CASE STUDY

CONTENTS

Spondylodiscitis – a silent infection with loud consequences

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ABSTRACT

The aim of this study is to present a case of a patient with spondylodiscitis. Spondylodiscitis is a infection of the spine involving the vertebral body and/or intervertebral disc, often caused by Staphylococcus aureus and Enterobacteriaceae. It most commonly affects the lumbar spine, begins in the vertebral body endplates and can lead to destruction of bone structures and involvement of the intervertebral disc and surrounding tissues. A 53-year-old man developed lumbar spine pain after an infection of unknown origin accompanied by fever and weakness. After two weeks, when the pain worsened and radiated to the left buttock, an MR examination showed features of L3/L4 spondylodiscitis. During hospitalization, a follow-up MR examination revealed destruction of the L3 and L4 vertebral bodies, inflammatory changes within the L3/L4 intervertebral disc, and widening of the intervertebral space. Inflammatory granules in the anterior part of the spinal canal with slight pressure on the meningeal sac were identified. A CT scan confirmed vertebral destruction. Empirical antibiotic therapy (clindamycin, ceftriaxone, vancomycin, rifampicin) was followed by spinal stabilization. After a year, CT scan showed improvement – reduced destruction of the L3/L4 vertebral bodies and no palpable infiltrative lesions. The stabilizing material was removed. Spondylodiscitis is a infection of the spine, often diagnosed late because of nonspecific symptoms such as back pain and fever. The MRI is the gold standard for diagnosis. Diagnosis is based on clinical, laboratory and imaging findings. It requires the cooperation of surgeons, radiologists and microbiologists. Early detection improves prognosis and quality of life.

KEY WORDS: spine, intervertebral discs, magnetic resonance imaging, spinal inflammation.

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INTRODUCTION

Spondylodiscitis (SD) is a serious infection of the spine involving the vertebral body and/or intervertebral disc, which can also spread to the supraspinal space, posterior elements and spinal soft tissues [1]. Due to non-specific symptoms, such as persistent back pain, limitation of spinal mobility or general signs of infection (fever, weakness), making an accurate diagnosis is often challenging [2]. Delay in diagnosis can lead to serious complications, including epidural abscesses, neurological disorders and permanent damage to spinal structures [1].

The epidemiology of this disease shows an increasing number of cases, which is related to the prolongation of patients' lives, advances in interventional medicine and the increasing use of invasive diagnostic and therapeutic procedures [3]. Risk factors include previous infections such as endocarditis, urinary tract infections, septic arthritis and the presence of vascular catheters [3]. Etiologically, SD is most often caused by Staphylococcus aureus (about 60% of cases) and bacteria of the Enterobacteriaceae genus [3]. The infection most often involves the lumbar spine (60%), less often the thoracic (30%) and cervical spine (10%) [3]. The disease process usually begins in the vertebral body endplates and can spread to surrounding tissues, leading to destruction of bone structures and involvement of the intervertebral disc [3].

The diagnosis of SD is based on clinical signs of elevated inflammatory parameters in laboratory tests and advanced imaging methods [4]. The gold standard is a magnetic resonance (MR) imaging with gadolinium contrast, which allows for precise determination of the extent of inflammatory lesions and identification of soft tissue and bone abscesses [5].

