

## **Case Series**

### **Surgical decompression in massive cerebellar stroke**

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## **ABSTRACT**

### **Objective**

To present the local experience of decompressive surgery in massive cerebellar strokes at Shifa International Hospital, Islamabad.

### **Patients and Methods**

This retrospective non-randomized case series included six patients operated at Shifa International Hospital, Islamabad from October 2003 to July 2008 for cerebellar stroke. Decompression with or without external ventricular drain was performed. Functional status at follow up was evaluated by Glasgow Outcome Scale.

### **Results**

Out of six patients with cerebellar stroke, one had cerebellar hemorrhage while others had cerebellar infarction. Mean age was 60 years and male to female ratio was 1:1. In three cases ventricular drainage with posterior decompressive craniectomy was done. All were operated when they developed deterioration in conscious level. Five patients were discharged with Glasgow Outcome Scale of 3 to 5 (severe disability to good recovery). One patient who died had simultaneous brain stem involvement.

### **Conclusion**

Early decompressive surgery is life saving for massive cerebellar strokes, while concomitant brainstem involvement carries poor prognosis. All patients with cerebellar stroke should be co-managed by neurology and neurosurgery services in the initial phase to help with quick intervention, if indicated. (Rawal Med J 2010;35: ).

### **Keywords**

Cerebellar stroke, decompressive surgery, external ventricular drain.

## **INTRODUCTION**

Acute cerebellar stroke can present with severe headache, nausea, vomiting, vertigo, ataxia, and dysarthria. It may initially be clinically indolent and can quickly deteriorate into life-threatening catastrophe, whereas some patients can present in a moribund comatose state.<sup>1</sup> Surgical intervention may be necessary if mass effect develops.<sup>2</sup> Cerebellar stroke, therefore, requires constant vigilance in decision making to yield better outcome. It is often difficult to determine whether ensuing coma results from potentially reversible causes such as evolving hydrocephalus, brain stem compression by mass effect or by the irreversible brainstem infarction.<sup>1</sup> The overall risk of poor outcome depends on the level of consciousness and hence it is the most powerful indicator for decisive surgery, superior to any other clinical sign.<sup>2</sup> In one series, patients with acute cerebellar hemorrhage who developed neurological deterioration, 90% of cases treated with surgical decompression had promising outcome as compared to medically treated patients, of whom 75% died within one week.<sup>3</sup> In the same series, patients with acute cerebellar infarction who deteriorated acutely, 88% undergoing decompressive craniectomy survived, whereas only 20% medically treated patients survived.<sup>3</sup> Age more than 60 years, low Glasgow Coma Scale (GCS) and initial brainstem involvement have fatal or disabling outcome.<sup>4</sup> Aim of this study was to report our experience of decompressive surgery in massive cerebellar strokes at our institution.

## **PATIENTS AND METHODS**

The study included six adult patients with massive cerebellar stroke operated for cerebellar decompression between October 2003 to July 2008 in Shifa International Hospital, Islamabad, Pakistan. Families were educated about the risks and benefits of decompressive surgery and informed consent was obtained. All were monitored in high dependency or intensive care units after admission and were operated as soon as they developed progressive deterioration of consciousness.

Cerebellar decompressive craniectomy with or without external ventricular drainage (EVD) was performed. In decompressive surgery occipital bone was removed ipsilateral to the stroke. Duramater was opened, necrotic tissue or hemorrhage was evacuated and then duraplasty was done. All patients were initially co-managed by the neurologist, neurosurgeon and intensivist. Those patients whose neuroimaging showed hydrocephalus had ventricular drainage done either through external ventricular drain or ventriculoperitoneal shunt (VP shunt). GCS was monitored before and after the surgery. Neurological assessment was done and functional outcome was measured using Glasgow Outcome Scale (GOS), in which higher scores indicate good outcome.

## **RESULTS**

The demographic and clinical characteristics of patients are summarized in table 1. Case summaries of all 6 patients are provided below.

**Patient 1:** A 55 year old woman presented with sudden onset of vomiting and drowsiness on the day of presentation. Patient was known hypertensive who was not taking medications. On

examination her blood pressure was 220/130 mm Hg. She was drowsy, localized to pain and both plantars were up going and GCS was 11/15. An urgent CT scan of brain showed left cerebellar hemispheric hemorrhage with hydrocephalus (Fig 1a). Due to impending mass effect and decreasing level of consciousness, posterior cerebellar decompressive craniectomy was performed with external ventricular drainage to relieve hydrocephalus (Fig 1b). Patient had a prolonged and complicated hospital course with development of deep vein thrombosis and sepsis which was appropriately treated. She required tracheostomy and was discharged on 43<sup>rd</sup> day of surgery. Tracheostomy was closed at 2 months and her GOS was 3.

