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Intracardiac Abscess and Pacemaker Lead Infection Secondary to Hematogenous Dissemination of Methicillin-Sensitive *Staphylococcus Aureus* from a Prior Diabetic Foot Ulcer and Osteomyelitis

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Male, 77
Final Diagnosis: MSSA intracardiac abscess and pacemaker lead infection
Symptoms: Syncope • fever • dyspnea • lethargy • rigors • chills • malaise
Medication: —
Clinical Procedure: —
Specialty: Infectious Diseases

Objective: Rare disease
Background: Intracardiac abscesses are an unusual occurrence in developed countries. With the increase in use of implantable cardiac devices, the increase use of and advancements in antibiotics, and the longevity of patients with cardiac devices, one may expect an increase in such infections; however, case reports are rare. We are presenting a case in which hematogenous dissemination of methicillin-sensitive *Staphylococcus aureus* (MSSA) infection from a lower extremity diabetic ulcer propagated into an infected pacemaker lead and ultimately an intracardiac abscess of the right atrium.





Case Report: A 77-year-old male with a history of MSSA diabetic foot infection complicated by osteomyelitis presented with fever, syncope, and wide complex tachycardia, and he was found to have an intracardiac abscess and fibrinous lead vegetations. The patient was deemed too ill for invasive surgical intervention given his comorbidities, pacemaker generator replacement requirement, and intermittent ventricular tachycardia. The patient was subsequently sent home with oral antibiotics and home hospice per patient and family wishes.

Conclusions: This case demonstrated how hematogenous dissemination of MSSA infections from a diabetic foot ulcer and osteomyelitis can seed pacemaker hardware resulting in an intracardiac abscess. Unfortunately, our patient was too ill to undergo all procedures required to eradicate the abscess and infected pacemaker hardware. The standard of care would be complete hardware removal. Conservative management would include indefinite or prolonged antibiotic therapy, with the notion that intracardiac abscesses cannot be cured with antibiotics alone. This conservative management approach would be deemed necessary in a select population that cannot undergo surgical intervention.

MeSH Keywords: Abscess • Endocarditis, Bacterial • Pacemaker, Artificial • *Staphylococcus Aureus*

Abbreviations: AICD – automatic implantable cardiac defibrillator; ATP – appropriate anti-tachycardia pacing, BIPAP – biphasic positive airway pressure support; ED – Emergency Department; ECG – electrocardiogram; EP – electrophysiology; LV – left ventricle; LVEF – left ventricular ejection fraction; IABP – intra-aortic balloon pump; ICU – Intensive Care Unit; MSSA – methicillin-sensitive *Staphylococcus aureus*; NT-proBNP – N-terminal pro-brain-type natriuretic peptide; PCI – percutaneous coronary intervention; PO – per os; QTc – corrected QT interval; TEE – transesophageal echocardiogram; TTE – transthoracic echocardiogram; VT – ventricular tachycardia

Full-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/906227>

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Background

Intracardiac abscesses are an unusual occurrence in developed countries. With the increase in use of implantable cardiac devices, increase use of and advancements in antibiotics, and longevity of patients with cardiac devices, one may expect an increase in such infections; however, case reports are rare. In this case report we present a case in which hematogenous dissemination of methicillin-sensitive *Staphylococcus aureus* (MSSA) infection from a lower extremity diabetic ulcer propagated into an infected pacemaker lead and ultimately resulted in an intracardiac abscess of the right atrium.

Myocardial abscesses are often described as a complication of infective endocarditis. About 3,000 to 4,000 new cases of myocardial abscesses are diagnosed in the USA annually [1]. Infective endocarditis has an incidence of approximately 7 per 100,000 patient years [1]. Traditionally, aortic valve involvement of infectious endocarditis is seen with more frequency, followed by mitral valve involvement. However, reports of pacemaker infection rates vary: some suggest 2% to 4% annually, while others suggest 13% to 80%. Between 2004 and 2006, the rate of pacemaker infections increased 57% [2]. Cardiac implantable device infection incidence has been reported to be 0.7% to 2.2% [3]. *S. aureus* is the second most common cause of hospital acquired bacteremia in the USA [4]. *S. aureus* infective endocarditis for left-sided native valves has been associated with higher mortality rates than other organisms [5]; one-year survival rates were 37% lower (57% versus 80%) [6]. It is estimated that 60% of endocarditis due to *S. aureus* is due to MSSA [4]. The same consideration can be extrapolated from infective endocarditis to infected pacemaker or defibrillator leads and wires. Cardiac abscesses are suspected to arise from contiguous spread from infected valves and hardware as well as from possible hematogenous dissemination of thrombi into coronary arteries [1]. Non-intravenous drug related right-sided endocarditis accounts for only 5% to 10% of all cases [6]. The overall mortality of right-sided endocarditis has been reported to be 17% [4]. Conventional treatment with antibiotics alone has been found to be inferior to surgical intervention when comparing all-cause mortality and thrombotic events [7].

