Short report

Thoracic radiculopathy related to collapsed thoracic vertebral bodies

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SUMMARY Three cases are presented of thoracic radiculopathy related to collapsed thoracic vertebral bodies. In all cases proximal weakness of the legs was present, leading to the diagnosis of myopathy in two cases. Sensory symptoms were present in two cases. In one, anterior thigh paresthesias lead to a diagnosis of meralgia paresthetica. This diagnostic entity must be remembered if appropriate corroborative tests are to be performed. In cases of trauma this diagnosis should be considered if thoracic vertebral collapse is present. Conversely, an evaluation of proximal weakness should include a review of thoracic radiographs for vertebral collapse, especially in the presence of sensory findings in the lower abdominal or proximal thigh region.

The literature on the neurological complications of thoracic vertebral body fracture focuses on spinal cord or cauda equina injury.¹⁻⁴ The only other symptoms mentioned are poorly localised pain, stiffness, or back weakness.⁵ Typical statements are that simple wedge fractures "can be ignored"⁶ and "are not commonly associated with neurological injury".⁷ In only one case were "radicular symptoms" mentioned.⁸ Three patients are now reported with collapsed thoracic vertebral bodies who, electromyographically, had denervation restricted to lower thoracic-upper lumbar roots.

Case reports

Case One

A 76-year-old Chinese woman slipped two weeks before admission to hospital with subsequent back pain radiating to the right of the abdomen, and walking difficulty. She suffered from Parkinson's disease. On examination there was proximal leg weakness. Cranial nerves, sensation, and reflexes were normal. There were pill-rolling tremor, cog-wheel rigidity, and Myerson's sign. Radiographs revealed compression fractures of T12 and L1, and fractures of ribs and right ischium. On bone scan uptake was increased over T12. CT showed that T12-L1 antero-posterior diameter was decreased. Serum enzymes (CPK, LDH) were mildly elevated.

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Electromyography revealed fibrillation potentials, positive sharp waves, bizarre high-frequency discharges, with normal and long-duration polyphasic motor unit potentials in bilateral lower abdominal, right upper abdominal, left iliopsoas, and right lower thoracic paraspinal muscles. Extensive sampling elsewhere (including tensor fasciae latae, quadriceps, hip adductors, tibialis anterior, gastrocnemius muscles) did not reveal further abnormalities. There were no brief small abundant polyphasic potentials (BSAPPs). Conduction studies (including motor, F waves, H reflexes, and sural sensory nerve action potentials) were normal. These tests indicated bilateral lower thoracic-upper lumbar (approximately T11-L1) radiculopathy.

Case Two

An 80-year-old man had suffered for a year from insidiously progressive leg weakness. He had fallen 4 years before, fracturing his left knee, and 15 months before, suffering compression of the L1 vertebral body. On examination, hearing acuity was found to be decreased and upward gaze limited. Weakness was restricted to iliopsoas muscles bilaterally. Sensation and reflexes were normal. Radiography showed a compression fracture of the L1 vertebral body. Serum SGOT and LDH were normal.

Bizarre high-frequency discharges with normal and long-duration polyphasic motor unit potentials were present in bilateral iliopsoas and lower abdominal, left upper abdominal, and lower thoracic paraspinal muscles. No abnormalities were found elsewhere after extensive sampling, and no BSAPPs were present. Conduction studies were normal. These tests indicated bilateral T11-L1 radiculopathy.

Case Three

A 72-year-old woman had suffered for several years from

right thigh paraesthesias and difficulty arising when seated. A low back injury had occurred 35 years before, and right mastectomy 15 years before with no additional therapy. On examination there was hypaesthesia on the distal part of the right anterior thigh. Cranial nerves, sensation, strength, and reflexes were normal. Radiographs revealed an old T12 vertebral collapse, and several fractured ribs. Bone scan was normal. Thoraco-lumbar CT confirmed T12 compression with no thoracic stenosis; degenerative disease was present (T11-12 and T12-L1), and stenosis below L3.

Fibrillation potentials, positive sharp waves, with normal motor unit potentials were present in the iliopsoas and lower thoracic paraspinal muscles bilaterally. No other abnormalities were present elsewhere during extensive sampling and no BSAPPs were found. Conduction studies were normal. These tests indicated bilateral L1,2 radiculopathy.

Discussion

All three patients had proximal lower extremity weakness and collapsed thoracic vertebral bodies which were dismissed as unrelated and insignificant. A diagnosis of meralgia paraesthetica was made in one case and myopathy in the others. Only when electromyographic sampling was extended to T11-L2 muscles was any radiculopathy demonstrated.

A myopathy was specifically sought on electromyography by evaluating motor unit potentials for the presence of BSAPPs. In none of these cases were they present. Severe denervation was isolated to lower thoracic-upper lumbar roots after extensive electromyographic sampling.

Sensory complaints occurred in two cases. The first patient experienced back pain with right abdominal radiation. The third case was diagnosed as meralgia paraesthetica. On careful consideration, the distribution was not typical of the lateral femoral cutaneous nerve involving the anterior rather than the lateral thigh. Motor findings were present in all cases with proximal distribution suggesting a primary diagnosis of myopathy in two cases, but only the legs were involved.

In one case, myopathy was suggested by mild CK elevation. It is known, however, that such elevation can occur in neuropathic processes.⁹⁻¹¹

Proximal weakness, in addition to suggesting myopathy, should also bring thoracic radiculopathy into consideration. There are conditions associated both with myopathy and with vertebral collapse.¹² These include steroid therapy,^{13 14} Cushing's syndrome,¹⁵⁻¹⁷ hyperthyroidism,¹⁸ hyperparathyroidism,¹⁹⁻²² chronic anticonvulsant therapy,²³ malabsorption syndromes,²⁴ and alcoholism.²⁵

Only a high index of suspicion can lead to a diag-

nosis of thoracic radiculopathy. Two situations especially should draw attention to this possibility. Firstly, in those after trauma with a thoracic vertebral compression, especially in the presence of proximal leg weakness or lower abdominal-proximal thigh sensory complaints. Secondly, when evaluating the aetiology of proximal leg weakness or sensory complaints, thoracic radiographs should be reviewed for vertebral body collapse.

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