

Clinical Workshop

S. G. HERSHEY, M.D., Editor

Diagnostic Features of Fat Embolism— Results in Six Cases

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Traditionally, the early diagnosis of fat embolism has been difficult. In the classic cases described by Van Bergman in 1873 and Christian Finger in 1879, petechiae, cyanosis, dyspnea, pulmonary rales and coma appeared in previously healthy patients following fractures of long bones. These clinical criteria have remained the cornerstone of the diagnosis.¹ Unfortunately, once these symptoms appear mortality approaches 85 per cent.² Therefore, efforts have been directed toward the establishment of early diagnoses by the use of laboratory tests. These have not been as discriminating as might be desired, but when used in conjunction with clinical criteria they can provide a diagnostic profile. This allows an early provisional diagnosis. Confirmation of the clinical impression by blood-gas measurements has now superseded all previous diagnostic methods. This report discusses diagnostic findings in six patients with fat embolism who made complete recoveries.

CLINICAL MATERIAL

All six patients were young healthy adults. None had a history of previous circulatory, pulmonary, or neurologic disease. All had been injured in traffic accidents. Each had received a fracture of a long bone of a lower extremity. All but one were lucid when admitted to the hospital emergency room. The exception was a patient thought to have a concussion and believed to be intoxicated. All pa-

tients were given early medical and surgical treatment for their injuries and admitted to the intensive care unit.

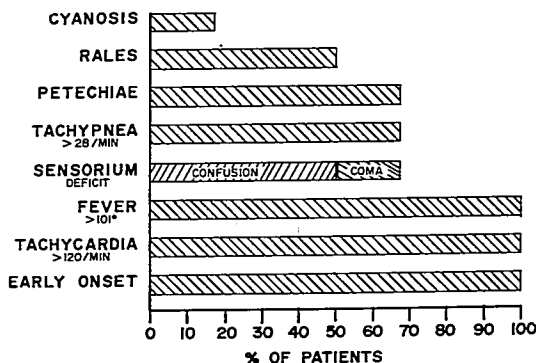
Signs and Symptoms. Fat embolism was suspected within 48 hours of injury in the cases of five patients, and 12 hours later in the sixth case (fig. 1). Unexplained fever and tachycardia were the earliest and most reliable findings, and these were found in all patients. Tachypnea, petechiae, and deficits in sensorium appeared later in four of the six patients. The changes in sensorium were evidenced by confusion and agitation, except in the case of one patient who became semicomatose for 36 hours. Pulmonary rales, present in 50 per cent, were not easily detectable, and were not associated with copious secretions. Mild hemoptysis occurred in one patient. Cyanosis occurred in one patient. Shock, as evidenced by hypotension, did not occur.

Laboratory Evidence. Despite early replacement of blood losses, continued decreases in hematocrit were observed in all patients (figs. 2 and 3), even in the face of brisk diuresis (fig. 4). Typical small fluffy infiltrates (snowstorm or salt-and-pepper appearance) were seen on roentgenograms of the chests of four patients. At the time of presumptive diagnosis of fat embolism, only two patients had positive roentgenograms. The patient with coma had an electroencephalographic abnormality consisting of a diffuse disturbance. Two patients had nonspecific EKG abnormalities (axis change and T-wave inversion). Unexpectedly, fat globules were found in the urine of only one patient, and elevation of serum lipase occurred in none. The blood-gas abnormalities were found early, and always before roentgenographic evidence of embolism.

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FIG. 1. Signs and symptoms.



These consisted of hypoxia, hypobarbia, and moderate-to-severe loss of bicarbonate (figs. 2 and 5).

Discussion

Our findings, like those of others, have shown the unreliability of symptomatic evidence in the diagnosis of fat embolism.² No single symptom or sign can by itself provide a diagnosis; in fact, all of them are substrata common to all illnesses. It is the failure to attribute them collectively to any other pathologic state that lends validity to the diagnosis.

Tachycardia despite replacement of estimated fluid losses has special significance in alerting the clinician to impending circulatory or respiratory failure. Each of our patients had a pulse rate greater than 120/min on the day of provisional diagnosis. The tachycardia

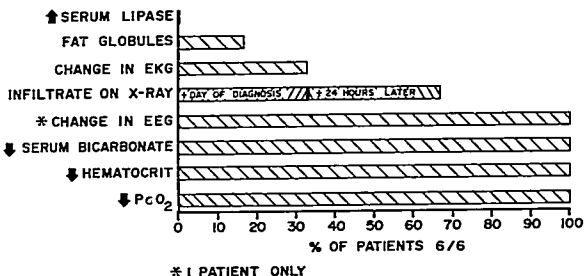
preceded the high fever of sudden unexplained onset, which was difficult to control with aspirin.

