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Management of epidural hematomas of the posterior Cranial Fossa

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1. Introduction

Epidural hematomas of the posterior fossa (EDHPF) are rare lesions associated with trauma to the posterior skull base. They have been reported to occur in 5.7–9.9 % of traumatic epidural hematomas.[1,2] The advent of Computed Tomography (CT) has facilitated the diagnosis of EDHPF. It has also contributed to a substantial drop in mortality associated with these lesions, with 40 % mortality in the pre-CT era to 15.6 % immediately after the introduction of CT imaging.[3] Generally, larger contemporary published series have treated the majority of these lesions surgically.[2,4–7] Conservative management has mostly been reserved for only smaller-size EDHPF.[2,4–6,8] These lesions are believed to have a venous origin typically from a tear in the venous sinus or the site of overlying skull fracture.[1,2,5,9–12] Due to their bleeding site being typically venous, our approach to these lesions by default has been conservative. Therefore, we present our results of management of EDHPF.

2. Methods

Between 2007 and 2018 all blunt, acute-to-subacute (diagnosis of EDHPF within index admission and patient had to be admitted to our facility within 96 h of index trauma) traumatic EDHPF were prospectively collected. Any chronic presentation of EDHPF was excluded. Medical records, radiographic records, operative notes, and clinic documentations were applied to retrieve data retrospectively.

Our method in conservative management of EDHPF has focused on early head elevation to greater than 30°. Furthermore, we paid early attention to minimize or alleviate any constriction to venous outflow from the head by removing any constriction of the neck vessels. We released any bands or collars that could have impeded venous outflow.

3. Results

3.1. Demographics

A total of 30 consecutive patients were enrolled in this study. Of these patients, 22 were male and 8 female, with an average age of 17.1 years (range 2–47 years). A total of 19 patients were younger than 18 years of age. The mechanisms of trauma were vehicular accidents (n = 14), falls (n = 9), assaults (n = 3), and other (n = 4).

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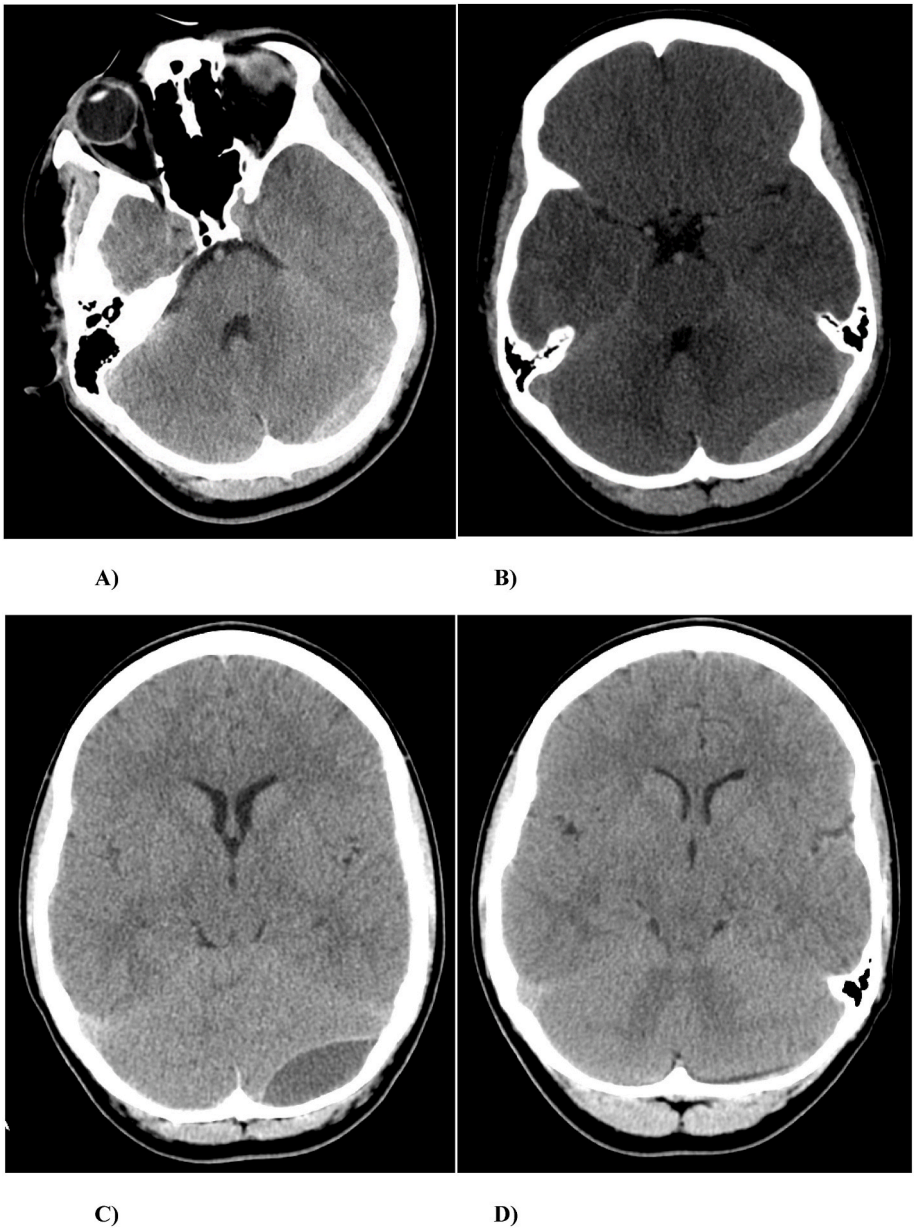


