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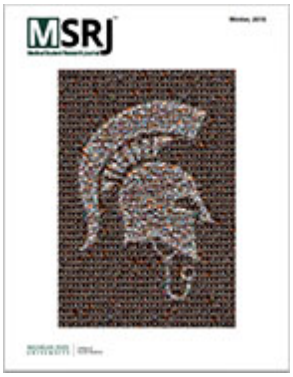


Aerococcus Viridans Infectious Endocarditis Complicated by Splenic Infarction

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# *Aerococcus Viridans* Infectious Endocarditis Complicated by Splenic Infarction

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**Abstract**

In this case report we discuss splenic infarction as a presentation for infectious endocarditis. While not unheard of, splenic infarctions are usually incidental findings and are not usually used to diagnose infectious endocarditis. Since our patient was on hemodialysis, had AIDS and blood cultures tested positive for *Aerococcus viridans* and *Streptococcus parasanguis*, we propose that atypical presentations of IE should be considered in immunocompromised patients.

**Introduction**

Infectious endocarditis (IE) is an infection of the endocardium which can present with various vascular phenomena ranging from major arterial emboli, intracranial hemorrhage, septic pulmonary infarcts and Janeway lesions. Vascular embolization is present in up to 22% to 50% of cases of IE[1].The risk of embolization increases with vegetations > 1 $cm^2$ . In our case report we describe a 40 year old HIV positive female on hemodialysis who presented with infectious endocarditis manifesting as multiple splenic infarctions in the absence of vegetations detected by transthoracic echocardiogram (TTE) or transesophageal echocardiogram (TEE). This raises the possibility of unusual vascular manifestations of infectious endocarditis in immunocompromised patients.

**Case Report**

Our patient is a 40 year old female with a past medical history significant for HIV with a CD4 count of 58, end stage renal disease on hemodialysis with AV fistula, chronic pancreatitis, seizures (controlled with levetiracetam) and type 2 diabetes mellitus who presented with a two day history of abdominal pain radiating to back. She also had diarrhea, diminished appetite, nausea, vomiting, fever, chills, chest discomfort and generalized malaise. On presentation her vitals were: Blood Pressure: 188/127 mm Hg, Temperature: 38 °C (100.4 °F), Heart Rate: 114, Respiratory Rate: 16, SpO2: 98 % (room air), Height: 160 cm (5' 3"), Weight 54 kg (119 lb 0.8 oz). On initial physical exam the patient was in mild distress, no skin lesions were noted, she had diffuse crackles auscultated in bilateral lung fields, no murmurs or rubs were heard on initial cardiology exam, she had tenderness to palpation in epigastric region, abdomen was soft, non-distended and bowel sounds were present. She was admitted for chronic pancreatitis exacerbation. Abdominal CT was performed, blood cultures collected and the patient was empirically treated with vancomycin and piperacillin/tazobactam.

Blood cultures taken on admission came back positive three days later for *S. parasanguis* and *A. viridans*. Five days after admission, a new grade III/VI holosystolic murmur at the mitral focus exacerbated on expiration was noted on cardiovascular examination. Patient also had a (+) rheumatoid factor during the admission, intermittent fevers and new splenic infarct found on

abdominal CT (figure 1). TTE and TEE four days after admission did not reveal any valvular vegetation or abscess. Repeat blood cultures also confirmed infection with *S. parasanguis* and *A. viridans*. The patient fulfilled Duke's criteria with two major criteria of new valvular regurgitation, blood cultures positive for *S. parasanguis* (part of the *S. viridans* group) and three minor criteria of vascular phenoma of splenic infarction, positive rheumatoid factor, and fever during her stay. She then began treatment for infective endocarditis by continuing the IV vancomycin with gentamicin for six weeks. The piperacillin/tazobactam was discontinued. The patient's hospital course was complicated by uncontrolled abdominal pain and social work placement; she was eventually discharged on hospital day 17 and within the next 30 days, she was readmitted twice for unrelated causes. She eventually finished her six weeks of vancomycin and gentamicin during the third hospital admission. Her blood cultures at seven days from the initial positive blood culture were consistently negative throughout her current and future hospitalization.

