Surgical and medical management of patients with massive cerebellar infarctions: Results of the German-Austrian Cerebellar Infarction Study

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M. Jauss
D. Krieger
C. Hornig
J. Schramm
O. Busse,
for the GASCIS study centers

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M. Jauss
Department of Neurology,
University of Lübeck,
Ratzeburger Allee 160,
D-23538 Lübeck, Germany
e-mail: jauss@usa.net

D. Krieger
Department of Neurology,
Cleveland Clinic Foundation,
9500 Euclid Avenue, Cleveland,
Ohio 44195-5001, USA

C. Hornig
Department of Neurology,
Klinik Weilmünster, Weilstrasse 10,
D-35789 Weilmünster, Germany

J. Schramm
Department of Neurosurgery,
University of Bonn,
Sigmund-Freud-Strasse 25,
D-53105 Bonn, Germany

O. Busse (✉)
Department of Neurology,
Community Hospital Klinikum Minden,
Friedrichstrasse 17,
D-32427 Minden, Germany

Abstract 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. The clinical course and neuroradiological features of 84 patients (aged 22–78, mean 58.5 years) with massive cerebellar infarction confirmed by computed tomography were prospectively observed for 21 days after admission and at 3-month follow-up using a standardized protocol. Data were gathered from 1992 to 1996 in 17 centers. The patients were assigned to three treatment groups depending on the decision of the primary caretaker: 34 underwent craniotomy and evacuation, 14 received ventriculostomy, and 36 were treated medically. Treatment groups differed regarding the level of consciousness, signs of mass effect in computed tomography and signs of brainstem involvement. The overall risk for poor outcome depended on the level of consciousness after clinical deterioration (odds ratio = 2.8). Subgroup analysis of awake/drowsy or somnolent/stupor patients revealed no relationship to treatment. The vascular territory involved did not affect outcome. Surgical treatment for massive cerebellar infarctions was not found to be superior to medical treatment in awake/drowsy or somnolent/stupor patients. Half of all patients deteriorating to coma treated with ventricular drainage or decompressive craniotomy had a meaningful recovery. We were unable to compare surgical versus medical therapy in this subgroup due to lack of control group. This study supports the notion that the level of consciousness is the most powerful predictor of outcome, superior to any other clinical sign and treatment assignment. Deterioration of consciousness typically occurred between days 2 and 4, with a maximum on day 3.

Key words Cerebellar infarction · Treatment · Decompression surgery · Prognosis

Introduction

Cerebellar infarcts constitute 1.5–4.2% of cases in clinicopathological series [1, 29] and 1.9–10.5% of those in clinical series of patients with cerebral infarctions [2, 3]. A subgroup of patients with large cerebellar infarctions deteriorate after a variable interval of relatively stable deficits [12]. The main predisposing factor for subsequent deterioration is thought to be the infarct size; however, other factors, including type of underlying vascular lesion, hemorrhagic transformation, and inadequate collateral blood flow may be involved. Patients presenting with areas of hypodensity on computed tomography (CT) ex-

tending to two-thirds of the posterior inferior cerebellar artery territory are currently considered to be at risk for subsequent deterioration [15]. Neurological deterioration occurs as a result of mass effect of swollen brain tissue in the posterior fossa [9] compressing the brainstem, with associated occlusive hydrocephalus [14, 18].

Mechanisms of clinical deterioration in cerebellar infarcts are recognized and are addressed in current therapeutic guidelines [12, 14, 19, 24, 27]. 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., ventriculostomy, posterior fossa craniotomy), and timing of the procedures are widely disputed [7, 9].

Craniotomy is considered to be essential in obtunded patients with massive cerebellar infarcts, and this has therefore prohibited any controlled therapeutic trial design among these patients [10]. Other argue that such treatment cannot be advocated in the absence of evidence from randomized controlled trials. Consequently we decided to carry out an observational design to provide prognostic factors and outcomes on the efficacy of therapeutic interventions used in patients with massive cerebellar infarcts [19, 27].

