

## Traumatic Cerebellar Hematoma: A Tertiary Care Experience of 23 Conservatively Managed Cases

### Abstract

**Context:** Traumatic cerebellar hematomas are rare in comparison to nontraumatic cerebellar hematomas. **Aims:** The aim of this study is to evaluate the prognostic factors and outcome determining factors with regard to conservatively managed isolated traumatic cerebellar hematoma. **Settings and Design:** Retrospective cohort study. **Materials and Methods:** A retrospective study of 23 patients of conservatively managed isolated posterior fossa hematoma, admitted between August 2018 and May 2020, was conducted in the Department of Neurosurgery, Institute of Medical Sciences, Banaras Hindu University, Varanasi. Each of the patients was evaluated in terms of age, sex, mode of injury, clinical presentation, comorbidity, the severity of injury, best motor response, Glasgow Coma Scale (GCS) at admission, computed tomography findings, and Glasgow Outcome Scale at discharge/death. **Statistical Analysis:** Chi-square test and unpaired *t*-test were used.  $P < 0.05$  was deemed statistically significant. **Results:** Mean volume of posterior fossa contusion was 8.9 ml. The cerebellar hemispheric (60.9%) location of hematoma was more common. Age at presentation ( $P = 0.0086$ ), best motor response ( $P < 0.0001$ ), severity of injury ( $P = 0.0002$ ), GCS at admission ( $P < 0.0001$ ), effacement of basal cistern ( $P < 0.0001$ ), fourth ventricular compression and intraventricular hemorrhage ( $P = 0.0008$ ), presence of hydrocephalus ( $P = 0.0142$ ), subarachnoid hemorrhage ( $P = 0.0008$ ), and volume of posterior fossa contusion ( $P = 0.0002$ ) were significantly associated with outcome of posterior fossa contusion. **Conclusion:** Traumatic cerebellar hematoma is rare. Conservatively managed cerebellar hematoma patients must be monitored closely for neurological and radiological status. Patients who show deterioration in neurological or radiological status require surgical intervention.

**Keywords:** Conservative management, outcome, traumatic cerebellar contusion, traumatic cerebellar hematoma, traumatic posterior fossa contusion, traumatic posterior fossa hematoma

### Introduction

Traumatic cerebellar hematomas or hemorrhagic contusions are rare in comparison to nontraumatic causes. The small capacity of the posterior fossa compounded with packed critical structures often leads to a catastrophic outcome in these hematomas. These lesions may present as an isolated solitary bleed or in association with a subdural hematoma (SDH), extradural hematoma, or supratentorial lesions. Traumatic cerebellar hematomas may present acutely or in a delayed fashion when they are referred to as delayed traumatic intracerebellar hematoma. Management of such hematomas has changed from predominantly surgical intervention to a combination of both conservative and surgical approaches based

on the hematoma characteristics and the clinical condition of the patients.

In comparison to posttraumatic intracerebral hematomas, traumatic cerebellar hematomas are rare and are reported in <1% of all head injuries.<sup>[1,2]</sup> Various studies had reported the incidence of cerebellar hematoma from 0.4% to 3.7% of total intracranial hematoma.<sup>[3-6]</sup> Computed tomography (CT) scan is the investigation of choice to diagnose traumatic cerebellar hematoma. It reveals the location of the bleed, superficial or deep, size of hematoma, status of the fourth ventricle and cisterns, presence or absence of hydrocephalus, associated posterior fossa EDH, SDH, or any supratentorial lesion [Figures 1 and 2]. It is used to follow-up the changing radiological picture where the conservative approach

