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Case Report

Clinical case report: discitis osteomyelitis complicated by inferior vena cava venous thrombosis and septic pulmonary emboli

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ABSTRACT

Viridans group streptococcus is an infrequent cause of osteomyelitis that is found in association with infective endocarditis. Only a few studies report viridans osteomyelitis in the absence of endocarditis. Vertebral pyogenic osteomyelitis can sometimes be complicated by psoas or paraspinal abscesses. These intra-abdominal and/or pelvic collections can very rarely result in venous thrombosis. A paraspinal abscess resulting in inferior vena cava (IVC) thrombosis has only been reported once in the literature. We report a case of a young female with a history of polysubstance abuse and chronic back pain, who was found to have extensive vertebral osteomyelitis and discitis with epidural, paraspinal, and psoas abscesses caused by viridans streptococci. These abscesses compressed on the IVC causing IVC thrombophlebitis extending to the iliac veins distally. Imaging also demonstrated multifocal bilateral septic pulmonary emboli and pleural effusions secondary to septic IVC thrombus; a transesophageal echocardiogram showed no evidence of infective endocarditis.

Introduction

Intravenous (IV) drug use is a risk factor for vertebral osteomyelitis and discitis. Studies have reported an increasing incidence of vertebral osteomyelitis; some of the etiologies for this change in incidence include increased instrumentation and indwelling catheters, aging population, and rising IV drug use [1,2]. Psoas and paravertebral abscesses, as a complication of osteomyelitis, are frequently associated with Pott's disease; however, pyogenic vertebral osteomyelitis can also result in formation of these abscesses [3]. Such intra-abdominal collections can in rare occasions cause compression of adjacent venous structures and result in deep vein thrombosis [4–8].

Case report

A 39-year-old woman with a history of polysubstance use including IV drug abuse presented to the emergency department and was found to be disoriented and agitated with shortness of breath. She also reported chronic back pain that

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had worsened recently and new right leg pain. In addition, there was a recent history of heroin and amphetamine inhalation.

She was febrile with a pulse of 100/min and respiratory rate of 20/min. Physical examination revealed 5/5 strength in upper extremities and 4/5 strength in bilateral lower extremities. Saddle anesthesia was also noted in a S2-3 distribution.

She had a white blood cell count of 24,000 $\times$ 10^3/mcL with an absolute neutrophil count of 22.0 $\times$ 10^3/mcL, bands 6%, and hemoglobin of 6.7. Head computed tomography (CT) was normal at this time, and urine toxicology screen was positive for amphetamine and benzodiazepines. Chest x-ray showed bibasilar opacities. Her blood culture came back to be positive for gram-positive cocci which was later confirmed to be viridans group streptococcal infection.

With acutely worsening back pain, right leg pain, and signs of cord compression on physical examination, magnetic resonance (MR) imaging of thoracic and lumbar spine was performed which demonstrated active discitis osteomyelitis at the L4-5 level with paraspinal, right psoas and anterior epidural abscesses and resultant severe canal stenosis with compression of the cauda equina nerve roots (Fig. 1). In addition, there were extensive conglomerate necrotic masses in the paraspinal region, highly suggestive of necrotic lymphadenopathy and/or infected fluid collections. Furthermore, thrombosis within the vena cava extending to bilateral iliac veins was identified (Fig. 1).

Limited visualization of the brain on the MR counting sequence demonstrated T2 hyperintense lesions in a periventricular distribution, which were not present on the initial emergency department CT scan performed a few days before the MR examination. In addition, a long segment of T2 hyperintense cord signal abnormality extending from T8 to T10 was also identified. Unfortunately, dedicated imaging to better characterize these lesions was unable to be performed as the patient became agitated and refused further imaging.

Bilateral pleural effusions were also found on this study, and thus, further imaging with a CT of chest, abdomen, and pelvis was performed (Figs. 2-4). The study demonstrated multiple cavitary rounded collections containing gas in the right lower lung. Additional rounded opacities were also seen distributed throughout bilateral lung fields suggestive of septic pulmonary emboli (Fig. 3). This study also corroborated the findings of prior MR imaging and redemonstrated venous thrombosis involving inferior vena cava (IVC) and iliac veins (Figs. 2 and 3).

