Gonococcal Endocarditis

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Bacterial endocarditis secondary to infection caused by *Neisseria gonorrhoeae* was the leading cause of infectious endocarditis prior to the antibiotic era, accounting for anywhere from 4% to 26% of all cases.[1–5] Since the onset of the antibiotic era, however, the incidence of gonococcal endocarditis has declined. Between 1942 and 1985, fewer than 35 cases were reported in the available worldwide literature. In two large modern series dealing with endocarditis, no cases involved *Neisseria gonorrhoeae*.[6,7] It should be noted that the worldwide incidence of other gonococcal infections is increasing markedly, with 70 million cases yearly.[5]

Disseminated gonococcal infection follows a local infection approximately 1% to 3% of the time.[5] It is interesting that of the approximately 1 million cases of disseminated gonococcal infections, so few present as endocarditis. This report describes a case of gonococcal endocarditis with aortic valve involvement in a 25-year-old man with no previously known cardiac abnormality. In spite of appropriate intravenous antibiotic therapy and supportive care, the patient deteriorated rapidly and died before valve replacement could be performed.

CASE REPORT

A 25-year-old man came to the Good Samaritan Family Practice Center with generalized muscle aches, headaches, decreased appetite, dizziness, and mild cough. His only significant medical problem was a seizure disorder. He was afebrile and his blood pressure was 108/80 mmHg. He appeared tired, but findings on physical examination were unremarkable.

The patient was thought to have a viral syndrome, and he was seen again a week later. At that time his clinical condition appeared to be worse. He complained of some vomiting and loose watery stools, and had a temperature of 37.6°C. There was no recent weight loss, but a 10-lb loss over 6 months was noted. He denied smoking, alcohol use, intravenous drug abuse, or homosexual partners. He reported having contracted gonorrhea by a female partner in the remote past, which had been treated appropriately. Further examination showed that his mucous membranes were dry and there was a slightly prominent liver edge. Findings on rectal examination were unremarkable. Tests for fecal leukocytes and stool guaiac were negative. There was no inguinal lymphadenopathy or urethral discharge. Examination of the heart and lungs was normal. His leukocyte count revealed 166×10^9/L (16.6×10^3/mL) with 0.67 neutrophils and 0.18 band cells. Hemoglobin and hematocrit were 123 g/L (12.3 g/dL) and 0.36, respectively. Chemistry profile revealed an abnormally low albumin of 27 g/L (2.7 g/dL) and the urinalysis revealed trace protein, 5 to 10 white blood cells, and 0 to 1 red blood cells per high power field.

Eight days later, despite treatment with fluid hydration and antiemetics, the patient had not improved. His temperature was 38.4°C, blood pressure was 140/46 mmHg, and a new grade 2/6 systolic ejection murmur with a loud S_4 gallop was detected. The patient was admitted with a presumptive diagnosis of bacterial endocarditis and mild dehydration. At admission, a chest x-ray examination revealed mild cardiomegaly and slight congestion of the pulmonary vasculature. A hepatitis screening profile was negative. An electrocardiogram revealed sinus tachycardia. A VDRL test was nonreactive, and purified protein derivative was also negative. Antistreptolysin O titer was less than 200 todd units, and teichoic acid antibody was negative. Arterial blood gases were normal.

The patient was rehydrated intravenously. Blood cultures were obtained and antibiotics were started (nafcillin, 1 g intravenously every 4 hours, and gentamicin, 60 mg intravenously every 8 hours following a loading dose of 120 mg). By the second day of hospitalization, with blood cultures initially negative, clindamycin, 600 mg every 6 hours, was added to the regimen. At approximately day 5 of the hospitalization, the blood cultures revealed β-lactamase-negative *Neisseria gonorrhoeae*. The previously mentioned antibiotic regimen was changed, and intravenous aqueous penicillin G, 5 million units every 6 hours, was begun.

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Consultation with a cardiologist was obtained. A physical examination then revealed a change in the quality of murmur. There was now a grade 4/6 systolic murmur consistent with aortic stenosis, a grade 3/6 diastolic murmur consistent with aortic insufficiency, and a grade 1–2/6 diastolic murmur consistent with functional mitral stenosis (a so-called Austin Flint murmur). Echocardiogram revealed a vegetative process at the aortic valve and thickening of the aortic valve leaflets. A functionally bicuspid aortic valve and probable abscess in the left coronary sinus of Valsalva were also noted. The mitral valve revealed minimal prolapse and some thickening. Left ventricular function was consistent with an overload pattern.

Computerized tomography of the chest did not give additional information. Cardiac catheterization was performed, which confirmed the diagnosis of abscess or aneurysm of the left coronary sinus of Valsalva. This examination also revealed wide-open aortic insufficiency with a high wedge pressure of 40 mmHg, suggesting pulmonary hypertension.

The patient remained on an intravenous penicillin regimen at a total dose of 20 million units daily. Because of increasing congestive heart failure, he also received intravenous furosemide, 25 mg of captopril orally every 6 hours, 25 mg of apresoline orally every 6 hours, and 0.25 mg of digoxin orally daily. Surgical consultation was obtained for aortic valve replacement.

Before surgery the patient had an unexpected cardiac arrest with electromechanical dissociation. Resuscitation was attempted unsuccessfully. At autopsy, pulmonary edema, cardiomegaly (cardiac weight 600 g), and acute infarction of the left ventricular papillary muscles were evident. The vegetative growth on the damaged left coronary cusp was actually overlying the ostium. The growth could have functionally blocked the blood circulation into the left coronary artery. No congenital anomalies of the heart or great vessels were identified. The aortic valve was damaged to the extent that diagnosis of bicuspid valve could not be made.

DISCUSSION

This case demonstrates several aspects of disseminated gonococcal infections. The patient presented with a protracted illness, yet once hospitalized, the infection became fulminant. Although gonococcal endocarditis in the antibiotic era is rare (2% of disseminated infections), the potential for the gonococcus to attack and destroy otherwise normal heart valves still exists.[4,8,9] This case also illustrates the ability of the gonococcus to destroy a heart valve in hours to days in spite of appropriate antibiotic therapy.[5] Death from gonococcal endocarditis prior to the antibiotic era was usually due to congestive heart failure, cerebral embolism, or uremia.[10] The death rate is twice as high in patients developing aortic insufficiency.[11] In fact, acute aortic insufficiency is now the most common cause of death in patients with infectious endocarditis.[12,13] The aortic valve is the most commonly affected in gonococcal endocarditis, followed by the mitral valve.[14]

Another factor complicating gonococcal endocarditis is that blood cultures are sometimes negative in spite of fulminant valve destruction.[14] Identification may sometimes be made only from cultures taken from the destroyed valve itself.[14] The patient presented here had positive blood cultures relatively early in the course and proper antibiotic therapy. His clinical condition, however, deteriorated so rapidly that potentially life-saving surgery could not be accomplished.

In summary, the case presented illustrates one aspect of the life-threatening nature of disseminated gonococcal infection. Although the incidence of gonococcal endocarditis is rare, the increasing incidence of local gonococcal infections and the aggressiveness the gonococcus demonstrates in the destruction of heart valves would suggest caution in the treatment of any gonococcal infection.[15–18] Accordingly, disseminated gonococcal infections should be treated aggressively. Valve replacement in cases of gonococcal endocarditis should not be delayed, especially when acute aortic insufficiency develops.[8,14]

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