ORIGINAL ARTICLE

Delayed Epidural hematoma, presentation and treatment outcome, the experience of King Abdullah Hospital – Bisha in 2018

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Received:
3rd January 2019

Accepted:
27th June 2019

Abstract:

Introduction: Delayed Extradural Hematoma (DEDH) is a rare condition comprises 9-10% of all Extradural Hematoma (EDH)s in several series, but it is associated with very high mortality and morbidity rates.

Material and Methods: This is a one-year descriptive study done in a single center (King Abdullah Hospital - Bisha) in the period from January to December 2018. Only those with no or a small extradural hematoma that increased in size in the follow up CT scan were included in the study. Initial, follow up and post-operative CT scans for the patients satisfying the inclusion criteria above were all collected.

Results: During the period from January to December 2018, we received 62 cases with post-traumatic EDH. Four cases (6.5%) were found to satisfy the inclusion and exclusion criteria of DEDH. Three of the cases were initially managed conservatively, but when hematoma size increased, immediate surgical evacuation was done. In the 4th patient the hematoma size was large from the beginning with an overlying depressed fracture. Post-operative the hematoma was recollected again but from a different source of bleeding source (the sagittal sinus) and the patient was re-operated again for hematoma evacuation.

Discussion: All mechanisms of injury can cause delayed extradural hematoma including iatrogenic causes from any post-operative cranial procedure. The indications for doing urgent follow up CT brain includes, increased headache, irritability, any deterioration in the level of consciousness, change in the pupil's reactivity to light and if there is new neurological deficit.

Conclusion: All patients with EDH should be initially admitted to the ICU for at least 24 hours and should get their follow up CT Brain done before shifting them to the ward. Up to now, there is no constant features suggesting the development of DEDH and further studies are needed to establish standard management guidelines for early detection of patients potential to develop DEDH.

Keywords: Delayed extradural hematoma, craniotomy, head injury, post-traumatic

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Introduction:

Delayed extra-dural hematoma (DEDH) can be defined as "an extradural hematoma (EDH) that is not present on the initial CT scan, but is found on subsequent CT."[1] Sometimes the EDH is present in the initial CT scan, but its size is small and insignificant. Therefore, a more precise definition would be "a hematoma that is insignificant or not present on the initial computerized tomography (CT) scan made after trauma but subsequent CT scan shows sizeable epidural bleeding."[2]

DEDH comprises 9-10% of all EDHs in several series. Although some authors think that there is no difference in the clinical presentation between those with EDH and those with DEDH, but there are some risk factors should be seriously considered to avoid any unnecessary delay in delivering the appropriate management.[3] The risk factors for developing DEDH include lowering of the intracranial pressure (ICP) in a

rapid way either through medications like mannitol[4] or surgically like in evacuating a contralateral hematoma which can release the tamponade effect and invite more bleeding and increase in hematoma size 4-8 specially when there is an associated skull fracture contra-lateral to the site of hematoma.[9,10] Rapid correction of shock (a concept known as hemodynamic surge may cause an increase in any hematoma in the body including EDH)[4] and coagulopathies were also reported as risk factors for developing DEDH. Although spinal DEDH is a rare entity, but still few cases were reported in the literature but in almost most of the cases, the spinal DEDH occurred post-operatively within the first 3-days[11] specially when performing an aggressive removal of a malignant vertebral tumor like Ewing's sarcoma for example.[12]

In the literature, DEDH can occur with mild, moderate or severe head injuries,[13] but it was mainly reported in those with mild head injury[2] and skull fracture was reported as a common feature in almost all DEDH patients.[4,6,9,10] The other reported common features of DEDH patients include; young age,[4,14-16] a mode of trauma of either a fall or a pedestrian involved in motor vehicle accident[4] besides EDH seen in an unusual sites (like in posterior fossa hematoma).[4,17,18] Post-operative posterior fossa craniectomy also may cause a DEDH mostly secondary to blood oozing from the transverse sinus and therefore a special caution and close post-operative monitoring should be attained for such patients.[19]

Material and Methods:
This is a one-year descriptive study done in a single center (King Abdullah Hospital - Bisha) in the period from January to December 2018. The cases operated outside King Abdullah Hospital-Bisha were excluded from the study. Those who had small extradural hematoma that did not increased in size and managed conservatively and those with a large extradural hematoma from the beginning were excluded from the study. Only those with no or a small extradural hematoma that increased in size in the follow up CT scan were included in the study. Initial, follow up and post-operative CT scans for the patients satisfying the inclusion criteria above were all collected.

