Bilateral Asymptomatic Subacute and Chronic Epidural Haematoma of the Posterior Cranial Fossa

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Abstract: Post traumatic bilateral posterior fossa epidural haematomas (PFEDH) are relatively rare.
A case of bilateral symmetrical PFEDH one in the subacute and the other in the chronic phase is presented. The patient was managed conservatively.
The case is discussed with the pertinent literature.

Key words: Epidural haematoma, subacute, chronic, bilateral, posterior fossa.

INTRODUCTION

Posterior fossa epidural haematoma (PFEDH) is an important complication of craniocerebral trauma (16) and is relatively rare, the incidence ranging between 1.2% and 12.9% of all epidural haematomas (EDHs) (4, 17, 23).

Most PFEDHs are unilateral (4, 6, 21), but bilateral PFEDHs have been reported (1, 3, 20). Up to 1991, 62 cases of asymmetrical PFEDHs have been reported in the literature, of which 47 cases were subacute and 15 chronic (7-9, 13, 21). Only Saeki et al. presented a case of bilateral subacute PFEDH (20). We believe that, ours is the first report of a case presenting with bilateral symmetrical PFEDH; one in the subacute and the other in the chronic phase.

CASE REPORT

A16-year-old boy was hit by a car on 10 October, 1994. He was kept in a local hospital for 24 hours and had no complaints except for headache. A fortnight after the accident, on October 24, 1994 he attended a check-up at our hospital. He had no complaints and neurological and physical examinations were normal. Direct skull x-rays revealed an occipital linear fracture crossing the midline. Computerised tomography (CT) of the brain revealed a bilateral EDH of the posterior fossa; but the EDHs were in different phases, that on the right side was subacute (relatively more hyperdense than the other) and that on the left side chronic. The fourth ventricle was open and no hydrocephalus was detected (Fig 1). The patient was hospitalised. CBC, blood chemistry and brainstem auditory evoked responses (BAER) were within normal limits. A CT scan of brain taken on November 4, 1994 showed complete resolution of the chronic EDH on the left side. On the right side a chronic EDH was detected (Fig 2). The patient was discharged in good condition. In a third CT scan on December 5, 1994; complete resolution of both of the EDHs was observed (Fig 3).

DISCUSSION

Most EDHs reported in the literature are acute cases (1, 2, 5, 11, 12, 17-19) and the injury site is mostly the anteroposterior regions of the brain (2). Most bilateral EDHs are symmetrical, crossing the midline in the frontal, parietal or occipital regions. They occur as a result of venous bleeding due to injury of the saggital sinus or diploic vessels, which may also cause

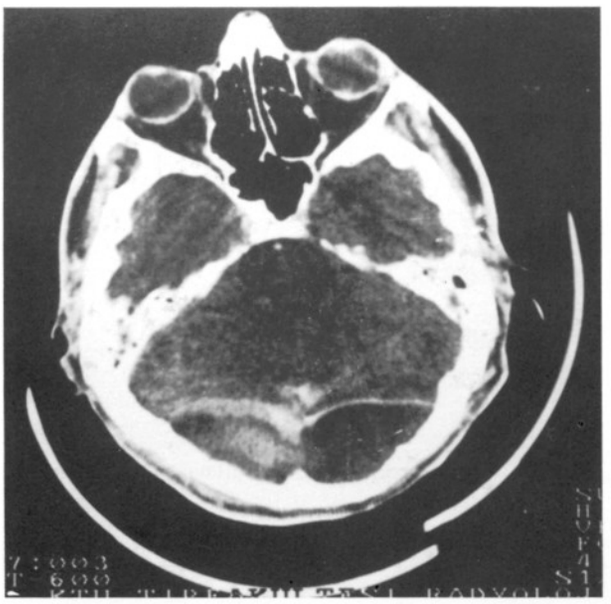

Fig. 1: Initial CT scan showing a bilateral PFEDH. The EDH on the right relatively more hyperdense than that on the left.

