A week later atrial fibrillation reappeared and her blood calcium concentration had risen to 2·7 mmol/l (10·8 mg/100 ml). Frusemide 20 mg and 500 ml physiological saline were administered intravenously. Carotid massage was performed without success. Repeated doses of frusemide 20 mg and 5 mg verapamil (injected slowly) were given intravenously. Nine minutes later her cardiac rhythm converted to sinus rhythm. Serum calcium concentration was 2·55 mmol/l (10·2 mg/100 ml). She was advised to stop taking the supplementary calcium and calciferol.

Comment

Verapamil is an antiarrhythmic and antianginal drug used to control supraventricular arrhythmias and the anginal syndrome. Fleckenstein showed that verapamil inhibited the transmembranal influx of calcium into cardiac cells and the release of calcium from the endoplasmic reticulum,[1] causing a negative inotropic effect. This effect may be reversed by increasing the extracellular concentration of calcium or promoting its transmembranal influx with catecholamines.[1 2]

Successful resuscitation of patients poisoned with intravenous calcium has been reported.[3] Verapamil is beneficial in atrial fibrillation because it slows the conduction of impulses through the atrioventricular junction by a direct cellular effect that is unrelated to autonomic influences.[4] Our patient was successfully treated with verapamil when atrial fibrillation recurred after the ingestion of calcium and calciferol in doses that lead to hypercalcaemia. The appearance of atrial fibrillation prompted a dual therapeutic approach aimed at reducing her blood calcium concentration while increasing her blood verapamil concentration. This was based on the assumption that hypercalcaemia antagonises the effect of verapamil on the atrioventricular node and favours re-entrant currents. We suggest that calcium should be used cautiously in patients receiving verapamil. In the event of "calcium poisoning" calcium intake should be stopped, hypercalcaemia treated, and a supplemental dose of verapamil administered if a toxic dose has not been reached.

[1] Fleckenstein A. Specific inhibitors and promoters of calcium action in the excitation contraction coupling of heart muscle and their role in the prevention of production of myocardial lesions. In: Harris P, Opie L, eds. *Calcium and the heart*. London: Academic Press, 1971:135-8.

[2] Grossman A, Furchgott RF. The effects of various drugs on calcium exchange in the isolated guinea pig left auricle. *J Pharmacol Exp Ther* 1964;**145**:162-72.

[3] Perkins CM. Serious verapamil poisoning: treatment with intravenous calcium gluconate. *Br Med J* 1978;ii:1127.

[4] Wit AL, Cranfield PF. Effects of verapamil on the sinoatrial and atrioventricular nodes of the rabbit and the mechanism by which it arrests re-entrant AV nodal tachycardia. *Circ Res* 1974;**35**:413-25.

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Fatal fat embolism after minor trauma

The clinical fat embolism syndrome—classically breathlessness, tachypnoea, fever, petechial rash, tachycardia, and cerebral effects—is well recognised in trauma. The severity of the syndrome is generally directly related to the degree of trauma.[1] Cases are usually seen in accident units and orthopaedic wards. I describe three patients admitted to general medical wards who were found to have severe systemic fat embolism after minor trauma. In each case the diagnosis was made only at necropsy.

Case reports

Case 1—An 80-year-old man was found semiconscious in the toilet of a local lodging house. Chest radiography showed a "snowstorm" appearance, which is a recognised feature of fat embolism (table). He died five hours after admission. At necropsy a bruise 8 cm diameter was noted in the scalp, but there was no skull fracture.

Case 2—A 23-year-old woman with spina bifida suffered a minor injury to her left chest when she was dropped while being carried. She became progressively breathless and was admitted 24 hours later. She died three hours after admission despite 35% oxygen. No fracture was found at necropsy but there was severe kyphoscoliosis with a hypoplastic left lung.

Case 3—A 62-year-old man who had suffered from atypical multiple sclerosis for 15 years fell and hit his head in the bathroom. He became progressively breathless and despite 35% oxygen died five hours after admission to hospital. At necropsy no skull fracture or scalp bruise was identified.

Details of three patients studied

Case No	Age and sex	Clinical features	Evidence of trauma	Necropsy findings
1	80 M	Cyanosis, coma, "snowstorm" on chest radiography	Large scalp bruise, no skull fracture	Severe fat embolism in brain, kidneys, and lungs
2	23 F	Dyspnoea, tachypnoea, tachycardia, cyanosis	History of minor trauma to ribs	Severe fat embolism in brain, kidneys, and lungs
3	62 M	Dyspnoea, tachypnoea, tachycardia, cyanosis	History of fall with minor head injury	Severe fat embolism in brain, kidneys, and lungs

Comment

All three patients had signs or symptoms consistent with a clinical fat embolism syndrome, though in no case was the diagnosis suspected. At necropsy all showed evidence of severe fat embolism, which was the cause of death. Although there was evidence or a good history of trauma, it was apparently minor and there was neither clinical, radiological, nor pathological evidence of any appreciable bony injury. The cases were also characterised by a rather fulminating clinical course.

Most evidence at present supports the original suggestion that the fat is derived from the marrow.[2] Certainly most cases are seen in people with a fracture or multiple fractures of long bones. In one study of 100 cases,[3] 93 showed multiple fractures or a major fracture of the tibia, femur, or pelvis. In only three patients was no bony injury seen, and all had severe soft-tissue injuries. The present three cases suggest that skeletal trauma, even in the absence of a fracture, can produce fat embolism, presumably due to rupture of small vessels in the marrow as a result of forces transmitted along the bones. Such cases, however, would not be expected to have a fulminating course leading to death.

The fat emboli first lodge in the pulmonary capillaries, and it is now established that this may produce subclinical hypoxaemia. In severe systemic fat embolism the arterial oxygen pressure may fall to 50 mm Hg or less.[4 5] All three patients cited here were cyanosed on admission, indicating appreciable hypoxaemia. Pre-existing lung disease may also be associated with a poor prognosis and predispose to systemic fat embolism. It was interesting to find on reviewing these cases that both men had radiological evidence of severe chronic bronchitis and emphysema, confirmed at necropsy, while the other patient had severe kyphoscoliosis and a hypoplastic left lung.

These cases suggest that even minor trauma may precipitate fat embolism, and that people with pre-existing lung disease may be predisposed to severe effects.

I thank Dr R A Fraser for allowing me to include one of his patients in this report.

[1] Sevitt S. *Fat embolism*. London: Butterworths, 1962.

[2] Watson AJ. Genesis of fat emboli. *J Clin Pathol* 1970;**23**, suppl 4:132-42.

[3] Gurd AL, Wilson RI. The fat embolism syndrome. *J Bone Joint Surg (Br)* 1974;**56B**:408-16.

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[5] Prys-Roberts C, Greenbaum R, Nunn JF, Kelman GR. Disturbances of pulmonary function in patients with fat embolism. *J Clin Pathol* 1970; **23**, suppl 4:143-9.

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