Thallium-201 Myocardial SPECT in Myocardial Bridging

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Myocardial bridging is an uncommon condition where a major coronary artery is bridged by a band of muscle and narrows during systole, particularly during rapid heart rates. We present a patient with typical angina and angiographically proven 60% systolic bridging of the left anterior descending artery distal to the first perforator. Postexercise SPECT ^{201}Tl scanning demonstrated a severe reduction of perfusion to the septum and a moderate reduction of perfusion to the anterior wall of the left ventricle. Redistribution images demonstrated good reversibility of the perfusion defects indicating reversible myocardial ischemia. This case provides additional supportive evidence that myocardial bridging may cause myocardial ischemia.

Key Words: myocardial bridging; thallium-201; single-photon emission computed tomography; myocardial ischemia

J Nucl Med 1995; 36:1031–1033

The major coronary arteries normally pass through the subepicardial layer of the heart. In myocardial bridging, however, a segment of a coronary artery is embedded in the myocardium for a variable length or bridged by a band of muscle (*1*). At coronary angiography the artery is seen to be narrowed in ventricular systole (*2*). Whether myocardial bridging causes myocardial ischemia and whether treatment is necessary is controversial (*3*). Few cases of ^{201}Tl scintigraphy have been reported in this condition none have dealt with SPECT imaging, so its role in the evaluation of myocardial bridging has not been established. We present a case of myocardial bridging causing reversible myocardial ischemia demonstrated by exercise SPECT ^{201}Tl scintigraphy.

CASE REPORT

A 40-yr-old man presented with typical angina of increasing frequency. A diagnosis of myocardial bridging was previously made according to coronary angiography. The patient had mild hypercholesterolemia (249 mg/100 ml). He was not hypertensive and did not smoke. The patient was treated medically with calcium channel blockers with symptomatic improvement. Physical examination was unremarkable. The electrocardiogram demonstrated sinus rhythm, left axis deviation, left ventricular hypertrophy on voltage criteria and small Q-waves in leads II, III and AVF. Echocardiography demonstrated normal cardiac chambers and normal left ventricular wall thickness with no significant hypertrophy of the left ventricle or systolic anterior motion of the mitral valve. The left ventricular wall motion was also normal. There was no evidence of mitral valve prolapse.

In view of the change in symptoms, an exercise ^{201}Tl study was performed to exclude myocardial ischemia as a cause of the patient's exertional chest pain. The patient, exercised to Stage 4 of the standard Bruce protocol for 11 min, had a peak heart rate of 167 bpm. Exercise was terminated due to chest pain similar to his previous episodes. No ischemic changes were detected on the stress electrocardiogram.

Images were obtained 10 min after injection of 90 MBq ^{201}Tl at peak exercise using a low-energy, multipurpose collimator. Data were collected from an elliptical orbit for 30 min on a single-headed gamma camera with a 180° arc (LAO to LPO). Reconstruction was performed with a Butterworth prefilter with a frequency cutoff of 0.10 and order of 8. Single pixel-thick slices of the short, horizontal and vertical long-axes were generated from the transaxial images. Thallium-201-chloride (20 MBq) was then reinjected 3 hr later and reinjection images were obtained after 10 min.

Postexercise SPECT ^{201}Tl images demonstrated severe perfusion reduction to the septum and moderate perfusion reduction to the anterior wall. Delayed imaging showed good reversibility of these defects, indicating reversible myocardial ischemia (Fig. 1). Subsequent coronary angiography demonstrated normal coronary arteries with no evidence of atherosclerosis and normal septal wall thickness. The left anterior descending artery, however, demonstrated 60% systolic narrowing distal to the first septal perforator, which is typical of myocardial bridging (Fig. 2). Further coronary angiography with ergonovine administration was not performed to evaluate coronary spasm.

DISCUSSION

Myocardial bridging is an uncommon condition noted on approximately 1%–12% of coronary angiograms, with the left anterior descending artery most commonly affected (*4,5*). At normal heart rates the majority of blood supply to the myocardium occurs during diastole with only 20% occurring during systole. At rapid heart rates, however, the systolic contribution becomes more dominant, and myocardial bridging may result in reduction of coronary blood flow to the myocardium. This is supported by studies with rapid atrial pacing in patients with myocardial bridging who

Received May 6, 1994; revision accepted Oct. 24, 1994.

