

ATYPICAL SPONDYLODISCITIS

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ABSTRACT

Infections with coagulase-negative *Staphylococcus* correspond to a small percentage of the total pyogenic spondylodiscitis and are associated with immunocompromised conditions. We present a case of a 58 year old male with a L1-L2 spondylodiscitis by a very atypical microorganism, the *S. hominis*. Only after two surgeries and specific antibiotic therapy it was possible to eliminate the disease.

Keywords: *Staphylococcus*, *Staphylococcus hominis*; Discitis.

RESUMO

As infecções por *Staphylococcus coagulase-negativo* correspondem a uma pequena percentagem do total das espondilodiscites piogênicas, estando associados a estados imunocomprometidos. Apresentamos o caso clínico de um homem de 58 anos, com uma espondilodiscite L1-L2, por um micro-organismo bastante atípico, o *S. hominis*. A resolução da patologia só foi possível após duas cirurgias e terapia com antibióticos específicos.

Descritores: *Staphylococcus*; *Staphylococcus hominis*; Discite.

RESUMEN

Las infecciones por *Staphylococcus coagulasa-negativa* corresponden a un pequeño porcentaje del total de las espondilodiscitis piogénicas, estando vinculadas a estados inmunocomprometidos. Presentamos el caso clínico de un hombre, de 58 años de edad, con una espondilodiscitis L1-L2, por un microorganismo muy atípico, el *S. hominis*. La solución de la patología fue posible solamente después de dos cirugías y de terapia con antibióticos específicos.

Descriptores: *Staphylococcus*; *Staphylococcus hominis*; Discitis.

INTRODUCTION

In spondylodiscitis, the main route of infection is hematogenous. Symptoms primarily consists of pain in the spine, inflammation, whether or not it is associated with a fever.¹ It may arise spontaneously (most commonly in pediatric cases), but it is significantly more common to arise accompanied by other infections, malignancy, or collagenosis. Spondylodiscitis often occurs as a complication of sepsis, urinary tract infection, respiratory tract infection, gastrointestinal pathology or even after amygdalectomy.²⁻⁷ The clinical picture is analytically completed with leukocytosis (with neutrophilia), and increased sedimentation velocity (SV) and C-reactive protein (CRP). With regards to imaging, X-ray (XR) provides some information, but complementation by computed tomography (CT) and especially magnetic resonance (MR) allows us to make a diagnosis. Note that the most frequently encountered pathogen is *St. aureus* and the lumbar spine is most often affected.

CASE

Patient, male, 58 years old, with a history of lung tuberculosis and chronic obstructive pulmonary disease (COPD) treated with corticotherapy, was referred to the emergency room (ER) due to progressive low back pain without radiation or apparent triggering factor that is refractory to medication. After physical examination, only lumbar pain on palpation without associated neurological or systemic changes was observed. Laboratory tests showed increased CRP and SV, and a lytic lesion at L1-L2 was seen in XR. (Figures 1 and 2) We subsequently performed a CT scan of the

lumbar spine (Figure 3) followed by MR (Figure 4A-B), and changes consistent with spondylodiscitis were found at the level of the L1-L2 intervertebral disc, in the presence of anterior paravertebral phlegmon at the same level. Given the clinical picture, the patient was admitted to conduct antibiotic therapy and to collect samples for a blood culture.

Due to a fruitless attempt at drainage guided by CT (Figure 5), clinical deterioration (with a 38°C fever) and progressive analytical



Figure 1. Lateral XR on the day of admission to the ER.

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Figure 2. AP XR on the day of admission to the ER.



Figure 3. Sagittal CT image on the day of emergency, showing lytic lesion on the upper surface of L2.

deterioration, we performed during the first surgical period costotomy, surgical drainage of the anterior paravertebral abscess, L1-L2 discectomy and interbody grafting via an anterior approach, about three weeks after arriving at the ER. Two weeks later, and after having a confirmed negative bacteriological examination and anatomical-pathological examination of the material excised evidencing only nonspecific inflammation, we proceeded to posterior pedicle D12-L3 instrumentation. (Figure 6 A-B) Bacteriological and anatomical-pathological samples were taken again and one *St. hominis* was isolated only during this last intervention, resistant to penicillin and its derivatives.

The patient underwent directed antibiotic therapy during the rest of the hospitalization, and some improvement of the clinical and analytical picture was observed during this time. The patient was discharged at the end of 2 months after the initial episode, continuing the specific antibiotic treatment (clindamycin + levofloxacin) at home.

After 3 months postoperatively the patient is without complaints of pain or neurological changes, and is afebrile, monitored in external consultation.

