

Traumatic Supra- and Infra-tentorial Extradural Hematoma: Case Series and Literature Review

Abstract

Traumatic supra- and infra-tentorial extradural hematoma (TSIEDH) is a rare lesion constituting <2% of all extradural hematomas. There are only a few published articles about TSIEDH. This study included three patients with TSIEDH who were treated and operated at Dr. Soetomo General Hospital, Surabaya, Indonesia, from August 2015 to July 2016. Two patients sustained injuries in traffic accidents and one patient was injured by fall. The male to female ratio was 1:2. Glasgow Coma Scale (GCS) score ≤ 8 was present in one and GCS score of 9–12 was present in two patients. The brain computed tomography scan verified linear fracture of occipital bone in one and linear fracture of occipital bone with lambdoid suture separation in two patients. Early diagnosis and early surgical intervention of TSIEDH are imperative because the deterioration of TSIEDH is sudden and quick. We presented our experience in treating patients with TSIEDH in Dr. Soetomo General Hospital, Surabaya, Indonesia.

Keywords: Extradural hematoma, infratentorial, posterior fossa, supratentorial, traumatic

Introduction

Extradural hematoma (EDH) is an uncommon but serious complication of brain injury. It is found in 1%–4% of traumatic brain injury cases.^[1-3] EDH is defined as lesions that typically develop immediately after trauma and expand in volume in minutes. Following the alleviation of the tamponade effect on intracranial volume, EDH constitutes a threat to life.^[4] Based on data from Bogdan Asanin, EDH of the posterior cranial fossa accounted for 0.11% of craniocerebral injuries or 7.9% of EDH recorded.^[3] EDH comprises the most frequent traumatic space-occupying lesion of the posterior fossa.^[5] Hooper classified traumatic posterior fossa EDH as acute, subacute, and chronic, with the beginning of symptoms within the first 21st h of trauma, between 2nd and 7th days after trauma, and later, respectively.^[3,6,7] According to the literature, there has been a certain decrease in mortality in the acute and subacute course patients.^[5]

EDH is classically arisen from a blow to the thin temporal bones of the cranium. Trauma to underlying middle meningeal artery leads to EDH.^[8] Traumatic EDH in posterior fossa is infrequent. EDH on both sides of tentorium at the same time is much

more infrequent. In our experience, traffic accidents and falls are the major causes of EDH. Relying on clinical symptoms only to identify the site of hematoma on traumatic supra- and infra-tentorial EDH (TSIEDH) is difficult since there may be no symptom initially. The diagnosis after the onset of medullary complications usually is too late for surgical treatment and the outcome usually is death. Computed tomography (CT) scan is a quick tool to detect the lesion in traumatic cases and improving the prognosis of patients.^[9] In the literature, there are only a few articles published related on TSIEDH. The purpose of this article is to present our experience in treating patients with TSIEDH in Dr. Soetomo General Hospital, Surabaya, Indonesia. Correct diagnosis and early surgical intervention will give the patient with TSIEDH a good chance to recover.^[6]

Case Report

The study included patients with TSIEDH who were treated and operated on at Dr. Soetomo General Hospital, Surabaya, Indonesia, from August 2015 to July 2016. Consent of the patients and institutional approval from medical research unit Dr. Soetomo General Hospital have been achieved. There were three patients with TSIEDH [Table 1].

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Two patients sustained injuries in traffic accidents and one patient was injured by fall. The male to female ratio was 1:2. Glasgow Coma Scale (GCS) score ≤ 8 was present in one and GCS score of 9–12 was present in two patients. The brain CT scan verified linear fracture of occipital bone in one and linear fracture of occipital bone with lambdoid suture separation in two patients. All patients were operated on by standard approach, one of them with lethal outcome.

Case 1

A 50-year-old woman referred to our hospital with decrease of consciousness after traffic accident (pedestrian hit by motorbike) 17 h before admission. Her initial GCS score was E3V2M5 (10/15), pupils bilaterally were in normal size, and there was no hemiparesis. The patient had left occipital swelling. The brain CT scan examination showed the presence of infra- and supra-tentorial EDH [Figure 1],

Table 1: Cases of traumatic supra- and infra-tentorial extradural hematoma in Dr. Soetomo General Hospital from August 2015 to July 2016

Parameters	Total
Number of cases	3
Sex	
Male	1
Female	2
Mechanism of injury	
Traffic accident	2
Fall	1
Skull fractures	
Linear	1
Diastasis	-
Linear + diastasis	2
GCS	
13-15	-
9-12	2
≤ 8	1
Recovery	
Good	2
Death	1