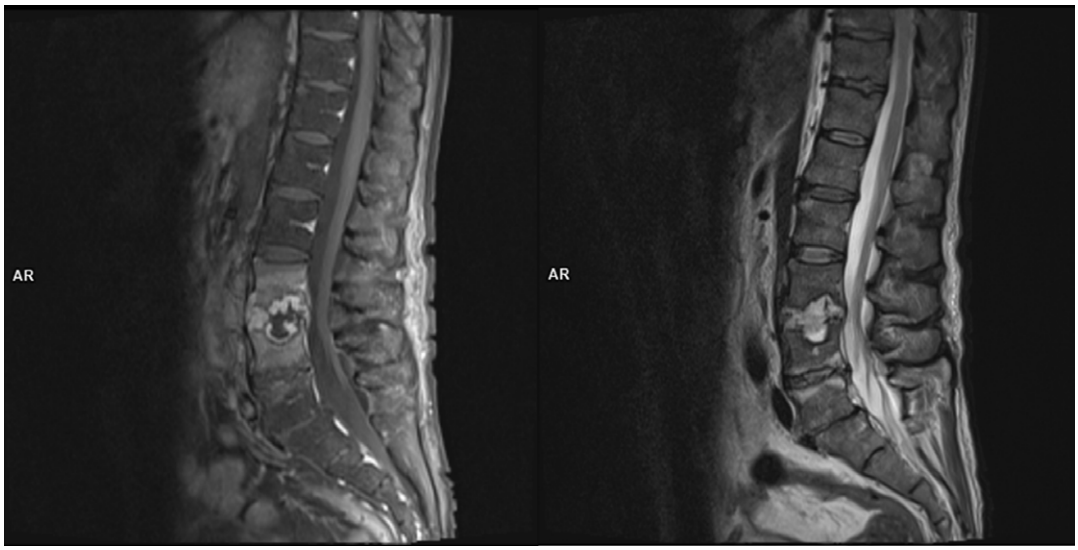

Fig. 1. Magnetic resonance imaging (MRI) of the lumbar spine . T1-weighted (Left) and T2-weighted (Right) sagittal images after contrast administration showing destruction of the L3 and L4 vertebral bodies, widening of the L3/L4 intervertebral space and inflammatory changes in the perivertebral soft tissues.

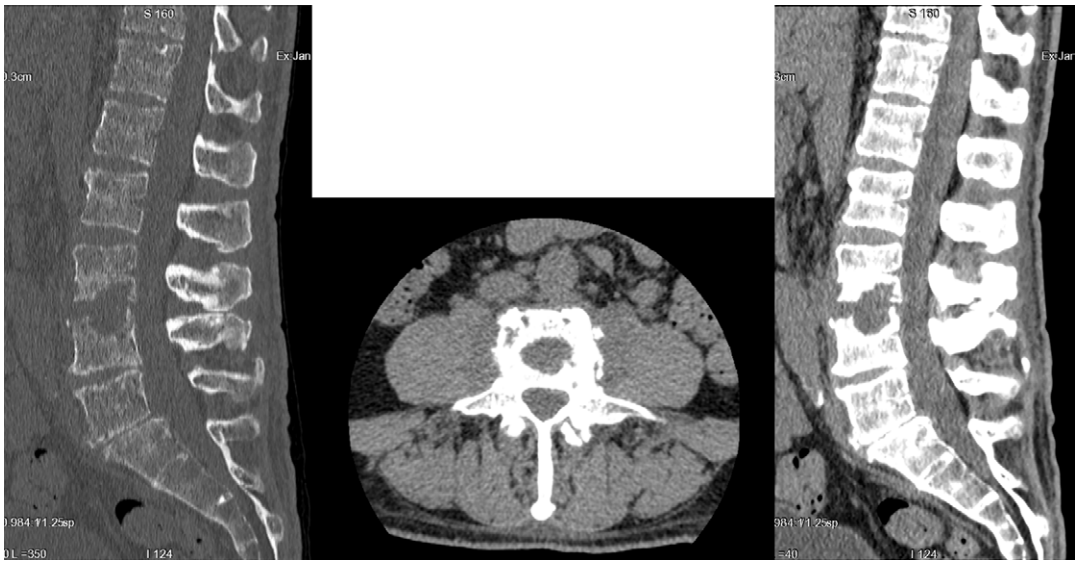

Fig. 2. A CT scan showing destruction of the adjacent vertebral body endplates of the L3 and L4 vertebral bodies (Left – bone settings) , with small fracture gaps of the cortical layer and widening of the L3/L4 intervertebral space. An inflammatory infiltrate was visualized in the spinal tissues at the L3/L4 level (Middle and Right- soft tissue settings).

Treatment of spondylodiscitis includes both conservative and interventional approaches. Long-term antibiotic therapy (lasting 4-12 weeks) is sufficient for most patients [6]. In complicated situations, such as spinal instability, the presence of epidural abscesses or new neurological deficits, surgical intervention is indicated [7, 8]. Treatments include decompression of nerve structures, drainage of abscesses and stabilization of the spine [6]. Despite the serious course of the disease, the implementation of early, targeted treatment achieves a complete cure in more than 90% of patients in developed countries [8].

CASE REPORT

A 53-year-old man underwent an infection with an unknown exit point in the fall. It was accompanied by fever, chills and general weakness of the body. A few days after the infection, the patient also developed lumbar back pain, which was treated on an outpatient basis with little improvement. A few weeks later, the patient experienced severe pain radiating to the left buttock following a sneeze. After an outpatient neurological evaluation, the patient was referred for an MR examination of the spine. Features of L3/L4 spondylodiscitis were found. The man had been experiencing night sweats, a feeling of weakness for several days and was mainly lying down due to the accompanying pain. When attempting to walk, he had to maintain a forward leaning position and required the assistance of an additional person. A decision was made to refer the patient to the hospital's neurology department.