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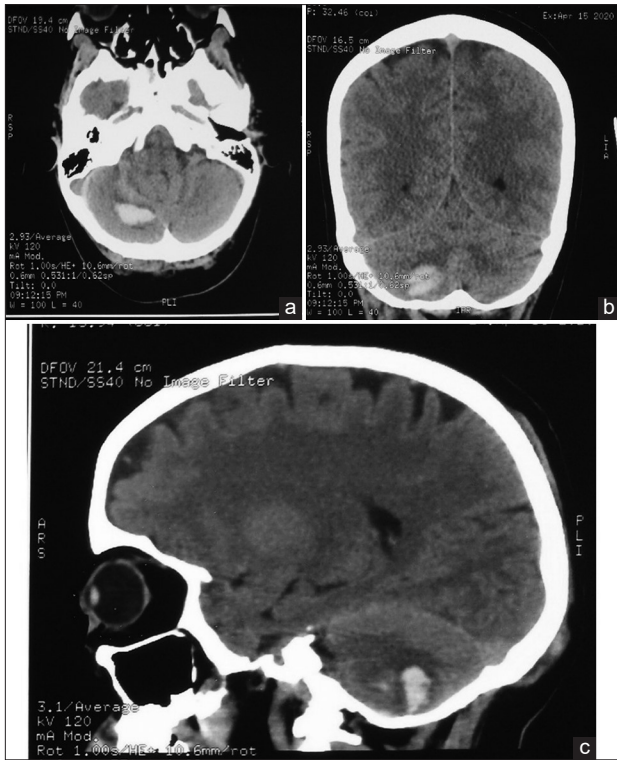


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**Figure 1: Non contrast computed tomography scan showing right lateral cerebellar bleed, with perilesional edema. (a) axial, (b) coronal and (c) sagittal views**

is followed. Volumetric assessment of cerebellar hematoma is done by using the formula,  $ABC/2$ , where A is the maximum hematoma diameter by CT, B is the perpendicular diameter to A, and C is the CT slice counts with hematoma, multiplied by the thickness of CT slice.<sup>[7]</sup>

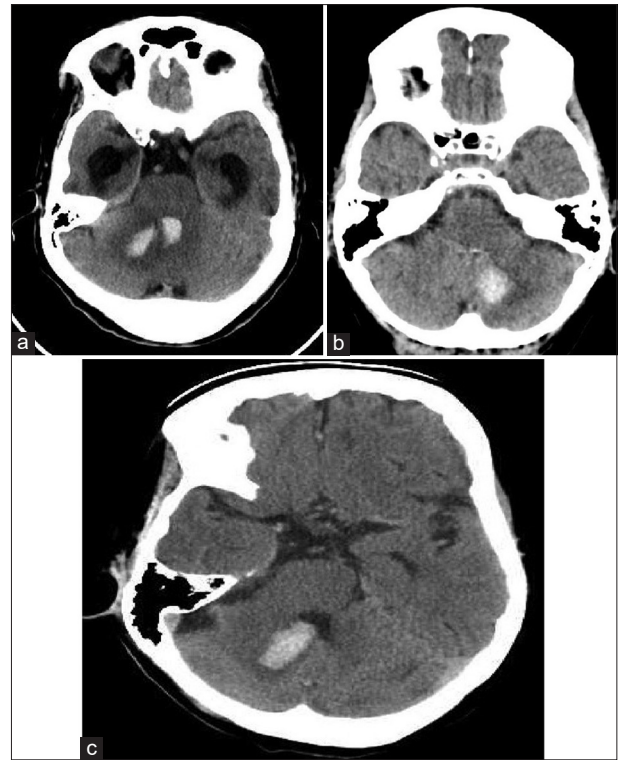
Traumatic cerebellar hematomas may result from local injury to the occipital area. Strong muscles of the neck, strong bone with smooth internal surfaces, the flexible cerebellar tentorium, and the CSF cushioning effect in the 4<sup>th</sup> ventricle and cisterns significantly reducing the amount of energy delivered at the moment of impact. Hence, traumatic cerebellar hematomas are less common than that of cerebral hemispheres.<sup>[8]</sup>

In this study, we aim to evaluate the prognostic factors and outcome determining factors with regard to conservatively managed isolated traumatic cerebellar hematoma.

## Materials and Methods

A retrospective analysis of 3210 patients of head injury, admitted between August 2018 and May 2020, was conducted in the Department of Neurosurgery, Institute of Medical Sciences, Banaras Hindu University, Varanasi. Twenty-three adult patients were found with isolated traumatic posterior fossa cerebellar hematoma that were conservatively managed.

