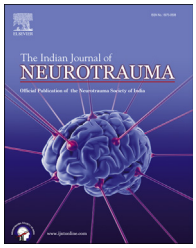
Available online at www.sciencedirect.com

SciVerse ScienceDirect

journal homepage: www.elsevier.com/locate/ijnt



Review Article

Traumatic cerebellar haematoma: A review

Ashis Patnaik [a,*], *Ashok Kumar Mahapatra* [b]

[a] *Assistant Professor, Dept. of Trauma & Emergency, All India Institute of Medical Sciences (AIIMS), Bhubaneswar, Odisha, India*
[b] *Director, All India Institute of Medical Sciences (AIIMS), Bhubaneswar, Odisha, India*

ARTICLE INFO

Article history:
Received 30 March 2013
Accepted 26 April 2013
Available online 18 May 2013

Keywords:
Traumatic
Cerebellum
Haematoma

ABSTRACT

Haematomas in the cerebellum are usually caused by hypertension. Traumatic cerebellar haematomas, although rare, are important cause of morbidity and mortality. This can be prevented by timely intervention to evacuate the haematoma. However, recently the protocol is gradually changing from only surgery approach to either surgery or conservative method. We discuss and review the literature for the incidence, mechanism of injury, clinical features, investigations, treatment and outcome for the traumatic cerebellar haematoma in detail.

1. Introduction

Traumatic cerebellar haematomas are rare in comparison to their counterpart, haematomas caused by the hypertension, in the cerebellum. They also include the cerebellar contusions. Although they constitute a small fraction of head injuries, they often lead to devastating outcomes including death. This is due to small capacity of the posterior fossa to accommodate any further increase in volume, due to haematoma. They may be isolated lesions or may be associated with other lesions of the posterior fossa such as subdural haematoma, extradural haematoma or supratentorial lesions. Traumatic haematomas of cerebellum may present with acute onset after trauma or in delayed manner (delayed traumatic intracerebellar haematoma). Various factors such as location, volume of haematoma, initial GCS score, status of fourth ventricle and cisterns etc. control the final outcome of traumatic cerebellar haematomas. Management of such haematomas has changed from all surgery approach to both conservative and surgery depending upon the haematoma characteristics and condition of the patients.

2. Incidence

In comparison to post-traumatic intracerebral haematomas, traumatic cerebellar haematomas are rare and are reported in <1% of all head injuries.[1,2] Few numbers of case series have been published in the literature describing these rare lesions of the posterior fossa.[1–11] Most of these have found the incidence of traumatic cerebellar haematomas to be less than 1% (Table 1). Liu,[12] reported the incidence of traumatic intracerebellar haematoma to be 3.7% of total intracranial haematomas, in his series.

Mechanisms responsible for these traumatic lesions remain unclear. They may be caused by motor vehicular accidents or by fall. Takeuchi et al,[3] categorized the types injuries causing traumatic cerebellar haematomas into:

* *Corresponding author.*
E-mail address: drashispatnaik@gmail.com (A. Patnaik).
0973-0508/$ – see front matter
http://dx.doi.org/10.1016/j.ijnt.2013.04.009

Table 1 – Incidence traumatic cerebellar haematoma.

Author	Traumatic cerebellar haematoma (% of head injury)
Takeuchi et al[3]	0.4% (17/4315)
D'Avella et al[4]	0.54% (18/3290)
Sato et al[5]	0.7% (8/1176)

1. Coup injuries, where the occiput was the site of impact – most frequent type.
2. Countercoup injuries, with a site of impact other than the occiput such as frontal or temporal.
3. Acceleration–deceleration injuries.

All age groups can be affected by traumatic cerebellar haematoma. Although young adults are more susceptible to these injuries due to motor vehicle accidents caused by rash driving and sports related fall, Zuccarello et al,[7] reported 5 cases of cerebellar injury in their series of 10 cases of traumatic posterior fossa haemorrhage in children.

3. Clinical signs and symptoms

Traumatic cerebellar haematoma can be totally asymptomatic to be followed by sudden and rapid deterioration of the neurological status.[13] This is in correlation with the small reserve capacity of the posterior fossa. Symptomatic cases can present with

1. Features of cerebellar dysfunction like ataxia, nystagmus.
2. Features of increased intracranial pressure like headache, diminished consciousness, vomiting, pupillary irregularity, deranged respiratory pattern.

