtentorial herniation, as this can be eliminated by regulating the CSF flow rate and monitoring the patient's neurological status.^{5,8,15} The clinical improvement after drainage must occur within a short period of observation to justify not proceeding to craniectomy. If external drainage fails to improve consciousness, then immediate decompressive craniectomy with removal of necrotic tissue become mandatory. We performed such a procedure in 3 of our patients and 2 of them survived.

In this series, 5 patients had evidence of brainstem compression and/or tight posterior fossa on admission. In these patients posterior fossa decompression with removal of necrotic tissues was done immediately without delay, 4 of these patients had a good outcome, this was the same as reported by other authors.^{13,14,17}

Therefore, early diagnosis and prompt surgical decompression are mandatory before the development of irreversible brainstem damage or severe hypertensive hydrocephalus.

We concur with other authors that evidence of tight posterior fossa justifies immediate surgical decompression in the absence of brainstem compression.^{13,16} Conversely, if deep coma has been present from the onset and further severe deterioration occurs during transfer, in the absence of hydrocephalus, then treatment limiting decision becomes justified.

Conclusion

The debate regarding the optimal treatment for patients with cerebellar infarction can not be completely solved until a prospective study eliminating selection bias for comparison of the surgical outcome after external ventricular drainage and decompressive craniectomy.

Our small series demonstrates that external ventricular

drainage can rapidly solve brainstem compression and improve patient's neurological status. Patients with brainstem compression from the beginning and/or with tight posterior fossa may benefit from decompressive craniectomy.

Moreover, the successful treatment of a patient with cerebellar infarction depends on awareness that clinical deterioration may occur some days after infarction onset, early diagnosis and immediate initiation of treatment.

Urgent surgical decompression of cerebellar infarction can be a life-saving and rewarding surgical procedure in well-selected patients.

References

1. Amarenco P: The spectrum of cerebellar infarctions. *Neurol* 1991, 41: 973-979
2. Amarenco P, Hauw JJ, Gautier JC: Arterial pathology in cerebellar infarction. *Stroke* 1990, 21: 1299-1305
3. Amarenco P, Hauw JJ, Henin D, et al: Cerebellar infarction in the area of the posterior cerebellar artery: Clinicopathology of 28 cases (French). *Rev Neurol (Paris)* 1989, 145: 277-286
4. Caplan LR: Vertebrobasilar disease: Time for a new strategy. *Stroke* 1981, 12: 111-114
5. Chen HJ, Lee TC, Wei CP: Treatment of cerebellar infarction by decompressive suboccipital craniectomy. *Stroke* 1992, 23: 957-961
6. Duncan GW, Parker SW, Fisher CM: Acute cerebellar infarction in the PICA territory. *Arch Neurol* 1978, 78: 129-140
7. Hinshaw DB, Thompson JR, Hasso AN, Casselman ES: Infarctions of the brainstem and cerebellum: A correlation of computed tomography and angiography. *Radiol* 1980, 137: 105-112
8. Horing CR, Rust DS, Busse O, Jauss M, Laun A: Space-occupying cerebellar infarction: Clinical course and prognosis. *Stroke* 1994, 25: 372-374
9. 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: 257-264
10. Jennett B, Bond M: Assessment of outcome after severe brain damage: A practical scale. *Lancet* 1975, 1: 480-484
11. Jones HR, Millikan CH, Sandok BA: Temporal profile (clinical course) of acute verobrobasilar system cerebellar infarction. *Stroke* 1980, 11: 173-177
12. Krieger D, Busse O, Schramm J, Ferbert A: German-Austrian space occupying cerebellar infarction study (GSCIS): Study design, methods, patient characteristics. *J Neurol* 1992, 239: 183-185
13. Madonnel RAL, Kalanis RM, Donna GA: Cerebellar infarction: Natural history, prognosis and pathology. *Stroke* 1987, 18: 849-855
14. Mathew P, Teasdale G, Bannan A: Neurosurgical management of cerebellar hematoma and infarct. *J Neurol Neurosurg and Psychiatry* 1995, 59: 287-292
15. Raco Antonino, Caroli Emanuela, Isidori Alessandra: Management of acute cerebellar infarction: one institution's experience. *Neurosurg* 2003, 53: 1061-1066
16. Rousseaux M, Devos P, Lesion F, Petit H: "Pseudotumoral" cystic cerebellar infarction with slow evolution. *Neurosurg* 1985, 16: 61-63
17. Sypert GW, Alvord EC Jr: Cerebellar infarction. A clinicopathological study. *Arch Neurol* 1975, 32: 357-363
18. Thogi H, Takahashi S, Chiba K, Hirata Y: Cerebellar infarction: clinical and neuroimaging analysis in 293 patients - Tohoku cerebellum infarction study group. *Stroke* 1993, 24: 1697-1701
19. Weisberg LA: Acute cerebellar hemorrhage and CT evidence of tight posterior fossa. *Neurol* 1986, 36: 856-860

GENTLE REMINDER

Distribution of cavernous and venous angiomas

Location	Cavernous angioma (%)	Venous angioma (%)
Frontal	28	42
Parietal	7	24
Temporal	17	2
Occipital	4	4
Basal ganglia/thalamus	7	11
Corpus callosum	0.6	
Brain stem/cerebellum	6	3