Transient Mitral Regurgitation: An Adjunctive Sign of Myocardial Ischemia During Dipyridamole-Thallium Imaging

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A patient developed transient exacerbation of a mitral insufficiency murmur and a reversible posterior wall perfusion defect during dipyridamole-thallium imaging. Coronary angiography showed significant stenoses of both the right and the circumflex coronary arteries that supply the posterior papillary muscle. Cardiac auscultation for transient mitral incompetence, a sign of reversible papillary muscle dysfunction, is a simple and practical adjunctive test for myocardial ischemia during dipyridamole-thallium imaging. It may confirm that an isolated reversible posterior wall myocardial perfusion defect is truly ischemic in nature as opposed to an artifact resulting from attenuation by the diaphragm.


Transient mitral regurgitation caused by papillary muscle dysfunction has been well documented during both spontaneous (1) and exercise-induced (2) myocardial ischemia. We describe a patient who developed reversible exacerbation of mitral regurgitation, orthopnea, and scintigraphic evidence of posterior wall ischemia during dipyridamole-thallium imaging.

CASE REPORT

A 66-yr-old woman was admitted for vascular surgery. Past history included one episode of pulmonary edema of unknown etiology treated in another institution and a 20-yr history of hypertension. Except for the one episode of pulmonary edema, she gave no history of myocardial infarction, angina, or congestive heart failure. An electrocardiogram showed left ventricular hypertrophy with strain. Dipyridamole-thallium imaging was ordered for pre-operative cardiac risk assessment. Before the infusion of 0.14 mg/kg/min of dipyridamole for 4 min she was lying comfortably supine, the heart rate was 80 min and regular, the blood pressure was 170/100 mmHg, the respiratory rate 14 min, the lungs were clear and the rest of the physical examination was unremarkable except for a grade 1/6 apical soft blowing murmur in the left lateral decubitus position compatible with mild mitral incompetence. Five minutes after the end of the infusion, as

the patient was being positioned under the camera, she developed increasing dyspnea with no chest pain, at which time the blood pressure was 160/100 mmHg, the heart rate 100 min, and regular and auscultation revealed a loud grade 3-4/6 holosystolic apical blowing murmur. As the dyspnea progressed to orthopnea, a bolus of 100 mg of aminophylline was administered intravenously. Two minutes later the patient could resume the supine position and the apical systolic murmur had regressed to grade 1/6. Planar scintigraphic images in three views showed a reversible perfusion defect of the posterior wall visible on the left anterior oblique 70-degree view only (Fig. 1). Coronary angiography revealed a complete obstruction of the proximal right coronary artery that opacified through contralateral collaterals, a 90% stenosis of the first diagonal branch of the left anterior descending, a 60% stenosis of the middle third of the left anterior descending, and an 80% stenosis of the distal circumflex between the third and fourth marginal branches. Left ventricular angiography showed akinesis of the anterolateral segment and normal contraction of the other segments. Unfortunately, the injection of dye induced a run of ventricular tachycardia with artifactual mitral regurgitation that interfered with the evaluation of the mitral valve.

DISCUSSION

Dipyridamole-thallium imaging is widely used for the detection of coronary artery disease and the evaluation of cardiac risk before noncardiac surgery in patients unable to complete a standard exercise test. Apart

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from the diagnostic inhomogeneous distribution of thallium, which may be attributed either to relative hypoperfusion or true regional ischemia, there are few adjunctive signs of myocardial ischemia. Changes in heart rate and blood pressure are obviously nonspecific. Chest pain is also nonspecific and does not appear to be related to either the extent or the severity of the coronary artery disease (3). ST segment depression is uncommon, appears to be relatively characteristic of a subgroup of coronary patients with good collateral vessels that promote the coronary steal effect, and is of no prognostic value (4). Both transient new onset (1) and reversible exacerbation (2) of mitral regurgitation have been described during myocardial ischemia, but not, to our knowledge, during dipyridamole-induced ischemia. It is not characteristic of any specific coronary involvement: the posterior papillary muscle is usually involved (as in our case) and receives its blood supply from both the right and the circumflex coronary arteries in 70% of cases, exclusively from the right in 20% and exclusively from the circumflex in 10%. Both the left anterior descending coronary artery and its diagonal branches supply the anterior papillary muscle which is less frequently involved (5).

Systematic auscultation for mitral regurgitation is impractical during exercise stress testing. However, during dipyridamole-thallium imaging, conditions are ideal for detection of transient papillary muscle dysfunction; the surroundings are quiet and the patient is supine. We believe that careful auscultation for transient mitral insufficiency or reversible exacerbation of mitral incompetence is a simple and specific adjunctive test for myocardial ischemia during dipyridamole-thallium imaging. Further studies are necessary to determine the frequency of its occurrence in patients with dipyridamole induced myocardial ischemia.

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