Traumatic Posterior Fossa Hematoma, A Rare Entity: Study of 21 Cases

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Abstract

Background Traumatic posterior fossa hematoma is a rare entity. Traumatic posterior fossa hematomas are associated with considerable morbidity and mortality and their surgical management remained controversial.

Methods From August 2011 to August 2017, approximately 5,100 patients with head injury were managed. Authors reviewed clinical and radiological findings, management criteria, and outcome of posterior fossa hematoma in 21 patients.

Results Out of 21 cases, 13 survived with our management. The Glasgow Coma Scale (GCS) on admission was higher in favorable group than in poor outcome group. Factors associated with Glasgow Outcome Scale in two groups were status of fourth ventricle, basal cisterns, subarachnoid hemorrhage (SAH), hematoma volume, and their location (hemispheric or midline). Similarly, associated supratentorial lesions, age, gender, lesions in other parts of body, and timing from injury to reporting to hospital were taken into consideration.

Conclusion The factors correlated with patient outcome were age, sex, mode of injury, GCS at admission, associated intracranial hematomas, associated SAH, hematoma volume, hematoma location, basal cisterns, status of fourth ventricle, and associated multiple injuries on other body parts. It is hereby concluded that timely surgical intervention should be employed whenever indicated without delay. Posterior fossa hematomas were rarely observed in the pediatric age group.

Keywords
- traumatic posterior fossa hematoma
- subarachnoid hemorrhage
- Glasgow Coma Scale

Introduction

Traumatic posterior fossa hematoma is very less as compared with spontaneous hematomas associated with hypertension. They are associated with significant mortality and morbidity. They constitute a small subset of total head injury patients.[1] As we know, posterior fossa have small volume, and any increase in volume due to any cause may cause increase intracranial pressure, herniation, and deterioration. Traumatic posterior fossa hematomas account for < 1% in all head injury patients.[2,3] Liu[4] reported the incidence of traumatic posterior fossa hematoma to be 3.7% of total intracranial hematomas. These hematomas may be totally asymptomatic, with a sudden increase in size can lead to rapid deterioration of neurological status.[5]

Traumatic cerebellar hematoma may be isolated or may be associated with subdural hemorrhage (SDH), extradural hemorrhage (EDH), and subarachnoid hemorrhage (SAH) in posterior fossa. They may present acutely or in delayed manner. The hematoma location, volume, initial Glasgow Coma Scale (GCS) score, status of fourth ventricle, and basal cisterns are important in final outcome. Management of these hematomas nowadays have changed from all surgery to conservative management.[1]

Authors evaluated outcome of patients on GCS score at admission, location, volume, status of cisterns, fourth ventricle, SAH, and supratentorial lesions. The surgical indications were posterior fossa hematoma with mass effect, midline shift, size greater than 3 cm, effaced basal cisterns, fourth ventricle compression, and associated supratentorial lesions with significant mass effect with midline shift.

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Similarly, patients were managed conservatively with respect to hematoma location, size less than 3 cm, normal basal cisterns, and fourth ventricle. In some of these hematomas, early surgical intervention is deemed necessary.[5-9] The early results of surgery are good, so surgical intervention if required should not be delayed. The course and prognosis of midline hematoma is worse, so evacuation should be done early.

Methods

From August 2011 to August 2017, approximately 5,100 patients with head injury were admitted in our institute. This study was focused on 21 patients (0.41%) with traumatic posterior fossa hematomas. The clinical findings, mode of injury, initial GCS, various radiological parameters, and final outcome were evaluated in 21 patients using Glasgow Outcome Scale (GOS). GOS was graded into good recovery (GR), moderate disability, severe disability (SD), vegetative state (VS), and death (D).

