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CASE REPORT

Fatal fat embolism following an isolated vertebral fracture

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The authors report an unusual case of a fat embolism following an isolated lumbar vertebra wedge fracture. A 66-year-old gentleman sustained a lumbar wedge fracture following a fall. During routine mobilisation some days later he collapsed suddenly and became unresponsive. Shortly after, he suffered a fatal cardiac arrest. A post-mortem was performed which attributed the cause of death to fat embolism. Most previously reported cases of fat embolism are associated with long bone fractures and major bony surgery. No apparent case of fatal fat embolism following an isolated vertebral fracture has previously been described.

Keywords : fat embolism ; vertebral fracture.

CASE REPORT

A 66-year old gentleman was admitted to our hospital after a fall he sustained while crossing the road. He claimed to have fallen backwards, landing heavily onto his lower back. He complained of severe lower back pain, which was worse when he attempted to mobilise.

He was known to have a past medical history of non-insulin dependent diabetes and ischaemic heart disease, but denied any previous history of back pain.

On clinical examination he was found to be locally tender posteriorly over the L1 lumbar vertebra, with no obvious bruising, swelling or step deformity. Neurological examination of the lower limbs was normal. Plain radiographs of the lumbar spine showed a wedge type fracture of the L1 vertebra, with an approximate loss of 50% in anterior height (fig 1). A subsequent CT scan confirmed the fracture configuration to be stable (fig 2). The patient was fully mobilised in a thoracolumbar support orthosis, no thromboprophylaxis was administered as his level of mobilisation was satisfactory.

During mobilisation with the physiotherapist four days later, the patient became acutely confused and collapsed. He was conscious, hypotensive, tachycardic and dyspnoeic. Biochemically, he was noted to be in renal failure (Na+ 130 mmol/l, K+ 6.2 mmol/l, Creat 387 mg/dl, Urea 23.8 mmol/l), arterial blood gas analysis confirmed a respiratory acidosis (pH 7.2) and hypoxia (pO2 68.9 mmHg).

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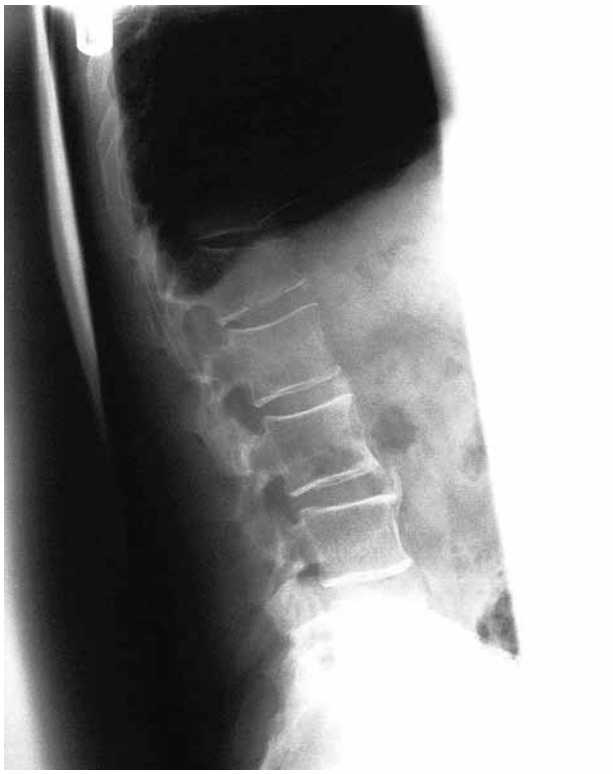

Fig. 1. – Lateral lumbar radiograph illustrating stable anterior wedge fracture of L1.

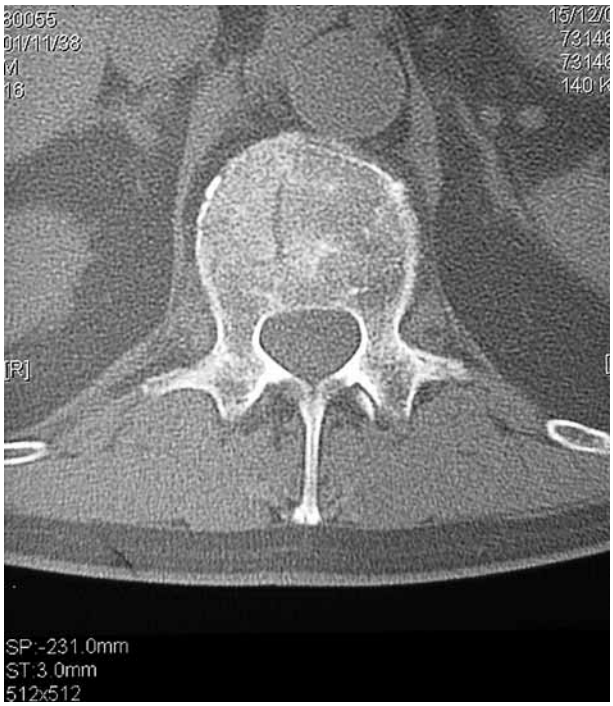

Fig. 2. – Axial CT scan showing vertebral body fracture with no involvement of the posterior elements.

ECG changes revealed a new onset right bundle branch block and a provisional diagnosis of pulmonary embolism was given. He was transferred to the coronary care unit for closer monitoring. An echocardiogram failed to demonstrate the presence of a PE and the cardiac enzymes were normal ; despite this he was anticoagulated with unfractionated heparin. The patient continued to generally deteriorate, he had a mildly elevated temperature (37.3°C), raised CRP (400 mg/l), deteriorating renal failure, hypotension and increasing confusion and agitation with no evidence of any focus of infection, and he was commenced on Cefuroxime. Given the history of trauma, a CT scan of the brain was performed and was normal. Three days later he suffered a cardiac arrest. Resuscitation attempts were unsuccessful and the patient died, seven days post admission.

