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Case Report

Double spinal cord injury in a patient with ankylosing spondylitis

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Ankylosing spondylitis patients are more prone to spinal fractures and these fractures commonly result in mobile nonunion. We report a patient with a 30-year history of ankylosing spondylitis who sustained double spinal cord injuries following minor trauma. The first injury occurred at the lumbar level due to pseudoarthrosis of an old fracture, and the second at the thoracic level following cardiopulmonary arrest and an episode of hypotension. The possible mechanisms of the injuries are discussed and maintaining normal blood pressure in these patients is emphasized.

Keywords: spinal cord injury; ankylosing spondylitis; spinal cord infarction; spinal fractures

Introduction

Ankylosing spondylitis (AS) has a prevalance of 1 per 1000 in the general population and primarily involves the vertebral column.[1] Spinal rigidity due to long-standing AS renders the patient susceptible to vertebral trauma, so that even minor trauma may cause fractures.[2–9] There are only a few reports in the literature describing AS patients with spinal cord injury (SCI) without vertebral fracture, and all of these report SCI secondary to either post-traumatic epidural hematoma[8–10] or cauda equina syndrome due to vertebral scalloping.[11–13] In this paper, we describe a patient with long-standing AS who sustained thoracic and lumbar SCIs following minor trauma. The trauma caused neither vertebral fracture nor epidural hematoma at the thoracic level, but aggravated spinal stenosis due to an old unstable fracture at the lumbar level.

Case report

A 66-year-old man with a 30-year history of AS had slipped and fallen on a patch of ice. He was transferred to the emergency room (ER) immediately. On initial examination, he was conscious, able to move his arms and legs but not his feet, had symmetrical positive deep tendon reflexes, but had impaired sensation in both feet. Plain radiographs of the spine were consistent with severe AS and L3 fracture. His arterial blood pressure was 60/30 mmHg and his heart rate was 92/min. Shortly after arrival at the ER, the patient suffered cardiopulmonary arrest. He responded to resuscitation and was transferred to the intensive care unit (ICU) where he was monitored and underwent a diagnostic workup. His arterial blood pressure stayed below normal and his central venous pressure remained below 5 cmH_2O for about 12 h. ECG, chest X-Ray and cranial computed tomography (CT) were normal. When the patient awoke and was in a stable condition, he could not feel or move his legs. His neurological examination was consistent with a T9 complete paraplegia. He then underwent thoracic and lumbar CT, which revealed normal spinal and paraspinal structures at the thoracic level, and bone destruction and severe spinal stenosis at L2–3 levels. The patient's past medical history included a lumbar vertebral fracture following a fall at home 15 years earlier. He had refused surgical stabilization at that time and recovered completely after 3 months of bed rest. He reported having no neurological symptoms or signs before his second fall. No surgical intervention was proposed and the patient was transferred to the authors' facility where he had comprehensive rehabilitation including physical, occupational and respiratory therapy. The patient was placed on an intermittent catheterization program every 6 h, which worked very well, and his bowel program was carried out with daily digital stimulation. In the sixth week after admission, the patient complained of abdominal cramps and back pain, and experienced autonomic dysreflexia.

Abdominopelvic ultrasonograpy (USG) was normal except for aortic calcification. The pain was relieved with paracetamol and TENS application. Urodynamic studies revealed a hyporeflexic, low-pressure bladder with a maximum cystometric capacity of 550 ml and no detrussor-sphincter dyssynergia. Pain began to radiate to his lower extremities and gained a neurogenic character. The patient underwent a magnetic resonance imaging (MRI) in the second month following injury, which revealed myelomalacia secondary to

