Fat Embolism

Pages with reference to book, From 122 To 126

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Abstract

Multiple injuries including fracture of the bones and crush injuries of the limbs pose many problems in their management. Fat Embolism, a mere 2.5% of the total number of cases presented here, remains a dreaded complication. Early diagnosis and management has a significant effect on the eventual morbidity and mortality. In the present study we have tried to standardize the diagnosis and management of these cases in the light of present knowledge. This series also highlights the fact that it is not a common complication but one must always be on the look in relevant cases (JPMA 34: 122, 1984).

Introduction

Fat embolism is a rare though serious complication of trauma, specially fractures of the long bones. It is a significant cause of post traumatic respiratory insufficiency (Riseb Rough and Edward, 1974). According to some estimates 10% of autopsies following trauma show evidence of fat embolism. Patients with extensive soft tissue injury and hypovolamic shock have an incidence of upto 60%. In war wounds and injuries the incidence may even go upto 80% (Scully, 1956).
Our experience with fat embolism occurring in patients admitted with multiple fractures is presented in this study.

Material and Methods

Two hundred and fifty three patients with multiple injuries were admitted through emergency during a period of 2 years (1st Jan. 1981- 31 Dec , 1982) of these 10 (7 males &3 females) developed fat embolism.
The laboratory investigations done on each case consisted of chest x-ray E.C.G., complete blood examination including platelet count, serum fibringen and albumin levels, Arterial P02, urine for fat glubules and Fundoscopy.

Results

Age and Sex

Of 10 cases with fat embolism there were 7 males and 3 females (male to female ratio was 2.3: 1). Age varied from 17 - 60 years.

Table I

Fracture Sites.

(Fracture of Tibia & Fibula were taken as fracture of single major bone).

1.	Fracture of one bone:		2
	a) Femur	1	
	b) Tibia	1	
2.	Fracture of two bones:		5
	a) Femur + Tibia	3	
	b) Femur + Humerus	1	
	c) Tibia + Humerus	1	
3.	Fracture of three bones:		3
	a) Femur + Tibia + Humerus	1	
	b) Tibia + Radius + Ulna	1	
	c) Femur + Radius + Ulna	1	
		Total:	10

Table I presents an analysis of fractures of long bones during the accidents. Two patients had fracture of single bone, 5 fracture of two major bones, and 3, fracture of 3 bones.

Signs and Symptoms

All patients were in hypovolemic shock when first seen, and recovered after resuscitation (I.V. fluids

and blood transfusions), remained well for about 48 has.,, and then suddenly got worse. This rapid deterioration in the absence of haemorrhage or sepsis was considered to be due to fat embolism. The time of onset of fat embolism is given in fig I.

