Treatment of Acute Bacterial Endocarditis by Valve Excision and Replacement

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The advent of a wide spectrum of bactericidal antibiotic agents has enabled physicians to treat many cases of bacterial endocarditis with a high likelihood of success. There remain, however, a significant number of patients with endocarditis in whom the infection is more resistant to antimicrobial therapy, valve destruction more rapid, and a satisfactory response to medical therapy sufficiently infrequent to warrant consideration of a new therapeutic approach. The purpose of this report is to describe a patient with bacterial endocarditis involving the aortic valve who failed to respond to antibiotics alone, but who has been cured by valve excision and replacement.

Case Report

The patient is a 45-year-old bricklayer. On June 2, 1963, he entered a hospital with a history of suprapubic pain and burning on urination. These symptoms had not responded to prostatic massage, antibiotics, or repeated soundings. At the time of admission his blood pressure was 124/70, temperature 98 F., pulse 86, and respiratory rate 18. The physical examination including complete cardiac evaluation was within normal limits except for an enlarged prostate gland. Laboratory studies revealed a hemoglobin of 15.6 Gm. per cent and a normal chest x-ray. On June 3 a transurethral prostatectomy was performed.

On June 15, 1963, the patient re-entered the hospital with a 36-hour history of shaking chills and fever. His temperature was 106 F., blood pressure 100/60, pulse 100, and respiratory rate 22. The remainder of the physical examination was normal. The hemoglobin was 13.0 Gm. per cent and the white blood cell count was 14,400, with 85 per cent polymorphonuclears. One blood culture grew out Proteus. He was treated with Kanamycin, 1.0 Gm. a day for 12 days. After 2 afebrile days antibiotics were discontinued and he was discharged from the hospital.

On July 11, 1963, chills and fever recurred. He saw his physician who found no abnormalities other than fever. On July 13 examination revealed a loud harsh diastolic murmur at the aortic area, petechiae on the palate, and a splinter hemorrhage on the ring finger. The pulse was 130, and the blood pressure was 120/60. Laboratory studies showed a hemoglobin of 10.8 Gm. per cent and a white blood cell count of 7200 with 93 per cent polymorphonuclears. Chest x-ray revealed that the transverse diameter of the heart had enlarged by 2 cm. over previous examinations. Blood cultures on several occasions were positive for Klebsiella, sensitive to Colymycin, Polymixin B, and Kanamycin. He was treated with Kanamycin, 2 Gm. a day, and Colymycin, 300 mg. a day. On July 30 a repeat blood culture was positive for Klebsiella and despite continued antibiotic therapy his temperature rose to 38 C. or over nearly every day. In addition he continued to lose weight, his anemia progressed and the aortic diastolic murmur increased in intensity. On August 2 Kanamycin therapy was discontinued. On August 7 his temperature rose to 103 F. and all antibiotics were discontinued.

On August 10, 1963, the patient was transferred to Duke University Medical Center. Physical examination revealed a blood pressure of 110/0, pulse 110, respirations 20, and oral temperature 37.5 C. He was poorly nourished and chronically ill. His weight was 125 pounds, a loss of 20 pounds from his usual weight. Examination of the eyes was within normal limits. The lungs were clear. The heart was enlarged; the point of maximal impulse was 11.5 cm. to the left of the midsternal line in the sixth intercostal space. P₂ was greater than A₂. There were a grade-II ejection murmur over the aortic area, which radiated to the neck, and a grade-III diastolic decrescendo murmur at the aortic area, which radiated to the apex. There was a protodiastolic gallop. The liver and spleen were both felt below the costal margins. The remainder of the physical examination was normal.

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Laboratory data revealed a hemoglobin of 8.6 Gm. per cent, hematocrit level of 26 per cent, and white blood cell count of 10,660 with 78 per cent polymorphonuclears. Six blood cultures were negative. Chest x-ray showed an enlarged heart. The electrocardiogram indicated digitalis effect.

Because of persistent fever and signs of progressing aortic insufficiency, it was concluded that the patient's bacterial endocarditis was still active and had failed to respond to combined therapy with Colymycin and Kanamycin. Following admission to Duke Hospital he continued to lose weight, developed edema, and progressive anemia and had daily fever. It was elected to treat the patient for 3 weeks before removing the aortic valve and replacing it with a Starr prosthesis. Three days after admission he was placed on Polymyxin B, 50 mg., intramuscularly every 6 hours. He was digitalized and given a total of seven units of packed red cells with a rise in hematocrit level to 38 per cent. Four days prior to surgery, Kanamycin, 0.5 Gm. intramuscularly, every 12 hours was added to the antibiotic regimen.

On September 5, 1963, the patient was taken to the operating room for replacement of the aortic valve by means of extracorporeal circulation and hypothermia. With the patient on complete bypass the aorta was opened. There were large, soft vegetations measuring 3 to 4 mm. in diameter on both the left and right coronary cusps with perforations of these cusps. The noncoronary cusp was essentially normal. The annulus of the aortic valve did not appear involved; however, the left sinus of Valsalva was dilated. The aortic valve was excised totally and replaced with a no.-9 Starr valve by interrupted sutures of 3-0 Teflon impregnated Dacron (fig. 1).

