

Management of Cerebellar Stroke: Factors Affecting Surgical and Non-Surgical Treatment

Tariq Muhammad,^{1*} Nadeem Ahmad Memon¹

ABSTRACT

Objective To present the outcome and factors affecting both surgical and non-surgical decisions in the management of cerebellar stroke.

Study design Descriptive case series.

Place & Duration of study Ziauddin University Hospital Clifton Campus Karachi, from January 2010 to June 2019.

Methodology A group of selected patients with cerebellar hemorrhage and infarct were included in the study. At admission clinical status was recorded using Glasgow coma scale (GCS) score and outcome was measured using Glasgow outcome scale (GOS). The management was both conservative and surgical. The treatment was dependent on correlation of both clinical and radiological findings. No prophylactic intervention was carried out if there were disparity between clinical and radiological findings. Surgical intervention mostly included suboccipital decompressive craniectomy (SDC) with external ventricular drain (EVD). No duroplasty was performed. Follow up period was between 3 to 9 months.

Results A total of 36 patients were included in the study. Only 41% patient required surgery and remaining were managed conservatively. Surgical intervention was predominantly in hematoma patients (66%) as compared to infarct (23%) patients. The Glasgow outcome scale (GOS) was largely between 4 to 5 with minimal disability. Mortality rate was 13.88% in both surgically and conservatively managed patients.

Conclusions In a selected group of patients, surgical intervention is safe and lifesaving if performed timely. Equally good results are predictable with conservative management if instituted wisely.

Key words Cerebellar hemorrhage, Cerebellar infarct, Decompressive craniectomy, External ventricular drain.

INTRODUCTION:

Cerebellar hemorrhages comprise about 10% of all intracerebral hemorrhages, while cerebellar ischemic stroke accounts for about 3% of all cerebral ischemic

strokes.¹⁻³ Cerebellar infarctions are approximately two-thirds as common as cerebellar hemorrhage hence cerebellar hemorrhages are about 15% of all cerebellar strokes.⁴ In around 36% to 89% cases the identifiable risk factor is hypertension.⁵

¹ Department of Neurosurgery, Ziauddin University Hospital (Clifton Campus) Karachi

Correspondence:

Dr. Tariq Muhammad ^{1*}
Department of Neurosurgery
Ziauddin University Hospital (Clifton Campus)
Karachi
E mail: tariqm khan2001@hotmail.com

Management of cerebellar stroke either hemorrhagic or ischemic has been a hot topic of debate since long time among individuals and institutions. The posterior fossa, being the most confined space with the delicate contents like brain stem, even slight damage of which can result in catastrophic outcomes, making the decision more serious and crucial between surgical versus non-surgical management. Latest neuroradiological diagnostic tools like CT

scan and MRI has made the diagnosis very easy and accurate but surgical or non-surgical decisions are closely related to both clinical and radiological findings. Failure to correlate radiological and clinical findings results in poor outcomes. With time, suboccipital decompressive craniectomy with or without external ventricular drain insertion have become the main surgical options for patients requiring surgical intervention.⁶ Literature is heavily loaded with low to high thresholds for surgical decompression with favorable results.⁷ No uniform rigid criteria or guidelines for surgical or nonsurgical treatment has ever been laid down.⁸

Straightforward decisions are made in two groups of patients; one group with both satisfactory clinical presentation and radiological findings with no indication for surgical intervention, while the second group with positive clinical and radiological findings with clear indications for surgery. The third group is always a challenge where there is disparity between clinical scenario and radiological findings. This study was conducted to find the outcome of managing cerebellar stroke with already laid down and widely practiced parameters for both conservative and surgical management at our setup.

METHODOLOGY:

This descriptive case series was conducted at Ziauddin University Hospital Clifton Campus Karachi. A total of 36 cerebellar stroke patients who were managed from January 2010 to June 2019 were included. There were 21 cases of cerebellar infarct and 15 cases with cerebellar hemorrhages. Glasgow coma score was recorded at the time of admission and for further neurological assessment. Follow up period was from 3 months to 9 months. Postoperative recovery was assessed by using Glasgow outcome scale. All those cerebellar hemorrhages were excluded from the study if they were secondary to arteriovenous malformations (AVMs), aneurysms, tumors, blood dyscrasias and trauma. Strokes with brain stem extension were also excluded from the study. GCS 3 with dilated and nonreactive pupils were excluded from the study.

