

# A big challenge in the diagnosis of spondylodiscitis with multiple risk factors: a case report

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**Abstract.** Spondylodiscitis is a combination of discitis (inflammation of one or more intervertebral disc spaces) and spondylitis (inflammation of one or more vertebrae), the latter generally involving the areas adjacent to the intervertebral disc space. Spondylodiscitis is the most common spinal infection and the most common complication of sepsis or local infection, usually in the form of an abscess and is characterized by an increasing incidence. The main causative organisms are staphylococci, but potential organisms include a large number of bacteria, fungi, zoonoses. Spondylodiscitis frequently develop in immunocompromised individuals, such as by a cancer, infection, or by immunosuppressive drugs used for organ transplantations. Chronic back pain is the most common nonspecific presentation and diagnosis is often delayed. We present the case of a patient admitted for pain in the lumbar spine of increased intensity, initially without imaging signs of spondylodiscitis, but with the subsequent development of changes compatible with this diagnosis. The patient was treated with appropriate antibiotic therapy, with an undulating evolution determined by multiple complications that evolve to the present comorbidities.

**Key Words:** spinal infection, spondylodiscitis, cirrhosis

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## Introduction

Spondylodiscitis is characterized by an inflammatory process that occurs in the vertebral bodies, intervertebral spaces and discs, and adjacent paraspinal tissue. The first cases of pyogenic spinal infections were described in 1936 by Kulowski (Waheed et al 2019). The incidence of spondylodiscitis has increased considerably in recent years, a fact explained by the increase in the number of patients who may be susceptible, but also by the sensitivity and specificity of modern imaging. At European level, the incidence increased from 2.2 to 11% per year, per 100,000 inhabitants. (Gentile et al 2019; Herren et al 2012; Waheed et al 2019). Gender-differences in disease distribution are disproportionate, being more common in males (56% men, 34% women) with an average age of 59.6 years (a 0.82 higher susceptibility for males to develop the infection) (Gentile et al 2019; Herren et al 2012). Some of the risk factors for spondylodiscitis are: advanced age (patients over 65 years of age are almost 4 times more susceptible), diabetes, immunosuppression, history of infectious pathologies and HIV, polypharmacy, chronic kidney disease and liver cirrhosis (Gentile et al 2019; Herren et al 2012; Waheed et al 2019). The etiology is multifactorial and requires a multidisciplinary approach. In the initial stage of the disease, the intervertebral disc is affected, so that later the infectious process extends to the vertebrae and the adjacent paraspinal tissue (Waheed et al 2019).

The mode of transmission of the infection is hematogenous (most common from another source), contiguous spread (adjacent infectious process) or external inoculation. The most common is the hematogenous route as patients often require multiple vascular approaches, mostly central vein approaches, which can produce the inoculation of the microbial agent (Cervan et al 2012; Herren et al 2012). Most often, spondylodiscitis is caused by *Staphylococcus aureus* (over 50% in Europe) and *Mycobacterium tuberculosis* worldwide. In addition to the stated bacteria, spondylodiscitis positive for other staphylococci (*S. epidermidis*), streptococci (*Streptococcus* spp), enterobacteriaceae (*Escherichia coli*) or other gram-negative bacteria (*Brucella* spp) are cited (Gentile et al 2019; Menon et al 2014), being in most cases monobacterial. The site of development of the infectious process is most often in the lumbar region, but there are citations and cases of the infection occurring in the cervical region, thoracic region or multilocular (Cervan et al 2012; Stoop et al 2021). Clinical manifestations are nonspecific, often with hyperalgesia with a spinal starting point (one or more vertebral segments), loss of appetite to weight loss, night sweats, and fever (Gentile et al 2019; Herren et al 2012; Stoop et al 2021).

Routine biological investigations most often show signs of infection (leukocytosis, reactive inflammatory syndrome markers). An acute manifestation is accompanied in more than 80% of cases by increased PCR with or without the presence of leukocytosis; in the chronic stages, the blood count may not suggest

changes in the leukocyte profile and inflammatory markers may remain unchanged (Herren et al 2012; Foreman et al 2017).