Yokota et al,[14] reported 2 cases of traumatic cerebellar injury, one complicated with medial longitudinal fasciculus (MLF) syndrome and the other with cerebellar mutism. They suggested that the MLF syndrome was due to the neurovascular injury caused by the trauma. Cerebellar mutism was ascribed to the injury to cerebellar vermis or hemisphere.

4. Investigations

CT scan is the initial and gold standard investigation to evaluate the traumatic cerebellar haematoma. It shows the location, whether in superficial (Fig. 1) or deep part of cerebellum (Fig. 2), size of haematoma, status of 4th ventricle and cisterns, presence of hydrocephalus (Fig. 3), associated posterior fossa EDH, SDH or any supratentorial lesion (Fig. 4). It can be used to follow-up the changing radiological picture when the conservative approach is being taken or to know the cause in case of sudden neurological deterioration. Haematoma volume is determined using the ABC/2 formula, where A was the greatest haemorrhage diameter by CT, B was the diameter 90° to A, and C was the approximate number of CT slices with haemorrhage, multiplied by the slice thickness.[15] However, it may sometimes fail to show small cerebellar contusions due to less sensitivity of CT scan in posterior fossa.

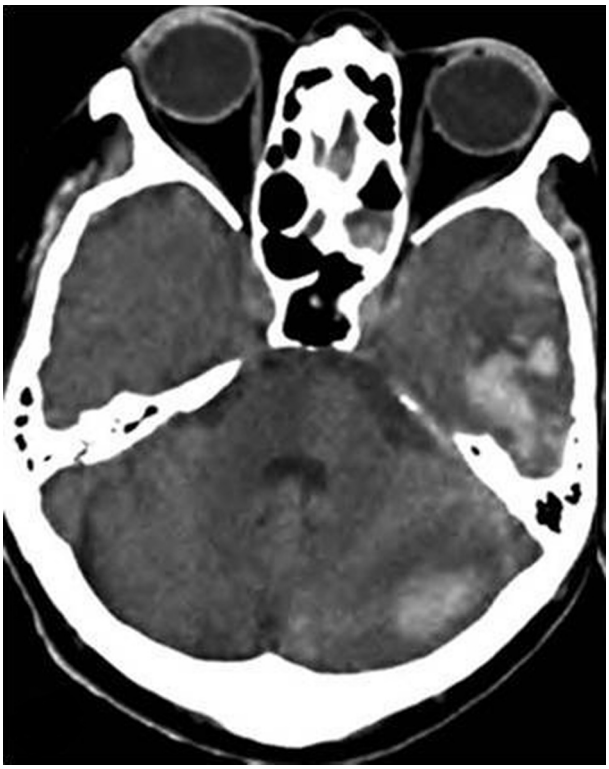


Fig. 1 – Haematoma located in the superficial cortical region of cerebellum.

MRI brain is usually not required as most of the clinically significant haematomas are visible in CT scan. Plain X-rays do not provide any specific picture except fracture lines.

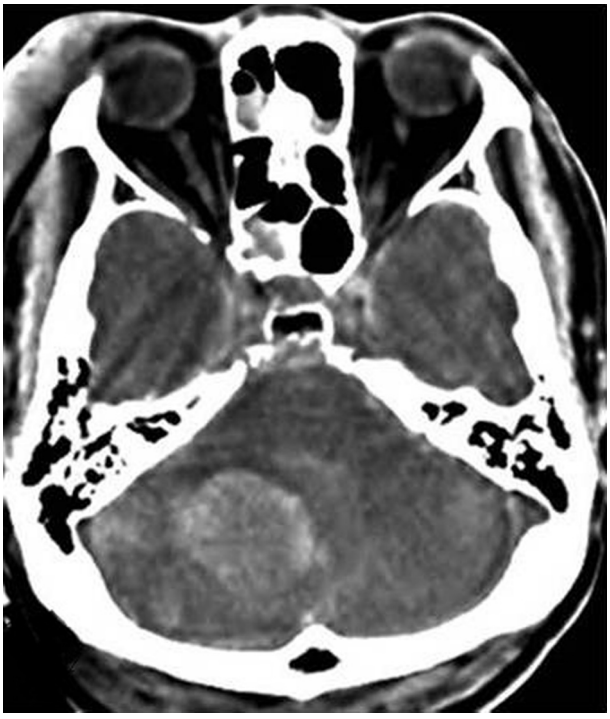


Fig. 2 – Haematoma in the central vermian area with compression of 4th ventricle.