On admission, the man underwent laboratory tests. Blood culture results were negative, with a C-reactive protein (CRP) level of 31.2 mg/L, a leukocyte count (WBC) of 11,100/μL and a procalcitonin (PCT) of ,0.2 ng/ml. Additional tests ruled out Lyme disease and tuberculosis as the cause of the infection. Broad-spec-

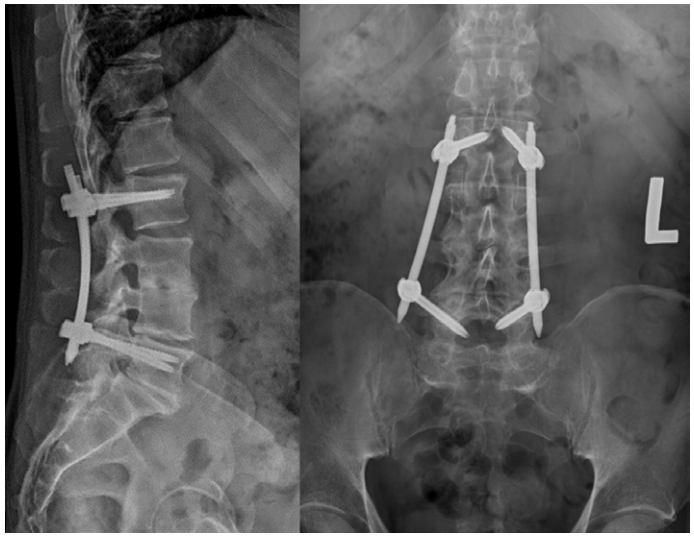

Fig. 3. Lateral (left) and AP (right) X-ray showing L2-L5 percutaneous transspinal stabilization.

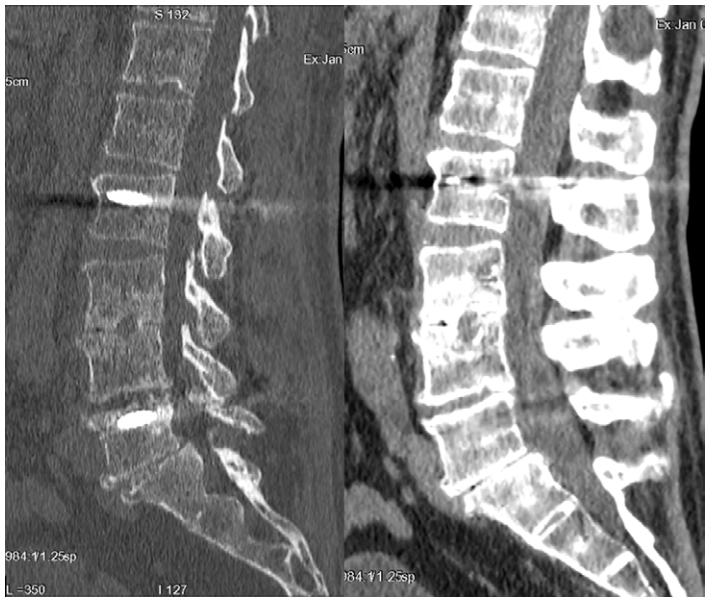

Fig. 4. A follow-up post stabilization CT scan (Left – bone settings) showing reduced destruction of the L3-L4 vertebral bodies and regression of infiltrative soft tissue changes (Right – soft tissue settings).

trum intravenous antibiotic therapy (clindamycin, ceftriaxone, vancomycin) was administered. In the following days, the patient's condition remained stable, with a decrease in CRP and a reduction of pain. Repeat MR examination of the lumbar spine showed progression of lesions compared to the previous examination, destruction of the L3 and L4 vertebral bodies at the L3/L4 intervertebral disc, with uneven contours and widening of the intervertebral space, inflammatory changes of the L3/L4 intervertebral disc, and the presence of inflammatory granulation tissue in the anterior part of the spinal canal, causing slight pressure on the meningeal sac (Fig. 1). In addition, inflammatory changes were visualized in the perivertebral soft tissues, especially on the left side. Modic II-type degenerative changes were also found in the vertebral body endplates of the L4 and L5 vertebral bodies, wide-set protrusion of the intervertebral disc with compression of the meningeal sac, and bilateral narrowing of the intervertebral foramina. The L5/S1 disc showed reduced height and features of dehydration.

