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CASE REPORT: Aortitis from the Unusual Culprit of S. pneumoniae, Serotype 23B

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Case Report

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Abstract

Introduction:

Aortitis is a rare but potentially life-threatening condition involving inflammation of the aortic wall that may lead to aneurysm formation or rupture. Infectious aortitis caused by *Streptococcus pneumoniae* is an uncommon but severe entity associated with high morbidity and mortality.

Case Presentation:

We report a case involving a Caucasian female in her 60s who presented with fever and severe left-sided flank pain. Initial laboratory work revealed leukocytosis and elevated inflammatory markers. Imaging identified a rapidly expanding aneurysm of the descending thoracic aorta with intramural thrombus formation. Blood cultures confirmed *S. pneumoniae* bacteremia, specifically serotype 23B, a strain not covered by current pneumococcal vaccines. The patient was managed with targeted antimicrobial therapy and underwent thoracic endovascular aortic repair (TEVAR) due to aneurysm progression.

Conclusions

A follow-up PET-CT scan one year later confirmed the resolution of the infection. Early imaging, multidisciplinary management, and tailored antimicrobial therapy are essential for optimizing patient outcomes in cases of infectious aortitis.

Introduction

Aortitis refers to inflammation of the aortic wall, presenting with variable morphologies. It may be localized to specific aorta segments, such as the thoracic or abdominal regions, or involve the entire vessel. While considered rare, its true incidence is likely underestimated. [1] The clinical presentation of aortitis is highly variable, ranging from non-specific constitutional symptoms—including fever, weight loss, and localized abdominal or back pain—to more severe manifestations. Due to the often-insidious onset and chronic nature of symptoms, diagnosis is frequently delayed, with many patients seeking medical attention only when complications, such as aneurysmal rupture, have occurred. [2] Aortitis may first present as a life-threatening emergency, such as hemorrhagic shock or septic shock secondary to infection, typically manifesting with tachycardia and/or hypotension. [3] Infectious aortitis and the subsequent formation and rupture of aortic aneurysms (mycotic aneurysms) are rare but life-threatening conditions, often associated with high morbidity and mortality. Historically, in the pre-antibiotic era, mycotic aneurysms commonly arose as complications of infective endocarditis, with causative pathogens mirroring those responsible for endocardial infections. In contrast, in the modern era, the most frequently implicated pathogens in infectious aortitis are Staphylococcus aureus and non-typhoidal Salmonella species. [4]

Streptococcus pneumoniae is a pathogen responsible for a broad spectrum of infections, including otitis media, sinusitis, pneumonia, bacteremia, and meningitis. [5] Introducing the pneumococcal conjugate vaccine has significantly reduced the incidence of severe infections caused by vaccine-included strains. [6]

This case report describes a case of aortitis caused by Streptococcus pneumoniae in an otherwise healthy woman in her mid-60s, leading to severe complications.

Case report

A Caucasian female in her 60d presented in the Emergency Department with a fever reaching 39°C and severe left-sided flank pain. Physical examination revealed decreased respiratory sounds at the lung bases, predominantly on the left, along with epigastric tenderness upon deep palpation. Laboratory tests showed significantly elevated inflammatory markers and D-dimer levels (Table 1). Initial Chest X-rays and abdominal ultrasound did not reveal significant pathological findings, underscoring the urgency and severity of the patient's condition.

Table 1
Laboratory Workup.

Lab	Value	Reference Range
White Blood Cell Count	13.000 /µL	4.6–10.20 /µL
C-Reactive Protein	310 mg/dL	< 5 mg/dL
D-Dimers	1895 µg/mL	< 500 µg/mL

The patient’s medical history included mild dyslipidemia, osteoporosis, and hypothyroidism, all well-controlled with medication, as well as a COVID-19 infection two months prior to admission. Notably, she was unvaccinated against Streptococcus pneumoniae.

