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Case Presentation

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Polymicrobial Infective Endocarditis in the Setting of Intravenous Drug Use

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Summary

The pathogenesis of infective endocarditis (IE) involves the interaction of microorganisms such as bacteria or fungi with the endothelial lining of heart valves. Patients with IE typically have a pre-existing valvular defect that allows highly virulent organisms to attach to valve leaflets. In patients with a history of intravenous drug use (IVDU), a leaflet of the tricuspid valve is typically involved in the setting of bacteremia or fungemia as the right heart is the site of venous return. Recent data shows that 80-90% of IE cases are caused by microorganisms such as staphylococci or streptococci, but the isolation of more than one microorganism is only seen in around 1-6.8% of cases. Here we discuss a case of a patient suffering from polymicrobial IE growing both methicillin-sensitive staphylococcus aureus (MSSA) as well as group A streptococcus (GAS) pyogenes with a history of IVDU.

Background

Polymicrobial IE is a less common phenomenon than monomicrobial IE but has been seen more frequently in patients with comorbidities such as diabetes, prosthetic values, or IVDU. The prognosis of polymicrobial IE depends on the speciation of microorganisms as well as the degree of antimicrobial/surgical intervention rather than the number of organisms identified **[1-11].** Therefore, the proper identification and treatment are necessary for patients susceptible to polymicrobial IE to maximize outcomes and reduce morbidity/mortality.

Case Presentation

This is a male patient in his 30s with a past medical history of tobacco use, IVDU, major depressive disorder, generalized anxiety disorder, and chronic left lower extremity wound presenting to the Emergency Department (ED) with shortness of breath. During the first admission, the patient was tachycardic to 114 and tachypneic to 20. He was otherwise hemodynamically stable with a blood pressure of 117/60, afebrile at 97.6, and saturating 98% on room air. Transthoracic Echo (TTE) was obtained that did not show signs of vegetation, however, CT chest showed innumerable, predominantly peripheral nodular opacities bilaterally, some of which were cavitating, that were highly suggestive of septic emboli **(Figure 1).** Blood cultures grew GAS pyogenes as well as MSSA x2. Infectious Disease (ID) was consulted who recommended repeating the TTE which showed a small, 0.5 cm (W) x 0.6 cm (L) vegetation on the right atrial aspect of the posterior leaflet of the tricuspid valve with mild to moderate regurgitation **(Figure 3).** Despite these findings, the patient was insistent on leaving against medical advice due to family reasons. Infectious disease recommended discharging with oral amoxicillin/clavulanate, however, the patient left prior to initiation. The patient then returned to the ED later that day for continuation of therapy, however, left against medical advice again due to family reasons. The patient presented to the ED for a third time to resume IV antibiotics four days from the initial presentation. He was reporting symptoms of opioid withdrawals, musculoskeletal point tenderness to his mid-back, and left lower extremity pain secondary to chronic wound ulcerations. He denied chest pain, shortness of breath, headache, fever, chills, nausea, vomiting, diarrhea, dysuria, abdominal pain, or neuropathy. The patient was afebrile with a temperature of 97.9, heart rate of 90, respiratory rate of 16, blood pressure of 122/78, saturating 97% on room air. His complete blood count was notable for a white cell count of 14.9. Erythrocyte sedimentation rate/C-reactive protein was 118/128.21. Chest X-ray showed scattered patchy nodular opacities bilaterally and hazy opacification of the bilateral lower lung zones, suggestive of septic emboli **(Figure 2).** He was admitted to medicine for further work-up. Gram stain of blood culture continued to show GAS pyogenes as well as MSSA. CT thorax was obtained given persistent back pain that demonstrated abnormal cortical irregularity and widening of the L4-5 facet joints suspicious of septic facet arthritis. The patient remained hemodynamically stable on room air throughout his admission.