Mental changes ranged from slight loss of affect and decrease in memory span to frank psychosis and coma. However, confusion was the most common mental symptom in our patients and did not immediately clear with improvement of oxygenation.

Petechiae, present in four of the six patients, could not be elicited by tourniquet occlusion in the remaining two. This test is less successful in provoking petechiae than the use of negative pressure, according to Garner and Peltier.⁴ Yet our group had a higher incidence of petechiae than the 20 per cent cited by most investigators.

Tachypnea cannot be equated with normal or hyperventilation. Minute volumes, mea-

FIG. 2. Laboratory evidence.



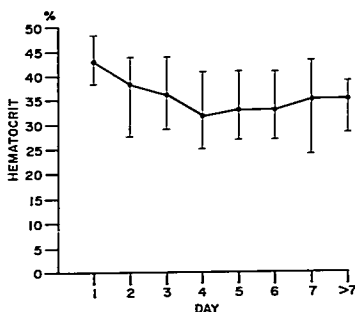


FIG. 3. Average and range of decreases in hematocrit following injury. Day 1 is day of admission and initial hematocrit determination.

sured in three of our cases, were slightly to greatly elevated (7.3 l, 15.2 l and 46.8 l). The clinician should be aware that minute ventilation may be tremendously altered by changes in tidal volume while rate remains normal or decreases.

There may be no significant auscultatory findings in the chest even in the presence of severe hypoxemia. They do not coincide with roentgenographic findings, either, I have pointed out the absence of detectable respiratory abnormalities in a previous paper.⁵

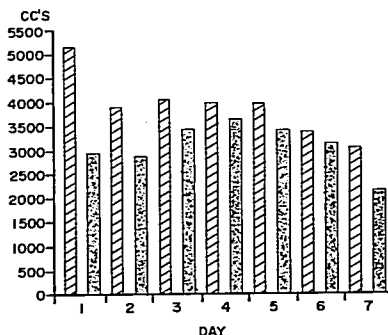


FIG. 4. Fluid intake (including blood) and output in five patients with fat embolism following injury. Day 1 is day of injury. Striped bars = fluids; dotted bars = urine.

The lack of specific laboratory indicators of fat embolism in our group of patients was most disappointing. The EKG was nonspecific and merely provided confirmatory evidence of circulatory distress, and this in only two of six patients. Fat globules, sought in both sputum and urine daily, were found in only one patient. Daily lipase determinations were performed, but we discovered no elevations. Although Peltier has been enthusiastic in promoting the use of this test for diagnosis and prognosis, recent work by Ross casts doubt on its validity.⁶ He compared serum lipase levels in a group of 53 patients who had fractures with those in a control group of 40 patients undergoing elective soft-tissue surgery and was unable to find a significant difference. In fact, he found no elevation in three patients with clinical evidence of the syndrome.

The decrease in hematocrit has been observed by all investigators. All admit inability to explain it solely on the basis of blood loss. Fuchsig believes that more blood is lost in the soft tissues than usually estimated and that further dilution is consequent to extravascular shifts into the circulation, when we examined the decreases in hematocrit in relation to fluid intake in five of our patients we found decreases in the face of polyuria (figs. 3 and 4). This observation became even more striking when we added a conservative volume of 900 ml to cover insensible loss (at times when all patients had temperatures above 101 F). We then compared the ratio of fluid loss to fluid intake and noted that although intake was greater than loss the first day, the situation subsequently reversed itself (fig. 6). We saw a decreasing erythrocytic mass at the time when fluid losses exceeded intake. It is inconceivable, therefore, that the decrease in hematocrit could be explained solely on the basis of hemodilution. Careful studies with isotope tagging may eventually explain the fluid shifts and the decrease in erythrocytic mass.

Roentgenograms of the chest have perhaps been more highly regarded than any other investigative technique, yet two of our patients failed to show the usual diagnostic features of infiltration. These patients' roentgenograms did show changes in pulmonary vascular markings, however, and we suggest that evidence

for the disappearance and reappearance of segmental vessels should be more carefully sought. We have also observed an oligemic appearance in the lung field preceding a congested phase followed by infiltration. Changes in hilar and cardiac shadows occur, but are usually slight.

