Delayed Diagnosis of Acute Extradural Haematoma

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ABSTRACT

An 18-year old man presented with persistent left frontal headache, ptosis, blurring of vision and early morning vomiting 3 weeks after involvement in a road traffic accident. A computed tomography scan of the brain showed left frontal extradural haematoma. The patient recovered following craniotomy and evacuation of the haematoma.

KEY WORDS: Extradural haematoma, delayed diagnosis

Introduction

The risk of developing an intracranial haematoma following head injury with loss of consciousness is 1 in 29.¹ Extradural haemorrhage may be evident in 15% of cases and is accompanied by skull fracture in 90%.² Extradural haematoma usually presents acutely following trauma.

Case Report

An 18-year old man presented with persistent left frontal headache 3 weeks after involvement in a road traffic accident (RTA). There was loss of consciousness for about 30 minutes and he sustained bruises of the frontal scalp and lacerations on the upper and lower limbs. There was no convulsion or leakage of cerebrospinal fluid from the nostrils or ears. The patient reached a hospital conscious but lapsed into unconsciousness after 6 hours. He was resuscitated and the lacerations sutured. He regained consciousness after 3 days but continued to complain of frontal headache, which was treated with analgesics and he was discharged home after 5 days in hospital. Two weeks later, the patient developed early morning vomiting and blurring of vision in the left eye, leading to referral to the neurosurgical unit.

Physical examination showed no pallor or fever. The pulse rate was 80 per minute and blood pressure 110/70 mmHg. Chest and abdominal examinations were normal. The Glasgow coma score was 15. There was ptosis of
the left eyelid and weakness of the right lateral rectus muscle. The right eye and associated muscles were normal. The pupils were equal and both reacted normally to light. Fundoscopy showed bilateral papilloedema. Power and sensations were normal in all limbs. A skull X-ray showed a linear fracture of the frontal bone.

Computed tomography (CT) scan of the brain showed the fracture and a left frontal, lentiform, hyperdense extraparenchymal lesion displacing the frontal lobe, frontal horn of the lateral ventricle and a midline shift (Figure 1). There was brain oedema. These features were consistent with those of an extradural haematoma. Complete blood count and serum electrolytes and urea were normal.

At craniotomy, the findings were; 4cm linear fracture of the left frontal bone; partially liquefied extradural haematoma (150mls) and intact dura matter. There was no active meningeal bleeding. The haematoma was evacuated and there was visible intraoperative expansion of the frontal lobe. Postoperatively, the headache, early morning vomiting and ptosis subsided over 5 days. The patient was discharged home on the 7th postoperative and has remained well a 7 months of follow up.

Discussion

In this environment, due to lack of awareness and financial constraints, only few head injured patients, especially those with intracranial collection reach a neurosurgical centre, and then, frequently too late to benefit from any surgical intervention.

The operative outcome for intracranial haematoma is directly related to early diagnosis and evacuation and it is important that these patients are referred early. Extradural haematoma usually presents within 6 – 12 hours of injury and is located in the temporal region in about 70% of cases and the frontal region in 10%. The classical history of deterioration in level of consciousness after a lucid interval occurs in about 50% of children but less than one third in adults. The patient in this report had a lucid interval after an initial loss of consciousness but this was probably disregarding at the initial assessment. However, despite the extradural haematoma, the patient was able to regain consciousness and remain so for 3 weeks before referral; this may be due the location of the haematoma in the frontal region, so that the pressure from the haematoma is not transmitted to the directly brain stem. Extradural haematoma around the temporal, parietal or posterior fossa region are rapidly fatal due to the direct compression of the brain stem and tentorial herniation. It may well be that the haematoma in this patient was venous in origin, and this is associated slow progression of symptoms.

It is important that these patients are monitored closely, including CT scan of the brain, which may need to be repeated if symptoms persist. To achieve a good outcome, early referral of patients who sustain head injury associated with loss of consciousness or persistent symptoms is of optimum importance.

References
