Direct erosion of lumbar spine by an abdominal aortic aneurysm, resulting in paraparesis: unusual presentation. Case report

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Abdominal aortic aneurysm is a condition affecting nearly 4% of the elderly population.¹ It has a potential for producing a wide range of symptoms, including abdominal pain and back pain.² The latter is particularly difficult to interpret in patients with chronic rheumatological conditions, and delayed diagnosis may be associated with a poor outcome. We present a patient with rheumatoid arthritis and chronic low back pain, who developed bilateral leg weakness and hesitancy of micturition, due to an abdominal aortic aneurysm invading the spine.

Keywords: abdominal aortic aneurysm; vertebral erosion; paraparesis; rheumatoid arthritis

Case report

A 69-year-old female with chronic seropositive erosive rheumatoid arthritis of 28 years duration was admitted with a 4 year history of pain in the lower back, becoming more severe and radiating to the right thigh, a feeling of abdominal distension and intermittent constipation.

She had been seen by a general surgeon 2 years previously because of diarrhoea and low abdominal and back pain, thought to be due to diverticular disease. There was a past history of peptic ulcer, and she had developed diabetes mellitus 4 years previously. She had taken prednisolone 5 mg daily for the previous 28 years.

On examination she was very frail, with well established chronic rheumatoid deformities, but with no evidence of active inflammatory disease. She walked with difficulty, using one stick, but neurological examination was normal. She was tender in both iliac fossae and the suprapubic region, but no masses were palpable, and was also very tender over the third, fourth and fifth lumbar vertebrae. A provisional diagnosis of osteoporotic vertebral collapse was made.

A lumbar spine radiograph revealed an abnormal L5 vertebral body. The superior end plate was ill-defined, with apparent erosion of the inferolateral aspect of the lateral part of the body.

Repeated blood cultures were negative, but, a technetium bone scan showed increased uptake involving the inferior aspect of the body of L4 and the body of L5 suggestive of an infective process.

In view of the bone scan findings, she was commenced on intravenous antibiotics but her condition continued to deteriorate, and she required increased doses of opioids for pain. She developed recurrent abdominal pain and constipation and was referred for a general surgical opinion, as it was thought that she had developed intestinal obstruction.

About a month after admission, she developed bilateral

lower limb weakness, faecal incontinence and hesitancy of micturition over 2 days. Neurological examination revealed decreased power in both lower limbs. The limb reflexes were present but reduced, and the plantar responses were equivocal. There was decreased pinprick sensation in the perineal region and rectal examination revealed decreased sphincter tone.

An urgent myelogram revealed complete spinal canal obstruction at the L4–5 level, and she was promptly transferred to a neurosurgical unit, where an urgent exploration and decompression of the L4–5 region was performed. At operation it was noted that the bodies of the fourth and fifth lumbar vertebrae were 'crumbly and eaten away'. No convincing evidence of tumour or infection was found. The histology showed no evidence of malignancy or infection. Despite operative intervention the patient failed to improve.

A further myelogram 10 days after operation revealed extensive osteolysis of L4–5 around the disc space, with moderate retrolisthesis of L4. Computed tomography (CT) revealed diffuse bone destruction of the bodies of L4 and L5 by a large anterior soft tissue mass, with bilateral paraspinal extension, particularly on the right side. In addition there appeared to be a large aortic aneurysm immediately anterior to the lower vertebral bodies, extending down to the right common iliac artery. The distinction between aneurysm and soft tissue mass immediately posterior and lateral to it was not, however, clearly demonstrated on the non-contrast examination. It was thought that the underlying pathology was a metastatic lesion causing destruction of the bodies of L4 and L5 with associated paraspinal masses, and that the aneurysm was an incidental finding.

CT was repeated a week later with contrast. The main abnormality was a large paravertebral mass present bilaterally and almost symmetrically from L3 to S1. Anteriorly this incorporated a grossly enlarged terminal portion of the abdominal aorta, with no obvious wall separating the lumen from the retroperitoneal fat or the abscess posteriorly.

Bilateral hydronephrosis was noted, more marked on the right side.

The conclusion was that the appearances were those of osteomyelitis of the fourth and fifth lumbar vertebrae, with a paravertebral abscess and a mycotic fusiform aneurysm of the terminal abdominal aorta, complicated by hydronephrosis. At operation a large aortic aneurysm eroding the vertebrae was found, but no evidence of infection was seen. She was treated by resection of the aortic aneurysm with a right axillobifemoral graft. At the same operation she also had acrylic cement put into the bodies of L4 and L5. She made a slow recovery and was subsequently able to weight bear and walk a few steps with a walking frame.

Discussion

Several unusual manifestations of abdominal aortic aneurysm have been described. These include chronic contained rupture, inflammatory aneurysm, aortovenous fistula, 'mycotic' or infected aneurysm, aortoenteric fistula and atheroembolism,² recurrent spinal cord ischaemia,³ paraparesis due to an anterior spinal artery syndrome as a result of an infrarenal aortic aneurysm,⁴ and direct erosion of the vertebral body and spinal cord compression by an infected aneurysm.⁵

Although spinal cord ischaemia is a recognised complication of an abdominal aortic aneurysm, such a presentation is extremely rare and unless one is alert to this fact, it can be easily missed.

Desai *et al*³ described recurrent spinal cord ischaemia in a 75-year-old woman with a known abdominal aortic aneurysm, who 2 years later developed an acute episode, resulting in paraparesis. She was treated

conservatively and the paraparesis partially resolved, only for further deterioration to occur after 8 months. She died 3 years later due to rupture of the aneurysm. It was postulated that either she had had repeated embolisation from thrombosis in the aneurysm to the radicular arteries, or that the mouths of the regional radicular branches were progressively occluded due to the enlarging thrombus.

Our patient was different in that she had a destructive process in the vertebrae, which was initially thought to be due to osteomyelitis, and it was only the lack of response to antibiotics that prompted further investigations to be done. Once the aneurysm had been resected she made a slow but partial neurological recovery.

We can only conclude that the destructive process seen in L4 and L5 was caused by direct pressure from the aortic aneurysm.

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