Idiopathic Hypertrophic Subaortic Stenosis

Coexistence with Calcific Aortic Valvular Stenosis and Calcified Mitral Annulus in an Elderly Patient

To the Editor:

Our communication has the purpose of emphasizing the significance of the echocardiogram in a 79-year-old patient with idiopathic hypertrophic subaortic stenosis proven by catheterization, calcification of the mitral annulus, and aortic valvular stenosis. Idiopathic hypertrophic subaortic stenosis in the elderly is not a frequent occurrence; and when it is associated with other mild preexistent valvular disease (aortic stenosis) or with certain valvular changes related to the aging process, such as a calcified mitral annulus, idiopathic hypertrophic subaortic stenosis may represent a diagnostic challenge for the clinician.

On examination the dominant lesion may mask an associated milder one; for instance, in our patient the carotid pulse was brisk and bifid, and the systolic ejection murmur increasing with Valsalva’s maneuver supported the diagnosis of idiopathic hypertrophic subaortic stenosis but masked the aortic stenosis. The echocardiogram (Fig 1, left) showed an anterior systolic motion of the mitral valve and hypertrophy of the interventricular septum. Dense echoes of the aorta (Fig 1, right) probably represent deposits of calcium on the aortic leaflets. Also demonstrated was a dense echo posterior to the mitral valve but anterior to the posterior free wall of the ventricle, representing a calcified mitral annulus. The systolic anterior motion of the mitral valve was smaller than that typical of idiopathic hypertrophic subaortic stenosis, possibly because of the coexistent aortic valvular stenosis, as previously noted by Nanda et al. The diagnosis was confirmed by fluoroscopic examination and by catheterization, which showed an intracavitary left ventricular gradient of 95 mm Hg and a valvular systolic gradient of 25 mm Hg across the aortic valve.

We believe that the combined occurrence of idiopathic hypertrophic subaortic stenosis, aortic valvular stenosis, and calcification of the mitral annulus in the elderly may be more frequent than reported, and in-

Figure 1. Echocardiograms. Left, Anterior systolic motion of mitral valve (SAM) and hypertrophy of interventricular septum (S). RS, Right septum; LS, left septum; AML, anterior mitral leaflet; AC, annular calcification; and PH, posterior heart. Right, Dense echoes of aorta (AO) probably represent deposits of calcium on aortic leaflets. LA, Left atrium.
Intravascular Hemolysis with Pulmonic Stenosis

Occurrence after Correction of Subpulmonary Ventricular Septal Defect

To the Editor:

We have seen a case of severe intracardiac hemolysis with pulmonic stenosis following correction of subpulmonary ventricular septal defect. At the superior margin of the ventricular septal defect, a Dacron patch was sewn to the pulmonic valvular annulus. Valvulotomy was performed.

Immediately after the operation, the hemoglobinuria, hemosiderinuria, and progressive anemia persisted. The peripheral blood cells showed schistocytosis.

Right cardiac catheterization disclosed severe pulmonic stenosis, and the peak systolic pressure gradient between the pulmonary artery and right ventricle was 115 mm Hg. The patient underwent a second operation, and a Dacron patch was sewn into the incision to widen the area of pulmonic stenosis. The second operation brought cessation of intravascular hemolysis.

Severe pulmonic stenosis was produced in closure of a ventricular septal defect with a Dacron patch and became a cause to increase sheering stress. The sheering stress for erythrocytes increases as the systolic pressure gradient increases. When erythrocytes are subjected to a sheering stress exceeding the limit of the tensile stress of the erythrocytic membrane, the membrane ruptures, with the formation of schistocytes.

When mechanical hemolysis results from inadequate intracardiac correction, recatheterization and a second operation should be considered.

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Acute Gouty Arthritis Associated with the Use of Ethambutol

To the Editor:

Although the incidence and possible mechanism of hyperuricemia due to therapy with ethambutol have been discussed in the literature, clinical case reports of acute gouty arthritis associated with administration of ethambutol, to our knowledge, are nonexistent. We report three cases suggestive of ethambutol-induced gout.

CASE REPORTS

Case 1

A 47-year-old man was hospitalized in April of 1976, complaining of a warm, swollen, extremely tender right elbow of one day's duration. Six days earlier, the patient had had a tender, erythematous, swollen great and second toe of the right foot, which improved with heat and elevation of the foot. He denied trauma and recent ingestion of alcohol. There was no past medical or family history of gout or any arthritis. The only drugs taken by the patient were 800 mg (15 mg/kg) of ethambutol and 600 mg of rifampin daily, which had been started as therapy for active tuberculosis approximately one year prior to admission.

Remarkable findings from the physical examination were limited to a temperature of 38.8°C (101.8°F) and a severely painful, inflamed nonphaceous elbow. Pertinent laboratory data included a white blood cell count of 23,000/cu mm, a serum uric acid level of 8.0 mg/100 ml, a 24-hour urinary uric acid level of 0.37 gm/790 ml, and negative cultures and Gram's stain of the joint aspirate for bacteria or mycobacteria. Microscopic examination of the joint fluid revealed many urate crystals in the leukocytes.

Therapy with colchicine (0.6 mg orally every hour) was begun, and a good response was seen after nine doses.

Case 2

A 59-year-old woman was hospitalized in May 1974 with pneumonia. The patient had been maintained on therapy with