



## SUBOCCIPITAL DECOMPRESSIVE CRANIECTOMY AND EXTRAVENTRICULAR DRAINAGE IN CEREBELLAR STROKE

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**ABSTRACT** **INTRODUCTION AND AIM:** Cerebellar ischemic stroke are important causes of acute neurological morbidity, accounting for 2% to 3% of the 600 000 strokes occurring annually. Majority of the CI can be managed conservatively. Surgical intervention may be necessary if mass effect develops. Surgical therapy includes ventriculostomy and suboccipital decompressive craniectomy (SDC). Local surgery protocols differ greatly, and the questions of whether and when ventriculostomy or SDC should be performed and whether surgery should be performed in a single intervention, in combination, or in a stepwise approach remain unanswered. This study is proposed to formulate a protocol for optimal timing of surgical intervention. **MATERIALS AND METHODS:** Retrospective & prospective study including 83 patients, were managed as per a proposed protocol after diving them based on their presenting GCS and the observations were recorded and tabulated. **RESULTS:** We found out in our study the GSC / the consciousness level is the single most factors that should be taken into account before taking the decision for surgery. Although mass effect on imaging is an additional finding to decide the correct timing of surgery, alone it does not hold much significance. **CONCLUSIONS:** Majority of the patients can be managed conservatively and when possible (as per GCS) all the patients should be given a conservative trial. SDC remains the only type of management that can be administered in case of Space occupying CI with acceptable outcomes.

### KEYWORDS :

#### INTRODUCTION

In 11% to 25% of all cerebellar infarcts ischemic edema becomes space occupying within the posterior fossa, causing brain stem compression and obstructive hydrocephalus.<sup>(1)</sup> Cerebellar edema can result in marked hydrocephalus and brain stem compression by upward transtentorial or tonsillar herniation. Cerebellar infarctions (CIs) without primary brainstem or cerebral involvement are rare; they constitute 1.5 to 8.1% of cases in clinicopathological series.<sup>(5)(6)</sup> 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.<sup>(1)(7)(8)</sup> The recognition of the full spectrum of clinical presentation in cerebellar infarction has occurred since the advent of computed tomography (CT) and magnetic resonance imaging (MRI).<sup>(9)</sup> These techniques have made possible the diagnosis of minimally symptomatic patients as well as the delineation of patterns of clinical-anatomic evolution.

The early clinical stage of cerebellar infarct is characterized by signs from cerebellar dysfunction due to infarction, followed by a stage punctuated by brain stem compression in which the level of consciousness fluctuates, emerging into a final stage of coma. Both brain stem infarction and brain stem compression, caused by the mass effect resulting from oedema, may have similar clinical presentation.<sup>(4)</sup> This distinction remains important in the decision to proceed with occipital craniotomy.

Contemporary medical modalities include steroids, mannitol, barbiturates, and hyperventilation.<sup>(2)</sup> Since antiedemic agents are sometimes ineffective for the acute edema following infarction and surgical decompression by suboccipital craniectomy offers an effective alternative when medical modalities fail.

The exact timing of the surgical procedure in the form of Extra Ventricular Drain insertion and Suboccipital Decompressive craniectomy is debatable. There are no Randomized control trials stating the patient selection.

This study is proposed to review the operated cases and the ones that will be admitted during the course of the study at our institute and formulate a protocol for optimal timing of surgical intervention in the

form of Extra ventricular drain insertion or Sub Occipital Decompressive Craniectomy in patients with Acute Cerebellar Infarcts with mass effect.

#### MATERIAL AND METHODS

Duration of the Study August 2007 to December 2014. Type of Study:- Retrospective & prospective

#### Inclusion Criteria

All the patients irrespective of age and sex admitted and evaluated under departments of Neurology and Neurosurgery at AIMS and diagnosed (on imaging) with acute cerebellar stroke will be included in the study. Patients who have undergone Sub-occipital Decompressive Craniectomy (with the diagnosis of Cerebellar stroke) in the last 8 years (from August 2007) and patient admitted with the diagnosis of cerebellar stroke in the period of August 2012 - December 2014 were included in the study.

