Perforated Aneurysm of the Anterior Mitral Valve*

A Doppler and Two-Dimensional Echocardiographic Report

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Antemortem diagnosis of a perforated MV aneurysm by Doppler echocardiography, as well as two-dimensional echocardiography (2-DE), has not been reported. Herein we present such a case. (Chest 1990; 97:753-54)

Formation of an aneurysm of the mitral valve (MV) is rare, and it develops usually during a course of infective endocarditis.1,2 Perforation of such an MV aneurysm bears a poor prognosis and usually requires surgery. Although 2-DE is an established technique for imaging, localizing, and providing prognostic information related to an aneurysm of the MV, perforation of the MV aneurysm heretofore has primarily been sought at surgery3,4 or autopsy.5 The recent advent of the Doppler technique in combination with 2-DE enables a search for unusual flow patterns across the MV aneurysm to detect the presence of perforation. This report presents the Doppler echocardiographic manifestations of a perforated MV aneurysm.

CASE REPORT

A 29-year-old man with a four-month history of exertional dyspnea was admitted seven days after a sudden worsening of shortness of breath. His blood pressure was 90/70 mm Hg, the heart rate was 90 beats per minute and regular, the respiratory rate was 20/min, and body temperature was 37°C (98.6°F). The heart was enlarged. There was a grade 3/6 pansystolic murmur at the apex, with transmission to the left axilla and the back. An ECG showed right axis deviation and findings consistent with left atrial enlargement and biventricular hypertrophy. The 2-DE demonstrated dilatation of the left atrium (LA) and the left ventricle. There was a remarkable systolic bulging of the MV into the LA, appearing as a 2.5-cm ring-like structure with central echolucency visible at apical four-chamber views (Fig 1). An echo dropout at the center of the ring was well illustrated during systole at parasternal long-axis view (Fig 2). The Doppler examination revealed moderate mitral regurgitation, and a separate turbulent jet was also detected inside the ring structure during systole at parasternal long axis (Fig 3). There was leukocytosis (11,400/cu mm). The blood cultures were negative, and the patient had remained afebrile. Nevertheless, a full course of antibiotic treatment was administered. Thereafter, cardiac catheterization and left ventriculography elucidated severe mitral regurgitation with a systolic round structure over the MV leaflet protruding into the LA. Surgery revealed a dilated mitral annulus and markedly elongated chordae tendineae, with perforation at the anterior leaflet. Histologic examination revealed chronic inflammation and fibrosis.

DISCUSSION

Two-dimensional echocardiographic features of an MV aneurysm present as an aneurysmal bulge of the MV leaflet toward the LA.6 An MV aneurysm should be differentiated from the rare forms of blood cyst of the papillary muscle7 and nonendothelialized cyst of the MV.8 Differential diagnosis for such a ring-like structure in the vicinity of the MV by the absence of flow pattern inside or across the ring by Doppler technique or color flow mapping, the nature of motion throughout the cardiac cycle, and the location of the lesion can aid in the exclusion of the latter two rarer entities. Our case had the typical aneurysmal appearance during systole, but during diastole the aneurysm disappeared. This was consistent with the surgical finding of marked elongation of the chordae. This was different from those reported by De Luca and Colonna9 or by Reid et al.2 In their cases, aneurysms were all secondary to aortic regurgitation, and the regurgitant jet rushed to the MV during diastole. This may be the reason why those aneurysms remained visualized throughout the cardiac cycle; however, Doppler echocardiographic findings of perforated MV aneurysm have not been reported. Herein we add a criterion for the diagnosis of perforation of an MV aneurysm, which is the presence of abnormal regurgitant flow detectable inside the ring by Doppler technique or the presence of an abnormal color

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FIGURE 1. Apical four-chamber view showing ring-like structure (arrow) with central echolucency in LA. LV, Left ventricle; RA, right atrium; and RV, right ventricle.

FIGURE 2. Parasternal long-axis view showing echo dropout area (arrow) at center of ring during systole. LV, Left ventricle; RV, right ventricle; and AO, aorta.
Systemic Embolization following Thrombolytic Therapy for Acute Myocardial Infarction*

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We describe a patient with acute inferior myocardial infarction who developed a "saddle" aortic embolus during streptokinase infusion. Three months previously, this patient had sustained an anterior infarction, and an apical aneurysm was found. This patient's embolus had most probably originated from a left ventricular mural thrombus that had been dislodged by streptokinase. As fibrinolytic treatment is gaining wide acceptance, physicians should be aware of this rare, but possible, complication.

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PTT = partial thromboplastin time; rtPA = recombinant tissue plasminogen activator

Left ventricular thrombus is an important complication of acute myocardial infarction, especially when it involves the anterior wall. A minority of such thrombi will cause systemic emboli in the year following the infarction. It has been suggested that thrombolysis may be beneficial in lysing such thrombi. We report an unusual case of systemic embolization that occurred during streptokinase infusion in a patient who had sustained an anterior myocardial infarction, complicated by an apical aneurysm, three months previously.

Case Report

A 65-year-old woman was admitted to the coronary care unit of Hadassah University Hospital, Mt. Scopus, for acute inferior myocardial infarction. Three months previously, she had been hospitalized in another hospital for a large anterior infarction, complicated by pulmonary edema. No thrombolytic therapy was given at that time. Her medical history was remarkable only for insulin-treated diabetes.

The findings from physical examination on admission to our unit were normal; the femoral, posterior tibial, and dorsalis pedis pulses were easily palpable bilaterally. The ECG showed evidence of an acute inferior infarction and an old anterior infarction. Five hours after the onset of chest pain, an intravenous infusion of streptokinase was initiated, according to our current protocol, which calls for the administration of 1.5 million units of the agent within the first hour and another 1.5 million during the next 24 hours. Following treatment the patient's chest pain soon subsided; the ECG and the enzyme curves were suggestive of successful reperfusion. Coagulation studies at this time showed a very low level of fibrinogen and a prolonged PTT.

Eleven hours after starting thrombolytic therapy, the patient experienced the abrupt onset of severe bilateral leg pain, from the groin down. Examination disclosed disappearance of both femoral pulses and evidence of rapidly progressing leg ischemia. The differential diagnosis that we considered included mainly the

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FIGURE 3. Parasternal long-axis view of MV area, illustrating presence of turbulent jet during systole inside the ring structure where sample volume is placed. LV; Left ventricle; AO, aorta; and LA, left atrium.

Flow pattern at the site stated previously. This would be further supported by demonstrating an interrupted segment across the ring.

An MV aneurysm frequently has been associated with infective endocarditis. The infective and subsequent healing processes may weaken a circumferential portion of valvular tissue, which yielding to the intraventricular pressure, may form a true aneurysm or perforate (or both). Thus, perforation of the aneurysm may result in worsening of mitral regurgitation and should be considered in patients with left-sided endocarditis who have sudden hemodynamic deterioration. Early preoperative recognition of a perforated MV aneurysm by Doppler echocardiography and 2-DE thus plays a vital role in the therapeutic management of such a patient.

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