These imaging findings and her viridans bacteremia raised suspicion for infective endocarditis. However, no embolic features were present on physical examination of the extremities. Only a faint early ejection systolic murmur

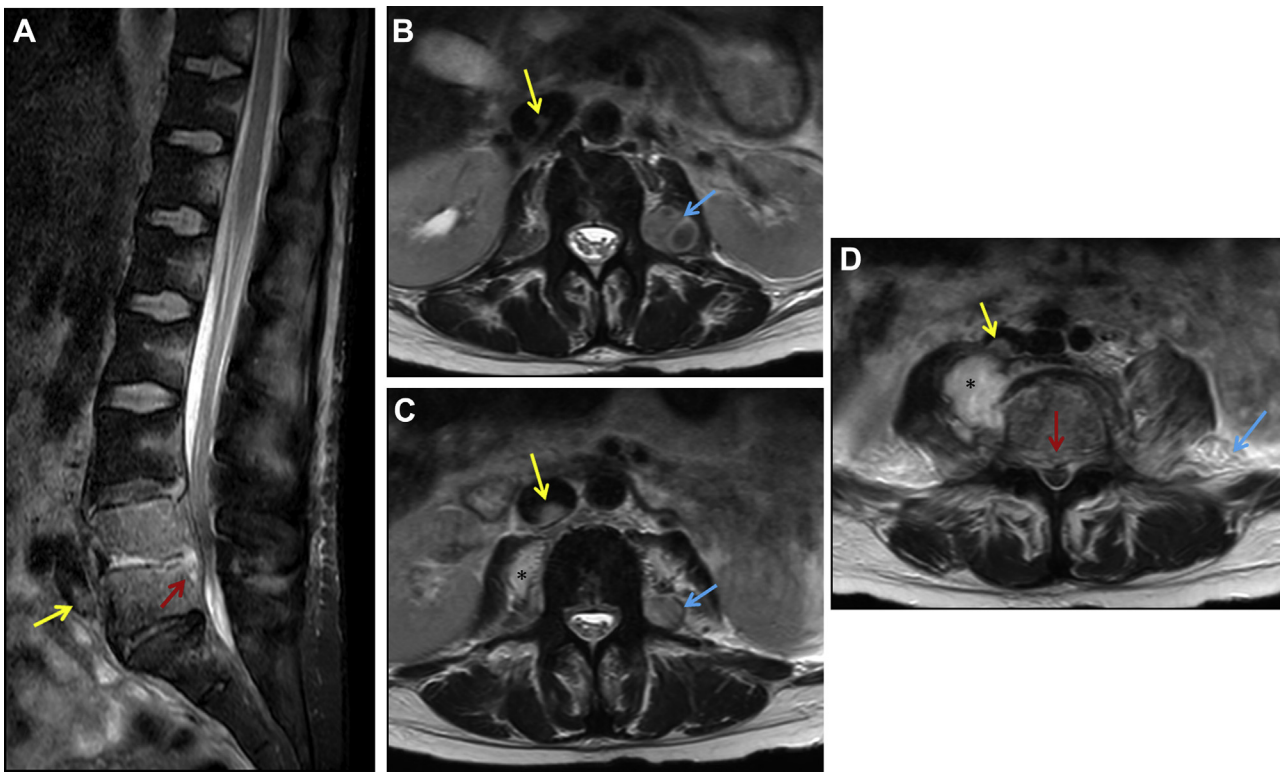

Fig. 1 – Sagittal T2 with fat saturation MR (A) and axial T2 MR (B-D) images from MR imaging of the lumbar spine demonstrate abnormal signal within the L4 and L5 vertebral bodies and L4-5 disc space with extension into the anterior epidural space (red arrows). Extensive paravertebral T2 hyperintense inflammatory change extending into the psoas muscles. T2 hyperintense collection is present within the right psoas muscle consistent with an intramuscular abscess (*) and similar signal collections within the paraspinal region reflecting either additional abscess collections or necrotic lymph nodes (blue arrows). Incidentally noted are T2 hyperintense filling defects within the right IVC and the bilateral iliac veins (yellow arrows).