Results:
During the period from January to December 2018, we received 62 cases with post-traumatic EDH, some of them were managed conservatively and some of them were surgically evacuated.

Of these 62 cases only 4 cases (6.5%) were found to satisfy the inclusion and exclusion criteria of DEDH. Three of the cases were initially managed conservatively, but when the patients clinically deteriorated and the hematoma size increased, immediate surgical evacuation was done. In the 4th patient the hematoma size was large from the beginning with an overlying depressed fracture. The hematoma was evacuated, the depressed bone was elevated, and the bleeding was found to be from the main stem of the middle meningeal artery. Post-operative the hematoma was recollected again but from a different source of bleeding source (the sagittal sinus) and the patient was re-operated again for hematoma evacuation.

All of the 4 cases had excellent post-operative outcome and they were all discharged home in a good condition.

The first case was 25-years old male involved in a motor vehicle accident "MVA" (car crash against the wall), had no history of loss of consciousness, convulsions, vomiting or bleeding per ear, nose or mouth. He came to the ED complaining of occipital headache and chest pain. The companion died in the scene. His past medical history (PMH) and drug history were not significant. On examination the patient was fully conscious, oriented, GCS 15 both pupils were equal and reactive to light, no neurological deficit. He had superficial scalp lacerations in the occiput and multiple chest bruises. He was hemodynamically stable, primary survey revealed right 3rd, 4th and 5th rib fractures with underlying lung contusion found in CT chest.

Figure-1:The initial CT Brain of case No.1. (a) shows a small rightretro cerebellar extradural hematoma. (b) shows no associated supratentorial hydrocephalus and a small right temporal hemorrhagic contusion. (c) 3D bone reconstruction showing no bone fracture overlying the hematoma

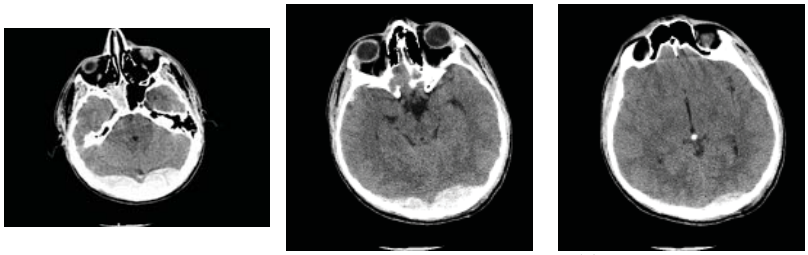

Figure-2:The follow up CT Brain done for case No.1 after 12 hours. (a) showed an enlargement of the right EDH and a newly seen left retro cerebellar acute EDH. (b) the CT cut just above the tentorial hiatus at the level of midbrain showing the supratentorial extension of the hematomas in both sides. (c)Higher cut at the level of the 3rd ventricle showing the final superior extent of the Lt acute EDH

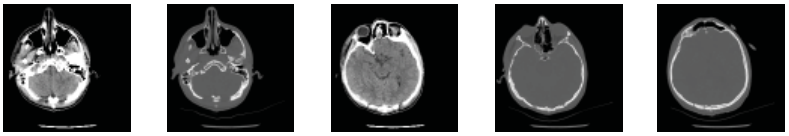

Figure-3: Follow up CT Brain done in the 1st post-operative day. (a) Both retro cerebellar hematomas were adequately evacuated and in the bone window you can see the size of craniectomies done and the bone ridge left in between. (b) Both occipital hematomas were also completely evacuated and in bone window the 2 small craniotomies were made few millimeters away from the midline