The diagnosis is based on the accumulation of clinical, para-clinical and imaging data, but most of the time biological investigations do not signal significant changes, and identifying the causative agent can be a real challenge (Gentile et al 2019; de Graeff et al 2017). For this reason, imaging investigations are a standard in making a positive diagnosis of spondylodiscitis. Magnetic resonance imaging is the gold standard for establishing the diagnosis of spondylodiscitis, with a specificity of over 90%.

We report a case with non-specific clinical presentation (neurological deficits were not present from the beginning), signs and symptoms of decompensation of the other comorbidities were present, and the diagnosis was delayed by the heterogeneity characteristics of spondylodiscitis.

## Case Presentation

A 72-year old woman with previous history of multiple cardiovascular comorbidities (arterial hypertension, atrial fibrillation, ischemic cardiomyopathy, valvulopathies such as severe mitral and tricuspid insufficiency, first-grade aortic insufficiency, large aortic stenosis and heart failure), gastrointestinal diseases (cirrhosis Child-Pugh C with unknown etiology, encephalopathy West-Heaven 2nd grade, esophageal varices), metabolic pathology (diabetes mellitus II) presented to the Emergency Unit in our hospital. The patient was recently hospitalized in another department of internal medicine for drowsiness, temporal and spatial disorientation, and hematochezia. The following procedures were carried out: esophagogastroduodenoscopy (that shows esophageal varices, micropolyps, without active bleeding), colonoscopy (digested blood easily removed by washing, colonic mucosa with an infiltrated and edematous appearance suggestive for portal hypertensive gastropathy) and contrast-enhanced chest computed tomography (CT) - without hematic extravasation.

In the last few days, she developed lower limb edema, back pain (more accentuated at the lumbar level described as 7/10 intensity), and loss of appetite. Physical examination revealed a cooperative obese patient, conscious, in a slightly altered general state, and no spatial and temporal disorientation, no changes during pulmonary auscultation, rhythmic heartbeats with holosystolic tricuspid murmur, and no changes observed during abdominal exam. The patients had associated bilateral lower limb "pitting" edema and paravertebral contracture lumbar. Her vital signs included a blood pressure of 102/54 mmHg, a heart rate of 65 beats per minute, and 99% oxygen saturation at rest in room air.

Upon admission to the hospital, the peripheral blood count revealed moderate anaemia (Hb=9g/dl), severe thrombocytopenia ( $44 \times 10^3 \mu\text{L}$ ), lymphopenia, inflammatory syndrome (elevated C-reactive protein), impaired renal function based on elevated serum creatinine and urea (creatinine clearance - CKD-EPI = 70 ml/min/1.73 m<sup>2</sup>), hepatocytolysis, cholestasis, hyperglycemia and elevated N-terminal prohormone of brain natriuretic peptide (NTproBNP).

The case was initially interpreted as decompensated liver disease and heart failure with further investigation of the precipitating factors and the exacerbation of the decompensation. Chest X-ray

and cardiac ultrasound were thus performed, showing moderate left pleural collection, partially secluded, and a pulmonary pattern accentuated by a linear vascular mechanism (Kerley B lines in the periphery of the lungs). Cardiac ultrasound revealed the progression of valvulopathies over time (mild aortic insufficiency, severe mitral and tricuspid insufficiency, severe secondary pulmonary hypertension with preserved ejection fraction -60%). Abdominal ultrasound did not detect any additional changes as compared to the previous examinations. During the hospitalization, the lumbar pain persisted even with analgesic medication, which is why an X-ray of the pelvis was performed and it revealed joint changes. The diagnostic assessment was completed with CT of the pelvis, showing bilateral coxarthrosis, changes in the L4-L5 segment, with intrasomatic L4 disc herniation and posterior L4 disc vacuum, left hemisacral osteosclerosis, calcifying enthesopathy (Fig.1).