The data from arterial blood gas analyses provide the most objective evidence of pulmonary embolization. Moderate-to-severe hypoxemia was present in every patient. This was most severe on the day of diagnosis, and increased gradually, with all patients returning to normal by ten days (fig. 5). Estimations of intrapulmonary shunts were made for four patients and were 7, 7.7, 17, and 24 per cent. A deadspace study, done for one patient, showed deadspace increased by 55 per cent. The hypoxemia is secondary to maldistribution because of increased deadspace as well as to the presence of right-to-left shunts. The increased deadspace is consistent with pulmonary embolization, and the shunting corresponds to the loss of alveoli secondary to infiltration and atelectasis.

The presence of hypocarbia in all patients (average P_{CO_2} 24, range 20–28 mm Hg) reflects the ventilatory response to hypoxemia. Patients with diseased lungs (emphysema, bronchitis or asthma) might be unable to increase ventilation and therefore arterial P_{CO_2} could be normal or increased. Initially five of the six patients had alkalosis (average pH 7.48, range 7.32–7.57). However, the pH values were not as alkalotic as might have been predicted from initial P_{CO_2} values (aver-

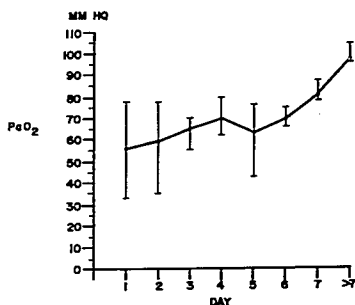


FIG. 5. Average and range of PaO_2 values in six patients with fat embolism while breathing room air. Day 1 is day of initial blood-gas studies.

age 24 mm Hg). The calculation of bicarbonate revealed considerable decreases in all patients. Thus, an underlying, but respiratory-compensated, metabolic acidosis had occurred. This corrected acidosis conceivably was secondary to poor peripheral perfusion, and lends validity to the theory of compensated or borderline shock said to be present in all patients. Early diagnosis and treatment undoubtedly accounts for the absence of frank shock in any of our patients.

Although the foregoing discussion has concerned itself with familiar diagnostic methods, widespread interest in the fat embolism syndrome is leading to the proliferation of new theories and investigation of many physiologic variables.⁷ Of particular interest are the similarities between the lungs in shock and the

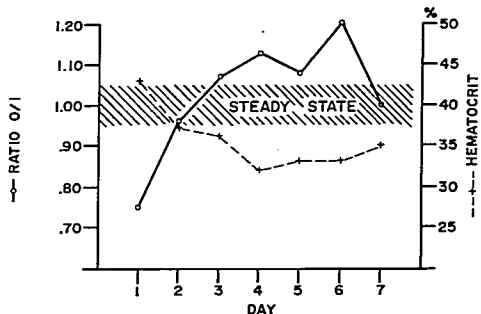


FIG. 6. Shifts in erythrocytic mass and fluid balance occurring in fat embolism. See text for details.

lungs in patients who have diffuse intravascular coagulation with fat embolism. This speculative connection between stress, hyperlipemia, and coagulation disturbances has stimulated investigators to look at levels and patterns of catecholamines, serum lipid fractions, and coagulation factors. Although the evidence is by no means consistent, it is anticipated that findings in these areas may provide more sensitive indicators of the fat embolism syndrome.^{8,9}

CONCLUSION

The diagnosis of fat embolism can be considered for any patient who develops tachycardia, fever, tachypnea, petechiae, or changes in sensorium early following fractures of a long bone. The diagnosis is supported by roentgenograms and decreasing hematocrit despite apparently adequate blood and fluid replacement. If these occur, arterial blood samples should be taken for analysis. The presence of hypoxemia without other explanation is diagnostic. Alterations in P_{CO_2} and pH are variable, but usually a compensatory respiratory alkalosis with an underlying bicarbonate deficit is found.

If morbidity and mortality are to be eliminated, careful observation of all patients for

this disease is needed. Aggressive therapy should virtually eliminate mortality in the group at risk.

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CASE REPORTS

Problems Related to Aldosteronism during Cesarean Section

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Hypersecretion of aldosterone results in retention of sodium and urinary loss of potassium, with development of hypokalemic alkalosis. The clinical manifestations are hypertension, headache, paresthesias, intermittent

tetany, periodic muscle weakness, polyuria and polydipsia. Primary aldosteronism (Conn's syndrome) is caused by an adenoma (adult form) or hyperplasia (juvenile form) of the adrenal cortex, while secondary aldosteronism is a complication of malignant and renovascular hypertension with excessive secretion of aldosterone due to stimulation by the renin-angiotensin mechanism. Primary aldosteronism rarely leads to peripheral edema or papil-

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