

Left-sided *Pseudomonas* Endocarditis with Disseminated Septic Emboli

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ABSTRACT

A 59-year-old man presented with sharp chest pain, hypoxia, and tachycardia. His past medical history included intravenous drug use (IVDU). *Pseudomonas aeruginosa* was isolated from his blood. *Pseudomonas aeruginosa* is a rare cause of endocarditis. Patient revealed that he injected drugs intravenously with tap water. Transesophageal echocardiogram revealed vegetation on the anterior mitral leaflet with associated mitral regurgitation. Patient was successfully treated with meropenem and tobramycin and underwent mitral valve replacement without complications. Majority of IVDU-related endocarditis caused by *Pseudomonas* involve right-sided valves, but our case is unique as it demonstrates left-sided endocarditis in a patient with IVDU. A combination of aggressive medical and early surgical treatment with valve replacement has enabled this patient to successfully recuperate.

KEYWORDS: endocarditis, pseudomonas, intravenous drug abuse

INTRODUCTION

Infective endocarditis (IE) is a life-threatening infection of the endocardial surfaces of the cardiac valves. Gram-positive cocci, as Staphylococci and Streptococci, are the leading causes of IE. Gram-negative rods and fungi are uncommon causative pathogens.¹ In this case, we diagnosed IE secondary to *Pseudomonas aeruginosa*, a very unusual cause of endocarditis. Involvement of the mitral valve is a unique presentation of our case.

CASE

A 59-year-old man with a past medical history of alcohol abuse and ongoing IVDU, presented to the emergency department with intermittent chest pain. The pain started 4 days prior to the encounter, was sub-sternal, pleuritic, sharp, and non-radiating. He also reported diaphoresis and decreased appetite. He denied associated dyspnea, fever, or chills. He has a history of smoking and denied prior history of cardiac disease. Family history was unremarkable.

The patient's initial oxygen saturation was 91%, improving

to 98% with two liters of oxygen via nasal cannula. His respiratory rate was 22 and heart rate was 120 beats per minute, and he was afebrile with a blood pressure (Bp) of 115/65. On exam, funduscopy revealed normal appearing retinas, lungs were clear to auscultation, heart exam significant for regular tachycardia with no added sounds or murmurs, and extremities with no edema, rash, or nail changes. The rest of the physical exam was otherwise unremarkable. Work-up in the emergency department included a normal complete blood count with a white cell count of 4,100/ μ L, hemoglobin of 13.6 gm/dl, mean corpuscular volume of 92 fl, and platelets of 269,000/ μ L. Patient's blood chemistry was significant for a sodium of 139 mmol/L, potassium of 4.2 mmol/L, bicarbonate of 24 mmol/L, urea of 9 mmol/L and creatinine of 0.7 mmol/L. His international normalized ratio was 1.3. Initial troponin level was 5.78 (Normal <0.03), with no dynamic ST/T wave changes or right ventricular strain pattern on electrocardiogram. Computed tomography angiogram was ordered and revealed emboli in the sub-segmental right lower lobe pulmonary arterial branches. The patient was started on heparin infusion for his pulmonary embolism. His troponin elevation was attributed to demand ischemia, but a trans-thoracic echocardiogram (TTE) was ordered for follow-up. It showed an ejection fraction of 40 to 45% with apical hypokinesia. Overnight, the patient's chest pain improved, but he had a febrile episode with dysuria. He was started empirically on ceftriaxone for a urinary tract infection, and blood and urine cultures were collected. Two sets of blood cultures grew gram-negative bacilli and the urine culture was negative. Over the next 24 hours, he became hypotensive but was fluid responsive. Given his active septic picture, antibiotics were escalated to piperacillin/tazobactam (Zosyn).

On hospital day 3, the patient developed severe persistent abdominal pain and a CT scan of the abdomen revealed a small splenic infarct. By that time the blood cultures grew pan-sensitive *Pseudomonas aeruginosa*. This raised suspicion for a possible source showering emboli, specifically infective endocarditis, given his IVDU history. A transesophageal echocardiogram (TEE) was ordered, and revealed a large highly mobile 2.2 x 1.1 cm vegetation of the anterior leaflet of the mitral valve with accompanying mild to moderate mitral regurgitation (**Figures 1,2**). No valve abscess was appreciated and no other vegetations were seen over the rest of the cardiac valves.

