ORIGINAL ARTICLE

Is the presence of linear fracture a predictor of delayed posterior fossa epidural hematoma?

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ABSTRACT

BACKGROUND: Though traumatic posterior fossa epidural hematoma (PFEDH) is rare, the associated rates of morbidity and mortality are higher than those of supratentorial epidural hematoma (SEDH). Signs and symptoms may be silent and slow, but rapid deterioration may set in, resulting in death. With the more frequent use of computed tomography (CT), early diagnosis can be achieved in patients with cranial fractures who have suffered traumatic injury to the posterior fossa. However, some hematomas appear insignificant or are absent on initial tomography scans, and can only be detected by serial CT scans. These are called delayed epidural hematomas (EDHs). The association of EDHs in the supratentorial-infratentorial compartments with linear fracture and delayed EDH (DEDH) was presently investigated.

METHODS: A total of 212 patients with SEDH and 22 with PFEDH diagnosed and treated in Göztepe Training and Research Hospital Neurosurgery Clinic between 1995 and 2005 were included. Of the PFEDH patients, 21 underwent surgery, and 1 was followed with conservative treatment. In this group, 4 patients underwent surgery for delayed posterior fossa epidural hematoma (DPFEDH).

RESULTS: Mean age of patients with PFEDH was 12 years, and that of the patients with SEDH was 18 years. Classification made according to localization on cranial CT, in order of increasing frequency, revealed of EDHs that were parietal (27%), temporal (16%), and located in the posterior fossa regions (approximately 8%). Fracture line was detected on direct radiographs in 48% of SEDHs and 68% of PFEDHs. Incidence of DPFEDH in the infratentorial compartment was statistically significantly higher than incidence in the supratentorial compartment ($p=0.007$). Review of the entire EDH series revealed that the likelihood of DEDH development in the infratentorial compartment was 10.27 times higher in patients with linear fractures than in patients with supratentorial fractures ($p<0.05$).

CONCLUSION: DPFEDH, combined with clinical deterioration, can be fatal. Accurate diagnosis and selection of surgery modality can be lifesaving. The high risk of EDH development in patients with a fracture line in the posterior fossa on direct radiographs should be kept in mind. These patients should be kept under close observation, and serial CT scans should be conducted when necessary.

Key words: Delayed epidural hematoma; head trauma; posterior cranial fossa.

INTRODUCTION

Traumatic posterior fossa epidural hematoma (PFEDH) is less common, compared to supratentorial epidural hematoma (SEDH). PFDEH accounts for 0.3% of all intracranial hematomas and 4–12% of all epidural hematomas (EDHs).[1–6] PFDEH usually develops secondary to trauma to the occipital, suboccipital, and retromastoid regions. Though clinical progression is slow, neurological progression is rapid and fatal in the absence of timely intervention. Diagnosis of PFEDH can be established by cranial computed tomography (CT) or magnetic resonance imaging.[7] Hematoma that induces mass effect should be surgically treated.

Delayed posterior fossa epidural hematoma (DPFEDH) is extremely rare, but can occur following head injury. DPFEDH is defined as the absence of EDH with or without linear fracture on initial CT, followed by development or deterioration of clinical symptoms, or detection of EDH on serial CT scans.

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Presence of linear fracture is a demonstrated risk factor for delayed supratentorial epidural hematomas (DSEDHs). In the literature, DSEDH accounts for 9–10% of all EDHs. However, its potential as a risk factor for DPFEDH has yet to be addressed.[8–13]

The present patient series included 234 patients with traumatic cranial EDH treated in Göztepe Training and Research Hospital Neurosurgery Clinic between 1995 and 2005, 22 of whom were diagnosed with PFEDH, and 212 of whom were diagnosed with SEDH. Patients were retrospectively compared and evaluated based on clinical and radiological findings. The present objective was to investigate whether linear fracture was a risk for DPFEDH development, and whether there was a difference in risk severity between linear fracture in the infratentorial and supratentorial compartments.

MATERIALS AND METHODS

A total of 234 patients treated for traumatic cranial EDH between 1995 and 2005 were retrospectively analyzed based on clinical and radiological findings. From this series, findings of 22 patients with PFEDH (9.4%) and 212 (90.6%) with SEDH were retrospectively evaluated.

