Radiology Case Reports 19 (2024) 2719–2723

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/radcr

Case Report

Lumbar vertebral diskitis-osteomyelitis with mycotic abdominal aortic aneurysm caused by Streptococcus mitis ☆,☆☆

Anas Sayed Suliman Atassi, MD[a], *George K. Vilanilam, MBBS*[a,*], *Rangarajan Purushothaman, MD*[a], *Razvan Zemianschi, MD*[a], *Ishan Pandey, BBA*[b], *Kurt J. Messer, MD*[c], *Surjith Vattoth, MD, FRCR*[d]

[a] *Department of Radiology, University of Arkansas for Medical Sciences, Little Rock, AR 72205, USA*
[b] *Baylor Hankamer School of Business, Baylor University, Waco, TX 76706, USA*
[c] *Division of Body Imaging, Department of Radiology, University of Arkansas for Medical Sciences, Little Rock, AR 72205, USA*
[d] *Diagnostic Radiology & Nuclear Medicine, Division of Neuroradiology, Rush University Medical Center, Chicago, IL 60612, USA*

ARTICLE INFO

Article history:
Received 8 December 2023
Accepted 12 February 2024

Keywords:
Osteomyelitis
Aortic aneurysm
Mycotic aneurysm
Discitis
Lumbar spine

ABSTRACT

Vertebral osteomyelitis is a well-documented disease entity in literature with various known etiologies. However, vertebral diskitis-osteomyelitis secondary to an infected aortic aneurysm is an uncommon and life-threatening complication. We present the case of a 65-year-old male patient who presented with chronic low back pain that acutely worsened for 1 to 1.5 months and was diagnosed with vertebral diskitis-osteomyelitis secondary to a contiguous infection from an adjacent mycotic aortic aneurysm. To our knowledge, this is one of the few cases reported of vertebral diskitis-osteomyelitis secondary to mycotic aortic aneurysm. We discuss the findings on CT and MRI, as well as the value of imaging in guiding management.

Introduction

Vertebral osteomyelitis is a well-documented disease entity in literature with various known etiologies. However, vertebral diskitis-osteomyelitis secondary to an infected aortic aneurysm is an uncommon and life-threatening complication. Infected aortic aneurysms carry a reported prevalence ranging from 0.6%-2.6% [1–3]. An infected aortic aneurysm can result from a primary infection from a direct or

☆ Acknowledgments: The authors received no financial support for this article's research, authorship, and/or publication.
☆☆ Competing Interests: Drs. Atassi, Vilanilam, Purushothaman, Zemianschi, Messer, Vattoth, and Mr. Pandey declare no potential conflicts of interest concerning this article's research, authorship, and/or publication.
* Corresponding author.
E-mail address: George.koshy.vilanilam@gmail.com (G.K. Vilanilam).
https://doi.org/10.1016/j.radcr.2024.02.039

lymphatic spread of infection involving the aortic wall. This usually involves a diseased portion of the aortic wall with either a preexisting aneurysm or traumatic pseudoaneurysm. Secondary infection is due to vascular spread through bacteremia or septic emboli involving the vasa-vasorum. This can involve an area with a pre-existing aneurysm, pseudoaneurysm, or plaque. Eventually, damage to the intimal and medial layers, as a result, can progress to develop an aneurysm if not already present [2]. Mycotic aortic aneurysms (MAA) carry a high complication rate with a risk of rupture reaching around 60% [4]. Furthermore, mycotic aneurysms carry a risk of showering emboli, sepsis, and multiorgan failure [4]. However, contiguous spread to involve the vertebrae is a dangerous complication that is also under-reported in the literature. We present a case of vertebral diskitis-osteomyelitis secondary to contiguous spread from an adjacent mycotic aortic aneurysm.