The study included those patients that fit our institutional protocol for conservative management of posterior fossa



**Figure 2: (a) Axial computed tomography scan showing acute hematoma involving the medial aspect of the right cerebellar hemisphere/middle cerebellar peduncle with intraventricular extension. Significant perilesional edema and obstructive hydrocephalus also noted. (b) Axial computed tomography scan showing acute hematoma in superior aspect of the left cerebellar tonsil. Mild perilesional edema is also seen. (c) Axial computed tomography scan showing medial aspect of the right cerebellar bleed with evidence of intraventricular extension**

hematoma or those who did not give consent for surgery. Non-comatose patients with isolated intracerebellar clots  $\leq 3$  cm were managed conservatively, under close supervision. Any patient who presented with a depressed level of consciousness, focal neurological deficit, and/or ominous findings on CT scan (hydrocephalus, obliterated Perimesencephalic cisterns, and/or displaced the fourth ventricle) was proceeded for early surgical intervention. Of these 23 patients, 6 patients were given option for surgery, but the patient/attendants did not give consent.

Initial CT head was obtained at the admission of the patient and repeat CT head was done at 6 h if the patient deteriorated  $>2$  points of initial Glasgow Coma Scale (GCS) score at admission. On CT scan, contusion location, volume, status of basal cisterns (patent or effaced), status of the fourth ventricle (normal or compressed, presence or absence of intraventricular hemorrhage [IVH]) and associated hydrocephalus were evaluated. Each of the patients were evaluated in terms of age, sex, mode of injury, clinical presentation, comorbidity, the severity of injury, best motor response, GCS at admission, CT findings, and Glasgow Outcome Scale (GOS) at discharge/death. The outcome was also evaluated by further making dichotomized group using GOS in death/dependent (1-3) versus independent (4-5).

We also compared the two cohorts of patients, one who were managed conservatively as per our institutional protocol ( $n = 17$ ) and the other who did not give consent for surgery and hence were managed conservatively ( $n = 6$ ).

Statistical tests were done using GraphPad Prism version 8.3.0 for Windows, (GraphPad Software, San Diego, California USA) software. For dichotomous evaluation, Chi-square test and unpaired  $t$ -test were used according to data analyzed. For percentage or proportion data Chi-square test and unpaired  $t$ -test were used, respectively, for the testing level of significance.  $P < 0.05$  was deemed statistically significant.

## Results

A total of 23 patients with conservatively managed posterior fossa contusion were analyzed retrospectively. The mean age of patients was 54.5 years, with females being more commonly affected (60.9%). Male-to-female ratio was 1:1.56. Road traffic accident was the most common mode of injury (65.2%) followed by fall from height (21.7%) and physical assault (13.1%) in succession. Common associated comorbidities were hypertension (39.1%), diabetes (21.7%), and oral anticoagulant intake (17.4%). Patients with posterior fossa contusion commonly presented with altered sensorium (73.9%), vomiting (39.1%), headache (26.1%), and cerebellar signs (17.4%) [Table 1].

Most patients suffered a mild head injury (60.9%) followed by moderate (26.1%) and severe (13%) head injury. Mean GCS at admission was 12.3 with M6 (26.1%), M5 (43.5%), and M4 (17.4%) as the more common best motor responses [Table 2].

The mean volume of posterior fossa contusion was 8.9 ml. Vermian (17.4%) location of contusion was less common than cerebellar hemispheric (60.9%) contusion. Contusions involving both vermis and hemisphere were present in 5 patients (21.7%). Basal cisterns were patent in 18 patients (78.3%) and effaced in 5 patients (21.7%). Compression of 4<sup>th</sup> ventricle was present in 4 patients (17.4%), and IVH was present in 4 patients (17.4%). Hydrocephalus was present in 6 patients (26.1%) [Table 3].

There were two mortalities (8.7%) in the study group. Both these patients attendant had not given consent for surgery. Glasgow outcome score (GOS) of 5, i.e., good recovery was seen in 17 patients (73.9%) followed by GOS of 4 in 2 patients (8.7%) and GOS of 3 and 2 in 1 patient each (4.3%) [Table 4].

