Case Report

An Unusual Presentation of Infective Endocarditis

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Abstract

Introduction
Leukocytoclastic vasculitis is the inflammation of small blood vessels due to leukocyte migration that comprises a wide range of differentials. It can be caused by autoimmune disorders, infections, neoplasms, or certain medications and warrants prompt recognition and therapy for optimal patient outcomes.

Case Presentation
Here we present a case of a 37-year-old male who presented with a painful, petechial rash on his torso and extremities. Skin biopsy revealed leukocytoclastic vasculitis, thereby prompting a comprehensive investigation into the underlying etiology leading to the diagnosis of *Pseudomonas aeruginosa* infective endocarditis.

Conclusion
Pseudomonas aeruginosa is a rare cause of infective endocarditis with a high mortality and morbidity rate. This case highlights the importance of taking a thorough history and physical along with a complete workup of vasculitis as the underlying cause can be life-threatening.

Keywords
infective endocarditis; leukocytoclastic vasculitis; *Pseudomonas aeruginosa*

Introduction
Infective endocarditis (IE) caused by *Pseudomonas aeruginosa* is a rare clinical encounter. The majority of cases are associated with injection drug use, prosthetic heart valves, and pacemakers.[1] IE remains a diagnostic challenge due to its variable presentations. Prompt recognition and initiation of treatment are crucial due to the substantial morbidity and mortality in *Pseudomonas aeruginosa* endocarditis.[2] This case highlights an unusual presentation that led to the diagnosis of *Pseudomonas aeruginosa* IE.

Case Presentation
A 37-year-old male presented with a painful, petechial rash on his torso and extremities. The patient described the rash as burning, and he suffered from nosebleeds. The patient denied signs of fever, shortness of breath, chest pain, cough, diarrhea, or abdominal pain. The patient stated that 2 days prior to symptom development, he had lacerated his forearm on a rusty fence and rinsed the wound with water from a well. Vital signs were hemodynamically stable and without fever.

Physical examination revealed a diffuse non-blanching petechial rash along the bilateral lower extremities, upper extremities, abdomen, and back associated with +1 pitting edema; there was also a 2 x 1 cm left forearm laceration with uneven borders and surrounding erythema. Laboratory results revealed an elevated white blood cell count of 15 300 x 10^9/L, an elevated erythrocyte sedimentation rate of 34 mm/hr, and an elevated C-reactive protein of 15.8 mg/L. Lactic acid was within normal limits.

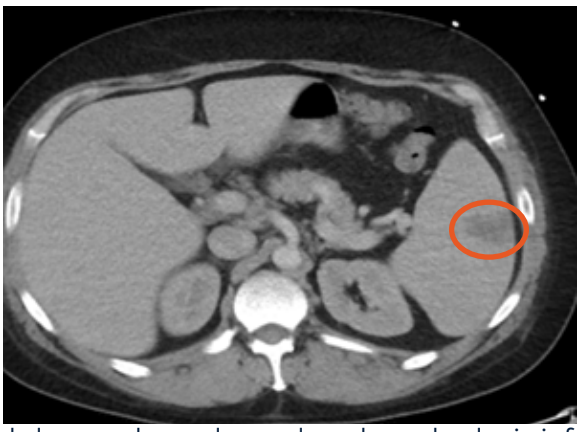

Figure 1. A CT scan of the abdomen showed a wedge-shaped splenic infarct (orange circle).

A punch biopsy of the left ankle and left leg revealed perieccrine neutrophilic infiltrate and leukocytoclastic vasculitis. The differential diagnosis was infection, vasculitis, erythema multiforme, Stevens-Johnson syndrome, or immunobullous reaction.

Computed tomography (CT) of the chest, abdomen, and pelvis revealed multiple wedge-shaped splenic infarcts and cavitary pulmonary nodules consistent with septic emboli. (**Figures 1**, **2**, and **3**) The infectious disease department was consulted, and the patient was started on vancomycin and cefepime for empiric bacteremia broad spectrum coverage. Initial blood cultures grew gram-negative rods. Subsequently, 2 sets of blood cultures grew *Pseudomonas aeruginosa*.

