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Case Report

Tricuspid valve gonococcal endocarditis: fourth case report

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ABSTRACT

Disseminated gonococcal infection (DGI) occurs in 1–3% of all gonococcal infections; endocarditis is a complication in 1–2% of patients with DGI. We present the fourth reported case of gonococcal tricuspid valve endocarditis, this one occurring in a 53-year-old male with a 2-month history of shortness of breath.

1. Introduction

Disseminated gonococcal infection (DGI) occurs in 1–3% of all gonococcal infections, and endocarditis is a complication in 1–2% of patients with DGI.[1–4] The number of reported cases has decreased with the advent of antibiotics.[1,5,6] There are only two case reports of tricuspid valve involvement occurring in the post-antibiotic era.[2,3] In both reports, the patients were young and had a past history of gonococcal infection.[2,3] We present herein a case of gonococcal tricuspid valve endocarditis in a 53-year-old male with no history of known gonococcal infection, heart disease, or valve replacement.

2. Case report

A 53-year-old male presented with a 2-month history of progressive shortness of breath, which was initially noted on exertion and then present at rest. He also complained of generalized weakness, subjective fevers, chills, and weight loss for several weeks. A review of systems was significant for night sweats, lower extremity edema, chronic cough, and abdominal discomfort for 2–3 months. He denied paroxysmal nocturnal dyspnea and orthopnea. His past medical history was significant for alcoholism and remote intravenous drug abuse. He initially denied any sexually transmitted diseases or high risk sexual behavior. However, during a primary care visit 6 months later, the patient acknowledged unprotected heterosexual encounters with multiple partners.

Physical examination revealed an acutely ill appearing male with an oral temperature of 37.8 °C, a blood pressure of 135/73 mmHg, a heart rate of 115 per min, a respiratory rate of 20 per min, and an oxygen saturation of 98% on room air. A skin examination did not reveal any rashes or abnormalities. Cardiac examination revealed a non-radiating, soft 2/6 systolic murmur in the left parasternal region, an undisplaced point of maximal impulse, and no jugular venous distention. He had hepatomegaly and bilateral pitting edema up to the knees. The patient did not have findings of tenosynovitis or septic arthritis. Laboratory data were significant for microcytic anemia with a hemoglobin level of 7.6 g/dl and a leukocyte count of 20.4×10^9 cells/l, with 74% neutrophils, 6% bands, and 12% lymphocytes in the differential. A toxicology screen was negative and a 12-lead electrocardiogram was normal.

The admitting diagnosis was congestive heart failure (CHF) secondary to anemia. The patient received 2 units of packed red blood cells and empiric antibiotics (vancomycin and piperacillin/tazobactam). The latter were started because the patient was febrile and acutely ill, even though there was no obvious source of infection. A transthoracic echocardiogram showed a large tricuspid valve vegetation with moderate regurgitation. On the third day of admission, three different sets of blood cultures drawn in a span of

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Table 1
Summary of reported tricuspid valve gonococcal endocarditis in the post-antibiotic era

Author [Ref.]	Age (years)/sex	Previous gonococcal infection	Clinical presentation	Antibiotics	Complications	Outcome
Fernandez et al. [6]	35/M	Treated for urethritis	Cough, malaise, arthralgia, CHF	Vancomycin, penicillin (6 weeks)	None	Alive
Jackman and Glamann [2]	22/M	Treated for urethritis	Chest pain, chills	Penicillin (4 weeks)	None	Alive
John et al. [4]	18/M	No	Fever, abdominal pain	Penicillin (6 weeks)	None	Alive
Current case	53/M	No	Fever, chills, weight loss, shortness of breath	Ceftriaxone (6 weeks)	None	Alive

M, male; CHF, congestive heart failure.

4 h on the day of admission were reported positive for Neisseria with DNA probe confirmation. The bacteria in all the cultures were sensitive to ceftriaxone (minimum inhibitory concentration (MIC) dilution 0.04 μg/ml) and resistant to ampicillin/penicillin by β-lactamase screening. No ciprofloxacin sensitivities were conducted. The urethral and throat swabs cultured in Thayer–Martin medium were negative for gonorrhea; however, these samples were collected after the patient had already received empiric antibiotics. No urine PCR was obtained.

The patient improved on intravenous ceftriaxone. Surveillance blood cultures drawn 12, 46, and 84 days after admission (the latter set drawn 6 weeks after completion of antibiotics) were negative. A second echocardiogram done 6 weeks later revealed no increase in the size of the tricuspid vegetation or worsening of regurgitation. The patient has been asymptomatic for the last 6 months.

3. Discussion

Gonorrhea remains a major bacterial pathogen with high disease incidence in developing countries and an increasing rate of incidence in developed countries.[7] One of the reasons for a steady increase in the incidence of gonorrhea worldwide is because of the ability of gonococcus to develop antibiotic resistance.[7] In the USA, gonorrhea remains the second most commonly reported communicable disease with over 300 000 cases reported annually, and there are many cases that are not reported.[1,8] The highest reported rates of gonococcal infection are seen among adolescents, young adults, and among the population of southeastern USA.[8] After dissemination of penicillinase-producing and fluoroquinolone-resistant *Neisseria gonorrhoeae*, the recommendations in the USA have recently been modified to exclude quinolones as a standard treatment for gonorrhea.[7] This resistance pattern is alarming and suggests that the incidence of the infection may continue to rise in the future if strong preventive measures are not implemented.

In the pre-antibiotic era, *N. gonorrhoeae* was the third leading cause of infective endocarditis, accounting for 11–26% of all cases. It was reported to have very high mortality rates.[2,3] Even with the advent of antibiotics the mortality rates are high, around 20%.[2] Patients with gonococcal endocarditis (GE) usually present with non-specific symptoms, such as myalgias, arthralgias, fever, chills, a maculopapular rash, and back pain. However, most patients have prominent cardiac abnormalities, such as CHF and/or a new murmur. The aortic valve is involved in 50% of cases, followed by the mitral valve in 30% of cases.[6] Historically diagnosing GE has been challenging, because Neisseria is difficult to grow even with appropriate culture media. On average, more than six blood cultures are required.[10] With the advent of nucleic acid amplification techniques, the detection of bacteremia has become easier. GE is an aggressive infection that may cause valve destruction in hours to days, even if the patient has been treated with appropriate antibiotics.[1,2,9] Fifty percent of patients require valve replacement.[1]

GE differs from other causes of bacterial endocarditis in some respects. The duration from the onset of symptoms to diagnosis is usually longer for GE than for other pathogens like *Staphylococcus aureus* and pneumococcal endocarditis, but shorter and more aggressive than viridans Streptococcus, enterococcal, and other Gram-negative endocarditis.[5]

Our case illustrates the fact that patients with GE present with non-specific symptoms (see Table 1). Our patient came with fever, weakness, night sweats, and weight loss. Although GE is rare, it should be suspected in patients with risk factors for sexually transmitted diseases and arthritis. Lack of prior urethritis does not rule out the diagnosis. Unlike the common presentation of aortic GE, which requires valve replacement, tricuspid GE is cured by medical therapy.

Conflict of interest: No conflict of interest to declare.

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