

## CASE REPORTS

## Silent orbitocranial penetration by a pencil

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**Abstract**

**Objective**—To emphasise the value of computed tomography even in the absence of symptoms in a case of penetrating injury of the upper eyelid.

**Methods**—Case report.

**Results**—Although clinically asymptomatic, penetration of upper eyelid was associated with intracranial penetration that left a track in the brain parenchyma.

**Conclusions**—Computed tomography of orbit and brain is an important investigation, even in seemingly trivial eyelid injury, to reveal the full extent of the damage.

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Keywords: eyelid; orbitocranial; computed tomography

Penetrating orbitocranial injury can cause serious consequences and is potentially fatal. Penetration is usually caused by long, thin, and relatively hard objects. These injuries are rare and may need neurosurgical intervention.

**Case report**

A 5 year old boy was brought to the accident and emergency department with an eyelid laceration. He had fallen onto a pencil being carried in his hand. Ophthalmic examination showed presence of a laceration on the right upper eyelid with no evidence of injury to the eyeball. Exploration before suturing the wound revealed fragments of the lead of the pencil. A radio-opaque shadow seen on radiography before exploration was persistent after exploration (fig 1). Due to the possibility of this being part of the roof of the orbit computed tomography was requested. This showed a defect in the roof of the orbit and the shadow was a bony fragment. There was also a track leading from the roof of the orbit and terminating just lateral to the lateral ventricle (fig 2). The patient was observed and followed up by the neurosurgeons. At 10 months there were no ocular or neurological sequelae.

**Discussion**

Intracranial penetration can cause potentially fatal injuries. Neurosurgical intervention may be required to manage intracranial haematoma, abscess, and to remove foreign bodies. A delay in recognition could cause serious consequences.<sup>1</sup> Loss of sight and ophthalmoplegia have been reported after penetration by a pencil.<sup>2</sup> Magnetic resonance imaging (MRI) should be used as an adjunct if a wooden foreign body is suspected.<sup>3</sup> The main issue is one of recognition of the actual extent of the injury so that appropriate management can be planned. Our case demonstrates the fact that seemingly trivial injury of the eyelid can be associated with damage to the intracranial



Figure 1 The defect in the roof of orbit and the corresponding bony fragment is shown.

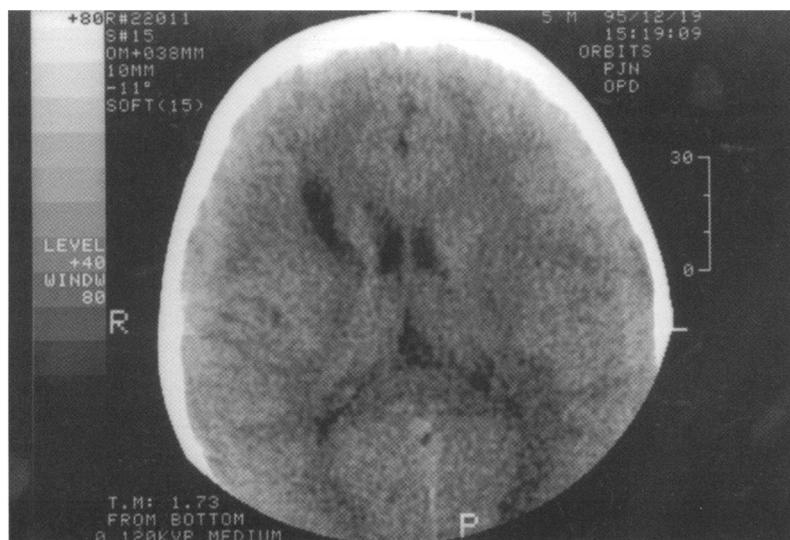


Figure 2 The low density track in the brain terminating near the right lateral ventricle is shown.

tissue and still remain asymptomatic. One should suspect intracranial penetration in all penetrating orbital injury and evaluate the extent of damage with computed tomography and MRI if a wooden foreign body is suspected. Plain radiographs have limited value.

