

## Myocardial Bridging

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Human myocardial bridging is a normal anatomic variation in which a coronary artery is bridged by a short segment of myocardium. It can cause variable degrees of systolic obstruction. The majority of patients are asymptomatic. A wide variety of syndromes can occur, including myocardial infarction and sudden death. All patients with myocardial bridges have systolic artery compression, but it is postulated that ischemia develops only in those who have a concomitant decrease in diastolic coronary artery blood flow. Surgical removal of the myocardial bridge can be curative, and various other treatments can alleviate symptoms. The overall prognosis is good.

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### CASE REPORT

In 1997, a 68-year-old man applied for a modified single-premium variable life insurance policy. The single premium payable was \$65,000, with a net amount at risk of approximately \$50,000. The history admitted on the application was that of a possible myocardial infarction in 1990. At that time, the proposed insured admitted to a catheterization with a finding of myocardial bridging. He was treated with atenolol 25 mg/d, and since that time he had no further symptoms. The only other admitted history was that of remote surgery on his left knee.

Because of the admitted history, an Attending Physician's Statement was obtained. The information was quite interesting. There were numerous visits during 1985-1997 because of

ongoing low back pain with intermittent radiculopathy. The statement included documentation of degenerative disk disease of the lumbar spine and intermittent treatment with various types of nonsteroidal anti-inflammatory drugs. The proposed insured's weight was 157 lb. In August 1987 it was noted that he had a positive thallium stress test with no chest pain during the test. There was no indication of why the test was performed, but it may be presumed that he was experiencing chest pain. He was prescribed diltiazem daily and nitroglycerin as required. The remainder of the notation states that the patient would be followed clinically, and if further chest pain occurred, angiography would be performed. The proposed insured did well for the next 3 years without any apparent chest pain. In 1990, a resting electrocardiogram

(ECG) showed the development of ST depression and T wave changes. Because of the changed ECG and his previous history, a repeated thallium treadmill stress test was performed. This test showed ECG changes of ischemia (amount of ST depression not noted), as well as reversible areas on the thallium scan in the septal area plus a fixed defect in the inferior wall consistent with an old scar. After this, cardiac catheterization was performed. The catheterization showed the proposed insured to have a left dominant system, with the inferior left ventricle supplied by a branch from the left coronary artery. The right coronary artery was clear. The proximal left anterior descending artery showed minimal plaque formation. The middle left anterior descending artery was described as having an area of severe bridging with 75% to 95% narrowing. In the circumflex artery, the proximal marginal branch was also described as having an area of severe bridging, with 75% to 95% narrowing. The chamber sizes were all normal, as was the ejection fraction. The recommendation was to treat the patient with a  $\beta$ -blocker, and atenolol 50 mg/d was started. The proposed insured continued on atenolol without apparent chest pain. In February 1995, a resting ECG showed the new development of a complete right bundle branch block (RBBB). Because of this, a repeated thallium treadmill stress test was performed. The proposed insured exercised for 9 minutes, achieving a maximum heart rate of 154 beats per minute with a normal blood pressure response. The thallium scan showed reversible areas of ischemia in the anterior and apical portions, but the previously described fixed inferior defect was not present. The recommendation was to continue atenolol and diltiazem. The subject continued to do well, with no further notations of any chest pain. The only other data in the Attending Physician's Statement were lipid measurements. In 1990, the subject's cholesterol was 221 mg/dL, high-density lipoprotein was 42 mg/dL, low-density lipoprotein was 160 mg/dL, and triglycerides were 75 mg/dL. In 1995, repeated lipid measurements showed

the cholesterol to be 196 mg/dL, high-density lipoprotein 42 mg/dL, low-density lipoprotein 132 mg/dL, and triglycerides 112 mg/dL. Thyroid function studies were also normal at this time.

## CASE DISCUSSION

Typically, the coronary arteries are epicardial in location, running on the surface of the myocardium. Myocardial bridging describes an anatomic variation in which a portion of the coronary artery lies within the myocardium itself and is no longer epicardial in location. The myocardial bridge (MB) usually consists of a short segment of a superficial muscular band that runs across a coronary artery, thus forming a "bridge."

Myocardial bridging was first described in 1737, when Reyman noted this particular anatomy in human hearts.<sup>1</sup> In 1961, Polacek was the first person to use the term *myocardial bridge*.<sup>2</sup> With the development and increasing frequency of coronary angiography, an increasing interest in this entity developed. There has been a great deal of debate regarding whether MB is related to a true myocardial abnormality or a normal variant of coronary anatomy. Animals have been divided into 3 groups depending on the location of their coronary arteries.<sup>3</sup> In type A, the coronary arteries are entirely within the myocardium (eg, squirrels, rabbits). In type B, the coronary arteries are predominantly epicardial, but frequent MBs are found (eg, sheep, dogs). In type C, the coronary arteries are completely epicardial (eg, horses, cows). Humans are type B. Most researchers now consider MB to be a normal variation of human coronary anatomy.