Investigations and Treatment

Upon admission, the patient was initiated on empirical antibiotic therapy with ceftriaxone and azithromycin for a preliminary diagnosis of possible left-sided pneumonia. However, given the atypical and severe nature of her back pain, radiating to the epigastrium, an abdominal CT scan with oral contrast was performed. The scan revealed atelectasis, consolidation in the lower left lung fields, and peri-aortic opacities. A follow-up CT aortography with intravenous contrast identified an aneurysm of the descending thoracic aorta, accompanied by dissection and an intramural thrombus (Fig. 1). In response, the patient was placed on continuous vital sign monitoring and initiated on β-blocker therapy.

Blood cultures subsequently confirmed S. pneumoniae bacteremia, with the isolate demonstrating susceptibility to β-lactams and glycopeptides but resistance to macrolides and lincosamides. Accordingly, antimicrobial therapy was adjusted to vancomycin and ceftriaxone (MIC < 0.5 µg/ml for both

vancomycin and cefotaxime). Shortly thereafter, the patient developed acute renal failure, likely attributed to nephrotoxic effects from the combination of medications and intravenous contrast. Vancomycin was discontinued and replaced with linezolid, leading to a rapid resolution of febrile episodes. Immunological tests, including ANA and p/c ANCA, were negative. Further serotyping of the blood culture isolates identified S. pneumoniae serotype 23B.

Differential Diagnosis

Aortitis can result from both infectious and non-infectious etiologies. Among infectious causes, Streptococcus pyogenes, Streptococcus pneumoniae, Staphylococcus species, and Salmonella species are the most prevalent pathogens. In contrast, non-infectious aortitis is commonly associated with giant cell arteritis, Takayasu arteritis, and various rheumatologic diseases [7].

Outcome and follow-up

The diagnosis in this case was particularly challenging, as aortic dissection is exceedingly rare in otherwise healthy females. The underlying bacteremia is believed to have triggered the aneurysm's expansion. This was most likely a rapidly progressing mycotic aneurysm resulting from hematogenous seeding of the intima or vasa vasorum. The possibility of a preexisting aneurysm is unlikely, given the acute onset of symptoms, the absence of a family history of vascular disease, and the patient's negative personal history for hypertension, smoking, or underlying connective tissue disorders.

Despite antimicrobial therapy, the patient continued to experience intermittent retrosternal pain and episodes of vomiting. A repeat aortography demonstrated progression of the lesion, with an increase in aneurysm diameter (Fig. 2). Given the severity of findings, a multidisciplinary team—including a cardiac surgeon and an interventional radiologist—was consulted. After receiving 14 days of adequate intravenous antimicrobial therapy and achieving sterile blood cultures for 11 days, the patient underwent an emergent thoracic endovascular aortic repair (TEVAR). A PET-CT scan was performed to assess the extent of the infection, revealing no additional inflammatory foci beyond the descending thoracic aorta (Figs. 3a and 3b).

The patient's condition gradually improved, and she was discharged after a total hospitalization of 35 days. She was transitioned to long-term oral antimicrobial therapy with cefixime and monitored closely. She remained asymptomatic over the following 12 months, with monthly clinical and laboratory follow-ups demonstrating stable inflammatory markers. A follow-up PET-CT scan showed no residual inflammation (Fig. 4), and cefixime therapy was discontinued after completing a 12-month course. She was monitored monthly for three months and then every three months post-discontinuation. At two years post-infection, she remains symptom-free, with normal laboratory parameters.

Discussion

As a major blood vessel, the aorta is relatively resistant to damage compared to other vasculature. However, several factors can weaken the aortic wall, increasing susceptibility to aneurysm formation and infectious processes. These predisposing factors include uncontrolled diabetes mellitus or hypertension, malignancy, iatrogenic inoculation due to surgical interventions or medical devices, atherosclerosis, vascular malformations, and medial cystic necrosis of the aortic wall. Four different mechanisms have been postulated to produce infection of the arterial wall: (a) septic micro-emboli to the vasa vasorum, (b) extension from a contiguous infected focus, (c) hematogenous seeding of the intima during bacteremia from a distant infection, and (d) trauma to the arterial wall with direct contamination. However, identifying a primary source of infection remains challenging, with a definitive etiology determined in only approximately 50% of reported cases [8].

