SERRATIA OSTEOMYELITIS CAUSING NEUROLOGICAL DETERIORATION AFTER SPINE FRACTURE

A REPORT OF TWO CASES

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We report two cases of *Serratia marcescens* infection at the sites of spinal fractures and emphasise the fact that neurological deterioration soon after spinal fracture may be due to acute vertebral osteomyelitis.

The spontaneous appearance of haematogenous osteomyelitis in a fractured vertebra appears to be a very uncommon event (Atsatt 1939; Milgram and Romine 1982; Eismont et al. 1987). *Serratia marcescens* is a rare cause of vertebral osteomyelitis (Fishbach, Rosenblatt and Dahlgren 1973; Mintz and Mollett 1975). We present two cases in which an acute *serratia* osteomyelitis developed in recently fractured vertebrae and discuss the implications.

CASE REPORTS

Case 1. A 40-year-old building worker fell from a height of six metres, and sustained a burst fracture of the body of the second lumbar vertebra (Fig. 1). On admission, 10 days after the accident, he had mild weakness of the knee and toe extensors in the left leg, normal peri-anal tone and sensation, and 700 ml of residual urine.

On the following day he developed a spiking fever and chills, and *Serratia marcescens* was cultured from blood and urine samples. The urinary sepsis was temporarily controlled by bladder drainage and parenteral antibiotic therapy, but, over a period of one week, he developed progressive paraplegia with loss of anal sphincter control. At an urgent operation for anterior decompression the appearance of the fractured body of L2 was different from that expected in a three-week-old

fracture. The fragments were free and easily resectable, being bathed in a clear fluid, and the disc material in the L1/L2 disc space appeared to be partially digested. Culture of material from this area grew *Serratia marcescens* which had the same antibiotic sensitivity as that from the urine and blood cultures. Anterior stabilisation was achieved with iliac and rib isografts (Fig. 2), and six weeks later a posterior stabilisation was performed with Harrington distraction rods and tension band wiring between L1 and L3 (Fig. 3). The patient made a slow, but uneventful and complete neurological recovery.

Case 2. A 39-year-old woman was struck by a lawnmower blade which penetrated her abdominal wall in the right epigastrium and lodged firmly in the bodies of L2 and L3 vertebrae. The blade was removed with some difficulty by the general surgeon who performed the explorative laparotomy. Lacerations were found in the pancreas, superior mesenteric artery, and inferior vena cava. The vessels were repaired, and hemipancreatectomy and splenectomy performed.

The patient subsequently developed a biliary fistula and septicaemia. *Serratia marcescens* was cultured from the fistula and from blood samples, and the patient was treated by local washouts and appropriate antibiotics. Fistulography showed no evidence of involvement of the lumbar spine (Fig. 4). The fistula closed slowly but a low grade fever persisted.

Two months after her injury the patient developed severe low back pain, more pyrexia and signs of bilateral irritation of lumbar nerve roots. Radiographs now showed L2/L3 discitis, and scintigraphy confirmed an osteomyelitic lesion in the body of L3. Computerised tomography showed a prevertebral mass in the area of the head of the pancreas and myelography revealed mild extradural compression at L2/L3. A diagnosis of vertebral osteomyelitis was made and, based on the sensi-

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© 1989 British Editorial Society of Bone and Joint Surgery
0301-620X/89/2076 \$2.00
J Bone Joint Surg [Br] 1989;71-B:256-8.

Fig. 1

Fig. 2

Fig. 3

Figure 1 - Lateral radiograph showing a typical burst fracture of L2 with retropulsion of the posterior superior part of the vertebral body. Figure 2 - Lateral radiograph after L2 decompression showing subtotal vertebrectomy and the strut graft in place. Figure 3 - Radiograph after Harrington instrumentation and interspinous wiring.

Fig. 4

Fig. 5

Figure 4 - Case 2. Biliary sinogram showing lack of communication with the vertebral column. Figure 5 - Five years later, the lateral radiograph shows solid fusion from D11 to L3.

tivities found during the *serratia* septicaemia, the same antibiotics were given. There was no clinical change except that mild paraparesis developed.

Computerised tomography, three weeks after the initial scan, now showed obvious destruction of the body of L2 and disruption of the L2/L3 interspace. At anterior decompression, the paravertebral tissues from L2 to L3 were found to be acutely inflamed with fibrin exudate and granulation tissue; cultures grew *Serratia marcescens*. Discectomy was performed at D12/L1, L1/L2 and L2/L3. There was slow neurological recovery with resolution of fever, decrease in sedimentation rate, and radiological evidence of fusion (Fig. 5).

DISCUSSION

These two cases are interesting for two main reasons: first, the delayed onset of neurological deficit after a vertebral fracture, a familiar clinical presentation, was here associated with osteomyelitis and abscess formation in a recently damaged spine. We could find only three previously reported instances of osteomyelitis after vertebral fracture (Atsatt 1939; Milgram and Romine 1982; Eismont et al. 1987). In these cases the organisms were *Staphylococcus aureus* or *Pseudomonas aeruginosa*, and none had presented with acute neurological deterioration.

Secondly, vertebral infection by *serratia*, which has been reported as a spontaneous event in intravenous drug addicts (Fishbach et al. 1973; Mintz and Mollett 1975), occurred here in previously healthy patients. The route of infection in addicts has been reported to be by contaminated implements used by immune-compromised individuals (Knutson et al. 1982; Mahan and Kalish 1982; Watanakunakorn 1986). In our cases the infection may have been acquired by haematogenous seeding into the locus minoris resistens, the damaged vertebrae (Wilkowske et al. 1970). The fact that a normally commensal organism such as *Serratia marcescens* was involved could have been an indicator of a degree of immune depression in these seriously ill patients.

Neurological deterioration soon after vertebral fracture is usually attributed to oedema of the cord, and

the appearance of progressive neurological signs several days later is commonly ascribed to displacement of fracture fragments or increasing deformity. Our cases show that vertebral osteomyelitis should be considered as an additional cause of neurological deterioration after vertebral trauma. As reported by Eismont et al. (1983), anterior decompression led to favourable results.

There have been increasing reports of musculoskeletal infection caused by *Serratia marcescens* (Burton and Nagel 1972; Kelly, Wilkowske and Washington 1973). Our cases highlight a hitherto undescribed and potentially serious consequence of such infection in patients with a spinal fracture.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

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