On further history review, the patient confirmed injecting methamphetamine using tap water and occasionally his own saliva a few weeks prior to his admission. Thorough examination did not reveal any new skin lesions or visual defects. There were no signs of heart failure or new neurological deficits. Based on TEE results, antibiotics were changed to intravenous meropenem and tobramycin. By the fourth day of hospitalization, the patient had a new onset progressive headache with dizziness, but a non-focal neurological exam.

Figure 1. Trans-esophageal echocardiographic image of the vegetation (red arrow) over the anterior leaflet of the mitral valve.

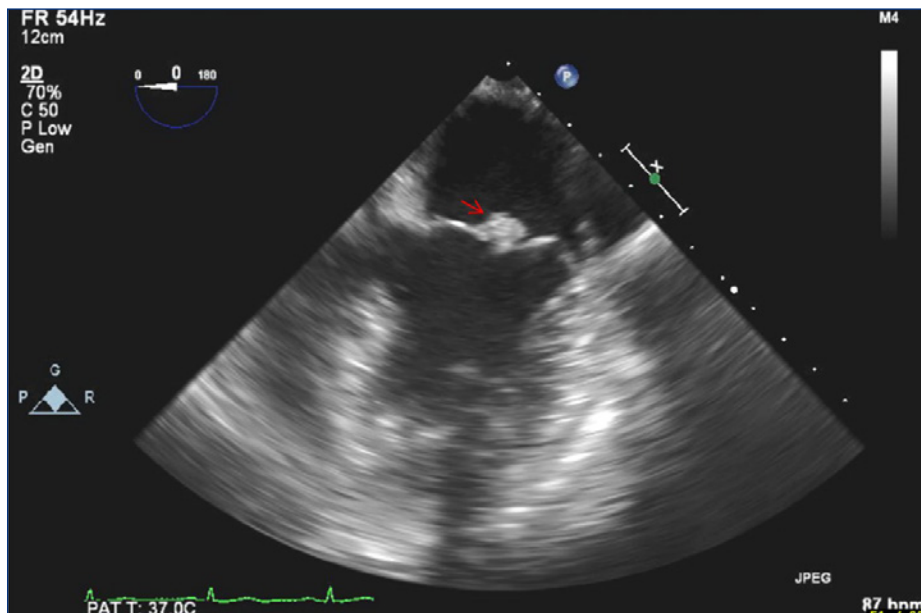
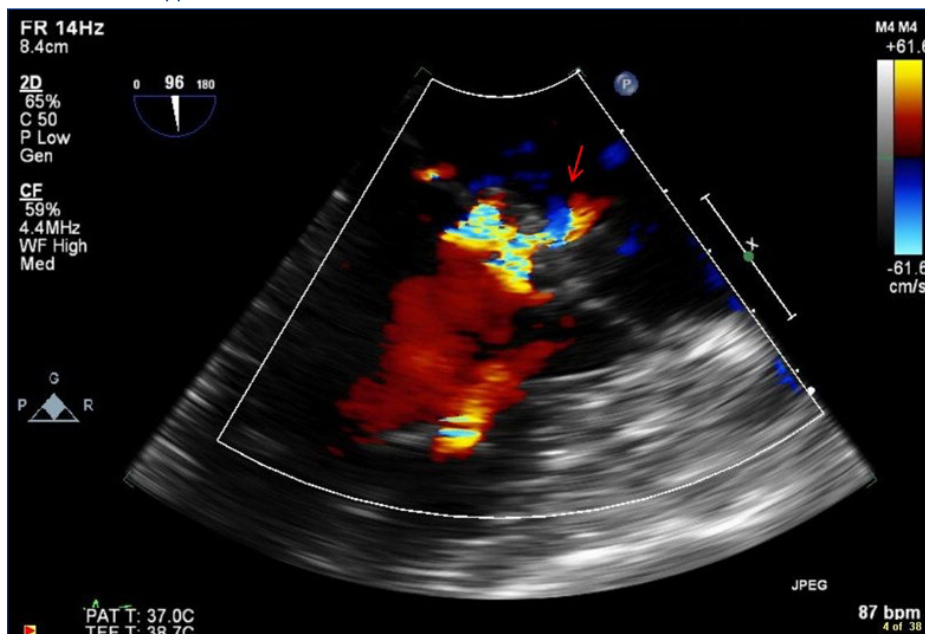


Figure 2. Trans-esophageal echocardiographic image of regurgitant flow (red arrow) across the mitral valve on color doppler view.



Computed tomography of the brain revealed high-density foci overlying the left frontal and parietal cortex, which were reported as possible meningeal abnormalities versus hemorrhagic emboli. Anticoagulation was stopped and he was found to have magnetic resonance imaging (MRI) findings consistent with multifocal acute emboli, possibly septic, with accompanying hemorrhage.