Fig. 1. (A–D) A) A 14-year-old male fell off a driving truck and presented with headache. He was otherwise neurologically intact. Initial CT head revealed a left EDHPF. B) A follow up CT head done 3 h later disclosed expansion of the hematoma, which was managed conservatively, since the patient did not worsen clinically. Eventually after 1 week of conservative management in the hospital the patient was discharged. C) On day 17 after the trauma a CT head disclosed further expansion of the hematoma but with breakdown of the clot (hypodense hematoma). D) Finally, the hematoma resorbed on CT head 6 weeks following the trauma, with the patient remaining stable and unchanged clinically throughout the period of conservative treatment.

Patients presented within 24 h from the trauma in 27 cases, within 48 h in 2 cases, and a single case presented at 4 days post-trauma.

Clinical symptoms consisted of headache ($n = 11$), nausea with or without vomiting ($n = 6$), initial loss of consciousness ($n = 7$), diminished mental status ($n = 9$), coma ($n = 8$), ataxia ($n = 2$), failure to thrive ($n = 1$), diplopia ($n = 1$), and seizure ($n = 1$).

4. Radiological data

All EDHPF were diagnosed using CT scanning of the head. Half of EDHPF extended supratentorial. In 27 patients the EDHPF was located lateral to midline over the cerebellar hemisphere, in 2 it crossed midline, and in a single case it was located over the clivus.

The EDHPF was not present on the initial image in 6 patients. In 14 patients an immediate (within hours or days) progression of the hematoma was documented (Fig. 1). The thickness of the EDHPF ranged from 2 mm to 29 mm. Thickness of less than 10 mm was present in 17 patients.

Hydrocephalus was registered in 3 instances, of which in one case the hydrocephalus was not considered to be caused or aggravated by the EDHPF. Basal cisterns were compromised in 7 patients, of which in 5 cases the EDHPF was not considered causing or significantly aggravating the obstruction of the basal cisterns. Associated traumatic intracranial injuries were noted in 20 patients and consisted of subarachnoid hemorrhage ($n = 13$), subdural hematoma ($n = 4$), cerebral contusion ($n = 14$), cerebrospinal fluid leak ($n = 1$), stroke ($n = 1$), intraventricular hemorrhage ($n = 2$), and diffuse brain edema ($n = 1$).