Infected aortic aneurysms, or mycotic aortic aneurysms (MAAs), account for approximately 0.7% to 26% of all aortic aneurysms [9]. MAA typically results from hematogenous bacterial colonization of a pre-existing aneurysm or an adjacent infectious site. Following bacterial colonization of the compromised vascular endothelium, collagenolytic and elastolytic enzyme activation leads to suppuration, ultimately facilitating aneurysm formation within weeks. Notably, COVID-19 has been implicated in the activation of metalloproteinases and collagen degradation within the aneurysmal wall, potentially contributing to aneurysm development [10].

Currently, there are no specific laboratory tests that can definitively diagnose MAA. While blood cultures are critical in identifying causative pathogens, they are only positive in 50% to 82% of cases [11]. CT angiography (CTA) remains the imaging modality of choice for diagnosing mycotic aneurysms, offering high-resolution three-dimensional visualization of vascular anatomy, which is essential for both diagnosis and surgical planning [12]. CTA findings suggestive of infectious aortitis include aortic wall thickening, loss of normal wall contour, periaortic fluid collections, rapidly enlarging saccular aneurysms or pseudoaneurysms, and, in some cases, intramural air [13]. Magnetic resonance angiography (MRA) is a reliable alternative imaging technique. The early detection of vascular wall changes via CTA enables prompt diagnosis and intervention [1, 13].

Delays in diagnosis or inadequate treatment of mycotic aneurysms often result in significant morbidity and mortality, primarily due to sepsis or catastrophic hemorrhage following rupture [14]. Furthermore, COVID-19 infection has been reported to exacerbate patient outcomes, as multiorgan failure associated with severe COVID-19 pneumonia may increase the risk of aortic rupture and delay necessary interventions, ultimately elevating mortality rates. Given these risks, excluding non-infectious causes of aortitis is imperative before establishing a final diagnosis. [10]

Previous experience suggests poor outcomes in the absence of surgical repair when pain is present, and rupture is a concern. Open surgical repair with debridement and graft placement has traditionally been considered the gold standard for infected aneurysms [8]. Endovascular repair of mycotic thoracic aneurysms is increasingly being utilized, particularly in patients with high surgical risk or in cases where the anatomical location of the aneurysm presents challenges for an open procedure [15, 16].

The selection and duration of antimicrobial therapy following TEVAR remain challenging, with limited evidence to guide clinical decisions [10]. FDG PET-CT has emerged as a valuable tool in guiding antibiotic duration, potentially eliminating the need for lifelong suppressive therapy, especially when an initial positive scan becomes negative during treatment. Although establishing a definitive cut-off value for positivity in borderline cases remains challenging, the test's high negative predictive value makes it particularly useful for the follow-up of known aneurysms [17].

A noteworthy aspect of this case is the pneumococcal serotype responsible for aortitis. While pneumococcal strains not covered by available vaccines have occasionally been associated with severe infections, serotype 23B was not included in the vaccines available at the time of this case. This is the first documented case of aortitis caused by serotype 23B, to our knowledge, although similar cases have been reported with serotype 23A [18, 19]. Emerging evidence suggests that serotype 23 has been increasingly linked to severe infections in the post-vaccine era, highlighting the need for further research into its virulence and clinical impact.