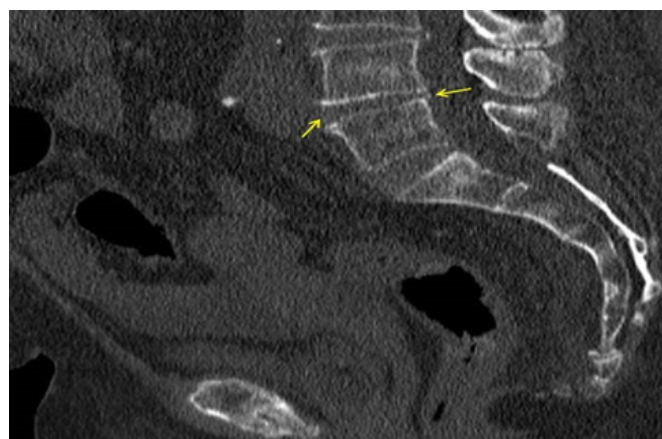


Fig.1. First unenhanced CT of the pelvis (reconstructed sagittal image) shows moderate loss of L4 disc height with mild degenerative changes of the adjacent endplates L4-L5 (arrows), without any bone destruction, therefore with no suspicious signs of spondylodiscitis.

It was considered opportune to supplement the analgesic mediation, with obtaining partial control of the symptomatology. During hospitalization, the patient presented a progressive degradation of the state of consciousness, with episodes of drowsiness - framed in the context of hepatic encephalopathy. When biological tests were repeated, elevated inflammatory markers, altered renal function and worsening anemia (hemoglobin drop to 6.4 g/dl, without signs of active bleeding) with severe hypoalbuminemia were observed. Infectious screening was performed (blood cultures, lingual secretion, pharyngeal secretion, rectal swab, urine culture) and 3 units of erythrocyte mass (ER) were dynamically administered with oral and intravenous supplementation of protein and albumin deficiency - with slow favorable evolution. Following the screening, a urinary tract infection was detected (confirmed with *Klebsiella* spp and *Candida* spp), which is why treatment with beta-lactams (amoxicillin + clavulanic acid) and antifungals was initiated. Considering the persistence of lumbar hyperalgia, the upward curve of inflammatory markers and positive blood cultures with coagulase-negative staphylococci, it was decided to change the class of administered antibiotics - carbapenem (Meropenem) based on the antibiogram and to repeat noncontrast-enhanced CT of the abdomen and pelvis (Fig.2). The following were detected: dorso-lumbar spondylarthrotic changes, areas of cortical disruption

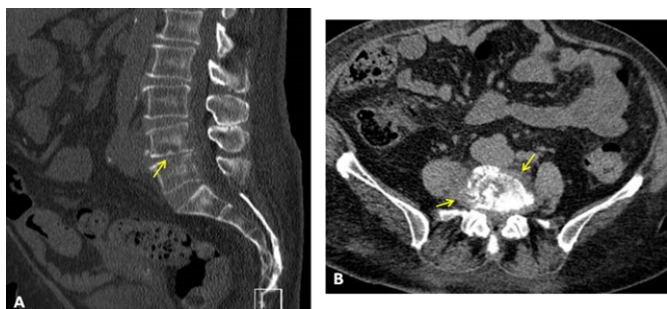


Fig.2. Second unenhanced CT of the abdomen and pelvis: On the reconstructed sagittal bone window image (image A) there is newly developed small areas of ill definition and reduced bone density of the L4-L5 vertebral endplates (arrow). Also, there is progression of the loss of L4 disc height. On the axial soft tissue window image (image B), beside the end plate erosions, there is perivertebral swelling with a loss of peripheral fat planes (arrows), in keeping with probable soft tissue inflammation.

