Gastric outlet following acid ingestion


Pancreatitis, which made the pancreas susceptible to inflammation. It is also possible that hyperlipidaemia due to parenteral nutrition contributed to pancreatitis. The disparity between the clinical symptoms and severity of pancreatitis is also well known. The onset of disseminated intravascular coagulopathy in this patient is a known complication of severe pancreatitis.7,8

Paraplegia due to thoracic disc herniation

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Summary

Disc herniation at the thoracic spine level is more common than generally thought. Localisation of pain may be vague and may erroneously point to cardiopulmonary, gastrointestinal, genito-urinary or even psychiatric disease. Magnetic resonance imaging is the investigation of choice, especially if spinal cord compression is suspected.

Keywords: disc herniation, paraplegia, spinal cord compression, magnetic resonance imaging

Disc herniation at the thoracic spine level has been thought to be an uncommon problem until recently. Although in life less than 1% incidence has been quoted, autopsy series show a higher incidence of thoracic disc herniations of between 7 and 15%. Most series of patients with dorsal disc herniations suggested incidences under 2% of all operated discs. It has been postulated that a number of factors at the thoracic spine level are responsible for the low overall incidence of thoracic disc herniation, for example, the relatively limited mobility of the thoracic spine secondary to the small sizes of the thoracic discs, the relative restraint of the thoracic spine because of the ribs and sternum, and the orientation of the facet joints in the coronal plane. The variable clinical manifestations can obscure the diagnosis, and until recently the lack of radiographic sensitivity and specificity for assessment of complaints referable to the thoracic spine had compounded the issue. We wish to report on a disc herniation at the thoracic spine level in a patient whose presenting features were vague or confusing enough to suggest an initial diagnosis of a functional problem by a number of physicians, including casualty doctors, a consultant neurologist, and a consultant radiologist, until paraplegia developed due to cord compression.
Case report

The patient was a 36-year-old woman working as a care assistant. She first presented to the Casualty department with a 12-month history of back pain radiating down mainly the left leg but also occasionally the right leg which had been gradually getting worse for the previous six weeks. She also gave a history of paraesthesia and felt that her problems had been aggravated by lifting a patient in a nursing home. The pain was unrelieved by analgesics and nonsteroidal anti-inflammatory agents prescribed by her general practitioner.

At this stage, examination revealed paravertebral spine muscle spasm, stiffness of movements, but normal power, sensation, and reflexes including a plantar reflex response in the feet. She was, however, tender in the thoraco-lumbar region. Radiographs of the lumbar and lumbosacral spine were normal. No clear diagnosis could be made but for her pain she was prescribed analgesics, low-dose diazepam and advised to have physiotherapy with plans to review her as necessary.

When reviewed two weeks later she was mobile, and relatively painfree; examination was normal and she was discharged. However, in just less than a month she had to be admitted to the orthopaedic unit with a two-week history of worsening back pain with pain down the left side. This caused her difficulties in mobilising and sleep; she had occasional falls but no bowel or urinary symptoms. Examination at this time revealed spastic lower limbs with an unusual posture, in particular, ankles and toes looked as if she were having a cramp, and extensor plantar response was queried. She had no sensory abnormalities and radiographs were again normal. She was treated with pelvic traction.

Three days later she experienced increased pain and paraesthesia in the left leg and also some paraesthesia in the right foot. Her reflexes were again found to be normal but there was once again equivocal response to plantar responses bilaterally. Straight leg raising was limited to about 30° on both sides, sensory function was normal. Ten days later she was examined by a consultant neurologist who found her difficult to assess and suggested a functional disorder. As a result she was assessed by a psychiatrist who felt that there were some underlying psychological problems but still felt that it was necessary to exclude organic spinal pathology. She was therefore sent for magnetic resonance imaging (MRI) but could not co-operate with the examination, and no diagnostic images could be obtained. The radiologist undertaking this examination also felt that there was a functional problem and considered that sedation was not warranted to undertake further assessment and imaging.