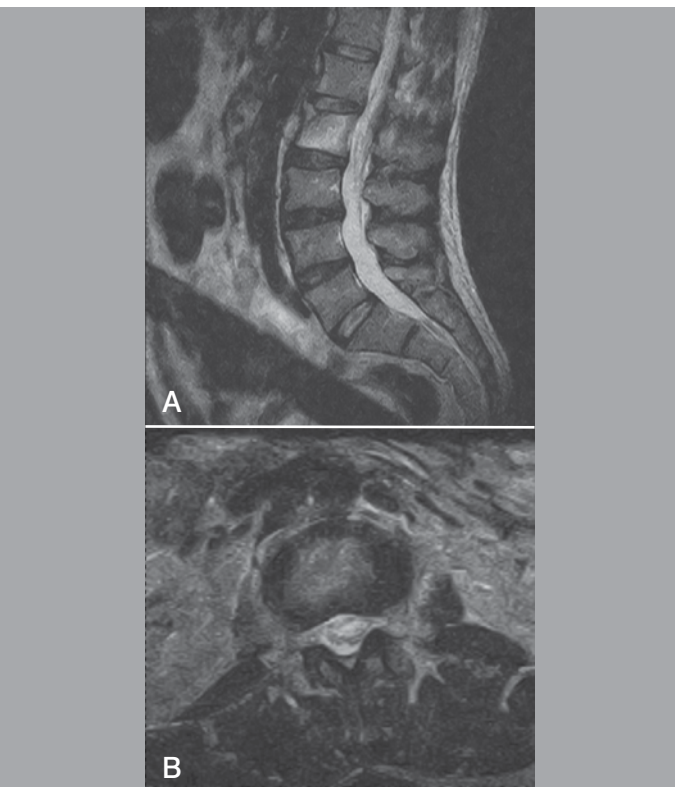


Figure 4 A and B. Sagittal (4) and axial (5) plane MR demonstrating L1-L2 spondylodiscitis, with a slight anterior paravertebral phlegmon at this level.



Figure 5. CT-guided drainage at the L1-L2 intervertebral disc level.

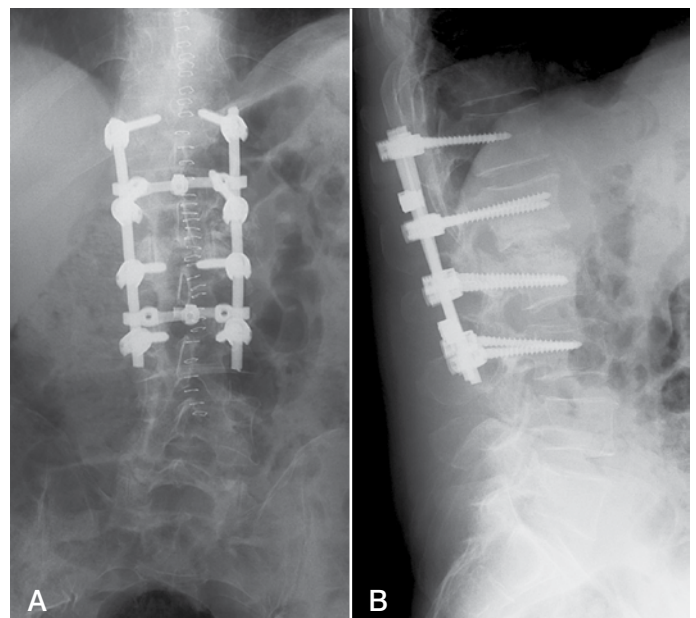


Figure 6 A and B. Postoperative XR.

DISCUSSION

In spondylodiscitis, as well as in infectious arthritis, the main route of infection is hematogenous, and the principal cause is staph infection in 40-60% of cases, especially *Staphylococcus aureus* among all species, through the production of the coagulase enzyme. In the past, coagulase-negative staphylococci rarely caused infections of clinical relevance, but the continued use of cardiac and joint catheters and prostheses has led to an increase in its pathogenic capacity, especially in immunocompromised patients. Currently, coagulase-negative staphylococci are responsible for about 3-18% of infectious spondylodiscitis cases;⁸⁻¹² this percentage increases to 25-30% in post-surgery cases.^{13,14} The most common coagulase-negative subspecies is *St. epidermidis* (72%) followed by *St. lugdunensis* (7%).⁸⁻³⁰

Spondylodiscitis by *St. hominis* is clinically rare. There are few cases described in the literature;³¹ the microorganism is resistant to penicillins and their derivatives. It is associated with immunocompromised states or invasive procedures, but may arise as a result of a contamination of a microbiological examination.

Although the history of tuberculosis in this patient leads one to suppose infection by *Mycobacterium tuberculosis*, this diagnosis was not confirmed.

It is concluded that a *St. hominis* infection is very uncommon, and that there is a fundamental need for directed antibiotic therapy.

All authors declare no potential conflict of interest concerning this article.

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