CLINICAL ARTICLE

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Surgical Management of Acute Cerebellar Infarction

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Objective : The aim of this study is to determine which patients with progressively deteriorating acute cerebellar infarction would benefit from surgical treatment and which surgical procedure would best benefit them.

Methods : Seventy six patients were treated at our hospital for cerebellar infarction over the past 3 years. Sixty nine patients received conservative management in the neurological department of our hospital. Among them, 7 patients (5 males and 2 females; average age, 49 yrs) were referred to neurosurgical department because of mental deterioration and underwent emergency surgery. Five patients underwent external ventricular drainage with suboccipital craniectomy and two patients underwent suboccipital craniectomy alone.

Results : Of the 7 surgically treated patients, 4 patients experienced good recovery and 2 patients experienced moderate disability (disabled but independent) and 1 patient experienced severe disability (conscious but disabled). There was no death.

Conclusion : In patients conservatively treated for cerebellar infarction and showing mental deterioration and radiologically evident brainstem compression and ventricular enlargement, we strongly recommend suboccipital craniectomy (plus optional external ventricular drainage in case of showing hydrocephalus) as a first treatment option.

KEY WORDS : Cerebellar infarction · Suboccipital craniectomy · External ventricular drainage.

Introduction

Stroke is the most prevalent disease involving the central nervous system[3]. Cerebellar infarcts constitute 1.5~4.2% of cases in clinicopathological series and 1.9~10.5% of those in clinical series of patients with cerebral infarctions[6,9]. Many patients are in a responsive state early after stroke and deteriorate secondarily after various periods of time (24~168 hours; median time, 72 hours)[3].

Clinical features and the time profile of neurological symptoms in cerebellar stroke have been subdivided into a useful three-stage scale by Heros[1,5,8]. The early stage is of pure cerebellar symptoms, such as vertigo, nausea and vomiting, unsteadiness of gait and clinical signs including ataxia of gait and trunk, nystagmus, dysmetria of the limbs and dysarthria. The intermediate stage is of normal consciousness and includes focal signs of brainstem displacement, such as sixth, seventh nerve palsy, gaze paresis, forced gaze deviation. Late stage patients are stuporous or comatose and demonstrate signs of global brainstem disturbance including posturing, cardiovascular instability, ataxic breathing pattern and pinpoint pupils.

This neurological deterioration occurs as a result of mass effect of swollen brain tissue in the posterior fossa compressing the brainstem, with associated obstructive hydrocephalus. Aforementioned mechanisms of clinical deterioration in cerebellar infarcts are recognized and are addressed in current therapeutic guidelines[5,6,8]. Although there is good evidence that surgery is required to salvage the majority of patients with imminent infratentorial herniation, patient selection criteria, type of surgery (i.e., suboccipital craniectomy, external ventricular drainage), and timing of the procedures are widely disputed[6].

We report our experience with the management of 7 patients with massive cerebellar infarcts during past 3 years.

Materials and Methods

From December 2001 to July 2004, seventy six cerebellar infarction patients received platelet-inhibitory medication (aspirin) initially. Among them, sixty nine patients whose consciousness remained intact received conservative therapy with mannitol and aspirin. But seven cerebellar infarction patients (5 males and 2 females; average age, 49 yrs) exhibited mental deterioration during the course of medical therapy. Computed tomography imaging revealed progressively enlarging infarction

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Table 1. Summary of clinical cases

Case No	Sex/age	GCS score		Interval between onset and preop	EVD timing	CT finding		GOS
		Onset	Preop			Brainstem compression	Hydroce phalus	
1	F/48	15	6	1 day	intraop	−	+	3
2	M/61	9	8	1 day	intraop	+	+	4
3	M/32	13	7	1 day	no	+	−	4
4	M/61	15	8	2 days	intraop	+	+	5
5	M/40	14	8	1 day	no	+	−	5
6	M/36	15	8	2 days	intraop	−	+	5
7	F/68	15	8	6 days	no	+	−	5

Abbreviation : GCS(Glasgow coma scale), EVD(External ventricular drainage), GOS(Glasgow outcome scale), CT(Computed Tomography), M(male), F(female)

Table 2. The treatment and patients number of cerebellar infarction in other literature

	Total patients	Conservative management	Surgical intervention		
			SC	EVD	SC after EVD
Hornig CR[5]	52	12	30	10	4(40%)
Jauss M[6]	84	36	34	14	4(29%)
Raco A[9]	42	25	4	13	5(38%)
Taneda M[10]	15	5	7	3	2(67%)

Abbreviation : SC(suboccipital craniectomy), EVD(external ventricular drainage)

with fourth ventricle displacement and brainstem compression or obstructive hydrocephalus. The range of time that elapsed between the onset of symptoms and secondary neurological deterioration was between 1~6 days (median time, 2 days). All patients were thought to be responding poorly to aggressive medical treatment. These patients were treated with suboccipital craniectomy with or without temporary external ventricular drainage. The patients were closely monitored by repeated neurological and CT examinations in order to evaluate the efficacy of the treatment (Table 1).

