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Case Report

Spondylodiscitis - Related Endocarditis

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A B S T R A C T

Spondylodiscitis refers to an infection of the musculoskeletal system, whereas endocarditis is an infection of the endocardium or heart valves. Through the hematogenous spread of pathogenic microorganisms, one condition can be concomitant with the other. A 35-year-old male patient was admitted to our hospital with complaints of lumbar pain that was later determined to be caused by spondylodiscitis (SD). The patient's hospital stays also revealed the presence of infective endocarditis (IE), as indicated by the presence of vegetation on the tricuspid valve and the tip of the central venous catheter (CVC). The patient's medical history included deep vein thrombosis (DVT), post-COVID syndrome, hepatitis C, thrombosis of the left femoral artery, and a history of intravenous drug use. The patient was treated with antibiotics, and the central venous catheter was removed, with a PICC line placed instead. Key findings and points from this case include the importance of thorough examination and diagnosis of spondylodiscitis and infective endocarditis, as well as the need for customized treatment plans based on the underlying etiology. The use of appropriate antibiotics and surgical intervention can significantly reduce the mortality rates in such cases.

Keywords: Spondylodiscitis; Endocarditis; Hematogenous spread; Antibiotics; Pathogenic microorganisms

Abbreviations: IE: infective endocarditis; SD: spondylodiscitis; CBC: Complete Blood Count; CRP: C- reactive protein; DVT: deep vein thrombosis; CT: computed tomography; TTE: transthoracic echocardiogram; MRI: magnetic resonance imaging; TTE: transoesophageal echocardiogram; HIV: human immunodeficiency virus; ESR: erythrocyte sedimentation rate; WBC: white blood count; CVC: central venous catheter; PICC line: peripherally inserted central catheter; 18F-FDG-PET/CT: Positron emission tomography (PET) with 2-deoxy-2-[fluorine-18]fluoro-D-glucose (^{18}F-FDG)

1. Introduction

The correlation between IE and SD was first established in 1965. Both of these infections are quite challenging to diagnose, and therefore, individuals who are predisposed, and report symptoms as described below, need to be thoroughly examined. Infection arises due to bacteremia, as a result of various causes. To diagnose both conditions, a protocol needs to be followed, including a series of examinations that can reveal an active infection, such as hematologic tests [erythrocyte sedimentation rate (ESR), Complete Blood Count (CBC) and C- reactive protein (CRP)], blood cultures to identify the pathogenic

agent, and imaging studies that may reveal abnormalities. It is critical to identify the etiological factor, for the therapeutic plan to be targeted and customized for the specific pathogen. With the use of appropriate antibiotics, mortality has significantly decreased, while surgical intervention takes place in cases where it is deemed necessary. In this article, we present the case of a 35-year-old man who was admitted to our hospital with findings of spondylodiscitis, and during his hospitalization, concomitant endocarditis was identified.

Case Report

A 35-year-old male patient was admitted to the Emergency Room of AHEPA Hospital with progressively worsening lumbar pain that initially developed four months ago. The patient's medical history included deep vein thrombosis (DVT) of the right lower limb, post-COVID syndrome, hepatitis C, thrombosis of the left femoral artery, and intravenous drug use (IVDU).

The patient was admitted to the Internal Medicine Department. The physical examination showed lumbar pain, with motor difficulty that was attributed to the pain, without any neurological deficit. The laboratory results showed increased CRP levels at 10.21 mg/dl, and anemia (hemoglobin: 10.6 g/dl).

Blood cultures and complete serological tests were performed. A computed tomography (CT) scan with a contrast agent showed significant vertebral endplate lesions in the L3-L4 disc space, architectural changes, subchondral damage, endplate sclerosis, anterior syndesmophyte, and intervertebral disc space narrowing (**Figure 1**).

