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Case report

The uncommon delayed neurological deficit in posterior fossa chronic epidural hematoma: A case report

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ARTICLE INFO	A B S T R A C T
<i>Keywords:</i> Chronic epidural hematoma Posterior fossa epidural hematoma Delayed neurological deficit Surgical	Background: Chronic epidural hematoma (CEDH) is uncommon and therefore, less well characterized. The incidence of CEDH ranges from 3.9 % to 30 % of all epidural hematomas. Posterior fossa epidural hematomas represent a rare clinical entity. It has been reported in only 4–7 % of all extradural hematomas. This rare condition may present with rapid clinical deterioration by quick increase in size that may cause brain stem compression. This study aims to provide a case of chronic epidural hematoma with uncommon sign of delayed neurological deficits, specifically in the posterior fossa region.
	<i>Case presentation:</i> We report a case of a 34-years-old male with left upper and lower extremities weakness for 3 days before admission. The patient had a history of falling from a height of approximately 3 m about 3 weeks ago. Craniotomy epidural hematoma evacuation was performed on the patient. <i>Conclusion:</i> Chronic epidural hematoma is uncommon and therefore, less well characterized. The results of surgical care of symptomatic chronic posterior fossa EDH are often excellent. Early diagnosis and emergent

evacuation provide better outcome.

1. Introduction

An epidural hematoma (EDH) is an extra-axial collection of blood within the potential space between the outer layer of the dura mater and the inner table of the skull. An epidural hematoma occurs in 2 % of all head injuries and up to 15 % of all fatal head traumas [1,2]. Males are more often affected than females. Furthermore, the incidence is higher among adolescents and young adults. Acute epidural hematoma was first reported by Jacobson in 1886 and is a well-known clinical entity [3].

Epidural hematoma diagnosed more than 14 days after head injury is classified as a chronic epidural hematoma (CEDH). Chronic epidural hematoma (CEDH) is uncommon and therefore, less well characterized. The incidence of CEDH, which may range from 3.9 % to 30 % of all epidural hematomas, is also unknown from the literature. In the post-computed tomography (CT) era it is often considered that CEDH is a rare entity [4-6].

Posterior fossa hematoma has been reported in 3 % of all operated extradural hematoma and 0.3 % of all intracranial hematomas. A history

of occipital trauma was present in nearly all cases. Interestingly, most cases of epidural hematoma were not related to motor vehicle accidents [7,8]. Occipital bone fractures were found in nearly 80 % of cases. In more than 50 % of EDH, no source of bleeding was found. Diffuse venous oozing from the edges of the fracture line or the surface of the stripped dura, torn dural branches of the vertebral artery, and laceration of a dural sinus and emissary veins have all been described as sources of bleeding in EDH of the posterior fossa [9–11].

We report a rare case of posterior fossa chronic epidural hematoma with delayed neurological deficit after trauma. This case may provide further information and examples of chronic epidural hematoma with uncommon signs of delayed neurological deficit, specifically in the posterior fossa region.

2. Case presentation

A 34-years-old man came to the emergency department with left upper and lower extremities weakness in the last 3 days before admission. He had complaints of headache and dysarthria. The patient had a

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history of falling from a height of approximately 3 m about 3 weeks ago and loss of consciousness approximately 2 h after incident. After the accident, the patient was brought to the nearby hospital and was reported to regain his consciousness with good orientation. Patient had a history of vomiting at the hospital and was hospitalized for 5 days. During hospitalization, the patient was fully conscious with no neurological deficit and there was no head CT Scan examination. The patient was discharged and after 3 weeks, he presented himself to the emergency department with left upper and lower extremities weakness.

In the emergency department, the patient was fully conscious with full Glasgow Coma Scale (GCS) E4V5M6. Neurological examination was found with left hemiparesis and motoric score of 4. There was left hypoglossal cranial nerve paresis. Head CT Scan was performed on the patient. A hyperdense biconvex inhomogeneous lesion in the right side and the volume of hematoma 22 ml with the thickness 2.2 cm were reported. There was also compression of the right quadrigeminal cistern that caused non communicating hydrocephalus (Fig. 1).

Craniotomy epidural hematoma evacuation was performed on the patient. The aim of this surgery was to release the compression of the cerebellum and the brainstem resulted by the hematoma. During surgery, the hematoma was found to be capsulated with fluid and clot components (Fig. 2). There was no bone fracture above the lesion. The source of bleeding whether from bone fracture or sinus laceration was unidentifiable. After surgery, the patient was fully conscious with no neurological deficit. Hemiparesis and hypoglossal cranial nerve paresis were recovered. The patient later was able to carry on normal activity and go to work, without special care needed. This case report is presented based on the Surgical Case Report (SCARE) Guidelines [12].

