# MANAGEMENT OF ACUTE CEREBELLAR INFARCTION: ONE INSTITUTION'S EXPERIENCE

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**OBJECTIVE:** The management of cerebellar infarctions is controversial. The aim of this study was to determine which patients require surgical treatment and which surgical procedure should be performed when a patient with a cerebellar infarction exhibits progressive neurological deterioration.

**METHODS:** A total of 44 patients (24 male and 20 female patients; average age, 56 yr) were treated at our institution for cerebellar infarctions in the past 8 years. Twenty-five patients received conservative treatment; two patients who were deeply comatose received no treatment. The remaining 17 patients underwent emergency surgery. Of those 17 patients, 8 underwent external ventricular drainage alone, 5 underwent external ventricular drainage as the first treatment plus secondary suboccipital craniectomy, and 4 underwent suboccipital craniectomy, with removal of necrotic tissue, as the first treatment.

**RESULTS:** Of the 25 conservatively treated patients, 20 experienced good outcomes, 4 experienced moderate outcomes, and 1 died as a result of pulmonary embolism. Of the 17 surgically treated patients, 10 experienced good functional recoveries (7 treated with external ventricular drainage only and 3 treated with drainage followed by suboccipital craniectomy) and 3 survived with mild neurological deficits (one patient underwent ventriculostomy, one suboccipital craniectomy plus external ventricular drainage, and one suboccipital craniectomy only). The overall mortality rate was 13.6% (6 of 44 patients).

**CONCLUSION:** For patients with worsening levels of consciousness and radiologically evident ventricular enlargement, we recommend external ventricular drainage. We reserve surgical resection of necrotic tissue for patients whose clinical status worsens despite ventriculostomy, those for whom worsening is accompanied by signs of brainstem compression, and those with tight posterior fossae.

**KEY WORDS:** Brainstem compression, Cerebellar infarction, Treatment

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Cerebellar infarctions (CIs) without primary brainstem or cerebral involvement are rare; they constitute 1.5 to 8.1% of cases in clinicopathological series (2, 3, 13, 16, 20, 29, 31). CIs are often fatal. The main determinant of outcomes among patients with CIs is the extent of infarction; however, other factors, such as hemorrhagic transformation and reflow of blood into damaged vessels, may also be involved (1, 16, 22). When secondary edema becomes space-occupying, brainstem and fourth ventricle compression occurs, resulting in neurological deterioration. Worsening of the clinical course may be delayed for several days after the onset of the CI (29).

The choice of treatment for patients with CIs is still controversial. Opinions differ mainly with respect to the best option if conservative therapy fails. Although many investigators favor suboccipital decompressive craniectomy for some patients (7–9, 21, 31), others recommend external ventricular drainage (8, 14, 18, 21, 25).

In this study, by reviewing data for a series of 44 patients who were treated at our institution in the past 8 years, we attempted to determine whether neuroimaging data and close neurological surveillance would enable the surgeon to decide whether and how to surgically treat patients with CIs. In addition, we

present our management algorithm (*Fig. 1*) and analyze data from the literature with respect to our treatment policy.

## PATIENTS AND METHODS

Between January 1991 and August 1998, 44 consecutive patients presented to our department with CIs. Patients for whom the ischemic area extended to the brainstem were excluded from this series. There were 24 male and 20 female patients. The patient ages ranged from 9 to 83 years (median, 56 yr). The mean time elapsed between the onset of symptoms of infarction and admission to our hospital was 15 hours (range, 3–75 h). The typical presenting symptoms were vertigo, nausea, and vomiting (42 patients), sometimes accompanied by headaches (16 patients); cerebellar signs were noted for 18 patients. Two patients presented to their initial hospitals in comas. Those two patients exhibited deterioration during transfer to the neurosurgical unit. They presented to us with Glasgow Coma Scale (GCS) scores of 3, and magnetic resonance imaging (MRI) scans did not demonstrate hydrocephalus. Eleven patients had potentially embolic diseases (*Table 1*). Thirty-two patients were hypertensive. All patients underwent GCS assessments at the time of admission (27, 30) (*Table 2*). All patients underwent initial computed tomographic and MRI evaluations. All patients except two underwent angiography or magnetic resonance angiography. The CI was attributable to occlusion of the posteroinferior cerebellar artery in 25 cases (56%), occlusion of the anteroinferior cerebellar artery in 7 cases (15%), occlusion of the superior cerebellar artery in 2 cases (4.5%), and massive infarction (with occlusion of two or more arteries) in 7 cases (15%). For three patients, the involved artery remained unknown.

