Surgical Unroofing a Long-Segment Myocardial Bridge of the Left Anterior Descending Artery Leading to Complete Angiographic and Symptomatic Improvement: A Case Report

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Abstract

A 52-year-old man presented to our institution with a one year history of intermittent, progressive chest tightness with activity. He underwent stress test which was positive. Coronary angiography revealed a long segment myocardial bridge of the left anterior descending artery (LAD). Initially, he was trialed on maximal medical therapy, however, symptoms continued. As a result, he was brought to the operating room for unroofing of the myocardial bridge with excellent clinical and radiographic outcome. The diagnosis, medical, and surgical management of myocardial bridge is complicated. Long-term data surrounding best options for management is scarce.

Background:

Myocardial bridging is an anatomical variant, occurring when an epicardial coronary artery contains an intramyocardial segment that becomes compressed during systole.[1] Most commonly, myocardial bridging occurs in the mid-portion of the LAD. The incidence and prevalence vary widely due to variation in diagnostic methods and definitions of myocardial bridging.[2,3] Currently, no diagnostic gold standard exists for clinically significant myocardial bridging.

Longstanding debate surrounds the clinical significance of myocardial bridging. Since myocardial perfusion occurs mainly during diastole, it was thought that systolic compression in the setting of a myocardial bridge would not have significant clinical impact. However, angiography and intravascular ultrasound (IVUS) have shown that compression extends into diastole, inducing symptoms from delayed diastolic relaxation and decreased perfusion. Additionally, myocardial bridging may cause persistent reduction in diastolic luminal diameter, retrograde systolic flow, reduced coronary flow reserve, and increased velocity of blood flow.[2,4] Often, the tunneled artery segment in symptomatic patients has endothelial dysfunction. The portion of the artery proximal to the bridge may be prone to atherosclerosis due to alterations in flow patterns. The bridged segment has a propensity toward coronary spasm due to endothelial dysfunction.[1,3-5] Patient specific features such as the depth and length of myocardial bridge, septal branch involvement, and degree of systolic diameter reduction influence the clinical impact.

Case Presentation:

A 52-year-old man presented to our institution with a one year history of intermittent, progressive chest tightness. Past medical history was notable for hypertension, hyperlipidemia, post-traumatic stress disorder and family history of coronary artery disease (CAD). Prior to developing symptoms, he was quite physically active; however, became less so prior to presentation due to symptomatic limitations.

He underwent an exercise stress test that showed good functional capacity (exercise time of 9 min 47 seconds) but demonstrated 2-3 mm ST depressions starting in Stage 2 of the bruce protocol in leads II, III, aVF and V4-V6. The patient also expressed dypnea with exercise that resolved 5 minutes into recovery. Coronary angiography was then performed, demonstrating non-obstructive CAD . However, a long segment myocardial bridge in the mid-LAD was observed (Figure 1) Transthoracic echocardiogram (TTE) demonstrated normal left ventricular size, normal systolic function and an estimated left ventricular ejection fraction of 60-65%.

Initially, the patient was trialed on maximal medical therapy for symptomatic relief, including amlodipine, aspirin and rosuvastatin. His symptoms persisted. He was subsequently referred for cardiac surgical evaluation. He was deemed an appropriate operative candidate, and was brought to the operating room for surgical unroofing of the myocardial bridge on April, 2020.

Standard aortic and dual stage venous cannulation was performed. The aorta was cross clamped and the heart was arrested with antegrade Del Nido cardioplegia. Upon surface inspection, a long segment of intramyocardial LAD was easily identified (Figure 2). The LAD was unroofed with the use of a 15-c blade over the course of 10 mm. The aortic cross clamp was removed and the patient was easily separated from cardiopulmonary bypass. He underwent coronary angiography on post-operative day one to evaluate the results of the surgery radiographically. Angiography revealed that the myocardial bridge had been entirely relieved (Figure 3). The patient had an uneventful post-operative course and was discharged home on postoperative day number four. He was seen in follow-up one and two months post procedure and is doing well without recurrence of symptoms.

Discussion:

The Schwarz classification is used for guiding therapeutic considerations in patients with an identified myocardial bridge. This classification is broken down into types A, B, and C. Type A patients have an incidental finding of myocardial bridging on angiography without objective signs of ischemia and do not require treatment. Type B patients demonstrate ischemia during stress testing and should receive treatment with beta-blockers or calcium-blockers. Type C patients have significantly altered intracoronary hemodynamics, with objective signs of ischemia, including symptoms. These patients should be managed with medical therapy initially, and if that fails, operative intervention should be pursued.[6]

Beta-blockers are useful in patients with myocardial bridging due to their negative inotropic and chronotropic effects coupled with reduced sympathetic drive, allowing for increased length of diastolic coronary filling and reduced compression. Calcium-channel blockers can be used as an alternative. Nitrates should be used with caution in these patients, as they increase systolic compression within the bridge while vasodilating the proximal segment. These altered hemodynamics may exacerbate retrograde flow thereby reducing the threshold for myocardial ischemia.

In patient's refractory to medical therapy, stenting may be considered. However, significant rates of in stent restenosis have been identified small numbers of patients. Additional concerns surrounding stenting include stent fracture, thrombosis, and increased risk of perforation during deployment, all of which has limited the utility of this treatment.[4,5]

A surgical gold standard has not been established for patients with an isolated myocardial bridge and refractory symptoms due to the infrequent finding of need for surgical intervention. Coronary artery bypass graft (CABG) with use of the left internal mammary artery (LIMA) to the LAD has been described. Concerns regarding the ability of the CABG graft to remain open due to competitive flow in a patient with non-obstructive CAD limits enthusiasm for this approach. Alternatively, the literature suggests that for patients with isolated myocardial bridge, the surgeon may carefully unroof the intra-myocardial component of the affected vessel. A perceived benefit of unroofing is that it can be done in isolation or in combination with CABG if necessary. The unroofing technique has been shown to be safe and effective in improving symptoms in a small number of patients. Interestingly, very little is in the literature regarding the actual angiographic outcome of patients who undergo surgical unroofing of a myocardial bridge as a result of significant symptoms. This case report demonstrates complete unroofing as confirmed by post-operative angiography in addition

to significant symptomatic improvement.

Conclusion:

In young patients with exertional chest pain, myocardial bridging should be considered in the differential diagnosis. While many patients with myocardial bridge respond well to medical therapy, those who do not should explore surgical options for symptomatic improvement and treatment.

The surgical management of a symptomatic patient with an isolated myocardial bridge is debated. CABG has been performed, however concerns regarding graft patency in the setting of non-obstructive CAD is warranted. Unroofing of the bridge has been described and performed in case reports throughout the literature, however post-operative symptomatic improvement and angiographic demonstration of complete bridge relief have been infrequently reported. Here, we describe a patient with a symptomatic myocardial bridge involving the LAD for which medical therapy was ineffective. Our patient underwent successful surgical unroofing of a long segment myocardial bridge, leading to total symptomatic improvement and complete resolution on post-operative coronary angiography.

Multi-institutional registries and randomized clinical trials are warranted to shed light on optimal strategies for patients with myocardial bridging refractory to medical therapy.

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