Delayed EDH was detected in 4 of the 22 PFEDH patients and 2 of the 212 SEDH patients. Underlying causes were investigated. Direct radiographs were obtained in the lateral and anteroposterior planes in the 234 patients with head injury. Those with trauma to the occipital, suboccipital, or retromastoid regions were also examined with Towne's radiography. In patients with fractures, fracture type and whether the fracture had crossed venous sinuses were noted. CT scan was performed in all patients, and recorded findings included enlargement of the trigone, temporal horn, and 3rd ventricle, 4th ventricle compression and shift, basal-ambient and quadrigeminal cistern compressions, and whether hematomas were bilateral. In addition, EDH volume was calculated using the equation: 0.5 height x depth x length.

Upon admission, severity of head injury was classified according to the Glasgow Coma Scale (GCS) by Narayan,[14] as mild (GCS: 13–15), moderate (GCS: 9–12), or severe (GCS: 3–8). SEDH patients with a hematoma of less than 30 cm^3 in volume, less than 15 mm in thickness, and with a midline shift of less than 5 mm were clinically observed and followed up, as were those with GCS greater than 8 but with no focal neurological deficit. In patients with PFEDH of greater than 5 mm in thickness on CT, and greater than 15 cm^3 in volume, with mass effect resulting in fourth ventricle shift, ventricle compression and perimesencephalic cistern compression were surgically evacuated.

Level of consciousness and severity of head injury on admission were considered during postoperative follow-up. Initial Glasgow Outcome Score (GOS) and examination results of all patients were recorded. Of the patients diagnosed with PFEDH, 21 received surgical treatment, and 1 received conservative treatment. While type of surgery varied, depending on hematoma localization, paramedian or median suboccipital craniotomy was performed, as was evacuation of the EDH, followed by detection of the bleeding site and bleeding control, after which surgery was terminated. Of the 22 PFEDH patients, only 4 had DPFEDH, all of whom had linear fractures. Two SEDH patients developed DSEDH, none of whom had linear fractures.

Statistical Analysis

Statistical analysis was performed using the GraphPad Prism program (version 3; GraphPad Software, Inc., San Diego, CA, USA). Mann-Whitney U-test was used to compare data with descriptive statistical methods (mean, SD), as well as with paired data. Fisher's exact test and relative risk were used to compare qualitative data. A p value of $p<0.05$ was considered statistically significant.

RESULTS

A total of 234 patients were analyzed retrospectively based on clinical and radiological features. Eight patients diagnosed with PFEDH (36%) were female, and 14 (64%) were male, with a ratio of 2:3. Fifty-one patients with SEDH were female (24%), and 161 (76%) were male, with a ratio of 1:3. Of the patients with EDH, 22 were diagnosed with PFEDH, and 212 with supratentorial EDH. Of the patients with PFEDH, 4 were diagnosed with DPFEDH (Fig. 1a–d). Of the patients with SEDH, 2 were diagnosed with DSEDH.

Mean age of patients with PFEDH was 12 years, while that of patients with SEDH was 18 years. Incidence of supratentorial and infratentorial EDH in the 1st decade of life was significantly high ($p<0.05$), while incidence in the 2nd decade of life was quite considerably high: 56% and 82% in SEDH and PFEDH patients, respectively. Demographic characteristics are presented in Table 1.

The most common causes of traumatic posterior fossa EDH include fall from a height, traffic accident, assault, and collision of the head with a solid object. Signs and symptoms of the 22 PFEDH patients were headache (17 patients), nausea and vomiting (15), impaired consciousness (14), and retromastoid, occipital, and suboccipital swelling (12). Though less common, symptoms including loss of consciousness, cerebellar dysfunction, diplopia, abducens paralysis, otorrhagia, neck stiffness, and anisocoria were encountered in otherwise asymptomatic patients (Table 2). Two of the 4 patients with DPFEDH presented with headache and nausea/vomiting, the other 2 with neurological disorientation. All were discharged in GOS 5 condition.

Of the 212 patients with SEDH, 169 had swelling at the site

Table 1. Demographic factors

	Supratentorial EDH	Infratentorial EDH
Gender		
Male	161	14
Female	51	8
Age (median)	18	12
Trauma type		
Fall from height	63	10
Traffic accident	90	4
Assaults & collisions	59	8

ED: Epidural hematomas.