5. Treatment

Buczek et al,[16] suggested that although before the introduction of CT the prevailing view was that all intracerebellar haematomas should be treated surgically, the present opinion is that some of them meeting certain criteria (small size, peripheral situation in the cerebellar hemispheres laterally to the midline, absence of symptoms of intracranial hypertension, and the possibility of frequent CT control) may be treated successively without surgical intervention with advantage to the patient.

Treatment of traumatic cerebellar haematomas falls into two categories:

1. Conservative.
2. Surgical evacuation.

5.1. *Conservative*

Conservative approach can be considered for following conditions:

1. Fully conscious patient.
2. Superficial location.
3. Maximum diameter less than 3 cm or volume less than 15 ml.

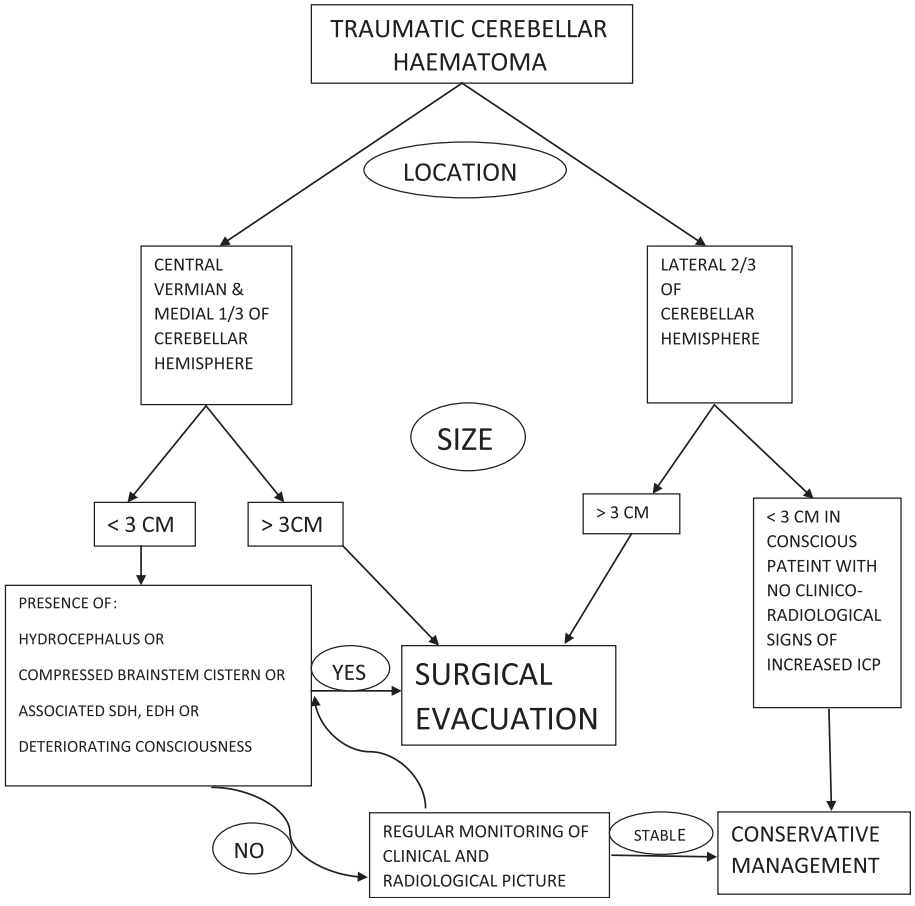
Patients who fulfil all the above criteria, should be regularly followed up clinico-radiologically to detect any new haematoma development, increase in the size haematoma or compression of 4th ventricle or brainstem cisterns.

5.2. *Surgical evacuation*

Surgical evacuation is recommended for all patients with:

1. Larger clots with size >3 cm.
2. Larger haematomas causing cisternal and 4th ventricle compression.
3. Associated extradural or subdural haematomas of posterior fossa.
4. Associated acute hydrocephalus.