**Patient 2:** A 42 year old woman presented with the sudden onset of blurred vision, diplopia, decreased vision, vertigo and 7 episodes of projectile vomiting. She had started using oral contraceptive pills for 4 months prior to developing symptoms. Examination showed blood pressure of 146/86 mm Hg. She had symmetric, reactive pupils, nystagmus with fast component towards the right side. Past pointing and finger nose ataxia was present on right side with GCS of 12/15. An MRI of brain showed acute large infarct in right cerebellum with mass effect on the brain stem, effacement of fourth ventricle and mild obstructive hydrocephalus (Fig 2a). She had worsening of GCS during hospitalization and underwent posterior decompressive craniectomy with EVD. Post-operative CT scan showed reappearance of 4<sup>th</sup> ventricle and decrease in mass effect (Fig 2b). She showed improvement in immediate post operative period, was mobilized early and discharged on 9<sup>th</sup> day of admission. At 3 weeks follow up visit, she was found to have wound infection with methicillin sensitive staphylococcus aureus, which was treated conservatively and she recovered completely. Her GOS at six weeks follow up was 5.

**Patient 3:** A 75 year old woman with previous history of uncontrolled hypertension presented to the emergency with sudden onset of drowsiness, slurring of speech, and vomiting. On examination her blood pressure was 190/120 mm Hg and GCS was 11/15. She was lethargic, had diplopia and nystagmus. CT scan of head showed right cerebellar hemisphere infarct with minimal hydrocephalus. On deterioration in level of consciousness, right posterior fossa craniectomy was done with evacuation of necrotic tissue. She made uneventful recovery with discharge on 8<sup>th</sup> day of surgery. At 2 weeks follow up her GOS was 4.

**Table1. Demographic and clinical characteristics of patients.**

	<b>Patient 1</b>	<b>Patient 2</b>	<b>Patient 3</b>	<b>Patient 4</b>	<b>Patient 5</b>	<b>Patient 6</b>
<b>Gender</b>	Female	Female	Female	Male	Male	Male
<b>Age</b>	55	42	75	45	70	71
<b>Type of stroke</b>	Hemorrhagic	Ischemic	Ischemic	Ischemic	Ischemic	Ischemic
<b>Side of stroke</b>	Left	Right	Right	Right	Right	Left
<b>GCS at presentation</b>	11/15	12/15	11/15	8/15	10/15	10/15
<b>Blood Pressure at presentation (in mm Hg)</b>	220/130	146/86	190/120	150/70	150/80	150/90
<b>Risk Factors</b>						
<b>Dyslipidemia</b>				Yes		
<b>Oral contraceptive use</b>		Yes				
<b>Hypertension</b>	Yes		Yes			
<b>Diabetes mellitus</b>					Yes	
<b>Ischemic heart disease</b>					Yes	Yes
<b>Atrial fibrillation</b>					Yes	
<b>Developed hydrocephalus</b>	Yes	Yes	Yes	Yes	Yes	Yes
<b>Indication for surgery</b>	*	*	*	*	*	*
<b>Surgical procedure</b>	**	**	** *	** *	****	***

<b>Post-operative complications</b>	deep vein thrombosis sepsis tracheostomy	MSSA surgical site infection	Nil	Acute ischemic changes in pons & cerebellum  death	MRSA infection tracheostomy	Nil
<b>Glasgow outcome scale</b>	3	5	4	1	4	4

\*decreased level of consciousness, \*\*posterior decompression with external ventricular drain, \*\*\*posterior decompression without external ventricular drain, \*\*\*\*posterior decompression with ventriculoperitoneal shunt, MSSA – methicillin sensitive Staphylococcus aureus, MRSA – methicillin resistant Staphylococcus aureus

**Patient 4:** A 45 year old man presented to the emergency room with the complaint of headache and rapid deterioration in conscious level. He had no known co-morbidity. Examination showed a blood pressure of 150/70 mm Hg. Neurological examination showed GCS of 8/15, with bilateral symmetric pupils, and bilateral up going plantar responses. He was intubated and an urgent CT scan was done which showed massive right cerebellar infarct with obliteration of cistern and concomitant hydrocephalus. His total cholesterol was 288 mg/dL and LDL of 211 mg/dL. After initial stabilization, he had urgent decompressive craniectomy done with removal of infarcted tissue. A post surgical MRI scan showed acute ischemic changes in right cerebellar hemisphere as well as pons. He did not recover and died on day 6 of admission.

