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Case report

Spinal cord ischemia in Scheuermann disease: A report of three cases



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ABSTRACT

Background: Neurological complications in Scheuermann's disease are rare but serious.

Case reports: We report three cases of severe neurological deficit due to medullar ischemia attributable to the compression of a radiculomedullary artery by thoracic (two cases) and lumbar (one case) disc herniations associated with Scheuermann's disease. They were not treated surgically because of the absence of direct spinal cord compression or definitive spinal cord ischemia. Those young patients still have severe neurological damage. An earlier management could have prevented them.

Conclusion: When doubting about any compressive sign, MRI should be performed with diffusion weighted imaging (DWI) and apparent diffusion coefficient (ADC) sequences in emergency.

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1. Introduction

Scheuermann's disease (juvenile kyphosis dorsalis) is a painful structural kyphosis of the thoracic spine [1,2] which occurs in teenagers.

It is characterised by defective growth of the vertebral cartilage endplate. Scheuermann's disease may result from excessive mechanical stress during the growth of the spine on a weakened vertebral endplate from a genetic background [3]. The radiographic aspects are related to the vertebral endplate lesions and include vertebral wedging of more than 5° of at least three adjacent vertebrae at the apex of the kyphosis, irregularity of the vertebral endplate and Schmorl's node (intraosseous disk herniation). This is the original definition of Scheuermann's disease but the criteria had evolved since then.

Disc alterations are frequent and may be secondary to dysfunction of the disc-vertebra complex. Disc impairment is more frequently observed in Scheuermann's disease than in normal spines [4]. Disc ruptures tend to occur at the apex of the thoracic kyphosis [5]. This observation can be explained by defective disc nutrition because of altered exchange between the disc and the vertebral endplate [3]. Disc impairment can induce pain, but neurological complications have only been described in a small number of

patients with severe and untreated kyphosis [1]. They were caused by dural cysts [6,7], or spinal cord compression by thoracic disc herniation [1,6], and were often triggered by trauma. Those neurological complications may occur more frequently when the curve is small and angular [1].

We report four cases of severe neurological deficit due to spinal cord ischemia attributable to the compression of a radiculomedullary artery by thoracic and lumbar disc herniation associated with Scheuermann's disease.

2. Case reports

2.1. Case 1

A 40-year-old female was admitted for a brutal posterior left thoracic pain with under breast irradiation. She was in good health except for hypothyroidism and obesity. Two hours after her admission, she suddenly had paraesthesia and flaccid paraplegia in lower limbs with sensitivity level at T4. No trigger was identified. Tendon reflexes were abolished. Proprioceptive sensitivity was spared.

The spine MRI did not reveal any sign of spinal cord compression. It showed intramedullary reworking in the T3 to T8 region with bilateral anterior intramedullary hypersignal that could reveal spinal cord ischemia. Two left posterolateral disc herniations T6-T7 and T7-T8 with signs of Scheuermann's disease were observed (Fig. 1). Thoracic kyphosis on MRI was 43°.

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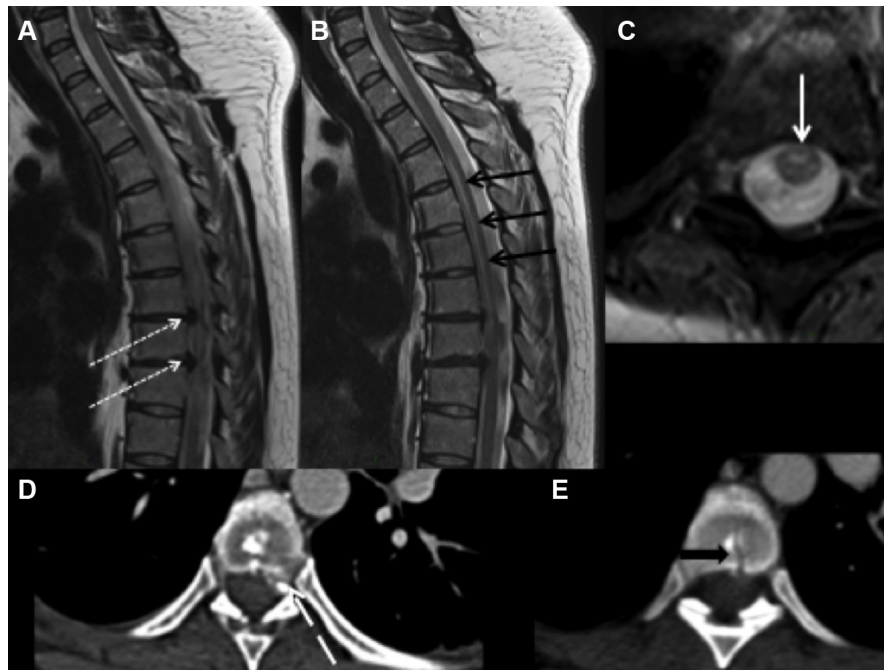


