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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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A 38-year-old Caucasian man with a history of uncontrolled diabetes mellitus type 2 and asthma presented to the emergency department at a local hospital with a 1-week duration of fevers and chills associated with nausea, vomiting, and non-productive cough. He had a history of left ischiorectal abscess requiring incision and drainage in the emergency department a month prior to admission. At that time, the culture of his abscess was positive for methicillin-susceptible *Staphylococcus aureus* and he had completed 1 week of antibiotic therapy. He denied other respiratory symptoms, abdominal symptoms, recent dental work, sick contacts, or recent travel history. He was diagnosed with gastroenteritis and was prescribed ranitidine with improvement of his nausea and vomiting. However, his fevers and non-productive cough continued. Two days later, he presented to the emergency department again. He was found to have a leukocytosis and minimal linear opacity at the left lung base on chest X-ray. He was admitted to the local hospital and was started on ceftriaxone and azithromycin for community-acquired pneumonia. The blood cultures grew *Streptococcus agalactiae*. A transthoracic echocardiogram (TTE) was limited due to poor image but revealed no evidence of vegetation. A contrast-enhanced computed tomography (CT) scan demonstrated the mass within the right ventricle, a cavitary lung lesion, and a splenic infarction. On the fifth day of admission, the patient was transferred to our hospital for further evaluation and treatment.

Family history was significant for diabetes mellitus type 2 in his mother and metastatic cholangiocarcinoma in his father. He denied tobacco, alcohol, or intravenous drug use.

On admission, his temperature was 37.2°C, respiratory rate was 20 breaths per minute, pulse was 101 beats per minute, blood pressure was 134/96 mmHg, and body mass index was 39.6 kg/m^2. Physical examination revealed caries on a left upper molar, left lung crackles, splenomegaly, and multiple tattoos, but no heart murmur or evidence of micro-emboli in the retinae, skin, or mucous membranes. A well-healed scar was present on the left buttock without signs of fluctuance, tenderness, or fistula formation. The rest of the examination was unremarkable.

Laboratory testing showed a hemoglobin of 14.1 g/dl; a leucocyte count of 11,510 cells/μl with 67.7% neutrophils, 19.8% lymphocytes, 6.8% monocytes, 1.7% eosinophils, and 0.3% basophils; a platelet count of 221,000 cells/μl; a serum sodium of 133 mEq/l, a serum potassium of 3.6 mEq/l, a serum chloride of 102 mEq/l, a serum urea of 8.0 mg/dl, and a serum creatinine of 0.4 mg/dl.

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Liver function tests and coagulation panels were normal on admission. A transesophageal echocardiogram (TEE) revealed a large, complex, multilobar, highly mobile echodensity 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).

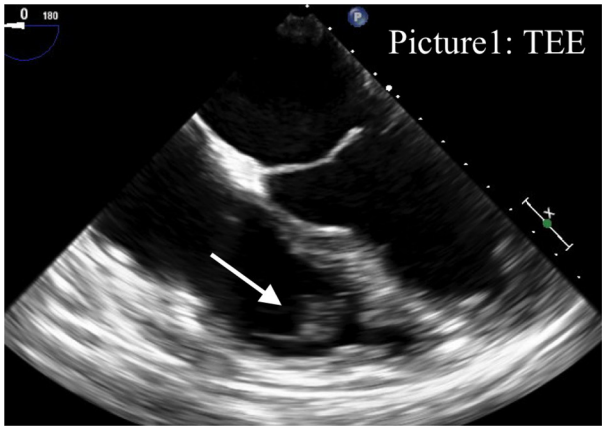

Fig. 1. A large, complex, multilobar, highly mobile echodensity in the right ventricle measuring up to 4 cm at the longest dimension that was attached to chordae tendineae.

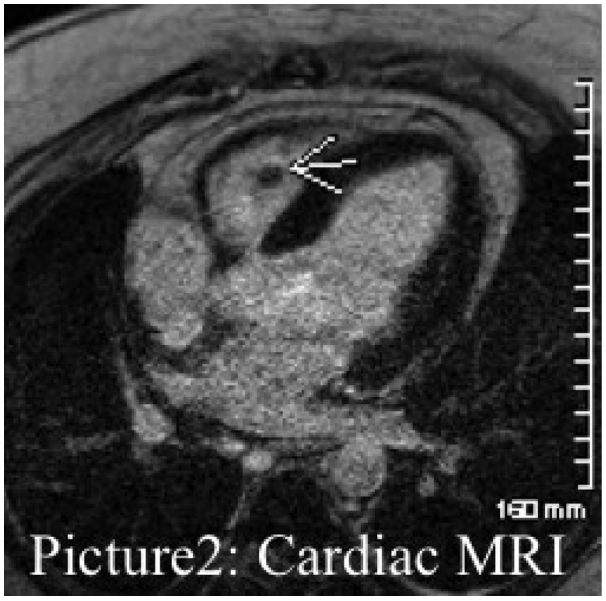

Fig. 2. No definite delayed gadolinium enhancement of the mass.

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

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