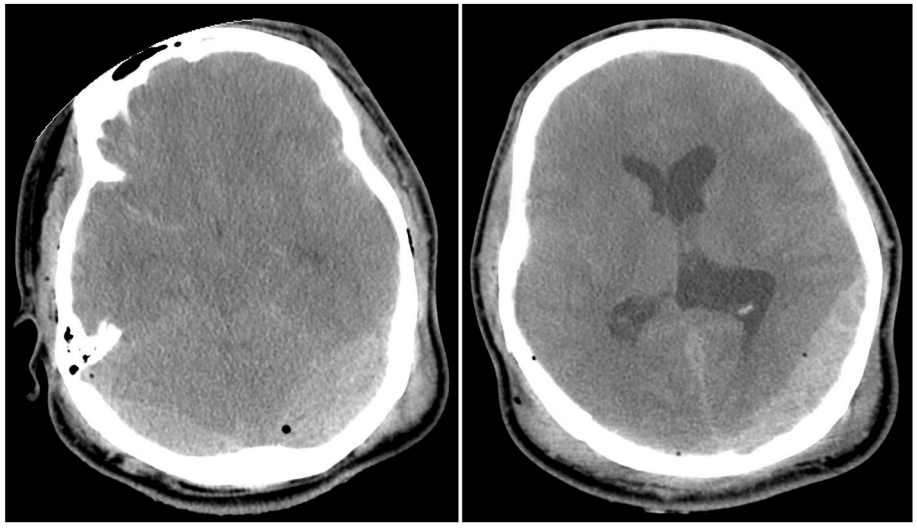
Table 1
Initial GCS score and method of treatment.

Initial GCS Score*	Patients Treated Surgically	Patients Treated Conservatively
13–15	1	20
9–12	2	0
3–8	6	1

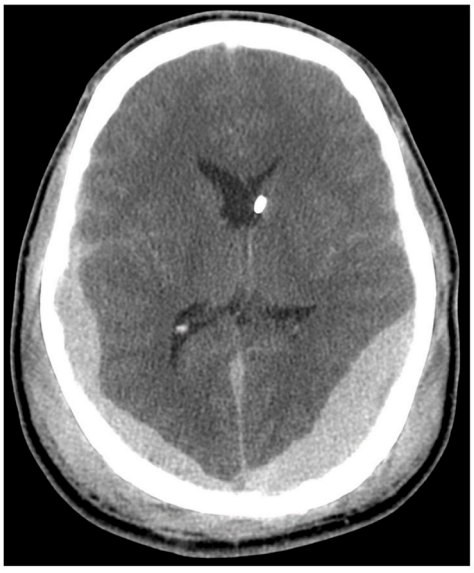
*The initial GCS score upon presentation is divided into three categories resembling mild (GCS 13–15), moderate (GCS 9–12), and severe (GCS 3–8) head injury. As depicted in the table, most of the surgical interventions (craniotomy and/or EVD placement) were performed in the groups with moderate and severe head injury, whereas most patients with mild head injury (GCS 13–15) underwent conservative therapy.

4.1. Neurosurgical intervention

Neurosurgical interventions were applied in 9 patients. Four patients had a procedure specifically to address the EDHPF either indirectly by placing an external ventricular drain (EVD) to alleviate hydrocephalus caused by the EDHPF ($n = 2$), or directly by evacuating the EDHPF ($n = 2$). In the cohort that was operated on to directly evacuate the EDHPF, one case was done primarily to evacuate a large supratentorial component of the EDHPF that was associated with elevated intracranial pressures. Intra-operatively, laceration to the transverse sinus as source of bleeding was identified. The laceration was treated in onlay fashion by placing an oversized piece of synthetic dural substitute graft epidurally over the site of tear, which readily stopped the bleeding with a small amount of gentle compression applied to region. The other patient underwent evacuation of the EDHPF associated with radiographic

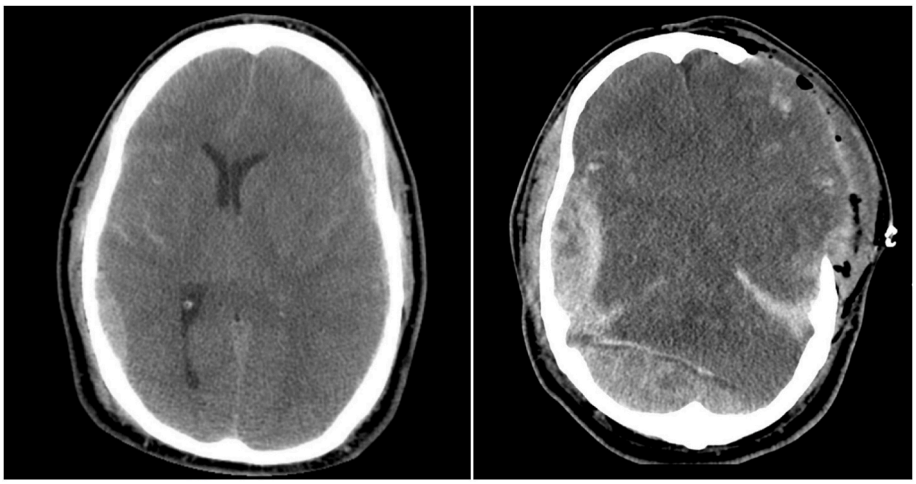


A) B)