On dichotomized analysis, age at presentation ( $P = 0.0086$ ), best motor response ( $P < 0.0001$ ), severity of injury ( $P = 0.0002$ ), GCS at admission ( $P < 0.0001$ ), effacement of basal cistern ( $P < 0.0001$ ), fourth ventricular compression and IVH ( $P = 0.0008$ ), presence of hydrocephalus ( $P = 0.0142$ ), subarachnoid

**Table 1: Patient demography, mode of injury, comorbidities, and clinical presentation ( $n=23$ )**

Patient characteristics	Number of patients (%)/value
Sex	
Male	9 (39.1)
Female	14 (60.9)
Age (years)	
Mean age	54.5
Range	35-68
Mode of injury	
RTA	15 (65.2)
FFH	5 (21.7)
Physical assault	3 (13.1)
Coexisting comorbidities	
Diabetes mellitus	5 (21.7)
Hypertension	9 (39.1)
Respiratory disease	2 (8.7)
Stroke (previous history)	1 (4.3)
Seizure disorder	1 (4.3)
Cardiovascular disease	2 (8.7)
Psychiatry illness	1 (4.3)
Gastrointestinal diseases	1 (4.3)
Oral anticoagulant intake	4 (17.4)
Previous head injury	1 (4.3)
Clinical presentation	
Headache	6 (26.1)
Ataxia	5 (21.7)
Cerebellar signs	4 (17.4)
Seizure	1 (4.3)
Loss of hearing	2 (8.7)
Vertigo	4 (17.4)
Tinnitus	1 (4.3)
Facial weakness/paresis	1 (4.3)
Vomiting	9 (39.1)
Dysarthria	2 (8.7)
Altered sensorium	17 (73.9)

RTA – Road traffic accident, FFH – Fall from height

hemorrhage (SAH) ( $P = 0.0008$ ), and volume of posterior fossa contusion ( $P = 0.0002$ ) were significantly associated with outcome of posterior fossa contusion [Table 5]. However, we did not find any significant association of gender, mode of injury, and location of contusion with outcome [Table 5]. The mean duration of injury and reporting to the hospital was 4.8 h in the favorable outcome group and 6.7 h in poor outcome group.

Moreover, we did not find any association between the location of contusion and GCS at admission. No association was found between hypertension ( $P = 0.1058$ ), diabetes ( $P = 0.8619$ ) and oral anticoagulant intake ( $P = 0.0583$ ) and outcome of posterior fossa contusion [Table 5].

Of 6 patients who did not consent for surgery, mean volume of hematoma was 11.73 ml. 4 patients had hydrocephalus, 5 patients had effaced basal cisterns, mean GCS at admission was 10.9, and there were 2 mortalities in this group.

**Table 2: Severity of injury, Glasgow Coma Scale at admission, and best motor response**

Patient characteristics	Number of patients (%) /value
Severity of injury	
Mild (14-15)	14 (60.9)
Moderate (9-13)	6 (26.1)
Severe (3-8)	3 (13)
GCS at admission	
Mean	12.3
Standard deviation	2.49
Best motor response	
M6	6 (26.1)
M5	10 (43.5)
M4	4 (17.4)
M3	2 (8.7)
M2	1 (4.3)
GCS – Glasgow Coma Scale	

**Table 3: Radiological findings**

Radiological findings	Number of patients (%) /value
Volume of contusion	
Mean	8.9 ml
SD	2.4
Maximum diameter of hematoma	
Mean	23 mm
SD	2.8
Location of contusion	
Vermian	4 (17.4)
Hemispheric	14 (60.9)
Both	5 (21.7)
Basal cisterns	
Patent	18 (78.3)
Effaced	5 (21.7)
Fourth ventricle	
Normal	19 (82.6)
Compressed	4 (17.4)
IVH	4 (17.4)
Hydrocephalus	
Present	6 (26.1)
Absent	17 (73.9)
SAH	4 (17.4)

SD – Standard deviation; SAH – Subarachnoid hemorrhage; IVH – Intraventricular hemorrhage