Initial computed tomography (CT) head was obtained at admission of patient and repeat CT head was done at 6 hours if the patient deteriorated more than 2 points of initial GCS score at admission. On CT scan, hematoma location, size, status of basal cisterns (normal or compressed), status of fourth ventricle (normal or compressed), associated SAH, and supratentorial lesions were evaluated. Hematoma volume was assessed by using formula A*B*C/2, where A is maximum transverse diameter of hemorrhage on CT, B is anterioposterior diameter, and C is number of CT slices showing hematoma.[10]

Hematomas were classified as type 1 vermian hematoma (►**Fig. 1**) and type 2 hemispheric hematoma involving superficial two-thirds of the hemisphere (►**Figs. 2** and **3**). Injury is classified as coup, countercoup, and acceleration–deceleration type. Time to reach hospital is important as it affects overall morbidity and mortality of patient.

Results

Clinical and Radiological Findings

Out of 21 patients, 18 were male and the rest females. Most of the patients were in the age range of 30 to 60 years. The mode of injury was road traffic accident (15 patients) and fall (6 patients). Duration between sustaining injury and reporting to hospital was 4 hours in 11 patients and 5 to 8 hours in 10 patients. GCS score on admission ranged from 3 to 15 as shown in ►**Table 1**. The location of hematoma was type 1 (8 patients) and type 2 (13 patients). Nine patients had isolated posterior fossa hematoma and 12 patients presented with associated intracranial lesions.

Intracranial lesions were supratentorial contusion (11), SDH (2), EDH (2), SAH (9), IVH (1), diffuse axonal injury (DAI) (1), hydrocephalus (HCP) (2), and SDH in posterior fossa in 1 patient (►**Fig. 3C**). Basal cisterns were compressed in 9 and