Conclusions

Pneumococcal aortitis, though rare, represents a life-threatening condition with high mortality that often presents with non-specific clinical features. Prompt diagnosis through laboratory tests, blood cultures, and advanced imaging—particularly CT and FDG PET-CT—is crucial for evaluating aneurysmal involvement and monitoring therapeutic response. Early initiation of targeted antibiotic therapy, in combination with surgical or interventional management, remains the cornerstone of treatment. Importantly, pneumococcal aortitis may occur in the absence of traditional risk factors, highlighting the need for heightened clinical suspicion. FDG PET-CT also plays a pivotal role in guiding the duration of antibiotic therapy. Effective management requires a multidisciplinary approach to ensure timely diagnosis, individualized treatment, and improved patient outcomes.

Declarations

1. Funding: Not applicable.
2. Conflicts of interest/Competing interests: The authors declare that they have no conflict of interest.
3. Ethics approval: No ethical committee approval was required for this case report by the Department, as this article does not contain studies with human participants or animals.
4. Consent to participate: Informed consent was obtained from the patient included in this study.
5. Written Consent for publication: The patient gave written consent to use personal data for the publication of this case report and any accompanying images.
6. Availability of data and material: All data underlying the findings are fully available.
7. Code availability: Not applicable.
8. Authors' contributions: All authors contributed significantly to the work and agree with the manuscript content. SCK, NT and IB were responsible for writing and design; ET, YM, and CSK

performed analysis and interpretation; HS provided writing and overall monitoring. All authors approved the final version.