#### Exclusion Criteria

Patients with simultaneous multiple infarcts will not be included in the study. Patients who did not complete the treatment at AIMS Kochi will be excluded from the study. Patients with deaths due to unrelated reasons will be excluded from the study.

#### Patient Assessment

All the patients will undergo through clinical assessments (age, symptoms, signs etc.), most importantly Glasgow Coma Scale (GCS), at the time of admission and were closely monitored throughout the stay. Clinical deterioration was considered as decrease in GCS score of 2 points. Follow up imaging was done with respect to the drop in GCS.

#### Radiological Assessment

A tight posterior cranial fossa will be defined according to the criteria proposed by Weisberg, 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).<sup>(64)</sup>

Serial CT was performed at least once before and once after intervention. Likewise, medically treated individuals were scanned three times.

A CT score ranging from 0 to 9 (65) was calculated by adding the following items (Fig. 1):

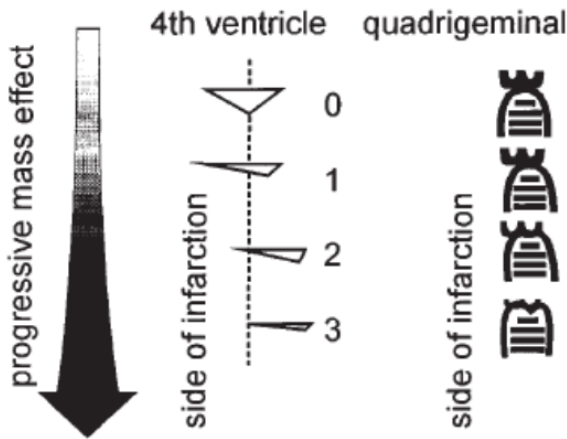


Figure 13<sup>(10)</sup>

1. 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)

**CT scores**

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

For analytical purpose, score of 4-6 and more was considered as presence of mass effect and < 4 was considered as no mass effect.

These patients will be followed by a stroke service team composed of Neurologists, Neurosurgeons. When necessary they will be observed in neurological / surgical intensive care units. All patients will receive antiedema measures (such as mannitol, steroids, and hyperventilation). All the patients will undergo imaging in the form of CT & MRI stroke protocol. The need for EVD insertion and Suboccipital decompressive craniectomy will be evaluated as per the following protocol.

**Data Analysis**

At the end of the study the data regarding the number of patients which needed surgical intervention and the ones which could be managed conservatively will be analyzed. The prognostic factors important for determining the guidelines for surgical intervention will be laid out.

**Statistical applications**

To test the statistical significance of the association between the type of presentation and various radiological and clinical parameters Chi-square test was done. Multivariate logistic regression analysis was done to take into configuration the effect of confounding factors.

**CONSERVATIVE MANAGEMENT**

Guidelines for conservative treatment are derived from those for acute ischemic stroke in general and include airway protection, adequate oxygenation, blood pressure management, management of blood glucose levels, body temperature, and prevention of deep venous thrombosis and pulmonary embolism. (66) (67) The level of consciousness was monitored closely. Immediate imaging was prompted in the case of clinical deterioration (GCS of 2) or occurrence of brainstem signs. Escalation of conservative treatment included ventilation, deep sedation, and other intensive care measures. Medical treatment strategies for increased ICP were in accordance with those used in the management of supratentorial stroke and included osmotic therapy, artificial coma, hyperventilation, barbiturates, buffers, and steroids. (68)(51)

**SURGICAL TREATMENT**

Surgical therapy includes ventriculostomy and suboccipital decompressive craniectomy (SDC). In contrast to the controversial debate about surgical treatment of space-occupying supratentorial stroke, surgical intervention is widely accepted as the treatment of choice in cerebellar stroke with mass effect and strongly recommended in current stroke guidelines. (66)(67)

**Ventricular Drainage**

Ventriculostomy was performed by inserting an extraventricular drain in a lateral ventricle. This procedure represents the gold standard for measuring and managing the ICP. The indication for Extra Ventricular Drain (EVD) insertion was low GCS (<9) or a fall in GCS of >2 with features of early Hydrocephalus & basal cisterns open.

