



## Case Report

### Low Molecular Weight Heparin Induced Delayed Traumatic Clival Subdural Hematoma Associated With Isolated Abducens Nerve Palsy in a Child

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## Summary

Traumatic clival subdural hematoma is a very rare clinical entity. This rarity can result in delayed or missed diagnoses. Although several hypotheses are proposed to explain this lesion, the exact pathophysiology remains to be unknown.

We present a case of low-molecular-weight heparin induced clival subdural hematoma, extending to the left cerebellopontine angle and causing left abducens nerve palsy which occurred six days after trauma.

**Key words:** Clival Subdural hematoma, low-molecular-weight heparin, trauma, mechanism

**Bir çocuk olguda düşük molekül ağırlıklı heparin kullanımına bağlı gelişen ve izole abducens sinir felci ile birliktelik gösteren gecikmiş travmatik klival subdural hematom**

## Özet

Travmatik klival subdural hematom çok nadir olarak görülmektedir. Bu durum tanı koymada güçlüğe veya gecikmeye sebep olabilmektedir. Klivus bölgesi subdural hematomların oluşum mekanizmaları ile ilgili olarak birçok hipotez öne sürülmüş olmasına rağmen, esas patofizyoloji hala tam olarak bilinmemektedir. Biz burada, travma sonrası takip edilen ve düşük molekül ağırlıklı heparin tedavisi alan çocuk hastanın travmadan 6 gün sonra izole VI. sinir felcinin eşlik ettiği, sol pontoserebellar bölgeye uzanım gösteren klival subdural hematom olgusunu sunmaktayız. Konu ile ilgili makalelerin ışığında düşük molekül ağırlıklı heparine bağlı gelişen klival subdural hematomun mekanizmasını açıklamaya çalıştık.

**Anahtar Kelimeler:** Klival subdural hematom, düşük molekül ağırlıklı heparin, travma, mekanizma

## INTRODUCTION

Traumatic retroclival hematoma is a rare entity and usually occurs in the pediatric population<sup>(4,6)</sup>. Most of these reported cases are extradural, and developed after motor vehicle trauma or falls from height<sup>(3,6,8-12,14-18)</sup>. To our knowledge, late onset low-molecular-weight heparin (LMWH) induced traumatic clival subdural hematoma (SDH) associated with

isolated abducens nerve palsy has not been published in the English literature.

We present a 12 year old multiple trauma patient who was administered with LMWH on admission and developed SDH after 6 days.

## CASE PRESENTATION

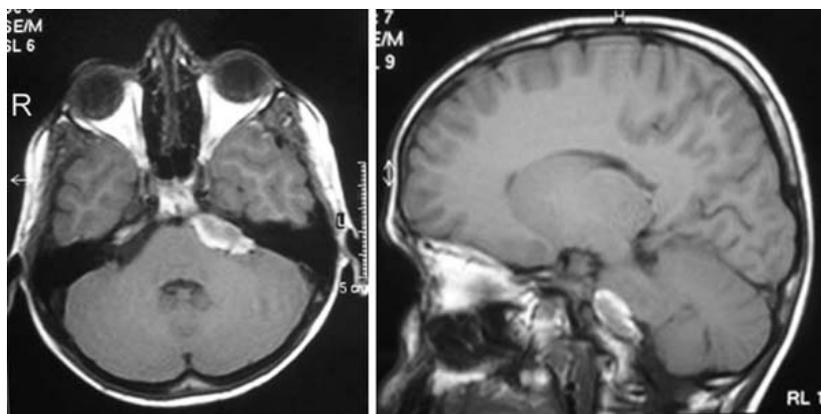
A 12-year-old boy fell from the second-story window of an apartment building. On admission, his Glasgow Coma Scale (GCS) was 15. Fractures of the right tibia-

fibula, left calcaneus and right distal radius were detected. Physical examination was unremarkable besides fractures. Brain and cervical computed tomography (CT) revealed no abnormality (Figure 1A). The patient was hospitalized by the department of orthopedy and prophylactic LMWH (enoxaparin 0.4mg, twice a day subcutaneously) was applied by the trauma surgeons, with weight-adjusted dosing without laboratory monitoring, to decrease the risk of venous thrombosis in multitrauma patient. On the posttraumatic 6th day, the patient complained of sudden headache, nausea and vomiting. His physical examination revealed isolated abducens nerve palsy on the left eye. CT of the brain showed clival subdural hematoma extending to the left cerebellopontine angle (Figure 1B). MR imaging study and