Investigations

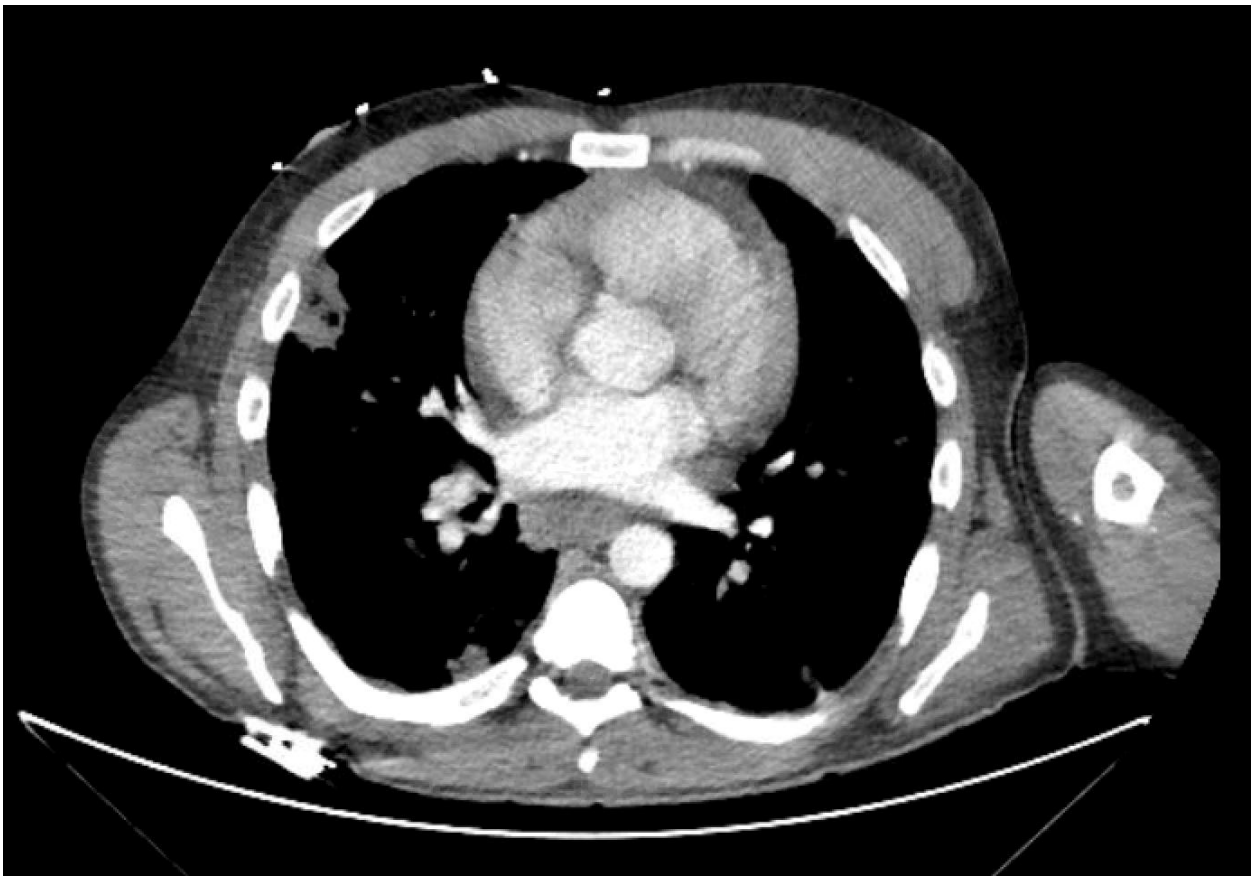

Figure 1: CT Chest showing peripheral nodules suspicious for septic emboli.