A post-mortem examination was performed. Gross visualisation of the brain revealed multiple petechial haemorrhages present throughout the white and grey matter. Frozen sections of the brain revealed fat droplets within the cerebral vasculature. The cardiac findings were of an acute myocardial infarct secondary to thrombus within underlying coronary artery disease with no evidence of a septal defect. Respiratory system was normal. The right kidney was pale and congested. These findings were concluded to be consistent with fat embolism syndrome.

DISCUSSION

Fat embolism syndrome is a well recognised complication following trauma and bone surgery. It had been repeatedly documented following the insertion of an intramedullary device into the medullary cavity of a long bone, cemented fixation of a femoral stem following total hip arthroplasty (*1, 4, 8*) and less frequently following spinal surgery (*9, 10*).

Fat embolisation is thought to be often subclinical, but is also a recognised cause of sudden

intraoperative and perioperative death. The incidence in patients with fractures is reported to range from 0.25% to 1.25% (*7*), with reported mortality between 10% and 20% (*3*).

Symptoms may be immediate or delayed up to three days after trauma. The diagnosis of fat embolism syndrome is based on the patient history, supported by abnormal clinical findings in the cardiac, pulmonary, cerebral and cutaneous systems. As in our case, hypotension associated with tachycardia, shortness of breath and acute confusion are well recognised clinical findings.

It is thought to occur when fat emboli originating from the medullary cavity within bone enter the systemic circulation and via the pulmonary tree embolise, most commonly to the brain and kidneys. The underlying pathological mechanisms that have been proposed include both a mechanical and biochemical theory. Pell *et al* (*6*) were strong proponents of the mechanical theory. They demonstrated by transoesophageal echocardiography that the insertion of an intramedullary guide wire and intramedullary nailing of a femur resulted in echogenic material passing to the right side of the heart. This was associated with reduction in oxygen saturations and elevation in right heart pressures. Takahashi *et al* (*10*) demonstrated that probing the vertebral body via the pedicle was sufficient to produce fine embolic events to the right atrium. The biochemical theory suggests the possibility of free fatty acid toxicity. The release of catecholamines associated with trauma promotes the release of free fatty acids, C-reactive protein may cause chylomicrons to coalesce and form fat globules (*5*), and this is postulated to activate a systemic immune response.

Unfortunately, no pathognomonic tests are available to aid in the diagnosis of this syndrome. A high index of suspicion in combination with appropriate clinical signs and symptoms is all the treating physician can rely on, more often it being a diagnosis of exclusion. Treatment options are aimed at supportive measures ; maintaining oxygenation, splintage of fractures and correction of electrolyte disturbances. There is no evidence to support the use of adjuvant treatments such as steroids or heparin either for treatment or prophylaxis.

Although previously described in a lung transplant recipient with multiple vertebral body compression fractures (*2*), a literature search has revealed no previous accounts of fat embolism occurring following an isolated vertebral fracture in a patient who has not undergone bony surgery. More recently there have been a number of reports that have focused on bone marrow embolisation following vertebroplasty which is becoming an increasingly frequent procedure (*9*). This unusual diagnosis was made retrospectively given the post-mortem results and documented clinical course.

We suggest, therefore, that fat embolism may occur even following an apparently innocuous isolated fracture, and not necessarily following major trauma or surgery.

REFERENCES

1. **Aoki H, Soma K, Shindo M *et al.*** Evaluation of potential fat emboli during placement of intramedullary nails after orthopaedic fractures. *Chest* 1998 ; 113 : 178-181.
2. **Day JD, Walden SM, Stuart SR *et al.*** Fatal fat embolism after numerous vertebral body compression fractures in a lung transplant recipient. *J Heart Lung Transplant* 1994 ; 13 : 785-790.
3. **Fabian TC.** Unravelling the fat embolism syndrome. *N Engl J Med* 1993 ; 329 : 961-963.
4. **Herndon JH, Bechtol CO, Crickenberger DP.** Fat embolism during total hip replacement : a prospective study. *J Bone Joint Surg* 1974 ; 56-A : 1350-1362.
5. **Hulman G.** Pathogenesis of non-traumatic fat embolism. *Lancet* 1988 ; 1 : 1366-1367.
6. **Pell ACH, Hughes D, Keating J *et al.*** Fulminating fat embolism syndrome caused by paradoxical embolism through a patent foramen ovale. *N Engl J Med* 1993 ; 329 : 926-929.
7. **Peltier LF.** Fat embolism. A current concept. *Clin Orthop* 1969 ; 66 : 241-253.
8. **Pitto RP, Koessler M, Kuehle JW.** Comparison of fixation of the femoral component without cement and fixation with use of a bone-vacuum cementing technique for the prevention of fat embolism during total hip arthroplasty : a prospective, randomized clinical trial. *J Bone Joint Surg* 1999 ; 81-A : 831- 843.
9. **Syed MI, Jans, Patel NA *et al.*** Fatal fat embolism after vertebroplasty : identification of the high-risk patient. *Am J Neurorad* 2006 ; 27 : 343-345.
10. **Takahashi S. Kitagawa H, Ishii T.** Intraoperative pulmonary embolism during spinal instrumentation surgery. A prospective study using transoesophageal echocardiography. *J Bone Joint Surg* 2003 ; 85-B : 90-94.