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Fat embolism — a review

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SUMMARY

The subject of fat embolism is of recurring interest to those managing trauma. This article covers the topic of fat embolism in general, and presents a case of fulminant fat embolism syndrome which highlights the importance of clinical expertise, and whatever technological aids are available to diagnose and appropriately treat this relatively rare, but highly significant form of the syndrome. Fulminant fat embolism syndrome has a very high mortality and should be watched for in patients who have experienced major trauma.

INTRODUCTION

Any case in which fat globules are demonstrated within the lung parenchyma or peripheral microcirculation can be described as 'fat embolism' (Van Besouw *et al.*, 1989). The fat embolism syndrome has been recognized for over 100 years, but there remains considerable doubt as to its incidence and clinical significance. Post mortem studies of trauma victims have suggested that the incidence of fat embolism can be very high, (up to 100% in some studies) and occurs very rapidly after severe trauma (Tanner *et al.*, 1990), but the significance of this finding as a possible cause of death has yet to be demonstrated.

Trauma to the fat-containing bony or soft tissues is the major cause of clinically significant fat embolism. Fractures of the long bones or the pelvis are particularly implicated. In many conditions, as diverse as decompression injury and diabetes, lipectomy (Christman, 1986), bone marrow transplantation (Lipton *et al.*, 1987) and joint replacement (Hagley *et al.*, 1986), there is histological evidence that fat globules cause blockage of blood vessels. In most of these cases, however, the emboli are of little clinical significance, and are very few in number.

The fat embolism syndrome is a recognizable systemic reaction which occurs in some patients with fat embolism.

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Ernst von Bergmann, in 1873, made the first clinical diagnosis of fat embolism (Bergmann, 1873) based on his knowledge of the pathology of fat embolism, and the similarity of his patient's symptoms with those of cats which he had used in scientific research 10 years before, when he studied the effects of intravenous injection of oil (Bergmann, 1863). In the case reported by Bergmann, the patient developed dyspnoea, cyanosis, and coma some 60 h after falling from a high roof and sustaining a comminuted fracture of the distal femur. He died 79 h post trauma, and a massive pulmonary fat embolism was discovered at autopsy.

In 1911, the term 'cardiac syndrome' was used by Grandahl to describe the early acute onset of tachycardia and hypotension seen with fat embolism. He attributed this to blockage of the pulmonary arteries by fat (Sevitt, 1962).

In 1962, Sevitt devised the current classification of the fat embolism syndrome into three recognizable systemic reactions (Sevitt, 1962). Of the three distinguishable clinical spectra, the first, subclinical, is characterized by decreased PaO_2 and minor haematological changes around 3 days post trauma. The second, non-fulminant, which occurs any time up to 6 days post trauma, consists of any or all of fever, petechiae, tachycardia, respiratory failure, and signs of central nervous system (CNS) embolism. Thrombocytopaenia, anaemia, and coagulation abnormalities can be found, as can pulmonary alveolar and interstitial opacities on chest X-ray. There is no definitive test for this version of the syndrome, and as most of the changes described can occur as a result of trauma as well as a result of fat embolism, the diagnosis remains a clinical one, and the significance is uncertain.

The third form of fat embolism syndrome, the fulminant variation, is of considerable significance to those treating post traumatic patients. It occurs very suddenly and rapidly after injury, and progresses very quickly, often resulting in death within a few hours of the initial trauma. The major clinical features are acute respiratory failure, acute cor pulmonale and embolic neurological changes. These occur shortly after injury and often result in the death of the patient. Those who have suffered multiple fractures are particularly susceptible to this form of the syndrome, which, although it is relatively rare, is of immense clinical significance because of its high mortality.

There are many who do not agree with Sevitt's view of fat embolism syndrome. Peltier represents a popular theory when he states that essentially, fat embolism is a pulmonary disease. He emphasizes that fat emboli are capable of causing acute cor pulmonale (Peltier, 1965). He suggests that humeral and cellular factors such as platelets and vasoactive amines might play an important role in the creation of the vascular obstruction in addition to the mechanical blockage from the fat globules.

Peltier's theory of rapidly progressive right heart failure accompanied by pulmonary hypertension, appears to be the most widely held current view of fulminant fat embolism syndrome (Evarts, 1970; Weiss, 1974).

CASE REPORT

The following case recently presented to our Hospital. A 31-year-old male was brought to the Accident and Emergency Centre (A&E) with multiple injuries after

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jumping from a highway overpass which was 15 m above the ground.