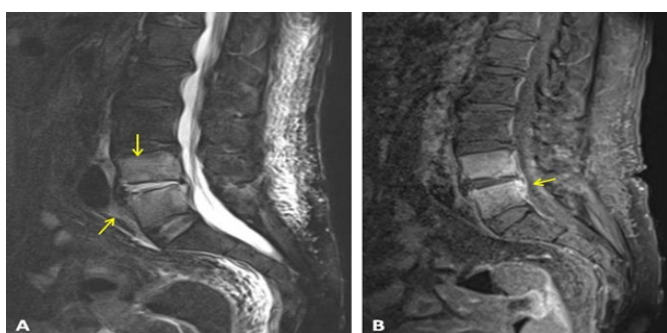


Fig.3. Contrast enhanced MRI of the lumbar spine: Sagittal STIR image (A) shows high signal in L4-L5 disc space (fluid), high signal in adjacent L4-L5 endplates and vertebra bodies (bone marrow edema), small areas of loss of low signal cortex at endplates and high signal in paravertebral soft tissues (arrows). On the contrast enhanced T1 image (B), there is enhancement of L4-L5 vertebra (including the endplates) and adjacent paravertebral soft tissues and anterior epidural fat (arrow), suggestive of inflammation.

in the lower L4 segment, which could correspond to a fracture or to spondylitic changes and therefore the diagnostic assessment was completed by a lumbar MRI scan.

Changes suggestive of spondylodiscitis at L4-L5 level were thus highlighted, with the presence of intradiscal fluid and edema of the L4-L5 vertebral bodies, with postcontrast capture at the level of the meninges, marked narrowing of the right L4-L5 foramen and L4 nerve root compression (Fig.3).

Clinical findings included intermittent paresthesia and lower limb hypotonia with progressive limitation of mobilization. In order to establish the therapeutic conduct, the case was submitted to a multidisciplinary team (specialists in internal medicine, neurology, neurosurgery, infectious diseases, radiology), and the decision was to refrain from any invasive measure (age, comorbidities, severe thrombocytopenia), monitoring the neurological status, the association to carbapenem and glycopeptide (Targocid) and caution regarding renal function (both with a view to adjusting antibiotic doses and due to the fact that, per se, renal insufficiency represents a negative predictor of the evolution of the patient with spondylodiscitis). The dynamic of the evolution was slowly favorable under the newly

initiated treatment, without new neurological symptoms, with the progressive decrease of inflammatory markers; renal function was carefully monitored and neurological and infectious disease consultation was resumed periodically.

Approximately 7 weeks after the initiation of antibiotic therapy, the patient presented an episode of hematochezia manifested by multiple episodes of diarrhea with blood in stool. A possible infection with *Clostridium difficile* was suspected - with a negative result. Lower GI bleeding persisted for about a week, causing the patient's hemodynamic instability, with hemoglobin dropping to 5 g/dl. Lower GI endoscopy was performed, which revealed multiple areas of the colon mucosa with diffuse bleeding in the lining and ulceration with an adherent clot in the rectum. In the context of severe anemia, isogroup erythrocyte mass, isoRH, fresh frozen plasma, antihemostatics, hydroelectrolyte rebalancing solutions were administered with the limitation of bleeding after about a week and the partial correction of the anemic syndrome, with the increase of hemoglobin up to 10 g/dl. From a neurological point of view, the evolution was favorable, with no new complaints, with a significant improvement of the inflammatory syndrome and the progressive resumption of mobilization, and therefore, after 9 weeks of hospitalization, the patient was discharged.