Fig. 1. Case 1. A (MRI) shows two left posterolateral disc hernia T6-T7 and T7-T8 (thin discontinuous white arrows). B (MRI) shows intramedullary hypersignal, illustrating medullar ischemia (thin black arrows). C (MRI) shows intramedullary hypersignal with “snake eyes” aspect, illustrating an ischemic etiology (thin white continuous arrow). D (CT scan) shows partly calcified posterolateral disc hernia (large discontinuous white arrows). E (CT scan) shows trail sign (large black arrow), very evocative of Scheuermann's disease.

Complementary exams have eliminated neurological, cardiologic and infectious diseases. In particular, aorta CT did not identify any thrombosis, including lumbar branches arteries. Multi-level endplates irregularities with invagination of cortical endplate into sub-cortical bone of the vertebral bodies as well as typical “trail sign” are better evaluated by CT than MRI. On the contrary, decreased signal in T2 weighted images and narrowing of disc spaces were more visible on MRI (Fig. 1).

The diagnosis was vascular compression of Adamkiewicz artery or of one of its branches by a thoracic disc herniation on Scheuermann disease, causing an anterior spinal cord ischemia.

There was no surgical care because of definitive medullar ischemia. There was an incremental incomplete recovery of neurological deficit in about three months.

2.2. Case 2

A 57-year-old female was admitted with a brutal thoracic back pain after a wrong movement associated with walking difficulties. There was no preceding illness except an obesity.

Clinical exam revealed paraparesis of lower limbs below T10, tendon reflexes were abolished.

MRI showed a mostly anterior intramedullary hypersignal in T2 sequence below T10 reflecting a spinal cord compression. It also revealed an extra spinal cord image with no contrast enhancement, no mass effect and signs of Scheuermann's disease. The thoracic kyphosis was 39° on MRI.

Complementary exams have eliminated neurological, cardiologic and infectious diseases.

The diagnosis was vascular compression of anterior afference of spinal artery by an excluded thoracic disc hernia. There was no surgical care because of an absence of direct spinal cord compression.

A CT angiography revealed a thin artery originating at a left T11 inter-coastal artery, thus corresponding to radiculomedullary artery.

CT also confirmed the presence of an excluded T10-T11 left lateral pre-foraminal disc herniation with a forward migration and

a classical “trail sign”. It also enhanced the multi-level endplates irregularities, predominantly from T7 to L1, with invagination of cortical endplate into sub-cortical bone of the vertebral bodies and Schmorl nodes.

The MRI that was performed after 2 months revealed pathognomonic sign of an ischemic etiology of the spinal cord (with a “snake eyes” aspect) and a persistent hypersignal in T2 sequence of T11 to L1.

The final diagnosis was spinal cord ischemia of T10 to conus medullaris made by vascular compression of spinal cord anterior artery on thoracic disc herniation.

There was an incremental incomplete recovery of neurological deficit of left lower limb in about three months. No improvement has been observed after three months. She still has severe neurological damage, like neurological flaccid bladder.

2.3. Case 3

A 32-year-old patient in good general health was admitted for brutal calves' pain associated with progressive motor deficit and walking disorders. Clinical exam revealed an almost complete deficit of lower limbs, an abolition of tendon reflexes, a decrease of thermoalgesic sensitivity, the hypoesthesia of sacral metameric levels and a weakness of anal sphincter.

MRI showed an anterior intramedullary hypersignal in T2 sequence reflecting a T12-L1 spinal cord ischemia, a L2-L3 under ligament disc hernia. Signs of Scheuermann's disease consisted on multi-levels endplates irregularities more pronounced in thoracolumbar junction (Fig. 2). The thoracic kyphosis was 47° on an X-ray.

No previous spine injection was performed previously. Complementary exams have eliminated neurological, cardiologic and infectious diseases.

The diagnosis was an anterior spinal cord ischemia caused by the compression of Adamkiewicz artery (lumbar part) by a L2-L3 disc herniation or a compression of the Desproges Gotteron artery.

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