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Case report

Fulminant fat embolism associated with closed fracture reduction

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Fulminant fat embolism is well recognised although uncommon. This case report of a patient developing pulmonary oedema during surgery and anaesthesia and dying four hours later is rare, unusual and disturbing. Two similar but non-fatal cases, both young males, of intraoperative pulmonary oedema associated with closed fracture reductions, where fat embolism was implicated, have been previously reported in the literature.[1]

CASE HISTORY

A 60-year-old woman was admitted following a road traffic accident, having sustained a fracture of the right tibia and fibula, a compound fracture of the right olecranon, a fractured right clavicle and fractures of the seventh, eighth and ninth ribs on the right side. She was conscious and fully orientated with a blood pressure of 120/60 mmHg and a pulse rate of 60/min. Chest X-ray showed a contused right upper lobe.

Three hours later she was referred for anaesthesia for reduction of her compound limb fractures. Pre-operative examination showed her to be drowsy and pale with cold cyanosed peripheries. There had been persistent oozing from her fracture sites since admission, with an estimated blood loss of 1.5 litres. Her haemodynamic status had deteriorated, with a blood pressure of 100/60 and a pulse rate of 96/min. Anaesthesia was deferred pending fluid replacement with 500 ml of gelatin solution (Haemacell), 840 ml blood and one litre of compound lactate solution over three hours while central venous pressure was monitored. Supplementary 40% oxygen by mask and 500 mg hydrocortisone intravenously was also given. Her clinical state improved but urinary output remained poor.

At this stage she was considered sufficiently improved for reduction of her badly displaced fractures. Anaesthesia was induced uneventfully with 2 ml intravenously of a mixture of alphaxalone and alphadolone (Althesin), and maintained by spontaneous ventilation of 50% nitrous oxide in oxygen via a face mask with intravenous increments of the induction mixture given as required. Throughout

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the 45-minute procedure, she was haemodynamically stable until the final 10 minutes when she suddenly became centrally cyanosed and tachypnoeic, even though the inspired oxygen concentration had been increased to 100%. Although the continuous ECG record and blood pressure were normal, the heart rate had risen to 120/min. A tracheal tube was inserted and pink frothy sputum was aspirated. There was no evidence of gastric contents on endotracheal suction, the trachea was centrally located and on auscultation diffuse bilateral pulmonary crepitations were audible.

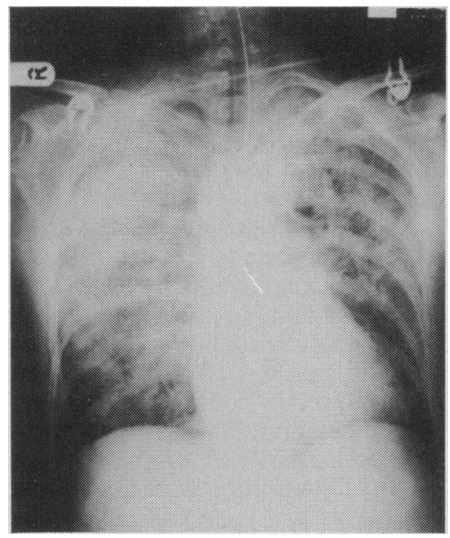

Fig 1. Post-operative chest film showing extensive 'snow-storm' infiltrates over both lung fields.

Post-operatively she remained cyanosed. Controlled ventilation with 100% oxygen was started and 80 mg frusemide and 1 g hydrocortisone were given intravenously. Chest X-ray (Fig 1) showed extensive bilateral pulmonary infiltrates consistent with pulmonary oedema. The differential diagnosis included aspiration pneumonitis, fat embolism syndrome, fluid overload, a severe hypersensitivity reaction to the induction mixture or acute cardiac failure secondary to possible cardiac contusion. Arterial blood gas analysis revealed gross hypoxaemia, PaO_2 3.8 kPa (normal range 12.6 – 13.3 kPa), PCO_2 5.23 kPa (4.5 – 6.1 kPa), pH 7.23 (7.36 – 7.44). Central venous pressure had risen from 9 cm H_2O pre-operatively to 14 cm H_2O. Urine output, scanty throughout, had now stopped. Peak inflation pressure was 25 cm H_2O and a positive end-expiratory pressure of 10 cm H_2O was applied without any improvement in oxygenation. Despite supportive treatment the patient had repeated episodes of asystole and died four hours post-operatively.

At autopsy, both lungs were grossly oedematous. Microscopic examination revealed massive pulmonary fat embolism (Fig 2) with the alveolar walls clearly outlined by fat-laden capillaries. Early reactive changes and interstitial oedema were also evident. There was also extensive deposition of fat in myocardial, renal glomerular and cerebral capillaries.

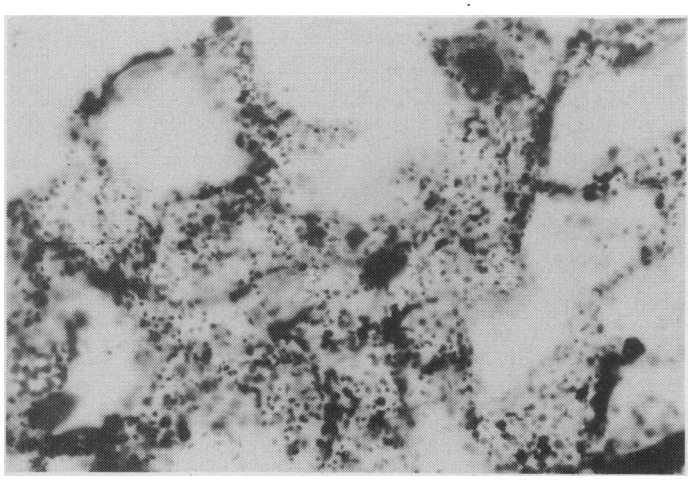

Fig 2. Histological section of lung showing fat emboli (black) in vessels of the alveolar walls, and early reactive changes.

DISCUSSION

Fulminant fat embolism appears to be an accelerated version of the classical fat embolism syndrome, differing only in severity and rapidity of onset. It occurs within a few hours of injury and is rapidly progressive with a fatal outcome in 90% of cases.[2] It can present with pulmonary hypertension, acute cor pulmonale, shock and sudden respiratory failure. The diagnosis is usually confirmed at autopsy.

In our patient, fat embolism was not initially suspected: the first arterial blood gas analysis was performed only after the clinical signs of hypoxaemia became apparent. The sudden onset of intra-operative cyanosis and pulmonary oedema in our patient supports the view that closed fracture reduction can produce repeated showers of fat emboli from traumatised tissues.[3] The filtering capacity of the lungs and the compensatory ability of the pulmonary circulation especially by capillary recruitment are normally very effective, so that moderate degrees of occlusion only produce a slight rise in pulmonary vascular resistance.[4] However, in a patient already compromised by fat embolism, additional fat emboli released by operative manipulations can overwhelm the compensatory capacity of the lungs, resulting in sharp rises in pulmonary vascular resistance and right ventricular pressure.[5] While operative interference in the presence of fat embolism exacerbates the syndrome, other studies have shown that early effective immobilisation can prevent fat embolism.[6] Herein lies the central dilemma. In retrospect it might have been wiser to delay surgery for at least 24 hours to allow further assessment of the patient in view of her initial hypovolaemia, lung contusion and rib fractures.

We would emphasise that a high index of suspicion for fat embolism and careful patient observation, including serial arterial blood gas analysis, is important in all severe trauma cases before operative intervention should be contemplated.

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