Seventeen centers were recruited throughout Germany and Austria to participate in the study. All sites were tertiary referral centers, either university hospitals or large community hospitals with departments of neurology, neurological surgery, and critical care facilities. The study protocol provided case report forms and agreement on a standardized medical protocol, specified surgical approaches, and serial standardized radiographic studies among participants [19]. This report focuses on the clinical characteristics, treatment decisions with respect to clinical and radiographic findings, and prognostic criteria in patients with massive cerebellar infarcts.

Patients and methods

Recruitment and intervention patterns

The study was designed as a prospective, observational, multicenter trial. We included patients aged between 18 and 80 years, admitted within 5 days after onset of a cerebellar infarction with mass effect by CT. Patients presenting with acute onset of coma, quadriparesis, or other indicators of underlying basilar occlusion, or prior significant neurological impairment (Rankin > 2) and unrelated terminal conditions were excluded from the study.

Baseline characteristics of patients

Case reports forms and CT scans of 95 patients were obtained over the 4-year period and made available for analysis. After evaluation of the CT scans by the neuroimaging committee and clinical data by the steering committee, 11 patients were excluded who either did not meet the predetermined criteria for massive cerebellar infarction ($n = 8$) or presented with clinical and CT evidence of basilar occlusion ($n = 3$). Consequently 84 patients (mean age 58.5 ± 12.5, 56 male) were included for further analysis.

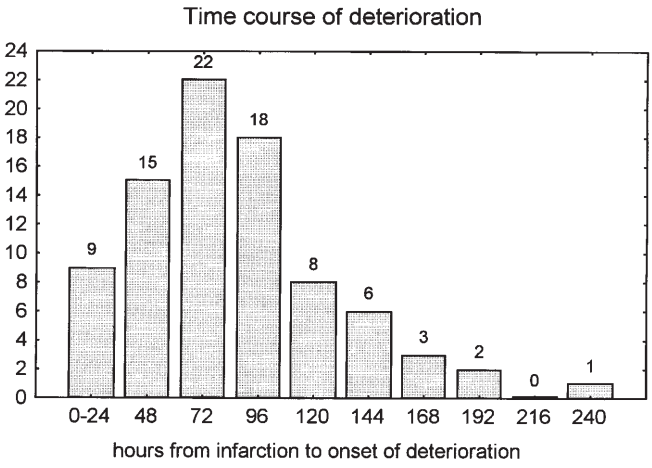

Fig. 1 Time course of deterioration during the first 10 days after stroke onset

Signs and symptoms at cerebellar stroke onset included: vertigo (87%), gait disturbance (85%), nausea (82%), vomiting (66%), headache (65%), drowsiness (60%), slurred speech (44%), neck pain (37%), weakness (24%), double vision (22%), and sensory disturbances (19%).

Cranial nerve and focal neurological abnormalities at admission were occurred as follows: lower cranial nerve involvement (36%), unilateral extensor toe (20%), gaze palsy (21%), bilateral extensor plantar response, (16%), 6th nerve palsy (15%), 7th nerve palsy (9%), pupillary abnormalities (7%), hemiparesis (5%), quadriparesis (3%), and extensor posturing (1%).

Neurological symptoms developing during hospitalization were: lower cranial nerve involvement (14%), pupillary abnormalities (4%), 6th nerve palsy (4%), bilateral extensor plantar response (2%), gaze palsy (2%), hemiparesis (1%), and unilateral extensor response (1%). Development of 7th nerve palsy, quadriparesis, or extensor posturing not already present at admission did not occur.

The average time from onset to admission was 43.7 ± 39.1 h. Thirteen patients were comatose on admission. Eleven patients underwent craniotomy on the day of admission, one had surgery on day 2, and one had surgery on day 4.