The patient presented in this case study was deemed too ill for invasive surgical intervention, given his comorbidities, intermittent ventricular tachycardia, and the requirement for pacemaker generator replacement. The patient was subsequently sent home with oral antibiotics and transitioned to home hospice. His automatic implantable cardioverter-defibrillator (AICD) was transitioned to pacemaker mode only and he was discharged per patient and family wishes.

Case Report

A 77-year-old male presented to the emergency department (ED) with a chief complaint of syncope. The patient had a past medical history of hypertension, hyperlipidemia, uncontrolled diabetes mellitus type two, recurrent diabetic foot ulcers complicated by osteomyelitis, peripheral vascular disease, and coronary artery disease status post five-vessel coronary artery bypass grafting 17 years prior with concomitant pacemaker/defibrillator implantation around the same time, secondary to systolic heart failure and secondary to ischemic cardiomyopathy. He described his syncope as having prodromal symptoms; the most prominent being dizziness. He self-reported dyspnea and fevers of 101°F (38.3°C) to 102°F (38.9°C). His primary care evaluation occurred the week prior to admission. Three days after his primary care evaluation, the patient continued to have persistently elevated blood glucose readings with development of lethargy, rigors, chills, and malaise. On the day of his presentation to the ED, his family had found the patient down at bedside, with 2.5 hours of unwitnessed time, which prompted emergency medical services activation.

Upon arrival to the ED, that patient was found to have elevated troponin levels of 2.8 ng/mL and a NT-proBNP (N-terminal pro-brain-type natriuretic peptide) of 24,000 pg/mL in addition to a leukocyte count of 16,000/uL.

An electrocardiogram (ECG) identified wide complex tachycardia at 146 beats per minute (bpm) and he was found to be tachypneic at 22 breaths per minute (bpm). The patient was hypertensive at 157/136 mm Hg with a mean arterial pressure of 143 mm Hg. Cardiology was notified, and cardioversion ensued. Post cardioversion, the repeat ECG demonstrated a heart rate of 115 bpm with a right bundle branch and left anterior fascicular block, QTc of 445 msec and a normal axis. Levophed was initiated. Empiric antibiotic therapy with vancomycin and piperacillin/tazobactam was initiated after blood cultures were obtained. Agonal breathing developed post cardioversion and the patient was subsequently intubated and transferred to the intensive care unit (ICU). Upon arrival to the ICU, the patient was found to be hypotensive despite the initial dose of Levophed and the dose was titrated up, and dopamine was started. The patient was emergently taken to the cardiac catheterization laboratory. Cardiogenic and septic shock were both considered.

The cardiac catheterization revealed severe native vessel disease with a vein graft to the right coronary artery with 85% stenosis, which underwent percutaneous coronary intervention (PCI) and stent deployment. A pseudoaneurysm was identified, and if the patient's status deteriorated, he would need a repeat cardiac catheterization. A 50 cc intra-aortic balloon pump (IABP) was inserted from the right femoral approach.

Upon arrival back to the ICU, the patient continued to have persistent sustained ventricular tachycardia episodes despite lidocaine and amiodarone infusions. The defibrillator was reprogrammed with a lower threshold of 130 bpm and his Levophed was titrated down with the IABP programmed at a 1 to 1 ratio. Two days later, the blood cultures obtained in the ED were found positive for MSSA in both samples.

During this time, his liver function tests and creatinine were elevated. The IABP was removed given the finding of gram-positive bacteremia. His lactic acidosis slowly resolved; however, given his persistent fevers and worsening leukocytosis, vancomycin was transitioned to intravenous (IV) linezolid. The patient was known to have had multiple diabetic foot infections complicated with osteomyelitis in the past and had been on multiple courses of antibiotics, all of which were completed in their entirety. The Levophed was titrated down once more and the patient was extubated a day later. Given his persistent hypoxia, he was placed on BiPAP (bilevel positive airway pressure). During this time, the patient became volume overloaded given the total amount of fluids being infused. He then underwent monitored diuresis. The IV amiodarone was transitioned to oral amiodarone. Despite these interventions, the patient continued to experience episodes of ventricular tachycardia resulting in additional bolus infusions of amiodarone. Maintenance infusions of amiodarone were reinstated. The Infectious Disease Service was consulted, and the patient was transitioned to cefazolin and rifampin given the culture and sensitivity results identifying MSSA (see Table 1 for susceptibility profile). Rifampin was added due to the presence of an AICD/pacemaker. The leukocytosis had resolved and defervesce had occurred. The patient continued to have runs of ventricular tachycardia and received two appropriate defibrillations from the AICD. The patient was transitioned to oxygen via nasal cannula. Precedex was initiated at night given the patients disrupted sleep-wake cycle and disorientation. Repeat blood cultures were obtained with resin which identified no growth. Interrogation of the pacemaker was performed, and the lidocaine infusion was resumed.