An additional CT scan of the spine with 3D reconstruction showed destruction of the adjacent vertebral body endplates of the L3 and L4 vertebral bodies, with the presence of small fracture gaps of the cortical layer and widening of the L3/L4 intervertebral space. The intervertebral disc was depressed intradurally and toward the intervertebral foramina, causing pressure on the meningeal sac and nerve roots. At the L5/S1

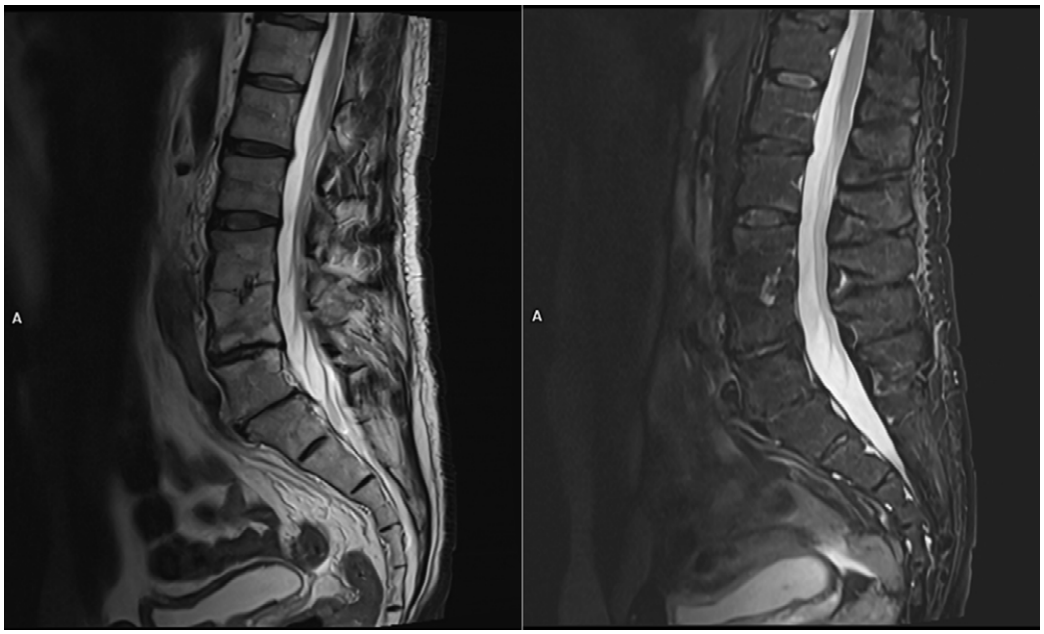

Fig. 5. After 2 years of incidence, a follow-up magnetic resonance imaging (MRI) examination of the lumbar spine (T2-weighted sagittal images) showing lesion regression at the L3-L4 level.

level, a first-degree slip, bilateral L5 spondylolysis and a flattened intervertebral disc were visualized, which protruded intrathecally, compressing the meningeal sac. An inflammatory infiltrate was visualized in the spinal tissues at the L3/L4 level (Fig. 2).

A decision was made to transfer the patient to the orthopedic ward for further treatment. A strict bed regimen was prescribed to limit the strain on the spine. In consultation with a microbiologist, antibiotic therapy was modified. Clindamycin was discontinued, intravenous ceftriaxone and vancomycin were continued, and after a few days it was decided to add rifampicin to increase the effectiveness of treatment of the infection. The patient's condition gradually improved – pain decreased and his overall condition stabilized. For verticalization, the patient was provided with a spinal orthosis.

A subsequent follow-up CT scan showed further narrowing of the L3/L4 intervertebral space and slightly greater sclerosis of the L3 and L4 vertebral bodies, indicating progressive degenerative and inflammatory changes in the lumbar spine.

After six weeks of antibiotic therapy, the patient underwent L2-L5 percutaneous transspinal stabilization surgery to stabilize the lumbar spine. The operation was aimed at limiting further destruction of bony structures and improving spinal stability (Fig. 3). Intraoperative histopathological examination did not detect pathological changes. The patient was discharged home with the recommendation of oral antibiotic therapy with rifampicin and levofloxacin.