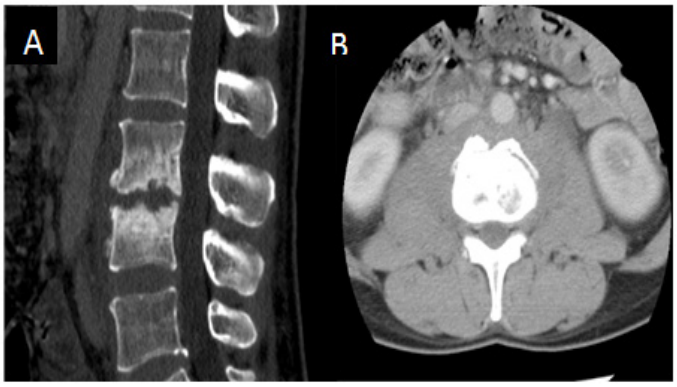

Figure 1: CT of the spine showing endplate sclerosis and irregularity (A). Mildly enhanced soft tissue between the spine and abdominal aorta (B).

Ceftriaxone, vancomycin, and gentamycin were initiated. Following the neurosurgical assessment, a three-point thoracolumbar brace was applied.

Transthoracic echocardiography (TEE) showed agile mitral valve formation. A transesophageal echocardiogram (TOE) was scheduled to further assess whether it was a vegetation or an artifact, which was not completed; however, due to lack of cooperation. Magnetic resonance imaging (MRI) confirmed spondylodiscitis in the L3-L4 disc space (**Figure 2**).

On the tenth day of hospitalization, the patient developed a high body temperature (37.5°C). The central venous line was replaced, a new set of blood and urine cultures were taken, and chest radiography was performed. The treatment was changed to piperacillin-tazobactam and daptomycin.

During the administration of piperacillin-tazobactam, the patient developed a maculopapular rash throughout his body and was treated with systemic administration of antihistamines (levocetirizine). A new MRI scan showed bone marrow edema and contrast enhancement of the L3 and L4 vertebral bodies, pedicles, pars interarticular, facet joints, and intervertebral discs (**Figure 3**).

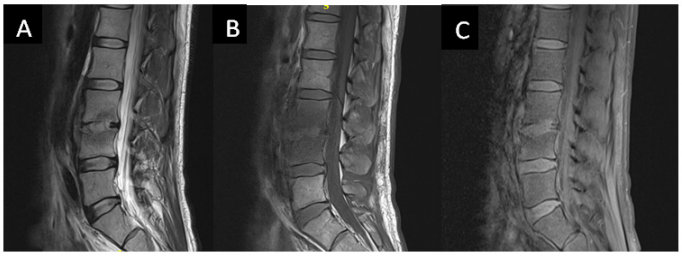

Figure 2: Spine MRI. The initial MRI showed a high T2 signal (fluid) in the L3-L4 disc space (A) and a high T2 signal/low T1 signal in adjacent end plates (bone marrow edema, B) with concomitant loss of low-signal cortex at the end plates and contrast enhancement (C).

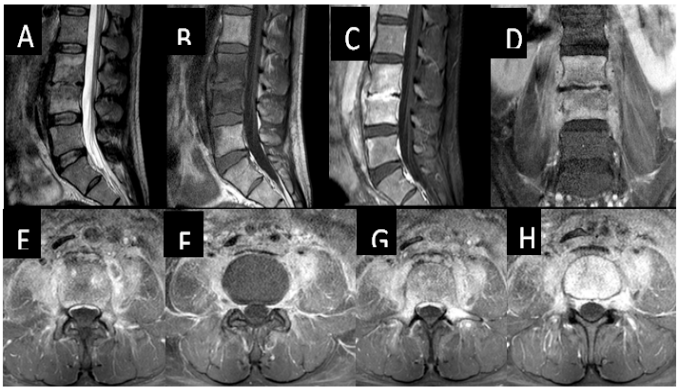

Figure 3: Spine MRI revealed bone marrow edema (A) and contrast enhancement of the L3 and L4 vertebral bodies, pedicles, pars interarticular, facet joints, and intervertebral disc (C, G). Inflammatory changes and enhancement of the adjacent psoas (D) with small intramuscular abscesses (E).