Surgical Approach: It has been have suggested that suboccipital craniectomy with haematoma evacuation is preferable to craniotomy, because the posterior fossa has less room to accommodate any postoperative bleeding than supratentorial sites after evacuating the haematoma.[17,18]



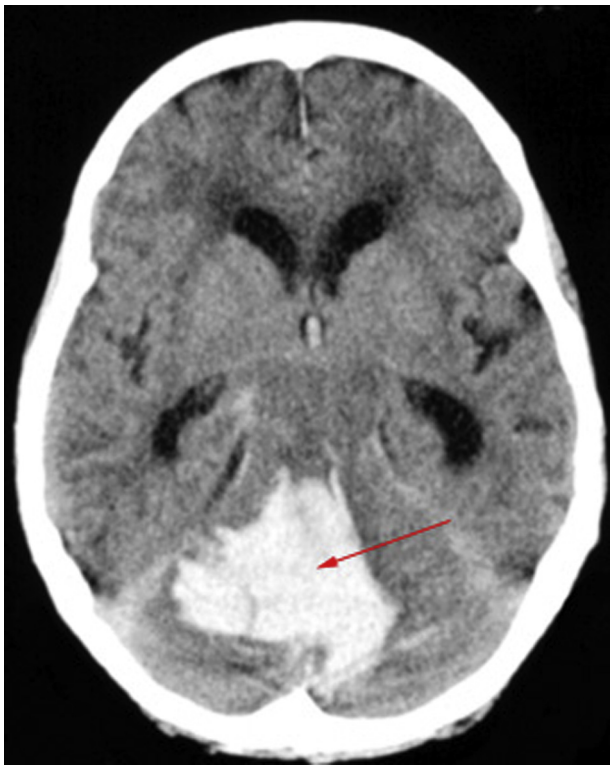


Fig. 3 – Large haematoma in central cerebellar region with hydrocephalus as evident by dilated frontal and temporal horns.

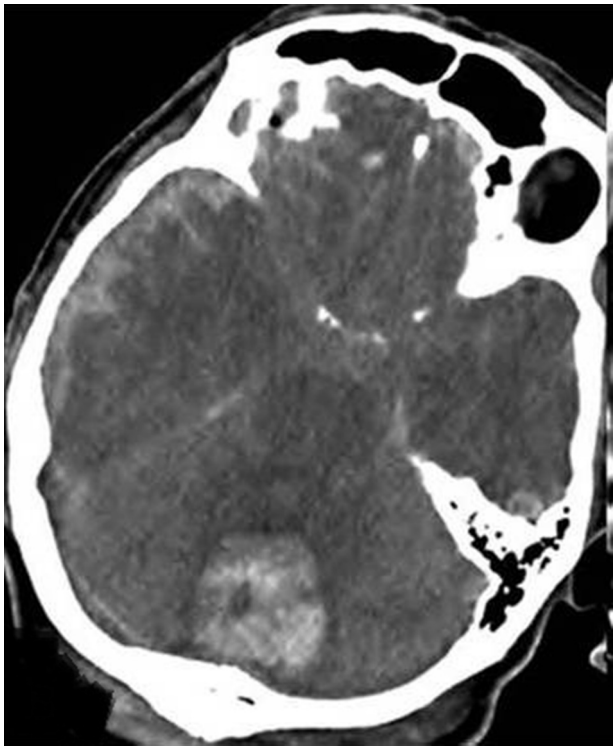


Fig. 4 – Cerebellar haematoma with associated subdural and subarachnoid haemorrhage in right side temporal region.

6. Outcome

Outcome in traumatic cerebellar haematomas is dependent upon certain factors (Table 2):

6.1. *Initial GCS score*

Initial GCS score at the time admission is the most important factor controlling the outcome. Patients with GCS score more than or equal to 8 have a good outcome, either managed conservatively or surgically. d'Avella et al,[1] reported of 81 patients of traumatic cerebellar haematomas, managed between 1996 and 1998, at 13 Italian neurosurgical centres. They divided them into Group I, consisting of 39 patients, who had initial GCS score of 8 or more. Group II consisted of 42 patients with GCS less than 8. They observed a favourable outcome in 95% of cases of group I and only in 19% of group II patients. Previously, they had also reported complete recovery in 8 of their patients, who had initial GCS score of 9 or more. Outcome was poor in 90% of cases of patients who had GCS of less than 9.[4]

6.2. *Haematoma location*

Location of haematoma in the cerebellum can be divided into deep and superficial location. Vermis and the innermost one third of each hemisphere constitute the deep portion.[19,20] Takeuchi et al,[3] classified the haematoma locations as either type 1 (haematoma localized in the superficial two-thirds of the hemisphere) or type 2 (haematoma extending into the vermis or the innermost third of the hemisphere). They reported that the larger haematoma volume, compressed cisterns around brainstem and the abnormal fourth ventricle were associated with type 2 haematoma location, which were statistically significantly ($p = 0.027$, 0.009, 0.035 respectively).

