Charcot spine: a complication of medullary arteriovenous malformation

Case illustration

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A Charcot joint of the spine results from a loss of joint protective mechanisms, which is generally caused by a condition affecting the deep pain sensibility.\(^4,5\) We report a case of spinal neurogenic arthropathy that occurred following surgical treatment of a medullary arteriovenous malformation (AVM) and discuss the differential diagnosis and therapeutic strategies that could be devised in similar circumstances.

This 27-year-old man was treated for an intramedullary AVM via a posterior approach following laminectomy of T10–L2. One year after initial treatment, his neurological status was considered to be stable. The patient later presented with deafferentation pain in the region extending from T-10 to L-4 on the left side. A scoliotic outline of deformity was demonstrated on clinical examination and x-ray studies of the area of laminectomy (Fig. 1 left). Twelve years after the first operation the patient’s symptoms progressed, with accentuation of the spinal deformity and L1–2 vertebral dislocation (Fig. 1 right). Results of a transpedicular bone biopsy revealed no tumor cells. Corrective fusion was performed via the posterior approach (Fig. 2 left). Fusion occurred within 6 months (Fig. 2 right).

Neurogenic arthropathy appears when a joint is deprived of its means of protective sensation, particularly deep pain sensation.\(^4,5\) Joint destruction could thus represent the exacerbation of the physiological degeneration process caused by stress conditions in the absence of sensory protective mechanisms. Spinal involvement occurs in 6 to 21% of cases.\(^6\) The case we report is, to our knowledge, isolated and involves a neurogenic arthropathy caused by a medullary AVM. Extensive laminectomy as part of the surgical intervention in treating the vascular malformation probably played a role in the development of the spinal deformity.\(^5\)

A differential diagnosis is sometimes difficult and often leads to the initial suspicion of an infection or tumor.\(^2\) In the featured case, histological studies performed via the transpedicular route showed signs of nonspecific bone necrosis only. The treatment of these lesions, which often cause considerable vertebral instability, usually involves surgery. It is necessary to achieve fusion of the affected vertebrae to suppress the movements responsible for the progressive destruction of the spine. The most appropriate strategies in the treatment of these dislocations remain anterior and posterior arthrodesis combined with osteosynthesis, usually posterior, by using a segmented material.\(^1\) In the present case, the deformity was not associated with major destruction of the vertebral bodies, and spontaneous intersomatic fusion was possible after stabilization and isolated posterior arthrodesis.

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References


Fig. 1. Left: Frontal radiograph of thoracolumbar junction obtained 6 months after surgical treatment of the vascular malformation. Note the right lumbar scoliotic curve. Right: Frontal radiograph of the thoracolumbar junction obtained 12 years after surgical treatment of the vascular malformation. Spinal deformity increased with a progressive L1–2 dislocation.

Fig. 2. Left: Anteroposterior radiograph obtained after surgical correction and fusion via the posterior approach. Right: Sagittal T1-weighted magnetic resonance image of the thoracolumbar junction obtained 6 months after surgery, demonstrating complete fusion.