A transthoracic echocardiogram (TTE) was first obtained revealing a normal tricuspid valve structure and normal leaflet separation. There was a possible mobile echo-density concerning for vegetation on the tricuspid valve. The mitral valve, aortic valve, and pulmonic valves showed normal structure without abnormal leaflet separation, cuspal separation, prolapse, or other mobile echo-densities present.

A transesophageal echocardiogram (TEE) was then obtained for further visualization and revealed multiple vegetations on the tricuspid valve consistent with endocarditis. (**Figure 4**) On hospitalization day 3, the patient's white blood cell count was normal. The infectious disease department followed the patient throughout the case. Subsequent patient samples did not grow any organism besides *Pseudomonas aeruginosa*, but the infectious disease department recommended empiric gram-positive coverage with vancomycin due to the nature of the wound. A third set of blood cultures was obtained with no additional organismal growth.

The patient was continued on intravenous antibiotic therapy for 6 weeks. No surgical replacement of the tricuspid valve was performed because the patient did not exhibit signs of

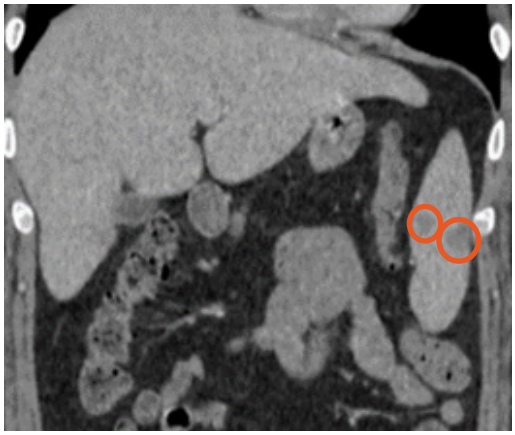

Figure 2. A CT abdomen sagittal view showed multiple splenic infarcts (orange circles).

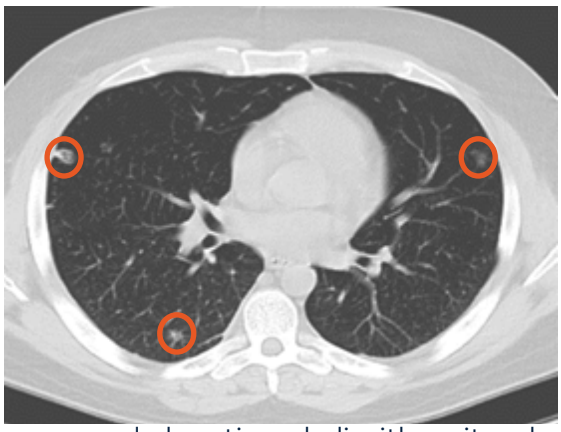

Figure 3. A CT scan of the lungs revealed septic emboli with cavitary lesions (orange circles).

heart failure and his sepsis had resolved within 3 days of hospitalization. On hospitalization day 22, vancomycin was switched to daptomycin by the infectious disease department due to a nephrotoxicity side effect. A 6-week course of antibiotics was completed with a resolution of his endocarditis and a marked reduction in his rash. The patient was counseled on appropriate wound care in the event of another traumatic wound and to clean properly with soap and water.

Discussion

IE is a rare infection of the endocardial surface of the heart. It is commonly associated with structural heart disease, congenital heart disease, prosthetic valves, intracardiac devices, and injection drug use. Prompt recognition and initiation of treatment are required as IE has a high mortality rate (~25%).[3] The Duke criteria, developed by Durack et al (Duke University), is a diagnostic tool used in the diagnosis of infective endocarditis.[4] Treatment is typically performed using 6 weeks of intravenous antibiotics after the first set of negative blood cultures. At least 3 sets of blood cultures should be obtained as they detect 96 to 98% of bacteremia.[5]