On comparing the two cohorts of patients, one who were managed conservatively as per our institutional protocol ( $n = 17$ ) and the other who did not give consent for surgery and hence were managed conservatively; age of the patients ( $P = 0.0003$ ), best motor response ( $P < 0.001$ ), severity of injury ( $P = 0.0005$ ), GCS at admission ( $P = 0.0279$ ), effacement of basal cisterns ( $P < 0.0001$ ), compression of the 4<sup>th</sup> ventricle ( $P = 0.0002$ ), hydrocephalus ( $P = 0.0085$ ), volume of hematoma ( $P < 0.0001$ ), location of contusion ( $P = 0.0144$ ), and mortality ( $P = 0.0127$ ) showed significant ( $P < 0.05$ ) difference between the two groups [Table 6].

**Table 4: Glasgow outcome score**

GOS	Number of patients (%)
5 ( good recovery)	17 (73.9)
4 ( moderate disability)	2 (8.7)
3 (severe disability)	1 (4.3)
2 ( persistent vegetative state)	1 (4.3)
1 (dead)	2 (8.7)

GOS – Glasgow outcome score

**Table 5: Dichotomized outcome analysis: Glasgow outcome scale categories (1-3 vs. 4-5)**

Characteristics	1-3 ( $n=4$ ), $n$ (%)	4-5 ( $n=19$ ), $n$ (%)	$P$
Age	61.3 (5.14)	51.6 (6.23)	0.0086*
Gender (%)			
Male	3 (75)	6 (31.6)	0.1058
Female	1 (25)	13 (68.4)	
Mode of injury			
RTA	3 (75)	12 (52.2)	0.6955
FFH	1 (25)	4 (21)	
Assault	0	3 (15.8)	
Best motor response	2.48 (0.81)	5.42 (0.3)	<0.0001*
Severity of injury			
Mild	0	14 (73.7)	0.0002*
Moderate	1 (25)	5 (26.3)	
Severe	3 (75)	0	
GCS at admission	6.4 (0.82)	12.8 (1.20)	<0.0001*
Basal cisterns			
Patent	0	18 (94.7)	<0.0001*
Effaced	4 (100)	1 (5.3)	
Fourth ventricle			
Normal	1 (25)	18 (94.7)	0.0008*
Compressed	3 (75)	1 (5.3)	
IVH	3 (75)	1 (5.3)	
Hydrocephalus			
Present	3 (75)	3 (15.8)	0.0142*
Absent	1 (25)	16 (84.2)	
Volume of contusion	9.8 ml (1.2)	7.6 ml (0.8)	0.0002*
Location of contusion			
Vermian	1 (25)	3 (15.8)	0.5034
Hemispheric	3 (75)	11 (57.9)	
Both	0	5 (26.3)	
SAH	3 (75)	1 (5.3)	0.0008*
Comorbidity			
Hypertension	3 (75)	6 (31.6)	0.1058
Diabetes mellitus	1 (25)	4 (21)	0.8619
Oral anticoagulation	2 (50)	2 (10.5)	0.0583

\* – Statistically significant ( $P < 0.05$ ), SAH – Subarachnoid hemorrhage; IVH – Intraventricular hemorrhage; GCS – Glasgow Coma Scale; RTA – Road traffic accident, FFH – Fall from height

## Discussion

Traumatic cerebellar contusion or hematoma is quite rare when compared to intracerebral hematoma account to <1% as quoted in various studies.<sup>[1-5]</sup> In this study, we found the incidence of conservatively managed isolated posterior

**Table 6: Comparison of conservative indicated and negative consented patients**

Characteristics	Patients with conservative management indications (n=17)	Patients who gave negative consent for surgery (n=6)	P
Age (SD)	52.4 (3.8)	60.45 (4.5)	0.0003*
Sex			
Male	12	2	0.1079
Female	5	4	
Best motor response (SD)	5.3 (0.59)	3.33 (0.82)	<0.0001*
Severity of injury			
Mild	14	0	0.0005*
Moderate	3	3	
Severe	0	3	
GCS at admission (SD)	12.8 (1.8)	10.9 (1.3)	0.0279*
Effaced basal cisterns	0	5	<0.0001*
Compressed 4 <sup>th</sup> ventricle	0	4	0.0002*
Hydrocephalus	2	4	0.0085*
Volume of hematoma (SD)	7.9 ml (1.2)	11.73 ml (2.3)	<0.0001*
Location of contusion			
Vermian	1	2	0.0144*
Hemispheric	14	1	
Both	2	3	
Mortality	0	2	0.0127*