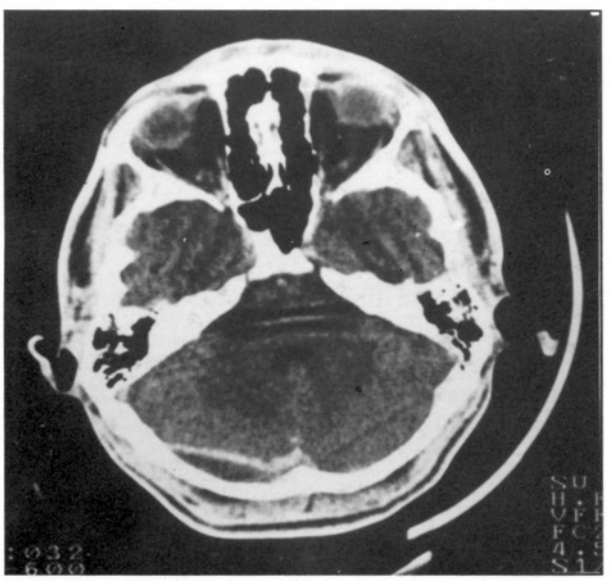

Fig. 2: Second CT scan showing complete resolution of the EDH on the left side and the EDH on the right is smaller and in the chronic phase.

delayed or subacute EDH (2). In the literature limited cases of subacute EDH have been reported (20) and chronic PFEDHs are more rare (8, 15, 22).

The mechanism of the two phases of the PFEDH in the presented case may be explained as: 1) Linear fracture crossing the dural sinus may injury it and the first EDH may occur. By the mass effect of the first EDH, the dural sinus may dissect and delayed EDH (or the second EDH) may occur; 2) There can

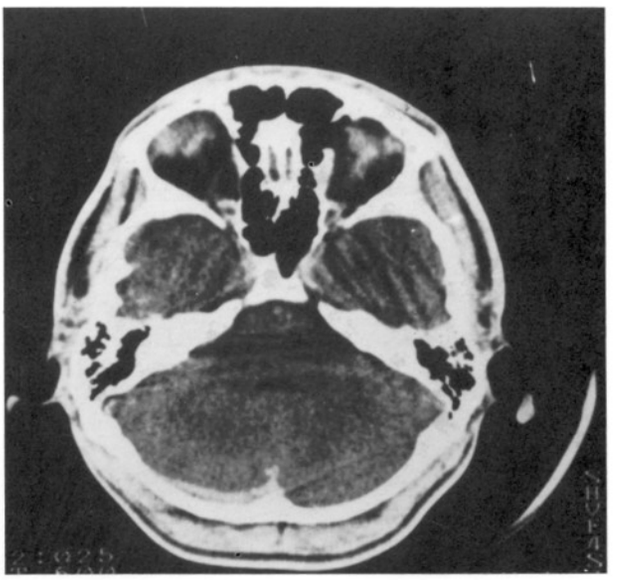

Fig. 3: Third CT scan showing complete resolution of both EDHs.

be multiple injury sites in the injured dural sinus and one may stop bleeding and the other continue to bleed.

Our patient was asymptomatic; which is also interesting for a PFEDH of this size. This can be explained by the venous origin of the bleeding or relatively large cisterns of the posterior fossa.

In the management of patients CT-based indications for surgery are: 1) The maximum thickness of the EDH is more than 15mm. 2) Poorly visualised posterior fossa cisterns. 3) Marked deformity and/or displacement of the fourth ventricle. 4) The haematoma extending to the supratentorial region and severely compressing the brain (3, 21). These criteria are very important especially in acute PFEDHs of arterial origin. Subacute cases may exhibit increased intracranial pressure and/or cerebellar, brainstem, long tract or cranial nerve signs (8, 14). We also believe that the development of hydrocephalus is a critical factor for chronic PFEDHs managed conservatively (10).

The presence of EDH can not be excluded by the absence of neurological deficits in posterior fossa fractures. The probability of a future PFEDH must be always kept in mind Therefore, linear fractures in the posterior fossa can be considered as a possible key of the PFEDH and need close observation.

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