Concomitant leptomeningeal enhancement (H). The second bone marrow lesion in the L5 body (C). On the 20th day of hospitalisation, the patient again developed high body temperature (39 °C) and complained of chest pain. He also reported numbness in the right upper limb and right lower limb combined with spontaneous and involuntary movements of the right upper limb that lasted approximately 5 min. We suspected endocarditis as the initial diagnosis. A new set of blood cultures were obtained. A transoesophageal echocardiogram (TOE) followed, which revealed vegetation on both the tricuspid valve and the tip of the central venous catheter (CVC), thus confirming the diagnosis of endocarditis. In such a case, we are dealing with right heart involvement; the patient has some artificial material on the right side of the heart (vascular port, as in this case). The CVC was immediately removed. A PICC line (peripherally inserted central catheter) was placed through the basilic vein of the arm. The antibiotic treatment was changed to meropenem, vancomycin, gentamicin, and micafungin. Neurological assessment and brain CT showed no significant findings. In the lungs, endocarditis on the right-side cause's lung abscesses. Interestingly, lung computed tomography (CT) screening results revealed no abnormalities or indications of disease. Brain magnetic resonance imaging (MRI) revealed findings indicating possible septic emboli (**Figure 4**).

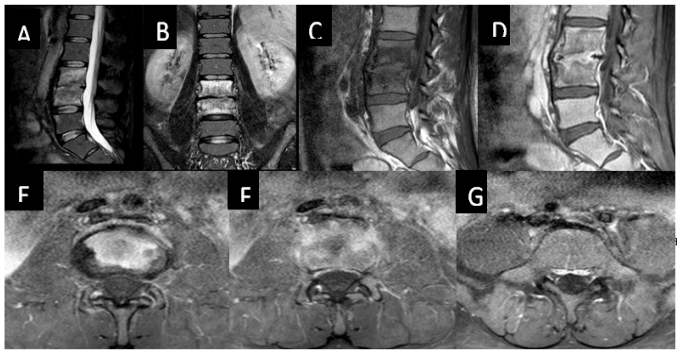

Figure 4: Spine MRI on 21st day depicted a further significant reduction in paravertebral inflammation (F) and dural enhancement (G). Bone marrow edema is also slightly reduced (A, B).

A new spinal MRI on the 30th day of hospitalisation showed significant improvement (**Figure 5**). A significant observation in this case was the absence of pathogenic organisms. Moreover, the patient recovered fully without any complications, which contradicts the initial prognosis.

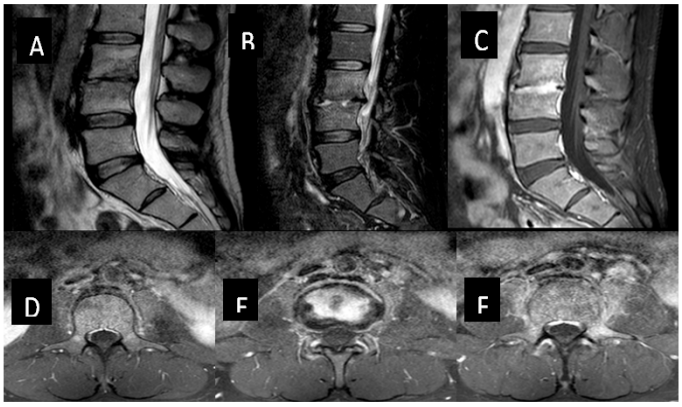

Figure 5: Spinal MRI on the 30th day showed a further reduction in bone marrow oedema (B). Adjacent psoas inflammatory changes are significantly reduced (F).

Discussion

Spondylodiscitis is an infection involving the intervertebral disc and adjacent vertebral bodies, resulting from pathogenic microorganisms that migrate through the bloodstream, either venous or arterial, with the latter being more common[1]. Additionally, it can be attributed to infections from a distant site (endocarditis, abscess, urinary tract infection, pneumonia, or pelvic infection), and it can develop postoperatively, following surgery at a distant site (pelvic, urological, vascular, cardiac, or internal organ surgery), with a local infection becoming systemic or arising from intravenous use of illicit drugs[2]. The most affected age groups are individuals under 20 years old, especially those infected with the human immunodeficiency virus (HIV), and adults aged 50-70 years[1].

When an intervertebral disc is infected, the condition is referred to as discitis, and when the infection involves the endplate, it is referred to as osteomyelitis or spondylitis. Typically, at the time of diagnosis, both anatomical regions are affected; hence, the term spondylodiscitis is used in most cases (95%), which affects the vertebral body more than the posterior elements (5%)[2].