Criteria for surgical intervention were deteriorating neurological status with radiological evidence of stroke either infarct or clot with mass effect, evident by 4th ventricle effacement or occlusion with acute hydrocephalus. Fourth ventricle compression was divided into three groups as described by Kirollos et al where Group I mean normal size and configuration, Grade II was partially compressed and shifted, and Grade III was completely obliterated.⁵ Grade III always required surgery as it was associated with acute hydrocephalus and

deteriorating neurological status, while stable Grade I and II were managed conservatively with the Grade II at high risk of deterioration. Grade II patients had some early signs of hydrocephalus with occasional complaints of headache and vomiting but with intact level of consciousness. Early signs of acute hydrocephalus were defined as loss of normal crescentic or triangular shape of temporal horns, blunting of frontal horns with loss of their sickle shape but without clear ballooning and with no signs of transependymal seepage, loss of slit shape of third ventricle but no clear rounding and effacement of 4th ventricle without complete occlusion. While enlarged temporal horns, ballooning of third ventricle and ballooned frontal horns with transependymal seepage and almost invisible 4th ventricle (Grade III) were taken as clear signs of established hydrocephalus.^{9,10}

All patients with cerebellar stroke were treated according to the worldwide practiced guidelines. Active and alert patients with cerebellar hemorrhage diameter less than 4 cm and without radiological evidence of clear and established hydrocephalus were treated conservatively. While unconscious patients or patients with deteriorating consciousness level and hemorrhage diameter of more than 4 cm were treated surgically. A frontal EVD was inserted and clot or necrotic brain tissue was decompressed with SDC without duroplasty or bone fixation. Decompression was considered satisfactory with evident brain parenchymal pulsations. In patients with hemorrhage size less than 4 cm with 4th ventricular extension and hydrocephalus, were treated only with EVD insertion without SDC. All those patients with 4th ventricle effacement without complete occlusion, with early signs of hydrocephalus and GCS 13 to 15 were managed conservatively till their improvement or surgical intervention after further deterioration.

Active and alert patients without hydrocephalus with cerebellar infarct were treated conservatively irrespective of the size of infarct. While patients with 4th ventricle occlusion and acute hydrocephalus were treated surgically by placement of EVD and performing SDC. Patients with GCS 13 to 15 without complete 4th ventricle occlusion and with signs of early hydrocephalus were managed conservatively till their recovery or surgery in case of further worsening of their clinical status.

Postoperatively, patients were kept on control mode ventilation for 24 to 72 hours. EVD was set to open to drain at 15 to 20 mmHg above external auditory meatus in supine or from mid sagittal line between

| Management Plan | No. of Patients (n) | Glasgow Outcome Score | | | |
|-----------------|---------------------|-----------------------|---------|-----|-----|
| | | (1) | (2 - 3) | (4) | (5) |
| Conservative | 5 | - | - | 1 | 4 |
| EVD only | 2 | 1 | - | - | 1 |
| SDC+EVD | 8 | 1 | 1 | 2 | 4 |

GOS = (1), death; (2), persistent vegetative state; (3), severe disability; (4), moderate disability; (5), low disability

| Management Plan | No. of Patients (n) | Glasgow Outcome Score | | | |
|-----------------|---------------------|-----------------------|---------|-----|-----|
| | | (1) | (2 - 3) | (4) | (5) |
| Conservative | 16 | 2 | - | 2 | 12 |
| EVD only | - | - | - | - | - |
| SDC+EVD | 5 | 1 | - | 1 | 3 |

GOS = (1), death; (2), persistent vegetative state; (3), severe disability; (4), moderate disability; (5), low disability

both eyebrows in lateral position without any fear of reverse cerebellar herniation.^{11,12} Within 3 to 7 days EVD was taken out after checking for development of hydrocephalus with serial CT scan brain in 12 to 48 hours, either by closing the drain or by elevating the level of drainage. Low molecular weight heparin was started within 24 to 48 hours in cases of cerebellar infarct.