Deep cerebellar haematomas, arising from vermis and inner part of the cerebellum tend to compress the 4th ventricle and brainstem early and easily, as they are close to it. This can lead to hydrocephalus and raised intracranial pressure earlier and more frequently than the superficial haematomas, which produce the same effect only when they are large enough to encroach upon the 4th ventricle at a later stage. Involvement of deep cerebellar nuclei, which are the principal output tract of cerebellum, could lead to poor prognosis. Thus, the superficial cerebellar haematomas have a better outcome, irrespective of managed either conservatively or surgically.

Table 2 – Factors correlating with outcome.

Initial GCS score
Haematoma volume
Haematoma location
Status of the brainstem cisterns and the fourth ventricle
Presence of associated SAH
Associated supratentorial traumatic lesions.[1]

6.3. *Haematoma volume*

More the haematoma volume, earlier is the appearance of signs of increased intracranial pressure and consequently the less favourable outcome. Deep cerebellar locations are also frequently associated with larger haematoma volume than their superficial counterpart. Takeuchi et al,[3] observed that the average haematoma volume of 12.4 (+/−) 11.9 cm^3 in their unfavourable outcome group in comparison to 2.2 (+/−) 2.6 cm^3 in the favourable outcome group ($p = 0.025$).

6.4. *Status of brainstem cisterns and fourth ventricle*

The cisterns around the brainstem are absent or compressed in cases of cerebellar haematoma, where there is excessive pressure within the posterior fossa causing them to be compressed or even absent. Consequently, these cases are associated with earlier neurological deterioration and poor outcome.

Fourth ventricle anatomy also correlates with pressure status within the posterior fossa. When compressed or completely absent, it leads to obstruction to the passage of CSF pathway and impending pressure on the brainstem, hydrocephalus. All this can lead poorer outcome in the long term. Takeuchi et al,[3] reported the association between abnormal brainstem cistern and fourth ventricle and the outcome, ($p = 0.002$, 0.010 respectively), which was significant.

6.5. *Presence of SAH*

Presence of subarachnoid haemorrhage along with the cerebellar haematoma is associated with the poorer outcome, probably due to the ischaemic effects of vasospasm induced by the SAH. Takeuchi et al,[3] observed their association to be significant ($p = 0.003$).

6.6. *Presence of supratentorial lesions*

d'Avella et al,[1] reported that the associated supratentorial lesions negatively affect the outcome of traumatic cerebellar haematomas. However, Takeuchi et al,[3] could not find this association as significant.

7. Delayed traumatic intracerebellar haematoma (DTICH)

Delayed traumatic intracerebellar haematoma (DTICH) is a clinical entity in which haematoma develops several hours after trauma. Few numbers of such cases have been reported in the literature.[14,21–25] The delay between the trauma and haematoma occurrence ranges from 4 h to 4 days. The mechanism responsible for delayed intraparenchymal haematoma is still controversial. Evans and Scheinker[26] proposed a sequence of vasoparalysis in the contused brain causing increased endothelial cell permeability followed by diapedesis and extravasation resulting in the formation of intracerebral haematoma. Tsubokawa et al[27] proposed that hyperoxidation caused by local cerebral circulation disorders was a possible mechanism of DTICH.

8. Conclusion

Traumatic cerebellar haematoma is a rare, but potentially life threatening condition, which requires careful and intensive management. Although most of them present acutely and shortly after the trauma, they may also present treacherously long hours after the trauma, in a delayed fashion. With wide and easy availability of CT scan and regular neurological status monitoring, conscious, small, hemispheric haematomas can be serially followed up avoiding unnecessary surgical evacuation. Large, midline or deep cerebellar haematomas or patients with deteriorating neurological status require definite surgical evacuation to save the life. Initial GCS score, haematoma size and volume, status of cisterns surrounding brainstem and 4th ventricle are the ultimate prognostic markers irrespective of the treatment modality.

Conflicts of interest

All authors have none to declare.