Infections of the spinal column are usually classified according to the causative microorganism, such as pyogenic, parasitic, fungal, or tuberculous. Tuberculosis has historically been the primary cause of spinal infections; however, due to the successful diagnosis and treatment of pulmonary tuberculosis, its incidence has decreased over the last 50 years[3]. According to an analysis by Grammatico L, et al., Staphylococcus spp. are the main infectious agents that cause spondylodiscitis (38%); gram-negative bacilli, including enterobacteria (20-30%); and streptococci, with Mycobacterium tuberculosis and Brucella spp. being less prevalent[4].

However, as observed in this case, as many as one-third of the infectious agents may remain unidentified[3]. Additionally, pyogenic spondylodiscitis predominantly affects the lumbar region of the spinal column (58%), followed by the thoracic (30%) and cervical (11%) regions[4].

The adult intervertebral disc, being avascular, undergoes involution of intraosseous anastomosis around the third decade of life. With aging, this leads to the release of septic emboli, causing extensive vascular bone infarcts and spreading of infection to adjacent structures, resulting in characteristic imaging findings of spondylodiscitis, including erosion[4].

Patients suffering from spondylodiscitis develop local pain, accompanied by muscular spasms that are attributable to inflammation, fever, and neurological deficits. The clinical picture is nonspecific, with low-specificity indicators and symptoms, leading to delayed diagnosis, particularly in the tuberculous form, as well as delayed treatment[1]. Literature data report a delay of 2-6 months between the onset of initial symptoms and diagnosis[5,6]. Diagnosis is challenging, and clinical, laboratory, and imaging findings[1].

In clinical practice, the laboratory parameters that specialist medical practitioners focus on mostly refer to elevated levels of inflammatory markers, such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), where, similar to the case under study, values exceed the normal limits[5]. ESR is a sensitive indicator of infection, and it is high in the majority of patients (90%). However, it is a nonspecific marker of infection. CRP is an acute-phase protein with greater specificity than ESR and is also observed at increased levels in 90% of patients with spondylodiscitis[1].

The white blood cell count (WBC) may be elevated or within the normal range in patients with spinal column infection. However, although it is not a specific infection marker for patients with spondylodiscitis, it should be included in the diagnosis and treatment process, especially in cases accompanied by fever, thus providing general information about the patient's course of treatment[7].

The imaging examinations that are conducted in each case are crucial in disease diagnosis. Although it has low specificity (57%), a radiographic examination should be performed to all patients with suspected spondylodiscitis. In advanced cases, it may reveal abnormalities in the end-plates of the vertebrae, with eventual fragmentation, and reduced height of the intervertebral disc[5].

Axial tomography (CT) remains the preferred examination for evaluating bone changes, including the identification of early alterations in vertebral end-plates, the presence of bone necrosis, and pathological calcifications that indicate tuberculosis[5].

The only imaging method that combines high sensitivity (97%) with satisfactory specificity (94%) is gadolinium-enhanced magnetic resonance imaging (MRI), which provides

detailed anatomical information about the surrounding soft tissues, i.e., highlighting bone abnormalities, and detecting paraspinal or epidural abscesses[9]. It is highly useful in the early stages of infection when other imaging methods remain inconclusive (e.g. radiography)[5].

In cases of negative blood cultures, a recommended patient management algorithm involves withholding antibiotics until a CT-guided biopsy/aspiration is performed. Biopsy samples should be sent for polymerase chain reaction (PCR) testing, and checked for Gram stain, aerobic, and anaerobic cultures, tuberculosis, and fungi. The accuracy of CT-guided spinal biopsy is approximately 70%, with false negatives being possible, in cases of insufficient tissue sampling or antibiotic administration. Repeat biopsy may be considered, if antibiotic cessation is deemed safe and initial biopsy results are negative[7].

The most common comorbidities were sepsis (27%), and endocarditis (9%)[4]. On the other hand, infectious endocarditis is a rare, but highly morbid, and fatal, condition (30%)[9]. It is characterized by infection of a native or prosthetic cardiac valve, endocardial surface, or permanent cardiac device, causing inflammation in the heart's endothelium[10].

The infection is associated with healthcare interventions, resulting from the use of intravascular catheters, and intracardiac devices. The most common cause of the disease is the following pathogenic microorganisms, collectively accounting for 90% of endocarditis cases. Specifically, Staphylococcus aureus constitutes 26.6% of cases, followed by viridans group streptococci, at 18.7%, other streptococci, at 17.5%, and enterococci, at 10.5%[9].