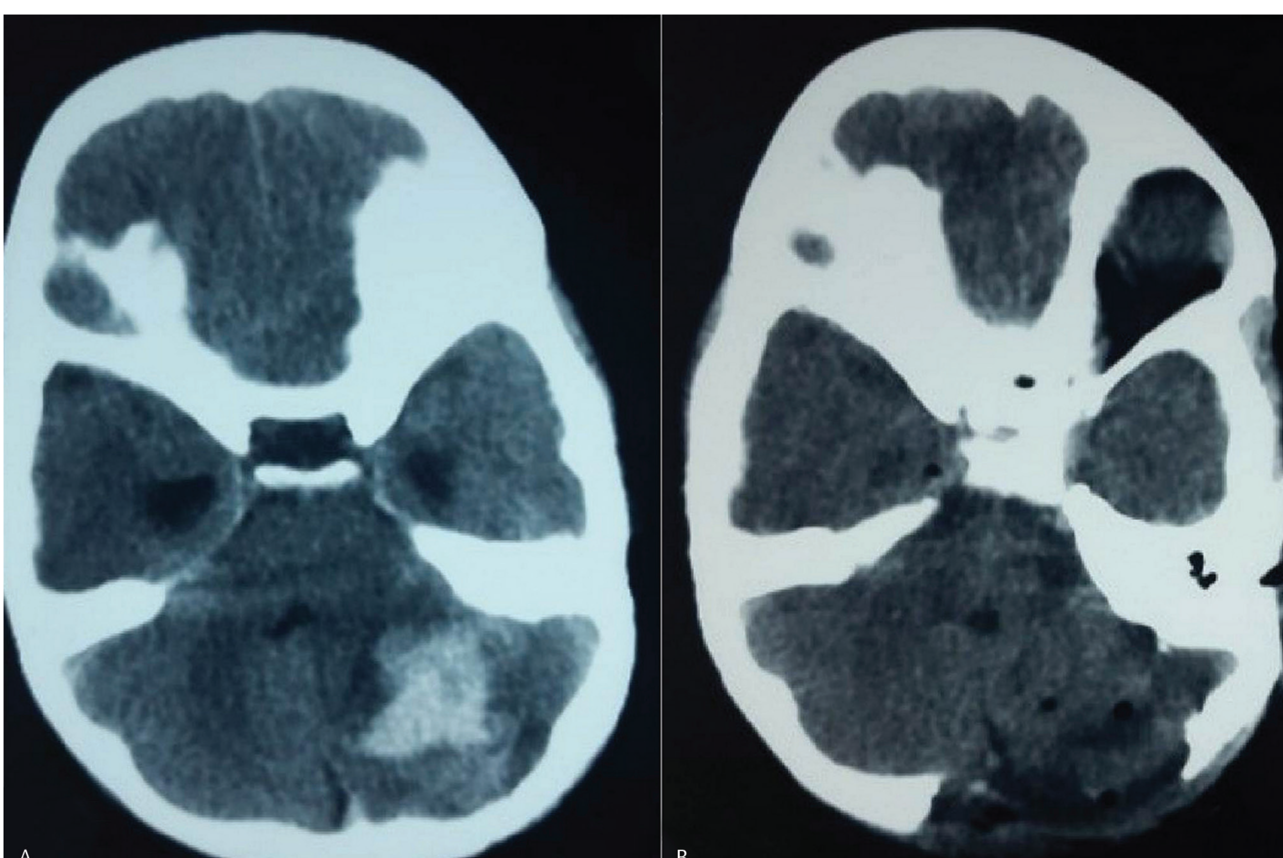

Fig. 1 (**A**) Computerized tomography scan revealing vermian hematoma (type 1). (**B**) Postoperative scan after suboccipital craniectomy and hematoma evacuation (type 1).

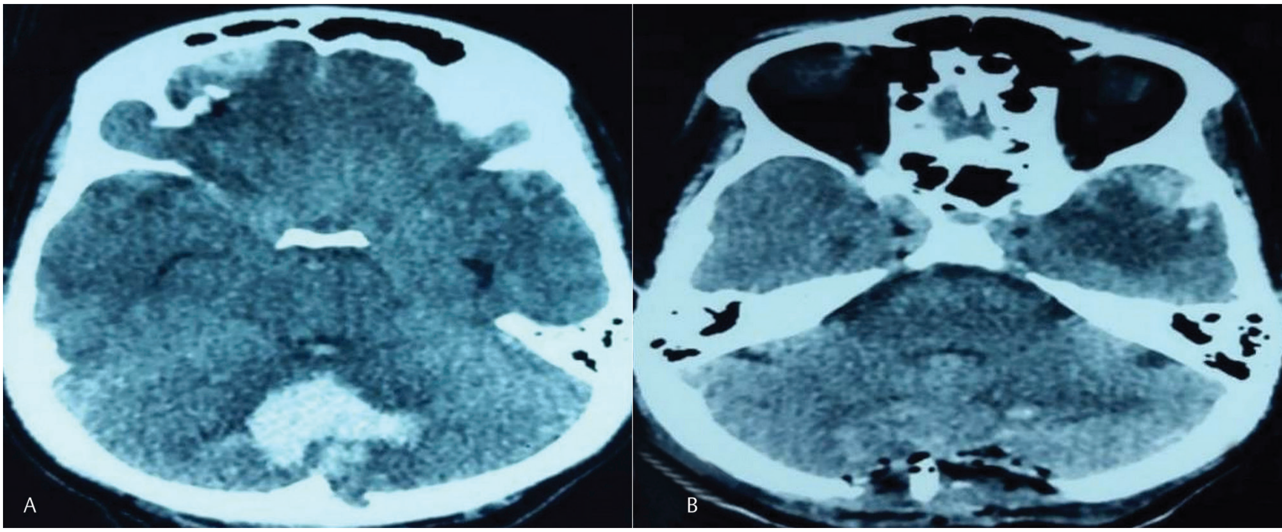

Fig. 2 (**A**) Computerized tomography scan revealing preoperative image type 2 hematoma. (**B**) Postoperative scan showing complete removal of hematoma.

