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Case Report / Приказ случаја

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A combination of acute and delayed contralateral epidural hematoma

Комбинација акутног и одложеног контралатералног епидуралног хематома

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SUMMARY

Introduction An acute bilateral extradural hematoma is an uncommon presentation of traumatic head injury; however, it leads to higher mortality rate than acute unilateral hematoma. A delayed epidural hematoma (DEDH) is a hematoma that is not present on initial computed tomography (CT) scan but is found on subsequent CT. While reviewing the literature, we could not find recently published papers considering supratentorial DEDH after primary operated contralateral EDH.

Case report A comatose 14-year-old male, with Glasgow Coma Scale score 4 and the right midriatic pupil on the side of the blunt trauma to the head, was admitted to ICU after he had survived a traffic accident. Initial brain CT scan showed an acute temporo-parietal epidural hematoma on the right side of the cranium, with impressive midline shift and bilateral linear skull fracture. Surgery was performed and intracranial pressure (ICP) monitor was implanted, which showed the increased values of ICP. A control brain CT scan performed within 24 hours showed a new contralateral occipitoparietal epidural hematoma. Another operation was performed. Second, control CT brain scan showed favorable finding. The patient was transferred after 25 days to the rehabilitation center, with the disability rating score of 11 and after 3 months it was 1.

Conclusion A contralateral DEDH is a life-threatening neurosurgical emergency case which can occur during the first 24 hours after decompressive craniectomy. Control and ICP monitoring is recommended in these cases, especially after surgical decompression.

Keywords: brain trauma; delayed bleeding; extradural hematoma; intracranial pressure monitoring

САЖЕТАК

Увод Акутни билатерални епидурални хематоми (ЕДХ) нису чест налаз након повреде главе, али су узрок знатно веће смртности у поређењу са акутним унилатералним хематомом. Одложени епидурални хематом (ОЕДХ) је епидурално крварење које није присутно на иницијалној КТ, али је нађено на накнадној КТ. У литератури нисмо нашли рад са описом супратенторијалног ОЕДХ након операције контралатералног ЕДХ.

Приказ болесника После саобраћајне несреће примљен је 14 година стар дечак у коми (Глазгов кома скор 4) и проширеном зенивом на страни тупе трауме главе. Иницијална КТ мозга показала је темпоропаријетални ЕДХ на десној страни кранијума, са израженим померењем средњелинијских структура и билатералном линераном фрактуром лобање. Повређени је оперисан и започет мониторинг интракранијалног притиска (ИКП) који је био повишен. Контролна КТ мозга унутар првих 24 часа показала је нови контралатерални окципито-паријетални ЕДХ. Предузета је нова операција. Други, контролни налаз КТ мозга је био уредан. Након 25 дана болесник је преведен у Рехабилитациони центар, са степеном неспособности оцењеним са 11. После три месеца, болесник је био готово без последица повређивања.

Закључак Контралатерални ОЕДХ је стање непосредне животне угрожености, који се може појавити унутар првих 24 часа од краниотомије. Потребно је брижљиво праћење стања болесника, укључујући контролну КТ и праћење ИКП.

Кључне речи: траума мозга; одложено крварење; епидурални хематом; мерење интракранијалног притиска

INTRODUCTION

An epidural hematoma (EDH) occurs when blood accumulates between the skull and the dura mater due to severe cranial trauma. A typical location of these hematomas is the temporal one, as the most common cause of bleeding is a lesion of the middle meningeal artery. The pterion region, which overlies it, is relatively weak and prone to injury. Nevertheless, other locations of EDH are not so uncommon and are reported in 20–30% of cases [1]. It is usually found on the same side of the cranium that was impacted by the blow, but very rarely it can be due to a counter coup brain injury. Epidural blood clot, while expanding, strips the dura from tabula interna of the skull, forming the mass that causes brain shifting, and consequently compression on the cerebral blood vessels and cranial nerves. Usually, EDH grows within the first 4 to 6 hours, after which profound deterioration starts in the form of contralateral limb weakness, coma, ipsilateral pupil fixation and dilatation.

This condition is reported in 1-3% of head injuries [2], and 15-20% of EDHs are fatal [3], while in children population only 5% [4].

Intracranial EDH is considered to be the most serious complication of head injury, requiring immediate diagnosis and surgical intervention. It usually occurs in young adults, and it is rare before the age of 2 and after the age of 60. Intracranial epidural hematoma may be acute (58%), subacute (31%) or chronic (11%) [5]. Bilateral EDH accounts for 2-10% of all acute EDHs in adults, being exceedingly rare in children [6].

A traumatic delayed epidural hematoma (DEDH) can be defined as a hematoma that is insignificant or not present on the initial computerized tomography (CT) scan made after trauma; however, a subsequent CT scan shows sizeable epidural bleeding [7]. The reported incidence of DEDH varies from 5.6% to 13.3% [8, 9].

While reviewing the literature, we could not find recently published papers considering supratentorial DEDH after primary operated contralateral EDH, only some cases after the operation of contralateral subdural or intracerebral hematoma [10–12].

