

Space-Occupying Cerebellar Infarction

Clinical Course and Prognosis

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Background and Purpose Because the timing and strategy of surgical intervention in massive cerebellar infarction remains controversial, we report our experience with the management of 52 such patients.

Methods Case records, computed tomographic scans, surgical reports, and angiograms of 52 patients with space-occupying cerebellar infarction defined by computed tomographic criteria were reevaluated with regard to clinical course, etiology, therapeutic management, mortality, and functional outcome.

Results In most cases clinical deterioration started on the third day after stroke, and a comatose state was reached within 24 hours. Sixteen patients were treated medically, and 30 by suboccipital craniectomy (22 plus ventriculostomy, 12 plus

tonsillectomy). Ten patients primarily had ventriculostomy, which in 4 patients was supplemented by craniotomy because of continuing deterioration. Twenty-nine patients made a good recovery, 15 remained disabled, and 8 died. Even comatose patients had a 38% chance of a good recovery with decompressive surgery. Age older than 60 years ($P=.0043$) and probably initial brain stem signs ($P=.0816$) and a late clinical stage ($P=.0893$) were linked with a fatal or disabling outcome.

Conclusions Decompressive surgery should be the treatment of choice for massive cerebellar infarction causing progressive brain stem signs or impairment of consciousness. (*Stroke*. 1994;25:372-374.)

Key Words • cerebellar infarction • hydrocephalus • prognosis • surgery

In 11% to 25% of all cerebellar infarcts ischemic edema becomes space occupying within the posterior fossa, causing brain stem compression and obstructive hydrocephalus.¹⁻⁴ Disturbance of consciousness and secondary brain stem signs develop in hours to days. Three stages of clinical deterioration due to the increasing infratentorial mass can be differentiated.⁵ In the early stage the patient shows signs and symptoms attributable to cerebellar dysfunction. In the intermediate stage consciousness is slightly impaired, and signs of brain stem compression occur. In the late stage the patient becomes stuporous or comatose with posturing and cardiovascular/respiratory instability. Mortality is high in this stage, but there is widespread agreement that neurosurgical intervention by external ventricular drainage and/or decompressive craniotomy improves prognosis significantly.⁶ Nevertheless, there is still controversy regarding the timing and strategy of surgery. We therefore report our experience with the management of 52 patients with space-occupying cerebellar infarcts.

Subjects and Methods

This report deals with 52 patients with space-occupying cerebellar infarcts admitted to two neurological departments and one neurosurgical department in a 10-year period. At least one of the following computed tomographic (CT) criteria must be fulfilled for the diagnosis of a massive cerebellar infarct: displacement of the fourth ventricle; obstructive hydrocephalus, documented by progressive dilatation of the

lateral ventricles on follow-up CT; and partial or complete obliteration of the basal cisterns. Patients with coma, severe hemiparesis, or tetraparesis from the onset of stroke due to severe brain stem infarction were not included in this study. Case records, CT scans, surgical reports, and angiograms were reevaluated with regard to clinical course, etiology, therapeutic management, and prognosis. The patients were assigned to three clinical stages according to a widely used classification⁵ considering their worst condition (in the period before surgery if this was undertaken). Functional status at hospital discharge was rated by a modified Rankin scale.⁷ Major stroke was assumed if the functional deficit exceeded 2 points on this scale, ie, the patient was unable to attend to personal affairs without assistance. Angiography of the vertebrobasilar system was performed in 16 cases; 34 patients had a Doppler ultrasound examination only.

Results

Baseline Data

Mean age of the patients was 61.2 ± 10.1 years (range, 38 to 82 years); 34 (65.4%) were male. Arterial hypertension was the most frequent vascular risk factor (33 patients; 63.5%), followed by diabetes (21 patients; 40.4%), and hypercholesterolemia (5 patients; 9.6%). Doppler ultrasound sonography or angiography revealed a unilateral or bilateral vertebral artery stenosis in 10 patients and occlusion in 2 patients. Four of these patients and another 18 with a normal Doppler and angiographic examination had a potential cardiac source of embolism, most by nonrheumatic atrial fibrillation (14 patients) or myocardial infarction (3 patients). Eleven patients (21.2%) presented with additional mild or moderate brain stem symptoms or signs (10 patients with lateral medullary syndrome, 1 patient with hemiparesis). Hydrocephalus was visible on at least one CT scan in 42 patients (80.7%).

