Case history

A 36-year-old female presented with acute onset right mid thoracic pain. Over the subsequent 30 min, she developed weakness of the right lower limb and contralateral numbness and paraesthesia.

On examination, there was increased tone in the right lower limb with pyramidal type weakness grade 1–2/5, hyperreflexia and an extensor plantar response. Pyramidal function on the left was normal. In relation to her sensory examination, there was a patch of sensory loss to pain on the right at T7. Below this, there was contralateral pain and temperature loss distal to the knee. Proprioception and vibration were lost on the right to the knee. These signs and symptoms were consistent with a partial Brown-Séquard syndrome on the right at T7.

Magnetic resonance imaging demonstrated a right para-central disc protrusion at T6/7 (Figure 1). There was however no significant compression of the spinal cord itself (Figure 2). On the T2 weighted sequences, there was high-signal change on the lateral aspect of the cord corresponding to her clinical symptoms. In addition, there was an area of high signal in the body of T7 suggestive of possible infarction (Figure 3). Spinal fluid examination was unremarkable and an inflammatory and prothrombotic screen was negative. Thus, a diagnosis of Brown-Séquard syndrome of probable vascular onset was made with associated right paracentral T6/7 disc protrusion and vertebral body infarction.

She was managed conservatively. Two years later, she was ambulant but had mild residual weakness of the right and numbness of the left foot.

In this case, we postulate that a protruding thoracic disc produced an acute onset Brown-Séquard syndrome by impeding the blood supply to the spinal cord. This is a unilateral lesion of the spinal cord. It is characterised by ipsilateral hemiplegia and the loss of tactile and proprioceptive sensation accompanied by contralateral loss of pain and temperature sensation.

The blood supply of the spinal cord is through the anterior and posterior spinal arteries, supplying the anterior two-thirds, and the posterior third of the cord, respectively. Below the cervical level, the anterior and posterior arteries are insufficient to supply the cord and as such they receive serial reinforcement through anastomosis with the radicular arteries that are derived from the segmental vessels including the ascending cervical, intercostal and lumbar arteries. The radicular arteries pass through the intervertebral foramen and divide into
Figure 2. T2 weighted transverse MRI of the spinal cord showing disc prolapse at T6/7 and vertebral body/spinal cord infarction without direct impact on the cord.

Figure 3. T2 weighted sagital MRI of spinal cord showing oedema and vertebral body infarction at T7.

Figure 4. Arterial blood supply to the spinal cord and vertebral body (adapted from Faig et al.).
anterior and posterior branches. In this case, the supply to both the anterior radicular and anterior medullary arteries was affected presumably by an acute disc prolapse compressing the spinal branch of the segmental artery (Figure 4). A previous report by Mansour et al. demonstrated a similar clinical scenario with compression of the artery of Adamkiewicz. Magnetic resonance imaging however was not performed. In our case, imaging suggested infarction in the spinal cord. The presence of infarction in the vertebral body has been shown to be strong supportive evidence of a similar event in the adjacent spinal cord and can serve as a confirmatory sign for spinal cord ischemic stroke.

To our knowledge, this constellation of clinical events has not been described previously using magnetic resonance, the clue to the diagnosis being infarction of the vertebral body.

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References