Case presentation

A 65-year-old male with a history of hypertension, abdominal aortic aneurysm, and peripheral vascular disease, including right external iliac artery and left superficial femoral artery stent placements, presented with chronic low back pain that acutely worsened over a period of 1-1.5 months. The patient reported that the pain was 7/10 in severity, exacerbated with activity and standing, and radiated to the lower extremities bilaterally to the level of the calves. There was no history of numbness or tingling. Physical examination was unremarkable except for mildly increased blood pressures up to 154/78 mmHg. Focused neurological examination did not reveal any abnormality. Laboratory evaluation demonstrated mild leukocytosis with a WBC level of 9.76 K/uL (normal range: 3.6-9.5 K/uL) and elevated inflammatory markers such as erythrocyte sedimentation rate (ESR) of 47 mm/hour (normal range: 0-20 mm/hour) and C-reactive protein (CRP) of 29 mg/L (normal range: less than 10 mg/L).

A contrast-enhanced magnetic resonance imaging (MRI) of the lumbar spine revealed a large 8 × 7 cm fusiform infrarenal abdominal aortic aneurysm at the L3 vertebral body level (Fig. 1A). An extensive hypointense nonenhancing soft tissue was also present posterior to the abdominal aortic aneurysm lumen and anterior to the vertebral bodies (Fig. 1A). There was edema and contrast enhancement in the L3 vertebral body, predominantly on the right side, with associated destruction of the anterior third of the L3 vertebral body and the anterior margin of the L4 vertebral body (Fig. 1B). T2/STIR edema without postcontrast enhancement was noted in the L3-L4 disc space. Additionally, there was a ring-enhancing collection in the left psoas musculature measuring up to 4 cm, concerning for an abscess. These findings were worrisome for a large mycotic aneurysm and lumbar L3-L4 diskitis, osteomyelitis, and erosion, along with a periaortic abscess extending into the left psoas musculature (Fig. 1C). A subsequent computed tomography (CT) angiography of the abdomen, pelvis, and lower extremities demonstrated similar findings, along with patent right external iliac artery stent, patent left SFA stent, and tandem multifocal mild stenosis of the right SFA with patent 3-vessel runoff to the level of the ankles bilaterally (Fig. 2).

The patient was initially treated with empiric broad-spectrum antibiotics. The patient underwent a biopsy and drainage of the left psoas collection, which grew *Streptococcus mitis*. The patient subsequently underwent open abdominal aortic aneurysm repair with a rifampin-soaked Dacron tube graft. The retroperitoneum was exposed, and the patient underwent L3-4 anterior complete lumbar diskectomy and interbody arthrodesis using autologous bone graft, partial L3 corpectomy, and resection of phlegmon, and insertion of a biomechanical cage at the L3-L4 level. Blood, anaerobic, fungal, and acid-fast bacilli (AFB) cultures taken from the aneurysm, vertebral body, and intervertebral disc during the open surgery did not grow any pathological organisms.

The postoperative period was clinically unremarkable, and the patient was discharged in good condition on postoperative day 14. However, the patient returned after 4 months with

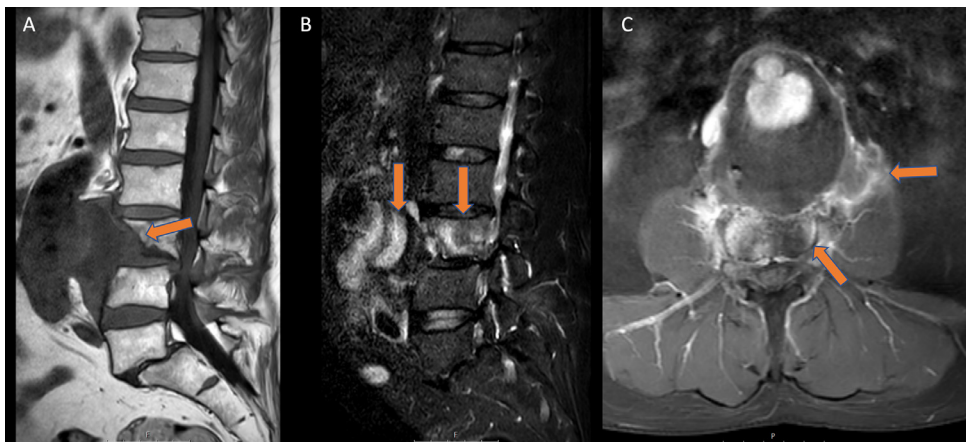

Fig. 1 – Sagittal T1-weighted (A) MRI demonstrating smooth destruction of anterior L3 and superior L4 vertebral bodies. Sagittal STIR (B) MRI demonstrating a high signal change in the soft tissue abscess posterior to the AAA (presumed Mycotic aneurysm). Inflammatory edema is also seen in the L3 vertebral body. Axial T1-weighted post-contrast MRI (C) demonstrates rim enhancement in the left psoas abscess and inflammatory enhancement in the L3 vertebral body. No epidural abscess was seen.