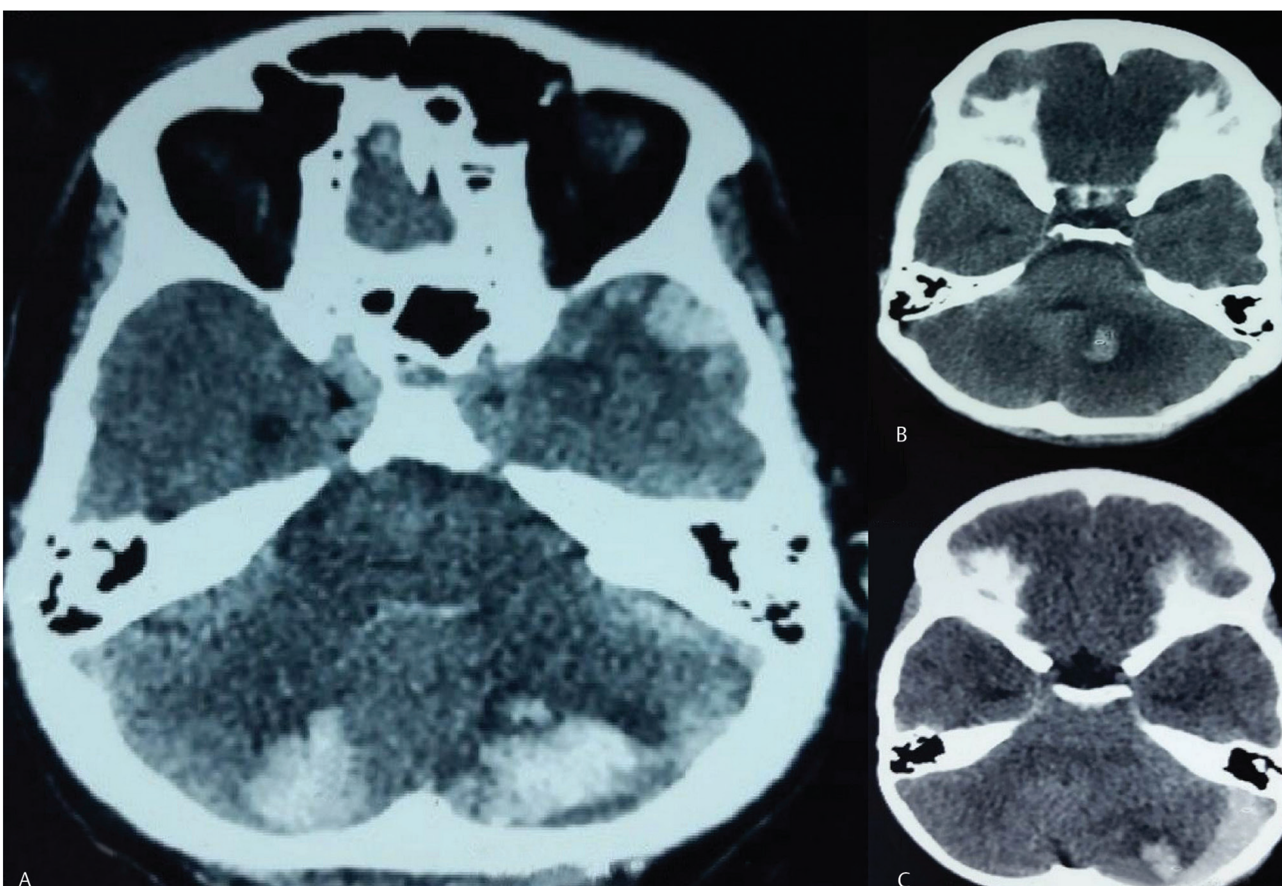

Fig. 3 (**A**) Computerized tomography scan revealing bilateral type 2 hematoma. (**B**) Computerized tomography revealing type 2 hematoma. (**C**) Computerized tomography revealing type 2 hematoma with subdural hemorrhage.

normal in 12 patients. Fourth ventricle had intraventricular hemorrhage (IVH) in 3, was compressed in 3, and normal in 15 patients. Associated injuries were found in 8, and skull fracture was seen in 15 patients. Out of 8 patients, only 1 had splenic injury which was managed conservatively and 1 patient had hemothorax which was managed by intercostal tube drainage.