**Procedure Details (EVD)**

For all the patients who underwent EVD insertion, a precoronal twist drill hole was made after identifying the side of more prominent lateral ventricle and the ventricle was tapped. The EVD bag was kept at a height of 10 centimeters for controlled drainage of the cerebrospinal fluid.

**Suboccipital Craniectomy**

The rationale for suboccipital decompressive surgery is to create space for the swollen tissue outside the narrow posterior fossa and thus avoid life-threatening compression of the brainstem and fourth ventricle. There are various surgical approaches: unilateral or bilateral craniectomy, additional opening of the foramen magnum with or without resection of the posterior arch of the atlas, and the use of different types of duraplasty. Necrotic cerebellar tissue is removed, to create extra space within the posterior fossa and reduce development of cytotoxic edema. (71)(51)(52)

**Procedure Details (Suboccipital craniectomy)**

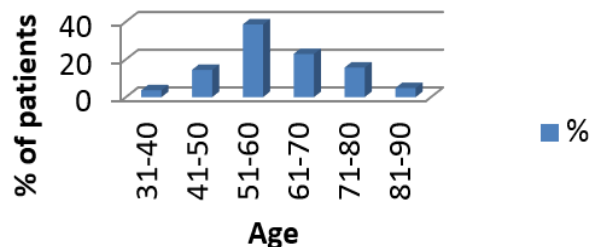
After vertical midline skin incision from the external occipital protuberance to the upper cervical spine, skin and subcutaneous tissue was separated from the underlying fascia, which was cut through laterally to the spinous process of the upper cervical spine, and muscles were disconnected from the spinous process and the occiput. Bilateral craniotomy was performed beneath the transverse sinus and enlarged laterally. Careful opening of the foramen magnum was made to sufficiently decompress the cerebellar tonsils.

Resection of the posterior arch of the atlas was done in order to achieve sufficient decompression. To maximize the decompressive effect, the dura was opened (Y-shaped) above the cerebellar hemispheres and the medulla oblongata. If the effect of decompression seems insufficient, removal of necrotic tissue was considered as an additional option. Finally, the dura was reconstructed by means of duraplasty with pericranial graft.

**RESULTS AND OUTCOME**

The incidence of male and female population was highest in the age group of 51-60 years 38.55%. The number was high for male and females both in the same range.

**Agewise Distribution**



Vomiting was the most common presenting complaint 79.52% and was always associated with vertigo 61.45%, gait disturbance 66.27% or headache 66.27%. A total of 51.81% of patients were drowsy or stuporous at the time of presentation and complete loss of consciousness (coma) was seen only in two patients 2.41%. Other symptoms seen at the time of presentation were double vision 46.99%, slurred speech 39.76%, neck pain 33.73% and limb weakness 22.89%.

Hypertension was the most common co-morbid condition seen in the patients followed by Diabetes Mellitus and Dyslipidemia. Hypertension was associated with one or more co-morbidities in most of the cases. 53% (RHD + DVT) of the patients admitted had history of potentially thrombo-embolic diseases. Majority of the patients 59.04% included in the study presented with a GCS of 13 (group A) or more, 22.89% of the patients were having GCS below 9 (group B) and the rest were either drowsy or stuporous. 18.07 % presented with their GCS ranging from 9 to 12 (group C). The patients were divided according to their presenting GCS:- Group A consisted of patients with GCS 13 or more (awake / obeying) 49 (59.04%). Group B had patients with GCS 9 to 12 (somnolent / drowsy) 19 (22.89%). Group C with patients of GCS less than 9 (Stuporous / Comatose) 15 (18.07%). The initial management was instituted according to the GCS and the mass effect that was calculated at the time of admission. (65) Group A and B patients were given conservative management initially. Group C patients were offered surgical management on the day of admission. Group A & B patients (68 out of 83) were admitted in the ward under close monitoring. They were treated conservatively initially. 26 (38.33%) out of the 68 patients, had further fall in GCS during their course in the ward. 20 (24.09%) of these patients were offered surgical intervention initially in the form of EVD placement, after repeating the imaging. Out of these 20 patients 11 (13.25%) patients showed signs of further deterioration and had to undergo SDC whereas the other 9 (10.8%) could be managed with EVD insertion alone.