GCS – Glasgow Coma Scale

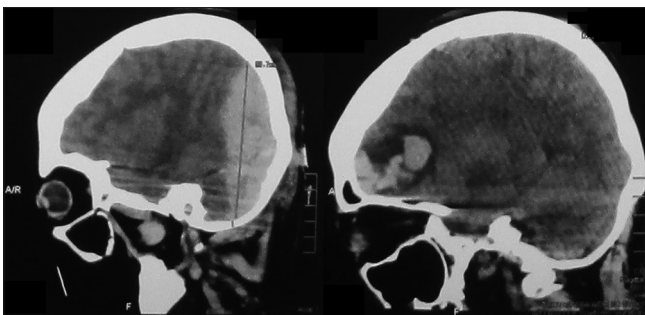


Figure 1: Preoperative and postoperative brain computed tomography scan of a 50-year-old woman with traumatic supra- and infratentorial extradural hematoma

linear fracture on left temporal bone, diastasis fracture on left lambdoidal suture, intracerebral hematoma (ICH) burst lobe on frontal and temporal base regions, and brain edema. The EDH was evacuated immediately. After surgery, the patient showed an increasing recovery without any neurological deficit in the postoperative period. On postoperative CT, there was no residual supra- or infra-tentorial hematoma [Figure 1]. She was discharged on the 17th postoperative day.

Case 2

A 38-year-old woman referred to emergency room with a history of fallen down 4 days before the admission from a tree of three meters height. On examination, her GCS score was E1V1M4 (6/15), pupils bilaterally were in normal size, and sluggishly reacted with no hemiparesis. The patient had left occipital swelling. The brain CT scan of brain was done and revealed infra- and supra-tentorial EDH [Figure 2], linear fracture on left temporal bone, diastasis fracture on left lambdoidal suture, and brain edema. In view of poor GCS score and CT scan finding, the patient was taken up for surgery for evacuation of EDH. After surgery, she was consulted to head-and-neck surgeon for tracheostomy. Her postoperative GCS was E2VtM4. Unfortunately, the patient died on the 11th postoperative day.

Case 3

A 31-year-old man was transferred from local hospital to emergency room in our hospital for further assessment after traffic accident in unconscious condition 20 h before admission. On initial assessment, his GCS score was E3V4M5 (12/15), pupils bilaterally were in normal size, and there was no hemiparesis. He had left occipital swelling and was very agitated. The brain CT scan was done and showed infra- and supra-tentorial EDH [Figure 3] and linear fracture on left parietal and occipital bone. The patient then was transferred urgently to operating theater and underwent evacuation of the hematoma. Postoperatively, the patient made a good recovery. He was discharged on the 5th postoperative day.

Surgical technique

The patients were intubated under general anesthesia during surgery. The position was prone with the head secured. Anatomic landmark (midline and transverse sinus) and the site of craniotomy were marked with a skin marker. A golf stick or linear skin incision was made. Multiple burr holes were drilled with a high-speed bone drill in supra- and infra-tentorial area. The bone flap was cut off using a gigli saw. A rongeur was used to open the bone to avoid sinus injury. The bone flap was then removed and parts of hematoma were explored. The supra- and infra-tentorial hematoma was cleaned and the dura was slinged, except toward the transverse sinus. In sinus region, the hematoma was cleaned near the bone first, preventing dissection of the hematoma by

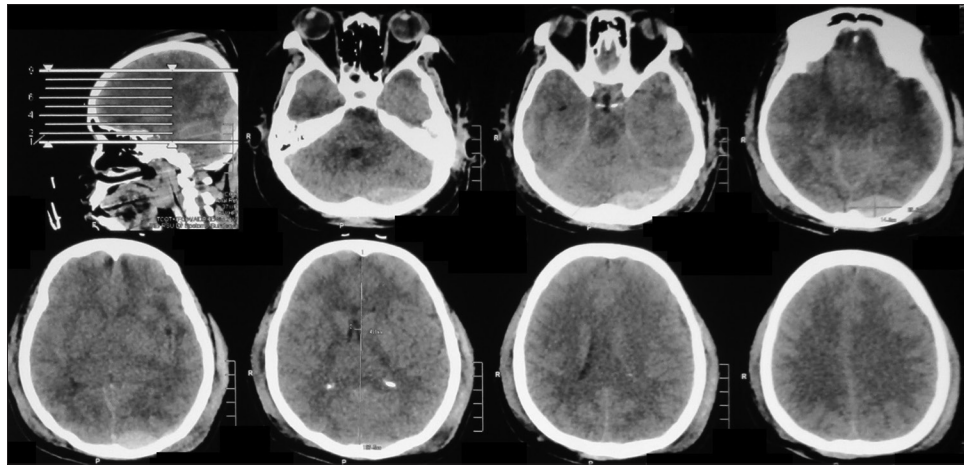


Figure 2: Left traumatic supra- and infratentorial extradural hematoma of a 38-year-old woman on brain computed tomography scan (Glasgow Coma Scale score E1V1M4)