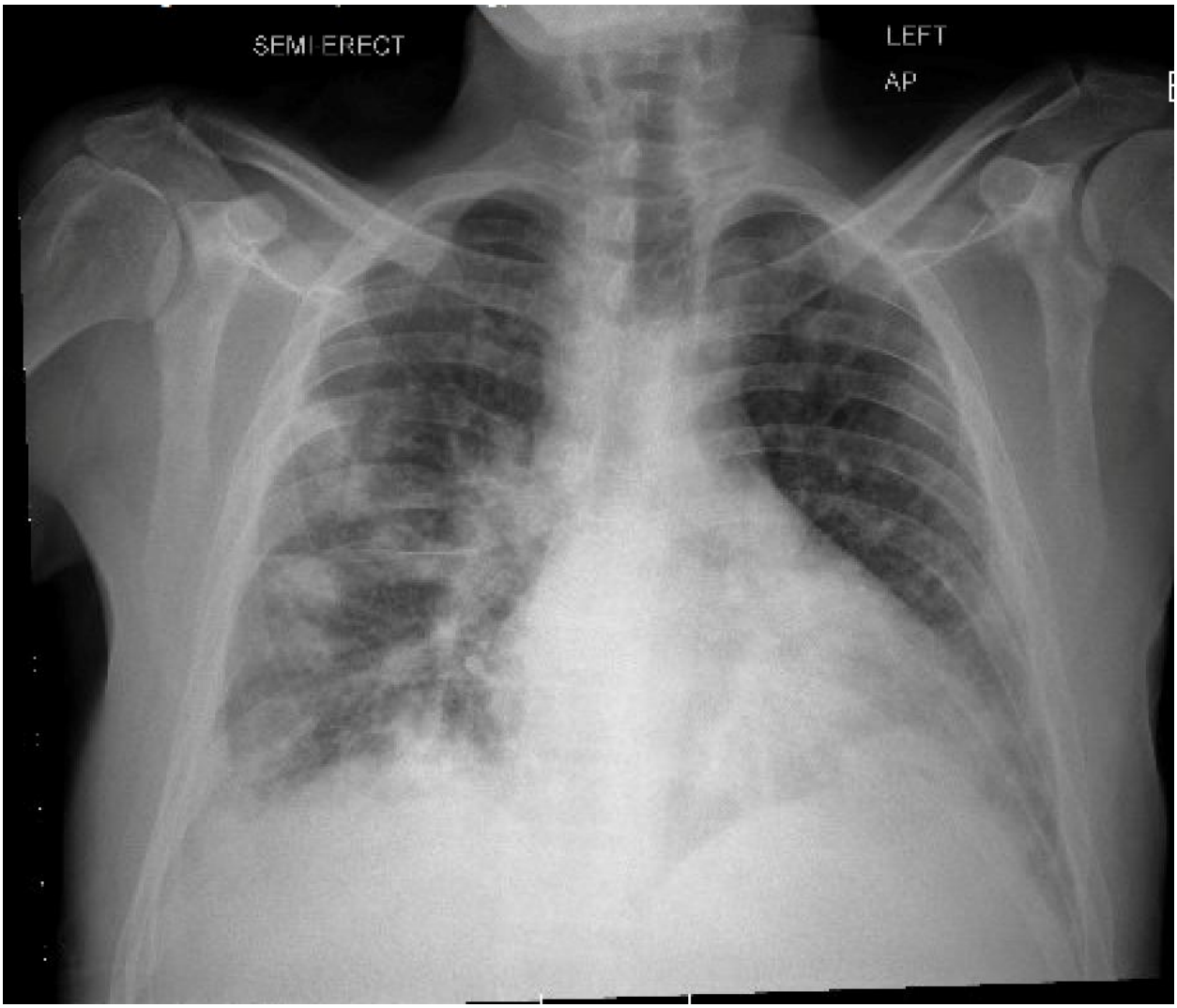

Figure 2: Chest X-ray showing bilateral nodular lung opacifications.