He was a paranoid schizophrenic who had attempted suicide some 9 months previously by jumping from the Sydney Harbour Bridge. At that time he had suffered severe injuries, including bilateral compound fractures of the tibia and fibula, fractured pelvis, crush fractures of the lumbar spine with an L3 partial paraparesis, retroperitoneal haematoma, subcapsular haematoma of the liver and pulmonary contusions. He required ventilation and tracheostomy. He was discharged after 9 months of intense in-patient treatment, still on walking sticks and calipers, and with a suprapubic catheter. Just 3 days later, apparently dejected as a result of his unrequited love for a member of the nursing staff who had nursed him during his stay, he jumped again. This time he was found on the roadway at the bottom of the Harbour Bridge.

On arrival at (A&E) he was shocked, with laboured respirations and confused verbal responses. His pulse was 170 beats min^{-1}, and blood pressure was $80 \times 40\,mmHg^{-1}$ in a MAST suit. The airway was clear and air entry was thought to be equal. He had an occipital laceration and a dilated right pupil, but moved both upper limbs symmetrically. The abdomen was soft, and a recent midline laparotomy scar was noted. There was gross haematuria from an indwelling suprapubic catheter. Chest and pelvic spring were normal. There was no evidence of long bone fractures or significant extracavitary haemorrhage.

A cross table lateral view of the cervical spine was normal. Supine chest X-ray demonstrated bilateral patchy opacities through both lungs. No rib fractures were seen.

Volume resuscitation was commenced with 3000 mls of Haemaccel and 4 units of packed cells. A right intercostal catheter drained 50 mls of blood. A left intercostal catheter drained a small amount of air. The patient was intubated and ventilated.

Despite these measures the patient's condition continued to deteriorate, with hypotension unresponsive to volume expansion. Diagnostic peritoneal lavage was thought to be contraindicated due to the recent surgery. A subxiphoid pericardial window evacuated 50 mls of blood from the pericardial cavity without improvement. Subsequent bradycardia progressed to an asystolic cardiac arrest 50 min after arrival in the department. Further attempts at resuscitation proved unsuccessful.

At autopsy, massive pulmonary fat embolism was found, with a single large embolus filling the pulmonary trunk as well as multiple smaller emboli in distal pulmonary arteries. A small haemopericardium, left pneumothorax and right haemopneumothorax were noted. There was a small laceration of the liver without significant haemoperitoneum. The pelvis was fractured. There were no long bone fractures. There was no intracranial mass lesion. Unexpected pulmonary and extrapulmonary tuberculosis was also found.

DISCUSSION

This case is an example of the rarely occurring fulminant fat embolism syndrome.

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The sudden onset within a few hours of injury, of respiratory failure and hypotension progressing rapidly to death from acute right ventricular failure is well described.

The fulminant course, and the need to exclude other, treatable, causes of hypotension following multi-trauma, make the clinical diagnosis difficult. The diagnosis remains essentially clinical.

In this particular case, fat embolism was diagnosed at post mortem examination. This is typical of the majority of described cases (Hagley, 1983), however, with modern investigative techniques, and a greater awareness of the condition, it should become more common to diagnose the syndrome earlier, excluding other possible causes of the symptoms.

Diagnosis

Fat embolism, in either adults or children is most likely to result in a patient who has suffered multiple fractures of the long bones. Inadequate initial treatment such as proper splinting, and rough transportation are important aetiological factors, as is hypovolaemic shock. Pre-existent cardiac and pulmonary disease, and osteopaenia are likely to predispose to the development of fat embolism, and a cirrhotic fatty liver can even be the source of fat emboli.

In the post-injury period, there are a number of clinical findings which are highly suggestive of fat embolism:

(a) *Tachypnoea, dyspnoea and profuse tracheobronchial secretion*. It has been reported that such symptoms of respiratory insufficiency occur, usually 2 or 3 days post trauma, in 75% of patients with fat embolism (Gurd & Wilson, 1974). In some 10% of patients the symptoms progress to respiratory failure (Guenter *et al.*, 1981);

(b) *Anxiety, apprehension, delirium, increasing unconsciousness*. These signs can occur up to 12 h before respiratory features and although they may be the primary presentation, it is rare for patients not to develop additional respiratory signs (Findley *et al.*, 1984). Jacobsen estimated that up to 86% of patients who have fat emboli develop neurological signs (Jacobsen *et al.*, 1986). While most patients develop a state of acute confusion, some develop additional focal neurological signs which can include hemiplegia, apraxia, aphasia, scotomas and anisoconia (Thomas *et al.*, 1974; Jacobsen *et al.*, 1986); and

(c) *petechial haemorrhages*. These are of a classical type described by Benestadd in 1911 (Benestadd, 1911). The rash, which occurs in some 60% of patients (Gurd & Wilson, 1974), develops early in the process, usually within the first 36 h, and it usually resolves within a week. It is most obvious in the conjunctiva, and mucous membranes and skin folds of the upper half of the body, especially around the neck and in the axilla.

It is essential that all clinicians who must treat patients following long trauma, develop and maintain a high level of suspicion, and watch for any early signs of development of these symptoms.