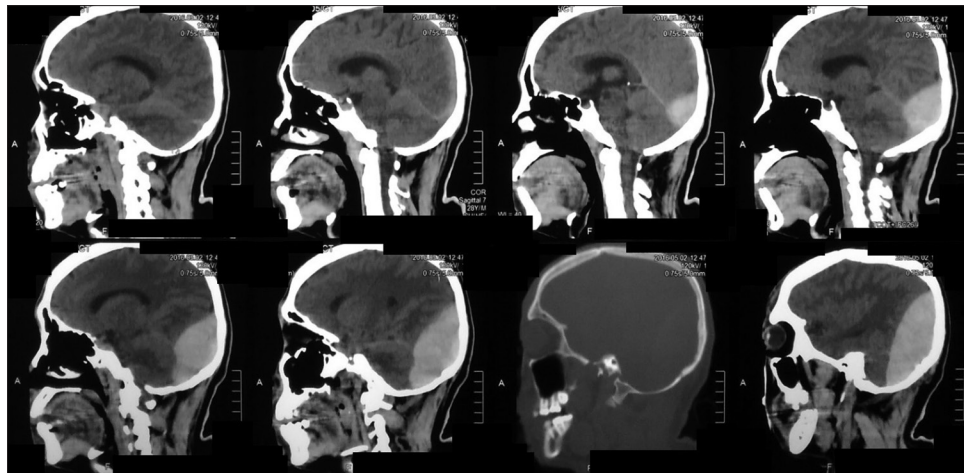


Figure 3: Brain computed tomography scan of a 31-year-old man showed traumatic supra- and infratentorial extradural hematoma (Glasgow Coma Scale score E3V4M5)

force. A bipolar was used to stop meningeal hemorrhage (if any). Compression with a gelfoam and surgicel was applied if sinus bleeding was encountered and the dura was secured with tack-up stitches. Finally, the bone was reset and the scalp was sutured.

Discussion

Even though the number of traumatic posterior fossa hematomas has been increasing with the invention of CT scan,^[10] the incidence of TSIEDH is still very rare constituting <2% of all EDH. In our hospital, from August 2015 to July 2016, there were only three cases of TSIEDH. According to Izumihara *et al.*, the reason may be the rarity of injury to the vein of Galen or the sinus and anatomical sparsity of bridging vein in the posterior fossa. TSIEDH appears both in front of lateral sinus and behind it, and in general, such cases are seen if lateral sinus injury is observed. One of the most significant criteria is the presence of fracture line that crosses the lateral sinus.^[9] TSIEDH mainly occurs due to occipital deceleration injury,

most often associated with occipital bone fracture or lambdoid suture separation.^[3,9,11] Venous sinus detachment, meningeal blood vessels leakage, and a fracture line across the transverse sinus all lead to TSIEDH.^[11] In our series, two cases had lambdoid suture separation and one case had occipital bone fracture.

Some authors suggested CT scan examination should be conducted in all traumatic injury patients with occipital soft tissue swelling and occipital bone fracture even if no clinical symptoms are present.^[5,9] Aggressive use of CT scan in the first 24 h (acute phase) and continual monitoring in patients with an occipital fracture and in patients with concomitant occipital trauma after the evacuation of hematomas is the logical way to reduce the mortality.^[5] The rate of occipital bone fracture or diastasis fracture of lambdoid suture ranges from 40% to 86%.^[3] In our series, all cases were present with linear fracture of occipital bone or diastasis fracture of lambdoid fracture. On CT scan, EDH appeared as a hyperdense biconvex lense, extracerebral lesion, sometimes with mixed densities

on account of clot reduction or rebleeding.^[5] CT scan also revealed mass effect caused by the hematoma with partial or total obliteration of the perimesencephalic cistern and compression of the fourth brain ventricle.^[3]