The outcome of these patients, managed conservatively, with respect to Rankin Score at discharge was good. Most of these patients were in the group A 42 (50.6%) and 6 (7.22%) were from Group B. 7 (8.43%) patients from the group A showed deterioration and were instituted surgical intervention (5 EVD and 2 SDC), all of whom had good outcome. 13 (15.66%) patients from the group B who needed surgical attention 4 underwent EVD placement and 9 underwent SDC. 3 (3.16%) patients out of the 9 who were in group B further progressed to coma and had poor outcome. There were 15 (18.07%) patients in group C, one patient was managed with EVD insertion and the rest were taken up for SDC without EVD trial. The outcome of these patients (according to the Rankin score) at the time of discharge was good for 9 (60%) out of 15 patients.

## DISCUSSION

The average duration of the patients to get admitted ranged from first to third day, but the fall in GCS was significantly higher on the third day, this can be again compared to the published studies. We also saw that the next major group of the patients who had drop in their GCS was in the first 24 hours. Thus it can be stated that majority of the patients will show fall in GCS in the first three days from the time of onset of symptoms. Patients who have HTN and DM associated with thromboembolic condition are at a higher risk of cerebellar stroke, this was a significant risk factor with respect to immediate outcome. That conservative therapy is the preferred treatment for patients who are alert (36) (69) (70) and in stable clinical condition of the patients who underwent conservative management showed good outcome at discharge. Close monitoring of these patients is needed as, early warning signs of increase in mass effect and ICP can be seen and picked up on imaging and fall in GCS. Patients with CIs exhibit deterioration after variable periods with relatively stable deficits. (54) (13) This is attributable to progressive expansion of cerebellar tissue necrosis, with consequent increases in water uptake and brain edema. (71) When a CI acts as a progressively enlarging mass, signs of brainstem compression or hydrocephalus occur. These two occurrences require immediate surgical treatment. Raco et al also stated that early diagnosis and prompt surgical treatment are mandatory, before the development of irreversible brainstem damage or severe hypertensive hydrocephalus.

More so on the 1st and the 3rd day the patients should be paid particular attention to, but a range of 1st to 7 days was seen in our study for the deterioration of the sensorium. Jutter et al reported the upper limit of drop in GCS and worsening of the mass effect to be 9 days.

Patients with a GCS of 9 to 12 (group B) are the ones who have to be carefully monitored and should be done in ICU. If they don't show any improvement in their GCS in 12 hours from the time of admission in the ICU, CT should be repeated to see presence of mass effect. External Ventricular drain insertion should be offered as the initial form of surgical intervention for patients with impaired consciousness, regarding it as a less-invasive surgical procedure than craniectomy.<sup>(58)</sup>

Any further deterioration or no improvement in the condition of the patient after insertion of the EVD in the next 12 hours should be taken as an indication for SDC.

## CONCLUSION

Patients presenting with GCS equal to or more than 13 can be managed conservatively and should be subjected to serial CT scans at an interval of 12 hours after admission (for first 24 hours) or when they show evidence of fall in GCS (> 2 points of GCS). Findings in CT had good predictive value of clinical deterioration also. Early surgical intervention in patients showing clinical deterioration (fall in GCS) with evidence of mass effect show good clinical outcome. Comatose patients should be managed with SDC without delay. Good outcome was seen in patients a. Who were managed with early surgical intervention within 6 hours of fall in GCS. b. Age < 60 years. Poor outcome was seen in a. Patients with brainstem infarcts b. Age > 60 years. c. Comatose patients (> 6 hours)