REFERENCES

1. d'Avella D, Servadei F, Scerrati M, 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;86:629–632.
3. Takeuchi S, Takasato Y, Masaoka H, Hayakawa T. Traumatic intra-cerebellar haematoma: study of 17 cases. *Br J Neurosurg*. 2011;25:62–67.
4. D'Avella D, Cacciola F, Angileri FF, et al. Traumatic intracere bellar hemorrhagic contusions and hematomas. *J Neurosurg Sci*. 2001;45:29–37.
5. Sato K, Hinokuma K, Matsuzawa Y, et al. Clinical study of traumatic cerebellar contusion. *No Shinkei Geka*. 1987;15: 1285–1289.
6. Pozzati E, Grossi C, Padovani R. Traumatic intracerebellar hematomas. *J Neurosurg*. 1982;56:691–694.
7. Zuccarello M, Andrioli GC, Fiore DL, Longatti PL, Pardatscher K, Zampieri P. Traumatic posterior fossa haemorrhage in children. *Acta Neurochir (Wien)*. 1982;62:79–85.
8. St John JN, French BN. Traumatic hematomas of the posterior fossa. A clinicopathological spectrum. *Surg Neurol*. 1986;25: 457–466.
9. Hamasaki T, Yamaki T, Yoshino E, Higuchi T, Horikawa Y, Hirakawa K. Traumatic posterior fossa hematoma. *No To Shinkei*. 1987;39:1083–1090.
10. 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.
11. Jagodziński Z, Dabrowska E, Buczek M. Post-traumatic cerebellar hematomas. *Neurol Neurochir Pol*. 1990;24:315–321.
12. Liu K. Characteristics of diagnosis and treatment of traumatic intracerebellar haemorrhage. *Zhonghua Wai Ke Za Zhi*. 1997;35:166–167.
13. Sokol JH, Rowed DW. Traumatic intracerebellar haematoma. *Surg Neurol*. 1978;10:340–341.
14. Yokota H, Mizunari T, Kuzuhara M, et al. Traumatic delayed intracerebellar hematoma. *Neurol Med Chir*. 1988;28:886–890.

15. Kothari RU, Brott T, Broderick JP, et al. The ABCs of measuring intracerebral hemorrhage volumes. *Stroke*. 1996;27: 1304–1305.
16. Buczek M, Jagodziński Z, Kopytek M, Dabrowska E. Conservative treatment of post-traumatic intracerebellar hematoma. *Wiad Lek*. 1989;42:550–555.
17. Pollak L, Rabey JM, Gur R, Schiffer J. Indication to surgical management of cerebellar hemorrhage. *Clin Neurol Neurosurg*. 1998;100:99–103.
18. Aghi M, Ogilvy CS, Carter B. Surgical management of intracerebral hemorrhage. In: Roberts DW, Schmidek HH, eds. *Schmidek and Sweet's Operative Neurosurgical Techniques Indications, Methods, and Results*. 5th ed. Philadelphia: Saunders/Elsevier; 2005:1061–1074. vol. 2.
19. Dimitrova A, Weber J, Redies C, et al. MRI atlas of the human cerebellar nuclei. *Neuroimage*. 2002;17:240–255.
20. Deoni SC, Catani M. Visualization of the deep cerebellar nuclei using quantitative T1 and rho magnetic resonance imaging at 3 Tesla. *Neuroimage*. 2007;37:1260–1266.
21. Nagata K, Ishikawa T, Ishikawa T, et al. Delayed traumatic intracerebellar hematoma: correlation between the location of the hematoma and the pre-existing cerebellar contusion–case report. *Neurol Med Chir*. 1991;31:792–796.
22. Seida M, Ito U, Kito K, Tomida S, Inaba Y. Traumatic cerebellar hematoma in children. Report of two cases. *Neurol Med Chir*. 1986;26:318–322.
23. Shigemori M, Shirahama M, Tokutomi T, Hara K, Yamamoto F. Delayed development of traumatic intracerebellar hematoma: case report – significance of serial CT scan and management. *Neurol Med Chir*. 1981;21:427–431.
24. Tibbs PA, Goldstein SJ, Smithson Jr JR. Delayed traumatic intracerebellar hematoma. *Surg Neurol*. 1981;16:309–311.
25. Martin AJ, Thomas NW. Evolving traumatic cerebellar hematoma. *Neurology*. 2001;57:1565.
26. Evans JP, Scheinker IM. Histologic studies of the brain following head trauma, II. Posttraumatic petechial and massive intracerebral hemorrhage. *J Neurosurg*. 1946;3: 101–113.
27. Tsubokawa T, Yamada J, Tomizawa N, Goto T, Shinozaki H, Moriyasu N. Classification of traumatic intracerebral hematoma by repeated CT -scan and clinical course. *Neurol Med Chir*. 1979;19:1127–1137.