Fifty patients presented on admission with awake/drowsy level of consciousness. During further observation 11 patients progressed to somnolence/stupor and 4 progressed to coma within an average of 24 ± 41 h (mean ± SD, range 0–168) after admission. Considering all patients, the most severe disturbance of consciousness was noted 18 ± 35 h (range 0–216) after admission or 61 ± 43 h (range 0–216) after onset of stroke.

The time course of decline in level of consciousness during the first 10 days after onset of infarction for all patients is shown in Fig. 1.

Therapeutic protocols

Treatment decisions were reached by the independent judgement of the local caregivers for each individual patient. Medical therapy directed against brain swelling included the use of glycerol 10% and mannitol 20%. A ventricular drainage was inserted in the right frontal horn and changed within 7 days if necessary. Decompressive surgery was performed as a large craniotomy over the infarct, duroplasty, and resection of the posterior atlas arch if tonsillar her-

niation was apparent. Resection of the infarcted tissue was not mandatory. Patients deteriorating despite ventriculostomy and requiring craniotomy later during clinical course were considered in the craniotomy treatment group.

Examination protocol

All patients were examined using a standardized examination protocol. Serial neurological examinations were performed on days 1–10 and 21 and 3 months after stroke onset. Level of consciousness was assessed using a three-point scale. Also assessed were cranial nerve abnormalities and focal neurological signs, including motor abnormalities, disturbance of coordination, sensory disturbances, and reflex status. Repeat CT was performed at least three times in the pre- and postoperative period. The functional deficit was evaluated in all survivors during an outpatient visit 3 months after onset.

Scoring

Disturbance of consciousness was rated as: 1 = awake/drowsy, 2 = somnolent/stupor, 3 = coma [26]. The outcome was assessed using the modified Rankin scale [31]: 1 = no symptoms, 2 = no significant disability despite symptoms, 3 = slight disability, impaired in previous activities but able to look after own affairs without assistance, 4 = moderate disability, needs help but able to walk without assistance, 5 = moderately severe disability, needs help in many activities of daily living, 6 = severe disability, bedridden, requiring constant nursing, 7 = death. A dichotomized Rankin score was used for logistic regression. Since previous studies have demonstrated a good outcome with only slight disability in the majority of patients suffering from cerebellar infarction [4, 33] a Rankin score of 2 or less was considered a good outcome and a Rankin score of 3 or more a poor outcome.

Radiographic studies

Serial CT was performed at least once before and twice after intervention. Likewise, medically treated individuals were scanned three times. CT were rated by a blinded neuroradiologist according to three particular features of infratentorial mass effect. A CT score ranging from 0 to 9 was calculated by adding the following items (see Fig. 2):

1. of the 4th ventricle (0 = no compression, 1 = unilateral compression, 2 = shifted midline, 3 = not visible)
2. Compression of the quadrigeminal cistern (0 = no compression, 1 = mild with asymmetric compression ipsilateral to the infarction, 2 = moderate with evidence of bilateral compression, 3 = severe bilateral compression with obscured quadrigeminal cistern)
3. Dilatation of the inferior horn of the lateral ventricle (0 = no dilatation, 1 = mild, 2 = moderate, 3 = severe)

Statistical analysis was performed after a trichotomization of overall CT scores:

– 0–3: no or slight mass effect
– 4–6: moderate mass effect
– 7–9: severe mass effect

To evaluate retest and interrater reliability, 28 randomly selected CTs from the entire sample were rated by a neurosurgeon and a neurologist who were naive to the patient's clinical state and not involved in their treatment. Results were compared to those of the neuroimaging committee (O.B., B.M., Minden). An intraclass correlation coefficient was calculated [21] to consider probable systematic deviations between observers. The intraclass correlation coefficient was 0.90 ± 0.04 for retest (overall CT score) and 0.78 ± 0.02 for interrater reliability.