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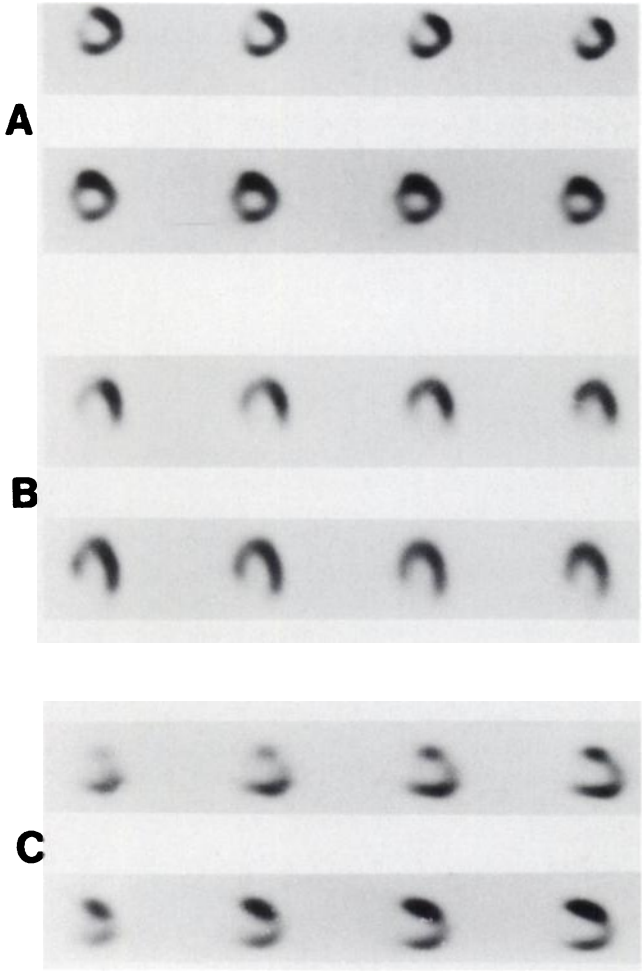

FIGURE 1. Corresponding SPECT sections of stress (upper) and 3-hr delayed (lower) images. (A) Short-axis. (B) Horizontal long-axis. (C) Vertical long-axis. Myocardial bridging of the LAD causes extensive reversible anteroseptal ischaemia.

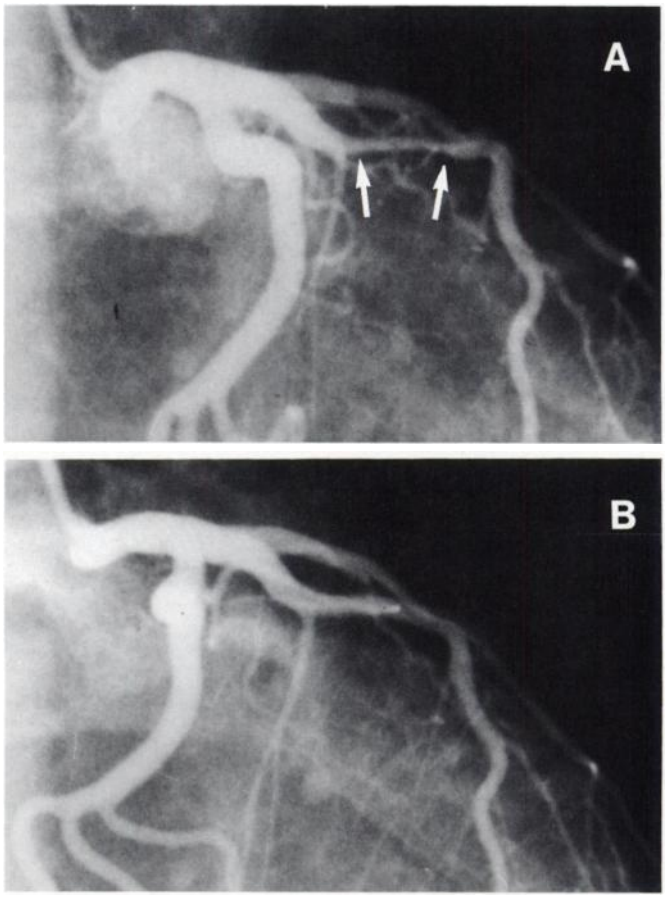

FIGURE 2. Coronary angiogram demonstrates (A) Narrowing of the LAD (between arrows) distal to the first perforator during systole and (B) return to normal caliber during diastole.

may develop chest pain, ST-segment depression or lactate production (*6*). Myocardial bridging has also been found in young individuals who died suddenly during heavy exertion with areas of myocardial fibrosis in the territory of the bridged vessel (*7*). In an autopsy series, myocardial bridging, either single or multiple, is found in 56% of consecutive cases with the left anterior descending artery most commonly involved (*8*). In the present case, myocardial bridging was the only cause found for the marked reversible ischemia demonstrated in the corresponding distribution on SPECT ^{201}Tl scintigraphy in the anterior wall and septum. There was no evidence of coronary atherosclerosis.

Previous studies utilizing planar ^{201}Tl scanning demonstrated normal perfusion in patients with myocardial bridging (*9,10*). In another study, dipyridamole ^{201}Tl scanning demonstrated a perfusion defect in a patient with myocardial bridging (*11*). They concluded, however, that the perfusion abnormality resulted from the hemodynamic effect of dipyridamole, not myocardial bridging.

Myocardial ischemia has been documented by exercise planar ^{201}Tl scanning in a patient with Wolf-Parkinson-White syndrome and myocardial bridging during an episode of paroxysmal supraventricular tachycardia (*12*). Ahmad et al. (*13*) demonstrated perfusion defects by performing planar ^{201}Tl scintigraphy on three patients with myocardial bridging of greater than 75% systolic narrowing. One of those patients developed chest pain. Our case further shows that myocardial bridging may cause myocardial ischemia, particularly during episodes of tachycardia, and should be considered when other causes of ischemia such as coronary atherosclerosis, spasm, arteritis and embolism are excluded. Previous studies utilized planar ^{201}Tl scintigraphy and not all studies detected myocardial ischemia in patients with myocardial bridging. The use of SPECT ^{201}Tl may increase the detection of ischemia in these patients.

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