Type 1 and Type 2 Hematoma Outcome

Comparison between type 1 and 2 hematomas is shown in ►**Table 2**. Age range in type 1 was 22 to 56 years, while in type 2 hematomas it was 12 to 79 years. In type 2 hematomas, age varied over a wider range as compared with type 1. Volume of hematoma was higher in type 2 hematomas as

Table 1 Summary of cases

S. no.	Age (y)	Sex (M/F) Duration[a]	Mode of injury, skull fracture	Associated lesions	Site of impact	GCS (3–15)	Volume, cm³, size (cm)	Type of location (½)	Basal cisterns (N/C)	Fourth ventricle (N/C/IVH)	Associated supratentorial lesions	SAH (A/P)	Intervention	GOS
1	40	M, 4 h	MVA,–	None	Occipital	14/15	16.8 (3.5 cm)	2	N	IVH	No lesion	A	Suboccipital craniectomy	GR
2	40	M, 6 h	MVA,+	None	Frontal	5/15	4 (2)	1	C	N	Right frontal contusion	P	Conservative	D
3	30	F, 7 h	MVA,+	None	Frontal, occipital	6/15	19.2 (4)	2	C	N	Right fronto-temporo-parietal acute SDH with IVH	P	Subocipital craniectomy	D
4	12	M, 2 h	MVA,–	None	Occipital	15/15	2 (2)	2	N	N	No lesion	A	Conservative	GR
5	65	M, 4 h	Fall,+	Abdominal injury	Occipital	12/15	15.4 (3.5)	2	N	N	No lesion	A	Suboccipital craniectomy	MR
6	47	M, 2 h	MVA,+	Fracture left ulna	Occipital	13/15	40 (5)	2	N	C	No lesion	A	Suboccipital craniectomy	MR
7	35	M, 3 h	Fall,–	None	Occipital	13/15	15.4 (3.5)	2	N	N	No lesion	A	Suboccipital craniectomy	GR
8	75	F, 6 h	Fall,+	None	Occiput	7/15	19.2 (4)	2	N	C	No lesion	P	Suboccipital craniectomy	D
9	22	M, 8 h	MVA,+	Fracture femur	Frontal, occipital	5/15	4 (2)	1	C	N	Left frontal EDH, with bilateral temporal contusions, with hydrocephalus	P	Supratentorial craniectomy	D
10	79	M, 7 h	Fall,+	None	Occipital	7/15	15.4 (3.5)	2	N	IVH	No lesion	P	Suboccipital craniectomy	VS
11	25	M, 1 h	MVA,–	None	Occipital	15/15	1 (1)	2	N	N	No lesion	A	Conservative	GR
12	55	M, 3 h	MVA,–	Fracture clavicle	Occipital	14/15	5.2 (3), 5 (2.5)	2 (bilateral)	N	N	Left temporal contusion	A	Conservative	GR
13	55	M, 2 h	MVA,+	Fracture humerus	Occipital	13/15	15.4 (3.5)	1	C	IVH	No lesion	A	Suboccipital craniectomy	MR
14	56	F, 6 h	MVA,+	Fracture femur	Frontal, occipital	7/15	15 (3)	1	C	C	Left frontotemporal contusion	P	Suboccipital and supratentorial craniectomy	D
15	25	M, 5 h	MVA,+	Fracture tibia	Temporal	9/15	1 (1)	2	N	N	Right basifrontal contusion, right temporal EDH, and left temporal contusion, right fronto-temporo-parietal SDH	P	Conservative	SD

(continued)