**TABLE 1. Potentially embolic diseases among patients in the present series**

| Potentially embolic disease | No. of patients |
|---|---|
| Recent cardiac infarction | 6 |
| Atrial flutter | 2 |
| Endocarditis with vegetations | 2 |
| Patent foramen ovale | 1 |
| Total | 11 |

**TABLE 2. Glasgow coma scale scores at the time of admission in the present series**

| Glasgow Coma Scale score | No. of patients |
|---|---|
| 3 | 2 (4.5%) |
| 6 | 2 (4.5%) |
| 9–12 | 15 (34%) |
| 13 | 15 (34%) |
| 14 | 7 (15%) |
| 15 | 3 (6%) |

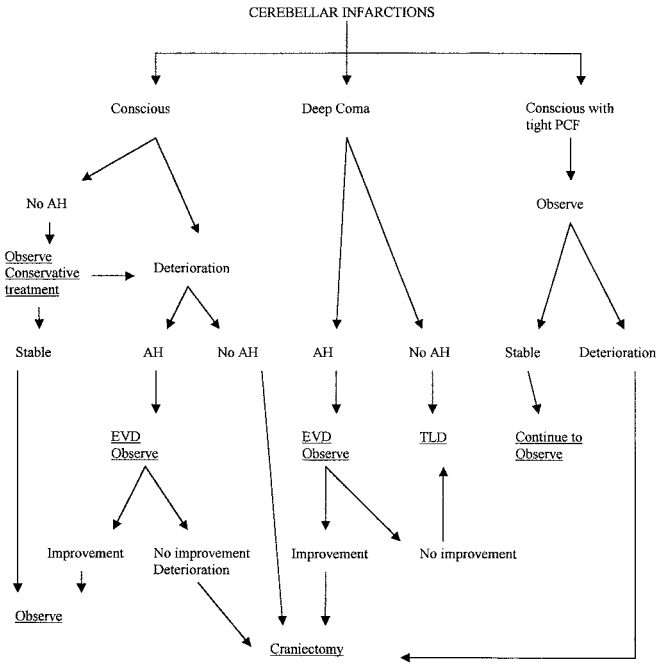


**FIGURE 1.** *Suggested algorithm for cerebellar infarction treatment.* PCF, *posterior cranial fossa;* AH, *acute hydrocephalus;* EVD, *external ventricular drainage;* TLD, *treatment-limiting decision.*

Neurological assessments with the GCS were repeated in the subsequent days. Deterioration was considered a decrease in GCS scores of 2 points. Intracranial pressure (ICP) was monitored for 18 patients. During the last 2 years of the study (1996–1998), ICP monitoring was performed with ventricular catheters (eight patients). Ventricular drains were placed if the ICP exceeded 30 cm $H_2O$. Computed tomographic or MRI scans were obtained at least four times in the pre- and postoperative periods. Fourth ventricle displacement of 3 mm and brainstem deformities were radiological criteria for early suboccipital decompression. We determined the fourth ventricle displacement with respect to the midpoint of a line drawn between the transverse sinuses. For patients with tight posterior cranial fossae, we preferred to perform surgical decompression if the aforementioned criteria were not met. A tight posterior cranial fossa was defined according to the criteria proposed by Weisberg (32), as a lack of observation of the basal cisterns in the posterior cranial fossa, an increase in the size of the third ventricle and the lateral ventricles (including the temporal horns), and a lack of observation of the fourth ventricle (not constant).

