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Constrictive Pericarditis from Methicillin-Sensitive *Staphylococcus aureus*: A Case of Source and Seeding with Persistent Bacteremia

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Abstract

Staphylococcus aureus pericarditis is a rare but life-threatening condition that often presents as purulent pericarditis and in rare cases can lead to constrictive pathophysiology. Methicillin-sensitive *Staphylococcus aureus* (MSSA) as a cause of constrictive pericarditis is uncommon. A 65-year-old male with diabetes presented with chest pain and was diagnosed with pericarditis. Blood cultures confirmed MSSA bacteremia, and imaging revealed loculated pericardial effusion with features consistent with constrictive pericarditis. Pericardiocentesis revealed MSSA-positive serous fluid, and the patient underwent prolonged antimicrobial therapy. Despite initial clinical improvement, he developed recurrent MSSA bacteremia with vertebral osteomyelitis and an epidural abscess. He underwent surgical spinal debridement and stabilization, followed by extended intravenous antibiotic therapy. The patient's symptoms ultimately resolved, and follow-up imaging showed significant improvement. This is an atypical presentation of constrictive pericarditis from community-acquired MSSA and this case demonstrates the diagnostic complexity of MSSA bacteremia and the importance of source control.

Keywords

Methicillin-Sensitive *Staphylococcus aureus*, Constrictive Pericarditis, Bacteremia, Epidural Abscess, Invasive Staphylococcal Disease

1. Introduction

Staphylococcus aureus (SA) pericarditis has a high mortality, and providers need

to be aware of the significant risks. The diagnosis of pericarditis is based on meeting at least 2 of the following criteria, including chest pain, pericardial friction rub, widespread ST-segment elevations of at least 1 mm, new PR depressions, and a new pericardial effusion [1]. SA pericarditis includes the criteria above as well as SA isolation in the pericardial space [2]. Most cases of SA pericarditis are thought to result from hematogenous spread and seeding of septic emboli within the pericardium as the main mechanism for infection if not contiguous foci [2]-[4]. Classically, SA pericardial pathology is purulent in nature [2] [5]. Treatment includes pericardiocentesis, and in the setting of purulent pericarditis, reaccumulation of fluid is common, often requiring drainage [2]. Additionally, recurrence of constrictive pericarditis has been demonstrated to lead to chronic constrictive pericarditis; chronic constrictive pericarditis is a disease of significant morbidity and mortality, and the only current definitive treatment is radical pericardiectomy, an intensive surgical procedure with perioperative mortality ranging from 10% - 50% [6].

Purulent pericarditis occurs in an estimated 1 in 18,000 hospitalized patients, whereby SA accounts for approximately 22% - 31% of bacterial pericarditis cases [7] [8]. In general, constrictive physiology rarely occurs with purulent pericarditis, documented in less than 3% of cases [2]. This has a significant mortality of up to 20.5% despite antibiotics and pericardial drainage [2]. Methicillin-sensitive *Staphylococcus aureus* (MSSA) as the cause of constrictive pericarditis is rare and this case emphasizes the importance of continued pursuit of source control. This case highlights a rare presentation of MSSA constrictive pericarditis and persistent community-acquired MSSA bacteremia with vertebral osteomyelitis and epidural abscess.

2. Case Report

A 65-year-old male with a past medical history of type 2 diabetes, hypertension, hyperlipidemia, stable proliferative diabetic retinopathy, and benign prostatic hyperplasia. He presented with complaints of chest pain worse with inspiration, associated with myalgias, general weakness, and night sweats. On physical exam, the temperature was 37.8˚C, otherwise, he was hemodynamically stable and oxygenating appropriately on room air. Laboratory tests were notable for glucose 409 mg/dL, WBC 15,390/μL with 83.8% neutrophils, troponin I 106 picogram/mL, BNP 145 picogram/mL, erythrocyte sedimentation rate (ESR) 117 mm/hr, and C-reactive protein (CRP) > 200.00 mg/L.