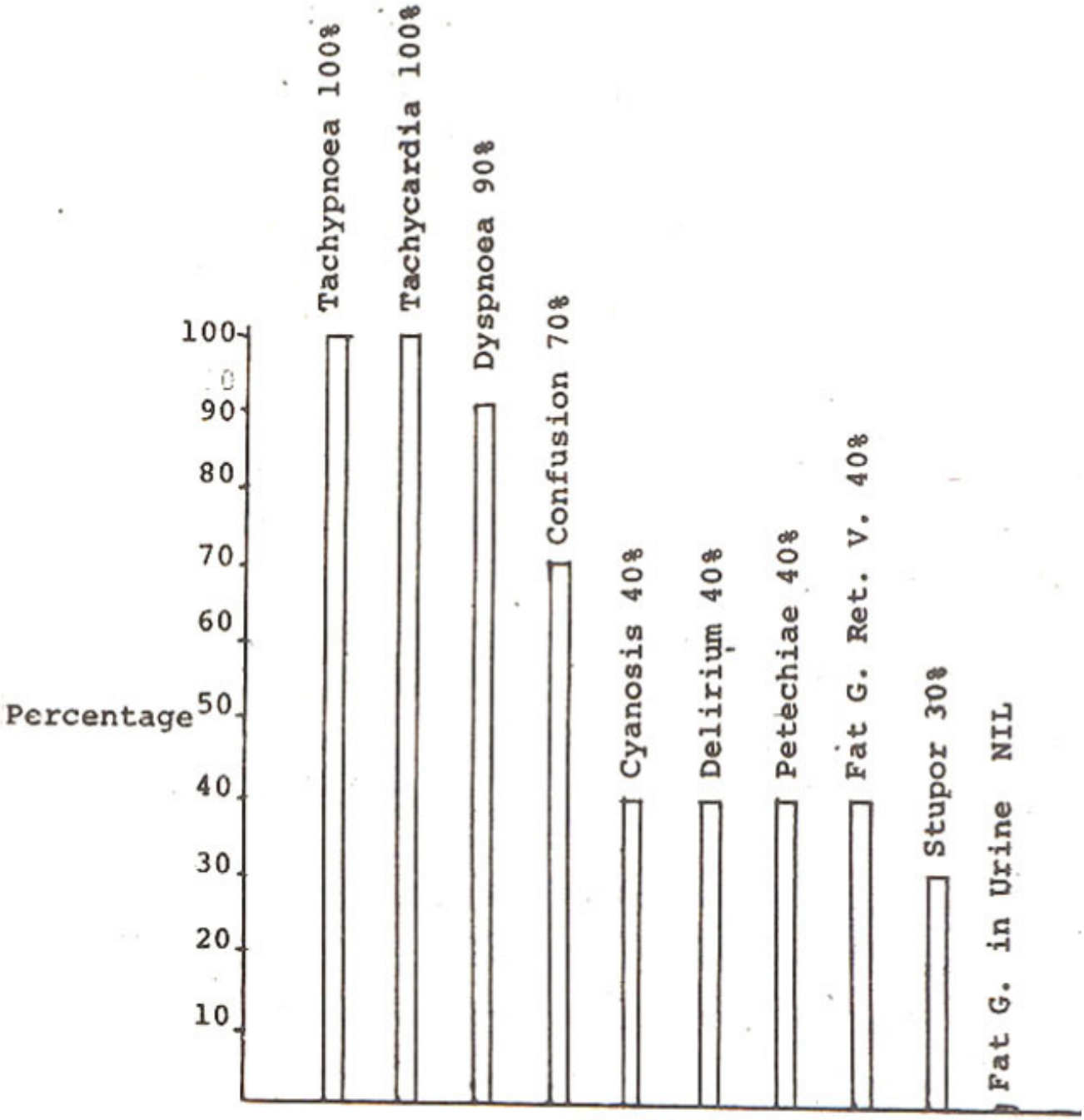

Fig. 2 Symptoms and signs of fat Embolism.

In 7 out of 10 patients, the time of onset was 2-3 days after the accident. The earliest possible time was 36 hours and latest 96 hours, average about 60 hours.

This deterioration in the clinical state was accompanied by confusion, delirium, dyspnoea, pyrexia going upto 102°F, hypotension, and evidence of diminished peripheral perfusion. The oliguria observed in these cases was perhaps due to a period of sustained hypotension and soon recovered in all those

cases who survived. The systems affected in the order of frequency were respiratory, cardio-vascular and nervous system. Tachyponea and tachycardia were present in all cases, dyspnoea in 90%, confusion in 70% and other symptom in 30 - 40% cases. Two patients developed coma and died (Fig. 2).