All patients received antiedemic therapy (dexamethasone and mannitol) and platelet-inhibiting drugs (aspirin). Twenty-

five patients whose consciousness remained intact received only conservative therapy with dexamethasone (8 mg/24 h) and mannitol (0.5 g/kg every 6 h). Thirteen patients with rapidly worsening consciousness levels accompanied by ICP increases and radiologically evident acute hydrocephalus, without transtentorial herniation, underwent external ventricular drainage for 48 to 96 hours. In one case, it was necessary to perform cerebrospinal fluid shunting after closure of the external drain. Five patients with further neurological deterioration despite external ventricular drainage underwent suboccipital craniectomy and removal of necrotic tissue. Three patients with clinical and radiological signs of brainstem compression from the onset of CI and one patient with a tight posterior cranial fossa underwent craniectomy, with removal of necrotic tissue, as the first treatment. A treatment-limiting decision was made for two patients who presented to us in deep comas (GCS scores of 3) (*Table 3*).

## RESULTS

Results and treatments in the current series are presented in *Table 3*. Independently of each other, two neurologists and one neurosurgeon evaluated the clinical outcomes; they were blinded to the treatments. Outcomes at the time of discharge from our department were classified according to the Glasgow Outcome Scale, i.e., good recovery, moderate disability (disabled but independent), severe disability (conscious but disabled), persistent vegetative state (unconscious, unable to follow commands), or death (17). Of the 25 patients (56%) treated with medical therapy alone, 20 experienced good recoveries, 4 survived with moderate disabilities (slight cerebellar signs), and 1 died as a result of pulmonary embolism.

Of the 13 patients (38%) treated with cerebrospinal fluid diversion, 10 experienced good recoveries (7 underwent external ventricular drainage only and three underwent drainage and subsequent craniectomy). One patient who underwent external ventricular drainage only and one patient who underwent external drainage followed by craniectomy survived with moderate disabilities (slight force deficits associated with ataxia). Patients who underwent suboccipital craniectomy (four patients) as the first treatment exhibited moderate (one patient) or severe (one patient) disabilities or died (two patients). Two patients for whom treatment-limiting decisions were made died. Those two patients arrived at the hospital deeply comatose (GCS scores of 3).

## DISCUSSION

There are differences of opinion regarding the optimal treatment for patients with CIs. Although there is general agreement that conservative therapy is the preferred treatment for patients who are alert and in clinically stable condition (4, 8, 10, 18, 27), the optimal treatment for patients with impaired consciousness remains controversial. Surgical techniques range from craniectomy and necrotic tissue removal to burr hole and external ventricular drain placement. For patients who became critically ill, some authors described successful outcomes after surgical resection of the necrotic tissue (6, 27, 29, 31), whereas others reported good results with external ventricular drainage (11).

We support the use of external drainage as the first treatment for patients with impaired consciousness, regarding it as a less-invasive surgical procedure than craniectomy (*Fig. 1*). Our data suggest that external drainage may be efficacious in the treatment of CIs that become life-threatening. The rationale for this is that consciousness deterioration is not always caused by secondary brainstem compression by the swollen cerebellar tissue. It may also result from hydrocephalus. In such cases, drainage of the hydrocephalus may interrupt a cascade of events that would otherwise increase the subtentorial mass effect (15, 26). This was observed for eight patients in our series, for whom drainage led to a complete recovery of neurological function.