The patient became afebrile on the day after surgery and had only one febrile day during the remainder of his hospital stay. A Gram-stain of the aortic valve revealed numerous gram-negative rods. Culture of the valve grew out Klebsiella, type 19. The organism was sensitive to Kanamycin in a dilution of 25 μg. per cent and to Polymyxin B in a dilution of 12 μg. per cent. The patient's serum was bactericidal in a 1:2 dilution for the organism grown from his valve. Kanamycin, 1 Gm. a day, and Polymyxin, 200 mg. a day, were continued for 26 days postoperatively. On the twenty-seventh postoperative day all antibiotics were discontinued and during the following week he remained afebrile. The heart returned to normal size and the patient gained weight. There were no diastolic murmurs and the blood pressure was 120/70 mm. Hg. Audiograms at the time of discharge revealed a severe hearing loss in the upper frequency range.

The patient has been followed for 15 months since surgery. A faint aortic diastolic murmur developed 2 months postoperatively. He was admitted to the hospital at that time for 3 days of observation, and was found to be afibrile with a normal hemoglobin, white blood cell count, and differential. Five blood cultures were negative. The diastolic murmur suggested a small leak around the valve margin; however, there were no peripheral signs of aortic insufficiency, the heart was normal in size, and there was no evidence of recurrent infection. The patient continues to feel well and has returned to full employment at his job as bricklayer.

Figure 1
Graph of the clinical course (June 1963 through March 1964). Included are the temperature curve, blood cultures, antibiotic data, body weight, and note of major events.
Discussion

Management of patients with bacterial endocarditis presents at least two outstanding therapeutic problems. The first is to eradicate the infection and the second is appropriate treatment of cardiac failure and structural valve damage secondary to the infection.1 In recent years, early diagnosis, potent bactericidal agents, and prolonged courses of therapy have reduced greatly the mortality from bacterial endocarditis.2 At the present time, congestive heart failure is the commonest cause of death in such patients,3 and the mortality of those who develop aortic insufficiency as a result of endocarditis may exceed 50 per cent.4 These considerations highlight the need for a vigorous and effective therapeutic approach in patients with perforation of the aortic valve and congestive failure. The problem is even more urgent when the infection resists standard antimicrobial therapy and when the indicated antibiotics have serious toxic side effects.

The patient described in this report had acute bacterial endocarditis that resulted in perforation of the aortic valve. The diagnosis was established by a history of chills and fever, physical findings of a new cardiac murmur and positive blood cultures. Despite approximately 3 weeks of intensive therapy with Kanamycin and Colymycin, and a subsequent 3-week course of Polymyxin B, the patient continued to have fever, progressive anemia, and signs of increasing cardiac decompensation. Although it is possible that the infection might have responded to continued antibiotic therapy alone, two considerations led us to replace the valve. First, and most important, it was clear that the patient had not compensated for his aortic insufficiency and uncontrollable cardiac failure was the major factor limiting his recovery. Second, there was no indication that the infection had responded despite 6 weeks of therapy with bactericidal antibiotics. Replacement of the aortic valve with a Starr prosthesis was carried out without serious difficulty and the patient has recovered without evidence of further infection or hemodynamically significant aortic insufficiency. To our knowledge this is the first report of successful surgical treatment of active bacterial endocarditis involving the aortic valve.

In 1940, Touroff and Vesell5 reported a patient with patent ductus arteriosus and Streptococcus viridans endarteritis, in whom ligation of the ductus was followed by recovery from infection. Subsequent reports6,7 indicate that active endarteritis is not considered a contraindication to surgery in patients with patent ductus. Another setting in which the surgical approach may be indicated is in patients with postoperative endocarditis in whom a suture forms the nidus of infection. Bahnson et al.8 and Teitel and Florman9 have noted that such patients demonstrate extraordinary resistance to antibiotic therapy and have reported several cures after removal of the infected suture. The above cases are not directly analogous to the situation presented by our patient, but they do establish the feasibility of surgical procedures, in the presence of active infection within the heart, when the procedure itself influences favorably the infection.

In retrospective analysis of our case, three factors appear worthy of note in regard to the successful outcome. First, at the time of surgery the infection did not appear to involve the aortic ring. Consequently, all or nearly all infected material could be removed by valve excision and the sutures used to attach the prosthesis were placed in uninfected tissue. Sheldon and Golden10 have recently emphasized the frequent occurrence of valve-venting abscesses in certain instances of acute bacterial endocarditis due to Pneumococcus and Staphylococcus. Such lesions are undoubtedly related to the highly invasive nature of the organism, and it seems probable that they would preclude successful surgical therapy. Second, our patient had no evidence of pre-existing myocardial disease. This is probably an important reason why the signs of heart failure cleared rapidly after surgery and his heart size returned to normal. Finally, an intensive program of bactericidal antibi-

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