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Case Report

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Rapid Spontaneous Resolution of Large Acute Extra Dural Hematoma

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ABSTRACT

Acute extra dural hematoma constitutes for approximately 1.5% of case treated for head trauma. Early diagnosis and rapid evacuation is needed for most of the large hematomas. Only few cases are reported which showed rapid spontaneous resolution. Herein, we report a case of spontaneous resolution of large EDH within 12 hours in 24 years old gentleman and possible mechanisms are discussed.

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Introduction

Acute extradural hematoma is one of the most critical neurosurgical emergencies which can be proved fatal if not evacuated urgently. They accounts for approximately 1.5% of cases treated for head injury [1, 2]. Asymptomatic patients with small EDH can be managed conservatively while most of the large, symptomatic EDH warrants early surgical evacuation because of rapid deterioration [1-3]. Rapid spontaneous resolution of large EDH is rare phenomenon and only few cases are reported. Here, we report a case of 24 years old male, whose repeat NCCT head showed decompression of EDH within 12 hours period. Possible mechanisms of spontaneous resolution are discussed here.

Case

A 24-year-old gentleman was presented in our emergency department at 5:00 A.M. with a history of road traffic accident at 7:00 P.M. of yesterday evening. He had history of brief loss of consciousness and two episodes of vomiting. He was first taken to nearby private hospital where NCCT head was done within first four hours of injury. The NCCT head was suggestive of large extra dural hematoma of left frontal region (max thickness-2.5 cm) with mass effect and midline shift of approximately 6mm and overlying communited fracture of left frontal bone. Urgent evacuation of hematoma was advised there but relatives of patients were not agreed upon and they brought their patient to our hospital.

On arrival in our hospital, patient was conscious and oriented. His Glasgow coma scale was E4V5M6. Vitals were stable (pulse rate-78/minute and blood pressure-110/70 mm Hg.). Both pupils were normal in size and reacting equally to light. There were no evidence of any external wound over scalp. Scalp swelling was present in left fronto temporal region. In view of early scan and stable condition of patient, a repeat NCCT head was done. This showed thin extra axial hematoma (volume-8 ml.) without any midline shift with overlying communited fracture of left frontal bone and increase in scalp hematoma compared to previous scan. His blood investigations including hemogram and coagulation profile were normal. Patient was managed conservatively.

Discussion

Several possible mechanisms have been proposed for the spontaneous resolution of an EDH. Usually, spontaneous resolution of hematoma occurs over a period of several weeks [1,4,5]. It is very rare for an EDH to resolve within hours [1,3,6,7]. Mechanism of resolution for chronic EDH is different from acute EDH. Pang et.al has proposed formation of fibrovascular membrane lining the dural side of the clot. It acts as an absorbing structure. The angioblasts form sinusoids which gradually connect with the dural vessels, so blood and blood products can return to the systemic circulation via the permeable membrane of these sinusoids. This mechanism could be comparable to that of chronic subdural hematomas. This process is particularly thought to be the mechanism in chronic EDH resolution because a time period is needed for its formation [1,2,4,6]. In the literature, various mechanisms responsible for early spontaneous resolution of EDH have been reported.

The presence of skull fracture can allow transfer of collection between the epidural region and subgaleal space. It can relieve compression caused by the hematoma [1-3,6,7]. Raised intracranial pressure creates a pressure gradient between the EDH and extracranial soft tissues, such that the EDH is forced out of the epidural space through the fracture [1,3,5]. Association of skull fracture with rapid resolution of acute sub dural hematoma has also been reported. Jasmit singh et.al reported that linear fracture may facilitates the redistribution of the acute subdural hematoma and improvement of the brain shift [8]. Dolgun et.al has proposed liquefaction of epidural clot by cerebrospinal fluid through the small dural tear [6]. Malek et al. reported that crush injury to epicranial tissues can be followed by transient elevation in interstitial fluid pressure, and in the presence of skull fracture, the elevated interstitial pressure can decompress into the epidural space. When interstitial subgaleal pressure decreases, the fluid

leaked back [9]. In our case, rapid resolution occurs because of possible pressure gradient between intracranial and extracranial compartment and communication between epidural space and subgaleal space through the communited fracture.

Conclusion

Management of EDH depends upon size of hematoma and neurological status of the patient. Smaller lesion can be managed non operatively with careful monitoring. Large EDH may need urgent evacuation as they may deteriorate rapidly and rapid spontaneous resolution is very rare. To conclude, small subgroup of patients, who present late with initial early scan and intact neurologically, should be scanned again even if the early scan suggests operable lesion, to look for the spontaneous decompression.

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