Table 2. Comparison of cranial tomographic findings of posterior fossa epidural hematomas (PFEDHs) vs delayed posterior fossa epidural hematomas (DPFEDHs)

	PFEDH	DPFEDH	p
Temporal horn			0.07
Normal	14	1	
Enlargement	4	3	
3th ventricle			0.259
Normal	14	2	
Enlargement	4	2	
Trigone			0.01**
Normal	17	1	
Enlargement	1	3	
4th ventricle			0.117
Normal	13	1	
Compressed or shift	5	3	
Basal cisterns			0.073
Normal	17	2	
Compressed	1	2	
Ambient cisterns			0.029**
Normal	12	0	
Compressed	6	4	
Quadrigeminal cisterns			0.09
Normal	11	0	
Compressed	7	4	
Localization			0.21
Unilateral	15	2	
Bilateral	3	2	

of injury. The other most common symptoms included headache, nausea/vomiting, impaired consciousness, loss of consciousness, lateralizing signs (hemiparesis, Babinski reflex), and herniation. Two patients had delayed SEDH in this group. There was no need to evacuate the hematoma, and a conservative approach was adopted. Both patients were discharged in GOS 5 condition.

Classification according to localization on cranial CT, in order of increasing frequency, revealed EDH in the temporal (16%), temporoparietal (22%), and parietal (27%) regions, while incidence of EDH in the posterior fossa was approximately 8% in the present series (Fig. 2). Overall incidence of DPFEDH was 2%. Regarding localization of PFEDH in the infratentorial compartment on CT scan, 5 EDHs showed bilateral and 17 unilateral localization. Two DPFEDHs showed bilateral and 2 showed unilateral localization. Mean PFEDH volume was 13.5 cm^3 (range: 2.3–45 cm^3).

Fracture line was detected on direct radiography of 102 (48%) of the 212 SEDH patients, while direct radiographs of 110 (52%) patients showed normal findings. DSEDH was detected during follow-up of 2 patients with no fracture line on direct radiography (0.09%). Linear fracture was identified in 15 (68%) of the 22 PFEDH patients. Twelve patients had paramedian occipital fracture and 3 had occipital fracture crossing the transverse sinus at the midline. DPFEDH was noted in 4 patients (18%) whose direct radiographs revealed the presence of a fracture line.

No statistically significant difference was found in fracture distribution between patients with PFEDH and those with SDEH (p=0.11). However, occurrence of EDH was 2.14 times more likely in the infratentorial compartment than in the supratentorial compartment in patients with linear fracture (p>0.05). Incidence of DPFEDH was statistically significantly higher in the infratentorial compartment than in the supratentorial compartment (p=0.007). Probability of DPFEDH development in the presence or absence of fracture was 4.66 times higher in the infratentorial compartment than in the supratentorial compartment (p<0.05). Incidence of DPFEDH in the infratentorial compartment, in the presence of fracture, was statistically significantly higher than in the supratentorial compartment (p=0.0002). A review of the entire EDH series revealed that the probability of delayed EDH (DEDH) development in the infratentorial compartment in patients with linear fracture was 10.27 times higher than in patients with supratentorial fracture (p<0.05).

PFEDHs and DPFEDHs were compared in terms of CT findings including ventricle compression and shift, and enlargement of the trigone, temporal horn, and third ventricle, indicating development of hydrocephalus. In cases of DPFEDH, enlargement of the trigone and compression of the ambient cistern, among basal cisterns, were significantly increased (p=0.01 and p=0.029, respectively) (Table 2).