The clinical picture varies, and clinicians should be alert to symptoms for prompt diagnosis. It may manifest as an acute and rapidly progressing infection, or as a subacute or chronic disease, with low-grade fever, and nonspecific symptoms that can be misleading. Fever, night sweats, fatigue, weight loss, and anorexia are common, with up to 85% of patients having cardiac murmurs. Notably, embolisation of the brain, lungs, and spleen occurred in 30% of the patients. Special attention should be paid to patients with predisposing risk factors (protrusion of the mitral valve, congenital heart disease, previous endocarditis, patients with implanted cardiac devices, etc.). Some of the comorbidities of infectious endocarditis include intravenous drug use, chronic kidney disease (especially in patients undergoing hemodialysis), chronic liver disease, advanced age, corticosteroid use, uncontrolled diabetes, central venous catheter, and immunocompromised status (including HIV infection)[9].

Diagnosis of infectious endocarditis relies on modified Duke Criteria (**Table 1**), with imaging studies, such as CT, MRI, and 18F-FDG PET/CT, playing a crucial role[11]. Transesophageal echocardiography (TOE) is performed to confirm diagnosis, in cases of no diagnostic transthoracic echocardiography (TTE) with a high clinical suspicion, especially in cases involving prosthetic devices, and Staphylococcus aureus bacteremia. Imaging repetition is generally unnecessary during treatment unless clinical deterioration or complications occur[9].

Table 1: Major and Minor Criteria for the diagnosis of infective endocarditis, and its complications.

Major Criteria		
Microbiological Criteria		
Isolation of typical microorganisms responsible for infective endocarditis (IE), from two different blood cultures. The microorganisms include Streptococcus bovis, the HACEK group, Staphylococcus aureus, and Enterococcus.	Persistent positive blood cultures, indicating the presence of microorganisms that are compatible with the diagnosis of IE. The criteria include 2 blood cultures taken with a minimum 12-hour interval in-between, or 3 positive blood cultures taken at different times. Additionally, isolation of microorganisms from most blood cultures, when four or more are taken, with the first and last cultures separated by at least one hour	Detection of Coxiella burnetii in a single positive blood culture, positive PCR, or serological evidence of Q fever.
Evidence of Myocardial Involvement		
Development of valve insufficiency not previously present. Exacerbation or alteration in pre-existing murmurs is insufficient.	Positive echocardiogram for microbial endocarditis, involving the visualization of intracardiac masses that are consistent with vegetation, without alternative anatomical explanations, or the presence of abscess.	New valvular regurgitation in prosthetic valves.
Minor Criteria		
Pre-existing cardiac damage or use of intravenous toxic substances.		
Fever ≥38.0°C		
Vascular phenomena	Arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, Janeway lesions	
Microbiological evidence	Positive blood cultures (different from those outlined in major criteria) or serological evidence of an ongoing infection with microorganisms compatible with the diagnosis of microbial endocarditis.	
Immunological phenomena	Glomerulonephritis, Osler's nodes, Roth spots, rheumatoid factor.	

Collaboration between various medical specialties, including cardiologists, internists, infectious disease specialists, neurosurgeons, neurologists, and cardiothoracic surgeons, is essential, for both the diagnosis and management of infectious endocarditis. Coordination among these specialists leads to prompt referral with appropriate therapeutic (conservative or surgical) protocols and close patient monitoring to reduce mortality (**Table 1**)[9].

Spondylodiscitis (SD) is a recognized complication of IE, possibly stemming from the hematogenous spread of infectious agents[12].

According to studies by Del Pace S. et al. and Carbone A. et al. a significant percentage of IE patients experience SD (8-30%). The correlation between these conditions is common, with specific microorganisms involved. In particular, Streptococcus Viridans and Enterococcus are prevalent in IE and SD cases. Neurological symptoms and embolic phenomena are observed in both conditions.

The majority of cases of endocarditis reported involvement of the aortic valve at a rate of (53%), with mitral (40%) and tricuspid (7%) valve involvement being less prevalent. In cases of spondylodiscitis, intense lumbalgia (low back pain) (26.90%)

was noted[13]. Timely diagnosis is crucial and relies on advanced imaging techniques such as magnetic resonance imaging (MRI) and PET/CT[14].

