

## Myocardial Bridging

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The author reports a case of an applicant whose myocardial bridging caused myocardial infarction. It had been thought that most instances of bridging were of little clinical significance. However, there have been reports showing that severe bridging of the left anterior descending coronary can produce myocardial ischemia or sudden death.

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The proposed insured is a 66-year-old man applying for a \$125,000 universal life product in May 2004. He is a non-smoker and non-drinker. On the application, he admitted to having had hip surgery followed by a heart attack and catheterization. The paramed examination showed his height to be 5'6", weight 158 lb, and his blood pressure was 112/74 mm Hg. The blood chemistry profile and urine specimen were normal. The cholesterol was 219 mg/dL (175–250), HDL cholesterol 65 mg/dL (35–100), LDL cholesterol 120 mg/dL (70–160) and the Cholesterol/HDL cholesterol ratio was 3.4 (<5.5). The electrocardiogram was unremarkable. An attending physician's statement (APS) was obtained. On October 25, 2002, the proposed insured had hip replacement surgery for his progressive osteoarthritis. Perioperatively, his hemoglobin fell to 9.5 gm, and he became hypotensive. When the proposed insured developed chest pain, cardiology was consulted. His risk factors were determined to be hyperlipidemia and a remote family history for heart disease.

He had no history of hypertension or diabetes. The evaluation included serial EKGs, troponin levels, and cardiac enzymes; they supported the diagnosis of a non-Q wave myocardial infarction having occurred October 25.

After he was stabilized, a cardiac catheterization was done. It showed normal systolic left ventricular function. There was mild diastolic dysfunction as suggested by the left ventricular end-diastolic pressure of 14–18 mm Hg (normal range 5–12 mm Hg). The coronary angiogram demonstrated a normal left main coronary artery. Consistent with a severe myocardial bridge was a long segment (2 cm) involving the mid-portion of the left anterior descending, which completely obliterated during systole. Plaquing was noted to involve the same area. Mild nonobstructive disease was present in the circumflex and right coronary systems. The impressions included: diastolic left ventricular dysfunction, most likely secondary to an occult cardiomyopathy; normal systolic function; severe

myocardial bridging; and mild plaquing involving the same segment, the circumflex system and right coronary system. The echocardiogram and Doppler findings included: normal systolic left ventricular function, left atrial size of 4.1 cm (normal less than 4.0 cm), aortic root was 3.8 cm (2.0–3.7 cm), and normal heart valves. He was discharged October 28, 2002, on Toprol-XL (metoprolol), Zocor (simvastatin), Coumadin (warfarin) and aspirin. No further clinical notes were available.

## DISCUSSION

The major coronary arteries normally are distributed over the epicardial surface of the heart. Occasionally, one of the arteries will have a segmental intramyocardial course; the artery is "bridged" by a band of myocardial fibers. During systole, this segment is compressed, a condition referred to as systolic myocardial bridging or milking. During systole, a segment of the artery is compressed; it reverses during diastole. This phasic nature of the obstruction helps differentiate bridging from fixed coronary stenosis.<sup>1</sup> This phenomenon was first described in depth in 1951 and was recognized angiographically in 1960.

Mohlenkamp et al report 11 autopsy studies. Of these 2666 autopsies, 663 (25%) showed bridging (the range was 5%–85%). They also report 16 angiography studies. Of these 28,485 angiographies, 496 (1.7%) had bridging (range 0.5%–12%). One of the reports was 658 angiographic studies showing bridging in 79 (12%) in otherwise normal coronary arteries. These 11 studies included 62 subjects who had angiographic normal coronaries and underwent provocative tests. In this group, 40% had myocardial bridging. In all the studies, the left anterior descending artery was most commonly affected. In those with hypertrophic cardiomyopathy and in recipients of cardiac transplants, the prevalence of bridging was high.<sup>1</sup>

Detecting myocardial bridging angiographically is influenced by medications affecting the contractile state of the ventricle. Beta-blockers given intravenously have been

shown to attenuate a systolic luminal reduction in these patients. Intravenous isoproterenol and intracoronary nitroglycerine have been shown to enhance the detection rate.<sup>2</sup> Because the affected region of the ventricle typically becomes akinetic or dyskinetic after an acute myocardial infarction, it is conceivable a myocardial bridge causing this condition may be unrecognized on angiogram during the acute phase of the myocardial infarction.<sup>3</sup> Cardiac meds and acute myocardial infarctions may help explain the wide range of reported frequency of myocardial bridging.

Coronary atherosclerosis in association with myocardial bridging of the left anterior descending artery has been studied. Generally, the segment proximal to the bridge shows atherosclerotic plaque, but the segment of the artery tunneled into the myocardium is spared.<sup>1</sup>

Myocardial bridging causes coronary artery obstruction during systole. There is usually no significant reduction in total myocardial perfusion since almost two thirds of blood flow in the left coronary system occurs in diastole. Myocardial bridging is capable of provoking ischemia, possibly by preferential shortening of diastole with increased heart rate. Also, the greater the degree of systolic narrowing, the more persistence of narrowing into early diastole occurs. This is referred to as "spill over" phenomenon. Myocardial bridging should be considered in the absence of coronary occlusive disease when the myocardial ischemia is evident on the myocardial SPECT scintigrams. The length of the coronary tunneling and the degree of systolic compression are factors that affect the production of myocardial ischemia.<sup>3</sup>

