Bacterial Endocarditis in a Patient with
Idiopathic Hypertrophic Subaortic Stenosis

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BACTERIAL ENDOCARDITIS generally has not been considered a threat to patients with isolated idiopathic hypertrophic subaortic stenosis (IHSS). Although there have been a very few reports of its occurrence in this disorder,1,2 these patients represent only a small fraction of those patients diagnosed as having this entity.3 A patient has recently been seen with bacterial endocarditis, who during subsequent hemodynamic and angiographic studies, was found to have IHSS. The rarity of this occurrence has prompted this report with a recommendation for prophylaxis for subacute bacterial endocarditis (SBE) in patients who have muscular subaortic stenosis.

Report of Case

G.D.M., a 29-year-old, white male, was admitted to the J. Hillis Miller Health Center of the University of Florida on September 10, 1964, because of malaise, chills, and fever of approximately 8 hours duration. Ten hours earlier he had had a broken tooth extracted by his dentist. He denied any other acute symptoms.

His past history revealed that the patient had been deferred from the army for heart trouble in 1958. Subsequent evaluations, including a cardiac catheterization, were believed to be compatible with the diagnosis of idiopathic myocardiopathy and he was digitalized. However, no stress tests or angiograms had been made.

Physical examination revealed a well-developed, well-nourished, white, male in mild acute distress. Blood pressure was 110/50 mm Hg, pulse, 120 per minute, respiration, 20 per minute, and oral temperature, 38.6 C. Pertinent findings included an extraction cavity in his left lower gum and pharyngeal inflammation. Cardiovascular examination revealed very brisk bifid peripheral arterial pulses. The point of maximal impulse was in the fifth left intercostal space 2 cm lateral to the midclavicular line with a 1+ left ventricular lift. The first and second heart sounds were prominent and a third heart sound was heard at the apex. A grade-III of VI holosystolic murmur was heard maximally between the lower left sternal border and the cardiac apex with radiation into the left axilla. No lymphadenopathy, clubbing, splinter hemorrhages, retinal changes, or splenomegaly were present.

Laboratory tests gave the following values: Hemoglobin, 14.1 g %; hematocrit, 44%; white blood cell count, 7,800 with 58 polymorphonuclear leukocytes, 8 bands, 31 lymphocytes, and 3 monocytes. Corrected erythrocyte sedimentation rate was 9 mm in 1 hour. Urine was within normal limits and the antistreptolysin-O titer was 100 Todd units. One of eight blood cultures grew out alpha hemolytic streptococci; however, the attending physician considered this to be a contaminant. Chest x-rays (fig. 1) and electrocardio-

Figure 1
Postero-anterior view of the chest demonstrating concentric hypertrophy of the left ventricle and slight enlargement of the left atrium.

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gram demonstrated no changes over previous examinations which also had revealed left ventricular hypertrophy and slight left atrial enlargement. The patient became afebrile despite absence of any specific therapy and was discharged. However, when seen, 3 days later, he was again febrile (38.5°C). The four blood cultures made at the time of discharge from the hospital were all positive for alpha hemolytic streptococci which were sensitive to penicillin.

Physical findings at this time, except for the healing of the left lower gum, had not changed appreciably. His condition responded rapidly and completely to penicillin therapy. Subsequent blood cultures were all negative and the patient had no further fever or other signs of relapse during follow-up examinations.

Three months following the episode of bacterial endocarditis, the patient was readmitted for another cardiac catheterization. Right heart pressures were entirely normal as was his cardiac index. Simultaneous left ventricular and right brachial artery pressures demonstrated a 30-mm gradient at rest (fig. 2). The pulse contours, response to isoproterenol and nitroglycerin, angiogram, and left ventricle-to-aortic root pullback were all characteristic of muscular subaortic stenosis.

Following this study, administration of digitalis was discontinued and the patient was discharged to be followed as an out-patient. Bacterial endocarditis prophylaxis is being used as necessary.

Discussion

This patient with all the hemodynamic and angiographic findings of IHSS developed acute bacterial endocarditis following extraction of a tooth. This rare complication occurring in a patient with IHSS has probably been seen on only three other occasions. As in this patient, one of the other three developed bacterial endocarditis following dental manipulation.

The small number of cases of IHSS known to be involved with bacterial endocarditis do not permit any conclusions to be drawn as to the site of the infection. In one of Boiteau and Allenstein's cases the mitral valve was thickened at autopsy and this area was felt to be damaged secondary to the endocarditis, although this could not be proven.

Whatever the locus of the bacterial infection on the endocardium, the significance of bacterial endocarditis in patients with IHSS cannot be underestimated. The possibility that any patient with IHSS may develop this entity is a real one. Patients with IHSS, therefore, should be added to the list of those in whom appropriate prophylaxis against SBE should be carried out.

Summary

A rare case of subacute bacterial endocarditis, due to a streptococcus viridans organism, is reported in a patient with idiopathic hypertrophic subaortic stenosis (IHSS). Subacute bacterial endocarditis prophylaxis is recommended for patients with IHSS.

References

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