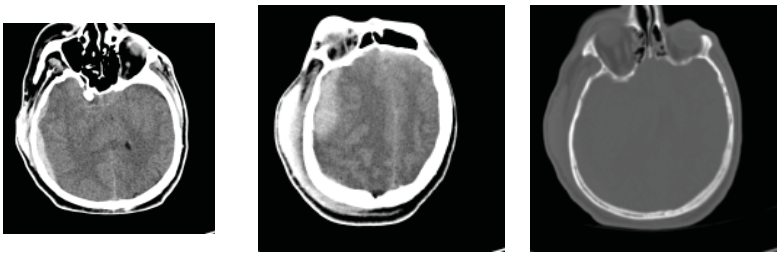

Figure-4: CT Brain done upon the patient arrival to RR. (a) 2 small right temporal and occipital small acute EDHs. (b) another right frontal small acute EDH. (c) temporal bone linear fracture that caused the hematoma

CT Brain showed a small right retro-cerebellar acute EDH with no associated hydrocephalus or an overlying bone fracture. (Fig.1) The patient was admitted under close observation in the ICU. 12-hours later the patient became agitated with severe headache. On examination the patient was still conscious and oriented. Pupils were still reactive to light. Follow-up CT Brain showed an increase in the hematoma size with another EDH in the opposite side, both hematomas were reaching to the supra-tentorial compartment (Fig.2). The patient was immediately shifted to the OR, 2 small occipital craniotomies for the occipital hematomas and for the cerebellar hematomas were done, the bone ridge overlying the posterior part of the superior sagittal sinus, the transverse sinuses and the confluence of the sinuses was left in place. Post-operatively the headache was completely resolved, the patient was fully conscious, oriented with no neurological deficit. Follow up CT Brain was obtained in the first post-operative day and it showed completely evacuated occipital and posterior fossa hematomas. (Fig.3) The patient was discharged home in stable condition.

Our case no. 2 was a 26-years old patient sustained electric shock while he was painting the wall. The electric shock threw the patient 2-meters away from the wall, he fell on a hard ground, but had no post-traumatic loss of consciousness, vomiting, convulsions or bleeding per ear nose or mouth. On examination the patient was fully conscious, oriented, GCS was 15, both pupils were equal and reactive, he had ecchymosis and swelling in the right eye with subgaleal swelling. There was deep burn over both hands and feet. The patient was adequately resuscitated and shifted to CT room. CT Brain showed small temporo-occipital acute extradual haematoma, temporal bone fracture and a large overlying sub-galeal hematoma.(Fig.4) The patient was initially admitted to the ICU and kept on conservative management under close observation. 6-hours later, the patient became agitated with detoriation in the level of consciousness, GCS dropped to 8/15 E2V2M4, Rt pupil was 4mm sluggishly reactive to light while Lt pupil was 3mm reactive to light. The patient was intubated and then was immediately shifted to CT room. Follow up CT Brain showed enlargement of the 3 hematomas with significant mass effect and midline shift.(Fig.5) The patient was immediately shifted to OR from the CT room and the EDH was immediately evacuated through a large craniotomy flap. Post-operatively the patient was successfully extubated, became fully

