Case Report

Streptococcus gordonii: an emerging pathogen as a cause of infective endocarditis?

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Infective endocarditis (IE) is a serious illness that affects the endocardial surfaces of heart valves, the mural endocardium, and septal defects. It can cause severe complications, such as abscesses, aneurysms, heart failure, renal failure, and sepsis. Right-sided endocarditis is more prevalent in persons who inject drugs, accounting for 10% of all instances of IE. *Streptococcus gordonii* is a gram-positive bacterium that colonizes the oral mucosa, skin, and gastrointestinal tract. It is an opportunistic pathogen and a rare cause of IE. We describe a case of *S. gordonii* IE in a 47-year-old man who injected drugs and had a history of prior tricuspid valve IE treated with tricuspid valve replacement and mitral annuloplasty.

Introduction

Infective endocarditis (IE) is an uncommon but potentially fatal cardiac condition. Its incidence is 3-7.5 per 100,000 person-years, and its 5-year death rate is 45%, with an in-hospital mortality rate of up to 22%.[1] Individuals with IE often exhibit non-specific systemic symptoms such as chills, fever, tiredness, and night sweats.[2,3] A new murmur and positive blood cultures are often present. Despite advancements in early detection and treatment, IE is linked to substantial complications, such as cardiac abscess and aneurysm, heart failure, septic pulmonary emboli and infarction, lung abscess, and empyema.[2,3] These are particularly prevalent in those with high cardiac risk factors, such as prosthetic valves, recent operations, or coexisting illnesses such as intravenous (IV) drug usage, cancer, steroid use, and uncontrolled diabetes.[2,4]

Staphylococcus aureus, viridans group streptococci, and enterococci are the most common causes of IE, which usually affects the left side of the heart.[3] *S. aureus* is the most common causal bacterium in right-sided IE, which often occurs in persons who inject drugs.[3] While viridans group streptococci have a low virulence and are usually found in damaged and prosthetic valves, *S. aureus* has a high virulence and frequently infects normal/native valves.[4] An emerging bacterium of the sanguinis group of commensals, *Streptococcus gordonii*, typically inhabits the human skin, mouth, upper respiratory tract, and gastrointestinal system.[4,5] Certain components of *S. gordonii*'s cell wall function as virulence and attachment factors, causing vegetations and biofilms to develop.[4-6] Due to its prevalence in the oral cavity, it is most commonly linked to periodontal disease and dental caries.[1,5] Yet, it may also cause systemic infection, which can result in a number of illnesses, including hepatic abscesses, endocarditis, spondylitis, and empyema.[1,4,7]

Case Presentation

A 47-year-old man with chills, fever, and increased frequency of urination over the previous four days was brought into the emergency room. He denied having a cough, shortness of breath, palpitations, arthralgia, skin lesions, dysuria, or urethral discharge. Prior history included IE requiring tricuspid valve replacement and mitral valve annuloplasty, treated Hepatitis C virus infection, asthma, chronic obstructive pulmonary disease, and hypertension. Social history was notable for daily IV heroin use. He endorsed occasionally lubricating the needles with saliva before heroin injection and licking the injection site following injection.

Vital signs were as follows: temperature 39ºC, blood pressure 158/66 mmHg, HR 70 bpm, RR 17 breaths per minute, and SpO2 98% on room air. Upon physical examination, a middle-aged man appeared acutely unwell without scleral icterus or conjunctival pallor. He had fair dentition on oral examination without visible oral lesions. The cardiovascular exam was unremarkable, without a murmur. Chest exam revealed clear breath sounds bilaterally. He had tender indurated right forearm phlebitis with visible needle marks at the sites of prior IV drug injection.

Laboratory investigation revealed leukocytosis of 10,230/mcL (4,800-10,800/mcL), normocytic anemia with Hgb 10.7 g/dL (14.0-18.0 g/dL), thrombocytopenia 100,000/mcL (150,000-450,000/mcL), serum lactate of 1.6 mmol/L (0.6-1.4 mmol/L), procalcitonin of 0.31 ng/mL (0.02-0.08 ng/mL), C-reactive protein of 89.7 mg/L (0.0-5.0 mg/L), and erythrocyte sedimentation rate of 32 mm/hr (0-15 mm/hr). HIV 1,2 Ag/Ab were non-reactive, and HIV-1 viral load was not detected. Urine toxicology was positive for opiates/methadone, while urinalysis was within normal limits.

Electrocardiogram showed normal sinus rhythm with non-specific T wave abnormalities in the anterolateral leads. A chest radiograph showed evidence of tricuspid valve replacement and blunting of bilateral costophrenic angles, with no focal consolidation. Bilateral upper extremity duplex ultrasound was negative for deep vein thrombosis.