CASE REPORT

Herein we present a case of a 14-year-old boy who sustained severe injuries as a pedestrian in an accident. His polytrauma injuries included the left lung contusion, extensive parietal bilateral skull fracture, and typically located, large EDH, temporoparietal on the right side, detected on computer

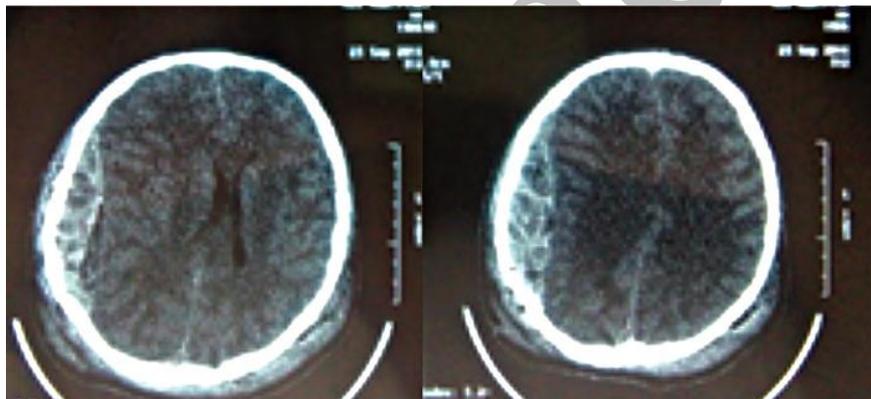


Figure 1. Initial epidural hematoma in right temporoparietal region.

tomography (CT) scan (Figure 1). On admission, his Glasgow Coma Scale score (GCS) was 4, the patient had the left-sided hemiplegia, and the right pupil was fixed and dilated.

Urgent craniotomy was performed as well as the evacuation and hemostasis of massive extradural hematoma which was combined with modest dura and brain laceration. Intracranial pressure (ICP) monitor was implanted in the subdural space, and it showed the values that indicated intracranial hypertension (ICHTN). The initial ICP was higher than 40 mmHg during the first postoperative hour and declined to around 24 (average ICP value was 24,3 mmHg) for the next 20 hours. The right pupil shrank to the normal size. During the first 12 hours, the patient was sedated, so that his state of consciousness was unavailable, except for the short period of time between two sedations. Due to ICHT and reduced possibilities of assessing GCS, a control CT brain scan was performed. A new, contralateral occipitoparietal DEDH was found, which was a bit smaller than the initial one (Figure 2). Another operation was performed,

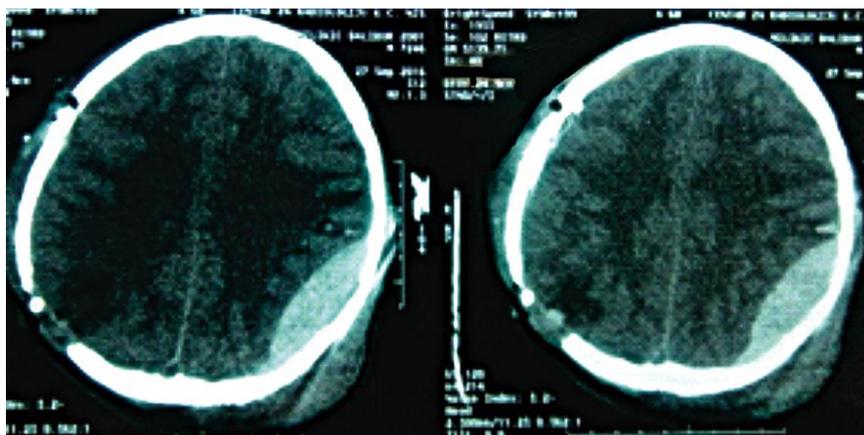


Figure 2. Contralateral delayed epidural hematoma in left occipitoparietal region – newly formed hematoma after first operation.

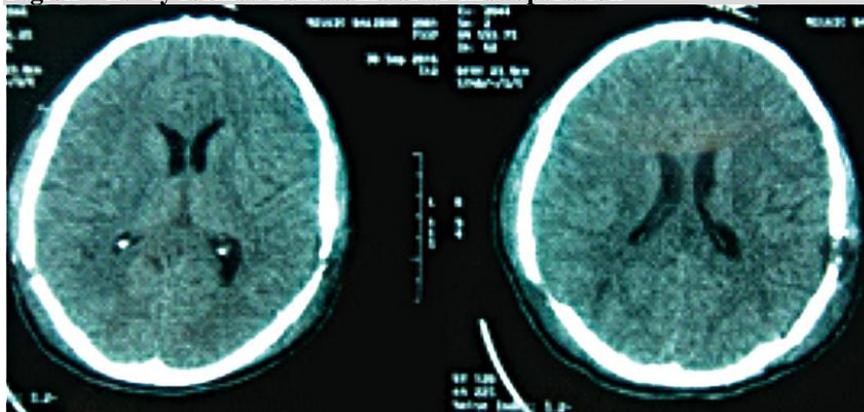


Figure 3. Control brain CT scan after the second operation – normal postoperative finding.