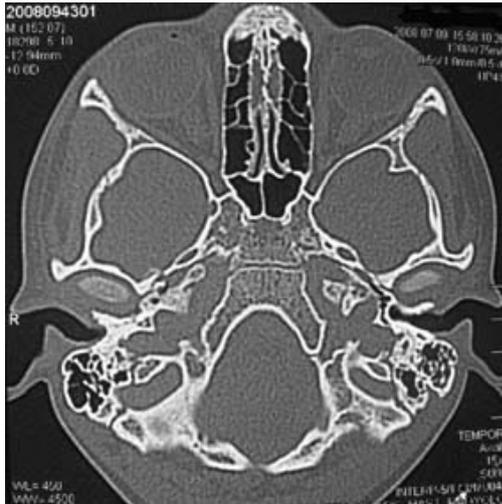
subsequent CT angiogram of the brain demonstrated no underlying vascular abnormality (Figure 2). A fine-cut CT scan through the skull base (Figure 3) and cervical spine and MR imaging of the spine did not reveal any evidence of bone and ligamentous injury. Plain flexion-extension radiographs of the cervical spine showed no abnormal movement at the occipitocervical junction or in the upper spine. Laboratory analysis revealed a normal platelet count and coagulation profile. The patient was reassured and treated conservatively. Further CT imaging of the brain on the 7.day after first determination of bleeding showed resolution of the hematoma. (Figure 4). Subsequent follow-up in forthcoming 6 months, he was found to be completely asymptomatic.



**Figure 1:** Normal computed tomography of an 12-year-old boy on admission to the Emergency room after falling from height (A), whereas posttraumatic 6.day computed tomography of the same patient demonstrating a hyperdense clival SDH extending to the left cerebellopontine angle (B).



**Figure 2:** Axial (left) and parasagittal (right) T1-weighted MR imaging sequences of the same patient showing hyperintense clival SDH extending to left cerebellopontine angle.



**Figure 3:** Axial CT scan of the bone showing normal radiological findings.



**Figure 4:** Control computed tomography of the patient on the 7.day after first determination of bleeding showed complete resolution of the hematoma.

## DISCUSSION

Traumatic clival hematomas are unusual and most reported cases involved pediatric patients whose hematomas occurred after motor vehicle trauma or fall from height<sup>(4,6)</sup>. Nevertheless, traumatic clival SDHs are very rare in case and constitutes approximately 0,3% of all acute SDHs<sup>(4)</sup>. The relative rarity in which SDHs occur in these locations can result in delayed or missed diagnoses. LMWH induced delayed traumatic clival SDH has not been published in the English literature, yet. To our knowledge, clival SDH due to trauma has been reported in a pediatric<sup>(1)</sup> and in two adult patients<sup>(4)</sup>, in which one of these described adult patients was in context of hemophilia<sup>(13)</sup>. The exact mechanism of injury is not well understood due to the rarity of such cases (Table 1). In hemophiliacs, frequency of bleeding episodes in the untreated individual are related to the factor VIII clotting activity<sup>(2)</sup>. LMWH mechanism of action is increasing the inhibitory effect of antithrombin on the serine proteases thrombin and factor Xa with greatest effect upon factor Xa<sup>(7)</sup>. So there is no similarity according to bleeding mechanisms between patients with hemophilia and LMWH intake. In our

case, LMWH was applied by the trauma surgeons for prophylaxis of venous thrombosis.