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Figures

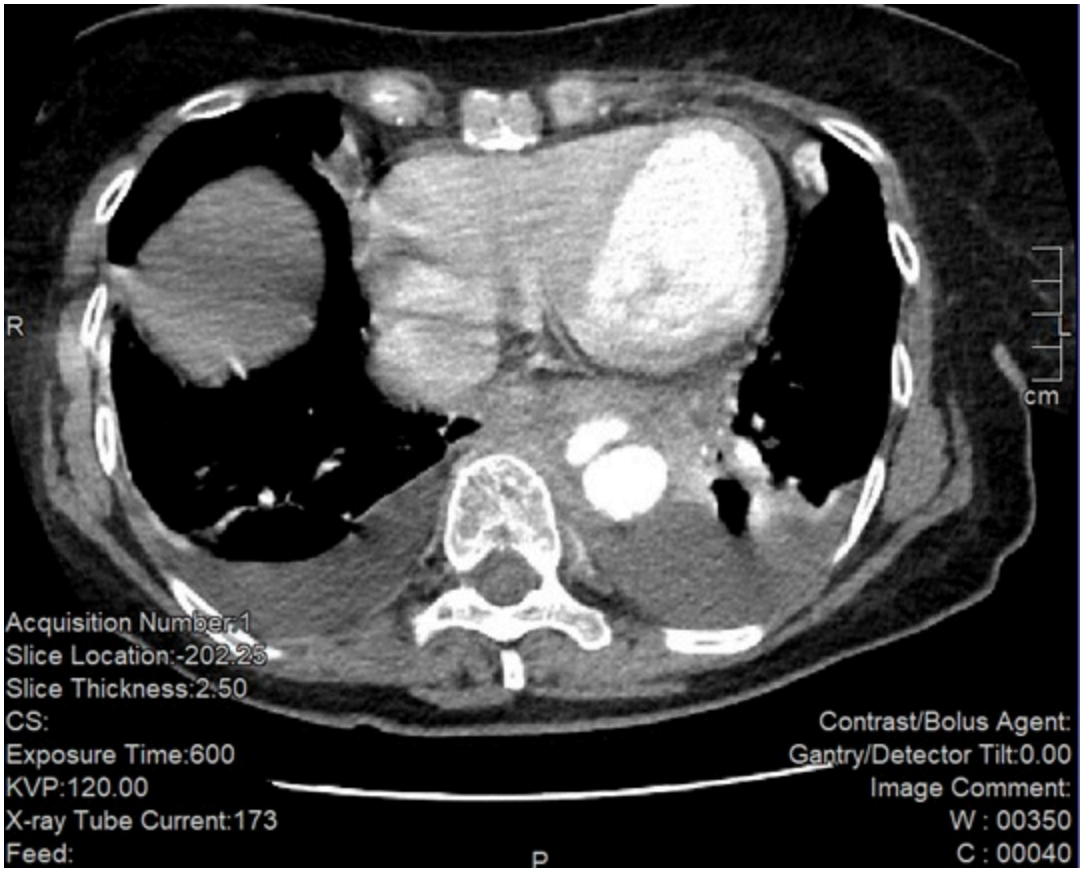


Figure 1

Chest CT: Mural thrombus inside the aorta

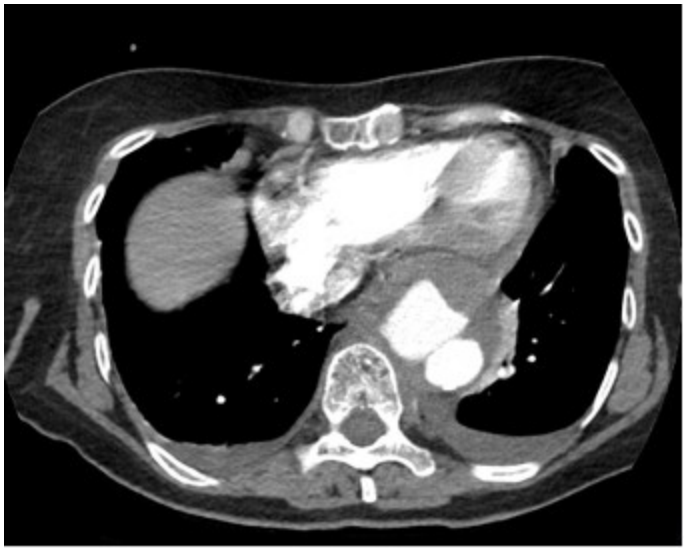


Figure 2

CT – Worsening of Mural thrombus inside the Aorta