3. Discussion

Traumatic posterior fossa epidural hematomas represent a rare clinical entity. Skull fractures are involved in the majority of the cases. Even without any fracture, epidural hematoma could still be formed. Following trauma, an epidural hematoma often forms when the periosteal duramater gets separated from the calvarium and the intervening veins rupture [13,14]. The size of hematoma may quickly grow as a result of the vascular rupture. The development of late and chronic clinical pictures is possible, nonetheless, if the venous structures are affected. Posterior fossa EDH has a venous origin in 85 % of cases and is caused by damage to the transverse or sigmoid sinuses secondary to occipital bone fracture [8,15,16]. There is no agreement on the specific time-based definition of CEDH, unlike subdural hematoma, which is referred as chronic subdural hematoma if discovered after more than 21 days following injury. In the literatures, chronic EDH is defined in a variety of ways. Sparacio et al. [4], used the term chronic EDH for epidural hematomas that are operated more than 48 h after the first injury and Clavel et al. [4], classified CEDH as EDH that are discovered more than 72 h after the initial injury. Bradley [4] recently defined chronic EDH as epidural hematomas recognised more than 14 days after a head injury based on hemoglobin breakdown products on magnetic resonance imaging [4,6,7]. This definition appears to be more recent, evidence-based, and scientific. In our case, the patient came after 21 days from initial accident. Based on the literatures and clinical timeline,



Fig. 1. Head CT scan examination (A) axial section and (B) sagittal section demonstrated inhomogeneous hyperdense lesion with hypodense rim in the right posterior fossa.



Fig. 2. During surgery there was capsulated encircled the hematoma, before the evacuation hematoma (A) and after the evacuation hematoma (B).

it is categorised as a chronic EDH.

The possible pathogenetic mechanisms that may explain chronicity in extra-axial hematomas include the existence of associated skull fractures, hematomas that are frontally located, age-related diffuse cerebral atrophy, venous sources of bleeding, and traumatic arteriovenous fistulae of meningeal vessels [1,2,5]. The CT scan for CEDH frequently reveals a low-density center encircled by a high-density border. In addition to the natural enhancement of the displaced dura itself, the granulation tissue producing a fibrovascular neo membrane on the exterior of dura mater is thought to be the mechanism of this rim enhancement. The misplaced dura mater may also become calcified [4–6]. Our CT scan finding demonstrated inhomogeneous hyperdense and hypodense lesion in the posterior fossa. There was hypodense lesion surrounded by hyperdense lesion in the CT scan.

Some CEDHs are discovered by chance, while others are found when persistent and/or progressive symptoms such as headache, dizziness, nausea, vomiting, memory loss, limb weakness, and alteration of consciousness are investigated [5,8,15]. The earlier surgical evacuation of symptomatic CEDH is performed, the better the results. However, spontaneous remission may be anticipated in patients with few or no symptoms, normal neurological function, and a modest CEDH without any mass effect. In these situations, a careful waiting may be necessary, but this entails expensive serial scans and protracted hospital stays. Even if the condition of the patient is satisfactory, surgical evacuation should be taken into consideration if CEDH is shown to not be naturally absorbed on serial scans due to the possibility of calcification [3-5,8,17]. Our case came with delayed neurological deficit after 21 days from accident. We performed surgical evacuation of hematoma based on the clinical condition and CT scan examination. Early after surgery, the neurological symptoms in the patient were improved. The patient was able to perform daily activity without any difficulties.

4. Conclusion

The results of surgical care of symptomatic chronic posterior fossa EDH are often excellent, while lesser ones can be treated conservatively. A watchful waiting may be warranted in minor chronic posterior fossa EDH patients, but it requires expensive serial scans and a protracted hospital stay. To rule out chronic posterior fossa EDH, a CT scan should be performed on any patient who has had a head injury and is alert and exhibiting mild, persistent symptoms and/or signs. The chronic posterior fossa EDH is a rare entity in the current period of comprehensive neuroimaging, it is often mentioned.

Submission statement

This manuscript is original and has not been submitted elsewhere in part or in whole.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Provenance and peer review

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Ethical approval

All ethical principles were considered in conducting this case report. All patient information kept confidential.

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CRediT authorship contribution statement

Galih Indra Permana - study concept, patient contribution, revising article, reviewed final version of article, study oversight, creation of figures.

Rizki Meizikri - study concept, patient contribution, revising article, reviewed final version of article, study oversight.

Tedy Apriawan - data collection, manuscript writing, critically revising article, reviewed final version of article.

Nur Setiawan Suroto - data collection, critically revising article, reviewed final version of article.

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Abdul Hafid Bajamal - study concept, patient contribution, revising article, reviewed final version of article.

Declaration of competing interest

The authors declare that they have no conflict of interest.

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