**Discussion**

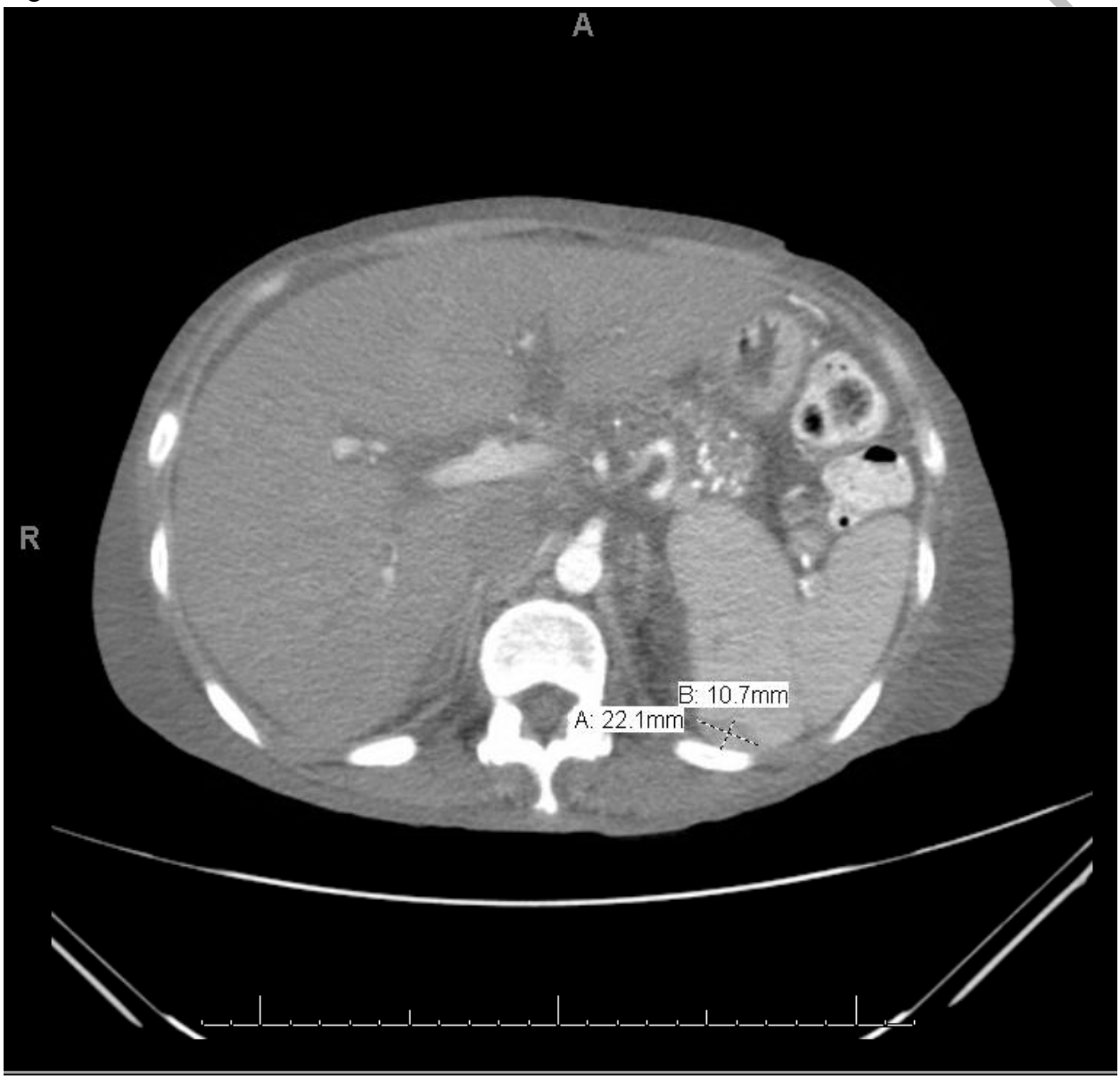
Vegetations in IE are composed of fibrin, platelets, microcolonies of microorganisms, and inflammatory cells[3]. These vegetations can then break off and embolize, leading to infection and infarction in distant sites. Embolization is common in IE with the lungs, central nervous system, bowel and spleen being the most common sites for embolization. Mitral valve vegetations have the highest chance of embolizing, followed by aortic and right sided vegetations. The infectious agent may also have a role in determining the risk of embolization, with staphylococcal and fungal IE having higher rates[4].

In this case, the HIV positive patient experienced multiple splenic infarcts without evidence of a vegetation on TTE or TEE. Complicating her immunocompromised status was our patient's end stage renal disease. Hemodialysis has been shown to significantly increase the risk of IE, with studies showing a relative risk of 16.9 of IE compared to the general population[5]. *Staphylococcus aureus* is the most common pathogen implicated in patients on chronic hemodialysis. Our patient had a polymicrobial IE with blood cultures that tested positive for a *S. parasanguis* and *A. viridans*. While *S. parasanguis* is a known cause of subacute bacterial endocarditis, *A. viridans* is a rare organism and concomitant coinfection suggests a nosocomial etiology. A study describing 11 cases of *A. viridans* IE showed a predisposition to embolization, with every case positive for vegetations[6]. Another case report postulated that immunocompromised status was a risk factor for *A. viridans* IE[7].

With our patient's uncommon presentation of splenic infarctions without vegetations as well as a polymicrobial IE with a rare organism, we posit that her immunocompromised status with chronic hemodialysis could have contributed to her presentation. Since our patient was started on empiric antibiotics, there is a possibility that our patient's vegetation could have embolized and

caused her splenic infarctions before TTE and TEE were done[4]. There could have also been a false negative result from the echocardiography, but this might be less likely because of the large sizes of vegetations described from previous cases of *A. viridans* IE[6]. While our patient fulfilled the Duke's criteria for IE, her uncommon presentation of splenic infarctions delayed her diagnosis and treatment until after her blood cultures returned positive and her new heart murmur was appreciated. We recommend that a lower threshold to diagnosis IE in immunocompromised patients with risk factors such as hemodialysis be used in order to facilitate effective treatment.

Figure 1




Abdominal CT of the patient. A 22.1mm x 10.7mm hypodense area is noted on the in the posteromedial spleen near the rib – a new finding compared to her previous CT. A small part of the pancreas is visible in this image, with calcifications indicative of her chronic pancreatitis.

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