CASE REPORT

EXTRADURAL HEMATOMA RESULTING FROM MIGRATION OF SUBGALEAL HEMATOMA

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ABSTRACT

Extradural hematomas are collection of blood between the dura and skull bone. These hematomas are common complication of head injury. Spontaneous resolution of extradural hematoma due to migration into the subgaleal space has been reported. We are reporting an unusual case of a 19-year-old male presenting with an extradural hematoma, which was formed because of migration of a subgaleal hematoma through the skull fracture. Our case emphasized the importance of assessment of patients presenting with subgaleal hematoma to prevent them from any neurological injury.

Keywords: Subgaleal Hematoma; Extradural Hematoma; Tight Bandage.

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INTRODUCTION

Extradural hematomas are collection of blood between the dura and skull bone. These hematomas are common complication of head injury. Most extradural hematomas are caused by bleeding from a meningeal artery, mostly the middle meningeal artery but some are also caused by bleeding from dural venous sinuses. Following skull fracture, extradural hematomas are known to achieve their full size very rapidly¹. However, some patients also reported late development of extradural hematomas and expansion of a pre-existing hematoma^{2,3}. latrogenic extradural hematomas are a known complication of cranial surgery.

Large symptomatic hematomas are considered a serious neurosurgical emergency and require urgent evacuation^{4,5} but small hematomas, which are asymptomatic, can be managed conservatively⁶. Many cases of spontaneous resolution of extradural hematomas have been reported in literature^{4,7,8} a common proposed mechanism of which is transmigration of the extradural hematoma to the subgaleal space through a skull fracture. This potential space extends from the orbital margins anteriorly to the nuchal lines posteriorly and laterally to the temporal fascia¹⁰. However, no case of migration of subgaleal hematoma to the extradural space has been reported in literature. We report an uncommon case of an extradural hematoma, which was formed because of migration of a

subgaleal hematoma through the skull fracture after application of tight head bandage.

CASE REPORT

A 19 years old male patient presented to the ER, 48 hours after having a motorcycle accident. He had come to the ER as he had developed massive subgaleal swelling. On admission, he was alert and oriented and had no focal neurological deficit. His hemodynamic status was stable. He had large scalp swelling bilaterally with bilateral raccoon eyes. His CT scan of head showed no evidence of brain injury and no intracranial hematoma. However, he had a large subgaleal hematoma (Figure 1). He also had fracture of the skull at right temporal area. The paramedic in the ER applied a tight head bandage and he was admitted for neurological observation. Three hours after admission, he started to become drowsy and he developed left hemiparesis. A CT scan was repeated and showed a large temporo-parietal extradural hematoma with midline shift. There was a marked reduction in the size of the subgaleal hematoma (Figure 2). An urgent craniotomy was performed and the extradural hematoma was evacuated. At surgery, a parietal fracture was identified (Figure 3); there was a small subgaleal hematoma but there was no discreet source of hemorrhage from the dura and the extradural hematoma was found to be liquefied. Following surgery, the patient made an uneventful recovery.

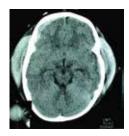


Figure 1: CT Brain Shows Large Bilateral Subgaleal Hematomas But No Extradural Hematoma

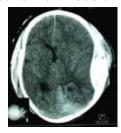


Figure 2: CT Brain Showing Markedly Reduced Subgaleal Hematomas Along With Newly Developed Extradural Hematoma.



Figure 3: CT scan (Bone Window) Showing Skull Fracture.

DISCUSSION

Post-traumatic extradural hematomas are known to be associated with skull fractures and in most cases result from bleeding from meningeal artery or dural sinus tear. As these hematomas are complications of skull fracture and are not due to brain injury they carry a good prognosis and immediate evacuation of symptomatic extradural hematomas is mandatory.

Subgaleal hematomas commonly occur in neonates, following forceps delivery or vacuum extraction¹¹. These hematomas can be of major clinical significance as the total blood volume in this age is small. These subgaleal hematomas are not associated with skull fractures and often resolve spontaneously. Subgaleal hematomas also occur as a common complication of head injury and may or may not be associated with skull fracture. Cases of spontaneous resolution of extradural hematomas have been reported and these are probably due to transmigration of hematoma from extradural to subgaleal space¹². Usually this is seen in small children.

In our patient, at the time of first presentation even after 48 hours of injury there was no extradural

hematoma. Following application of tight bandage of the head by the paramedic, this hematoma seems to have appeared three hours after injury. Slow accumulation of serosanguinous fluid due to transfer through the skull fracture resulted in the accumulation of this hematoma. By the time the surgery was performed, the subgaleal hematoma had reduced in size. In addition, there was no discreet source of hemorrhage from the dura.

In the context of spontaneous resolution of extradural hematomas a potential communication between intracranial and epicranial hematomas through the fracture has been suggested. While reporting a case of a 17 months old child whose extradural hematoma resolved spontaneously, Malek et al. postulated that extradural hematomas that evolved rapidly in the presence of normal intracranial pressure occur as a result of increased pressure in the subgaleal compartment with migration of fluid from subgaleal to extradural space¹³. They suggested that once the intracranial pressure increased the fluid shifted back from extradural to subgaleal space resulting in spontaneous resolution of the hematoma¹⁴.

Our case seems to support this hypothesis. It appears that although our patient had a skull fracture and a subgaleal hematoma, initially his intracranial pressure was normal. With the application of tight cranial bandage, the fluid shifted from the subgaleal to the extradural space resulting in the hematoma. As this clot had become symptomatic, it had to be evacuated. To the best of our knowledge, no report of formation of extradural hematoma due to transfer of a subgaleal hematoma has been reported in adults in literature. This hematoma did not resolve spontaneously and needed surgical evacuation as it had become symptomatic.

CONCLUSION

In conclusion, we suggest that patients who present with large subgaleal hematomas and associated skull fracture should not be managed with a tight head bandage unless otherwise indicated. In such case, these patients should be kept under observation as potential candidates for developing extradural hematomas.

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