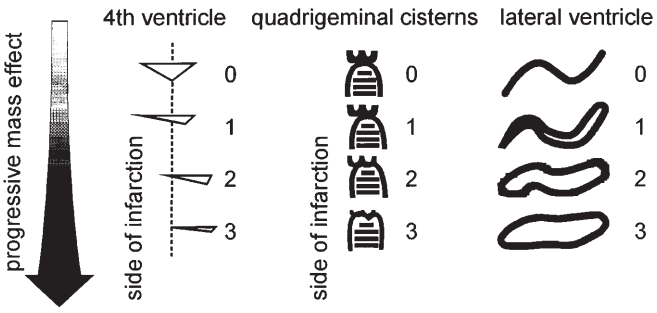

Fig. 2 Three items of the CT score during progressive mass effect compression of the 4th ventricle (assessed on the CT slice with the 4th ventricle best visible); compression of the quadrigeminal cistern (assessed on the most rostral CT slice with the cerebellar vermis largely visible); dilatation of the inferior horn of the lateral ventricle (assessed on the CT slice with the inferior horn of the lateral ventricle best visible)

Statistical methods

To compare clinical and neurological characteristics between the treatment groups, cross-tables were produced using Fisher's exact test to determine statistical significance ($P < 0.05$). Wilcoxon's rank sum test for dependent samples was used in comparisons of ordinal data, such as CT score during follow-up. Continuous variables, such as age and timing of operation, were compared between the treatment groups by means of Duncan's post hoc analysis of variance [22].

Logistic regression with stepwise selection was performed to determine relevant prognostic factors. The level for both entry and staying in the model was 0.05 [28]. The dichotomized Rankin score on day 90 was the dependent variable. Independent variables were: degree of impairment of consciousness at admission, worst degree of consciousness during deterioration in clinical course, CT criteria of mass effect (compression of 4th ventricle, compression of quadrigeminal cistern, dilatation of the inferior horn of lateral ventricle), age, and lower cranial nerve involvement at admission and at deterioration.

The patients were grouped according to their level of consciousness to compare treatment effects in patients with clinical states of varying severity. Subgroup analysis was performed using cross-tabulations and Fisher's exact test. Spearman's rank sum correlation coefficient was used to examine the relationship between the level of consciousness, outcome, and mass effect on CT.

Results

Clinical and neuroradiological presentation and treatment decision

Treatment groups differed with respect to level of consciousness on admission, lowest level of consciousness during course, mass effect on CT, and lower cranial nerve involvement and unilateral and bilateral extensor plantar response (Table 1). Four patients primarily treated with ventriculostomy required decompressive surgery during

Table 1 Clinical characteristics of 84 patients with massive cerebellar infarction at deterioration by treatment (percentages may not add to 100 because of rounding)

	Medical treatment (n = 36)		Ventricular drainage (n = 14)		Craniotomy (n = 34)	
	n	%	n	%	n	%
Age (years)	61.2 ± 10.3		54.5 ± 17.3		57.4 ± 12.0	
Male gender	22	65	9	64	25	71
Etiology						
Undetermined	26	72	9	64	23	68
Lower embolic (highly suspected)	10	28	5	36	10	29
Dissection	–	–	–	–	1	3
Vascular territory						
Superior cerebellar artery	4	11	–	–	1	3
Anterior inferior cerebellar artery	1	3	–	–		–
Posterior inferior cerebellar artery	26	72	9	64	26	76
Undetermined	4	11	–	–	–	–
Multiple	1	3	5	36	7	21
Clinical findings (at deterioration)						
6th nerve palsy	5	15	1	10	7	32
7th nerve palsy	2	7	1	10	2	9
Pupillary abnormalities	1	3	2	14	3	9
Hemiparesis	3	9	1	11	2	11
Quadriparesis	–	–	–	–	2	11
Unilateral extensor plantar response*	10	28	2	15	4	12
Bilateral extensor plantar response*	4	11	2	15	10	31
Posturing	–	–	1	8	–	–
Gaze palsy	3	9	1	12	7	30
Lower cranial nerve involvement*	2	6	7	50	27	75
Level of consciousness at admission*						
Awake/drowsy	28	78	8	57	14	19
Somnolent/stuporous	8	22	4	29	9	26
Comatose	–	–	2	14	11	32
Level of consciousness at deterioration*						
Awake/drowsy	27	75	3	21	6	18
Somnolent/stuporous	9	25	8	57	12	35
Comatose	–	–	3	21	16	47
Mass effect in CT*						
No or slight (score 0–3)	24	67	1	7	8	24
Moderate (score 4–8)	9	25	3	21	14	41
Severe (score 9–12)	3	8	10	71	12	35
Duration (hours)						
Onset, admission	41 ± 44		46 ± 52		45 ± 29	
Onset, surgery**	–	–	82 ± 61		62 ± 29	
Onset, deterioration	59 ± 49		79 ± 61		57 ± 25	