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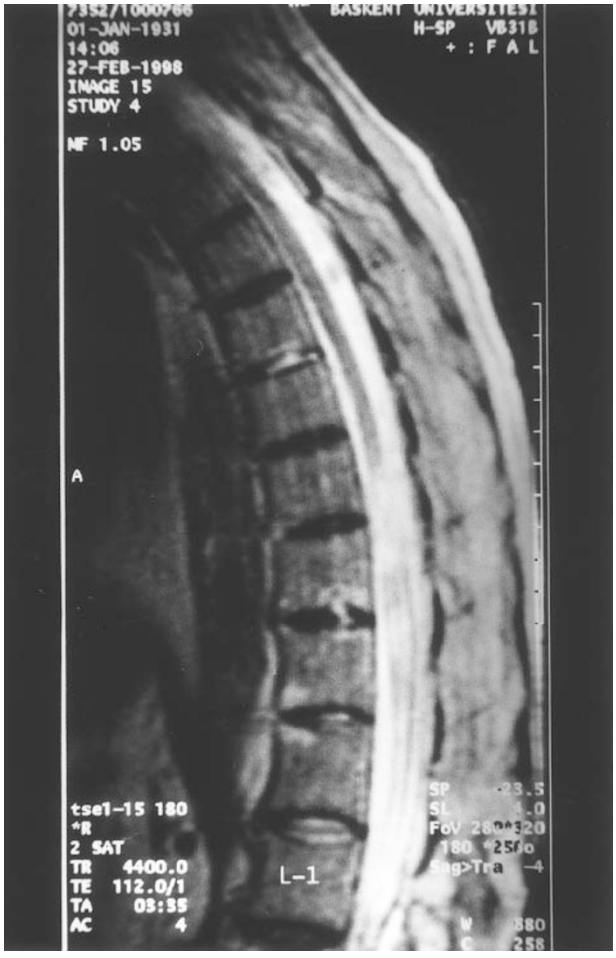

Figure 1 Sagital (TR: 4400 msec, TE: 112 msec) T2 weighted MR images demonstrate hyperintense lesions T9–12 levels consistent with spinal cord infarction

spinal cord infarction at levels T9–11 (Figure 1) and severe destruction of the L2,3 vertebrae, including neural arcs and bodies with fluid collection and soft tissue component causing spinal stenosis. Fine needle aspiration biopsy and culture confirmed the absence of osteomyelitis. The patient was discharged at a level of modified independence in activities of daily living, with a functional independence measure (FIM) score of 84. Follow-up physical examination in the third month after discharge revealed no change in the patient's level of neurologic function.

Discussion

It is well-documented that AS patients are more prone to spinal injury due to biomechanical changes in the vertebral column secondary to ligament calcification and osteoporosis.[2–9] Most fractures occur in the cervical spine, and in most cases the type of injury is minor hyperextension trauma.[6,14] Cervical spinal fractures are said to be three times more common in AS patients than in the general population.[2–4] Another characteristic feature of spinal lesions in AS patients is discovertebral destruction or pseudoarthrosis, as was seen in our patient, in which the development of fibrous connective tissue at the site of fracture results in mobile nonunion.[2,5,15,16] It is suggested that the radiological appearance of pseudoarthrosis can be very similar to discovertebral infection, and recognition of this entity in AS patients may eliminate the need for needle biopsy or surgical exploration.[16] On MRI we detected severe bone destruction, and soft tissue component causing marked spinal stenosis in our patient at L2,3. We postulated that the patient's previous fracture resulted in nonunion or pseudoarthrosis at the level of L2,3 and that his second fall caused a cauda equina syndrome due to the progression of stenosis, as he had described motor and sensory loss only in the distal portions of his lower extremities prior to cardiac arrest. Pederson *et al* point out that the ankylosed spine fractures like long bone, and the fracture is highly unstable so that even minor manipulation may cause cord damage.[6] Weinstein *et al* stated that AS patients with spinal pain should be handled carefully during transfer and transport because of the risk of fracture or dislocation at a previous fracture site.[16]

The second SCI in our patient was at T9, and was clearly detected as cord infarction on MRI performed in the second month after injury. The absence of any bone lesion and/or hematoma at that level on both CT and MRI indicated that the problem was vascular insufficiency. The fact that motor function and positive deep tendon reflexes were present after his fall and before cardiac arrest suggested that the SCI was a consequence of vascular insufficiency during systemic hypotension and cardiopulmonary arrest. We are uncertain whether the ankylosed spine played a role in this injury, but it is possible that traction of the spinal cord within the rigid ankylosed spinal column during resuscitation procedure or at the moment of fall might have caused damage, or at least facilitated vascular insufficiency at that level where the blood supply is poor.

The clinical presentation of this patient could also be explained by a single lesion at level T9 and prolonged spinal shock, as his superficial anal, bulbocavernous, and deep tendon reflexes are still absent and are associated with flaccid lower extremity muscles and bladder. However, the presence of severe bone destruction and spinal stenosis at Level L2,3 in addition to the physical examination findings in the ER just after the fall, suggest that a double SCI occurred, first at the lumbar and later at the thoracic level.

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