Further workup included chest radiography demonstrating linear consolidation in the left lower lobe. EKG noted diffuse ST elevations in leads I, II, III, aVF, and V2 - V6, with ST depressions in aVR and V1 (**Figure 1**). The patient was originally started on intravenous (IV) ceftriaxone and vancomycin. Blood cultures obtained on hospital day 1 were positive for MSSA, and the antibiotics transitioned to cefazolin. Subsequent urine culture showed >100,000 colony-forming units of MSSA.

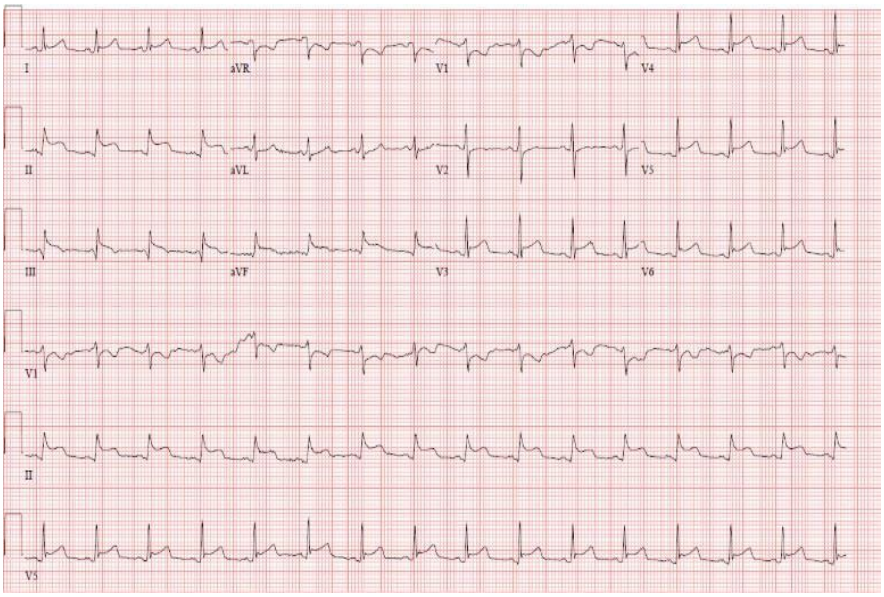

Figure 1. Electrocardiogram consistent with acute pericarditis. 12-lead EKG showing diffuse ST-segment elevations in leads I, II, III, aVF, and V2 - V6, with reciprocal ST depressions in aVR and V1. Additionally, widespread PR segment depressions are present. These findings are characteristic of acute pericarditis and were key to initiating diagnostic evaluation.

The patient underwent transthoracic echocardiogram (TTE), which demonstrated loculated pericardial effusion and possible constrictive pericarditis. A transesophageal echocardiogram (TEE) did not demonstrate evidence of endocarditis. Cardiac magnetic resonance imaging (cMRI) (**Figure 2**) demonstrated moderate to large pericardial effusion measuring approximately 1.5 cm in thickness with internal septations. Cine sequences revealed a septal bounce, consistent with pericardial constriction.

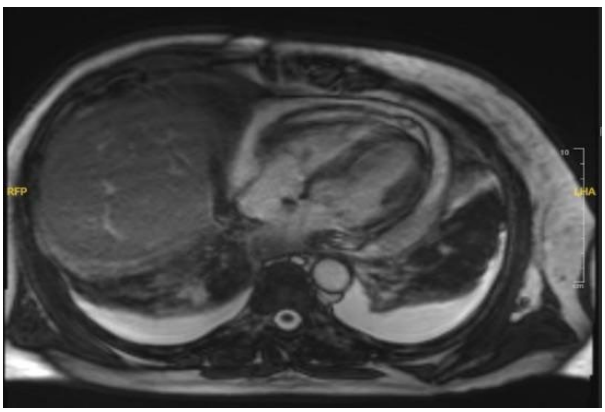

Figure 2. Cardiac magnetic resonance imaging (cMRI) demonstrating constrictive pericarditis. cMRI with T2-weighted and cine sequences showing moderate circumferential pericardial effusion with internal septations and thickened pericardium. Additionally, the presence of a septal bounce on dynamic imaging was consistent with constrictive physiology. These findings confirmed pericardial involvement and guided the decision to proceed with intervention.