Spondylodiscitis is an infectious pathology of the spine, which is dominated by non-specific features. Early diagnosis is essential for a favorable outcome and to avoid possible complications. From the early stages, it is necessary to concretely define the personal pathological antecedents (taking into account the quick evolution of spondylodiscitis due to invasive interventions) and the identification of risk factors (diabetes, cardiac pathology, renal failure). From the perspective of risk factors, a study carried out in the period 2005-2015 in patients with spondylodiscitis and liver cirrhosis (different stages of the Child-Pugh classification of cirrhosis - 16.7% of patients were classified in class A, 25% in class B and 58.3% in class C), demonstrated that these patients have an increased predisposition to the occurrence of infectious processes (five times higher than the patient with fully preserved immune functionality). Susceptibility to infection in patients with cirrhosis is closely related to the impairment of defense mechanisms, reticuloendothelial and portosystemic function and increased intestinal permeability. Moreover, an infectious process can aggravate a preexisting chronic liver disease, acting as a trigger for other complications such as: ICD, hepatorenal syndrome or hemorrhages. In addition to the fact that liver cirrhosis is a risk factor per se for spondylodiscitis, the study also reveals other factors such as diabetes mellitus (in 52.8% of patients), cardiac pathology (47.2% of participants) and renal failure (44.4% of patients) (Abderahman et al 2020). We mention the fact that our patient was also known to have liver cirrhosis (Child Pugh class C), with a recent episode of lower digestive hemorrhage and type 2 diabetes; moreover, towards the end of hospitalization, the patient also developed mild nitrogen retention (possibly due to prolonged antibiotic therapy). Spondylodiscitis can be classified according to the type of infectious agent (pyogenic, granulomatous and parasitic or fungal) or the mode of transmission. Thus, most often, the most frequent mode of infection transmission is hematogenous through inoculation (for example, the dialysis patient who requires multiple vascular access lines), which determines



the development of bacteremia (especially if these patients are also immunosuppressed). Moreover, long-term central venous catheters become colonized with staphylococcal species due to the formation of an intraluminal biofilm. Thus, this biofilm can cause embolization and further obstruction. Another mode of transmission is direct contact (an adjacent infectious process) which can cause the appearance of the primary foci of spondylodiscitis (for example a urinary tract infection or abscessed collection) (Abderahman et al 2020; Herren 2017; Madhavan et al 2019; Waheed et al 2019). Both the hematogenous mode of transmission and direct contact were plausible in the case of our patient. It must be taken into consideration that the patient was recently discharged from another medical service where, due to the inadequate venous vascular approach, the choice was for a central venous catheter (risk of hematogenous infection); the patient was also diagnosed with urinary infection with *Klebsiella* spp, which was also present upon admission to our medical unit.

*Staphylococcus aureus* is particularly pathogenic for spinal infections due to its molecular adhesion that allows it to attach and expose fibrinogen and collagen in the microcapsule (under the action of a polysaccharide) thereby preventing phagocytosis. Moreover, *S. aureus* contains a capsular ampholyte ion with a polysaccharide structure (zwitterion) that allows intracellular survival, thus leading to the formation of abscesses, persistent infection up to osteomyelitis (Cervan et al 2012). Gentile et al published a systematic review that included 1756 patients from different areas (Europe, North America and Asia). The most common etiological factors for spondylodiscitis have been cited as follows (in descending order as number of subjects): *Staphylococcus* spp, *M. tuberculosis*, other bacteria (over 25% of cases), or viral, fungal or parasitic infections (less than 1%). As for the place of occurrence of the pathology, the study reveals that in more than half of the patients the changes appeared at the lumbar level (followed by cervical or thoracic or multiregional). Therefore, the lumbosacral region is most often affected given the vascularity, increased mobility and the fact that anatomically, the peritoneum allows the development of the infectious process (Herren 2017). In the case of our patient, the blood cultures were positive with coagulase-negative staphylococcus, and the center of the infectious process was in the lumbar region.

In a study from 2019, carried out on 44 patients diagnosed with spondylodiscitis, the subjects presented low back pain (in 100% of cases) as the main symptom, with the onset of febrile syndrome in the following days. Of the patients included in the study, only 54% developed febrile episodes (Waheed et al 2019). Most often, the pain is described as continuous and accentuated by physical exertion, percussion of the spine and rushed by knee flexion (pain radiating from the spine) (Gentile et al 2019; Herren et al 2017; Stoop et al 2021). According to the data from the literature, lumbar pain dominated in our case, accentuated by movement, but without the appearance of febrile syndrome during hospitalization.