In addition to the common suggestive features, there are a number of signs, often related to fat embolism which, if present, may strengthen the basis of diagnosis. These include:

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(1) *Retinal Signs* — retinal haemorrhage, mucular oedema, fluffy exudates and, rarely, presence of fat droplets in the vessels;

(2) *Hepatic Signs* — rarely, jaundice may be the presenting sign (Gurd & Wilson, 1974);

(3) *Fever* — may be secondary to underlying infection or may result from the fat embolism (Murray et al., 1974);

(4) *Tachycardia* — again may result from an underlying infection; and

(5) *Renal Signs* — transient oliguria, lipuria, proteinuria, and haematuria may all result from fat emboli in the renal system.

In the laboratory, the essential diagnostic test is the measurement of blood gases. In any patient who has experienced bony trauma, measurement of blood gases should be performed as soon as possible after admission, and should be repeated frequently over the next 48 h. The other significant blood test is daily platelet counts as thrombocytes below 150 000 cubic ml^{-1} is diagnostic of fat embolism.

A number of other laboratory findings have been associated with fat embolism, however, the tests concerned are too sensitive to be of clinical value at this stage. These include lipuria (occurs in approximately 50% of all patients with significant bony injury); fat in the sputum (of no significance in diagnosis of fat embolism); serum lipase (elevations occur in about 50% of patients with fractures); and detection of fat droplets in the circulating blood (Peltier, 1988).

One of the major problems in dealing with fat embolism of a severe kind is that the three major elements of the fulminant fat embolism syndrome as described by Sevitt (1962) — acute respiratory failure, acute cor pulmonale and embolic neurological changes — can all occur as a result of the trauma itself, particularly as the patients who are most susceptible to the syndrome are those who have experienced the most severe trauma resulting in multisystem damage. A C.T. scan coupled with the clinical picture, may be able to differentiate primary intracranial injury from embolic neurological changes, and a Swan-Ganz Catheter can differentiate hypovolaemic shock from acute cor pulmonale using estimation of right atrial and pulmonary wedge pressures (Hagley, 1983). An ECG may suggest acute cor pulmonale or myocardial ischaemia, arterial blood gases estimation will show hypoxia and a normal or low $PaCO_2$, and there may be abnormal coagulation studies. Chest X-ray can demonstrate pulmonary oedema.

Treatment

The management of fat embolism is essentially preventive and supportive. These include careful and adequate first aid measures such as splinting and gentle transportation to hospital. Early immobilization of fractures (Baker, 1976), including immediate open reduction and internal fixation of multiple long bone fractures, is essential (Riska *et al.*, 1982; Saikku, 1954). Restoration of adequate blood volume, that is prevention or treatment of hypovolaemic shock, must be undertaken immediately as shock has been shown in animals to increase the incidence of fat embolism (Watson, 1970). Human albumin solution has been demonstrated to reduce the incidence of fat emboli in multiple trauma patients, and is therefore

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the choice for fluid replacement in such patients (Moylan, 1976). Early, and frequent measurement of the blood gases, with supplemented oxygen therapy where needed, may reduce the incidence and severity of fat embolism syndrome. Treatment of acute respiratory failure by standard methods of ventilatory support (intubation and respirator) should be undertaken in all patients whose blood gases cannot be maintained at acceptable levels by administration of oxygen (Lindeque *et al.*, 1987).

Unfortunately, treatment of acute right ventricular failure is rarely successful. Inotropes and careful fluid loading may be useful. Cardiac and pulmonary fat embolectomy for suspected fat embolism has been reported (Nelson, 1974), but has not been greeted with enthusiasm in most quarters. The use of corticosteroids in massive doses may be considered for initial prophylaxis, or as therapy if lung function deteriorates. It must be emphasized, however, that while steroids may be of benefit, there is no documented and standardized proof of this.

Other specific measures which have been proposed for the treatment of fat embolism include alcohol, heparin, dextran 40, and aprotinin. All of these agents have supporters, but no agreement has been reached as to the benefits of their use, largely because trials have shown conflicting results.

Outcome

Most patients with fat embolism syndrome can expect to return to normal, if adequate support, particularly of ventilation, has been provided. The overall mortality rate for this condition is low (5–15%), with the severity of respiratory problems being a close indicator of the risk of death. Cerebral complications, and especially focal neurological defects, provide for most of the long term morbidity associated with this condition (Jacobsen, 1986; Gurd, 1974). Fulminant fat embolism syndrome as described by Sevitt has a high mortality, and despite modern technology and improved practice, not a great deal has been achieved in its management.

Conclusion

It is essential that those involved in the acute management of severely traumatized patients are aware of the possibility of fat embolism syndrome, and use clinical expertise and whatever technological aids are possible to diagnose it and treat it appropriately. Particular attention must be paid to prevention, if possible, and to maintenance of adequate levels of oxygenation in any susceptible patient.

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