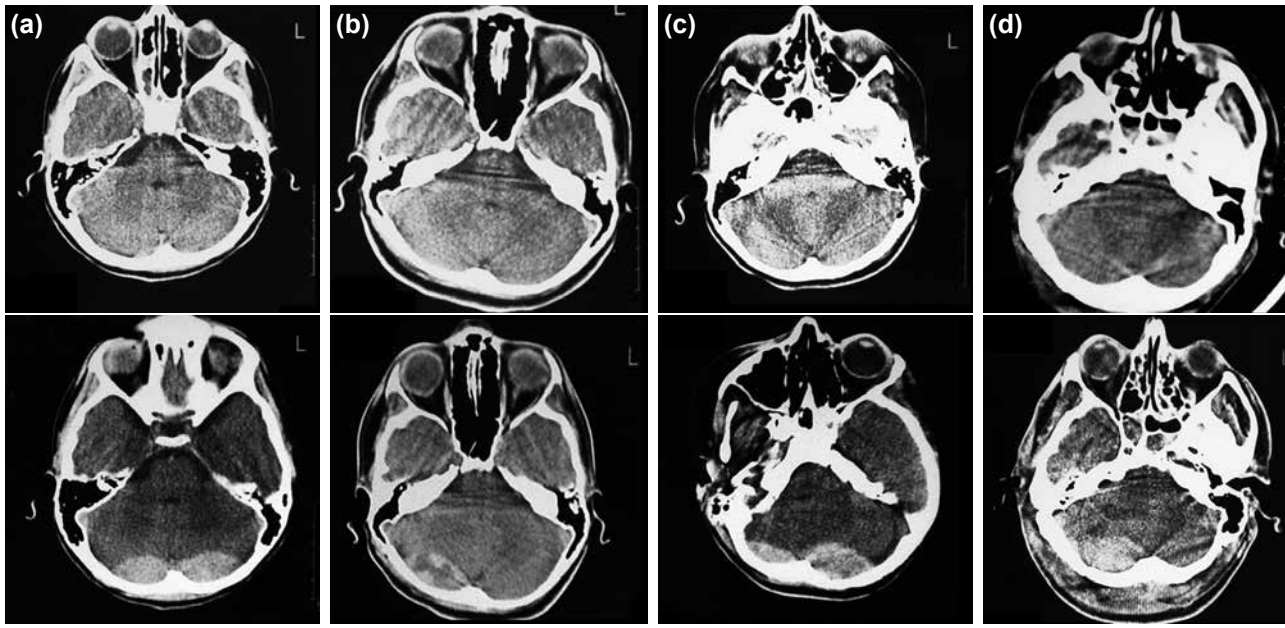

Figure 1. (a) Case 1: S.K., 9 years old, initial computed tomography (CT) view (above) and view after 24 hours (below). **(b)** Case 2: Ş.S., 13 years old, initial CT view (above) and view after 10 hours (below). **(c)** Case 3: S.Y.; 10 years old, initial CT view (above) and view after 12 hours (below). **(d)** Case 4: T.Ş., 33 years old, initial CT view (above) and view after 12 hours (below).

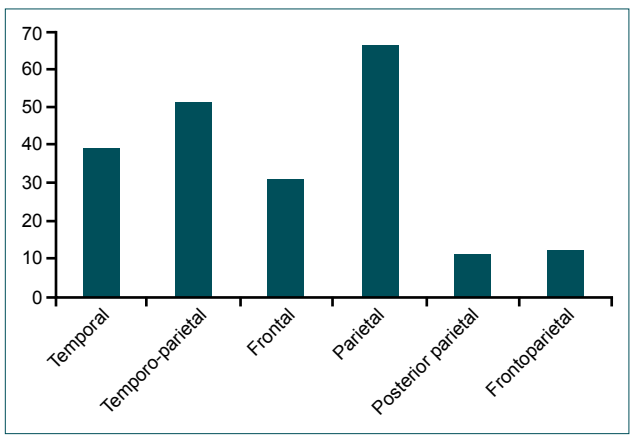

Figure 2. Distribution of epidural hematomas (EDHs) according to localization on cranial CT.

DISCUSSION

EDHs are 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, EDHs constitute a threat to life.[15,16] These lesions may develop slowly and can be manageable, though prediction of the course of EDH is challenging.

The posterior fossa is a rare location for EDH. In PFEDH, the deterioration of symptoms may be slow and silent. However, neurological progression is rapid and can be fatal if left untreated. Early diagnosis is crucial for survival. Diagnosis and cure of PFEDH was possible following the first case of successful surgery, reported by Coleman and Thompson in 1941.[17] However, until recently, diagnosis was challenging. As in the first published case series, documented by Campbell, Fisher, Hooper, and Petit-Dutaillis et al., the difficulty in establishing diagnosis resulted in the deaths of almost half the patient population.[18–21] These authors emphasized that half of the population was in the pediatric age group and had occipital fractures crossing the sinus. Recently, the frequent use of CT has facilitated PFEDH diagnosis in patients with head injury, and the number of patients diagnosed with DEDH has increasingly grown. Some authors suggest that a CT scan should be performed in each patient with swelling in the occipital region or fracture of the occipital bone.[22,23] In the present series, CT scan was performed, after neurological progression, in 4 patients with linear fracture, and DPFEDH was noted in all.