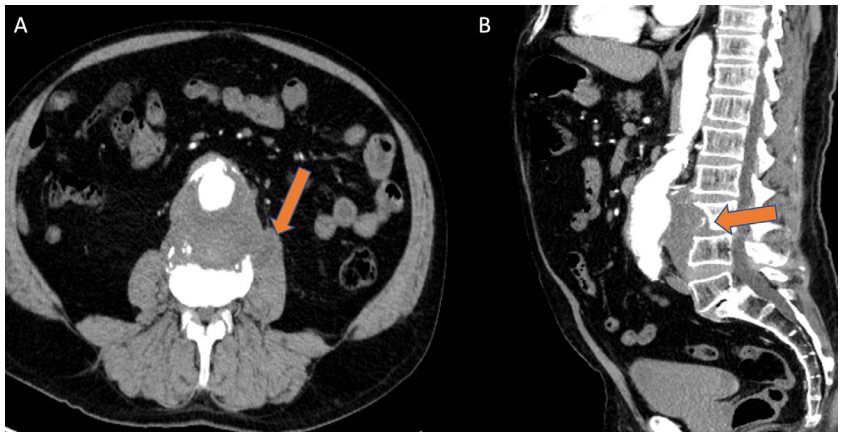

Fig. 2 – Axial contrast-enhanced CT (A) demonstrating abdominal aortic aneurysm with irregular walls and relatively well-demarcated soft tissue abscess on the posterior aspect of the aneurysm. A left psoas abscess is also seen. Sagittal CT image (B) demonstrates smooth destruction of the anterior aspect of the L3 vertebral body and the superior aspect of the L4 vertebral body.

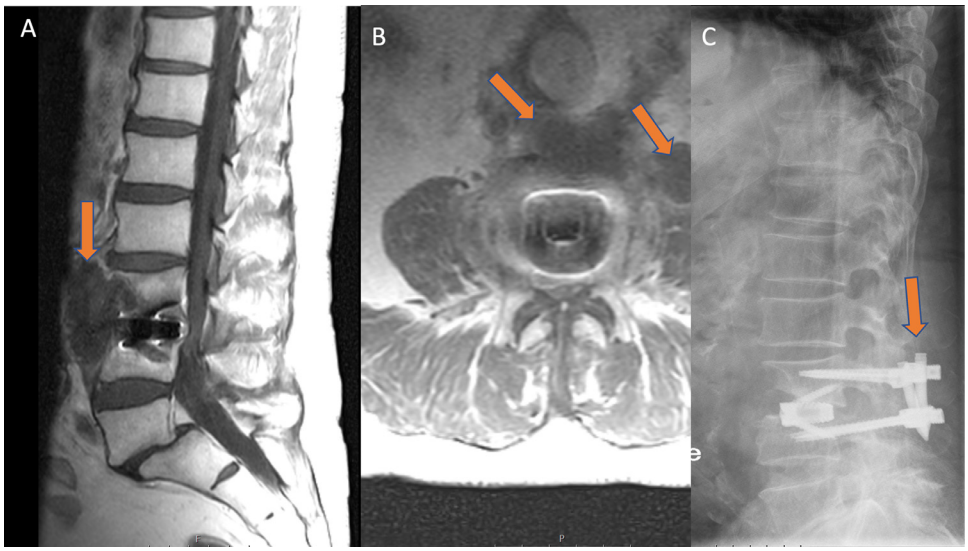

Fig. 3 – Sagittal (A) and axial (B) T1-weighted post-contrast MRI images utilizing metal artifact reduction sequence demonstrating post-surgical debridement of the abscess with placement of a disc spacer at L3-4 level with non-enhancing residual post-surgical soft tissue change at the operative bed and the left psoas muscle (arrows). Surgical repair of the mycotic abdominal aneurysm with a Dacron graft is also noted. Three-month postoperative lumbar spine radiograph (C) demonstrating staged posterior spinal fusion hardware at L3-4 (arrow).