After obtaining the first two sets of blood cultures, the patient was started on empirical vancomycin and piperacillin/tazobactam. A transthoracic echocardiogram showed normal ejection fraction but limited visualization of the heart valves. Forty-eight hours after starting antibiotics, two other sets of blood cultures were obtained. All four sets of blood cultures drawn grew *S. gordonii*, and based on sensitivities, antibiotics were switched to IV ceftriaxone and gentamicin. He underwent a transesophageal echocardiogram, which confirmed a bioprosthetic tricuspid valve with vegetation on the right atrial side measuring 1 cm x 0.4 cm. A chest CT scan was negative for any evidence of septic emboli or microinfarcts. The patient was discharged on six weeks of IV ceftriaxone and two weeks of gentamicin.

Follow Up

The patient completed six weeks of antibiotics and was also appropriately referred for addiction care. Discharge follow-ups were also scheduled with cardiology, primary care, and infectious disease. Twelve months after the completion of therapy, a wellness check-up phone call was conducted with the patient. The patient endorsed that he had not used any recreational drugs since his discharge from the hospital.

Discussion

IE is a rare systemic illness that affects the endocardial surfaces of natural and prosthetic heart valves, the mural endocardium, or septal defects. The clinical presentation may be insidious and develop acutely or subacutely.[1,2] The majority of patients have fever, chills, and night sweats for days to weeks, and around 25% have a new murmur or embolic phenomena.[2,3] Myocardial abscesses, valve insufficiency, and congestive heart failure are among the intracardiac sequelae.[2-4] IE is usually lethal without treatment. It has a mortality rate of up to 30%, even with optimal treatment.[1,2] Cardiovascular risk factors such as congenital, damaged, or prosthetic valves/devices, as well as comorbidities such as old age, malignancies, poor dentition, poorly controlled diabetes, chronic renal disease, steroid usage, and IV drug use, are examples of risk factors.[2]

Staphylococci, streptococci, and enterococci species account for more than 80% of all instances of IE worldwide.[5] While *S. aureus* is currently the most common cause of endocarditis, accounting for 26% of all cases,[2] there is a growing prevalence of streptococci-related infections due to an aging population and the increased use of prosthetic valves.[2,3] Poor dentition is a significant risk factor for streptococcal IE.[2] Our patient, in this case report, had a history of endocarditis with tricuspid valve replacement, putting him at high risk for additional episodes.

One-tenth of all cases of IE involve the right heart, with a substantial percentage occurring among persons who inject drugs.[3] Bacteria enter the systemic circulation by direct inoculation and common IV drug practices such as lubricating needle tips with saliva before use and licking the puncture wound site following injection. IE in persons who inject drugs typically affects the tricuspid valve.[2,3,8] This was in keeping with our patient's presentation because he was known to lick the needles or injection site, increasing the potential of systemic transmission.

Gram-positive *S. gordonii* is a member of the sanguinis group of bacteria known to colonize the skin, oral mucosa, and gastrointestinal tract.[4,5] They alkalinize and produce the protective biofilm in the oral cavity.[4] They are alpha-hemolytic, catalase-negative cocci in pairs and chains.[5] A key component of S. gordonii's pathogenicity is its cell wall. Cell wall proteins expressed by *S. gordonii* include Hs antigen (Hsa), collagen-binding domain protein (CbdA), gordonii surface protein B (GspB), and streptococcal surface protein (Ssp) A and B.[4,6] These cell surface proteins—serine-rich repeat adhesins, lipoproteins, peptidoglycans, and lipoteichoic acids—allow *S. gordonii* to bind platelets and erythrocytes when it enters the bloodstream.[4,6-7,9-11] This bacterium-platelet complex is more likely to adhere to the fibronectin-rich extracellular matrix of heart valves, which can result in the growth of vegetations and biofilm.[4,6,9] Moreover, this results in the production of inflammatory cells such as macrophages and cytokines like interleukin (IL)-6, IL-8, and NF-κB, which can cause a significant systemic inflammatory response.[4,6-7,9-11]

The modified Duke's criteria can help diagnose endocarditis, and our patient met both of the major criteria of positive blood cultures and vegetation on echocardiogram.[1,12,13] A tailored, long-term antibiotic regimen based on sensitivities and early pathogen identification is critical to treating IE.[12,13]

Conclusion

IE is a rare but life-threatening disease of the heart valves and endocardium. Practices like licking needles and injection sites can potentially cause oral bacteria to spread into the bloodstream, leading to systemic infections in patients with predisposing factors. This case highlights that *S. gordonii*, though uncommon, should be considered a potential cause of IE, especially in persons who inject drugs.

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Consent: The authors contacted the patient over the phone on Friday, January 12, 2024, at 10:45 a.m. The patient was informed about the case report/manuscript and reassured that no patient identifiers were listed in the manuscript. The patient wholeheartedly gave consent for the manuscript to be published. The authors gave the patient the option to receive a copy of the manuscript once it was published, to which he agreed.