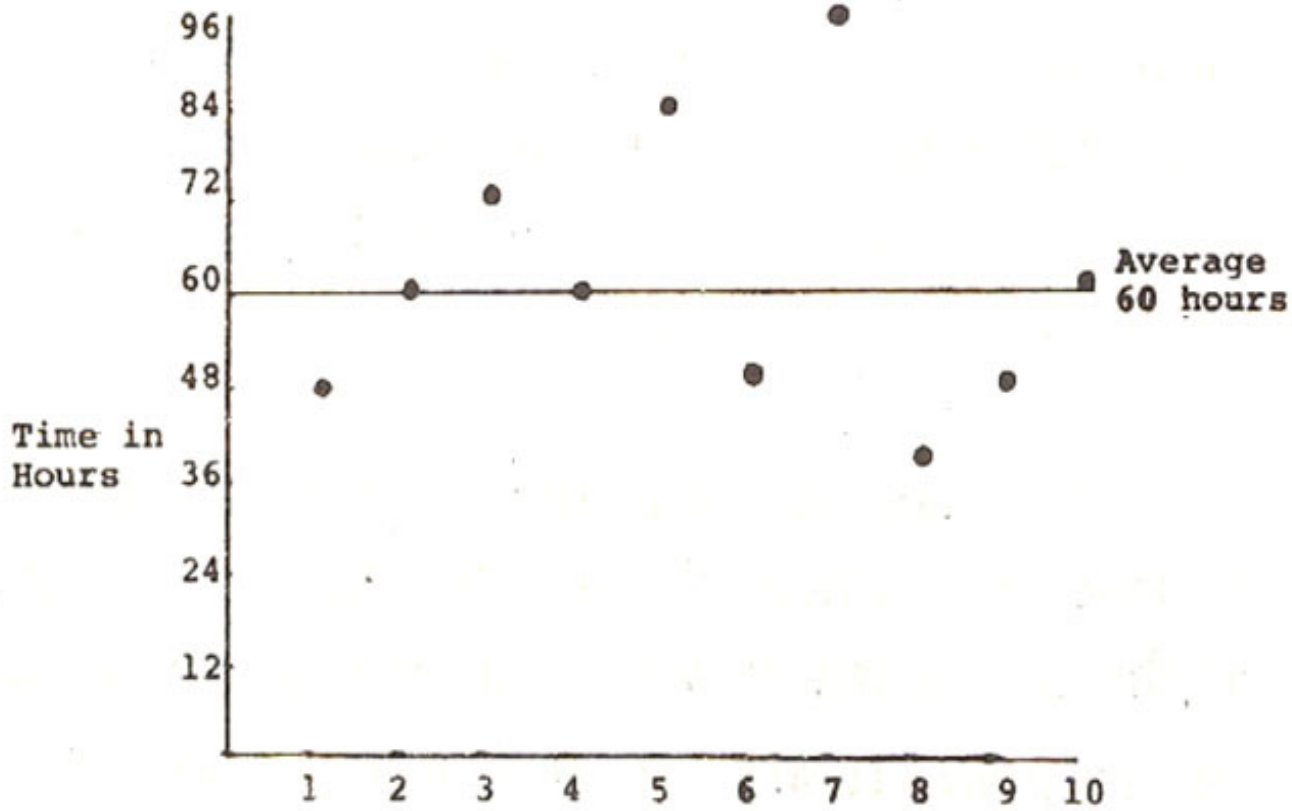

Fig. 1 Time of onset of Fat Embolism.

Fat embolism should be suspected in patients with multiple injuries, who presented with shock, tachycardia and neurological symptoms.

Investigations

X-ray chest in 40% cases showed fluffy densities, and similar number had right axis strain on electrocardiogram. Non specific S-T changes were seen in 30% cases.

Fat globules could not be demonstrated in a 24 hrs specimen of urine in any of the patients.

Artarial P02 levels dropped significantly in 9 out of 10 patients (fig 3)