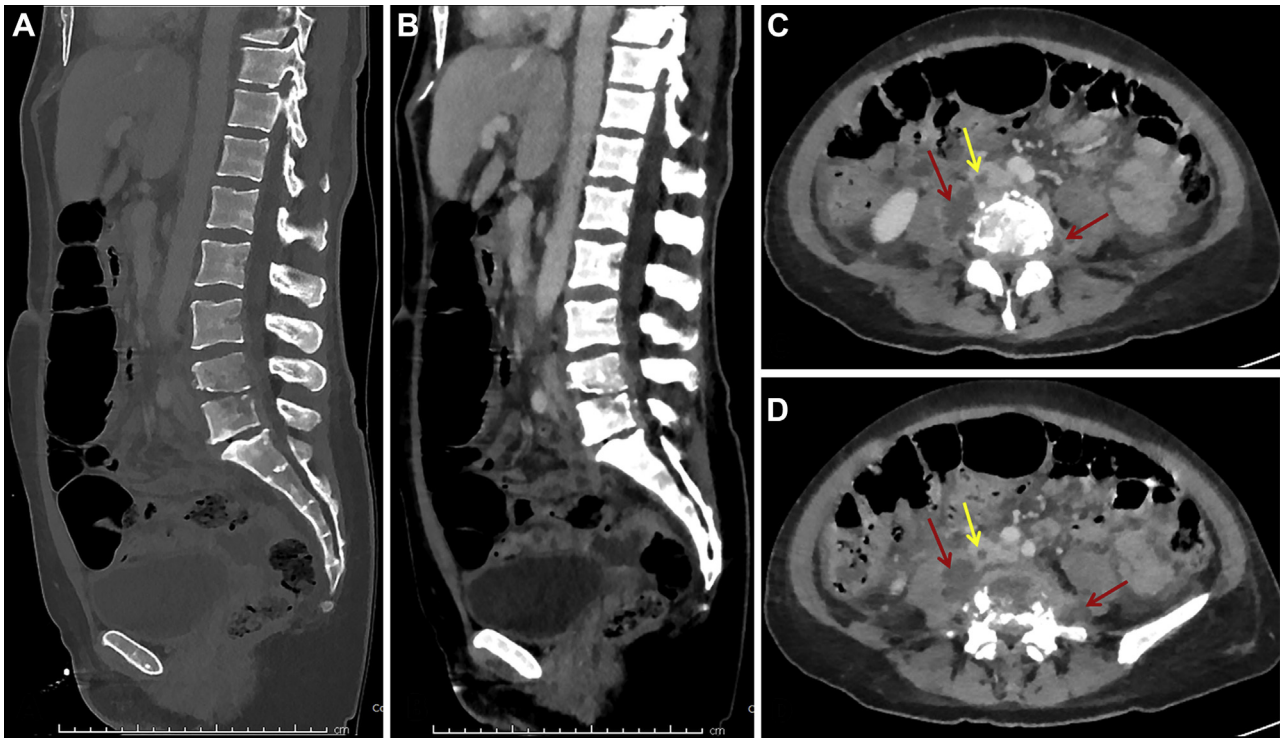

Fig. 2 – Sagittal CT images from abdominal pelvis CT in bone (A) and soft-tissue (B) algorithm demonstrate destructive end plate changes at the L4-5 level. Axial images in soft-tissue algorithm at the L4-5 level (C, D) demonstrate extensive perivertebral soft-tissue inflammatory change, loss of the fat plane between the vertebral bodies, and the psoas muscles as well as multiple paraspinal and psoas fluid collections (red arrows). In addition, there is a filling defect within the right IVC (yellow arrows).

was present that was attributed to her severe anemia; however, no holosystolic murmur was appreciated. In addition to this a transesophageal echocardiogram demonstrated no valve abnormality, thus effectively ruling out infective endocarditis.

The patient was started on IV vancomycin initially but was subsequently transferred to another facility.

Discussion

The incidence of vertebral osteomyelitis is increasing partly due to increase in IV drug use, in addition to other factors [1,2]. The most common organism involved in osteomyelitis in IV drug users is *Staphylococcus aureus*, followed by *Pseudomonas*

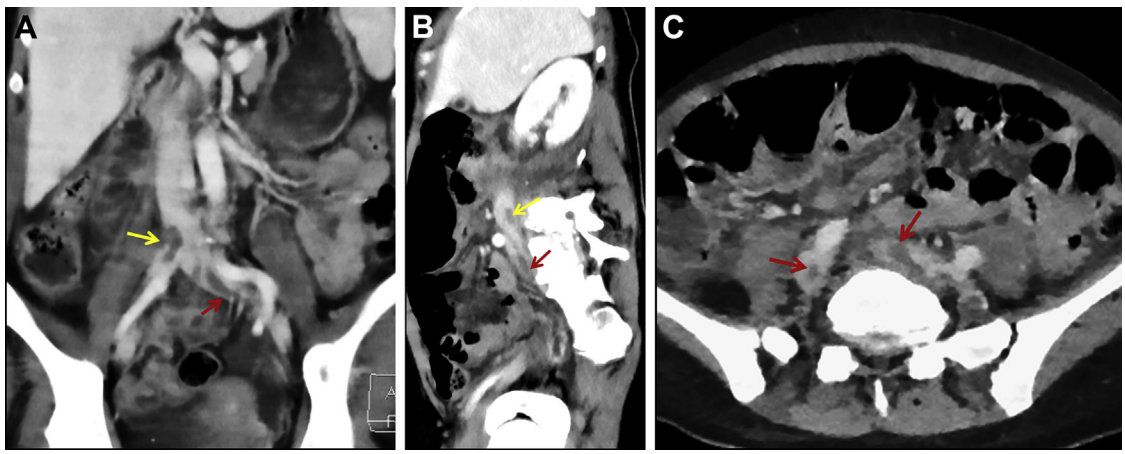

Fig. 3 – Coronal oblique maximum intensity projection (A), sagittal (B), and axial (C) CT images from abdomen pelvis CT in soft-tissue algorithm demonstrate low-density filling defects within the IVC (yellow arrows) and iliac veins (red arrows) consistent with subocclusive thrombus.