* $P < 0.05$ (Fisher's exact test)
** $P < 0.05$ (Duncan's test)

their later clinical course. If craniotomy was deemed necessary, surgery was performed earlier (mean time from admission to craniotomy: 62 ± 29 h) than in patients where ventriculostomy was felt appropriate (time from admission to ventriculostomy: 82 ± 61 h). The difference was statistical significant (Table 1).

Clinical and neuroradiological presentation and outcome

Among the 36 awake/drowsy patients 31 had a good outcome. In the 29 patients with somnolent/stupor levels of consciousness 22 had a good outcome. In contrast, of the 19 patients with progression to coma during clinical course only 9 had a good outcome.

Table 2 Outcome by state of consciousness at deterioration

Outcome	Awake/ drowsy (n = 36)	Somnolent/ stupor (n = 29)	Comatose (n = 19)
Good outcome (Rankin ≤ 2)	31 (86%)	22 (76%)	9 (47%)
Poor outcome (Rankin > 2)	5 (14%)	7 (24%)	10 (53%)

Factors affecting the outcome were calculated using a logistic regression model (Table 2). The state of consciousness at deterioration was the only factor retained in the logistic regression model, associated with a 2.8 times increased risk for poor outcome (95% confidence interval: 1.4–5.6) for each increase on the three-step scale of disturbance of consciousness. Age, lower cranial nerve involvement and CT criteria of mass effect were excluded by the stepwise regression. The latter of these was reversible by craniotomy, with a remarkable improvement from a preoperative median overall CT score of 5 (3.5–6; 25–75% quartile) to a postoperative median overall CT score of 2 (0–3). This difference between the pre- and postoperative CT scores (within 4.9 ± 3.0 days) was statistically significant ($P < 0.05$, Wilcoxon's rank sum test for dependent samples).

Treatment decision and outcome

Among 36 awake drowsy patients 27 were treated medically, 3 had ventriculostomy, and 6 underwent decompressive craniotomy. Four of the latter had lower cranial nerve involvement at the time of surgery. In contrast, only two patients of the remaining 27 medically treated patients had lower cranial nerve involvement ($P < 0.05$, Fisher exact test). The various regimens were evenly distributed among patients with somnolence or stupor (n = 29), but surgery was performed uniformly among comatose patients (n = 19), predominantly surgical decompression of the posterior fossa. Among comatose patients only three were treated exclusively with ventriculostomy. Vascular territory had no effect either on treatment decision or on outcome.

Among individuals treated exclusively medically 30 of 36 (83%) had a Rankin score of 2 or less (good outcome) at 3 months follow-up examination. Of 14 patients treated with ventricular drainage 10 (76%) had a good outcome. Of 34 patients with craniotomy 22 (65%) had a good outcome.

Since all patients progressing to coma were treated surgically, a comparative analysis could be carried out only among awake/drowsy and somnolent/stupor patients. Treatment effects in patients received the various treatment regimens for similar levels of consciousness were evaluated by a post hoc analysis of variance with Duncan's test. It is noteworthy that there was no difference in outcome after 3 months among awake and stupor patients in any of the treatments (Table 3, comatose patients are listed only in descriptive manner).