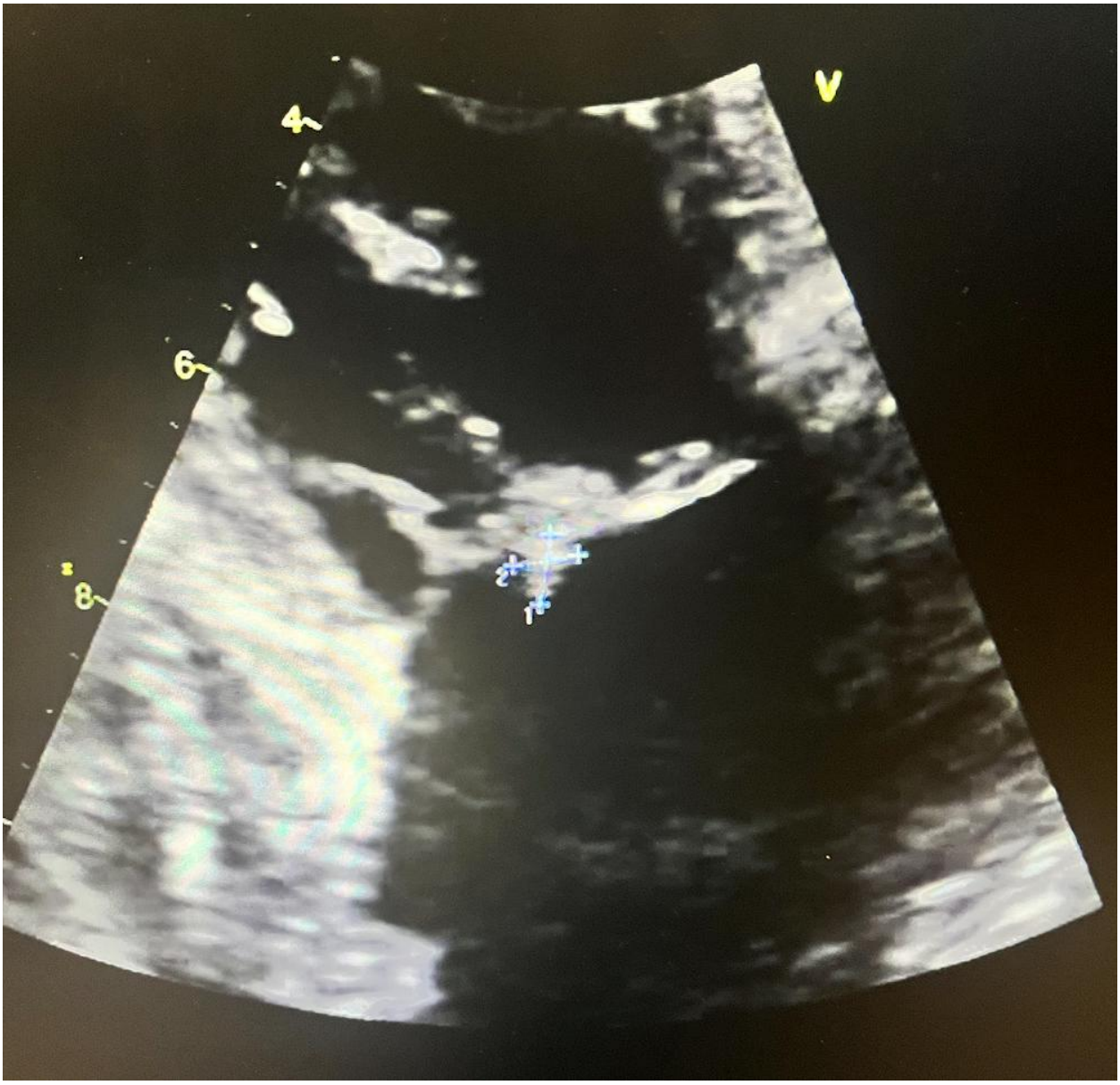

Figure 3: Transthoracic echocardiogram depicting tricuspid valve 0.5 cm (W) x 0.6 cm (L) vegetation.

Differential Diagnosis

Initial differential diagnosis consisted of multifocal pneumonia likely secondary to septic emboli given his IVDU and bacteremia. Concern for fungal or mycobacterial infection was warranted given cavitating nodular opacifications shown on CT chest as well as his social history of drug/tobacco use. Opioid withdrawal was ruled in given the patient's history of drug use requiring methadone, respiratory symptoms, and positive fentanyl screening on urinalysis. Major depressive/generalized anxiety disorder was considered given the patient's past medical history. Pulmonary embolism was less likely as his CT chest showed no evidence of central embolism.

Treatment

During the patient's first admission, he received one 57.5 ml (5 mg/kg) of gentamicin at 50 mls/hr, one 2 gm + one 1 gm of cefepime, one 100 ml at 200 ml/hr of cefepime, one 2 gm ceftriaxone 100 ml at 240 ml/hr, and two runs of vancomycin 250 ml/hr. On his second presentation returning after leaving against medical advice earlier that day, he received one 2 gm cefazolin and two 250 ml of vancomycin. On his third presentation where he agreed to remain at the hospital, he received two 2 g of cefazolin in the emergency department and was continued on 20 ml of cefazolin at 5 ml/min every 8 hours intravenously.

Outcome and Follow-Up

Patient had repeat blood cultures return as negative around 1 week after first admission. He remained afebrile without leukocytosis throughout his hospital course after continuing his IV cefazolin 2 g every 8 hours to complete a six week course. Follow up consisted of repeat CT chest to reassess the extent of septic emboli, and his ESR/CRP was monitored weekly.

Discussion

Endocarditis occurs when microorganisms colonize the endocardial surface of the heart, leading to inflammation and the formation of valvular vegetations **[1-3].** In polymicrobial endocarditis, the interaction between multiple pathogens can potentiate the virulence and pathogenicity of individual organisms. For example, synergistic effects between bacteria can result in increased adhesion, biofilm formation, and resistance to host immune responses, contributing to more severe disease presentation and complications **[4,5].** The diagnosis of endocarditis, including polymicrobial cases, relies on a combination of clinical criteria, imaging studies, and microbiological evidence. The guidelines provided by societies such as the American Heart Association (AHA) and the European Society of Cardiology (ESC) offer recommendations for the diagnosis and management of endocarditis typically based off the size and severity of the vegetation. These guidelines emphasize the importance of a multidisciplinary approach involving cardiologists, infectious disease specialists, and cardiac surgeons. In this particular case, the diagnostic pathway involved a comprehensive assessment of the patient's clinical history, physical examination, blood cultures, echocardiography, and CT/X-ray imaging.