One year later, the patient presented for a follow-up CT scan, which showed reduced destruction of the L3-L4 vertebral bodies and no palpable infiltrative changes compared to the earlier scan, indicating a progressive healing process (Fig. 4). Three months later, the man was again admitted to the hospital for removal of the anastomosis. At present, 2 years after the incident, the patient reports no pain, but only has a feeling of stiffness in the lumbar spine with some activities, such as bending down. A follow-up MR examination showed significant narrowing of the L3/L4 intervertebral space with destruction of the adjacent vertebral body endplates of the middle part, exacerbations at the edges of the vertebral bodies, and Modic II degenerative changes. Modic II-type degenerative changes in the vertebral body endplates lamellae were also visualized at the L4 and L5 levels. A significant reduction in the height of the L5/S1 intervertebral disc and a moderate reduction in the height of the L4/L5 intervertebral disc were visualized (Fig. 5).

DISCUSSION

The magnetic resonance imaging (MRI) is the recommended diagnostic method for patients with suspected osteomyelitis of the spine [9, 10]. It is distinguished by its high sensitivity (97%), specificity (93%) and accuracy (94%) [10]. Thanks to its precise imaging, it allows for early detection of inflammatory changes in spinal tissues especially in the acute phase. Characteristic changes in vertebral bodies and intervertebral discs are visible in T1- and T2-weighted sequences [10]. In addition, it allows for the selection of an appropriate treatment strategy, suggesting the need for conservative therapy or surgical intervention, depending on the severity of the disease and involvement of the spinal structures. On classic X-rays, inflammatory changes usually appear

about 2-8 weeks after the onset of symptoms [11]. Their diagnostic value is greater in the chronic phase of the disease. Computed tomography (CT) is particularly useful when magnetic resonance imaging (MRI) is contraindicated, unavailable or gives inconclusive results. It can be helpful in confirming suspected degenerative disc lesions in patients referred for biopsy, such as in cases of intervertebral space narrowing, which may preclude the need for biopsy [6]. CT can detect changes such as loss of intervertebral space height, obliteration of the vertebral body endplate structure, and soft tissue lesions, including abscesses, fractures or neoplastic processes [6].

The differential diagnosis of spondylodiscitis includes infectious diseases (tuberculosis, mycosis, brucellosis), inflammatory diseases, neoplastic diseases and Modic I degenerative changes [12-14]. It is crucial to distinguish infection from neoplastic and inflammatory processes by means of clinical picture, laboratory findings (CRP, ESR, cultures), imaging (MRI, CT) and biopsy, which allows for proper treatment [5, 15].

The diagnosis of intervertebral discitis is sometimes delayed or misclassified as a degenerative process, leading to inappropriate treatment. Although this condition is rare in adults, it should be considered in patients with chronic back pain insensitive to analgesic treatment, especially in the presence of risk factors [11]. Spondylodiscitis is more common in older patients (50-70-years-old), mainly men [6, 11]. Despite extensive testing, the pathogen and the source and route of infection are often not detected. In addition to age, risk factors include diabetes mellitus, malnutrition, weight loss, steroid therapy, HIV, immunosuppression, history of oncology, renal failure, rheumatic disease and history of spinal surgery [11]. In particular, long surgery times, high blood loss and the number of total surgeries increase the risk of spinal infection (1-4%) [15]. Obese patients with subcutaneous fat of more than 50 mm have a higher risk of postoperative infection [11].

Treatment begins with empirical therapy, the most common being amoxicillin/clavulanic acid, ampicillin/sulbactam, cephalosporins and vancomycin [16]. These preparations are used for a minimum of 6 weeks, but some sources report that antibiotic therapy should last from 4 to 12 weeks [6, 17]. Initially, antibiotics are administered intravenously and then orally. Lack of effectiveness of antibiotic therapy progressive destruction of bone structures with spinal instability are an indication for surgical treatment [2]. The most common is transpedicular stabilization [2]. Possibly early diagnosis allows for a better and more effective response to conservative treatment.

CONCLUSIONS

Spondylodiscitis is a serious spinal emergency that requires prompt diagnosis and treatment. Because of nonspecific clinical symptoms, such as back pain that worsens at night and fever, diagnosis is sometimes delayed. The use of MRI as the gold standard for imaging and the identification of the pathogen before starting therapy is crucial for successful management. The cornerstone of treatment is drug therapy with antibiotics tailored to the etiologic agent. Untreated or poorly managed cases of intervertebral discitis can lead to serious complications such as spinal deformities, paresis or paralysis, and even death from sepsis. A multidisciplinary approach to diagnosis and treatment ensures the best therapeutic results. Collaboration among physicians, orthopedists, neurologists, radiologists and microbiologists is essential to achieve a positive treatment outcome and minimize complications. Early detection of the disease, proper therapeutic management and close monitoring of patients with risk factors are key to improving the prognosis and quality of life of patients.

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CONFLICT OF INTEREST

The authors declare no conflict of interest

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