Paraclinical investigations, although not pathognomonic, are necessary for the early detection of the infectious process. A 2017 study shows that elevated CRP levels can shorten the time needed to formulate the diagnosis, as compared to other laboratory parameters (Jean et al 2017). Moreover, both CRP and ESR

are used to monitor treatment progress (with normalization of CRP levels 3 months after initiation of treatment and decrease of ESR levels to 1/4 of initial values). Procalcitonin levels play a minor role in the diagnosis of spondylodiscitis, being more expensive and not effective in evaluating subsequent treatment (Herren et al 2017; Waheed et al 2017). Once the infection is detected, it is imperative to identify the primary source. Thus, blood cultures are performed, detecting both anaerobic and aerobic bacteria. In the case of negative cultures (after at least 48h of incubation), detection by molecular methods (PCR) can be opted for, especially for patients with previous antibiotic therapy. The sensitivity of molecular detection can be improved by using tests specific to the bacterial species (eg *S. aureus*) (Herren et al 2017). Choi et al concluded that bacterial-specific PCR has twice the sensitivity in detecting the pathogen (compared to conventional PCR) (Choi et al 2014). However, the biggest disadvantage of molecular detection remains the impossibility of providing information related to antibiotic susceptibility and sensitivity. In addition to blood cultures and molecular identification, detection of the pathological agent can also be done by histopathology. The biopsy specimen can be obtained either by CT-guided aspiration (fine needle biopsy) or by surgical resection. Microbial detection by puncture has a rather unsatisfactory success rate (detection in less than 40% of cases) given the small amount of tissue available (Herren et al 2017). A radical improvement in the detection of the causative agent can be achieved by overlaying some virtual MRI/CT images, prior to performing the biopsy (Kim et al 2015). However, the most reliable method of identification remains biopsy by surgical resection (with an approximate success rate of 90%) (Herren et al 2017; Menon et al 2016). Studies show that the pathogen can be detected in more than 45% of cases, with a success rate in more than 75% for patients who have not previously undergone antibiotic therapy (Jean et al 2017). In our case, further repeated blood cultures were negative, the other detection methods not being an option for subjective reasons (additional risks for an unfavorable evolution - age, comorbidities, severe thrombocytopenia).

Spine radiography is the imaging test of choice (with extremely low sensitivity and specificity - approximately 80% and 50%, respectively) (Foreman et al 2017). In most cases, erosions of the vertebral plateaus can be initially highlighted which can later evolve into kyphotic changes with massive morpho-structural destruction (depending on the clinical manifestations, the immune status of the patient and the virulence of the pathogen) (Herren et al 2017). Nuclear magnetic resonance (MRI) remains the gold standard for diagnosing spondylodiscitis. To increase sensitivity, the use of Gadolinium enhancement (which has proved its effectiveness, although conventionally used to evaluate cardiac fibrosis) is suggested. In case of contraindications for MRI (such as pacemakers or prostheses), CT is chosen (Foreman et al 2017; Herren et al 2017). A true star of modern oncology, positron emission tomography (PET-CT) with <sup>18</sup>F-FDG radiolabeled glucose (fluorodeoxyglucose) has been receiving increased attention in recent years regarding its importance in the diagnosis of spondylodiscitis. As a principle of operation, the radioactive glucose particles will not accumulate at the level of the marrow or bone structures, but only at the level of the inflammatory process, determining aspects of "hot spots" of

the spine. 18F-FDG-PET-CT has both advantages and disadvantages, but it also represents an alternative for patients with contraindications to the administration of the contrast substance (allergic background, impaired renal function). For the detection of future sources of infection, bone scintigraphy with Tc99 and gallium citrate can be opted for, which has a reduced specificity due to remodeling processes in spondylodiscitis (such as osteochondrosis). Considering the costs, the reduced feasibility of such techniques, and the logistics required to perform such imaging investigations, most studies recommend contrast-enhanced MRI (Foreman et al 2017; Herren et al 2017; Skanjeti et al 2012; Treglia et al 2012).