**Patient 5:** A 70 year old man presented with vertigo, dizziness, slurred speech and decreasing level of consciousness. He had diabetes mellitus, atrial fibrillation and ischemic heart disease. His blood pressure was 150/80 mm Hg with a GCS of 10/15. A CT scan showed right cerebellar infarct with mild hydrocephalus. A follow up CT scan showed interval increase in the size of the infarct and compression of 4<sup>th</sup> ventricle with marked increase in hydrocephalus. A ventriculoperitoneal shunt was inserted. Three days later, he had worsening in neurological status, therefore an urgent posterior fossa craniectomy was done with evacuation of infarcted tissue. He had very slow post-operative recovery, therefore a tracheostomy was performed. He also developed multiorganism sepsis which was treated with appropriate antibiotics. He had prolonged hospitalization and was discharged on 41<sup>st</sup> day. He had a follow up at 3 months of surgery and had a GOS of 4.

**Patient 6:** A 71 year old man presented with sudden onset of headache, projectile vomiting, vertigo, and progressive drowsiness. He had history of hypertension and ischemic heart disease and had a right hemispheric stroke 11 years ago. His blood pressure was 150/90 mm Hg with GCS of 10/15 and ataxia on left. Initial CT scan showed an old right temporoparietal infarct. He suddenly deteriorated on 3<sup>rd</sup> day of admission. A follow up CT scan showed left cerebellar infarct with compression of 4<sup>th</sup> ventricle and hydrocephalus. He underwent urgent posterior

fossa craniectomy with evacuation of cerebellar infarct. He was discharged on 9<sup>th</sup> day and a follow up at 2 weeks showed a GOS of 4.

## **DISCUSSION**

Most of the patients with posterior fossa stroke make good recovery, however, certain patients may have fatal outcome secondary to mass effect on brainstem or development of hydrocephalus. The management choices include conservative management or decompression to relieve hydrocephalus or evacuation of lesion to relieve both local effects and hydrocephalus. Management that is either delayed or an unnecessary intervention may result in worse outcome.<sup>5</sup> Most of the literature is about decompressive surgery in cerebellar hemorrhage with little information is available on cerebellar infarcts. Taneda et al<sup>6</sup> described 3 patients groups leading to coma that vary by their clinical presentation, course and neuroradiologic appearance: (1) Alert at onset, with paroxysmal cerebellar symptoms, signs and subsequent stabilization for 10 to 36 hrs, and then rapid and progressive deterioration in 2 to 18 hours (caused by direct brainstem compression by the expanding hematoma or edema). (2) Sudden cerebellar symptoms with rapid deterioration of consciousness, representing hemorrhage into infarct. (3) Sudden coma within hours of onset, without substantial further deterioration (caused by extensive brainstem infarction at the time of cerebellar hemorrhage or infarction).

Several retrospective studies have analyzed clinical and imaging features that are predictive of poor outcome in cerebellar hemorrhage, these include hematoma size more than 3 cm,<sup>7</sup> decreased level of consciousness and large size of the lesion,<sup>8</sup> systolic blood pressure more than 200 mm Hg, bilateral gaze palsy and moderate hydrocephalus.<sup>9</sup> Our patient 1 presented with the systolic blood pressure of 220 and CT scan brain showed left cerebellar hemorrhage with impending hydrocephalus and posterior cerebellar decompression was done with EVD as soon as she developed decreased level of consciousness. A widely accepted neurosurgical adage is to evacuate a cerebellar hemorrhage that is more than 3 cm in cross-sectional diameter by CT scan.<sup>1,10</sup> Our patient 1 also had a large cerebellar hemorrhage, and after surgery patient made rapid recovery with decrease in mass effect.

In large series predictors of disability include a hospital admission systolic blood pressure > 200 mm Hg, a hematoma size >3cm in diameter, visible brainstem distortion and acute hydrocephalus.<sup>11</sup> Predictors of death were abnormal corneal reflex and oculocephalic reflex, a GCS score <8, motor response less than localization to pain, acute hydrocephalus and intraventricular hemorrhage on CT scan.<sup>11</sup> All of our patients were operated due to poor initial or worsening level of consciousness. Five of our patients initially presented with GCS of >8 but deteriorated in hospital. All of our patients had hydrocephalus either on first or subsequent neuroimaging done on deterioration of level of consciousness. Only 1 patients presented with blood pressure >200 mm Hg. The patient that died in our series had concomitant brainstem infarction. Age younger than 70 years is considered predictor of good outcome.<sup>1</sup> Three of our patients were more than 70 years of age.