We disagree with authors who suggest that the placement of external ventricular drains increases the risk of upward transtentorial herniation (5). This complication can be virtu-

**TABLE 3. Treatments and outcomes in the present series[a]**

| Treatment | No. of patients | | | | |
|---|---|---|---|---|---|
| | Total | Good recovery | Moderate recovery | Severe disability | Death |
| Conservative | 25 | 20 | 4 | 0 | 1 |
| EVD | 8 | 7 | 1 | 0 | 0 |
| EVD + SC | 5 | 3 | 1 | 0 | 1 |
| SC | 4 | 0 | 1 | 1 | 2 |
| TLD | 2 | 0 | 0 | 0 | 2 |
| Total | 44 | 30 | 7 | 1 | 6 |

[a] EVD, external ventricular drainage; SC, suboccipital craniectomy; TLD, treatment-limiting decision.

ally eliminated by regulating the cerebrospinal fluid flow rate and simultaneously monitoring the patient's neurological status (15). We emphasize that, when such a strategy is chosen, the clinical improvement must be dramatic and must occur within an extremely short period of observation to justify not proceeding with craniectomy. If external drainage fails to improve consciousness, then an immediate decompressive craniectomy, with removal of all necrotic tissue, becomes mandatory. We performed such a procedure for five patients in this series, all of whom survived.

In an analysis of the pertinent literature, we identified a large number of patients as being within our treatment algorithm (*Fig. 1*). In particular, several authors favor a gradual therapeutic approach consisting of medical therapy for conscious patients, ventriculostomy for patients with hydrocephalus, and decompressive craniectomy for patients with symptoms of brainstem compression. The effectiveness of external ventricular drainage as the first treatment option for patients with CIs and hypertensive hydrocephalus was demonstrated by Cioffi et al. (6). Those authors reviewed data for a series of 10 patients with CIs; ventricular drainage was used for 5 patients who presented with hydrocephalus, and medical treatment was used for the remaining patients. Of the five patients who were treated with extraventricular drainage, one died, two experienced full recoveries, one exhibited a partial disability, and one required surgical decompression because of a failure to exhibit improvement with ventriculostomy. In a review of data for 55 patients, Shenkin and Zavala (28) reported the use of external ventricular drainage for 6 patients with hydrocephalus, with excellent outcomes for 4 patients and good outcomes for 2. The preoperative conditions of those six patients ranged from lethargic to comatose. Mathew et al. (24), in a study of 50 patients with CIs, confirmed the successful results for patients with hydrocephalus who were treated with external ventricular drainage. Those authors treated nine patients primarily with external ventricular drainage, which was supplemented with craniotomy for two patients because of continuing deterioration. Those two patients experienced good recoveries after removal of the infarction. Of the seven patients treated with extraventricular drainage only, five made good recoveries, one remained moderately disabled, and one died.

Khan et al. (19) reported on a series of 11 patients, including 2 with stupor or coma, who experienced good outcomes with ventriculostomy. Jauss et al. (16) reported on a series of 84 patients with CIs. Fourteen patients were treated with ventriculostomy (eight patients were awake/drowsy, four were somnolent/stuporous, and two were comatose). Of those 14 patients, 10 patients experienced good outcomes and 4 patients who were primarily treated with ventriculostomy required decompressive surgery in their later clinical courses.

Alert patents in this series with initial signs of hydrocephalus were monitored with close clinical and radiological observation and ICP monitoring. Patients with CIs may exhibit deterioration after variable periods with relatively stable deficits (6, 12, 23). This is attributable to progressive expansion of cerebellar tissue necrosis, with consequent increases in water uptake and brain edema (29). When a CI acts as a progressively enlarging mass, signs of brainstem compression or hydrocephalus occur. These two occurrences require immediate surgical treatment. Early diagnosis and prompt surgical treatment are mandatory, before the development of irreversible brainstem damage or severe hypertensive hydrocephalus. Therefore, we monitor all patients who do not undergo surgery as the first treatment with close clinical observation, serial computed tomographic scans, and ICP monitoring.

