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INFECTIOUS SPONDYLODISCITIS EVOLVING WITH REFLEX AXIAL PARAPLEGIA: A CASE REPORT

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Abstract: Spondylodiscitis is a term that encompasses infectious involvement of the intervertebral body and disc. Infection can occur via hematogenous route, contiguity of nearby tissues or direct contamination as occurs in surgical procedures. The diagnosis most often happens late, since the initial symptoms are nonspecific and often confused with mechanical low back pain. The aim of this report is to describe a case of spondylodiscitis that evolved with areflex axial paraplegia, requiring decompressive laminectomy.
Keywords: Spondylodiscitis, paraplegia, laminectomy.

INTRODUCTION

Spondylodiscitis, also called vertebral osteomyelitis, refers to the infectious involvement of the intervertebral body and disc that often occurs hematogenously, reaching the vertebral body and, by contiguity, reaching the disc.[1] It is an uncommon infection, in which the symptoms are often nonspecific and confused with mechanical low back pain, causing a delay in diagnosis and, consequently, an increase in morbidity and mortality.[2]

The present study reports a case of infectious spondylodiscitis that evolved with areflex axial paraplegia in which multimodal treatment with antibiotic therapy and laminectomy for decompression was performed, evolving with good results.

CASE REPORT

A 75-year-old patient, coming from Picos-PI, admitted for invasive coronary stratification after AMI with anterior ST-segment elevation, thrombolysed. He spent 10 days in the intensive care unit and 24 days in the ward, while awaiting transfer to our service. History of hypertension, diabetes, dyslipidemia, as well as significant smoking and alcohol consumption. During

hospitalization at our service, he presented severe low back pain without irradiation and, in 5 days, he developed areflex axial paraplegia, approximately one day after the coronary angiography was performed. No fever, headache or visual changes were observed. He had a blood count without leukocytosis, normal cerebrospinal fluid analysis, in addition to blood and urine cultures without germ growth. There was no monoclonal peak in the serum protein electrophoresis.

The echocardiogram showed alterations in segmental contractility, moderate systolic dysfunction (LVEF 40%), but without valvular alterations suggestive of endocarditis. Lumbosacral tomography showed spondylosis, diffuse disc bulge at L3 to S1 levels touching the ventral surface of the dural sac. Electroneuromyography showed polyneuropathy with an axial pattern in the lower limbs, with a severe degree for the sensory component and a moderate to severe degree for the motor component, in addition to rarefaction of potentials on the proximal and mid-distal musculature.

In MRI of the thoracic and lumbar spine, a central intramedullary expansive lesion was described, involving the conus medullaris, at the level of T11-T12, extending to L2-L3, low uptake, with a slight expansive effect in the thoracic portion and important in the lumbar portion, in the which infectious, ischemic, or neoplastic etiologies must be considered.

Neurosurgical evaluation indicated decompressive laminectomy at L2-L3. Antibiotic therapy with vancomycin was started, first associated with meropenem, which was replaced by imipenem, due to nosocomial infection (UTI and pneumonia) by germs sensitive to these drugs, totaling a total of 4 weeks of treatment, with progressive improvement of the condition.

DISCUSSION

Spondylodiscitis is an uncommon infection that affects the vertebra and the intervertebral disc and can occur via hematogenous dissemination, by contiguity of nearby tissues, or even direct contamination after surgical procedures on the spine.[3] In most cases, the diagnosis is difficult, since it

