

SHORT REPORT

Bilateral frontal extradural haematomas caused by rupture of the superior sagittal sinus: case report

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Abstract

A 26-year-old male sustained simultaneous massive bilateral frontal extradural haematomas following a head injury as a result of a large tear of the superior sagittal sinus, without fracturing of the skull vault.

Key words: Bilateral extradural haematomas, CT, head injury, seizure, superior sagittal sinus.

Introduction

Although bilateral extradural haematomas (EDHs) are uncommon, there are several reports in the literature. 1-12 When compared with unilateral haematomas, it has been reported that they may develop slowly, often after a traumatic force orientated in the anteroposterior direction, have fewer overlying fractures and commonly contain venous blood.1

A case of massive, acute bilateral frontal extradural haematomas due to rupture of the superior sagittal sinus in the absence of a skull fracture is reported. The sources of haemorrhage in bilateral extradural haematomas are discussed.

Case report

A 26-year-old man collapsed at home, striking his head. He immediately had a tonic-clonic seizure, lasting 5 min. He awoke, complained of a headache and vomited. Apart from an episode of acute alcohol intoxication a year earlier there was no prior history of epilepsy or other illness. On arrival at hospital he was alert, orientated and obeying commands. The forehead was bruised. Blood and coagulation tests, ECG and chest radiographs were normal, apart from some mildly raised liver function test levels and a new rib fracture. A skull radiograph was not taken. Soon after admission he became confused. Over the next 12 h, he had a further six fits, treated with a total of 30 mg of diazepam rectally. During this time he deteriorated until both pupils became fixed and dilated. CT of the brain (Fig. 1) showed massive



FIG. 1. Axial CT scan showing very large bilateral frontal extradural haematomas.

bifrontal extradural haematomas.

On referral to our neurosurgical department he was immediately intubated and ventilated, and given 300 ml of 20% mannitol intravenously. A bicoronal skin flap was turned and the extradural haematomas

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were evacuated via separate bilateral frontal craniotomies. No fractures were found, but active bleeding was seen from the anterior third of the sagittal sinus into the extradural space. This was controlled by hitching the surrounding dura up against the remaining midline skull vault. The dura had to be opened in order to do this.

Postoperative serial CT showed gradual reexpansion of both cerebral hemispheres, but with some atrophy of both frontal lobes. Three months postoperatively, he remained completely dependent on nursing care with a tracheostomy and feeding gastrostromy. He obeyed simple commands, but had bilateral complete oculomotor nerve palsies and a severe spastic tetraparesis.

Discussion

In an attempt to identify the source of haemorrhage in cases of bilateral EDHs, the literature of the last 30 years was reviewed. There appear to be two separate groups: patients with bilateral separate EDHs over the frontal/temporal/parietal lobes²⁻⁹ and those with single midline EDHs spreading bilaterally. 10,11 In the first (lateral) group, separation of dura from bone independently on both sides of the head causes a mixture of arterial and venous bleeding into the extradural spaces bilaterally, either simultaneously or sequentially. Barlow suggested that if both clots were largely arterial, they would be present soon after injury, but if one or both haematomas are delayed, then they are venous in origin.

In the second and rarer (midline) group, of which our case is an example, the bleeding source would appear to be more exclusively venous, from a rupture of the sagittal sinus itself rather than from smaller extradural vessels nearby. In the two other cases in this group, Agbi's patient¹⁰ was seen to have bleeding from the sagittal sinus just above the torcula causing a midline EDH extending from vertex to foramen magnum, but in Kissel's case¹¹ the source for a smaller midline bifrontal EDH was not identified because of initial conservative management.

In order to initiate a midline EDH a tear of the

strong dural attachment over the sagittal sinus is needed. Ford & McLaurin, 12 following experimentation in anaesthetized dogs, doubted that venous pressure is sufficient to cause a unilateral extradural haematoma, except when there is decreased intracranial pressure secondary to cerebrospinal fluid leakage. However, they did find that the force required was less if it was pulsatile rather than static.

In our patient, the initial force was in an anteroposterior direction. This, we suspect detached the sinus from bone, creating a defect in the sinus. This defect subsequently may have acted as a one-way valve. The vomiting during the lucid interval, as well as the sequence of seizures would both have increased the central venous pressure transmitted to the sinus in a pulsatile fashion. This combination, we suggest, was sufficient to cause enlargement of the extradural haematomas to a substantial size.

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