In addition, subgroup analysis of awake/drowsy patients with a CT score of 3 or more (n = 14) revealed no significant difference in outcome with any particular treatment (Table 3). Spearman's rank sum correlation between the state of consciousness at deterioration and CT score was low ($\tau = 0.34$; $P < 0.05$). The correlation between outcome at 3 months and CT score at deterioration was even lower ($\tau = 0.16$; $P < 0.05$).

Discussion

The results of this exploratory analysis using a rather homogeneous group of CT-diagnosed cerebellar infarctions

Table 3 Outcome at follow-up 90 days after infarction by treatment

	Medical treatment (n = 36)		Ventricular drainage (n = 14)		Craniotomy (n = 34)	
	n	%	n	%	n	%
Outcome						
Good (Rankin ≤ 2)	30	83	10	71	22	65
Poor (Rankin > 2)	6	17	4	29	12	35
Subgroup analysis of Rankin score (day 90)						
Good outcome (Rankin ≤ 2)	23	85	3	100	5	83
Poor outcome (Rankin > 2)	4	15	0	0	1	17
Somnolent/stupor (n = 29)						
Good outcome (Rankin ≤ 2)	7	32	6	27	9	41
Poor outcome (Rankin > 2)	2	22	2	25	3	25
Coma (n = 19)						
Good outcome (Rankin ≤ 2)	–	–	1	33	8	50
Poor outcome (Rankin > 2)	–	–	2	67	8	50

with mass effect indicate that surgical therapy in comatose patients is safe and effective. However, since this study did not allow comparison of medical therapy and surgical therapy in this subgroup, surgery cannot be generally advocated solely on the basis of this study. In contrast, patients who have not yet deteriorated to coma did not seem benefit from any particular aggressive treatment options. Exploratory analysis revealed no clinical or radiographic signs of prognostic value to identify patients with somnolent/drowsy level of consciousness who might benefit from decompressive surgery.

In addition, there are no CT signs suggesting a particularly poor prognosis or indicating immediate surgery. The results of this study also do not make it possible to establish the superiority of any particular surgical technique (i.e., ventriculostomy or decompressive surgery) in somnolent and stupor patients The concern that surgical decompression of the posterior fossa with evacuation of infarcted tissue may mask spontaneous recovery or rehabilitation effects requires consideration but was not addressed in this study. Further studies must determine the appropriate treatment in relation to invasiveness in patients with massive cerebellar infarction. Our findings warrant controlled trials in patients of the intermediate group with CT signs of mass effect and only moderately decreased levels of consciousness.

Presentation and early clinical course

Only a small number of our patients with massive cerebellar strokes are comatose upon admission to hospital, but the majority deteriorate within 3 days after onset (Fig. 1). This finding is supported by other studies [4, 12, 15, 17] which also report neurological decline within 3–4 days after onset. The most prominent feature of deterioration is the declining level of consciousness, while focal neurological symptoms do not play a role. Gaze palsy was the most frequent focal neurological symptom, often encountered early on.

Treatment decision in massive cerebellar infarction

Treatment decisions in this study were based on CT criteria and clinical findings. Ventricular drainage was preferred in patients with CT evidence of inferior horn enlargement, while decompressive craniotomy was performed in those with CT signs of quadrigeminal cistern compression. Findings associated with decompressive surgery included clinical course and lower cranial nerve involvement. These procedures reflect the agreement of local participants with previously published studies [9, 27].

No conclusive data now exist on the appropriateness of particular therapeutic approaches for massive cerebellar strokes. Anecdotal case series focus on various treatment alternatives for cerebellar infarcts of varying severity [5, 8, 11, 13, 20, 23, 25, 30]. Khan et al. [17] reported a series of 11 patients including two with stupor or coma who had a good outcome with steroids and ventriculostomy. In this study the majority of patients treated with ventricular drainage improved; however, 4 of 18 patients progressed to coma and ultimately required decompressive surgery of the posterior fossa. This is in agreement with the observation that ventricular drainage in patients with posterior fossa mass lesions may enhance the risk of upward transtentorial herniation [9, 16]. Additionally, 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 [4].