RESULTS:

A total of 36 patients with cerebellar stroke were included in the study. Twenty-one (58.33%) patients had cerebellar infarct and 15 (41.66%) with hematoma. Mean age in both groups was 69 year. Glasgow coma score at the time of admission was recorded. They were divided into two groups. Eleven patients (30.55%) had GCS 4 to 12 and 25 patients (69.44%) with GCS 13 to 15. CT scan and MRI brain were the main tools for diagnosis. Two (5.55) patients showed vermian cerebellar hematomas with 4th ventricular extension and hydrocephalus without posterior fossa mass effect.

Eleven (30.55%) patients with cerebellar stroke (clot=6, infarct=5 patients) were operated with SDC and EVD due to their low level of GCS and massive hydrocephalus at the time of presentation. External ventricular drain as a sole surgical procedure was used in 2 (5.55%) patients with cerebellar hematoma with 4th ventricular extension and hydrocephalus but without local mass effect (table I). Both patients were initially managed conservatively. No EVD alone was used in cerebellar infarct patients and they were either managed conservatively or with both EVD and SDC together.

Twenty-three (63.88%) patients with both types of cerebellar strokes were managed conservatively whose GCS at presentation was between 13 to 15 either with no signs of brainstem compression or with early signs of acute hydrocephalus. Among these, 16 (69.56%) patients were from cerebellar infarct group and they received mannitol and steroids to control the posterior fossa swelling (table II). Two patients required EVD and SDC after 3 to 5 days of incident when their GCS dropped. They both were from hematoma group. Total 10 (66.66%) patients required surgery among 15 patients with cerebellar hemorrhage and 5 (23.80%) required surgery among 21 patients with cerebellar infarct. No cerebral ischemic infarct patient required delayed surgical intervention.

Outcome was assessed with Glasgow Outcome Scale. In both conservatively and surgically managed patients the GOS was largely between 4 to 5. One patient among operated cerebellar hematoma patients with SDC and EVD remained severely disabled even after more than two years of rehabilitation. Two (5.55%) patients died in hematoma patients; one patient died of multi-drug resistant bacterial ventriculitis after EVD insertion and the other had massive cerebellar edema even after decompressive craniectomy and EVD insertion. Three (8.33%) patients died in cerebellar infarct patients; two among conservative group due to extensive brain stem extension of infarct and one patient died of myocardial infarct in operated patients after a month of good recovery. Two patients required permanent ventriculoperitoneal shunts after 6 to 8 weeks. They both had cerebellar hematoma.

DISCUSSION:

The management of cerebellar strokes whether hemorrhage or infarct is completely different from supratentorial strokes. The aim of management in this situation is to protect brainstem direct damage, tonsillar herniation due to raised posterior fossa pressure and trans-tentorial herniation indirectly due to severe hydrocephalus. Several pathoanatomical studies explain the fact that in many cases cerebellar strokes do not cause any structural damage in brain stem, and similarly supratentorial telencephalic structures are otherwise intact, hence there are always good chances for intellectual and cognitive functions.^{13,14} That is why numerous case studies revealed good outcome even with GCS 3 or 4 with nonreactive pupils.^{4,15,16} In our study one patient with GCS 4 and pinpoint pupils responded well to suboccipital decompressive craniectomy and external ventricular drain to full cognitive functions after one month but later on died of myocardial infarction. But this fact has always been challenged by other large studies where poor GCS is always a predictor of poor outcome.¹⁷ The possibility of good outcome in very poor Glasgow coma score is probably possible only in highly selected cases where brain stem injury and cerebral/cerebellar herniations have been ruled out which is sometime practically impossible.

A clear criteria for conservative treatment is a patient with cerebellar clot size less than 4 cm (while no size matters in case of cerebellar infarct) with no hydrocephalus and intact level of consciousness; while clot size more than 4 cm with hydrocephalus and deteriorating level of consciousness is an indication for surgical decompression and external ventricular drain placement.¹⁶ Critical observation and decision making are required in patients with headache, nausea and vomiting with intact level of consciousness and certain degree of mass effect of clot or infarct. This group of patients is a challenge for the surgeon as neither CT scan nor clinical status could predict the exact path for surgical or non-surgical management. We kept this third group in intensive care unit under critical observation till the time their 4th ventricle opened, and hydrocephalus settled, or they end up in surgery. The ICU stay was 3 to 12 days.