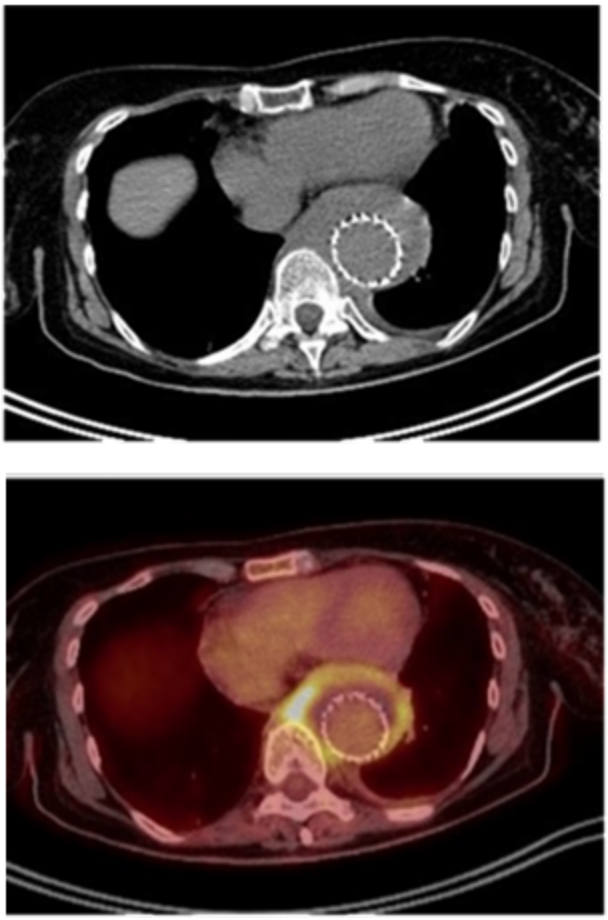


Figure 3

a: PET-CT: Imaging of the aorta

b: PET-CT: Imaging of the aorta

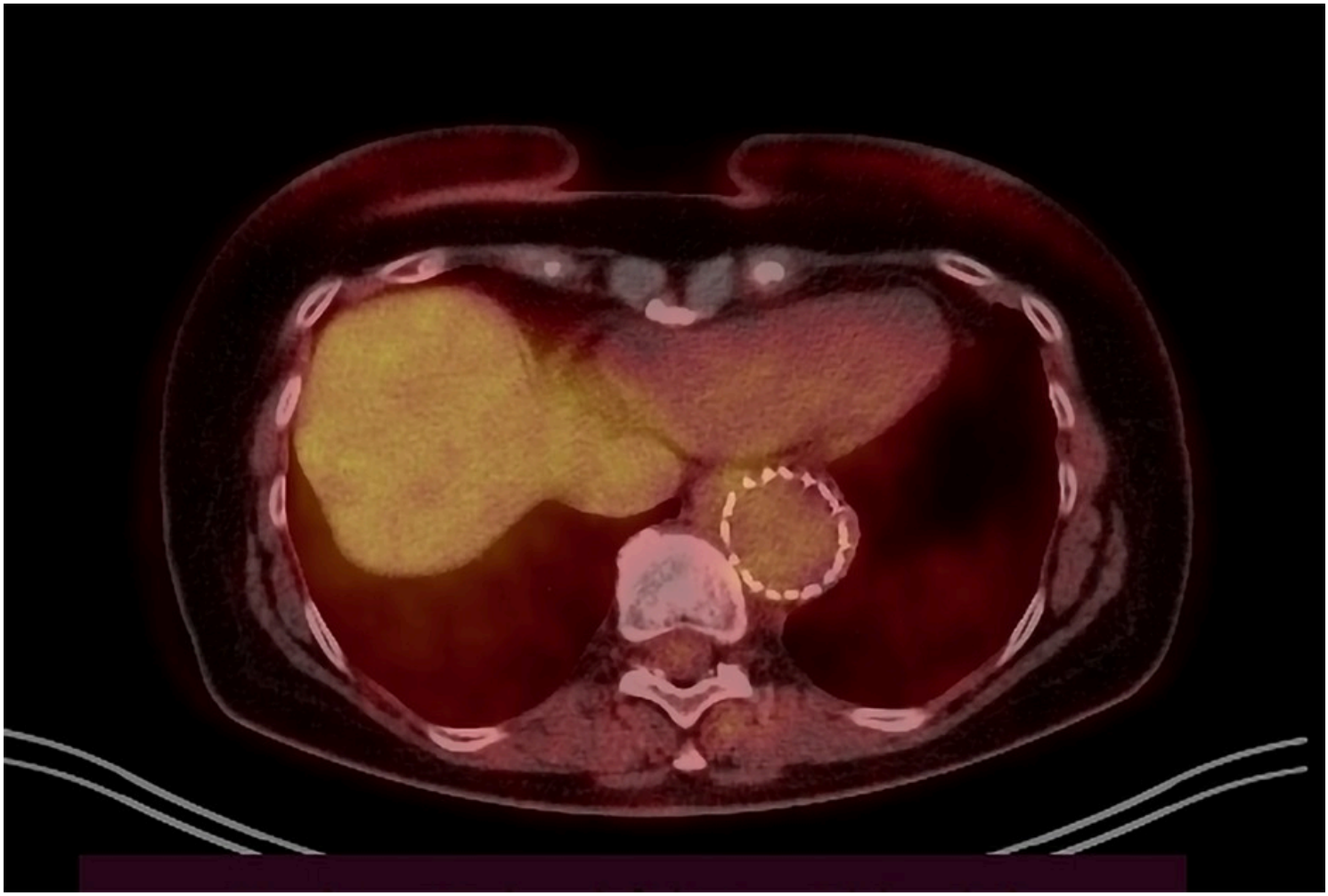


Figure 4

Follow-Up PET-CT without Inflammation of the Aorta

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