worsening low back pain radiating to the posterior thighs bilaterally, without weakness or sensory loss upon neurological examination. A repeat MRI demonstrated severe spinal canal stenosis with crowding and post-contrast enhancement of the cauda equina roots at the L3-L4 level (Fig. 3). There was an interval decrease in edema and enhancement within the L3 and L4 vertebral bodies and the prevertebral soft tissues, with a small volume of peripherally enhancing residual fluid collection within the left psoas musculature (Fig. 3). Post-surgical changes related to aortic aneurysm repair, lumbar diskectomy, and fusion were noted. The patient underwent L3-L4 decompressive laminectomy and posterior instrumentation using percutaneous pedicle screws (Fig. 3C). The postoperative course was unremarkable, and the patient was discharged on postoperative day 7. At the time of discharge, the patient reported significantly reduced pain.

Discussion

A few cases are reported in literature describing diskitis-osteomyelitis with associated mycotic aortic aneurysm, with the majority being postoperative or procedural complications.

To our knowledge, this is one of the first, if not the first, presentation of spontaneous vertebral diskitis-osteomyelitis secondary to contiguous spread from an adjacent mycotic aortic aneurysm.

MAA is an uncommon disease entity with a prevalence ranging from 0.6%-2.6% that also has multiple detrimental complications [1,2]. Diskitis-osteomyelitis as a complication due to contiguous spread is even less common and underreported in the literature. Diagnosing vertebral infection secondary to MAA can pose a challenge due to the vague nature of the initial symptoms, such as back pain or fever, and the difficulty of determining which started first; one of the main reasons being MAA is commonly managed as a fever of unknown origin initially [4]. The presence of a pulsatile mass or bruit can clue the diagnosing physician to the presence of an aneurysm but not to infection. Diagnosis is sometimes not made until after the extension of infection to involve the adjacent structures, such as the psoas muscle or vertebrae resulting in limping and flank pain warranting imaging [3,4].

In our case, the patient had a known aneurysm with no history of fever or other infectious symptoms. The patient, however, did complain of limping and flank pain, as well as neuronal radiation of the pain to involve the lower extremities warranting further investigation with imaging. Hence, a history of aneurysm with developing fever and elevated inflammatory markers such as ESR and CRP should raise suspicion for the possibility of an infected aneurysm. Additional physical exam findings can also provide valuable information to localize possible complications or infectious spread.

On the other hand, Vertebral osteomyelitis is usually a more obvious disease process, commonly diagnosed early enough before the infection would have had time to spread involving the aorta.

Furthermore, bacterial cultures can confirm the diagnosis but not exclude it, as cultures are positive in only 50%-85% of cases [5]. In our case, the initial blood cultures were negative, while the abscess culture grew *S. mitis*. This aligns with the most commonly known organisms to cause MAA, including *Staphylococcus, Streptococcus,* and *Salmonella,* with *Staphylococcus aureus* being the most common in Western countries [6–8].

Radiological imaging is the most reliable tool for diagnosis, with CT angiography being the modality of choice, demonstrating a sensitivity of 92%-96% and specificity of 93%-100% for diagnosing MAA [9]. CT Angiography findings that aid in diagnosis include, but are not limited to, peri-aortic edema, peri-aortic inflammatory soft tissue mass demonstrating uniform enhancement, and peri-aortic gas in the presence of aortic wall irregularity (prior to aneurysmal development) or presence of aortic aneurysm [10]. Additionally, MR can demonstrate similar features with low-T1/high-T2 signal intensity to demonstrate edema and inflammatory tissue, with the latter demonstrating uniform contrast enhancement [11]. However, the primary purpose of MR imaging is to reveal the extent of the spread of infection and the involvement of adjacent structures. Furthermore, MRI will aid in evaluating the spinal cord and help in decision-making as to whether surgery is required. Local invasion can usually be seen as fluid extending into the adjacent soft tissues or erosion of the neighboring vertebrae [10,11].