The pacemaker interrogation identified a ventricular tachycardia (VT) zone of 140–150 seconds. Appropriate anti-tachycardia pacing was identified 18 times with four AICD discharges. During this interval mexiletine (ATP) triggered V-tach intermittently. The patient's device was at elective replacement status and end-of-life (EOL) of the device was imminent. The patient recognized that his symptoms of dizziness and dyspnea were concordant with the VT episodes. A transesophageal echocardiography (TEE) was recommended once the patient's hemodynamics improved. Given the pacemaker EOL potential status, transcutaneous pacer pads were placed as a precaution. Palliative care was then considered and proposed to the family. Diuril was started and the Precedex was discontinued once the sleep-wake cycles improved. The lidocaine drip was

Table 1. Antibiotic susceptibility testing for *Staphylococcus aureus*.

Blood culture results (peripheral source)	Susceptibility testing for <i>Staphylococcus aureus</i>
Antibiotic	Sensitivity
Benzylpenicillin	≥0.5 Resistant
Beta-lactamase	Positive
Cefazolin	Sensitive
Cefoxitin Screen	Negative
Ciprofloxacin	≤0.5 Sensitive
Clindamycin	≤0.25 Resistant
Erythromycin	0.5 Resistant
Gentamicin	≤0.5 Sensitive
Inducible clindamycin	Positive
Levofloxacin	≤0.12 Sensitive
Linezolid	2 Sensitive
Oxacillin	0.5 Sensitive
Rifampicin	≤0.5 Sensitive
Tetracycline	≤1 Sensitive
Trimethoprim/sulfamethoxazole	≤10 Sensitive
Vancomycin	1 Sensitive

transitioned to amiodarone; however, seven more runs of VT were identified the following night. The patient was started on mexiletine and the amiodarone infusion was continued. A transthoracic echocardiogram (TTE) was obtained that noted a dilated left ventricle (LV) with segmental wall motion abnormalities with a LV ejection fraction (LVEF) of 35%, moderate concentric left ventricular hypertrophy, left atrial enlargement, and with pacemaker leads in the right side of the heart with mitral annular calcification with mild mitral regurgitation.

A TEE was performed once the patient was hemodynamically stable. Results demonstrated fibrinous lead vegetations and probable abscess along the lateral wall of the right atrium measuring 4.0×4.4×3.9 cm with a lucent core encasing the lead. (Figures 1, 2) Segmental wall motion abnormalities with moderate LV dysfunction and moderate mitral and tricuspid valve regurgitation were identified. Tertiary care transfer was proposed at this point given the need for further evaluation, including but not limited to lead removal, surgical drainage of the abscess, electrophysiology (EP) study for ablation of monomorphic V-tach, and pacemaker generator exchange. Gentamicin was initiated.

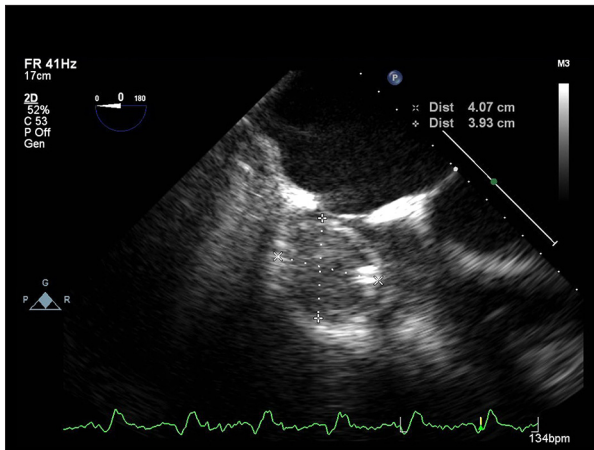


Figure 1. TEE (transesophageal echocardiogram) image of 4.07×3.93 cm intramuscular ovoid mass with lucent core with a circumferential hyperechoic ring in the wall of the right atrium encasing the atrial lead indicative of intra-atrial abscess.