We agree with other authors (21, 23, 24, 29) that, when clinical deterioration is accompanied from the onset by clinical and radiological signs of brainstem involvement without signs of hydrocephalus, the patient must undergo an immediate decompressive craniectomy with removal of the necrotic tissue, after MRI has excluded secondary brainstem ischemia. This policy was applied for two patients. In a review of CIs published by Feely (9) in 1979, 55 infarctions were reported to act as expanding mass lesions, compressing the brainstem. The author demonstrated that surgical decompression is mandatory if deterioration occurs or brainstem compression is already advanced.

We think that evidence of a tight posterior cranial fossa justifies immediate surgical decompression in the absence of brainstem compression. Conversely, if coma has been present from the onset and further severe deterioration occurs during transfer, in the absence of hydrocephalus, then a treatment-limiting decision becomes appropriate.

The debate regarding the optimal treatment for patients with CIs cannot be completely resolved until a 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 intracranial hypertension or brainstem compression should receive antiedemic therapy, with close clinical and radiological surveillance, whereas minimally invasive interventions such as external ventricular drainage can often rapidly resolve brainstem compression and improve the patient's neurological status. Moreover, the successful treatment of patients with CIs depends on the awareness that clinical deterioration may occur some days after infarction onset, early diagnosis, and immediate institution of treatment.

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## COMMENTS

Raco et al. present an 8-year experience with acute cerebellar infarction in 44 patients managed medically, with ventriculostomy, with suboccipital craniectomy, or with a combination of ventriculostomy and craniectomy. This experience demonstrates that a majority of these patients (61%) can be managed nonoperatively. For the rest, a stepwise approach was practiced, beginning with ventriculostomy in obtunded or deteriorating patients and resorting to decompressive craniectomy in patients who did not improve, who deteriorated further, or who showed signs of brainstem compression. Selection bias, retrospective data collection, and a limited patient cohort prevent this report from determining optimum management of cerebellar infarction, but a logical algorithm is presented that summarizes the approach used by many neurosurgeons. It should be emphasized that this approach works well only with close clinical and radiographic surveillance. These patients deteriorate rapidly, and a low threshold for craniectomy and resection of infarcted cerebellum should be maintained. Patients undergoing preventive craniectomy tend to recover better than those undergoing emergency craniectomy after deterioration.

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Urgent surgical decompression of cerebellar infarction can be a life-saving and rewarding surgical procedure in well-selected patients. Indications for surgical intervention in the setting of acute cerebellar infarction are controversial, as is the role of external ventricular drainage alone or in combination with surgical decompression. Raco et al. have retrospectively reviewed their 8-year experience with 44 patients presenting with acute cerebellar infarction at a single institution. On the basis of their review of that experience, they present an algorithm that is useful for clinical decision making when treating patients with acute cerebellar infarction.

The precise clinical features that lead to deterioration in cerebellar infarction are incompletely understood. Patients with mass effect demonstrated on the computed tomographic scan commonly deteriorate when obstructive hydrocephalus ensues. A number of radiological features have been shown to be predictive of neurological deterioration, including fourth ventricular distortion and shift, obstructive hydrocephalus, brainstem deformity, and basilar cistern compression.

In the present series, the authors treated 25 patients with intact consciousness with medical therapy consisting of corticosteroids and mannitol. Twenty of these patients had a good outcome, four experienced moderate disability, and one died of a pulmonary embolism. The authors chose to treat 13 patients with rapidly worsening consciousness accompanied by hydrocephalus without transtentorial herniation by external ventricular drainage alone. Five of these patients deteriorated further despite the placement of an external ventricular drain, and they subsequently underwent decompressive surgery. Of these five patients, three had a good recovery, one experienced a moderate recovery, and one patient died.

Although the data must be interpreted with the usual restrictions of a retrospective study in mind, the authors have provided a set of guidelines that can be of great benefit to physicians treating patients with acute cerebellar infarction.

