THALLIUM DESCENDING ANTERIOR

Figure 1. (Top), left coronary arteriogram in right anterior oblique view following ergonovine administration. A subtotal proximal left anterior descending coronary artery narrowing is present (arrow) with poor distal filling. Bottom, following the intracoronary administration of 300 μg of nitroglycerin, the spasm regressed leaving a 50 percent tubular organic narrowing of the proximal left anterior descending coronary artery.

The thallium perfusion scan showed a defect in the septal region during exercise which normalized by four hours. A long, proximal 50 percent left anterior descending stenosis, a 40 percent proximal stenosis of the first diagonal branch, and a 40 percent distal circumflex stenosis were present at coronary arteriography. Ergonovine administration induced angina and a subtotal occlusion of the left anterior descending coronary artery at the site of the fixed stenosis, as illustrated in Figure 1. A simultaneous 12-lead ECG revealed pseudonormalization of previously negative T waves in lead aVL. All evidence of coronary artery spasm disappeared immediately after the administration of nitroglycerin.

The patient was treated with diltiazem, 90 mg by mouth three times a day and has had no further episodes of angina.

Discussion

The cause of rest angina in this patient was undoubtedly coronary artery spasm because his organic coronary stenoses were not severe and because ergonovine induced coronary spasm, electrocardiographic signs of ischemia, and his usual angina. Treatment with the calcium antagonist drug diltiazem eliminated both his rest and effort angina. The cause of effort angina could have been either his organic stenoses or coronary spasm.1

Premature coronary artery disease causing angina, myocardial infarction,4,5 or sudden death6 has been reported after Mantel radiotherapy in young cancer patients not otherwise predisposed to ischemic heart disease. Although radiation causes coronary artery lesions in experimental animals, a cause-and-effect relationship has not been proven in man. Patients dying with coronary artery disease following radiation exhibit a variety of histologic changes, including fibromuscular proliferation of the arterial wall7 and medial atrophy with intimal and adventitial fibrosis.8,9 Some of these abnormalities might theoretically favor the development of coronary artery spasm, but others, such as diffuse fibrosis, could limit excessive vasomotion. Coronary spasm almost always occurs in association with an organic stenosis,9 thus, if radiation-induced coronary lesions are not severe enough to cause symptoms by themselves, they may provide the trigger or substrate for coronary spasm. Marzilli et al10 have postulated that coronary spasm may induce or accelerate the development of organic coronary stenoses, and coronary spasm has been implicated in the pathogenesis of myocardial infarction. If either of these theories is true, they might be relevant to the pathophysiology of radiation-induced coronary artery disease.

In summary, this case demonstrates that coronary artery spasm may cause angina in association with mild diffuse organic stenoses following mediastinal irradiation.

References


Fatal Embolism in Mitral Valve Prolapse

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The postmortem demonstration of fatal cerebral embolism arising from a thrombus detected at the base of a prolapsing and myxomatous mitral valve is reported. The clinical demonstration of recurrent atrial fibrillation in this patient lends support to the suggested use of anticoagulant therapy in similar patients.

The association of cerebral ischemic events and mitral valve prolapse (MVP) is widely accepted. Most reports discuss transient neurologic deficits ascribed to microemboli or arrhythmia, while postmortem confirmation of embolization in MVP remains a rarity.

CASE REPORT

A 46-year-old woman was first admitted with acute congestive heart failure. Having always been in good health, she gave no history of heart disease or murmur, diabetes, or hypertension. She had enjoyed eight normal pregnancies. Examination revealed atrial fibrillation with a rapid ventricular rate and a faint and intermittent systolic murmur and click. Digoxin and furosemide therapy cleared the congestive failure promptly, but several days of quinidine administration was required before sinus rhythm was restored. During this interval, anticoagulation with warfarin (Coumadin) was carried out. M-mode echocardiography displayed holosystolic MVP (Fig 1). Left ventricular size and function and left atrial size were within normal limits. A gated cardiac scan was also normal. Because of her alarming and unexplained presentation in cardiac failure without evidence of significant mitral regurgitation, the patient was referred for possible cardiac catheterization. This was deferred because of her improved and stable clinical course. About six months after discharge, Coumadin and quinidine therapy were discontinued. There was no evidence of recurrent arrhythmia on subsequent examinations or ambulatory ECG monitoring.

Six months later the patient suffered a sudden, massive hemiparesis. Atrial fibrillation was again present, but all other findings were unchanged. CT scan confirmed the presence of cerebral infarction, but, despite intensive management, the patient's condition deteriorated and she died in four days. At autopsy, the heart weighed 450 g, and the left ventricle was slightly thickened to 2 cm. The left atrium was normal. The mitral valve was floppy and demonstrated prolapse (Fig 2). Diffuse, myxomatous degeneration of the valvular structures was seen microscopically. An intracardiac thrombus was detected microscopically, embedded in the muscular trabeculae at the base of the mitral valve. There was no coronary or systemic atherosclerosis. There was total embolic occlusion of the
Cerebral cortex, displaying cystic and hemorrhagic infarction. Right middle cerebral and pontine arteries (Fig 3), with extensive cystic and hemorrhagic infarctions of the cerebral cortex, basal ganglia, and pons (Fig 4).

**COMMENTS**

In the absence of any significant heart disease other than MVP, both clinically and at autopsy, we believe that atrial fibrillation, detected during the attacks of congestive heart failure and fatal embolization, was a contributory factor in both of these complications. These observations lend support to the suggestion that patients with MVP and recurrent or persistent atrial fibrillation be considered for anticoagulation therapy. Knowing that MVP is a major cause of mitral valve replacement emphasizes that not all patients with MVP enjoy its usually benign course. Awareness of the possible complications of MVP and their prevention or treatment is desirable.

**REFERENCES**