The Infectious Diseases Society (IDSA) in the USA has identified 5 key terms that can contribute to improving the decision-making algorithm regarding spondylodiscitis: detection of causative agent, prevention of segmental spinal instability (due to changes such as alteration of adjacent bone or ligament structural integrity), avoiding the occurrence of localized infection and preventing neurological damage (Gentile et al 2019). According to the recommendations, our patient initially underwent pelvic x-ray that revealed arthritic changes; subsequently, contrast-enhanced MRI was performed since CT could not differentiate spondylodiscitis-specific lesions from possible fracture trajectories or other spondyloarthritic changes. Formulating the diagnosis is a real challenge, since non-specific manifestations tend to delay a correct diagnosis for up to 2-6 months (in the case of pyogenic spondylodiscitis, with a 5% rate of correct diagnosis from the early stages) (Cheung et al 2011). A series of diagnostic algorithms for spondylodiscitis are proposed in the literature (Fig. 4).

Spondylodiscitis is characterized by a remarkable heterogeneity in terms of severity that depends on the pathological pattern of each patient. The aim of the treatment is to eliminate the infection (antibiotic therapy), to restore the functionality and mobility of adjacent structures (surgical intervention) and, last but not least, to relieve hyperalgesia experienced from the early stages. Antibiotic therapy remains the mainstay of spondylodiscitis treatment and is initiated only after identification and isolation of the incriminating pathogen. The recommendations of

the Infectious Diseases Society in the USA (IDSA) emphasize the need for intravenous antibiotic therapy for 6 weeks (or oral administration as an alternative in case of good oral bioavailability); this aspect is also emphasized by the OVIVA study. Of the classes of antibiotics available, quinolones and clindamycin are preferred instead of beta-lactams (the latter having a more limited bioavailability). However, regarding infections with staphylococci or enterococci sensitive to beta-lactams, flucloxacillin, cefazolin, ceftriaxone or even penicillin (in the case of Enterococci spp) can be used. For MRSA infections, glycopeptide compounds such as vancomycin or alternatively daptomycin or linezolid are preferred. In addition to antibiotics, an important role in the treatment of spondylodiscitis is played by analgesics and conservative treatment (fixed or mobile orthoses). The latter provide satisfactory results in terms of quality of life, compared to a period of 2 and a half years post diagnosis (de Graeff et al 2017; Gentile et al 2019; Herren et al 2017; Pola et al 2017; Yoshimoto et al 2011). Bernard et al published an article that looked at the benefits and drawbacks of antibiotic therapy duration. The study included 359 patients with spondylodiscitis who were clinically and biologically monitored for 6 and 12 weeks, respectively. One-year outcome showed no benefit of high-frequency antibiotic therapy. (Bernard et al 2015). Herren et al outlined several factors that could lead to failure or unsatisfactory results of antibiotic therapy (age > 75 years and MRSA as the etiological agent) (Herren et al 2017). In the presented case, blood cultures were positive for coagulase-positive staphylococcus, with 7-week treatment duration, supplemented with pain reliever and ortezalombostat.

Besides infection control, the main purpose of surgical treatment is to restore segmental stability from a morpho-functional point of view. There are recommendations regarding surgical approaches (anterior, posterior or associated); it is also cited that minimally invasive transpedicular stabilization combined with debridement would lead to a faster recovery (Herren et al 2017). In most cases the motor segment is affected, which will subsequently lead to instability, reduced mobility and nerve compression that will require surgical intervention (Cottle et al 2008; Menon et al 2014). Herren et al propose a series of criteria for surgical treatment, the indications being: the presence of a neurologic deficit, the presence of abscess or empyema, sepsis, the ineffectiveness of conservative treatment and the morpho-functional instability of the spine. The morpho-functional instability component includes: segmental kyphosis > 15°, translation > 5mm and collapse of the vertebral plateaus > 50% (Herren et al 2017). Following the evaluation of the risks and benefits of the presented case, the need for surgical intervention was discussed. Surgical timing was decided given the associated risk factors (advanced age, liver cirrhosis, renal failure, diabetes, recent bleeding events, severe thrombocytopenia) and the unpredictable postprocedural evolution.