and under the contralateral fracture an epidural hematoma was evacuated. In the next three days, the values of ICP measured around 20 mmHg. The average daily ICP values were 19.4, 20.8 and 18.2, and in the last three days of ICP monitoring the values were around 15 mmHg. Meanwhile, blood samples routinely obtained in the emergency room were analyzed for coagulation parameters, including prothrombin time (PT), platelet counts (PLT), activated partial thromboplastin time

(APTT), fibrinogen level (Fbg), and the international normalized ratio (INR). All values were normal. One day after the second operation, the brain CT scan showed a favorable finding (Figure 3). Eye exam has been done at 7th postoperative day, but no signs of papiledema were found. A long-term recovery, tracheotomy and intensive physical rehabilitation followed the second operation. The patient was discharged from hospital after three weeks and transferred to the rehabilitation facility and had disability rating score (DRS) 11. Three months after the accident, his DRS was 1.

DISCUSSION

The question is whether a hematoma that is not present on initial CT scan but is found on subsequent CT represents an enlargement of an EDH from invisible to visible on high-quality CT or is a newly formed subacute extradural haemorrhage promoted by some risk factors.

The risk factors responsible for the delayed appearance of the EDH “tamponade” effect are usually increased endocranial pressure and post-traumatic arterial hypotension as well as coagulopathy in a limited number of cases [13, 14, 15]. Post-traumatic nasal and ear leakage of cerebrospinal fluid (CSF) may result in the occurrence of DEDH. Some of the risk factors are iatrogenic, including: operation of cerebellar expansive lesions, rapid intravenous infusion or transfusion after the development of hemodynamic shock in a traumatized patient, intraventricular

CSF drainage, overdrainage during ventriculoperitoneal shunting. A case of a DEDH in the spinal region, after facet joint infiltration in chronic back pain patient, has been also described [16].

Some authors suggest repeating a brain CT scan within the first 36 hours after sustaining an injury in the patient with a small, asymptomatic EDH, as mean time to EDH enlargement is about eight hours [17]. We performed the control CT brain scan within 24 hours as the neurological status of the patient remained unclear and potentially unfavorable. Nevertheless, ICP monitoring and continuous neurological monitoring could be crucial in early discovering of DEDH. Post-traumatic restlessness may lead to hyperventilation and blood pressure elevation with lowering of the ICP, both of which may provoke bleeding into EDH. However, in the series of Sakai et al. [18], all of the ICP monitored patients had either unchanged or increased ICP. Early diagnosis of DEDH can be facilitated by the liberal use of intracranial pressure monitoring as its sensitivity could be up to 80% [19, 20]. In the case we presented, after the first evacuation of the epidural clot, ICP dropped from 40 to 24 mmHg and that event could trigger the subsequent bleeding from a fractured skull contralateral. Still, the ICP values remained higher than normal (average ICP > 20 mmHg) until the time of the second evacuation of the epidural clot, although normalization of ICP values was to be expected. It turned out that DEDH maintained ICHTN. The ICP values were lower or equal to 20 mmHg for the next three days. Normalization of ICP occurred on the fourth postoperative day. Therefore, ICP monitoring can be of great help especially in a continuously sedated patient.

Eye examination did not show any signs of the papilloedema since it was performed a week after an accident. Normal results were to be expected according to the fundoscopic examination dynamic finding after severe brain trauma [21]. This dynamic is quite different from fundoscopic examination finding after severe spine injury in which papilledema occurs much later [22].

Enlargement of EDH in its acute phase is often explained by continuous hemorrhage and rehemorrhage from either the arterial or venous vessels. Some authors emphasize that venous hemorrhage does not generate enough pressure to strip dura mater from the bone or to overcome clot-induced tamponade effect [23]. A decrease in clot density combined with the formation of membranes with permeable sinusoids [24] usually after the fifth day after trauma could lead to new hemorrhage and further enlargement of hematoma. This slight increase in size between days 5 and 16 is manifested in 50% of cases, and some patients require emergency craniotomy when signs of herniation occur [24]. DEDH in our patient occurred much earlier, in the first 24 hours, probably due to extensive bilateral fracture.

Rarely reported in mild head traumas [25], DEDH is commonly associated with skull fractures [20]. DEDH development in the infratentorial compartment is tenfold higher in patients with fracture line in the posterior fossa than in patients with supratentorial fractures [26]. Rapid deterioration of injured patient, especially if DEDH is formed in the posterior fossa, could be lethal despite urgent CT brain scan and prompt operation [25]. This is explained by an irreversible damage to some of the vital centers located in the brain stem.

Atypical occipitoparietal location and modest volume of DEDH presented in this case report are relevant for the patient's prognosis, as it is unlikely to cause brain herniation or pressure on the brain stem. However, if it remains unrecognized timely, DEDH can be life-threatening.

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