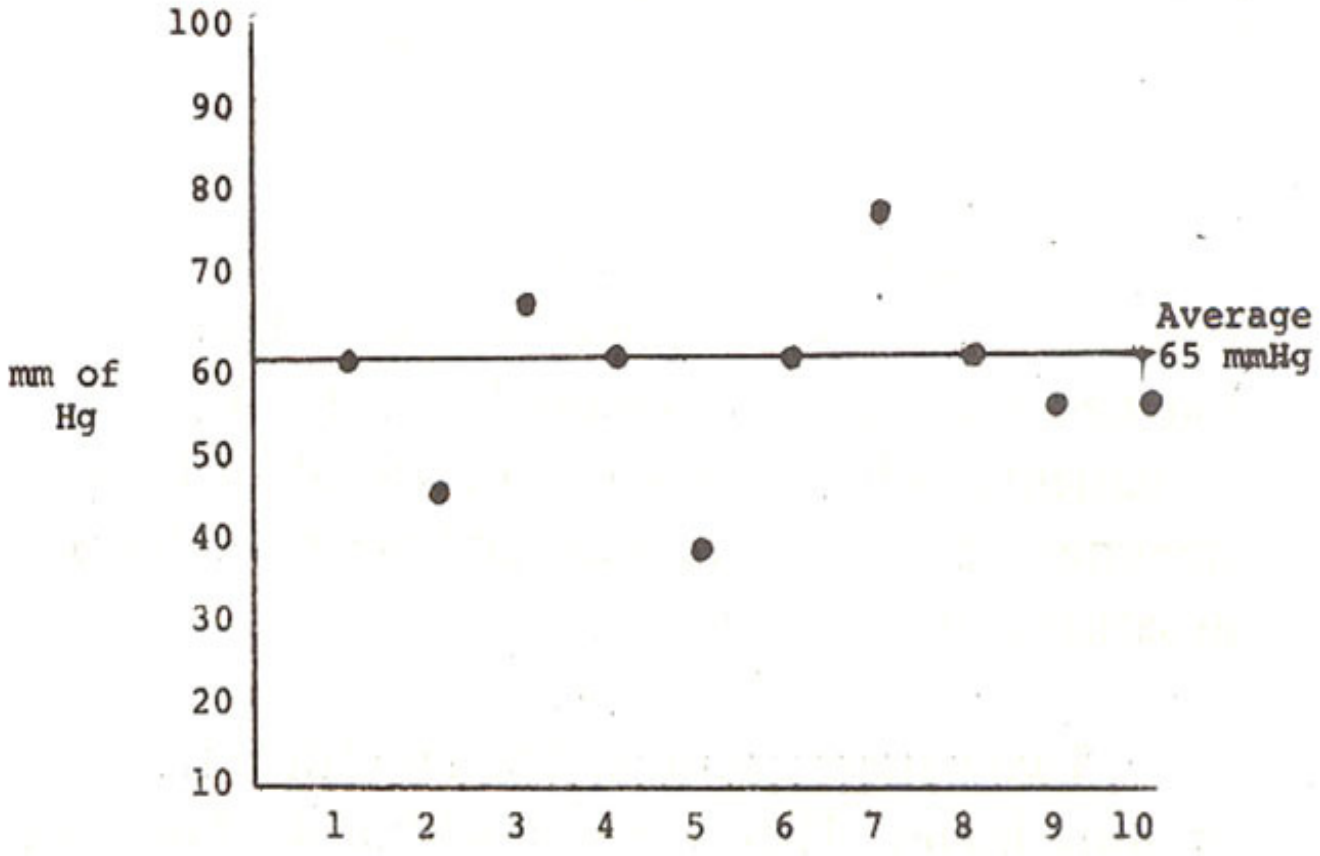

Fig 3 Arterial PO_2 level in 10 patients.

lowest level was 45 mm of Hg and highest 80mm of Hg (Mean 65mm of Hg)
Elevated levels of serum lipase (normal 1.5 units) were found in 4 cases while it was normal in 6 cases. Platelet count was normal in 4 and in the remaining it was less than 150,000/cu mm. Serum fibrinogen level was less than 200mg in 5 cases, 90% patients had hypoalbuminaemia (2.3 G).

Treatment

Supportive therapy consisted of measures to overcome respiratory distress. Oxygen had to be administered to most of the patients, some of them needed Aminophylline as well. Shock was treated by I.V. fluids and blood transfusion. Two patients needed tracheostomy and positive pressure ventilation.

Steroids were given to all patients during the state of shock. In 5 patients a single bolus of 500 mg of hydrocortisone sodium succinate was given. In three patients it had to be continued for 5 days and then gradually tapered off. In the two patients who expired, these were administered in increasing dosage till the time of their death.

Outcome

Eight patients made uneventful recovery and 2 patients died showing mortality of 20%. Four patients recovered within 72 hours of the onset of symptoms, 2 patients took another 12 hours, 1 patient recovered after 96 hours, and 1 became stable after 5 days.

The first sign to recover was tachypnoea, then the pulse rate became normal. Temperature touched the base line within 48 hours. All of the 8 patients who survived underwent definitive treatment for their fracture and were discharged in satisfactory condition.