Differential diagnosis represents a real challenge given the location of the infection and the ability to mimic other pathologies. Depending on the imaging option, differential diagnosis of spondylodiscitis must include rheumatic pathology (with polymyalgia), vertebral hemangioma, active osteochondrosis, vertebral tumor processes (with morpho-structural destruction), vertebral fractures and ankylosing spondylitis (Herren et al 2017, Skanjeti et al 2012; Treglia et al 2012). Contrast-enhanced

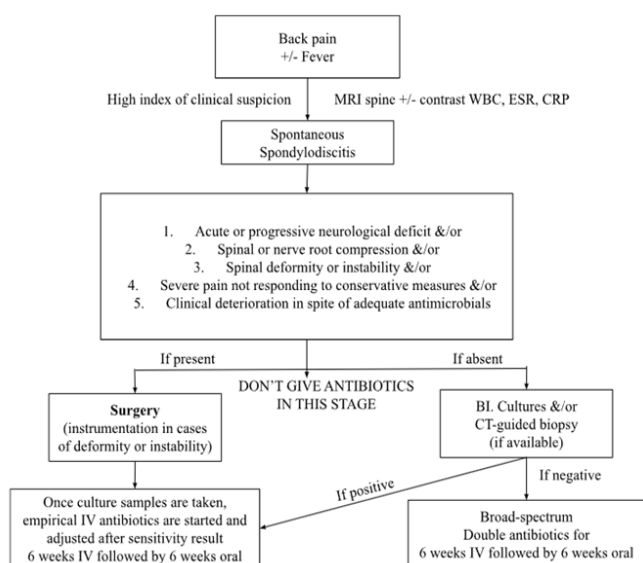


Fig.4. Diagnostic algorithm (Adapted from Waheed et al 2019)

MRI is a viable option as it distinguishes between degenerative changes in the spine, neoplastic processes and spondylodiscitis (Herren et al 2017). More recent studies consider MRI as equivalent to PET-CT, but which has the advantage of differentiating between degenerative changes and the presence of bone marrow edema in the vertebral body and hypervascularization. Considering that PET-CT has low specificity in differentiating spondylodiscitis from neoplastic processes and post-traumatic bone lesions, MRI remains the most viable option. (Herren et al 2017; Skanjeti et al 2012; Stoop et al 2021). The prognosis of spondylodiscitis without neurological implications is favorable only if antibiotic therapy is correctly and promptly initiated (Herren et al 2017). Regarding subsequent evolution, a systematic review followed the evolution of 143 patients with spondylodiscitis (49% men and 9% women) aged between 34 and 89 years, with the average age of onset of complications of 61.3 years. Of the patients included in the study, one-third developed localized collections (abscess) and less than one-fourth presented structural and functional (bone or ligament) instability (Gentile et al 2019). Different predictive factors for a poor prognosis in terms of mortality are cited in the literature (advanced age, presence of neurological deficit since admission and MRSA as an etiological agent). If inadequate treatment is used, mortality can even increase above 20% (Fantoni et al 2012; Herren et al 2017). Surgical treatment often causes complications, especially in the first 6 months, with a reserved 2-year prognosis (even in case of compliance with subsequent indications and warnings (Herren et al 2017).

## Conclusion

Spondylodiscitis is a vertebral infectious entity with a high risk of delayed diagnosis. It is recommended that any change in the character of back pain, new-onset fever, or any new minor neurological symptoms, including numbness or weakness, be viewed with great suspicion. Patients with multiple risk factors such as diabetes, cirrhosis, and neurologic deficits should undergo imaging and other studies to rule out spondylodiscitis, fractures, osteomyelitis, or epidural abscess.

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