\* - Statistically significant ( $P < 0.05$ ). GCS – Glasgow Coma Scale; SD – Standard deviation

fossa contusion/hematoma to be 0.71% of the head injury cases admitted at our center over the study duration. The mean age of the patient was 54.5 years, with females more common (60.9%) than males. Road traffic accident (65.2%) was the most frequent mode of injury, followed by fall from height. Although all age groups can be affected by traumatic cerebellar contusion/hematoma, studies have shown young adults more susceptible to these injuries due to road traffic accidents caused by rash driving and sports-related fall.<sup>[7]</sup> Posterior fossa hematoma is rare in children and the incidence of cerebellar hematoma is much rarer.<sup>[9]</sup>

Asymptomatic traumatic cerebellar hematoma may undergo sudden deterioration of the neurological status which may be attributed to small reserve capacity of the posterior fossa.<sup>[10]</sup> Cerebellar contusions/hematoma presents in a variety of ways. In adults, they will present with cerebellar signs such as nystagmus, dysarthria, hypotonia, ataxia, dysmetria, tremor, dysdiadochokinesis, and vertigo.<sup>[11]</sup> In addition, cerebral mutism may also be evident in children. One study regarding the neuropsychological sequelae in children with cerebellar trauma noted that most children with cerebellar trauma develop dyscalculia and exhibit lower visual recognition memory.<sup>[12]</sup> Features of increased intracranial pressure such as headache, diminished consciousness, vomiting, pupillary irregularity, and deranged respiratory pattern may be present.<sup>[7]</sup> In this study, patients most commonly presented with altered sensorium (73.9%), vomiting (39.1%), headache (26.1%), and cerebellar signs (17.4%) in decreasing order of presentation. Hypertension (39.1%) was the most common associated comorbidity followed by diabetes (21.7%) and

oral anticoagulant intake (17.4%), respectively. We did not find any significant association between comorbidities and the outcome of conservatively managed cases. Although these parameters might affect the surgical outcome, there is a paucity of literature in this regard.

Due to the rare incidence of traumatic cerebellar hematomas, the management approaches remain controversial. Conservative and surgical management are the mainstay of management. Conservative management is considered in conscious patients with superficial hematoma location and size <3 cm.<sup>[13]</sup> These patients should be serially followed up neurologically and radiologically to detect any delayed hematoma appearance, increasing size of cerebellar hematoma, compression of the fourth ventricle or brainstem cisterns, and development of hydrocephalus. Surgical evacuation is indicated in patients with hematoma size  $\geq 3$  cm, cisternal and fourth ventricular compression, associated extradural/subdural hematomas of posterior fossa and/or associated acute hydrocephalus.<sup>[7,14-16]</sup> In our study, the mean volume of contusion/hematoma was 8.9 ml. Basal cistern effacement, fourth ventricular compression, and acute hydrocephalus was seen in 21.7%, 17.4%, and 26.1% of patients, respectively, who refused for surgery. Hydrocephalus is usually not associated with traumatic intracerebellar clots.<sup>[17-19]</sup> Karasawa *et al.*<sup>[2]</sup> mentioned of acute hydrocephalus in 20% of intracerebellar hematomas, whereas in our series, it was (26.1%).

In this study, we found that age at presentation, best motor response, severity of injury, GCS at admission, effacement of basal cistern, fourth ventricular compression and intraventricular hemorrhage, presence of hydrocephalus,

associated subarachnoid hemorrhage and volume of posterior fossa contusion were significantly associated with outcome of conservatively managed isolated cerebellar contusion/hematoma. Studies have reported that GCS at admission,<sup>[4]</sup> location of cerebellar contusion/hematoma, volume of hematoma, status of ventricles and associated cisterns, and presence of associated lesions<sup>[7]</sup> are important determinants of the outcome of posterior fossa hematoma.