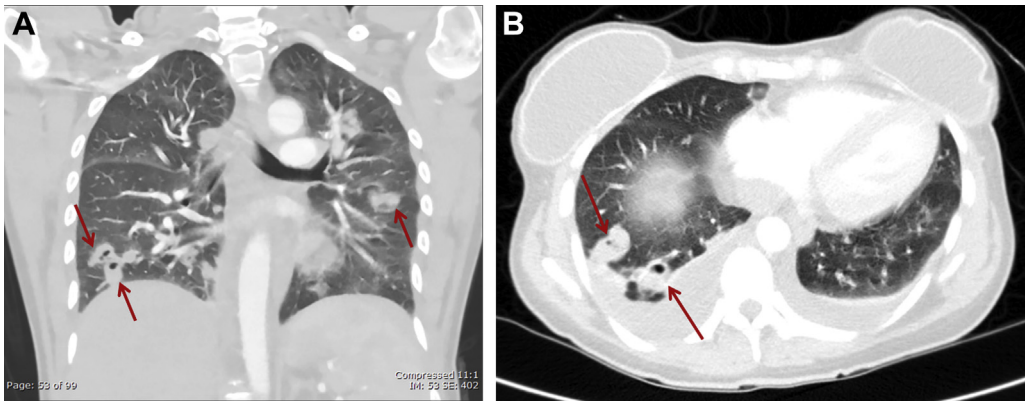

Fig. 4 – Coronal (A) and axial (B) CT images from chest CT in lung algorithm demonstrate numerous cavitary lesions consistent with septic emboli (red arrows).

aeruginosa; however, other organisms have also been identified in these patients [9,10].

First case of viridans osteomyelitis was reported in 1982 [11]. Since then more reports of osteomyelitis caused by viridans group streptococci have been coming up. Viridans osteomyelitis is reported to have a more subacute course [12]. This was corroborated by our patient's presentation as well; as she reported chronic back pain and new right leg pain, and despite imaging evidence of extensive disease, she was clinically functional.

Most cases of viridans vertebral osteomyelitis have been associated with endocarditis [13–16]. However, very few studies report vertebral osteomyelitis caused by viridans streptococci in the absence of infective endocarditis [17,18]. A study also suggests that endocarditis should be ruled out in any case of vertebral osteomyelitis regardless of the causative organism, reporting up to 30% incidence of infective endocarditis in these patients [19].

Psoas abscess is an uncommon condition which can be primary or secondary [20]. Vertebral osteomyelitis is a rare cause of secondary psoas abscess formation. A large number of these cases have been reported in association with tuberculous spine disease; however, other organisms have also been implicated [3]. Similarly, tuberculous osteomyelitis is more frequently associated with paraspinal abscesses as compared to pyogenic osteomyelitis [3]. However, our case demonstrated epidural, paraspinal, and psoas abscesses associated with pyogenic spondylitis.

Most cases of deep vein thrombosis involve lower extremities and thus involvement at the level of iliac veins or above should prompt further investigation to rule out any mass effect or extrinsic pathology [4]. Few cases of psoas abscess resulting in iliofemoral venous thrombosis have been reported [4–8]. We report a rare case of paraspinal abscess causing IVC thrombus, and the first as a result of viridans infection. IVC thrombus can cause bilateral lower-extremity swelling or back pain [21]. In the presence of extensive infectious vertebral disease in our case, it is not possible to ascertain if some of her back pain could be attributed to IVC thrombus; however, she did not have leg swelling on physical examination or any other symptoms from her IVC thrombus.

Our patient also developed extensive multifocal and bilateral septic pulmonary emboli as a result of IVC thrombus; as an infective endocarditis had been ruled out with a transesophageal echocardiogram which showed no vegetation. This suggests septic thrombophlebitis of the IVC. A few cases in adults have been recently reported where septic thrombophlebitis with contiguous infection resulted in septic pulmonary emboli [22,23]. However, most of these cases are either due to *S aureus* or rarely *Fusobacterium necrophorum*; both bacteria produce thrombogenic toxins [22,23]. No case of septic thrombophlebitis associated with viridans streptococcus has been reported.

Conclusions

Viridans bacteremia can occasionally be associated with vertebral osteomyelitis without any evidence of underlying infective endocarditis. Psoas or paraspinal abscesses can cause compression of adjacent venous structures causing thrombosis. This venous thrombosis may have a septic component to it as well due to the overwhelming adjacent infection and can manifest as septic pulmonary emboli. The report also reinforces the importance of radiological imaging in determining full extent of the disease process.

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