On day 5 of admission, the patient had persistent dyspnea and fever; repeat blood cultures 48 hours post-antibiotic provision were positive for MSSA. The patient was diagnosed with acute/subacute constrictive pericarditis with loculated pericardial effusion and persistent MSSA bacteremia. The patient then underwent pericardiocentesis with drainage of 50 mL serous fluid and placement of a pericardial drain (Figure 3). The culture from the pericardial fluid grew MSSA. He was diagnosed with loculated pericarditis in the setting of MSSA bacteremia with the differential diagnosis including seeded effusion from bacteremia of unknown source versus primary infection pericarditis/loculated abscess. He was treated with IV cefazolin 2 g every 8 hours for 6 weeks from the first postoperative negative blood culture and subsequently oral cephalexin 500 mg 3 times daily for 14 days. A detailed clinical course including treatment regimens, diagnostic findings and significant events is summarized in Table 1.

Table 1. Clinical course and treatment timeline.

Event Day	Clinical Course and Management
Day 1	Presentation with chest pain; blood cultures obtained; started on IV[a] ceftriaxone and vancomycin.
Day 2	Blood cultures positive for MSSA[b]; antibiotics switched to cefazolin.
Day 3	TTE[c] performed showing loculated pericardial effusion.
Day 4	cMRI[d] showed moderate to large pericardial effusion with septations; signs of constrictive pericarditis.
Day 5	Persistent fever and dyspnea; repeat blood cultures positive for MSSA.
Day 6	Pericardiocentesis performed; 50 mL serous fluid drained; pericardial fluid culture grew MSSA.
Weeks 1 - 6	IV cefazolin 2 g every 8 hours administered.
Weeks 7 - 8	Oral cephalexin 500 mg three times daily administered.
Day 62	New onset thoracic back pain; blood cultures positive for MSSA; toe wound culture positive for MSSA.
Day 63	MRI[e] of thoracic spine showed discitis and possible osteomyelitis.
Weeks 9 - 14	IV cefazolin 2 g every 8 hours administered.
Weeks 15 - 20	Oral cefadroxil 1 g twice daily administered.
Day 104	Worsening back pain; MRI showed distinct epidural abscess at T7 - T8.
Day 105	Surgical debridement with T5 - T10 pedicle screw fixation and laminectomy performed.
Weeks 21 - 26	IV nafcillin administered.
Weeks 27 - 34	IV cefazolin administered.

[a]IV: Intravenous, [b]MSSA: Methicillin-sensitive *Staphylococcus aureus*, [c]TTE: Transthoracic echocardiogram, [d]cMRI: Cardiac magnetic resonance imaging, [e]MRI: Magnetic resonance imaging.

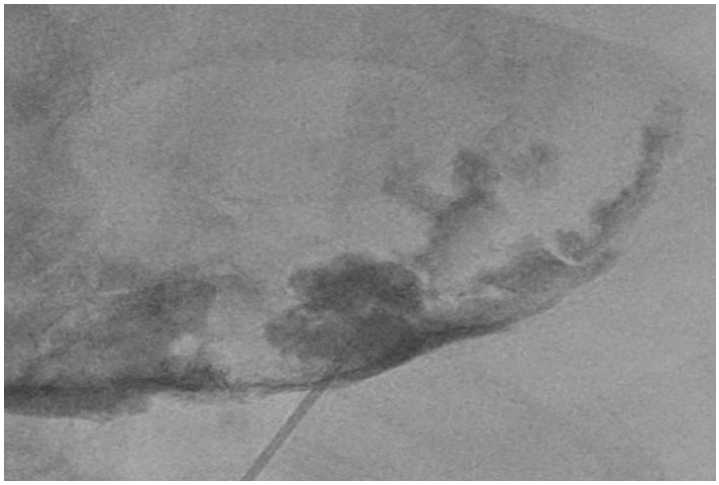

Figure 3. Pericardiocentesis and drain placement in loculated effusion. Intra-procedural imaging pericardiocentesis for loculated pericardial effusion and placement of a pericardial drain. Approximately 50 mL of serous fluid was aspirated. Procedure cultures subsequently grew methicillin-sensitive *Staphylococcus aureus*, confirming bacterial pericarditis.