Unlike EDH in the anterior and middle cranial fossa, EDH in the posterior fossa usually without any characteristic of clinical symptoms.^[3] Is *et al.* noted that clinical manifestations of supratentorial EDH were headache, nausea, vomiting, memory loss, hemiparesis, and unconsciousness; meanwhile, neck pain, cranial nerve palsy, and cerebellar dysfunction can be seen in infratentorial EDH.^[6] In our series, the chief complaint was the decrease of consciousness and other clinical findings were headache, nausea, and vomiting. Consciousness disturbance appeared was depended on the size and speed of hematoma formation and on additional intracranial injuries, such as brain contusion, ICHs of the contrecoup type in frontal and temporal regions, subarachnoid hemorrhage, subdural hygroma, subdural hematoma, and others.^[3] Although TSIEDH has associated typical characteristics, it might be difficult to diagnose as infratentorial acute EDH is clinically silent and has nonspecific symptoms.^[11] Infratentorial hematoma can lead to sudden death and quick deterioration while supratentorial hematoma can mask the features of infratentorial hematoma.^[9] An external injury to the occipital region, consisting of bruising, laceration, or cephalohematoma, can be the key finding in a head trauma that becomes complicated with a TSIEDH without specific neurological features.^[5] Some cases may occur without an associated fracture, but fracture is nearly always present and may even be depressed. Thus, TSIEDH should be strongly suspected in the presence of fracture line at the lateral or sigmoid sinus.

Special attention should be given to this type of hematoma since it can often be rapidly deteriorating, causing a sharp rise in intracranial pressure that leads to foramen magnum herniation, causing compression of the transverse sinus and brain stem, and subsequently death of the patient, a talk-and-die syndrome.^[3] The bleeding usually arises from the venous sinuses (transverse sinus, torcula herophili, mastoid emissary vein), the posterior branches of the middle meningeal arteries, or the diploe and the small dural vessel.^[5] It is noteworthy that when the space-occupying hematoma starts to grow, the rupture of a bridging vein may become an additional source of bleeding.^[5] Diploic bleeding may be responsible for some of the hematomas whose source could not be discovered.^[5] Injury of arterial blood vessel leads to fast filling of the hematoma with worse prognosis, while injury of the venous blood vessel with gradual bleeding below the fracture leads to slower formation of the hematoma with good prognosis after surgery.^[3] The source of bleeding in most cases was venous (transverse sinus).^[5] In our series, one case has source of bleeding from sinus transverse, one case from

site of fracture and mastoid emissary vein, and one case with unknown source of bleeding.

Diagnosis and cure of EDH of the posterior cranial fossa were possible following the first case of successful surgery, reported by Coleman and Thompson in 1941.^[4] Surgery remains the gold standard for the treatment of TSIEDH. This may be in the form of suboccipital craniectomy or craniotomy depending on the size of the hematoma.^[12] In our series, one case was done with suboccipital craniectomy and two cases underwent craniotomy.

Early diagnosis and early surgical intervention of TSIEDH are imperative. The reason why early surgery should be performed in patient with TSIEDH is to eliminate the hematoma and lift the compression to the transverse sinus.^[11] If the transverse sinus is compressed, causing venous return disorder, the symptoms of intracranial hypertension can be significant and can rapidly form tonsillar herniation, which is difficult to resolve. In general, overall mortality of TSIEDH is 17%, the operative mortality rate is 14%, and the morbidity rate is 6%.^[9] Morbidity and mortality has been shown to be affected by age with better prognosis in patients who are under 10 years of age, while old patients are unlikely to manifest signs or symptoms of increased intracranial pressure due to cerebral atrophy.^[9] Other prognostic factors are clinical course, state of consciousness, and time of surgical intervention. Koç *et al.* stated that the most important prognostic factors of mortality in TSIEDH were late diagnosis and late treatment.^[5] Coexisting intracranial lesions had no influence on mortality.^[5] According to Malik *et al.*, the factors predicting outcomes are as follows:^[7]

- Good prognostic factors: Good GCS score, children, midline shift <5 mm, clot thickness <15 mm, and clot volume <10 ml
- Bad prognostic factors: Sudden-onset, associated intracranial injuries, acute hydrocephalus (effaced fourth ventricle and posterior cranial fossa cisterns).

In our series, there was one case with lethal outcome.^[11] Poor initial GCS, old age, and delayed time to surgical intervention were factors of bad prognosis for the patient. In relation to the associated intracranial lesions coexisting with TSIEDH, we found ICH in one case and brain edema in two cases.

Conclusion

TSIEDH is a rare lesion constituting <2% of all EDHs. Clinical progress is silent and slow, but the deterioration is sudden and quick. It can be fatal if not promptly treated. Due to an early diagnosis is mandatory for good recovery, CT scan monitoring for all patients with occipital trauma should be carried out in the acute phase. Early diagnosis and timely surgical intervention could reduce the mortality and morbidity in TSIEDH.

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Conflicts of interest

There are no conflicts of interest.

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