Additional studies by Viezens L. et al. emphasize the value of echocardiography and TOE to the diagnostic process, demonstrating a tenfold increase in diagnosed IE, after applying transesophageal echocardiography (TOE) to patients with known spondylodiscitis[12].

Therapy is based on combined antimicrobial treatment, according to the sensitivities of the isolated pathogen cultured from material obtained through paracentesis and/or blood cultures. Antibiotics, including penicillin and first-generation cephalosporins, are used to treat common infectious organisms such as Staphylococcus and Streptococcus. For immunocompromised patients and intravenous drug users, broad-spectrum antibiotics are added, such as third-generation cephalosporins with better coverage against Gram-negative bacteria[3].

Clindamycin, vancomycin, quinolones, tetracycline, and cotrimoxazole also exhibit good bone-penetrating properties and should be considered in the treatment of spinal infections, especially in patients sensitive to beta-lactam antibiotics. Antibiotic treatment for less than four weeks may result in high recurrence rates. Empirical antibiotic therapy is recommended in the absence of pathogen detection, and the antibiotic spectrum should cover S. aureus and E. coli, which are the most common pathogens for pyogenic spondylodiscitis, taking into account local epidemiology and the possibility of colonization by resistant organisms[2,3].

The discontinuation criteria for treatment include the restoration of clinical and inflammatory markers, such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). CRP is considered a better indicator of treatment response, and its reduction is faster than that of ESR[15].

Hsiu-Yin Chiang et al. suggest that the initial ESR value and its variability in the first 4 weeks are useful indicators for predicting the duration of treatment and recurrence of spondylodiscitis. However, ESR levels normalize slowly and irregularly, even after successful infection treatment[15].

Surgical intervention was performed according to specific indications such as the presence of neurological deficits and deformity. Absolute surgical indications include spinal instability due to extensive bone destruction, severe kyphosis, and failure of conservative treatment. However, specific criteria, such as age and the general condition of the patient, should be considered[5].

According to Yoshimoto et al., in a study of 45 patients aged 65 years and older with pyogenic spondylodiscitis, some of whom presented with paralysis but were not operated on because of poor general health conditions and relevant problems, paralysis improved with conservative treatment in a significant percentage of the patients[16].

The use of antibiotics significantly reduced IE mortality of infective endocarditis. However, their use requires good renal function, which should be adjusted accordingly. The guidelines for empirical therapeutic regimens mention amoxicillin intravenous in stable patients, because it has better activity against enterococci and many HACEK microorganisms than benzylpenicillin. The role of gentamicin (IV) remains controversial until positive blood cultures are obtained, and should be used with caution in cases of nephrotoxicity or renal impairment. In severe sepsis without concerns for enterobacteria and Pseudomonas, vancomycin is preferred, and daptomycin is administered in allergic patients. In severe sepsis and risk factors for multidrug-resistant enterobacteria and Pseudomonas, vancomycin (IV) and meropenem are administered[17].

In several cases, we are dealing with right heart involvement, either with a fistula (emptied abscess) from the left heart or with a drug addict, or the patient has some artificial material on the right side of the heart (pacemaker leads, ICD, vascular port, as in this case). Vegetation on the right side can be removed endovascularly and is both curative and diagnostic (this can be seeded if vein cultures are still negative).

Regarding spondylodiscitis, patients who do not improve with conservative treatment and experience recurrence should undergo prompt surgery[18].

Conclusion

Although the association of infective endocarditis with spondylodiscitis is not rare, short-term mortality in infective endocarditis still exceeds 15%. Diagnosis is challenging, and clinical, laboratory, and imaging findings should be considered. The main targets of treatment are the isolation and identification of microorganisms, prevention of bacteremia and sepsis, elimination of infection, long-term pain relief, reversal of neurological deficits, restoration of spinal stability, and prevention of relapse. Rapid diagnosis and conservative or surgical treatment are key prerequisites for positive outcomes.

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Patient consent: The patient has given written consent to the inclusion of material pertaining to himself; he acknowledged that he cannot be identified via the paper, and we have fully anonymized the case report.

Ethical approval: Not applicable

Authors' contributions

AM: primary case-management, literature review and manuscript preparation.

EP, CK, VK: literature review

All authors read and approved the final manuscript.

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