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.

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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.
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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.[1] Most initial reports associated neurogenic pulmonary oedema as being precipitated by head injuries,[2–4] 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.[5] Isolated head injury is still the commonest association, but a variety of other precipitants have been reported including epilepsy,[6–9] subarachnoid haemorrhage,[10–12] cerebral emboli,[13] and induction of anaesthesia.[14]

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,[15] but a more recent report gives an incidence of 0.62% in isolated head injuries,[16] and one intensive care unit dealt with 20 cases over a 45 month period.[17]

Accident and Emergency Department, Royal Alexandra Hospital, Corsebar Road, Paisley PA2 9PN

Correspondence to:
Mr Kerr.

Although many of the case reports involve children or young adults,[3] [11] [14] [18] [19] a review of 20 patients[17] had an age range of 11 to 71 and demonstrated no clear relation to age. There is also no literature documenting any other association such as preceding alcohol or drug ingestion.

Typically the patient unexpectedly develops pulmonary oedema after a head injury or subarachnoid haemorrhage. In addition to the hypoxia and frothy sputum, there may be peripheral vasoconstriction with pallor, sweating and a rapid, weak pulse.[18] [20]

Despite experimental work with animals[21] [22] the pathophysiology is not fully understood but probably both hydrostatic pressure and altered capillary permeability play a part in the pathogenesis. An acute cerebral insult with raised intracranial pressure is followed by massive release of catecholamines by the sympathetic system probably caused by a disturbance of hypothalamic function. This leads to a large and rapid increase in systemic and pulmonary blood pressures while the cardiac output falls due to the massively increased peripheral resistance. As a result of this there is a redistribution of blood volume from the systemic to the pulmonary circulation and this leads to pulmonary venous and left atrial engorgement. It has been suggested that the subsequent rise in pulmonary capillary pressures results not only in hydrostatic oedema but also damages the pulmonary endothelium so that a permeability effect persists after resolution of the pressure changes.[23] [24] Other experiments have described how increased pulmonary permeability and oedema can follow a cranial injury without the pulmonary vascular pressures being raised.[22] Alternative mechanisms such as direct α adrenergic effects on endothelial cells or release of histamine due to sympathetic stimulation have also been suggested,[25] although experimental work has discounted the possibility of large doses of catecholamines directly increasing microvascular permeability.[26] However the protective effect of cervical transection[5] [27] and results demonstrating that adrenergic blocking agents inhibit the development of neurogenic pulmonary oedema[28] appear to confirm the key role of the sympathetic outflow in the pathogenesis.

As the pathophysiology is not fully understood the optimal treatment of neurogenic pulmonary oedema is not yet established. Generally, treatment is aimed at reduction of intracranial pressure, diuresis, and reversing the peripheral effects of the sympathetic stimulation.[19] Controlled hyperventilation with intermittent positive pressure ventilation and, if necessary, surgical decompression, are used to reduce intracranial pressure. Diuretics, usually frusemide or mannitol, are used to reduce blood volume and intracranial pressure. The depression of myocardial function generally associated with neurogenic pulmonary oedema has been shown to be reversed by dobutamine[17] and the combination of a positive inotrope and

a vasodilator is an established method of improving cardiac output.[3] The mortality of head injury patients with associated neurogenic pulmonary oedema may exceed 90%.[29] It is also commonly found in epileptics who die suddenly after an otherwise unremarkable fit.[6] [7]

Neurogenic pulmonary oedema is an uncommon complication of cerebral trauma but the diagnosis should be considered in appropriate cases. The mortality is high and early aggressive treatment is required to improve chances of recovery. Confusion with presentations such as aspiration may delay recognition and prevent optimal management thus compounding the effects of an already serious cerebral insult.

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