EDH is an accumulation of blood resulting from bleeding of extracerebral vessels, leading to an extra axial mass. Irreversible damage is difficult to prevent, due to epidural hemorrhage resulting in brain herniation and increased intracranial pressure. Today, the increasingly frequent use of CT in the differential diagnosis of patients with head injury has facilitated the diagnosis of EDH, resulting in a decreased mortality rate.[24] A control CT scan performed in the first 24 hours in patients kept under observation following head trauma can detect DPFEDH. Radiological changes precede clinical progression.[25] Though the mortality rate of DEDH has been reported as 5%, it was approximately 4.5% in the present series.

The majority of the present PFEDH patients were in the second decade of life. Harwood and Nash reported that 30% of patients with linear fracture developed EDH, and that EDH can develop following trauma to the occipital region due to abundant diploic and dural vascularization in infants and chil-

dren. Most of the present PFEDH patients (68%) were in the pediatric age group. Surgical intervention was not performed in 1 patient with PFEDH based on cranial CT findings, which showed a hematoma less than 5-mm thick, 10 cm^3, and without mass effect. Of the patients who underwent surgery, those with bilateral EDH underwent median craniectomy, while those with unilateral EDH underwent suboccipital craniectomy on the side where the lesion was located.

According to reports, incidence of bilateral PFEDH is approximately 30%, while it was approximately 23% in the present series.[8] In most cases, bleeding arises from the venous sinus, from posterior branches of the meningeal artery or diploic veins, and from the dural sinuses. The transverse or sigmoid sinus is usually responsible for acute bleeding.[26] A review of the literature revealed that prognosis of EDH is better in chronic and subacute cases. Prognosis is poor in acute cases, and perimesencephalic cistern-basal cistern compressions due to rapid mass effect caused by hematoma can be associated with mortality. In the present study, basal cistern compression was significantly higher in cases of DPFEDH, among all cases of PFEDH. Likewise, in spite of a significant increase in the width of the trigone, it is likely that mortality is associated with acute brain stem-pons compression and acute hydrocephalus due to ventricular occlusion.

The presence of a linear fracture is a risk factor for EDH. Results of a case series comparing 77 patients with primary EDH and DEDH by Poon et al. showed that primary brain damage was associated with linear fracture, and that hemorrhage arising from torn dura mater or venous injury was more common, compared to meningeal artery injuries. The authors also made clear that DEDH-related symptoms including hyperventilation, osmotic diuretics, otorrhea, surgical decompression, hypovolemia, or hypotension were not included in their study. Several studies have reported incidence of DEDH in patients with minor head trauma and linear fracture to be approximately 1%.[27] In most series, incidence of DEDH in patients with or without fractures was approximately 9–10%. This number is likely to increase as CT becomes is more widely used. Poon et al. reported that incidence of DEDH in traumatic EDH was about 30%.[24] Adeloye and Onabanjo suggested that DEDH could be of venous origin, and that administration of hyperosmolar agents such as mannitol may result in the formation of DEDH through loss of the intracranial pressure-related tamponade effect on small dural venules.[9] A case report by Koulouris and Rizzoli demonstrated that contralateral EDH developed after alleviation of the tamponade effect of EDH following surgical decompression.[22]

Goodkin and Zahniser,[16] in 1978, reported a case in which DEDH was documented by serial angiography. They suggested that DEDH could have been caused by increased blood pressure in a previously hypotensive and hypovolemic patient. A review of relevant studies showed that linear fracture line was present in 70% of PFEDH patients, and that most patients were in the pediatric age group. This finding supports the hypothesis that DEDH can be caused by a linear fracture, and is likely of diploic venous origin in this compartment.[4] In the present series, a linear fracture was present in 50% of EDH patients. In the present study, risk of PFEDH development in the presence of a linear fracture was 10.27 times higher than in patients with supratentorial fracture.