Results

We evaluated outcome at the time of discharge according to the Glasgow Outcome Scale, i.e., 5 : good recovery, 4 : moderate disability (disabled but independent), 3 : severe disability (conscious but disabled), 2 : persistent vegetative state (unconscious, unable to follow commands), 1 : death. 4 patients with hydrocephalus underwent suboccipital craniectomy with duroplasty and intraoperative external ventricular drainage. 3 patients without hydrocephalus underwent suboccipital craniectomy with duroplasty alone. All patients underwent surgical intervention immediately when there was a deterioration of neurological status and brain CT showed brainstem compression or obstructive hydrocephalus.

Of the 7 patients, 4 patients showed good recovery and 2 patients exhibited moderate disability but 1 patient experienced severe disability. The overall mortality rate was 0%.

The poor prognosis with the patient showing Glasgow outcome scale 3 was probably due to concomitant severe pulmonary complications (pneumonia and pulmonary congestion), complications of prolonged bedridden state (sacral sore, rigidity, etc), and uncontrolled diabetes mellitus after surgery.

Illustration case (Case 6)

A thirty six year old man previously in good health, presented to the emergency room with complaints of vertigo. He was alert and had no definite neurologic deficit at that time. As the headache became more pronounced, a CT scan and MRI were done (Fig. 1, 2). These showed an extensive infarction of the left cerebellar hemisphere. But the 4th ventricle was not compressed and the obvious hydrocephalus was not seen. Consequently, he was managed conservatively.

At about 48 hours after symptom onset he became stuporous and started to exhibit irregular breathing. The CT scan performed at the time of mental deterioration showed a more extensive low-density area in the left cerebellar hemisphere, and obstructive hydrocephalus(Fig. 3). Emergency suboccipital craniectomy and external ventricular drainage was performed. He gradually recovered and was alert and oriented. Though there was cerebellar ataxia, he was ambulant with minimal su-

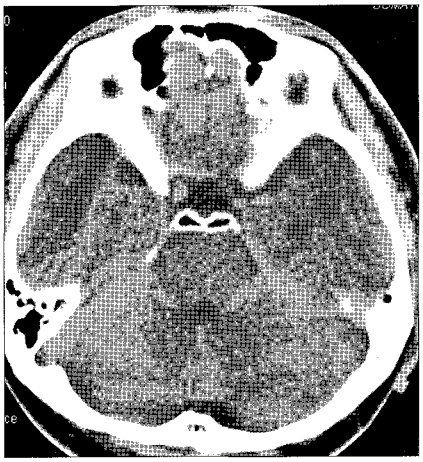

Fig. 1. Computed tomography showing an extensive infarction of the left cerebellar hemisphere.

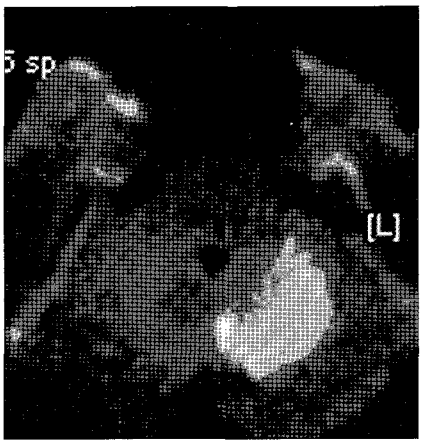

Fig. 2. Diffusion weighted axial magnetic resonance image showing an extensive infarction of the left cerebellar hemisphere.

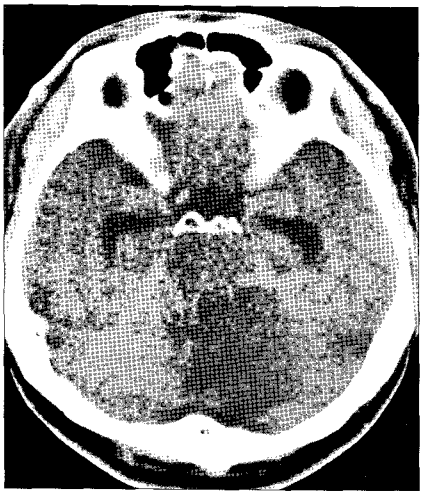

Fig. 3. (Computed tomography) : A more extensive low-density area in the left cerebellar hemisphere, and obstructive hydrocephalus with brainstem compression.

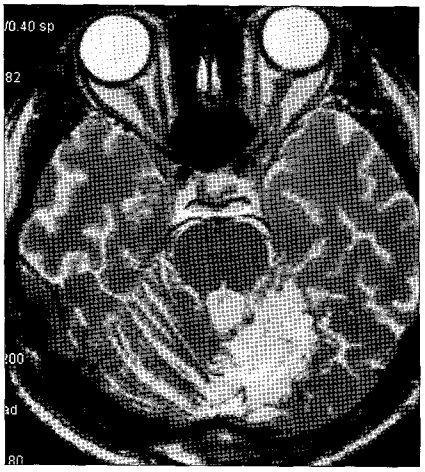

Fig. 4. (T2-weighted axial magnetic resonance image) : High signal change of the left cerebellar hemisphere due to old infartion without obstructive hydrocephalus or brainstem compression.

pport. At about 3 months after operation a follow-up MRI was performed (Fig. 4). This showed no obstructive hydrocephalus or brainstem compression.