In a study of 658 angiograms, systolic bridging was found in 12%, all being in the left anterior descending artery. Twenty-six of these had <30% systolic narrowing; 10 had normal exercise tests. Of the 55 with 30%–50% narrowing, 2 of the 12 who were stressed had EKG changes with exercise. Of the 11 with >50% systolic bridging, 1 of 3 had ischemic EKG changes with exercise. The group had a 5-year mortality rate of 98%. The

1 death was from aortic dissection. The investigators felt that, in general, myocardial bridging was a benign condition, though it could cause ischemia.<sup>4</sup>

In a group of 5250 undergoing angiography, another group of investigators found this condition to be less benign. The incidence of LAD bridging was 0.5%. There were 11 patients with bridging and no other coronary artery abnormalities. This group was paced and put on an exercise treadmill. Of these 11 subjects, 2 had <50% systolic narrowing. When stressed, none of them had EKG changes 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 beats per minute. Of the 5 patients with greater than 75% systolic bridging, 4 had ST depression with pacing at 150 beats per minute, 3 had angina with pacing at 150 beats per minute and 2 had exertional angina on treadmill testing.<sup>5</sup>

Bridging of coronary arteries that is observed in otherwise angiographically normal arteries generally is not hazardous to the patient. However, strenuous physical exertion results in compression of a portion of a coronary artery by a myocardial bridge. This may be associated with clinical myocardial ischemia and/or malignant ventricular arrhythmias.<sup>6</sup> A study of a group of patients who developed shock despite successful reperfusion of the infarct-related lesion showed that myocardial bridging may play a role in left ventricular function in the acute stage of inferior wall myocardial infarction as an independent predictor of shock in acute inferior wall myocardial infarction.<sup>7</sup> About 6% of patients with myocardial infarction have no evidence of coronary atherosclerosis by angiography or at autopsy.<sup>9</sup> A myocardial bridge is felt to be the cause of the infarction in some of these patients. Long-term prognosis in individuals with isolated myocardial bridging is good. In one small study of 81 people aged 46 years, the 5-year mortality was 97.5% (neither of the 2 deaths were related to myocardial bridging). Another group of 61 people aged 50 years with myocardial bridging, 11-

year survival was 98%. As in the first study, none of the deaths were related to myocardial bridging.<sup>1</sup>

However, it appears that deep and long myocardial bridges are often not benign. The presence of a myocardial bridge, in association with factors that increase the contractility or heart rate, have caused acute coronary syndromes and even sudden cardiac death. It would make sense to avoid drugs and activities that increase heart rate/contractility in patients with documented long and/or deep myocardial bridges. Profound anemia is another exacerbating factor. In combination with myocardial bridging, it has been shown to cause a myocardial infarction.<sup>9</sup>

Myocardial bridging is more common in cardiac disease associated with left ventricular hypertrophy. Its incidence seems to be related to the severity of left ventricular hypertrophy. Coronary thrombotic occlusion has been reported in patients with myocardial bridge, who present with an acute myocardial infarction.<sup>2</sup> Sorajja et al investigated the risk of sudden cardiac death and other mortality in adult patients with hypertrophic cardiomyopathy (HCM), whose coronary angiography also demonstrated myocardial bridging. This Mayo Clinic study involved 425 adults with HCM who underwent coronary angiography. Of these, 64 (15%) had myocardial bridging. Mean follow-up for the study was 6.8 years. This study found no increased risk of death, including sudden cardiac death, among adults with HCM, who had myocardial bridging diagnosed at coronary angiography.<sup>10</sup>

The medical management of angina caused by bridging includes beta blockers and possibly non-dihydropyridine calcium channel blockers (eg, verapamil, diltiazem), because they reduce heart rate and myocardial contractility. Nitrates such as nitroglycerine are contraindicated. Young people who have clinical evidence of ischemia shown to be related to myocardial bridging benefit from being on a beta blocker.<sup>11</sup> Surgical therapy is generally reserved for those who have persistent symptoms, proven ischemic changes, and those

with a high risk marker (such as nonfatal myocardial infarction and aborted sudden death) in how medical therapy has failed. Intracoronary stent placement may improve clinical symptoms. The incidence of in-stent restenosis requiring revascularization is about the same as seen with stenting an obstructive lesion >25 mm in length in the diseased coronary artery.<sup>12</sup>

Myotomy may be effective in treating patients with symptomatic myocardial bridging. This was done in a young woman with hypertrophic cardiomyopathy who presented with angina due to a myocardial bridge over the left anterior descending artery producing severe compression during systole. A percutaneous intracoronary stenting was done. Within 30 days of the procedure, she restenosed, and a myotomy was done.<sup>13</sup> Some have combined resecting the muscle bridge with bypass grafting.

Our proposed insured had myocardial bridging that was classified as significant by its angiographic appearance. It proved to be clinically significant. The angiogram showed a background of "mild" (nonobstructive) coronary disease both in the involved segment of bridging and in other areas, as well. Although the underlying disease process of myocardial bridging and atherosclerosis are very different, in this individual the mortality of his bridging was assumed to be similar to an intracoronary obstructive lesion. In addition, the proposed insured was felt to have a hypertrophic cardiomyopathy. While the literature suggests that this does not add to the mortality created by myocardial bridging, one still might question its significance. These aspects of the case and the lack of recent follow-up were considered when the overall mortality was assessed.

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