After 6 days of completion of the prolonged antibiotics, the patient developed new thoracic back pain with generalized weakness. Blood cultures were positive for MSSA, and a wound of the right great toe noted at that time was cultured and isolated MSSA. Magnetic resonance imaging (MRI) of the thoracic spine (**Figure 4**) demonstrated discitis and osteomyelitis at the T7 - T8 level with an associated epidural abscess measuring 1.2 × 0.8 cm on T2-weighted images. Neurosurgery was consulted and recommended the continuation of medical treatment, given that there were no signs of cord compression and no focal abscess. The patient again completed a course of 6 weeks of IV cefazolin 2 g every 8 hours and subsequently oral cefadroxil 1 g twice daily for 6 weeks.

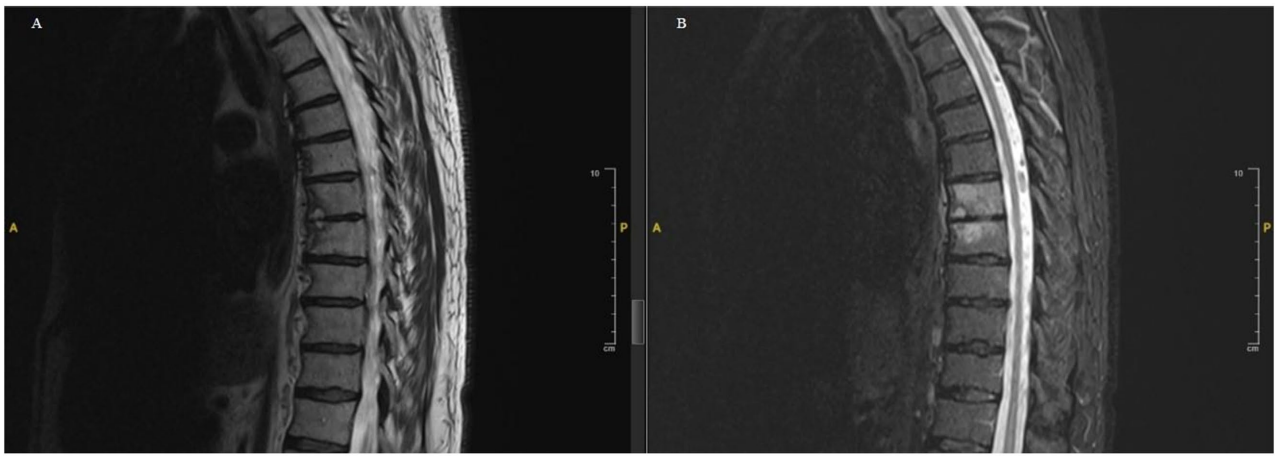

Figure 4. Thoracic spine magnetic resonance imaging consistent with discitis and osteomyelitis. Thoracic spine magnetic resonance imaging performed 10 days after completion of initial IV cefazolin therapy. (A) Sagittal T2-weighted image shows disc space narrowing and hyperintense signal at T7 - T8. (B) Sagittal STIR image reveals abnormal marrow signal and enhancement of the T7 - T8 vertebral bodies consistent with discitis and osteomyelitis. A left-sided epidural abscess is visible, measuring 1.2 × 0.8 cm. These findings prompted medical management and further monitoring.