Discussion

The brain tissue affected by ischemic necrosis usually undergoes edematous swelling. Sometimes, reflow of blood into damaged vessels following fragmentation and movement of the embolus or via collateral circulation may accelerate the enlargement of the mass lesion by promoting tissue swelling and/or hemorrhage in the infarcted area. Such a mass lesion in the cerebellar hemisphere directly compresses the brainstem, and the speed of its enlargement affects the rapidity of clinical deterioration[10]. In untreated cases, particularly in patients with a rapid alteration in the level of consciousness, mortality rates may reach 80 to 92%. Immediate and adequate treatment may drastically improve the prognosis of these patients[2].

There are differences of opinion regarding the optimal treatment for patients with cerebellar infarctions. Although there is general agreement that conservative therapy is the preferred treatment for patients who are alert and in clinically stable condition, the optimal treatment for patients that fail to respond to conservative therapy remains controversial. Surgical techniques range from craniectomy and necrotic tissue removal to external ventricular drainage[7]. For patients who became critically ill, some authors described successful outcomes after surgical resection of the necrotic tissue, whereas others reported good results with external ventricular drainage[9].

In an analysis of the pertinent literature, we identified that a large number of patients with primary external ventricular drainage underwent subsequent suboccipital craniectomy because of further deterioration (Table 2).

We favor decompressive suboccipital craniectomy plus temporary ventriculostomy instead of simple ventriculostomy alone for several reasons. The potential complications of simple ventriculostomy include upward herniation of the cerebellum through the tentorial hiatus. Also if posterior fossa mass effect does not resolve within 6 days, the ventricular drain must be changed or internalized to reduce the risk of infection[3,6].

We agree that external ventricular drainage is less invasive than suboccipital craniectomy. However, in cases where patients who receive primary external ventricular drainage do not show mental improvement, they go on to rapidly deteriorate into a state of confusion and subsequent coma within 3 days. The patients undergoing preventive craniectomy tend to recover better than those undergoing emergency craniectomy after deterioration[9]. Suboccipital craniectomy can avoid the complication of upward herniation by external ventricular drainage alone and may be sufficient to both remove pressure on the brainstem and allow sufficient CSF circulation through the aqueduct and fourth ventricle. Also decompressive craniectomy is a procedure which can be done rapidly and is usually fairly well tolerated[9].

Therefore, we assert that the following patients may benefit from early suboccipital craniectomy; patients exhibiting deterioration of neurological state, those with cranial nerve palsy with signs of brainstem compression, and cases showing obstructive hydrocephalus on follow-up brain CT.

The debate regarding the optimal treatment for patients with cerebellar infarction cannot be completely resolved until a large scaled prospective study eliminates selection bias for comparisons of the surgical outcomes after external ventricular drainage and decompressive craniectomy. However, our small series demonstrates that alert patients without signs of obstructive hydrocephalus or brainstem compression should receive conservative therapy, with close clinical and radiological surveillance, whereas rapidly deteriorating patients should receive aggressive surgical management in the early stage.

Conclusion

Because of the low intrinsic risk of suboccipital craniectomy and a sometimes rapid further deterioration after external ventricular drainage alone, we recommend decompressive craniectomy to be the treatement of choice for patients with acute

cerebellar infarction with ongoing progression of brain-stem signs or impairment of consciousness in the early stage.

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Commentary

Authors reviewed their clinical experiences of surgical management (decompressive craniectomy with or without ventricular drainage) of 7 patients with acute cerebellar swelling due to infarction. Preoperative Glasgow Coma Scales in their 7 patients ranged from 6 to 8. Other patient's characteristics (including age, sex, brain computed tomography finding, etc.) Surgical intervention (ventricular drainage or decompressive craniotomy) may be necessary in patients with cerebellar infarction if mass effect develops. However, patient selection and timing of surgery remain controversial, and there are few data on clinical signs in the early course that are predictive for outcome. Mohsenipour et al.[2] reported that In patient with a GCS<12 a reduction of mortality by 15% was obtained by surgical intervention and the outcome as measured by the GOS was significantly improved. Jauss et al.[1] reported that the level of consciousness is the most powerful predictor of outcome, superior to any other clinical sign and treatment assignment. They could not find surgical treatment for massive cerebellar infarctions to be superior to medical treatment in awake/drowsy or somnolent/stupor patients. In their study, half of all patients deteriorating to coma treated with ventricular drainage or decompressive craniotomy had a meaningful recovery.

Surgical outcome can be influenced by age, localisation and size of the lesion, space-occupying character on computed tomography, the progressive appearance of brainstem dysfunction and reduction of the level of consciousness as measured with the Glasgow Coma Scale. All this factors should be considered prior to surgical intervention of patients with massive cerebellar infarction.

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