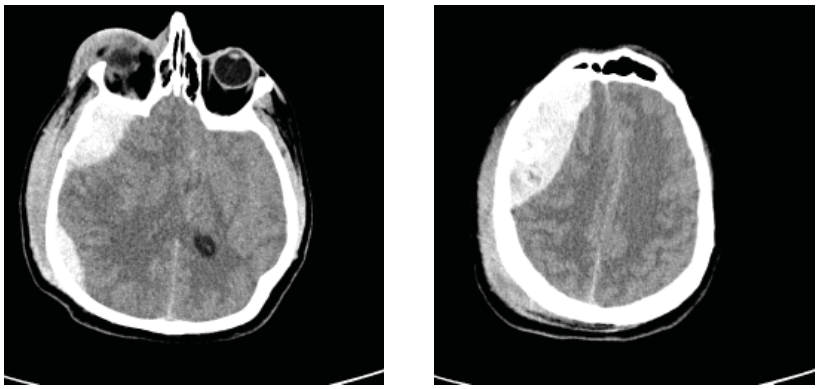

Figure-5: Follow up CT Brain done 6 hours later when the patient deteriorated clinically. (a) Larger right temporal and occipital acute EDHs with complete obliteration of the ipsilateral ventricle from the mass effect.(b) Enlargement of the right frontal EDH with significant midline shift

Figure-6: The follow up CT scan obtained in the first post-operative day. (a) Adequately removed temporal and occipital hematomas with no more mass effect on the ipsilateral ventricle, occipital horn started to appear after the evacuation of the hematoma. (b) After evacuating the frontal EDH no more midline shiftor displacement of the falx. (c) Bone window demonstrating the size and extent of the craniotomy bone flap used for hematoma evacuation

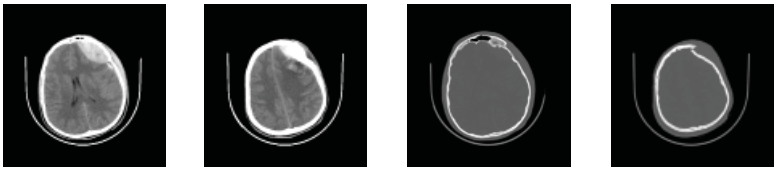

Figure-7: The initial CT brain obtained in the ED. (a) Large acute Edh with contusion just posterior to the hematoma. (b) The area of the depressed fracture showing the underlying brain contusion. (c) Bone window demonstrating the fracture line that is going through the pterion. (d) Bone window demonstrating the extent of the depressed fracture in the left frontal region

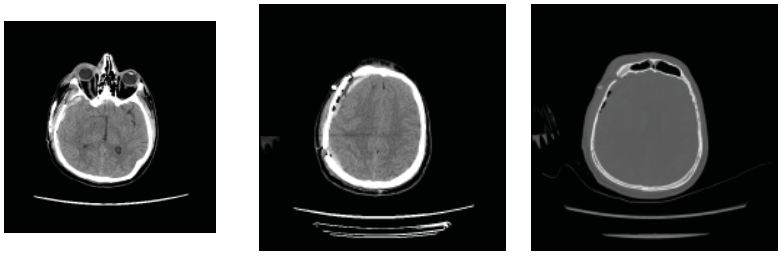

Figure-8: The follow up CT Brain done in the 1st post-operative day. (a) A more medially located EDH towards the superior sagittal sinus and the falx. (b) The contusion became clearer after the removal of the first hematoma with minimal pneumocephalus denoting the recent surgery. (c)The bone window is showing avulsed bone flap from the mass effect of the underlying EDH

conscious, pupils became equal and reactive and was hemodynamically stable. Follow up CT Brain showed adequate evacuation of the hematomas with no post-operative complications. (Fig.6) The patient was then discharged from neurosurgery after 3-days.

Our case no.3 was a 18-years old male with history of assault to the head with perfume bottle. No history of of loss of consciousness or convulsions. He presented to the ER with headache and vomiting for 3-times. On examination the patient was fully conscious oriented, GCS 15, both pupils were equal and reactive, no cranial nerve impairment and no neurological deficit at the four extremities. CT Brain showed large left frontal acute EDH with underlying left frontal hemorrhagic contusion and an overlying depressed fracture and a fracture line extending to the left pterion. (Fig.7) The patient was admitted to the ICU and prepared for urgent craniotomy. He was operated with elevation of the depressed skull fracture, evacuation of acute EDH (bleeding source was found from the main stem of the middle meningeal artery) and water-tight dural repair for the dural tear which was found beneath the depressed fracture. Post-operatively the patient was fine, both the headache and the vomiting resolved. As per routine, the patient was shifted for follow up CT Brain in the 1st post-operative day. Follow up CT Brain showed recollection of the hematoma with more medial position towards the anterior part of the superior sagittal sinus. (Fig.8) Re-exploration done again, and the bone flap was extended down to the anterior skull base and the whole hematoma was removed. Post-op the patient immediately extubated, he was fully conscious oriented with no neuro deficit. The patient discharged home 2-days later.