Despite theoretical concerns of upward herniation with the use of external ventricular drainage, the authors have demonstrated that in well-selected patients, this treatment can provide significant benefit. Decision making for patients must be individualized. In general, however, patients with progressive hydrocephalus, brainstem deformity, and basilar cistern compression should be considered for preemptive surgery before they become stuporous or comatose.

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Raco et al. describe their experience with management of large cerebellar infarctions. The results and recommendations are similar to those described in the literature. Hornig et al. (2) reported 52 patients with large cerebellar infarctions; 36 patients were treated surgically, with 30 undergoing some form of posterior fossa decompression and the remaining 6 undergoing ventricular drainage alone. Overall, 29 patients (56%) made a good recovery. Four of 10 patients who were treated initially by ventricular drainage deteriorated and underwent posterior fossa decompression. Even patients with huge infarctions of the cerebellum can recover, presumably if there is no brainstem infarction (4).

No randomized trials have been published on which to base treatment decisions. Some of the management strategies employed in this report may not be widely used. Guidelines for the treatment of patients with acute ischemic stroke recommended that corticosteroids such as dexamethasone not be administered to patients with acute ischemic stroke (1). This recommendation was based on Level of Evidence 1, Grade A, status. These guidelines did suggest that osmotherapy and hyperventilation were recommended for patients with herniation syndromes with increased intracranial pressure (Level of Evidence 2, Grade B) and that ventricular drainage might be indicated (Level of Evidence 3–5, Grade C). Surgical decompression was recommended for large cerebellar infarctions with brainstem compression (Level of Evidence 3–5, Grade C). In the German-Austrian Cerebellar Infarction Study, 84 patients with massive cerebellar infarction underwent craniotomy and decompression (34 cases, 41%), ventricular drainage (14 cases, 17%), or medical treatment (36 cases, 43%) on the basis of a decision made by the treating physician (3). Level of consciousness after clinical deterioration was the strongest predictor of poor outcome. Half of patients who deteriorated made a meaningful recovery. There was no difference in outcome in relation to treatment for awake or somnolent/stuporous patients. Deterioration was most common 3 days after onset.

One interpretation of the results is that because deterioration in consciousness predicted poor outcome, decompression should be performed early in patients with large infarctions that are likely to swell. Early prophylactic decompressive surgery has been advocated for supratentorial infarctions in clinical trials. It is hard to argue against it for cerebellar infarction in view of the outcomes that often can be achieved. It is difficult to be certain that ventricular drainage is as effective as suggested in the present report because of the lack of randomization. I prefer decompressive surgery in such cases as opposed to prolonged ventricular drainage.

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Raco et al. provide a retrospective review of 44 patients with cerebellar infarction. Twenty-five of the patients received conservative treatment, and two patients who were deeply comatose received no treatment. The remaining 17 patients underwent surgery. The authors developed an algorithm on the basis of their management strategy of insertion of an external ventricular drain or ventriculostomy if acute hydrocephalus is present. Of the 17 patients treated surgically, 10 had good functional outcomes. Three patients survived with mild neurological deficits, and the overall mortality rate was 13.6%.

A number of authors have described the use of ventriculostomy in their treatment of patients presenting with hydrocephalus and cerebellar infarction. The concern here is the potential risk for upward herniation. Raco et al. insert a ventriculostomy and observe the patient. If there is no improvement or if there is deterioration, then craniectomy is performed. The risk of craniectomy in these patients is fairly small. At our institution, we typically adopt the protocol of insertion of a ventriculostomy while decompressive craniectomy is performed. Decompressive craniectomy is a rapid procedure and is usually fairly well tolerated, even though the tissue being removed is infarcted, with little blood flow present. In terms of deterioration, the problem with an algorithm of insertion of a ventriculostomy, followed by a "wait-and-see" attitude, is that time can be lost in terms of potential for recovery. Although this single institution's experience does provide some insight into the management of cerebellar infarct, it should not stay the hand of the surgeon from proceeding rapidly to decompressive craniectomy in patients without brainstem infarction and with cerebellar infarction.

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