On comparing the two cohorts of the patients, one who were managed conservatively as per our institutional protocol and the other who did not give consent for surgery and hence were managed conservatively we found that in the latter group, there was significantly raised mortality, poorer GCS at admission, more severity of the head injury and older mean age. There is a paucity of literature in regard to such comparison.

The initial GCS score at the time admission is the most important factor controlling the outcome. Studies have reported that GCS score  $\geq 8$  have a better outcome, irrespective of conservative or surgical management.<sup>[1,3,4]</sup>

Contusions present in the deep regions of the cerebellum (in the vicinity of midline or vermis) are having increased morbidity and mortality.<sup>[3]</sup> However, we did not found any significant association of the location of cerebellar contusion/hematoma with the outcome. Traumatic cerebellar hematomas can be further classified based on their location into deep-seated and superficial. The deep portion comprises the vermis and the inner one-third of each cerebellar hemisphere. Whereas superficial traumatic hematomas are located in the superficial two-thirds of the cerebellar hemisphere. Takeuchi *et al.*<sup>[3]</sup> classified these cerebellar hematoma locations as either type 1 (deep) or type 2 (superficial). Deep cerebellar hematomas compress the fourth ventricle and brainstem and its cisterns early due to its vicinity. Due to such compression, hydrocephalus and increase in intracranial pressure are much earlier and more common than the superficial cerebellar hematomas. The superficial cerebellar hematomas may produce similar effects if they are large enough to compress the fourth ventricle at a later stage. The superficial cerebellar hematomas thus have a better outcome in comparison to deep cerebellar hematomas. Various studies have shown that greater the hematoma volume, earlier is the appearance of raised intracranial pressure features and resultant less favorable outcome. Deep cerebellar hematoma locations are having greater hematoma volume as compared to superficial location.<sup>[3,7]</sup> In this study, we did not found any significant association of location of contusion with the outcome, which may be due to the small size of hematoma volume in conservatively managed cases.

Studies have shown that SAH associated with the traumatic cerebellar hematoma is having a poorer outcome, probably due to the ischemic effects of vasospasm induced by the SAH.<sup>[3,7]</sup> In this study, we also found SAH to be significantly associated with poorer outcome.

The presence of supratentorial hematoma/injury along with traumatic cerebellar hematoma is associated with poorer outcome.<sup>[1]</sup> Nashimoto *et al.* reported that these patients should ideally be operated, and evacuation of cerebellar clot must be done.<sup>[14]</sup> In this study, we took isolated cerebellar contusion/hematoma cases without any other evident lesion supratentorially or infratentorially on noncontrast CT scans.

Delayed or evolving hematoma has been reported previously.<sup>[17,18]</sup> In this study, no cases of delayed or evolving hematoma was reported. Repeat CT examination is necessary, as delayed hematomas can develop. The timing of reaching the hospital is important and affects the overall morbidity and mortality of patients. Most of the patients in the favorable outcome group reported to the hospital within 4–5 h, whereas those in the poor outcome group had reported >4–8 h. Suboccipital craniectomy with cerebellar hematoma evacuation is preferred by most neurosurgeons for surgical candidates of traumatic cerebellar hematomas.<sup>[15,16]</sup> It should be considered in conservatively managed cases if neurological or radiological deterioration occurs in these patients.

## Conclusion

Traumatic cerebellar hematoma is rare among cases of traumatic head injury. They are potentially life-threatening condition which mostly presents in an acute fashion but may present as delayed hematoma. Conservatively managed cerebellar hematoma patients must be monitored closely for neurological and radiological status. Patients who show deterioration in neurological or radiological status require surgical intervention. Initial GCS score, hematoma size and volume, status of cisterns surrounding brainstem and 4<sup>th</sup> ventricle, fourth ventricular compression and intraventricular hemorrhage, SAH, and hydrocephalus are the ultimate prognostic markers for conservatively managed isolated cerebellar contusion/hematoma.

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## Conflicts of interest

There are no conflicts of interest.