Our case no. 4 was a 30-years old patient brought to the Resusitation Room in King Abdullah Hospital- Bisha with history of fall from height about 5-feet. On examination the patient was agitated, and irritable initial GCS was 10, pupils were equal and reating to light, moving both sides, PR 59, BP 135/77, O_2 saturation 92%. The patient was resuscitated in the RR and intubated by ICU team. CT Brain showed sub arachnoid haematoma (SAH) fisher grade-III, bi-frontal

Figure-9: The initial CT brain of the patient showing (a)a small Lt occipital acute EDH with a small hypodense spot ?? swirl sign. (b) SAH Fisher grade II in the Lt sylvian and interhemispheric with bifrontal hypodensity suggesting brain contusions and small size lateral ventricles. (c) interhemispheric SAH and loss of the frontal cortical sulci bilaterally from the brain oedema. (d) Lt occipital linear fissure fracture behind the mastoid bone with no blood inside the mastoid air sinus

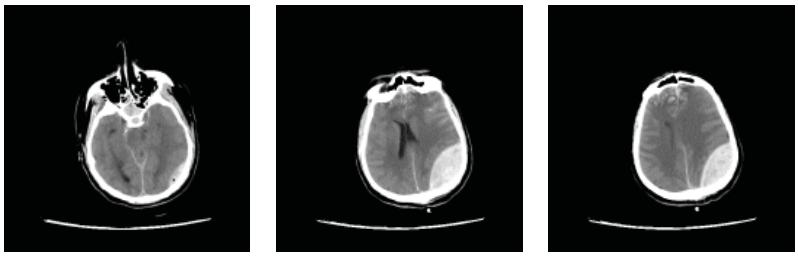

Figure-10: (a) A relatively larger more hyperdense acute EDH with more oedema obliterating the basal cisterns, hypodensities in the Lt lateral pons and the superior cerebellar surface in the tentorial hiatus "ischemia vs. small contusions". (b&c) Very large acute EDH with more pronounced oedema and compression on the ipsilateral ventricle with some midline shift and a clearer bilateral subfrontal hemorrhagic contusions

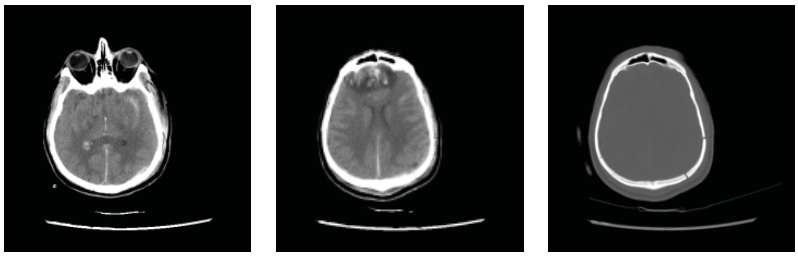

Figure-11: (a) Completely evacuated EDH with no more compression on the ipsilateral ventricle, there is minimal intraventricular hemorrhage with no hydrocephalus "SAH Fisher grade IV". (b) resolving bifrontal hemorrhagic contusions. (c) Showing the site for the bone flap of the craniotomy