C)

Fig. 2. (A–C) A) A 21-year-old male fell out of a driving vehicle and presented in a deeply comatose state with a GCS score of 3, anisocoric, non-reactive pupils, without other brain stem reflexes, but present agonal respiratory efforts. CT revealed an EDHPF along with diffuse brain edema, traumatic frontal contusion, and subarachnoid bleed, extensive skull base fractures involving bilateral petrous bones, B) as well as early developing hydrocephalous. C) An immediate external ventricular drain was placed to treat the associated hydrocephalus secondary to spinal fluid flow obstruction caused by the EDHPF. However, due to rapid progression of the EDHPF and the overall poor prognosis for recovery the family elected against further procedures and the patient died from his head injury.



A) B)

Fig. 3. (A and B) A) A 38-year-old male presented in coma with a GCS score of 7. CT head revealed an acute left hemispheric subdural hematoma (SDH) along with a small right posterior temporal epidural hematoma (EDH). Patient underwent an emergent left craniectomy and evacuation of the SDH. During this procedure a massive herniation of brain through the cranial defect was noticed. B) An immediate post operative repeat CT revealed a massive expansion of the right EDH with severe infratentorial extension and brain compression. The patient died shortly after from this EDH.

progression. In this case, it was specifically the patient's and family's request to operate. It was felt at the time that both options (surgery as well as conservative therapy) were equally reasonable.

In five patients, surgery was performed to primarily address other associated intracranial injuries. In one instance an associated EDHPF was also evacuated, but solely because it blocked access to a cerebellar contusion. In other terms, there was otherwise no need to evacuate the associated EDHPF.

In general, neurosurgical interventions (craniotomies and/or EVD placement) for head injuries in our patient population were typically applied in patients with lower Glasgow Coma Scale (GCS) scores (Table 1).

5. Patient outcomes

Follow-up was present in 21 patients (mean 22.4 months; range 1–85 months). Five patients were lost to follow-up after discharge. In these cases, their neurological status at time of discharge was used for their outcome evaluation.

At discharge and/or last follow up (range 1–85 months), a total of 23 patients were alive and fully neurologically intact. Two patients were alive with minor neurological deficit (facial palsy in one case and anosmia in the other). One patient remained in a permanent comatose state, and 4 patients died of their head injuries (13.3 % mortality). All patients that died or stayed in coma presented in coma.

In 2 instances the patients' deaths were caused or aggravated by their EDHPF. One patient presented in a deeply comatose state with non-reactive anisocoric pupils and agonal respiratory effort. GCS score was 3. An immediate EVD was placed to treat hydrocephalus associated with EDHPF. This failed to improve the patient's clinical condition. Surgery was offered but the family declined due to overall poor prognosis (Fig. 2). In a second patient who suffered from severe head injury with temporal-frontal contusions and subdural hematoma with shift, a decompressive hemicraniectomy was conducted. Intra-operatively severe brain herniation was encountered. An immediate CT obtained following hemicraniectomy disclosed a massive expansion of a contralateral epidural hematoma (initially small and limited to the supratentorial compartment), which post-craniectomy had also expanded to the posterior fossa causing massive brain stem compression. Patient rapidly succumbed to this hemorrhage and died prior to any further interventions being possible (Fig. 3).

The poor outcome of the remaining 3 patients who died or remained in coma was not caused by the EDHPF but rather was due to other associated traumatic intracranial findings.

6. Discussion

Prior to the advent of CT scanning, the mortality associated with EDHPF was high. With better imaging, this mortality was reduced significantly.[3] However, these lesions are still thought by many experts to require prompt surgical treatment by evacuation of the epidural hematoma located in the posterior fossa.[1,5,6,13–15] In larger modern case-series most of these lesions are typically operated upon.[2,4–7] This is generally due to the compact tight space of the posterior fossa harboring critical brain stem structurers. Amongst indications for surgery are thickness or volume of clot, progression of the size of the hematoma, crowding of the basal cisterns, and/or any mass effect on the 4th ventricle with or without associated hydrocephalus.[2,4–6,8,16,17]