As a result of immaturity of the craniovertebral junction in childhood, in the case of clival hematomas, injury may result from clivus fracture or ligamentous disruption<sup>(6)</sup>. Although, spontaneous posterior fossa SDHs due to arteriovenous malformation originating from the brain or spine have been described in the literature<sup>(5)</sup>, brain and spine MR imaging and CT angiogram study disclosed the presence of an underlying vascular abnormality<sup>(5)</sup>. The pathophysiological mechanisms of epidural clival hematomas in pediatric cases have been comprehensively reviewed and detailed by Guillaume and Menezes<sup>(6)</sup>. The biomechanical differences in the pediatric spine, which are increasing mobility and elasticity of the craniovertebral junction and upper spine, result with ligamentous injury in response to trauma<sup>(6)</sup>. However recent report of Tubbs et al, which is the largest series of pediatric retroclival epidural hematoma conclude that atlanto-occipital dislocation should be considered in all cases<sup>(17)</sup>. Retroclival epidural hematomas are anatomically limited from the

midportion of the clivus to the middle of the body of the axis because of the tectorial membrane boundaries<sup>(6)</sup>, whereas clival SDHs can be spread and effect a larger area even it may communicate with the spinal subdural space<sup>(4)</sup>.Clival SDHs in traumatic origin has been seen generally in acute onset and the patients have had focal neurological deficit, such as bilateral or unilateral palsy of the abducens nerve<sup>(6)</sup>. Our case, is an subacute onset of clival SDH in posttraumatic 6. day. However unilateral abducens nerve palsy was absent after trauma and developed 6 days after trauma with a clinical deterioration. Due to the ambiguity of the clinical and radiological presentation, the case is

unique and has to increase attentions on pediatric traumatic patients with LMWH therapy. To our knowledge, we could not find any reported case of LMWH induced traumatic clival SDH in the English literature. Therefore it is the only one case reported - the exact mechanism of this entity is not well understood. The probable mechanism for this case may be, LMWH induce bleeding tendency in a hyperflexion–hyperextension injury of the ligamentous structures of the craniovertebral junction, without any radiological findings.

**Table 1** Clinical features and possible mechanisms of reported cases of traumatic clival SDHs

Case –year	Age	Mechanism	Presentation time	Neurological examination	Treatment & Outcome
Myers et al- 1995	17	Probable consequence of the abundant venous plexus at the clivus, and propensitiy for hemorrhage with hemophilia A	Posttraumatic 5.day	GCS: 3	Brain death 12 hours after admission
Ahn et al – 2005	4	Redistribution of clival SDH to the spinal sudural spaces in evolution of these lesions	After trauma	GCS:10, four fifts strength in the left arm and leg	Conservatively, Discharged 1 week after admission in a stable condition
Casey et al – 2009	18	Stable right atlantooccipital joint injury	After trauma	GCS:13	Conservatively, Follow-up 2 months later, completely asytmomatic
Our Case	12	Mild hyperflexion-hyperextension injury of the craniovertebral junction or upper spine and propensity for hemorrhage with LMWH	Posttraumatic 6.day	GCS:15, late onset left abducens nerve palsy	Conservatively, Follow-up 2 months later, completely asytmomatic

## CONCLUSION

Rarity of traumatic clival SDH in pediatric age makes the exact pathophysiology unknown. We hypothesize that, similar to the mechanism which is related to epidural clival hematomas a history of a mild hyperflexion-hyperextension injury can even be a cause of clival SDHs in association with LMWH. Clival SDHs generally do not require surgical intervention. Further imaging should be performed to exclude late onset clival SDHs and clinical suspicion facilitate the management of this entity especially in pediatric age. Use of prophylactic LMWH in patients with multiple trauma should be carefully observed. This case is an evidence of prophylactic anticoagulation in multiple trauma which requires an attentive clinical and neurological follow-up. In condition of neurological deterioration this rare entity should be kept in mind, even in pediatric patients.

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**Received by:** 02 March 2011

**Revised by:** 13 July 2011

**Accepted:** 10 April 2012

## The Online Journal of Neurological Sciences (Turkish) 1984-2012

This e-journal is run by Ege University  
Faculty of Medicine,

Dept. of Neurological Surgery, Bornova,  
Izmir-35100TR

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Journal of Neurological Sciences (Turkish)

Abbr: J. Neurol. Sci.[Turk]

ISSNe 1302-1664

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