Lesson of the Week

Unrecognised femoral fractures in patients with paraplegia due to multiple sclerosis

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Patients with paraplegia may, owing to disuse of the legs, develop osteoporosis of the long bones of the leg and occasionally suffer fractures as a consequence despite their relative immobility. We report on two patients with paraplegia due to multiple sclerosis who sustained femoral fractures without any history of trauma and with minimal physical signs. This resulted in late diagnosis and inappropriate treatment in both cases. This complication has not to our knowledge been previously reported in patients with multiple sclerosis.

Case reports

Case 1—A 37 year old woman who had had paraplegia and complete sensory loss in the legs for three years due to multiple sclerosis was admitted with acute dyspnoea. She had cyanosis, tachypnoea (respiratory rate 28 breaths/minute), tachycardia (pulse rate 120 beats/minute), hypotension (systolic blood pressure 90 mm Hg), appreciable deterioration of her neurological signs, and crepitations in both lung mid-zones. A chest radiograph showed generalised fine mottling. Bronchopneumonia with possible bacteraemia was diagnosed and treated with oxygen, intravenous fluids, antibiotics, and hydrocortisone with considerable improvement. Blood cultures did not yield any growth. Three days after admission her right leg was noted to be swollen, warm, and slightly dusky in appearance but not tender. A deep venous thrombosis was thought to be an obvious possibility in a bedridden patient, and heparin was started. Nine days later her right leg remained swollen and there was bruising of the right thigh. Radiographs showed bilateral supracondylar femoral fractures, comminuted on the right. There had been no history of trauma. Anticoagulation was stopped, and she was treated with external splinting of the right leg. A second chest radiograph showed that the chest had cleared within 24 hours after the start of treatment and the picture was later thought to be highly consistent with fat embolism.

Case 2—A 63 year old woman who had paraplegia due to multiple sclerosis, confining her to a wheelchair, presented with a two day history of a painless, swollen left leg. There was no history of trauma. She was feverish, and the left leg was swollen and warm, though there was no tenderness or discoloration. She was presumed to have a deep venous thrombosis, and treatment with anticoagulants was started. The next day a bruise developed on her left thigh and radiographs showed a displaced fracture of the femoral shaft. The femur appeared osteoporotic. Anticoagulants were stopped, and she was referred to the orthopaedic surgeons.

Fractures of the leg may occur in patients with paraplegia due to multiple sclerosis without any history of trauma and with few physical signs

Comment

These two patients had several features in common. Both had paraplegia and sensory loss due to multiple sclerosis, both sustained femoral fractures with no history of trauma, and both were initially treated for deep venous thrombosis, which was thought to be the most likely cause of unilateral swelling of the leg in an immobile patient. In each case the fracture was not suspected and radiographs were obtained only after bruising had been seen.

Fractures of the leg are a recognised complication in patients with paraplegia, occurring in 4-8% of patients with paraplegia due to spinal cord injury.^{1,2} Supracondylar or femoral shaft fractures, as in our two patients, are most common² and usually occur in osteoporotic bones without known trauma or after trivial injury. Osteoporosis is known to occur in patients with paraplegia due to multiple sclerosis.³

These two patients presented within a few months of each other at different hospitals, suggesting that this problem may occur more often than is generally realised. Both cases were initially misdiagnosed as the usual symptoms and signs of fracture were not present. In patients with paraplegia who develop swelling of a leg the possibility of a fracture should be borne in mind even when there is no history of trauma, and, if a patient presents with acute respiratory distress, pulmonary fat embolism should be considered. Fat embolism is particularly likely to develop after fracture of atrophic bones, as these contain more fat than normal, and it may be mistaken for bronchopneumonia.⁴ Finally, these cases emphasise the importance of confirming a clinical diagnosis of deep venous thrombosis objectively before starting treatment with anticoagulants.⁵

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References

- Freehafer AA, Mast WA. Lower extremity fractures in patients with spinal cord injury. *J Bone Joint Surg* 1965;47A:683-94.
- Ragnarsson KT, Sell GH. Lower extremity fractures after spinal cord injury: a retrospective study. *Arch Phys Med Rehabil* 1981;62:418-23.
- Dechamps P, Potvliege R. L'ostéoporose dans la sclérose en plaques. *Acta Neurol Belg* 1972;72:215-25.
- Rains AJH, Capper WM. *Bailey and Love's short practice of surgery*. London: Lewis, 1971:130.
- Ramsay LE. Impact of venography on the diagnosis and management of deep vein thrombosis. *Br Med J* 1983;286:698-9.

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