However, there have been some efforts to apply a conservative approach towards these lesions.[17–20] A non-operative approach has mainly been used for smaller size hematomas, often measuring below 12 mm in thickness, or lesions that do not cause mass effect on the 4th ventricle or basal cisterns.[4,6,8,16,21] Furthermore, surgery is often applied if any progression of the EDHPF is noted.[2,6] Even in series more readily applying a conservative approach, a significant portion of patients typically undergo direct clot evacuation. The surgical portion often includes between 30 and 70 % of presented cases.[4,8,9,16,21–23]

Therefore, our cohort is unique because we treated the vast majority of our patients' EDHPF conservatively. We chose a conservative approach based on a number of criteria, such as GCS score, CT findings of clot thickness, mass effect, hematoma progression. Furthermore, in our series, we specifically focused up-front to optimize success for conservative therapy by aggressively minimizing any increase in intravenous sinus pressure. This was achieved by rigorous head elevation, as well as limiting any external obstruction around the neck (removing collars, avoiding constricting bands associated with the endotracheal tube). As these hematomas are largely due to a venous source of hemorrhage,[1,2,5,9–12] our goal was to minimize venous bleeding conservatively.

Applying this strategy, we could avoid surgery to evacuate EDHPF in most patients (90 %). Our mortality (13.3 %) was within the range of overall mortalities (0–17.9 %) associated with the management of these

lesions reported in major series in the literature.[1–9,21–23]

In the two instances where mortality was associated with the EDHPF (Figs. 2 and 3), an immediate surgical intervention was not believed to avert death.

7. Conclusion

The majority of EDHPF can be treated safely with conservative management. Measures to avoid an increase in intracranial venous pressures are key to success in conservative therapy. Indications for surgical evacuation should be considered on an individual basis.

CRediT authorship contribution statement

Michael A. Foster: Writing - review & editing. **Michael R. Collins:** Methodology, Investigation. **Thomas M. Wertin:** Methodology, Investigation. **Adrienne R. Azurdia:** Methodology, Investigation, Conceptualization. **Salvatore C. Lettieri:** Methodology, Investigation. **Iman Feiz-Erfan:** Writing - original draft, Investigation.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