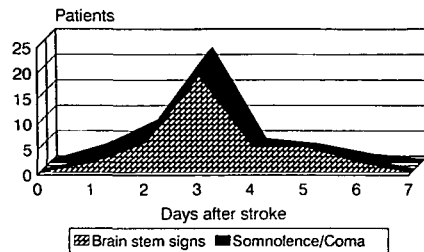
Clinical Course

Thirty-nine patients (75%) developed signs of brain stem compression and 41 (78.8%) a disturbance of

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Graph shows time between cerebellar stroke and first occurrence of signs of brain stem compression or an impairment of consciousness in 52 patients with space-occupying cerebellar infarcts.

consciousness. A total of 19 patients (36.5%) at least became comatose. No clinical deterioration was evident in 7 patients (13.5%) despite an infratentorial mass on CT scan. Signs of brain stem compression as well as impaired consciousness began during the third day after stroke in most cases (range, 1 to 6 days) (Figure). Babinski's sign and horizontal gaze disturbance were the most frequent indicators of a secondary brain stem dysfunction (Table 1). The time interval between the occurrence of first secondary brain stem signs or an impaired consciousness and coma or posturing was less than 24 hours in 14 of 19 patients who at least became comatose (range, 4 hours to 3 days).

Treatment

Thirty-six patients were treated surgically. In 30 cases suboccipital craniectomy was performed with resection of the infarcted cerebellar tissue. This procedure was combined with ventriculostomy in 22 of these patients because of occlusive hydrocephalus, and it was combined with resection of a tonsil because of tonsillar herniation in 12 patients. Ten patients had a ventriculostomy as the first surgical intervention. Six of these 10 patients were comatose and 7 in stage 3 compared with 10 (38.5%) and 16 (61.5%) of the 26 patients who at first had suboccipital craniectomy. In 4 cases ventriculostomy was supplemented by craniotomy after 1 to 3 days because of continuing clinical deterioration. Most patients underwent surgery on the third or fourth day after stroke (25 patients) (range, 1 to 7 days). The effect of surgery in most cases was clinically evident within 1

TABLE 1. Frequent Signs of Brain Stem Compression Due to Space-Occupying Cerebellar Infarction in 39 Patients With Secondary Brain Stem Signs

Sign	Patients	
	No.	%
Gaze deviation	13	33.3
Gaze palsy	8	20.5
Sixth cranial nerve palsy	3	7.7
Unilateral Babinski's sign	5	12.8
Bilateral Babinski's sign	19	48.7
Hemiparesis	11	28.2
Tetraparesis	3	7.7
Posturing	5	12.8
Bilateral miosis	3	7.7
Seventh cranial nerve palsy	2	5.1
Ataxic respiration	10	25.6

TABLE 2. Number of Patients With Space-Occupying Cerebellar Infarction and Nondisabling, Disabling, or Fatal Outcome According to Treatment and Clinical Stage

Outcome	Stage 1 or 2		Stage 3	
	Surgery	No Surgery	Surgery	No Surgery
Independent	9	11	9	0
Disabled	3	2	9	1
Died	1	0	5	2

or 2 days, although swelling on CT could be visible for several days. In 32 patients assessed by the Glasgow Coma Scale, the score improved in 26 cases, deteriorated in 2, and did not change within 48 hours after surgery in 4. Sixteen patients were treated nonsurgically by hyperosmotic agents. Most (13 patients) were in the first or intermediate stage without further clinical deterioration. The remaining 3 patients had been in the third stage for more than 1 day before admission to one of the departments and therefore did not undergo surgery.