Two patients who died bad multiple fractures of long bones. They expired within 48 hours of the onset of fat embolism despite the standard treatment.

Discussion

Zenker (1862) described the features of the fat embolism in the pulmonary tissues of persons dying following injury. Bergmann (1873) is credited for the first description of clinical findings following long bone fractures i.e. confusion, dyspnoea and patechiae.
Fenger and Salisbury (1879) presented the first American experience of the pathological process.
Dennis (1895) described the importance of fat embolism as a cause of collapse following injury. According to him the rule of three should be remembered as the cause of collapse after trauma:
1. Shock in first three hours.
2. Fat emboli after three days.
3. Pulmonary emboli after three weeks.
The management of fat embolism was first described by Hermann (1932) using a solution cf 5% Ethyle Alcohol and 5% Dextrose.
Although the condition has been documented for over a century, the pathogenesis of fat embolism remains obscure. Most cases occur following fracture of long bones, often of the lower extremity.
The brunt of fat embolism is borne initially by the lungs leading to wide spread tissue anoxia. The effects of tissue anoxia and direct damage caused to the brain by the fat emboli dominate the subsequent clinical picture. If there is extensive embolism of brain, it may prove fatal.
As to the source of fat emboli, Gauss (1924) described the mechanical theory according to which fat emboli were derived out of the bone marrow into the blood stream by the direct effect of trauma. These emboli when so released into the circulation produced a temporary occlusion of the pulmonary circulation by causing ischaemia and petichial haemorrhages.
It was further demonstrated in support of this mechanical theory that large veins are torn at the site of injury with a simultaneous disruption of adipose tissue in the bone marrow. Since pressure within the marrow cavity exceeds that of venous pressure at the time of injury and subsequently fat is easily transported into the torn veins by mechanical pressure. The mechanical theory was further supported by Arnim and Grant(1966) who demonstrated bone trabecules in the lung capillaries of laboratory animals containing fat emboli after experimentally subjecting them to trauma.
Lehman and Moore (1927) proposed the physiochemical hypothesis for fat embolism syndrome. They named it “Metabolic change theory”, according to which the changes seen in fat embolism are part of the metabolic change which occur after trauma. The fat can come either from the marrow cavity or other fat sources abundantly present in the body. The mechanism of emboli is that fat droplets in the range of 5 to 10 microns coalesce and form large chylomicrons which act as emboli following trauma. Serum free fatty acids are elevated in patients following major trauma. These have toxic effect on the lung tissue and cause disruption of the capillary alveolar membrane and alteration in the lung surfactant activity leading to oedema, haemorrhage and alveolar collapse (Peltier, 1956). Currently the “Metabolic Change Theory” favoured by most of the workers has an experimental proof (Kreis et aL, 1973). It is therefore possible that physiochemical chaiiges are responsible for fat embolism.
Mylan et al. (1976) have demonstrated that the patients following extensive trauma and fractures developing fat embolism syndrome have a significantly low serum albumin level in comparison with patients who do not show evidence of fat embolism. Fatty acids are released following extensive injuries which are bound to serum albumin when its levels are satisfactory i.e., above 3.5 grams per cent. In patients with low serum albumin during the first seventy two hours after injury there• is an elevation of free fatty acids which remain in circulation in unbound form and are responsible for fat embolism. It is clear therefore that the incidence of fat embolism is also related to the serum albumin levels.
Most of the patients with fractures have an unevenful course but a sudden turn for the worse in suth cases should raise the suspicion of fat embolism. The diagnosis of this condition should be based on

careful clinical examination and the treatment should commence without any delay.
The main presenting symptoms as seen in this series are dyspnoea, cyanosis and tachycardia followed by confusion, agitation and stupor. Two patients went into coma an4 died. Petichial haemorrhages were seen only in 40% cases, they were mainly visible on upper extremities chest, axillae and conjuctivae. Their appearance in patients with multiple injuries makes the diagnosis of fat embolism more evident. Multiple injuries including frictures with the above symptomatology were the yardsticks in making the clinical diagnosis in these patients.

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