1. Lui TN, Lee ST, Chang CN, Cheng WC. Epidural hematomas in the posterior cranial fossa. *J Trauma*. 1993;34(2):211–215. https://doi.org/10.1097/00005373-199302000-00005.
2. Bozbuğa M, Izgi N, Polat G, Gürel I. Posterior fossa epidural hematomas: observations on a series of 73 cases. *Neurosurg Rev*. 1999;22(1):34–40. https://doi.org/10.1007/s101430050006.
3. Pozzati E, Tognetti F, Cavallo M, Acciarri N. Extradural hematomas of the posterior cranial fossa. Observations on a series of 32 consecutive cases treated after the introduction of computed tomography scanning. *Surg Neurol*. 1989;32(4):300–303. https://doi.org/10.1016/0090-3019(89)90234-6.
4. Sencer A, Aras Y, Akcakaya MO, Goker B, Kiris T, Canbolat AT. Posterior fossa epidural hematomas in children: clinical experience with 40 cases. *J Neurosurg Pediatr*. 2012;9(2):139–143. https://doi.org/10.3171/2011.11.PEDS11177.
5. Bor-Seng-Shu E, Aguiar PH, de Almeida Leme RJ, Mandel M, Andrade AF, Marino Jr R. Epidural hematomas of the posterior cranial fossa. *Neurosurg Focus*. 2004;16(2):ECP1. https://doi.org/10.3171/foc.2004.16.2.10. Published 2004 Feb 15.
6. Karasu A, Sabanci PA, Izgi N, et al. Traumatic epidural hematomas of the posterior cranial fossa. *Surg Neurol*. 2008;69(3):247–252. https://doi.org/10.1016/j.surneu.2007.02.024.
7. Balik V, Lehto H, Hoza D, Sulla I, Hernesniemi J. Posterior fossa extradural haematomas. *Cent Eur Neurosurg*. 2010;71(4):167–172. https://doi.org/10.1055/s-0030-1249046.
8. Prasad GL, Gupta DK, Sharma BS, Mahapatra AK. Traumatic pediatric posterior fossa extradural hematomas: a tertiary-care trauma center experience from India. *Pediatr Neurosurg*. 2015;50(5):250–256. https://doi.org/10.1159/000438488.
9. Su TM, Lee TH, Lee TC, Cheng CH, Lu CH. Acute clinical deterioration of posterior fossa epidural hematoma: clinical features, risk factors and outcome. *Chang Gung Med J*. 2012;35(3):271–280. https://doi.org/10.4103/2319-4170.106144.
10. Yilmazlar S, Kocaeli H, Dogan S, et al. Traumatic epidural haematomas of nonarterial origin: analysis of 30 consecutive cases. *Acta Neurochir*. 2005;147(12):1241–1248. https://doi.org/10.1007/s00701-005-0623-2.
11. Charcos IB, Wong TW, Larsen BR, et al. Location of traumatic cranial epidural hematoma correlates with the source of hemorrhage: a 12-year surgical review. *World Neurosurg*. 2021;152:e138–e143. https://doi.org/10.1016/j.wneu.2021.05.052.
12. Holzschuh M, Schuknecht B. Traumatic epidural haematomas of the posterior fossa: 20 new cases and a review of the literature since 1961. *Br J Neurosurg*. 1989;3(2):171–180. https://doi.org/10.3109/02688698909002792.
13. Bullock R, Smith RM, van Dellen JR. Nonoperative management of extradural hematoma. *Neurosurgery*. 1985;16(5):602–606. https://doi.org/10.1227/00006123-198505000-00003.
14. Berker M, Cataltepe O, Ozcan OE. Traumatic epidural haematoma of the posterior fossa in childhood: 16 new cases and a review of the literature. *Br J Neurosurg*. 2003;17(3):226–229. https://doi.org/10.1080/0268869031000153071.
15. Mohanty A, Kolluri VR, Subbakrishna DK, Satish S, Mouli BA, Das BS. Prognosis of extradural haematomas in children. *Pediatr Neurosurg*. 1995;23(2):57–63. https://doi.org/10.1159/000120936.
16. Jamous MA, Samara QA, Jbarah OF, Ahmed YB. Management of traumatic posterior fossa epidural hematomas in pediatrics: our experience and review of the literature. *Childs Nerv Syst*. 2021;37(9):2839–2846. https://doi.org/10.1007/s00381-021-05248-y.
17. Otsuka S, Nakatsu S, Matsumoto S, et al. Study on cases with posterior fossa epidural hematoma–clinical features and indications for operation. *Neurol Med -Chir*. 1990;30(1):24–28. https://doi.org/10.2176/nmc.30.24.
18. Kawakami Y, Tamiya T, Tanimoto T, et al. Nonsurgical treatment of posterior fossa epidural hematoma. *Pediatr Neurol*. 1990;6(2):112–118. https://doi.org/10.1016/0887-8994(90)90044-2.
19. Pang D, Horton JA, Herron JM, Wilberger Jr JE, Vries JK. Nonsurgical management of extradural hematomas in children. *J Neurosurg*. 1983;59(6):958–971. https://doi.org/10.3171/jns.1983.59.6.0958.
20. Tuncer R, Kazan S, Uçar T, Açikbas C, Saveren M. Conservative management of epidural haematomas. Prospective study of 15 cases. *Acta Neurochir*. 1993;121(1–2):48–52. https://doi.org/10.1007/BF01405182.
21. Jang JW, Lee JK, Seo BR, Kim SH. Traumatic epidural haematoma of the posterior cranial fossa. *Br J Neurosurg*. 2011;25(1):55–61. https://doi.org/10.3109/02688697.2010.520759.
22. Hayashi T, Kameyama M, Imaizumi S, Kamii H, Onuma T. Acute epidural hematoma of the posterior fossa–cases of acute clinical deterioration. *Am J Emerg Med*. 2007;25(9):989–995. https://doi.org/10.1016/j.ajem.2007.02.041.
23. Chaoguo Y, Xiu L, Liuxun H, Hansong S, Nu Z. Traumatic posterior fossa epidural hematomas in children : experience with 48 cases and a review of the literature. *J Korean Neurosurg Soc*. 2019;62(2):225–231. https://doi.org/10.3340/jkns.2016.0506.007.

Abbreviations List

EDHPF: epidural hematoma of the posterior fossa
CT: computed tomography
GCS: Glasgow Coma Scale
SDH: subdural hematoma
EDH: epidural hematoma
EVD: external ventricular drain