CASE REPORT

Streptococcus agalactiae mural infective endocarditis in a structurally normal heart

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A 38-year-old Caucasian man with uncontrolled diabetes mellitus type 2 was admitted with a 1-week duration of fevers, chills, and a non-productive cough. He had a left ischiorectal abscess 1 month prior to admission. Physical examination revealed caries on a left upper molar and a well-healed scar on the left buttock, but no heart murmur or evidence of micro-emboli. Blood cultures grew Streptococcus agalactiae. A transesophageal echocardiogram revealed a mobile mass in the right ventricle that attached to chordae tendineae without valvular disease or dysfunction. A computed tomography (CT) with contrast revealed the mass within the right ventricle, a left lung cavitary lesion, and a splenic infarction. He was initially treated with penicillin G for a week. Subsequently, ceftriaxone was continued for a total of 8 weeks. A follow-up CT showed no evidence of right ventricular mass 8 weeks after discharge. This is the first reported case of S. agalactiae mural infective endocarditis in a structurally normal heart.

Keywords: infective endocarditis; Streptococcus agalactiae; streptokinase; rare disease; diabetes mellitus

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Received: 25 January 2016; Revised: 25 February 2016; Accepted: 1 March 2016; Published: 25 April 2016
Liver function tests and coagulation panels were normal on admission. A transesophageal echocardiogram (TEE) revealed a large, complex, multilobar, highly mobile echo-density seen in the right ventricle measuring up to 4 cm at the longest dimension that was attached to chordae tendineae (Fig. 1) and no evidence of intracardiac shunt. We found normal valves, normal cardiac dimensions and function, and no pulmonary hypertension. Differential diagnosis of intracardiac mass included thrombosis, neoplasm, and vegetation. A cardiac magnetic resonance imaging with gadolinium contrast was performed in order to differentiate them, and demonstrated no definite delayed gadolinium enhancement of the mass, making neoplasm unlikely (Fig. 2). We diagnosed *S. agalactiae* mural infective endocarditis involving right ventricular chordae tendineae.

He was initially treated with penicillin G for a week along with the extraction of the left upper molar with caries. After discharge, ceftriaxone was continued for a total of 8 weeks. Follow-up CT scan showed no evidence of right ventricular mass 8 weeks after discharge. We chose a CT scan as follow-up imaging of his vegetation because the initial TTE was limited due to poor image, a TEE was not available at his local hospital, and the initial CT scan revealed the vegetation.

**Discussion**

In spite of a comprehensive literature review, no definitive case has ever been reported regarding *S. agalactiae* right-sided mural infective endocarditis in a structurally normal heart (1). *S. agalactiae* is a rare cause of infective endocarditis causing 1.7% of all cases (2). It is a beta-hemolytic gram-positive bacteria that colonizes the female genital tract, the throat, and the rectum (3). In contrast to the incidence of *S. agalactiae* disease during pregnancy and the neonatal period, the incidence of invasive disease in non-pregnant adults has increased in recent years (2).

![Picture1: TEE](image1.png)

*Fig. 1.* A large, complex, multilobar, highly mobile echo-density in the right ventricle measuring up to 4 cm at the longest dimension that was attached to chordae tendineae.

![Picture2: Cardiac MRI](image2.png)

*Fig. 2.* No definite delayed gadolinium enhancement of the mass.

This increased incidence may be related to an increase in the prevalence of underlying medical conditions, such as diabetes, or to an aging population (2). A large, population-based analysis of the epidemiology of invasive *S. agalactiae* infection reported that almost 90% of patients had at least one underlying debilitating condition (2).

The source of infection in the majority of patients with *S. agalactiae* endocarditis was unclear (1, 2). However, our patient displayed signs of a systemic infection a week after completion of antibiotics for the ischiorectal abscess. Although his blood cultures revealed a different pathogen from his abscess culture, his ischiorectal abscess may have been polymicrobial. Thus, we concluded that either his ischiorectal abscess or caries of the left upper molar were the primary source of infection.

Mural endocarditis typically results from seeding of an abnormal area of endocardium during bacteremia or fungemia, or an extension of infection from underlying myocardial abscesses (4). However, Byung Joo Sun et al. (5) described that a large proportion of patients with infective endocarditis had no previous history of underlying heart disease: the pathophysiologic mechanism has not been clearly understood.

His right-sided vegetation could not adequately explain his splenic infarction because no intracardiac shunt including patent foramen ovale was observed. *Streptococcus* usually facilitates two factors: plasma-clotting factor and inhibitory factor of coagulation such as streptokinase (SK) (6). Human plasma which is coagulated by the bacteria’s plasma-clotting factor may become re-dissolved by their SK. The main sources of SK are beta-hemolytic streptococci of the Lancefield groups, A, C, and G, but not groups B (*S. agalactiae*) and F (7, 8). Therefore, the lack of SK
in *S. agalactiae* may explain the association with large vegetations and multiple septic emboli including his splenic infarction.

Well-established guidelines for treatment of valvular endocarditis point to aggressive and early surgical intervention when infection is associated with large vegetations, significant valvular or perivalvular complications, and thromboembolism. However, it is not clear whether this approach is appropriate for mural endocarditis (9). Our patient was treated successfully with antibiotics alone.

**Conclusion**

*S. agalactiae* is an uncommon cause of infective endocarditis. The large vegetations and frequent emboli in *S. agalactiae* have been attributed to the lack of streptokinase. The role of surgery in mural infective endocarditis is unclear and antibiotics alone may be sufficient.

**Conflict of interest and funding**

The authors have not received any funding or benefits from industry or elsewhere to conduct this study.

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