Despite prolonged treatment after clearance of bacteremia, 14 days after completing the parenteral cefazolin plus a course of cefadroxil, the patient had gradually worsening thoracic back pain. A repeat MRI of the thoracic spine (**Figure 5**) showed discitis osteomyelitis at the T7 - T8 level with an epidural abscess eccentric to the left side without significant spinal canal stenosis and significant erosion of the endplates of the T7 - T8 vertebral bodies. Patient underwent surgical debridement with T5 - T10 pedicle screw fixation with a laminectomy and complete facetectomy on the left side for disc space debridement with a partial corpectomy and interbody graft placement. He was provided 6 plus 8 weeks of IV antibiotic therapy (nafcillin and cefazolin, respectively), given the high risk of postoperative infection, especially in the setting of hardware implantation. In follow-up, repeat cardiac and thoracic imaging improved, and the patient had resolution of his symptoms. Prior to publication of this case report, written informed consent was obtained from the patient.

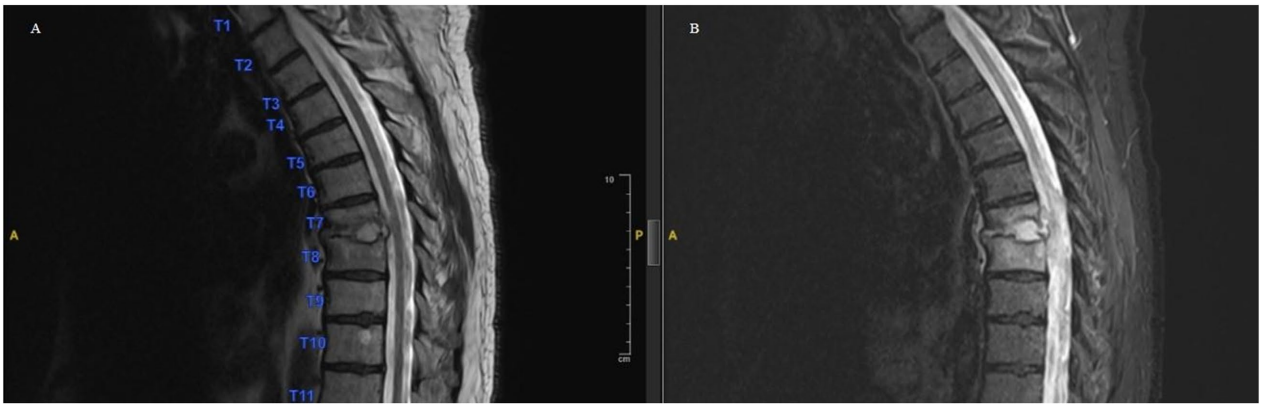

Figure 5. Follow-up thoracic magnetic resonance imaging showing progression of vertebral osteomyelitis and epidural abscess 6 months later. Thoracic spine magnetic resonance imaging performed approximately 15 days after completion of prolonged parenteral antibiotics and subsequent oral cefadroxil. (A) Sagittal T2 and (B) STIR images demonstrate worsening of T7 - T8 discitis and osteomyelitis, with increased vertebral endplate destruction and expansion of the epidural abscess into the left ventral epidural space. These findings led to pursuit of surgical intervention with debridement and stabilization.

3. Discussion

Constrictive pericarditis is rare in the setting of SA pericarditis and carries a significant risk for cardiac tamponade and often requires intervention [2] [9]. The majority of the documented SA pericarditis cases are Methicillin-resistant *Staphylococcus aureus* (MRSA), while MSSA is less frequently reported. Cases have been described of MSSA resulting in purulent pericarditis specifically in the setting of immunosuppression, from bacteriuria, and after COVID-19 pneumonia [10]-[12]. Moreover, loculated and constrictive pericarditis from MSSA remains rare.

Compared to idiopathic or neoplastic etiologies of pericarditis, MRSA pericarditis has a higher probability of leading to pericardial tamponade and thus greater mortality [2]. In contrast, the limited data on MSSA pericarditis preclude reliable prognostication at present. Some studies have suggested that MRSA bacteremia

may have a higher mortality compared to MSSA [13]. However, there have been smaller studies suggesting survival may be similar regardless of methicillin susceptibility with respect to endocarditis, although this is limited in the setting of sample size [14]. This case demonstrates the complexity of managing MSSA bacteremia when complicated by metastatic infections such as osteomyelitis and epidural abscess.