Table 1 *(continued)*

S. no.	Age (y)	Sex (M/F) Duration[a]	Mode of injury, skull fracture	Associated lesions	Site of impact	GCS (3–15)	Volume, cm^3, size (cm)	Type of location (½)	Basal cisterns (N/C)	Fourth ventricle (N/C/IVH)	Associated supratentorial lesions	SAH (A/P)	Intervention	GOS
16	47	M, 4 h	Fall,+	None	Frontal	3/15	2 (2)	1	C	N	Right frontal contusion with right frontotemporal acute SDH	P	Conservative	D
17	53	M, 6 h	MVA,–	None	Frontal	6/15	5 (2.5)	1	C	N	Bilateral basifrontal contusions, diffuse axonal injury	P	Conservative	D
18	42	M, 6 h	MVA,+	None, chest injury	Frontal	10/15	15.8 (3.5)	2	N	N	Right frontal contusion with mild hydrocephalus	A	Suboccipital craniectomy, ICD	SD
19	54	M, 8 h	Fall,+	None	Temporal	5/15	12 (3)	2	C	N	Left temporoparietal contusion	A	Conservative	D
20	43	M, 4 h	MVA,+	None	Occipital	9/15	4 (2)	1	C	N	Bilateral frontal contusion	P	Conservative	SD
21	32	M, 4 h	MVA,+	None	Occipital	15/15	5.2 (3)	1	N	N	Right frontal contusion	A	Conservative	GR

Abbreviations: A, absent; C, compressed; D, death; DAI, diffuse axonal injury; EDH, extradural hemorrhage; F, female; GCS, Glasgow Coma Scale; GOS, Glasgow Outcome Scale; GR, good recovery; HCP, hydrocephalus; ICD, intercostal tube drainage; IVH, intraventricular hemorrhage; M, male; MD, moderate disability; MVA, motor vehicle accident; N, normal; P, present; SAH, subarachnoid hemorrhage; SD, severe disability; SDH, subdural hemorrhage; VS, vegetative state.
Note: D–Site of impact could not be identified (acceleration–deacceleration injury), skull fracture (+, present;–absent).
[a]Duration: Timing from injury to reporting to hospital in hours

compared with type 1. SAH and supratentorial contusions were relatively higher in type 1 hematomas. There was no significant difference in other factors.

Treatment and Outcome

In our study, 10 patients were treated conservatively and 11 patients underwent surgery. All patients with surgical indications were operated immediately without any delay. Out of 11 patients, 10 patients underwent suboccipital craniectomy and hematoma evacuation, and 1 patient underwent both supratentorial and suboccipital craniectomy. CT head (►**Fig. 3**) suggestive of type 2 hematomas were managed conservatively and those suggestive of type 2 hematoma (►**Fig. 1**) managed by suboccipital craniectomy with hematoma evacuation. Outcome of 21 patients was GR in 6, MR (moderate recovery) in 3, SD in 3, VS in 1, and D in 8 patients. The incidence of poor outcome was 61.9% (13/21).

Prognostic Factors

Frequency, age (range), sex, mode of injury, GCS, hematoma location, basal cisterns, fourth ventricle, SAH, supratentorial contusions, SDH, EDH, IVH, DAI, lesion in posterior fossa, and their relationship with outcome are shown in ►**Table 3**. Agewise distribution shows that there were no females in the favorable group and all females were in the poor outcome group suggesting that females had higher mortality in posterior fossa hematoma.

In our study, GCS at admission was higher in the favorable group as compared with the other group. Brainstem cisterns were compressed in 2 patients in the favorable group as compared with 7 in the poor outcome group. Fourth ventricle was compressed in 1 patient in the favorable group. There was significant presence of SAH in the poor outcome group as compared with the favorable group.

Discussion

These hematomas approximately constitute one-fourth of all posterior fossa traumatic lesions,[3,11] but their frequency is almost between SDH and EDH.[9,12] Sato et al[13] established 0.7% incidence of cerebellar contusions. Nagata et al reported their frequency to be between 0.6 and 0.82%. The incidence observed in our series was 0.41%, which correlates well with other figures. Various mechanisms are responsible for these

Table 2 Comparison between type 1 and type 2 hematomas

Factors	Type 1	Type 2
Total patients	8/21	13/21
Age (range), y	22–56	12–79
Sex (M/F)	7/1	11/2
Mode of injury (Fall/MVA)	1/7	5/8
Skull fracture (present/absent)	7/1	8/5
Hematoma volume (cm^3), mean	6.8	14.1
Basal cisterns (N/C)	1/7	11/2
Fourth ventricle (N/C/IVH)	6/1/1	9/2/2
SAH (P/A)	6/2	4/9
Contusion (P/A)	7/1	4/9
EDH (P/A)	1/7	1/12
SDH (P/A)	1/7	1/12
IVH (P/A)	0/8	1/12
DAI (P/A)	1/7	0/13
SDH in posterior fossa (P/A)	0/8	1/12