Majority (27.77%) of our patients who required surgery immediately or after deterioration within first few days were among cerebellar hemorrhage group as compared to patients with infarcts (13.88%). This is a common finding among other studies as well and emphasize that patients with cerebellar hemorrhage needs more intensive monitoring as

compared to ischemic cerebellar stroke.^{11,18}

It is quite widely accepted that preoperative clinical status predicts the postoperative outcome.^{5,19-22} Those patients who are brought in coma (delayed arrival) or picked up late during neuro-observation are probably the poor candidates with poor outcome. Because of this consistent finding of poor outcome with patients in poor GCS, neurosurgeons took more aggressive surgical decisions while patients were in their good clinical status. In a series by Ott et al, the mortality was 17% in patients who were conscious at the time of surgery as compared to 75% who were unconscious.²⁰ Similarly, in another study mortality was 60% in patients whose arrival GCS was 6 and mortality was only 20% in patients with arrival GCS more than 10.²³ But most studies resist preventive surgeries where the patients not deteriorated. They found no benefit of preventive surgeries as in their practice excellent results were met even after their neurological deterioration.^{7,19} We operated on only those patients where both clinical and radiological indications coexisted.

We used EVD only as surgical procedure in patients with cerebellar bleed with intraventricular extension and hydrocephalus without any mass effect. Otherwise it was a concomitant procedure with SDC. This was because of the fear of upward herniation of cerebellar tissue in our practice. While some of the authors used EVD alone in majority of their patients where SDC was clearly indicated without any fear of cerebellar upward herniation.^{24,25} In the series of Mathew et al no patient required external ventricular drainage or delayed shunt placement after initial treatment with proper suboccipital decompressive craniectomy.¹¹ We followed the rule that EVD alone can decrease hydrocephalus but cannot decrease direct physical pressure of clot or infarct on the brain stem.²⁴ Hence we have never used EVD alone where there were clear indications for SDC.

Poor outcome has always been reported in groups of non-selected patients with predominant conservative approach. In one series timely surgical intervention saved 67% lives with 20% mortality rate as compared to >80% mortality in other studies with too much conservative approach.^{14,25} In our highly selected group of patients the mortality rate was 13.88%.

CONCLUSIONS:

The overall outcome is very promising in a carefully selected group of patients with cerebellar stroke in both surgical and nonsurgical management if applied logically. Unnecessary delay in surgery and very

aggressive surgical approach can further harm the health and life of the patients with cerebellar stroke.

REFERENCES:

1. Flaherty ML, Woo D, Haverbusch M, Sekar P, Khoury J, Sauerbeck L, et al. Racial variations in location and risk of intracerebral haemorrhage. *Stroke*. 2005;36:934-7.
2. Savitz SI, Caplan LR, Edlow JA. Pitfalls in the diagnosis of cerebellar infarction. *Acad Emerg Med*. 2007;14:63-8.
3. Edlow JA, Newman-Toker DE, Savitz SI. Diagnosis and initial management of cerebellar infarction. *Lancet Neurol*. 2008;7:951-64.
4. Heros RC. Cerebellar hemorrhage and infarction. *Stroke*. 1982;13:106-9.
5. Kirollos RW, Tyagi AK, Ross SA, van Hille PT, Marks PV. Management of spontaneous cerebellar hematomas: A prospective treatment protocol. *Neurosurgery*. 2001;49:1378-86.
6. Adams HP Jr, del Zoppo G, Alberts MJ, Bhatt DL, Brass L, Furlan A, et al. Guidelines for the early management of adults with ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups: The American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists. *Circulation* 2007;115: e478-e534.
7. Jauss M, Krieger D, Hornig C, Schramm J, Busse O: Surgical and medical management of patients with massive cerebellar infarctions: results of the German-Austrian Cerebellar Infarction Study. *J Neurol*. 1999;246:257-64.
8. Pfefferkorn T, Eppinger U, Linn J, Birnbaum T, Herzog J, Straube A, et al. Long-Term Outcome after suboccipital decompressive craniectomy for malignant cerebellar infarction. *Stroke*. 2009;40:3045-50.
9. Rekate HL, Blitz AM. Hydrocephalus in children. *Hand Clin Neurol*. 2016;136:1261-73.
10. Mathew P, Teasdale G, Bannan A, Oluoch-Olunya D: Neurosurgical management of cerebellar haematoma and infarct. *J Neurol Neurosurg Psychiatr*. 1995;59:287-92.
11. Broderick JP, Adams HP Jr, Barsan W, Feinberg W, Feldmann E, Grotta J, et al. Guidelines for the management of spontaneous intracerebral hemorrhage: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. *Stroke*. 1999;30:905-15.
12. Raco A, Caroli E, Isidori A, Salvati M. Management of acute cerebellar infarction: one institution's experience. *Neurosurgery*. 2003;53:1061-6.
13. Sybert GW, Alvord EC Jr: Cerebellar infarction. A clinicopathological study. *Arch Neurol*. 1975;32:357-63.
14. Heros RC. Surgical treatment of cerebellar infarction. *Stroke*. 1992;23:937-8.
15. Yanaka K, Meguro K, Fujita K, Narushima K, Nose T. Immediate surgery reduces mortality in deeply comatose patients with spontaneous cerebellar hemorrhage. *Neurol Med Chir (Tokyo)*. 2000;40:295-300.
16. Kobayashi S, Sato A, Kageyama Y, Nakamura H, Watanabe Y, Yamaura A. Treatment of hypertensive cerebellar hemorrhage -surgical or conservative management? *Neurosurgery*. 1994;34:246-51.
17. St. Louis EK, Wijdicks EFM, Li H, Atkinson JD. Predictors of poor outcome in patients with a spontaneous cerebellar hematoma. *Can J Neurol Sci*. 2000;27:32-6.
18. Auer LM, Auer T, Sayama I. Indications for surgical treatment of cerebellar haemorrhage and infarction. *Acta Neurochir*. 1986;79:74-9.
19. Dammann P, Asgari S, Bassiouni H, Gasser T, Panagiotopoulos V, Gizewski ER, et al. Spontaneous cerebellar hemorrhage-

- experience with 57 surgically treated patients and review of the literature. *Neurosurg Rev.* 2011;34:77-86.
20. Ott KH, Kase CS, Ojemann RG, Mohr JP. Cerebellar hemorrhage: diagnosis and treatment. A review of 56 cases. *Arch Neurol.* 1974;31:160-7.
21. Khan I, Janjua MB, Khatri IA, Nadeem M. Surgical decompression in massive cerebellar stroke. *Rawal Med J.* 2010;35:96-100.
22. Al Safatli D, Guenther A, McLean AL, Waschke A, Kalff R, Ewald C. Prediction of 30-day mortality in spontaneous cerebellar hemorrhage. *Surg Neurol Int* 2017;8:282. doi: 10.4103/sni.sni_479_16.
23. Donauer E, Loew F, Faubert C, Alesch F, Schaan M. Prognostic factors in the treatment of cerebellar haemorrhage. *Acta Neurochir (Wien).* 1994;131:59-66.
24. Seelig JM, Selhorst JB, Young HF, Lipper M. Ventriculostomy for hydrocephalus in cerebellar haemorrhage. *Neurology.* 1981;31:1537-40.
25. Horwitz NH, Ludolph C. Acute obstructive hydrocephalus caused by cerebellar infarction: Treatment alternatives. *Surg Neurol.* 1983;20:13-9.
- Received for publication: 01-10-2019
Accepted after revision: 30-11-2019
- Author's Contributions:
Tariq Muhammad: Study design, data collection and analysis, manuscript writing.
Nadeem Ahmad Memon: Study design, data collection and analysis, manuscript writing.
- Conflict of Interest:
The authors declare that they have no conflict of interest.
- Source of Funding:
None
- How to cite this article:
Muhammad T, Memon NA. Management of cerebellar stroke: Factors affecting surgical and non-surgical treatment . *J Surg Pakistan.* 2019;24 (3):116-21. Doi:10.21699/jsp.24.3.3.