Outcome

Twenty-nine patients made a good recovery until hospital discharge and were functionally independent. Fifteen patients still had residual symptoms requiring some help in personal affairs. Eight patients died, 7 of cerebral dysregulation and 1 of pulmonary embolism. The prognosis regarding survival and functional outcome did not differ in surgical and nonsurgical patients in stage 1 or 2. The decision for medical treatment of stage 2 patients depended on the impression of no further deterioration. If the patient had reached stage 3, only surgical intervention gave the patient a chance to survive without a major deficit. Nine of 23 patients in stage 3 who were treated by surgery had no or only minor persisting symptoms, whereas 2 of the 3 patients in stage 3 without surgery died, and 1 remained disabled (Table 2). Furthermore, 13 of 16 patients comatose before suboccipital craniectomy survived, 6 without major persisting symptoms. Two of the 6 patients with ventriculostomy only (3 each in stages 2 and 3) died, and 4 were discharged with a minor deficit. Four of the 30 patients with a suboccipital craniectomy died, 12 were disabled, and 14 made a good functional recovery. The following parameters were analyzed for their influence on mortality and functional outcome of patients treated by surgery: initial presence of brain stem signs, coma, posturing, pyramidal tract signs, age, mode of surgery, and clinical stage. Age older than 60 years (odds ratio [OR], 10; 95% confidence interval [CI], 2.06 to 48.57; $P=.0043$) was significantly linked with a fatal or disabling outcome. A tendency for fatal or major stroke was recognizable in patients with initial brain stem signs (OR, 7.5; 95% CI, 0.78 to 72.48; $P=.0816$) and those in stage 3 at the time of surgery (OR, 3.5; 95% CI, 0.82 to 14.85; $P=.0893$).

Discussion

Since the initial reports by Fairburn and Oliver⁸ and by Lindgren⁹ in 1956 regarding the successful treatment of progressively deteriorating patients with space-occupying cerebellar infarction by decompressive craniectomy, numerous anecdotal reports provided convincing evidence

that neurosurgical intervention can significantly reduce mortality due to massive cerebellar infarction.^{2-4,10-15} Nevertheless, the timing of surgery and the choice of procedure (ventriculostomy, suboccipital craniectomy, or both) are still controversial. Although a multicentered, open, prospective trial regarding the management of space-occupying cerebellar infarction was recently started in Austria and Germany,¹⁶ our experience with prognostic factors in 52 patients with massive cerebellar stroke may provide some hints for therapeutic decisions now.

Our data suggest that decompressive surgery is the only real chance for a patient with a space-occupying cerebellar infarct to survive without a disabling neurological deficit, if the patient has deteriorated to the late stage and become stuporous or comatose. On the other hand, we did not find a significant difference in mortality and functional outcome between surgically and nonsurgically treated patients in the early or even intermediate clinical stages. However, it must be considered that nearly all of our patients in stage 2 treated only medically remained clinically stable for some time. If further deterioration was noted in the intermediate stage, surgery was performed. Furthermore, in most patients the time interval between the first occurrence of secondary brain stem signs or an impairment of consciousness and a comatose state is less than 24 hours, so that time for intervention is often restricted. The functional prognosis seems to be worse for patients operated on in the late stage. Therefore, it has become our policy to perform decompressive surgery in the early intermediate stage, if the patient shows further deterioration.

However, if the patient is already comatose and posturing, decompressive surgery should still be considered in most cases. Approximately 38% of our surgically treated comatose patients were nondisabled at hospital discharge. This is in agreement with pathoanatomic studies that revealed surprisingly few structural changes due to brain stem compression in patients with fatal space-occupying cerebellar infarcts.¹⁷

Because advanced age and signs of a concomitant primary brain stem infarct were associated with poor functional prognosis, these two factors may influence the decision for surgical intervention if a patient with a massive cerebellar infarction has reached a late clinical stage. Magnetic resonance imaging may help in recognizing the extent of brain stem destruction in such cases.

Outcome concerning mortality and functional status was not different when results of external ventricular drainage and suboccipital craniectomy in this series were compared. This is in agreement with several studies that reported successful treatment of hydrocephalus due to massive cerebellar infarction by ventriculostomy without decompressive surgery.^{4,10,18-20} In contrast, subsequent suboccipital craniectomy was necessary in 4 of 10 patients with primary external ventricular drainage because of further deterioration. Possible reasons might have been the persisting compression of the brain stem in the posterior fossa or an ascending herniation favored by ventriculostomy.^{21,22}

A definite answer to the question of whether suboccipital craniectomy should be performed as the first surgical intervention or whether craniectomy should be

performed only in the case of further deterioration after ventriculostomy may be found in a prospective study.¹⁶ In the meantime, because of the low intrinsic risk of suboccipital craniectomy⁶ and a sometimes rapid further deterioration after ventriculostomy alone, we consider decompressive craniectomy to be the treatment of choice for patients with space-occupying cerebellar infarction with ongoing progression of brain stem signs or impairment of consciousness, even when patients are already comatose or posturing. This should be supplemented by ventriculostomy in patients with occlusive hydrocephalus and by tonsillar resection in patients with downward herniation. We believe that only older age and significant signs of primary brain stem infarction should significantly modify the decision for surgical intervention.

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