Follow-up blood cultures were negative, fever subsided, and repeat neurological exam was normal. Given the size of the vegetation and the multiple septic emboli, he was transferred to a facility with cardiothoracic services for valve replacement. Patient underwent left-heart catheterization revealing normal coronaries. Right-heart catheterization revealed low filling pressures and cardiac output at 6.5 L/min. After careful review of his reports and completion of an antibiotic course, the patient underwent a mitral valve replacement, with no immediate complications. Follow-up TTE revealed a well-functioning bioprosthetic valve.

DISCUSSION

Pseudomonas aeruginosa endocarditis is an uncommon condition, although its incidence increases with higher frequency of drug abuse, heart surgery, and *P. aeruginosa* bacteremia. Current evidence indicates that gram-negative bacilli are responsible for 5.3–12% of endocarditis cases, and *P. aeruginosa* is present in 30 to 50% of these cases.²⁻⁴ Estimates of the incidence of infective endocarditis in IVDU have been hampered by the lack of reliable data; approximately 2 to 4 cases per 1000 years of IVDU have been reported.⁵

Our patient was found to have *pseudomonas* bacteremia, with no obvious risk factor other than IVDU. Despite having urinary symptoms, his urine cultures were negative. Therefore, we hypothesized that his febrile illness may have been triggered by IE.

In the case of IVDU-related endocarditis, bacteria have a predilection for the right side of the heart, affecting the tricuspid valve in up

to 75% of cases as compared to 9% in cases of IE in non-IVDU patients. Tricuspid valve involvement presents with cough, hemoptysis, and discrete lung lesions. In our patient, the source of the pulmonary emboli was never identified, although a right-sided lesion would be most likely. And although the echocardiograms never confirmed concomitant right-sided vegetations, the pulmonary valves were reported as poorly visualized, and may have been the source of small vegetations causing sub-segmental pulmonary arterial occlusion. In contrast, the mitral valve involvement in IVDU patients is far less common, representing only 30% of cases.^{6,7} Compared to right-sided disease, left-sided *P. aeruginosa* endocarditis presents with fulminant, rapidly progressive symptoms with associated embolic events. According to a recent literature review summarizing 40 cases of the left-sided *P. aeruginosa* endocarditis in non-intravenous drug users, the overall mortality was about 64%.⁸⁻¹⁰ Tricuspid involvement is associated with better prognosis compared to left-sided valve involvement.

Our case is unique due to IE caused by pseudomonas with involvement of a left-sided valve. With disseminated septic foci, medical management becomes a challenge. The possibility of septic embolization in the setting of IE must always be considered with vigilance.^{11,12} In one large series, systemic arterial embolization or septic pulmonary infarction occurred in approximately 33% and 11% of cases, respectively.¹¹ Although mortality attributable to IE can exceed 30%,^{11,13} it is even higher among patients who experienced septic embolism events.¹⁴ Medical treatment alone is rarely successful in left-sided infective endocarditis caused by *Pseudomonas aeruginosa*, but we were able to clear his blood cultures with a combination therapy of meropenem and tobramycin due to early diagnosis.¹⁵⁻¹⁸

Early surgical consult is preferred in left-sided valve involvement and early valve replacement has been shown to improve prognosis. Most authorities now recommend treatment of left-sided endocarditis caused by *Pseudomonas aeruginosa* with early valve replacement accompanied by a 6-week course of high-dose combined extended spectrum β -lactam plus aminoglycoside antimicrobials.¹⁶⁻²⁰ Such a combined surgical and medical approach is associated with improved survival.^{17,19} Prognosis for medical therapy alone is worse compared to medical plus surgical therapy. Our patient's complicated hospital course led to a delay in surgical intervention, as the recent hemorrhagic cerebral emboli posed a challenge prior to taking the patient to surgery, especially with the expected need for anticoagulation afterwards. He eventually underwent a successful bioprosthetic mitral valve replacement, with complete resolution of his infection on both clinical and radiological grounds. The choice of the bioprosthetic valve was mainly due to the patient's active drug use and anticipated challenge with maintaining long-term anticoagulation.

CONCLUSION

In summary, we describe a male patient with history of IVDU presenting with left-sided endocarditis with *Pseudomonas*. This presentation is unique with respect to the bacterial pathogen involved and left-sided involvement. Early identification and aggressive treatment is key to successful recovery. Early surgical consultation is imperative as evidence demonstrates improved outcomes compared to medical treatment.

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