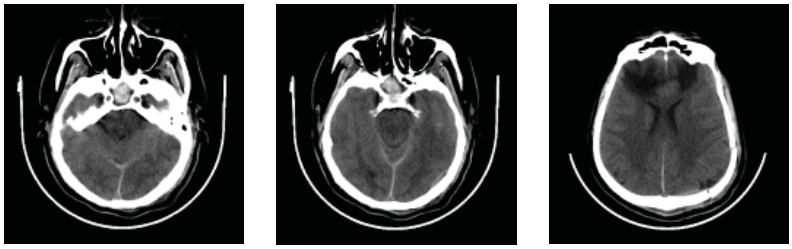

Figure-12: : (a & b) Showing hypodensity in the brain stem in the pons and extending to the junction between the pons and the midbrain just anterior to the aqueduct of Sylvius denoting ischemic changes and justifying the reason for the delayed post-operative recovery. (c) Bifrontal hypodensity at the site of the pre-existing bifrontal hemorrhagic contusions denoting resolved bifrontal contusions

hemorrhagic contusions, generalized brain edema and a small acute left occipital acute EDH with overlying linear skull fracture extending down to the base just behind the mastoid bone. (Fig.9) The patient was shifted to the ICU, connected to the mechanical ventilator and kept on conservative management. After 2-hours both pupils became fully dilated 5m non-reactive and he was immediately shifted to CT room. CT Brain showed larger EDH that was extending to the parieto-occipital region (Fig.10), the patient was immediately shifted to OR, craniotomy was done, and the hematoma was evacuated, and hemostasis was secured. The patient was shifted back to the ICU intubated. Follow-up CT Brain done (Fig.11). Follow-up CT Brain showed brainstem hypodensity just anterior to the aqueduct with encephalo-malacia at the site of old bifrontal hemorrhagic contusions.(Fig.12). He was discharged home in this condition, and he is currently under extensive rehabilitation physiotherapy and nursing care.

All of the patients in this series were males aging between 18 to 30 years old. Most of the patients had EDH in the occipital regions (3/4, 75%) associated with an overlying fracture (3/4, 75%), the source of bleeding was the dural venous sinus in 50% of the cases and arterial in the other half. The duration to DEDH was ranging from 2 to 24 hours and the indications for doing follow-up CT Brain were performed due to increase in the headache (1/4), deterioration in the level of consciousness (2/4) and as a routine post-operative follow up in an asymptomatic patient (1/4). All 4-patients were admitted and closely monitored in the ICU. The 3-patients were cured (3/4, 75%) while one improved and remained under rehabilitation physiotherapy for the associated other brain and brainstem injuries (1/4, 25%).

Discusion:

Delayed extradural hematoma has the same presentation of the regular EDH and is difficult to diagnose as there is no constant features suggesting its development.[20] It affects the same age group affected with EDH. All mechanisms of injury can cause DEDH including road traffic accidents, assault, fall from height and even iatrogenic from any post-operative cranial procedure.[7,8,10,12,19,20]

The presence of an associated skull fracture is not a constant feature and can only be detected intra-operatively.[21] The indications for doing urgent follow up CT brain includes, change in the magnitude or the frequency of the headache, irritability, any deterioration in the level of consciousness, change in the pupils reactivity to light and if there is new neurological deficit.[2,20]

Conclusion:

Delayed extradural haematoma is a rare condition but associated with a very high mortality and morbidity rates. All patients with EDH should be initially admitted to the ICU for at least 24-hours and should get their follow up CT Brain done before shifting them to the ward. The earlier the intervention, the better the outcome. Patients with DEDH associated with other brain injuries may have a poorer prognosis when compared to those without associated brain injuries. Up to now, there is no constant features suggesting the development of DEDH and further studies are needed to establish standard management guidelines for early detection of patients potential to develop DEDH.

Conflict of interest: None

Funding source: None

Role and contribution of authors:

Dr Mohammed Awad Elzain, the conception and design of the work, analysis, interpretation of data for the work, writing the article including the literature review and operating on some of the patients in the series as the main primary surgeon

Dr Umar Farooq Abdelhai, Revising the article critically for important intellectual content and final approval of the version to be published, besides operating in some patients as the primary surgeon

Dr Boussad Kermoud, revising the article critically for important intellectual content and final approval of the version, besides operating in some patients as the primary surgeon

Dr Naveed Akhter Chawla, Revising the article critically for important intellectual content and final approval of the version to be published, besides assisting the primary surgeons in operating the patients.