After adjustment of confounding factors, coma on hospital admission as (GCS <8), intraventricular hemorrhage and infarct extension to the brainstem were independently predictive of death.<sup>1,11</sup> Our patient 4 who died had GCS of 8 along with brainstem involvement. His preoperative CT scan brain showed massive right cerebellar infarct obliteration of cisterns and concomitant hydrocephalus and post operative MRI brain showed acute ischemic changes in right cerebellar hemisphere as well as pons. Infarct extension to the brain stem remains paramount in predicting outcome even after timely salvage decompression.

Surgical resection of necrotic tissue was reserved 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.<sup>12</sup> Long term functional outcome systematically analyzed by Pfefferkorn et al<sup>13</sup> also shares the comparable results. All 57 patients had decompression done. Five lost follow up but out of 52 patients, 21 (40%) lived functionally independent, 4 (8%) lived with major disability and 21 (40%) had died. Like wise of our results, the presence of additional brain stem infarction was associated with poor outcome. Quality of life in survivors was moderately lower than in healthy controls.

There are many retrospective studies and a few prospective trials that are helpful in guiding clinical decision making. In the absence of data from a randomized controlled trial, a reasonable frame work for patient care has been suggested by Kirolos et al,<sup>14</sup> who treated 50 consecutive patients using a protocol based on appearance of fourth ventricle and GCS score. If the fourth ventricle was completely effaced, the patient underwent surgical evacuation and ventricular drainage as our patients 2, 4 and 6 had 4<sup>th</sup> ventricle effacement and obliteration of basal cistern on initial CT scan brain while patient 5 developed 4<sup>th</sup> ventricle effacement on follow up CT scan. If the fourth ventricle appeared normal, the patient was treated conservatively, unless the GCS score deteriorated as was seen in our patient 3 who had no involvement of 4<sup>th</sup> ventricle but all were operated upon as they got deterioration in conscious level and GCS score.

## **CONCLUSIONS**

Early decompressive craniectomy with or without external ventricular drainage in our patients resulted in good outcomes. Involvement of brain stem was associated with poor outcome. All patients with acute cerebellar strokes should be monitored carefully and neurosurgical services should be involved early in the care of patient. Any clinical or radiological signs of worsening should lead to early decompression.

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Received: March 8, 2010 Accepted: April 8, 2010

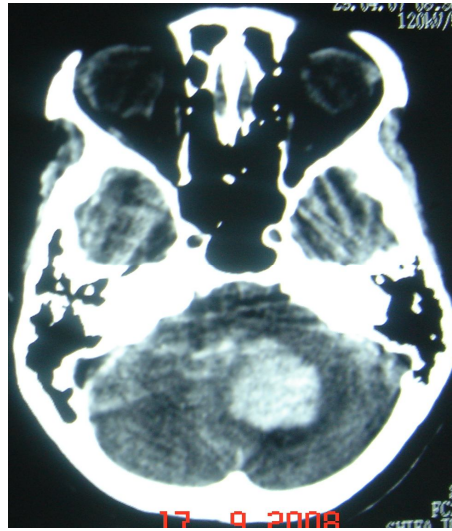
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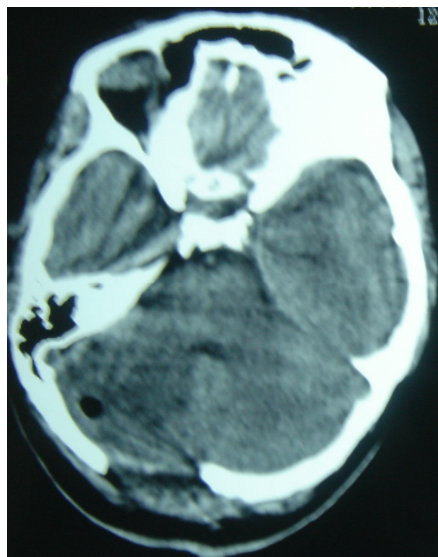
**Fig 1a. CT scan brain showing large left cerebellar and vermian hemorrhage with compression of 4<sup>th</sup> ventricle.**



**Fig 1b. Post operative CT scan brain showing evacuation of hemorrhage with reappearance of 4th ventricle.**



**Fig 2a. MRI brain showing large right cerebellar infarction with complete obliteration of 4th ventricle.**



**Fig 2b. CT head showing decompression of right cerebellar stroke with reappearance of 4th ventricle and decrease in pressure effect.**