## References

1. d'Avella D, Servadei F, Scerrati M, Tomei G, Brambilla G, Angileri FF, *et al.* Traumatic intracerebellar hemorrhage: Clinicoradiological analysis of 81 patients. *Neurosurgery* 2002;50:16-25.
2. Karasawa H, Furuya H, Naito H, Sugiyama K, Ueno J, Kin H. Acute hydrocephalus in posterior fossa injury. *J Neurosurg* 1997;86:629-32.
3. Takeuchi S, Takasato Y, Masaoka H, Hayakawa T. Traumatic intra-cerebellar haematoma: Study of 17 cases. *Br J Neurosurg* 2011;25:62-7.
4. D'Avella D, Cacciola F, Angileri FF, Cardali S, La Rosa G,

- Germanò A, *et al.* Traumatic intracerebellar hemorrhagic contusions and hematomas. *J Neurosurg Sci* 2001;45:29-37.
5. Sato K, Hinokuma K, Matsuzawa Y, Takehara S, Uemura K, Ninchoji T, *et al.* Clinical study of traumatic cerebellar contusion. *No Shinkei Geka* 1987;15:1285-9.
  6. Liu K. Characteristics of diagnosis and treatment of traumatic intracerebellar hemorrhage. *Zhonghua Wai Ke Za Zhi* 1997;35:166-7.
  7. Patnaik A, Mahapatra AK. Traumatic cerebellar haematoma: A review. *Indian J Neurotrauma* 2013;10:24-9.
  8. Glowacki JW. Posttraumatic cerebellar contusions and hematomas. In: Frowein RA, editors. *Cerebral Contusions, Lacerations and Hematomas. Advances in Neurotraumatology. Vol. 3.*, Vienna: Springer-Verlag Wien; 1991.
  9. Kulshreshtha V, Tripathi P, Jaiswal G, Gupta TK. Traumatic cerebellar haematoma in paediatric patient – A case report and review of literature. *Rom Neurosurg* 2016;30:566-72.
  10. Sokol JH, Rowed DW. Traumatic intracerebellar haematoma. *Surg Neurol* 1978;10:340-1.
  11. Van Gijn J. From the archives. *Brain* 2007;130:4-7.
  12. Braga LW, Souza LN, Najjar YJ, Dellatolas G. Magnetic resonance imaging (MRI) findings and neuropsychological sequelae in children after severe traumatic brain injury: The role of cerebellar lesion. *J Child Neurol* 2007;22:1084-9.
  13. Buczek M, Jagodziński Z, Kopytek M, Dabrowska E. Conservative treatment of post-traumatic intracerebellar hematoma. *Wiad Lek* 1989;42:550-5.
  14. Nashimoto T, Sasaki O, Nozawa T, Ando K, Kikuchi B, Watanabe M. Clinical study on cerebellar contusion: A report on 9 cases and literature review. *No Shinkei Geka* 2015;43:901-6.
  15. Pollak L, Rabey JM, Gur R, Schiffer J. Indication to surgical management of cerebellar hemorrhage. *Clin Neurol Neurosurg* 1998;100:99-103.
  16. Aghi M, Ogilvy CS, Carter B. Surgical management of intracerebral hemorrhage. In: Roberts DW, Schmidek HH, editors. *Schmidek and Sweet's Operative Neurosurgical Techniques, Indications, Methods, and Results. 5<sup>th</sup> ed., Vol. 2.* Philadelphia: Saunders/Elsevier; 2005. p. 1061-74.
  17. Pozzati E, Grossi C, Padovani R. Traumatic intracerebellar hematomas. *J Neurosurg* 1982;56:691-4.
  18. Yokota H, Mizunari T, Kuzuhara M, Kobayashi S, Yajima K, Nakazawa S, *et al.* Traumatic delayed intracerebellar hematoma. *Neurol Med Chir (Tokyo)* 1988;28:886-90.
  19. Tsai FY, Teal JS, Itabashi HH, Huprich JE, Hieshima GB, Segall HD. Computed tomography of posterior fossa trauma. *J Comput Assist Tomogr* 1980;4:291-305.