Dipyridamole Thallium-201 Myocardial Single Photon Emission Computed Tomography in Myocardial Bridging — A Case Report

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Myocardial bridging is a congenital anomaly of the myocardium in which a normally epicardial coronary artery is bridged by a band of muscle fibers. The artery is of normal caliber during diastole, but becomes compressed during systole. We present a patient with atypical chest pain, positive treadmill electrocardiographic test and angiographically proven 60% systolic bridging at the proximal portion of the left anterior descending artery. Dipyridamole thallium-201 myocardial SPECT demonstrated mild reduction of perfusion to the anterior wall of the left ventricle. Redistribution images demonstrated good reversibility of the perfusion defects, indicating myocardial ischemia. This case provides additional supportive evidence that myocardial bridging may cause myocardial ischemia.

Key Words: Dipyridamole • Myocardial bridging • Myocardial ischemia • Single photon emission computed tomography (SPECT) • Thallium-201

INTRODUCTION

Myocardial bridging is defined angiographically as systolic compression of an intramyocardial segment of a normally epicardial coronary artery. The artery is of normal caliber during diastole, but becomes compressed or “milked” during systole. Whether myocardial bridging causes myocardial ischemia and whether treatment is necessary are still controversial. To date, there have been only a few cases of abnormal thallium-201 (Tl-201) myocardial single photon emission computed tomography (SPECT) in this condition. We present a case of myocardial bridging causing myocardial ischemia demonstrated by dipyridamole Tl-201 myocardial SPECT.

CASE REPORT

A 41-year-old male was referred to our cardiology department for evaluation of atypical chest pain and positive exercise electrocardiographic test. He presented with a 2-month history of substernal chest pain. The pain lasted 5-10 minutes and occurred with exertion or when at rest. He had become unable to maintain his normal lifestyle. He was not hypertensive, did not smoke and had no family history of premature coronary artery disease. His blood sugar and lipid profiles were within normal limits. Physical examination revealed blood pressure 130/80 mmHg, pulse 72 beats/minute and regular, and respiratory rate 14 breaths/min. The lungs were clear and the heart sounds and peripheral pulses were normal. The 12-lead electrocardiogram (ECG) demonstrated normal sinus rhythm with normal tracing.

Because of atypical features of the chest pain and no major risk factors for coronary artery disease, a non-invasive diagnostic study was recommended. The patient underwent dipyridamole Tl-201 myocardial SPECT. Dipyridamole was infused intravenously over a 4-minute period, using the standard dose of 0.14 mg/Kg/min.
Baseline vital signs were blood pressure 130/80 mmHg and pulse 84 beats/min. Minimal blood pressure after infusion was 104/58 mmHg, with a maximal heart rate of 105 beats/min. There was 1 mm of down-sloping ST-segment depression in leads II, III, aVF and V3-V5 4 minutes after the end of dipyridamole infusion. The stress and 4-hr redistribution images demonstrated a reversible perfusion defect in the anterior wall of the left ventricle (Figure 1). Because of the abnormal thallium study, cardiac catheterization was subsequently performed. The left ventriculograms showed normal left ventricular wall motion. The coronary angiograms demonstrated widely patent coronary arteries with no evidence of atherosclerosis. The left anterior descending (LAD) artery, however, had 60% systolic narrowing in diameter at the proximal portion, which is a typical picture of myocardial bridging (Figure 2). Because the patient refused revascularization therapy, he was treated with beta-blocker and was followed up at the outpatient department. His symptoms became much improved after medical therapy.

DISCUSSION

Myocardial bridging is a congenital anomaly of the myocardium in which a normal epicardial coronary artery is bridged by a band of muscle fibers. Angiographically, the artery is of normal caliber during diastole but during systole becomes compressed or “milked”. Pathologic studies have reported myocardial bridging in 5%-86% of human hearts. All major epicardial coronary arteries have been involved with incidence at autopsy, being 5%-60% of the LAD artery, 12%-43% of the circumflex artery and 0.4%-41% of the right coronary artery. Angiographic studies in humans, however, have disclosed a much lower incidence of myocardial bridging. Bridging has been reported in 0.5%-12% of human angiograms, with the LAD artery most commonly affected.
Historically, myocardial bridging has been thought to be without hemodynamic significance, since most coronary blood flow occurs during diastole. However, myocardial bridging has been reported to cause myocardial ischemia in a few cases. Kramer et al. found systolic bridging in 12% of 658 angiograms, all in LAD. Of the 26 patients with less than 30% systolic narrowing, 10 had normal exercise tests. Of the 55 patients with 30%-50% systolic narrowing, 2 of the 12 who were stress tested had ECG changes with exercise. Of the 11 patients with greater than 50% systolic bridging, 1 of 3 had ischemic ECG changes with exercise. The 5-year survival was 98%, with 1 death from aortic dissection but none from other cardiovascular causes. These investigators concluded that myocardial bridging could cause ischemia but in general was a benign condition. Other investigators, however, have disagreed with the assertion that all myocardial bridging is benign. Noble et al. found an incidence of LAD bridging in 0.5% of 5250 patients undergoing angiography. The 11 patients with bridging but without other coronary abnormalities on angiography were studied with both pacing and exercise. Of the 2 patients with less than 50% systolic narrowing, none had ECG changes, lactate production or symptoms with either pacing or exercise. Of the 4 patients with 50%-75% systolic narrowing, 2 had angina and ST depression with pacing at 150 bpm. Of the 5 patients with greater than 75% systolic bridging, 4 had ST-segment depression and increased coronary sinus lactate production with pacing at 150 bpm, 3 had angina with pacing at 150 bpm and 2 had exertional angina on treadmill testing. The authors concluded that myocardial bridging was indeed capable of provoking ischemia, possibly via preferential shortening of diastole with increased heart rate and consequently diminished coronary blood flow. In a single patient with LAD bridging, Pichard and colleagues reported that pacing at 140 bpm caused angina, ST depression and reduced flow in the great cardiac vein, which drains the LAD territory. There also have been case reports linking myocardial bridging with angina, acute myocardial infarction, paroxysmal supraventricular tachycardia and ventricular tachycardia. In the present case, myocardial bridging was the only cause found for the reversible perfusion defect demonstrated in the corresponding distribution on TI-201 SPECT scintigraphy in the anterior wall.

In a previous study utilizing exercise planar TI-201 scanning, Greenspan et al. reported that all 7 patients with chest pain and 60%-80% LAD bridging had normal myocardial perfusion. However, myocardial ischemia has been documented by exercise planar TI-201 scanning in 3 patients with greater than 75% systolic bridging of the LAD and another patient with Wolf-Parkinson-White syndrome and myocardial bridging during an episode of supraventricular tachycardia. In another case report, exercise TI-201 myocardial SPECT demonstrated reversible perfusion defects in the anterior wall and septum in a patient with 60% systolic bridging of the LAD. Our case also shows that myocardial bridging may cause myocardial ischemia and should be considered in the absence of coronary occlusive disease when myocardial ischemia is evident on the myocardial SPECT scintigrams. Factors affecting the production of myocardial ischemia may include the length of the coronary tunneling and the degree of systolic compression.

REFERENCES