Ten days later she was assessed by a second neurologist who felt that there were organic problems. By this time the patient had bladder incontinence, increased tone in the lower limbs and extensor plantar reflexes. Sensory loss up to the level of T10 was also detected. A repeat MRI scan was therefore undertaken under sedation and a huge calcified disc was demonstrated at T6-T7 level causing marked cord compression and also myelomalacic changes in the spinal cord (figure). By this time she had all the neurological findings mentioned above plus absent anal reflex, decreased anal tone and mass movement of lower limbs to touch, pin prick or other sensory stimuli. She was incontinent of urine and unable to bear weight. After discussion with the neurosurgeon she opted for decompression surgery by the posterior approach. The operation itself was difficult but the spinal cord was freed from the projecting disc and was noted to be pulsatile. The spastic paraplegia did not improve, however, and the patient has been rehabilitated for wheelchair independence.

Discussion

There are a number of unusual features in this patient’s case history, including prolonged nonspecific complaints. Initially the localisation of the pain was vague enough for the casualty doctor to take X-rays of the lumbar and lumbosacral spine but not the thoracic spine, the actual site of her pathology. At presentation her symptoms and findings were also confusing enough for a number of physicians to question her complaints and, indeed, a functional disorder was suspected. Unfortunately, by the time correct diagnosis was reached she had developed a spastic paraplegia from a combination of spinal cord compression and associated ischaemic changes and it is unlikely that she will recover any meaningful function of her lower limbs.
Although thoracic disc herniations have until recently been thought to be relatively rare, with improved imaging techniques, including MRI, more cases are being diagnosed. The incidence of thoracic disc herniations is also higher than previously thought. In a recent report 48 patients were assessed for possible neoplasia in the dorsal spine and nearly 15% incidental thoracic disc prolapses were detected. The age range for thoracic disc prolapses is variable, most patients being in the fourth to sixth decade with equal sex distribution. Occurrence has not been implicated but the role of trauma is somewhat controversial. Electrical injury has caused thoracic disc herniation and paraplegia in a man. As at other sites, degenerative changes in the disc are an important cause of thoracic disc prolapse. Our patient is younger and the level of involvement (T6-T7) is somewhat higher than the usual reported level (T11-T12). The greater incidence at these levels may be due to these vertebrae being higher in cephalocaudal dimension than those above them and also because of their increased mobility due to free ribs at these levels.

It is perhaps not surprising that our patient's diagnosis was missed initially. Reports of pain are often vague, the level of involvement may be poorly defined and, indeed, the ambiguous and varying clinical presentations of disc herniation in this region have long been a cause for frustration and concern for clinicians. Not infrequently, other causes have been thought to account for dorsal spine pain, such as cardiac, pulmonary, gastrointestinal, genito-urinary or even psychiatric disease. In a few instances, thoracic or abdominal surgery has even been performed in an attempt to explore pathology.

With regard to diagnostic imaging, routine radiographs may not reveal any lesions but there may be obvious features of disc space narrowing and osteophyte formation. Calcification of the disc is a more important finding in this situation. Although myelography is more useful than plain radiographs the yield may vary considerably and occasionally may mimic intra- or extra-medullary neoplasm. A recent series reported approximately 67% sensitivity. CT scanning is a better technique and may accurately define encroachment of disc on the subarachnoid space and/or spinal cord. CT may also reveal lateral herniation not demonstrated by myelography. MRI is the investigation of choice at present. It is a low-risk non-invasive procedure, well tolerated by almost all patients, and can even be performed as an out-patient. It causes no ionising radiation and it provides excellent anatomical definition. MRI also surpasses other techniques in the detection of spinal cord compression and is able to detect multiple herniations in contrast to CT which requires scanning at multiple levels. As this technique is increasingly used the true incidence of thoracic disc herniations, both incidental and symptomatic, will probably reveal a higher frequency than previously suspected and missed cases or delayed diagnoses will diminish.