Duke's criteria for infective endocarditis are clinical criteria used to diagnose infective endocarditis. These criteria help healthcare professionals make a more accurate diagnosis of IE based on clinical and laboratory findings. Duke's criteria are divided into two major categories: major criteria and minor criteria. A diagnosis of IE can be made based on the presence of either two major criteria, one major criterion and three minor criteria, or five minor criteria. Major criteria include positive blood cultures and endocardial involvement. Minor criteria include a predisposing heart condition, fever greater than 38°C (100.4°F), vascular phenomena such as septic emboli, immunologic phenomena such as glomerulonephritis, and microbiological evidence other than the positive blood cultures mentioned in the major criteria like positive serological tests for organisms associated with IE **[6,7].** The case presented several noteworthy aspects. Firstly, it involved a man in his mid 30s with a history of intravenous drug

use, which is a known risk factor for polymicrobial endocarditis. Secondly, the echocardiographic findings revealed tricuspid vegetations, indicating the extensive nature of the infection. Group A Streptococcus pyogenes was one of the microorganisms present and is a less common cause of infective endocarditis compared to other pathogens such as Staphylococcus and Streptococcus viridans **[8,9].** Finally, the patient's clinical course was complicated by embolic events to the lung and lumbar vertebrae, highlighting the need for medical intervention. A review of the literature identified several published cases of polymicrobial endocarditis. These cases demonstrated similarities in terms of patient demographics, risk factors, and clinical presentations. However, variations were observed in the pathogens involved, the extent of valvular involvement, and treatment outcomes. One example of documented polymicrobial IE reported a patient with a history of IVDU suffering from three oral anaerobes: Actinomyces odontolytica, Veilloenlla species, and Prevotella melaninogenica, which was attributed to licking a needle prior to injection. This patient was treated with a 6-week course of penicillin G and metronidazole. Another case of an immunocompromised patient on chemotherapy grew multidrug resistant Staphylococcus epidermidis and Corynebacterium striatum which required surgical valve replacement. The current clinical guidelines for the management of endocarditis provide valuable recommendations for the treatment of polymicrobial cases. These guidelines emphasize the use of broad-spectrum antibiotics to cover a wide range of pathogens, individualized surgical intervention based on the extent of valve damage, and close follow-up to monitor treatment response and prevent relapse **[10,11].** However, given the rarity of polymicrobial endocarditis, specific guidance tailored to these cases is limited.

Learning Points/Take Home Messages

- Highlighting the importance of assessing for polymicrobial IE in patients at risk.
- Using clinical judgment to repeat investigations when warranted in order to properly diagnose patients.
- Understanding the variations of severity/treatment between monomicrobial versus polymicrobial IE.
- Acknowledging the difficulty of lengthy in-patient hospital therapies in patients unable/unwilling to stay.

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Patient's Perspective

According to the patient: I am frustrated with the six-week duration of IV antibiotic therapy required for IE. I have family affairs to attend to and was hoping to be discharged with oral medications. Although I was counseled that oral antibiotics were not as effective as IV, I am willing to leave against medical advice for a third time as since I cannot not remain in the hospital solely for IV antibiotics. I was hoping was to be discharged to a rehabilitation center to complete the antibiotics intravenously, but I am afraid this would not be an option for me. I also understood the rationale behind why leaving with a Peripherally Inserted Central Catheter (PICC) would be a risk to me given my history of IVDU, however, I want to leave as soon as possible. I am feeling weaker lying in bed each day and am hoping for alternative options to continue with therapy. This calls to mind the importance of proper rehabilitation centers or other avenues of receiving IV antibiotic therapy in the outpatient setting since most patients have trouble remaining inpatient for six weeks solely for IV medications. Notably in patients with a history of IVDU, compliance with lengthy inpatient courses could be difficult resulting in leaving prior to antibiotic completion. This could further worsen outcomes as patients would be receiving insufficient amounts of antibiotics which could ultimately increase their risk of morbidity/mortality.

Citation of this Article

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