- 1 Mutlukan E, Fleck BW, Cullen JF, *et al.* Case of penetrating orbitocranial injury caused by wood. *Br J Ophthalmol* 1991;75:374-6.
- 2 Oguz M, Aksungur EH, Atilla E, *et al.* Orbitocranial penetration of a pencil: extraction under CT control. *Eur J Radiol* 1993;17:85-7.
- 3 Specht CS, Varga JH, Jalali MM, *et al.* Orbitocranial wooden foreign body diagnosed by magnetic resonance imaging. Dry wood can be isodense with air and orbital fat by computed tomography. *Surv Ophthalmol* 1992;36:341-4.

## Neurogenic pulmonary oedema

Gary W Kerr

### Abstract

**Neurogenic pulmonary oedema is a relatively rare but significant complication of head injury. A case is described and the presentation, pathophysiology, and management are discussed.**

(*J Accid Emerg Med* 1998;15:275-277)

Keywords: neurogenic pulmonary oedema; head injury

### Case report

A 21 year old man was brought by ambulance to the accident and emergency department after having been assaulted while out drinking and left lying unconscious. He had a Glasgow coma score of 8 and soft tissue evidence of head trauma with abrasions and swelling over the right parietal area. The airway was clear with no blood, secretions, or vomit and he was tolerating a Guedal airway. There was no evidence of trauma anywhere else on his body. Examination was otherwise unremarkable apart from the presence of bilateral crackles on auscultation of the lung fields. Despite high flow oxygen via a reservoir mask his saturation on pulse oximetry was 91%.

The patient was intubated and ventilated but the saturation remained at 91% with an arterial oxygen tension of 8.5 kPa and a carbon dioxide tension of 5.1 kPa. A portable chest *x* ray was performed and this showed perihilar alveolar shadowing later reported as being consistent with pulmonary oedema. There was prolonged difficulty in oxygenating the patient, requiring a large tidal volume, positive end expiratory pressure, and frequent suction of frothy blood-stained fluid via the endotracheal tube. The oxygen saturation gradually improved and the patient was taken to the computer tomography room.

A computed tomogram was performed which showed multiple high attenuation areas in the grey white matter interface of both frontal lobes with marked soft tissue swelling and a little blood in the subarachnoid space. These appearances were thought to be consistent with diffuse axonal injury.

After the tomogram there was a sudden deterioration with hypoxia, hypotension, and an increased central venous pressure. The patient responded to vigorous ventilation and

adrenaline aliquots to maintain the blood pressure. As the chest *x* ray was thought possibly to show a globular heart shadow, an echocardiogram was performed. This revealed poor left ventricular function with marked anteroapical hypokinesia but no pericardial effusion.

He was transferred to the intensive care unit where he was treated with frusemide, dobutamine, adrenaline, and ventilation with positive end expiratory pressure. During the next 24 hours he had a further hypoxic episode with increased pulmonary artery wedge pressure and oedema. This responded to diuretics, dobutamine, and glyceryl trinitrate. Subsequently he was cardiovascularly stable with no further episodes of pulmonary oedema and he was extubated before being transferred from the intensive care unit eight days after admission. His neurological recovery was minimal and he was transferred to a long term rehabilitation unit.

### Discussion

When a patient with a head injury presents with the clinical features of pulmonary oedema there may not be an obvious explanation such as aspiration or excessive fluid administration. Neurogenic pulmonary oedema is a relatively uncommon complication of acute cerebral insults of various types and has been previously recognised as being the sole pathological mechanism in certain cases.<sup>1</sup> Most initial reports associated neurogenic pulmonary oedema as being precipitated by head injuries,<sup>2-4</sup> a finding emphasised by a report of 56 casualties from the Vietnam war with major head trauma that describes evidence of pulmonary oedema in 17 patients.<sup>5</sup> Isolated head injury is still the commonest association, but a variety of other precipitants have been reported including epilepsy,<sup>6-9</sup> subarachnoid haemorrhage,<sup>10-12</sup> cerebral emboli,<sup>13</sup> and induction of anaesthesia.<sup>14</sup>

The incidence is hard to determine as less severe cases may be unrecognised or attributed to aspiration. Graf and Rossi identified only two cases from a review of 2100 head injuries,<sup>15</sup> but a more recent report gives an incidence of 0.62% in isolated head injuries,<sup>16</sup> and one intensive care unit dealt with 20 cases over a 45 month period.<sup>17</sup>

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