Considering the patient's comorbidities and the requirements for open heart surgery, transfer to a tertiary care facility was declined. After 36 additional hours of the current medication regimen without any further VT episodes or VT storm, the lidocaine and amiodarone drips were discontinued. Mexiletine and amiodarone (oral) were continued. His defibrillator and detection functions were inhibited once hospice care had been elected. The pacemaker function was left intact. Antibiotic treatments to date had totaled 15 days of IV therapy and were to be continued for an additional two weeks' post discharge: rifampin 300 mg orally per day and Keflex 750 mg orally twice daily. The patient was then discharged home with hospice as requested, with outpatient follow-up.

Discussion

This case demonstrated how hematogenous dissemination of MSSA infections from a diabetic foot ulcer and osteomyelitis can seed pacemaker hardware resulting in an intracardiac abscess. Unfortunately, our patient was too ill to undergo all procedures required to eradicate the abscess and infected pacemaker hardware. The standard of care would be complete hardware removal. Conservative management would include indefinite or prolonged antibiotic therapy, with the notion that intracardiac abscesses and infected pacemaker leads/wires cannot be cured with antibiotics alone. This conservative treatment approach would be deemed necessary in a select population that cannot undergo surgical intervention. As extrapolated from Kang et al., early surgical intervention significantly reduces death from any cause and from embolic events. Poor prognostic factors were evident in our patient, including moderate congestive heart failure, altered mental status, and

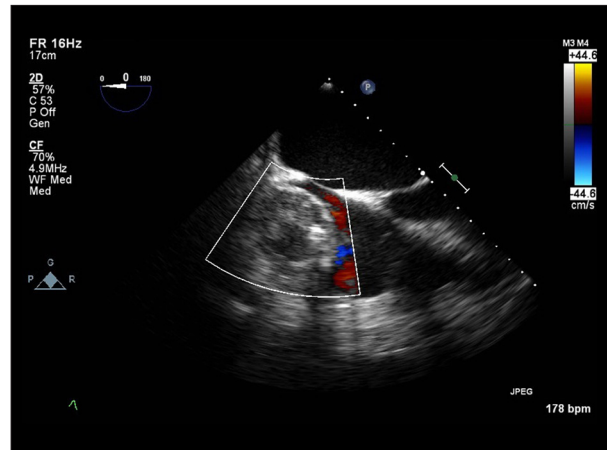


Figure 2. TEE (transesophageal echocardiogram) image utilizing color flow doppler function of the 4.07×3.93 cm intramuscular ovoid mass with lucent core demonstrating that the abscess is not contiguous with the intracardiac blood flow within the right atrial chamber and is confined within the right atrial myocardium.

staphylococcal inoculation and dissemination. The true origin of this patient's intracardiac abscess was never truly identified.

The foreign material of an AICD allows for a biofilm glycocalyx to be created, as is known to be produced by MSSA [2]. Phagocytosis by endothelial cells can convey protection against the host immune system, leading to persistent and difficult eradication of *S. aureus*. The lead implantation in the right atrium creates a direct route for inoculation into the myocardium. In our patient case, hematogenous dissemination to the coronary vasculature was less likely given the right atrial involvement and the lack of septic pulmonary emboli. Antibiotic bead implantation was not considered given the patient's comorbidities and deferment to a tertiary care center. In-hospital survival rates are considerably low for *S. aureus* in comparison to non-*S. aureus* infections (72% versus 88%, respectively). These patients have a 3-fold increase risk when LVEF <40% or abscess are evident, especially with prosthetic or foreign material present [5,7].

Our case reported is not the first to suggest an infection from a hematogenous dissemination from a lower leg wound [1]. Studies have found that 30% to 40% of patients with *S. aureus* bacteremia develop endocarditis [8]. Dicloxacillin, cefazolin, daptomycin, and vancomycin all exhibit activity against *S. aureus* and are used in conjunction with rifampin and/or gentamycin for synergism against *S. aureus*. After consulting with Infectious Disease Services, it was determined that clearing the infection would not be possible, especially given the patient's dependence on the implantable cardiac device to continue life maintenance, which is different than treating

for infectious endocarditis with IV antibiotics for at least six weeks if considering treatment for prosthetic valve endocarditis. Thus, it was considered that despite the length of duration of IV antibiotic therapy, this patient would not be able to eradicate the infection.

Conclusions

It is to no surprise that this patient's prognosis was dismal without surgical intervention; and the patient was discharged home with conservative management and hospice. The patient did not exhibit any further runs of VT, remained afebrile without leukocytosis with two negative blood cultures (with resin), and was stable clinically at discharge. This case report reiterates the importance in distinguishing between infectious

endocarditis and the less common intracardiac abscess with lead infection when considering long-term survival and prognosis in patients with known risk factors, prior infections, and inoculation with prior recurrent *S. aureus* infections.

Acknowledgements

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Conflicts of interest

None.

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