In our case, imaging not only revealed the infected aneurysm but also showed the extension of disease involving the vertebrae and psoas muscle. Given the absence of fever, the question of possible malignancy was raised. However, after drainage and culture of the psoas abscess, this was confirmed to be infectious.

Management of such cases includes a combination of prolonged antibiotic therapy in conjunction with an aggressive surgical approach. This includes either open surgical or endovascular repair, with recent data demonstrating no superiority of one over the other [12]. Our patient underwent open surgical repair, given the extensive involvement of the surrounding soft tissues, allowing the surgeons access to drain and debride all involved tissue. Literature has shown increased efficacy for prolonged antibiotic treatment of over six months, with 6-12 months being the recommendation, to eradicate infection and reduce recurrence [12].

Conclusion

Spontaneous vertebral osteomyelitis secondary to MAA is an uncommon disease entity and represents a meager percentage of vertebral diskitis-osteomyelitis. That, combined with the vagueness of its early clinical presentation, makes the diagnosis more challenging than expected. Moreover, progression to involve other surrounding tissues or even rupture carry critical consequences. This case, therefore, highlights the importance of early detection and diagnosis, as well as the consideration of infection-related complications. Prompt intervention and management are crucial in preventing further progression and severe outcomes.

Patient consent

The authors confirm that this report does not contain any personal information that could lead to the identification of the patients. We have obtained written informed consent for publication from the patient.

REFERENCES

[1] Sörelius K, di Summa PG. On the diagnosis of mycotic aortic aneurysms. Clin Med Insights Cardiol 2018;12:1179546818759678.
[2] Laohapensang K, Rutherford RB, Arworn S. Infected aneurysm. Ann Vasc Dis 2010;3:16–23.
[3] Pluemvitayaporn T, Jindahra S, Pongpinyopap W, et al. Concomitant mycotic abdominal aortic aneurysm and lumbar tuberculous spondylitis with cauda equina syndrome: a rare condition — a case report and literature review. Spinal Cord Ser Cases 2018;4:13.
[4] Majeed H, Ahmad F. Mycotic aneurysm., https://www.ncbi.nlm.nih.gov/books/NBK560736/ (2023, accessed 17 June 2023).

[5] Brown SL, Busuttil RW, Baker JD, Machleder HI, Moore WS, Barker WF. Bacteriologic and surgical determinants of survival in patients with mycotic aneurysms. J Vasc Surg 1984;1:541–7.
[6] Raavi L, Garg P, Hussain MWA, Wadiwala IJ, Mateen NT, Elawady MS, et al. Mycotic thoracic aortic aneurysm: epidemiology, pathophysiology, diagnosis, and management. Cureus 2022;14:e31010.
[7] Naktin J, DeSimone J. Lumbar vertebral osteomyelitis with mycotic abdominal aortic aneurysm caused by highly penicillin-resistant Streptococcus pneumoniae. J Clin Microbiol 1999;37:4198–200.
[8] Voulalas G, Makris S, Papacharalampous G, Maltezos C. Mycotic aneurysm due to leclercia adecarboxylata: a complication of vertebral osteomyelitis. Ann Vasc Surg 2016;33:229.e1–229.e5.
[9] Fisk M, Peck LF, Miyagi K, Steward MJ, Lee SF, Macrae MB, et al. Mycotic aneurysms: a case report, clinical review and novel imaging strategy. QJM 2012;105:181–8.
[10] Lee WK, Mossop PJ, Little AF, Fitt GJ, Vrazas JI, Hoang JK, et al. Infected (Mycotic) aneurysms: spectrum of imaging appearances and management. Radiographics 2008;28:1853–68.
[11] Macedo TA, Stanson AW, Oderich GS, Johnson CM, Panneton JM, Tie ML. Infected aortic aneurysms: imaging findings. Radiology 2004;231:250–7.
[12] Sörelius K, Budtz-Lilly J, Mani K, Wanhainen A. Systematic review of the management of mycotic aortic aneurysms. Eur J Vasc Endovasc Surg Off J Eur Soc Vasc Surg 2019;58:426–35.