Pseudomonas aeruginosa is an aerobic, gram-negative rod commonly found in soil and fresh water. It is an opportunistic pathogen with the ability to form biofilms increasing its ability to resist host defenses and form antibiotic resistance.[6] The patient in our case had rinsed a forearm laceration with contaminated well water, likely to be the source of his infection.[7] *Pseudomonas aeruginosa* is a rare cause of IE accounting for about 3% of all cases, but associated with high morbidity and mortality.[2] Typical bacteria isolated in IE include *Staphylococcal aureus*, *Viridans streptococci*, *Streptococcus gallolyticus*, *Haemophilus species*, *Aggregatibacter actinomycetemcomitans*, *Cardiobacterium hominis*, *Eikenella corredens*, and *Kingella kingae* (HACEK group). The tricus-

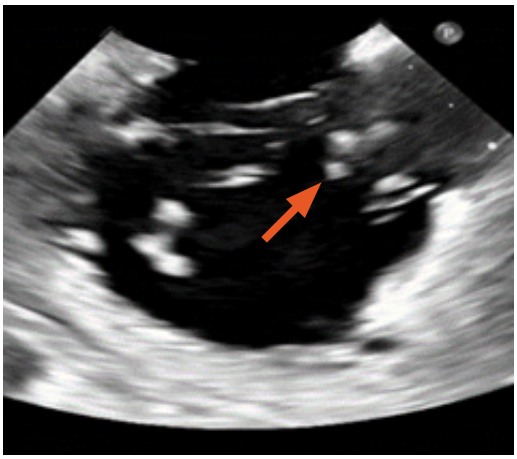

Figure 4. A transesophageal echocardiogram depicted multiple tricuspid valve vegetations (orange arrow).

pid valve is the most commonly affected valve, which was observed in our patient. The left ventricle can also be involved, and these patients have a poor prognosis often developing congestive heart failure or embolism of medium or large-sized arteries.[8] Prompt surgical evaluation for valve replacement is imperative as early valve replacement is associated with an improved prognosis.[9]

Echocardiography should be performed in all patients with suspected infective endocarditis. TTE is usually performed first with TEE afterward for further evaluation of cardiac complications, such as abscesses, leaflet perforations, and pseudoaneurysms. Chest radiography and computed tomography are useful for evaluation of septic emboli and infarcts. In our case, the punch biopsies of the patient's rash revealing leukocytoclastic vasculitis are unusual. Rashes usually associated with *Pseudomonas aeruginosa* include folliculitis and ecthyma gangrenosum.[6,10] Janeway lesions, which are non-tender hemorrhagic macules caused by septic emboli, and Osler nodes, which are tender purple-pink nodules caused by immune complex deposition, are skin findings associated with IE that were not observed in our patient.[3] One observational study revealed that patients with IE who have skin lesions have a higher risk of complications, including a higher rate of extracardiac complications, than those without skin lesions.[11]

Leukocytoclastic vasculitis is a small-vessel vasculitis due to immune complex deposition leading to activation of the complement system with the attraction of neutrophils causing damage to the vessel. It is a pathologic diagnosis with a large range of underlying causes making it a diagnostic challenge. Extracutaneous manifestations in leukocytoclastic vasculitis are rare with less than 30% of cases being involved outside of the skin. The patient in our case had no systemic symptoms, making the underlying cause of his vasculitis a diagnostic challenge. Infections commonly associated with leukocytoclastic vasculitis include *Mycobacterium*, *Staphylococcus aureus*, *Chlamydia*, *Neisseria*, and HIV.[11] The patient in our case likely developed *Pseudomonas aeruginosa* bacteremia from contaminated well water used to clean a wound. The bacteremia led to an immune response developing vasculitis of his skin.

Conclusion

Skin manifestations in IE are variable with Janeway lesions, Osler's nodes, and petechiae. None are pathognomonic for endocarditis. *Pseudomonas aeruginosa* is also a rare cause of IE with a high mortality and morbidity rate. This case highlights the importance of taking a thorough history and physical along with a complete workup of vasculitis as the underlying cause can be life-threatening.

Conflicts of Interest

The authors declare they have no conflicts of interest.

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