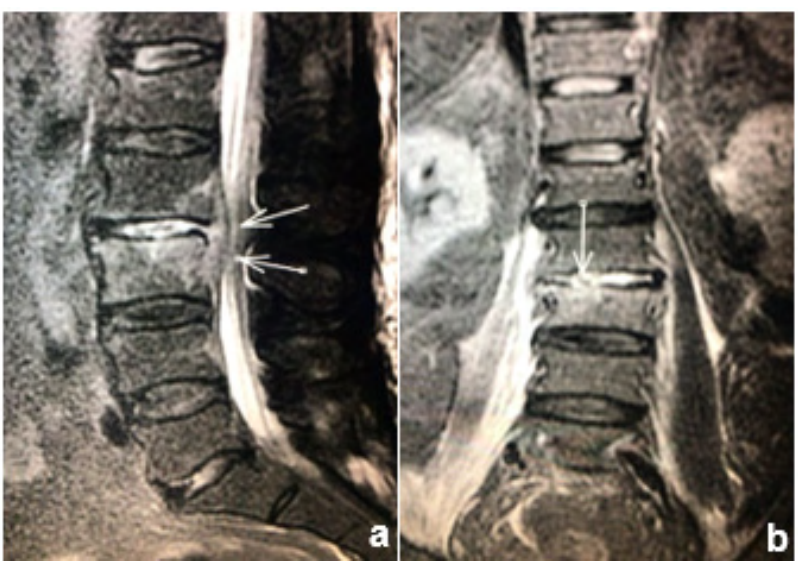

Figure 1. MRI of the lumbar spine. In a) sagittal T2, in b) coronal T2 showing expansive lesion.

presents with non-specific symptoms, which can delay the start of treatment by months. In some situations, evolution to severe cases can be observed, when it courses with neurological symptoms or generalized infection.[1,4]

Low back pain is often described as the initial symptom, appearing in 80% of cases. Absence of fever, together with normal laboratory tests, as in the patient under study, contribute to the differential diagnosis of mechanical low back pain, especially in spondylodiscitis by the *M. tuberculosis*, which leads to delay in treatment and negative impact on morbidity and mortality. The presence of low back pain associated with a sudden onset neurological deficit, as described, leads to high suspicion for spondylodiscitis.[2,5]

Among the most described risk factors, diabetes mellitus, alcoholism, HIV infection, sickle cell anemia, local or systemic infection, immunosuppression, spinal surgery, among others stand out.[6]. Patients on hemodialysis have additional risk factors, such as frequent vascular punctures and contamination from dialysis system water. For this reason, infectious spondylodiscitis must be included in the main diagnostic hypotheses for low back pain in CKD patients on dialysis.[4]

Most infections have only one causative agent, and in approximately one third of cases the microorganism is not isolated. During prolonged hospitalizations, the installation of central venous catheters, bladder catheters, substantially increase the risk of developing nosocomial infection. On admission, the patient had negative cultures; however, even during treatment with antibiotic therapy, a UTI by *Escherichia coli* sensitive to broad spectrum antimicrobials. Among the causative agents of pyogenic spondylodiscitis, *Staphylococcus aureus* is the most described, followed by the bacillus:gram negativo *Escherichia coli*. *Steptrococcus spp.*, *Pseudomonas aeruginosa*, *Enterococcus spp*, *Salmonella spp*. *Brucella spp.* also belong to the group of microorganisms that cause.[1,6]

Magnetic resonance imaging (MRI) is the diagnostic method of choice due to its high sensitivity (96%) and specificity (92%), in addition to being an important tool when axial spondyloarthritis or other inflammatory and non-infectious entities (SAPHO syndrome), Modic type 1 lesion, destructive spondyloarthropathy) enter the differential diagnosis[7]

In a prospective study published in 2020, 1755 patients diagnosed with endocarditis were selected, who were divided into two groups, with and without infective spondylodiscitis.[8] In the group with spondylodiscitis, patients with an older age group were observed, as well as a higher incidence of hypertension and autoimmunity diseases. A delay in diagnosis was also reported, which reached up to 35 days, which can be explained by the fact that 41% of the patients were asymptomatic.

Treatment is based on intravenous antibiotic therapy for at least 4-6 weeks, ideally guided by culture. In situations in which the causative microorganism is not isolated, as in the case in question, the therapy can be performed empirically, with coverage for the most commonly involved germs, and can be extended up to 3 months.[5] When there is development of motor deficit, generated by compression of nerve structures, the surgical approach is the method of choice in association with antimicrobial treatment.[9]

In the patient in question, the clinical presentation with sudden neurological deficit, associated with a collection with a compressive effect on the lumbar spine in an imaging exam, were decisive for the surgical approach. In addition to this criterion, motor instability, unsatisfactory response to drug treatment such as pain refractoriness and disease progression despite antibiotic therapy

are also cited as unequivocal indications for surgical decompression.[9, 10]

CONCLUSION

Infectious spondylodiscitis presents with nonspecific symptoms most of the time, however, in rare situations, it can evolve with severe neurological impairment. The non-specificity of symptoms shows the importance that must be given to this diagnosis, especially in groups with risk factors, since early treatment is crucial and prevents complications.

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