Abbreviations: A, absent; C, compressed; DAI, diffuse axonal injury; EDH, extradural hemorrhage; F, female; IVH, intraventricular hemorrhage; M, male; MVA, motor vehicle accident; N, normal; P, present; SAH, subarachnoid hemorrhage, skull fracture (+, present;–, absent); SDH, subdural hemorrhage.

hematomas but remain unclear. These could be coup and countercoup injuries. Coup injuries are thought to be the most frequent mechanism.[1,7,14] Countercoup injuries are considered to be rare.[15]

Another mechanism for traumatic intracerebellar hematoma involves acceleration and deacceleration injuries.[16] In our series, 7/21 patients had coup (33.3), 4/21 (19.0) had countercoup injuries, and the rest (47.6) had acceleration–deacceleration injuries. So, this leads us to the conclusion that acceleration and deacceleration is a common cause as reported previously by Takeuchi et al.[17]

Delayed or evolving hematoma has been reported previously.[18,19] In our series, no cases of delayed or evolving hematoma was reported as repeat scan was done within 6 hours of admission as mentioned previously. However, repeat CT examination is necessary, as delayed hematomas can develop.

The incidence of poor outcome in previously published series were around 20 to 100% (average 60%),[2] and in our series, it was 61.9%. The prognosis was better in good GCS patient and in patients who were promptly operated with surgical indication.[20] Summary of published series of posterior fossa hematomas are depicted in ►**Table 4**.

D'Avella et al have described protocol for traumatic intracerebellar hematomas as follows:

1. Conservative approach is a treatment option for noncomatose patients with intracerebellar clots 3 cm, except when associated with other EDH or SDH of posterior fossa.
2. Surgery should be done for any patient with any clot > 3 cm.

In our study, surgery was done in 11 patients, out of which 7 patients survived and 4 expired. In our series, type 1 hematomas were more frequent in the poor outcome group due to easy compression of brainstem or involvement of deep cerebellar nuclei which lead to bad prognosis.[17]

Table 3 Factors related to outcome in traumatic intracerebellar hematoma

Factors	Favorable outcome group	Poor outcome group
Frequency (patients), M/F	13/21, (13/0)	8/21, (5/3)
Mode of injury (fall/MVA)	3/10	3/5
Skull fracture (present/absent)	8/5	7/1
GCS (range)	7–15	3–7
Duration (timing from injury sustained to reporting to hospital), hours, median[a]	4–5 h	6 h
Hematoma location (½)	3/10	5/3
Hematoma volume (mean)	12.1	10.05
Basal cisterns (N/C)	11/2	1/7
Fourth ventricle (N/C/IVH)	9/1/3	6/2/0
SAH (P/A)	3/10	7/1
Supratentorial contusion (P/A)	6/7	5/3
HCP (hydrocephalus)	1/12	1/7
Supratentorial EDH (P/A)	1/12	1/7
IVH (P/A)	0/13	1/7
DAI (P/A)	0/13	1/7
SDH in posterior fossa	1/12	0/8

Abbreviations: A, absent; C, compressed; DAI, diffuse axonal injury; EDH, extradural hemorrhage; F, female; GCS, Glasgow Coma Scale; HCP, hydrocephalus; IVH, intraventricular hemorrhage; M, male; MVA, motor vehicle accident; N, normal; P, present; SAH, subarachnoid hemorrhage; SDH, subdural hemorrhage.
[a]Duration: Timing from injury to reporting to hospital in hours, skull fracture (+, present; – absent).