Though the posterior cranial fossa is the largest of the 3 cranial fossae, the comparison of supratentorial and infratentorial compartments reveals differences in the development of EDH in the presence of a linear fracture. In the present study, probability of EDH development in patients with fracture was 2.14 times higher in the infratentorial compartment than in the supratentorial compartment, and 64% of patients with infratentorial EDH were in the pediatric age group. In addition, risk of DEDH development was 4.66 times higher in the infratentorial compartment, compared to the supratentorial compartment. In cases of head trauma, severity is correlated with the presence of linear fracture and primary brain damage. In a study by Roda et al.,[28] infratentorial and supratentorial lesions were found in 39.7% of patients with head-injury-related EDH. In the present series, PFEDH accompanied 45% of lesions. Outcome of traumatic EDH, an extracerebral lesion, is much better than those of other traumatic intracranial bleedings, when treated properly. Early diagnosis and surgery, if necessary, is the key point of treatment. Patients should be closely observed and followed up with CT, particularly those with head injury and linear fracture in the posterior fossa.

Conclusion

DPFEDH, combined with clinical deterioration, can be fatal. Accurate diagnosis and choice of surgical modality can be life-saving. However, the high risk of EDH in patients with a fracture line in the posterior fossa on direct radiography should be kept in mind. These patients should be kept under close observation and serial CT scans should be performed when necessary.

Conflict of interest: None declared.

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ORİJİNAL ÇALIŞMA - *ÖZET*

Lineer kırık varlığı, geç gelişen posterior fossa epidural hematomu açısından bir risk faktörümüdür?

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AMAÇ: Travmatik posterior fossa epidural hematomları ender görülmelerine karşın mortalite ve morbiditesi suratentorial yerleşimli epidural hematomlardan daha yüksektir. Belirti ve bulguları silik ve belirsiz olmalarının yanında hızlı bir kötüleşme göstererek bilinç bozukluğundan komaya ve sonuçta ölüme yol açabilir. Bilgisayarlı tomografinin (BT) kullanımının posterior fossa travması geçirmiş kranium kırığı bulunan olgularda yaygınlaşması ile erken tanı konulabilmektedir. Ancak ilk çekilen tomografide görülmeyip seri çekimlerde yakalanan hematomlara geç gelişen epidural hematom'lar denmektedir. Çalışmamızda supratentorial-infratentorial kompartmanlarda gelişmiş epidural hematomların (EDH) lineer kırık varlığı ile ilişkisi ve geç dönemde gelişen epidural hematomlarla ilişkisi incelendi.

GEREÇ VE YÖNTEM: Bu çalışmada 1995–2005 yıllarında kliniğimizde 212 supratentorial epidural hematom (SEDH) ve 22 posterior fossa epidural hematomu (PFEDH) tanısı ile tedavi edilen olgular sunuldu. PFEDH olgularından 21'i ameliyat edildi, 1 olgu konservatif takip edildi, bu grubun içinden 4 olgu ise gecikmiş posterior fossa epidural hematomu (GPFEDH) nedeniyle ameliyat edildi.

BULGULAR: Posterior fossa epidural hematomu nedeniyle tedavi ettiğimiz hastaların yaş ortalamaları 12, SEDH nedeniyle tedavi ettiğimiz hastaların yaş ortalamaları 18 idi. Hematomlar, parietal bölgede %27, temporal bölgede %16 ve posterior fossada %8 oranlarında görülmekteydi. Supratentorial epidural hematom olan 212 hastanın %48'inde, PFEDH olgularının %68'inde lineer kırık mevcuttu. Posterior fossada geç gelişen eipdural hematom görülme sıklığı %2 idi. İnfratentorial kompartmanda geç gelişen epidural hematom görülme olasılığı ileri derecede supratentorial kompartmana nazaran anlamlıydı (p=0.007). Çalışmamızda lineer kırığı olupta posterior fossada EDH gelişme oranı 10.27 kat daha bulundu (p<0.05).

TARTIŞMA: Geç gelişen posterior fossa epidural hematomları klinik detoryantasyonla beraber hastayı ölümcül sonuçlara götürebilmektedir. Gecikmiş posterior fossa epidural hematomlarının tanısı ve cerrahi modalite hayat kurtarıcı olmakla beraber posterior fossa direkt grafilerinde kırık hattı tespit edilen hastalarda yüksek oranda EDH gelişebileceği göz önünde bulundurulmalı, bu tarzda olan hastaları yakın gözlem altında tutmalı gerektiğinde seri BT çekimleri yapılmalıdır.

Anahtar sözcükler: Geç gelişen epidural hematom; kafa travması; posterior kranial fossa.

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