## REFERENCES

1. The spectrum of cerebellar infarction. Amerenco P. 1991;41, Neurology, pp. 973-979.
2. Arterial pathology in cerebellar infarction. Amarencu P, Hauw JJ, Gautier JC. 1990, Stroke 21, pp. 1299-1305.
3. Surgical management of acute cerebellar infarction. Cioffi FA, Bernini FP, Punzo. 1985, Acta Neurochir (Wien) 74, pp. 105-112.
4. Surgical and medical management of patients with massive cerebellar infarction: Results of the German-Austrian cerebellar infarction study. Jaus M, Krieger D, Hornig CR, Schramm J, Busse O. 1999, J Neurol, pp. 246:257-264.
5. Infarctions of the brainstem and the cerebellum: A correlation of computed tomography and angiography. Hinshaw DB, Thompson JR, Hasso AN, Casselman ES. 1980, Radiology, pp. 137:105-112.
6. Cerebellar infarction: Comparison of computed tomography and magnetic resonance imaging. Simmons Z, Biller J, Adams HP, Dunn V, Jacoby CG. 1986, Ann Neurol, pp. 19:291-293.
7. Cerebellar infarction with brain stem compression: Diagnosis and surgical treatment. Lehrich JR, Winkler GF, Ojemann RG. 1970;22, Arch Neurol, pp. 490-498.
8. Spontaneous cerebellar strokes: clinical observations in 60 patients. Turgut M, Ozcan OE, Ozcan E, Okay S, Erbenig A. 1996;47, Angiology, pp. 841-848.
9. Acute cerebellar hemorrhage and CT evidence of tight posterior fossa. Weisberg LA. 1986, Neurology, pp. 36:856-860.
10. A computed tomography score for assessment of mass effect in space-occupying cerebellar infarction. Jaus M, Müffelmann B, Krieger D, Zeumer H, Busse O. 2001, J Neuroimaging, pp. Jul;11(3):268-71.
11. Guidelines for the early management of adults with ischaemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council working groups. Adams HP Jr, del Zoppo G, Alberts MJ, Bhatt DL, Brass L, Furlan A, et al. 2007, Circulation, pp. 115:e478-e534.
12. Guidelines for management of ischaemic stroke and transient ischaemic attack 2008. Committee, European Stroke Organisation (ESO) Executive. 2008, Cerebrovasc Dis, pp. 25:457-507.
13. Antiedema therapy in ischemic stroke. Bardutzky J, Schwab S. 2007, Stroke, pp. 38:3084-3094.
14. Long-term outcome after suboccipital decompressive craniectomy for malignant cerebellar infarction. Pfefferkorn T, Eppinger U, Linn J, Birnbaum T, Herzog J, Straube A, et al. 2009, Stroke, pp. 40:3045-3050.
15. Long-term outcome after surgical treatment for space-occupying cerebellar infarction: experience in 56 patients. Jüttler E, Schweickert S, Ringleb PA, Huttner HB, Köhrmann M, Aschoff A. 2009, Stroke, pp. 40:3060-3066.
16. Migraine and vertebrobasilar ischemia. Caplan, L.R. 1991, Neurology, pp. 41:55-61.
17. Temporal profile (clinical course) of acute vertebrobasilar system cerebral infarction. Jones HR, Millikan CH, Sandok BA. 1980, Stroke, pp. 11:173-177.
18. Cerebellar strokes: Mortality, surgical indications and results of ventricular drainage. Shenkin HA, Zavala M. 1982, Lancet, pp. 2:429-432.
19. Cerebellar infarction: natural history, prognosis and pathology. Macdonnell, R.A., Kalnins, R.N. and Donnan, G.A. 1987, Stroke, pp. 19: 847-55.
20. Cerebellar hemorrhage and infarction. RC, Heros. 1982, Stroke, pp. 13: 106-109.
21. Cerebellar infarction: A clinico-pathological study. . Syper GW, Alvord EC Jr. 1975, Arch Neurol, pp. 32:357-363.
22. Management of acute cerebellar infarction: one institution's experience. Raco A, Caroli E, Isidori A, Salvati M. 2003, Neurosurgery, pp. 53:1061-1066.