

Splenic and Kidney Infarct: Sequelae of Subacute *Streptococcus mitis* Bacterial Endocarditis

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Abstract

Infective endocarditis (IE) is caused due to the vegetation on the heart valves, myocardium wall, or the pacemaker leads. Vegetation is a lesion that appears as a consequence of successive deposition of platelets and fibrin on the endothelial surface of the heart. Colonies of microbes can be usually found under the vegetation. Heart valves are involved more frequently as compared to other places. *Streptococcus mitis*, formerly known as *S. mitior*, is a commensal of the oral flora, however, if there is loss of integrity of the mucous membrane, the infection may disseminate to the blood flow. We describe here a rare presentation of *S. mitis*, causing IE and its complications in an immunocompetent patient.

Keywords: Emboli, endocarditis, splenic infarct

INTRODUCTION

Infective endocarditis is one of the life-threatening complications of valvular heart disease associated with high mortality and morbidity. Splenic infarct is a complication of left side endocarditis. Splenic infarctions may present with nonspecific symptoms such as low-grade temperature, fatigue, abdominal pain, nausea and vomiting. Many micro-organisms can cause infective endocarditis including *Staphylococcus aureus*, Streptococci, Enterococci, HACEK group and many others. We report here infective endocarditis caused by *Streptococcus mitis* which is an α -hemolytic streptococcus, unique to the oropharynx. It is a normal commensal to the oral cavity and is usually recovered from clinical specimens in mixed culture with other organisms.

CASE REPORT

A 64-year-old male with a history of type II diabetes mellitus, hypertension, and hyperlipidemia presented with left-sided abdominal pain and fever 38.3°C. His symptoms started 2 months before the presentation and were flu-like. His abdominal pain was in left upper quadrant, dull in character, nonradiating and associated with nausea and unintentional weight loss of 20 pounds. He received azithromycin twice and Tamiflu once as an outpatient for upper respiratory tract infection without any relief. He denied sick contacts,

intravenous drug abuse, recent travel, and dental procedures. He was chronically ill looking, had a body mass index of 25.4, heart rate of 97 beats/min, respiratory rate of 22 breaths/min, and blood pressure of 115/60 mmHg. On examination, he had no palpable lymph nodes, had mild abdominal tenderness in the left upper quadrant and no rebound tenderness on palpation and revealed Grade III/VI holosystolic murmur at the apex of the heart on cardiac auscultation. He had a white blood cell count of 32.7 thousand (normal; 4000–11,000/ μ l) with a left shift. Computed tomography of the abdomen and pelvis with intravenous contrast showed diffuse infarction of spleen [Figure 1, white arrow], thrombus in the distal splenic artery branch, and wedge-shaped infarct of the right kidney. Transthoracic echocardiography showed 13 mm \times 7 mm mobile vegetation on the posterior mitral valve leaflet [Figure 2, white arrow], small vegetation on the anterior mitral valve leaflet, and moderate mitral regurgitation. Multiple blood cultures grew *Streptococcus mitis*, sensitive to penicillin. On day 7, he underwent mitral valve replacement with bioprosthetic valve. His postoperative course was uneventful

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Figure 1: Computed tomography of the abdomen and pelvis with intravenous contrast showing infarction of the spleen (white arrows)

and was discharged to rehabilitation center with intravenous antibiotics for 4 weeks.

A large number of microorganisms have been implicated in infective endocarditis (IE). Streptococci and staphylococci account for 80%–90% of the cases in which identification is made.^[1] Among streptococci species, viridans are the most commonly isolated pathogens in IE followed by species sanguinis, bovis, mitis, and mutans.^[1] A study from the New York Hospital analyzed 330 patients with *Streptococcus* IE from 1970 to 1978 and reported the various species responsible were as follows: *S. viridans*, 58%; *S. mitis*, 31%; *S. bovis*, 27%; *S. sanguinis*, 24%; *S. mutans*, 7%; Vitamin B6-dependent *S. mitis*, 5%; *S. anginosus*, 4%; and others, 2%.^[1] *S. mitis* is a low-grade, alpha-hemolytic pathogen commonly found in oropharynx and nasopharynx, especially in the gingival crevice. These bacteria gain access to the bloodstream following trauma to the mouth and cause IE. In intravenous drug addicts, *S. mitis* has an inclination for right-sided endocarditis and left-sided endocarditis in nondrug addicts as seen in the case.^[2] Liver, spleen, and kidney embolism are seen in 40% of population diagnosed with IE of the left side of the heart.^[3] Spleen is vulnerable to infarction due to the absence of collateral supply between the branches and sluggish blood flow within red pulp.^[4] Septic emboli can result in the formation of abscess in multiple organs. Computed tomography is most sensitive imaging tool in the diagnosis of splenic, renal, and other organ infarcts.^[5] *S. mitis* is highly susceptible to penicillin G potassium therapy. Once identified, the antimicrobial therapy should be considered for a minimum of 4 weeks from last negative blood culture.

The implication of presenting this case is to identify *S. mitis* also as a causative agent of IE early in the course. Being a part of the normal microflora of the oral cavity, the pathogenic aspect of *S. mitis* is often ignored. Likewise, the presence of

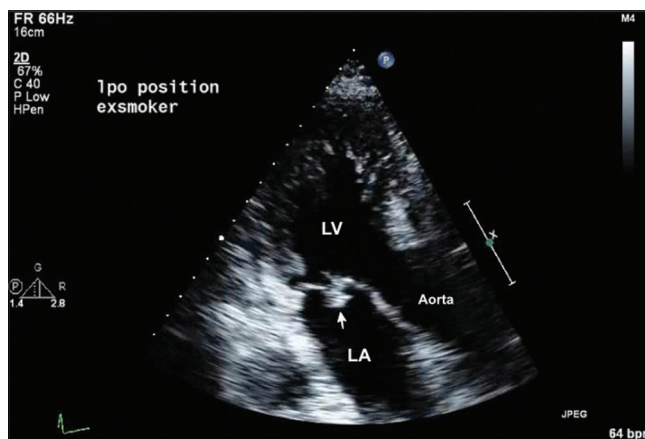


Figure 2: Transthoracic echocardiogram showing mobile vegetation (13 mm × 7 mm) on the posterior mitral valve leaflet (white arrow) and a small vegetation on the anterior mitral valve leaflet

S. mitis in the blood culture is believed to be a contaminant and underestimated; therefore, it is of prime importance that the two sets of blood culture are drawn from two different sites in a clear and sterile manner. Although *S. mitis* is of low virulence and it commonly affects the right side of the heart in patients with a history of intravenous drug abuse, it still carries the potential of damaging the native valves in an immunocompetent host if not diagnosed earlier. It is highly recommended to verify the presence of *S. mitis* in one set of blood culture, with a repeat set blood culture to avoid any confusion, and antibiotic sensitivity should be performed. Once identified, the antimicrobial regimen can be tailored as per the sensitivity to prevent antimicrobial resistance and adverse effects of broad-spectrum antibiotics such as *Clostridium difficile* infections.

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Conflicts of interest

There are no conflicts of interest.

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