Reference:

1. Fankhauser H, Uske A, de TN. [Delayed epidural hematoma. Apropos of a series of 8 cases]. Neurochirurgie 1983;29:255-260.
2. Radulovic D, Janosevic V, Rakic M, Durovic B, Slavik E, Lakicevic N. [Delayed epidural hematoma after mild head injury]. Vojnosanit Pregl 2005;62:679-682.
3. Rivas JJ, Lobato RD, Sarabia R, Cordobes F, Cabrera A, Gomez P. Extradural hematoma: analysis of factors influencing the courses of 161 patients. Neurosurgery 1988;23:44-51.
4. Milo R, Razon N, Schiffer J. Delayed epidural hematoma. A review. Acta Neurochir (Wien) 1987;84:13-23.
5. Feuerman T, Wackym PA, Gade GF, Lanman T, Becker D. Intraoperative development of contralateral epidural hematoma during evacuation of traumatic extraaxial hematoma. Neurosurgery 1988;23:480-484.
6. Kwan AL, Howng SL, Sun ZM, Lin CN. Delayed onset of traumatic epidural hematoma. Gaoxiong Yi Xue Ke Xue Za Zhi 1989;5:683-687.
7. Solomiichuk VO, Drizhdov KI. Contralateral delayed epidural hematoma following intracerebral hematoma surgery. Surg Neurol Int 2013;4:134.
8. Wu R, Shi J, Cao J, Mao Y, Dong B. Two occurrences of delayed epidural hematoma in different areas following decompressive craniectomy for acute subdural hematoma in a single patient: a case report. BMC Surg 2017;17:123.
9. Talbott JF, Gean A, Yuh EL, Stiver SI. Calvarial fracture patterns on CT imaging predict risk of a delayed epidural hematoma following decompressive craniectomy for traumatic brain injury. AJNR Am J Neuroradiol 2014;35:1930-1935.
10. Wang QP, You C. Predictive value of calvarial fracture for delayed epidural hematoma following decompressive craniectomy. AJNR Am J Neuroradiol 2015;36:E24.
11. Sokolowski MJ, Dolan M, Aminian A, Haak MH, Schafer MF. Delayed epidural hematoma after spinal surgery: a report of 4 cases. J Spinal Disord Tech 2006;19:603-606.
12. Kim B, Moon SH, Kim SY, Kim HJ, Lee HM. Delayed Spinal Epidural Hematoma after En Block Spondylectomy for Vertebral Ewing's Sarcoma. Asian Spine J 2010;4:118-122.
13. Radulovic D, Janosevic V, Djurovic B, Slavik E. Traumatic delayed epidural hematoma. Zentralbl Neurochir 2006;67:76-80.
14. Rappaport ZH, Shaked I, Tadmor R. Delayed epidural hematoma demonstrated by computed tomography: case report. Neurosurgery 1982;10:487-489.
15. Hahn YS, McLone DG. Risk factors in the outcome of children with minor head injury. Pediatr Neurosurg 1993;19:135-142.
16. Summers LE, Mascott CR. Delayed epidural hematoma: presentation in a pediatric patient. J La State Med Soc 2001;153:81-84.
17. Cervantes LA. Concurrent delayed temporal and posterior fossa epidural hematomas. Case report. J Neurosurg 1983;59:351-353.
18. Fumeya H, Ito K, Okiyama K et al. [MR imaging of traumatic cerebellar dysfunction]. No Shinkei Geka 1990;18:279-283.
19. Lee S, Park SK, Joo BE, Lee JA, Kong DS, Park K. The pathogenesis of delayed epidural hematoma after posterior fossa surgery. J Clin Neurosci 2018;47:223-227.
20. Radulovic D, Janosevic V, Djurovic B, Slavik E. Traumatic delayed epidural hematoma. Zentralbl Neurochir 2006;67:76-80.
21. Di RA, Ellis SJ, Landes C. Delayed epidural hematoma. Neuroradiology 1991;33:253-254.