Alternative causes of pericarditis, including viral, autoimmune, and neoplastic etiologies, were considered but deemed less likely based on the patient's clinical presentation and diagnostic findings. The growth of MSSA in both blood and pericardial fluid cultures, as well as the MRI evidence of loculated purulent effusion, supported a bacterial source. Viral and autoimmune workup was not pursued given the culture data. While the toe wound was a potential source, as well as the urine culture growth, the temporal relationship between the recurrence of bacteremia and initial presentation suggests these may have been secondary sites rather than the primary source. The initial source of bacteremia remains speculative, although hematogenous seeding leading to pericardial involvement is a plausible explanation.

As community-acquired SA infections continue to rise, clinicians should maintain a high index of suspicion for deep-seated complications in patients with persistent bacteremia [15]. Subacute progression and loculated fluid collections raised concern for hematogenous seeding from an indolent focus. Moreover, apart from diabetes mellitus, the patient lacked typical risk factors for invasive SA disease, including intravenous drug use, recent surgical procedures, or prosthetic devices.

In this patient, the development of discitis and subsequent epidural abscess after blood culture clearance and a prolonged antimicrobial course is most consistent with delayed metastatic seeding. Although an indolent discitis was discussed and considered as a potential source for initial seeding, the development of acute back pain, discitis, and subsequent epidural abscess after blood culture clearance and completion of a prolonged antimicrobial course is most consistent with delayed metastatic seeding from the MSSA bacteremia on presentation. Although spinal infections more commonly seed the pericardium, it is also plausible that the pericardial infection served as the source of bacteremia and subsequent spinal involvement [2].

Given the initial persistent bacteremia, this raises the question of the cefazolin inoculum effect and subsequently the decision to transition to nafcillin once the abscess was noted [16]. The cefazolin inoculum effect has been characterized in the setting of significant bacterial load, which can lead to reduced efficacy of cefazolin due to beta-lactamase-mediated hydrolysis. Although specific testing for this mechanism of resistance was not performed, the persistence of bacteremia despite cefazolin therapy and findings of deep-seated infection raised clinical suspicion and led to the transition to an alternative agent. Nafcillin, being less susceptible to the beta-lactamase degradation, was selected for its efficacy in high-inoculum infections

and its established use in MSSA bacteremia, specifically in the context of hardware implantation, whereby optimized bactericidal activity is essential to eradication [17].

Regardless, definitive management included source control through surgical debridement and an extended antimicrobial course. With rising SA infections, clinicians should remain aware of metastatic infections and closely monitor these complex patients with a low threshold for timely follow-up imaging and pursuit of source control. Persistent MSSA bacteremia continues to challenge clinicians, and ultimately, a thorough work-up may reveal deep-seated or metastatic infections that require prolonged therapy, multidisciplinary coordination, and timely source control to achieve resolution.

4. Conclusion

This case emphasizes the potential for delayed metastatic seeding in patients with persistent MSSA bacteremia, even after initial clinical improvement. Additionally, this case demonstrates the challenges in managing MSSA infections, particularly in the absence of typical risk factors, and underscores the necessity for a comprehensive diagnostic workup to identify and address all potential sources of infection. Early recognition and prompt source control are essential to improving patient outcomes.

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Informed Consent

Patient information was de-identified to protect privacy and informed consent for the publication of this case report was obtained in accordance with institutional guidelines.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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List of Abbreviations

SA	*Staphylococcus aureus*
MSSA	Methicillin-sensitive *Staphylococcus aureus*
MRSA	Methicillin-resistant *Staphylococcus aureus*
IV	Intravenous
MRI	Magnetic Resonance Imaging
TTE	Transthoracic echocardiogram
TEE	Transesophageal echocardiogram
cMRI	Cardiac magnetic resonance imaging
ESR	Erythrocyte sedimentation rate
CRP	C-reactive protein