Various factors influencing surgical decision were patients with poor GCS, advanced age, poor general condition, clinical signs of early brainstem damage, bilateral cerebral clots, and associated multiple supratentorial intracranial hemorrhages with evidence of coagulopathy. These factors reflect the peculiar heterogenecity of clinical radiological picture of this subset of posterior fossa hematomas.[21]

HCP is usually not associated with traumatic intracerebellar clots.[11,18] Karasawa et al[3] mentioned of acute HCP in 20% of intracerebellar hematomas, while in our series it was 9.52%. The timing of reaching hospital is important and affects overall morbidity and mortality of patients. Most of the patients in the favorable outcome group reported to hospital within 4 to 5 hours, whereas those in the poor outcome group had reported more than 4 to 8 hours. There was single case of posterior fossa hematoma with associated SDH in our series (►**Fig. 1C**). This suggests that associated lesions in posterior fossa hematoma such as SDH and EDH are less.

Posterior fossa hematoma is rare in children and incidence of cerebellar hematoma is much rarer.[22] Most frequent mode

Table 4 Summary of selected published series of intracerebellar hematoma and contusions

Author, year	No. of cases	Notes	Poor outcome (%)
Tsai et al, 1980	14	In 2 cases, associated with brainstem injury	85
Pozzati et al, 1982	7	All isolated clots	42
St John et al, 1986	3	One case associated with AEH, one with ASH	60
Hamasaki et al, 1987	4	3 cases isolated with ASH and one with both ASH and AEH	100
Sato et al, 1987	8	2 cases with concomitant diffuse cerebral contusions	50
Zuccarello et al, 1982	5	All children	20
Nagata et al, 1991	14	All delayed hematomas, one personal case, and literature review	64
Karasawa et al, 1997	13	11 cases with associated supratentorial ICH, SAH, or ASH; 2 cases with associated infratentorial ASH	54
D'Avella et al, 2001	18	8 cases of isolated intracerebellar clots	50
Present series	21	9 cases of isolated posterior fossa hematoma	61

Abbreviations: AEH, acute extradural hematoma; ASH, acute subdural hematoma; ICH, intracerebellar hematoma; SAH, subarachnoid hemorrhage.

of injury is fall followed by road traffic accident.[23] Trauma is a leading cause of childhood head injury, with Center for Disease Control, stating about half a million emergency department visits in the United States is for traumatic brain injury for children aged 0 to 14 years, with 0 to 4 years being the most vulnerable group.[24] Although most cases are seen in young adults, Zuccarello et al[25] reported traumatic posterior fossa hemorrhage in children also. Wright[9] reported 6 cases and Tsai et al[11] reported 2 cases of children in their series. Similarly, in our series, we had a single child. Management of such cases is similar to adult posterior fossa hematoma.

Suboccipital craniectomy with hematoma evacuation is the most preferred surgical approach in posterior fossa.[26,27] Clinical with radiological features, types of hematomas, prognostic factors, and final outcome are discussed and mentioned in ►**Tables 1–3**. In summary, patients who reported within 4 hours of trauma, with initial GCS score of between 9 and 15, absent SAH, normal basal cisterns, and normal fourth ventricle had better survival rate. However, older age group and females fared worse in our study as shown in ►**Tables 1–3**. In severely ill subjects, surgery should be individualized for each patient with reasonable salvageability.

Conclusion

Traumatic cerebellar hematoma is a life-threatening condition that requires timely management. Nowadays, with wider availability of CT and intensive neuromonitoring, smaller hematomas can be managed conservatively and larger ones with surgical evacuation in clinically deteriorating patients.

The factors correlated with patient outcome were age, sex, mode of injury, GCS at admission, associated intracranial hematomas, associated SAH, hematoma volume, hematoma location, basal cisterns, status of fourth ventricle, and associated multiple injuries on other body parts. It is hereby concluded that timely surgical intervention should be employed whenever indicated without delay. This study had some limitations, like sample size was relatively small to determine the actual prognostic factors. As these lesions are associated with higher morbidity and mortality, it should be evaluated further to explore pathophysiology for better clinical outcome of patients.

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Conflict of Interest
None declared.

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