Present reports have in common that no final conclusions can be drawn with either particular strategy. To overcome this to some extent we prospectively evaluated treatment strategies in patients with massive cerebellar infarcts of varying severity. We found that among the participants it is particularly the comatose patients who were treated surgically. We also observed a general trend during the study period towards craniotomy and a tendency to neglect ventriculostomy as a treatment option. The results of this study indicate general agreement on therapy guidelines and a significant overlap in the intermediate group.

We anticipated some bias in selection of participants and the Hawthorne effect, i.e., increased awareness of a potential deterioration leading to an early decision for aggressive treatment (see Tiffin and McCormick [32]). In fact, many surgical patients in this cohort were treated in anticipation of further deterioration while they were still awake/drowsy.

This study was not designed to establish an advantage of either surgical procedure or to develope specific guidelines for surgical interventions. However, it is noteworthy that almost one-half of patients proceeding to coma and treated with decompressive surgery survived and had a meaningful recovery.

Outcome after massive cerebellar infarction

In studying a variety of clinical and radiographic items and therapeutic interventions, we found that the outcome after massive cerebellar stroke depends principally upon the level of consciousness. In this series 15% of patients were comatose at admission, and another 7% progressed to coma after admission. The finding that level of consciousness is the key prognostic factor confirms the results of a recently published retrospective study [12]. Since other clinical parameters and radiographic signs are less significant than the level of consciousness, extreme caution should be used in administering sedatives to patients with massive cerebellar infarcts. This study shows

that patients with a level of consciousness better than coma do not benefit from surgery.

We also we evaluated prognostic factors in the subgroup of comatose patients who underwent craniotomy. Here we could not establish any clinical or radiographic sign of prognostic significance. This may in part be explained by the lack of variance in the independent clinical variables. Neurophysiological tests, in particular brainstem acoustic evoked potentials, have recently been shown to be correlated with clinical state and response to therapy [18] and should be explored further to determine how this modality can improve diagnostic certainty and therapeutic guidance.

CT signs of mass effect were not associated with poor outcome at 3 months, because of treatment interference. This finding is in line with clinicopathological evidence revealing surprisingly few structural changes secondary to brainstem compression in patients dying from cerebellar herniation [29]. Conversely, clinical studies have revealed significantly better outcomes with cerebellar than hemispheric or intrinsic brainstem strokes [6].

This study found no effect of the vascular territory on either clinical performance or outcome. However, it did confirm the frequent involvement of the posterior inferior cerebellar artery territory and multiple territories in cerebellar infarcts with mass effect. The relative paucity of superior cerebellar artery territory infarctions in this cohort may be due to their frequent association with intrinsic brainstem infarctions which consequently led to an exclusion from this study [33].

Conclusion

This study shows that surgical treatment in massive cerebellar infarctions is superior to medical treatment either in awake/drowsy patients or in somnolent/stupor patients. Comatose patients may benefit from ventricular drainage or decompressive craniotomy; however, we found no benefit of surgical versus medical therapy in comatose patients. On the other hand, clinical practice based on available level 1 and 2 evidence reveals that surgical therapy is not withheld from comatose patients. This practice is supported by the rate of good outcomes observed with surgery in this dire clinical situation. Not every therapeutic decision can be studied in a randomized manner. This study confirms that medical treatment is appropriate for patients whose level of consciousness is better than coma, and surgery for patients deteriorating to coma. Level of consciousness is thus the most powerful predictor of outcome predictor. Since clinical deterioration occurs between days 2 and 4, the patient should be kept under observation at least during this period, preferably in a stroke unit or intensive care environment.

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