MB findings are frequent. The occurrence has been estimated from 15% to 66%.<sup>4</sup> An average of 25% seems reasonable on the basis of prevalence studies. The length of MBs has been reported to range from 3 to 69 mm, and they occur predominantly in the proximal left anterior descending artery and rarely in the right coronary artery.<sup>5</sup> Interestingly, the MB muscle is different from other myocardial

muscle, with a distinctive spatial arrangement and the myofibers separated by an increased amount of connective tissue compared with normal muscle. MBs cause retardation of coronary artery systolic blood flow by way of compression, and this compression can be of variable degree. Noble et al classified this obstruction (called the milking effect on angiography) into 3 grades: grade 1 with <50% obstruction, grade 2 with 50% to 75% obstruction, and grade 3 with >75% obstruction.<sup>6</sup> The degree of obstruction is important because most clinical syndromes associated with MB are found in the grade 3 classification. However, patients who have the same degree of obstruction do not manifest any common clinical presentations, which can range from no symptoms to serious life-threatening consequences. The clinical syndromes associated with MB include chest pain, typical angina pectoris, myocardial ischemia as documented by objective testing (such as thallium testing), myocardial infarction, atherosclerosis of coronary arteries, ventricular arrhythmias, and sudden death. The vast majority of persons with MBs are asymptomatic.

Investigators have attempted to define the differences between symptomatic and asymptomatic patients who appear to have the same degree of systolic artery compression. Current research has focused on the use of intracoronary Doppler flow studies to measure actual decreases in flow seen on angiography.<sup>7</sup> All grades of MB milking effect cause a decrease in coronary artery blood flow during systole. This is more pronounced when tachycardia is present. Normally, after the systolic compression of the coronary artery, there is a fairly quick return of the diameter beginning in early diastole concomitant with an increase in blood flow. Early diastolic diameter increase has been shown to be delayed and accompanied by a decrease in mean coronary blood flow reserve distal to the MB. In the 42 symptomatic patients studied by Schwartz et al, all had grade 2 or 3 narrowing.<sup>7</sup> All patients also had a delayed increase in the diastolic diameter of the cor-

onary arteries, as described. It is reasonable to assume that myocardial ischemia could result in a setting in which there are decreases in both systolic and diastolic flow.

Another interesting finding has been that the intima of the coronary arteries is thinner at the site of the MB but is often accompanied by hyperplasia of the intima in the coronary artery just proximal (and occasionally distal) to the MB.<sup>8</sup> Hansen postulated that this hyperplastic area is more likely to develop atherosclerotic narrowing, thus leading to occlusion and infarct.<sup>9</sup> Actual atherosclerotic narrowing in the area of the MB itself occurs infrequently, perhaps related to the thinning of the intima, which may be protective. Various forms of therapy may be tried in symptomatic patients with MB. Bourassa et al first suggested surgical removal in 1975.<sup>10</sup> Patients with classic angina pectoris have shown relief of their symptoms with either removal of the MB or bypass grafting to the coronary artery distal to the lesion.  $\beta$ -Blockers have been used to alleviate symptoms by slowing the heart rate, thus allowing increased flow during the longer diastolic perfusion phase. Calcium channel blockers have also been used. Nitrates have been shown to increase the systolic obstruction from MB and actually worsen symptoms.

To summarize, MB is a fairly common and normal variation in human coronary artery anatomy. It has been associated with a variety of clinical syndromes, but overall this is quite uncommon. When it is associated with ischemia, the systolic compression is usually severe, and ischemia probably results from a reduction in diastolic coronary flow. Systolic compression alone does not cause ischemia under baseline conditions.

How can we apply this information to our proposed insured? Ten years before the application he had a positive thallium stress test, apparently performed because of chest pain. Three years later, he had repeated thallium testing because of a resting ECG abnormality. Catheterization at this time showed a myocardial bridge described as a class 3 narrowing of 75% to 95%. There were no other

significant atherosclerotic lesions to explain his chest pain. The long-term use of nonsteroidal anti-inflammatory drugs could cause gastrointestinal symptomatology that could mimic cardiac pain. The thallium stress test of 1990 suggested an old inferior infarction, but this was not apparent at the time of angiography and also was not present on a repeated thallium treadmill in 1995. When the patient developed a new complete RBBB in 1995, he was able to exercise for 9 minutes with a normal blood pressure response and no chest pain. He continued to be asymptomatic on atenolol and diltiazem daily. Because the patient's MB was severe, he belongs to the category in which clinical syndromes could develop. He had reversible areas of ischemia on thallium stress tests in 1987, 1990, and 1995. Despite this, he remained asymptomatic with no chest pain. He did develop RBBB. It is interesting to speculate on the significance of this RBBB. In 80% to 90% of human hearts, the right coronary artery has a large first posterior interventricular artery that supplies the atrioventricular node and the proximal right bundle. In our patient, the right coronary artery was clear on angiography. What conclusions are to be drawn from the 3 positive thallium scans during 1987–1995? With class 3 obstruction of both the left and circumflex arteries angiographically, the subject could be prone to a delay in diastolic filling, thus producing the abnormal thallium scan. At the same time, he was able to walk 9 minutes on a treadmill with no chest pain, no arrhythmias, and a normal blood pressure response. He is 68 years of age and has not had any life-threatening cardiac events, has reasonable lipid determinations, and has not developed any significant classic atherosclerotic narrowing of his coronary arteries. Taken together, all this

would suggest a reasonable prognosis in the near future.

In underwriting MB, one needs to know the degree of coronary obstruction occurring during systole. Additionally, one needs objective data that could suggest true ischemia (which would correlate with the delay in diastolic filling postulated to be the mechanism for ischemia in these patients). It is necessary to correlate this with the clinical presentation, if any, and it is advisable to know whether any of these clinical events were life threatening. Finally, one should take into consideration the treatment administered.

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