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A Bridge Too Far: A Case of Myocardial Bridge Requiring Unroofing and Complicated by Post-Cardiac Injury Syndrome

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Abstract

Myocardial bridging is a congenital heart defect in which a segment of a coronary artery tunnels underneath the myocardium and is also a rare cause of angina. The most commonly involved artery is the mid-segment of the left anterior descending artery (LAD) with a prevalence of 67-98% [1,2]. While medical management with negative inotropic/chronotropic agents such as beta blockers and calcium channel blockers are considered first-line therapy, surgical interventions including myocardial unroofing procedures can be safely performed and are associated with a 63% reduction in chest pain.

Background

The cases of angina that occur in patients with myocardial bridging occur during systole when the myocardium contracts and compresses the segment of the coronary artery that is tunneled underneath the myocardium. During this phase, the segment of the artery within the myocardium narrows, and blood flow supplying the heart decreases. The prevalence of myocardial bridging in literature varies significantly depending on the diagnostic method: coronary angiography reveals a prevalence of 6%, while autopsy reveals a prevalence of 42% [1]. This difference is due to the fact that myocardial bridging is typically benign and only incidentally found during autopsy. However, some patients with this condition may present with significant symptoms which require further evaluation and treatment [2]. This case report presents a patient with myocardial ischemia from myocardial bridging which required unroofing surgery. The patient subsequently developed post-cardiac injury syndrome.

Objective

Present myocardial bridging as an uncommon presentation of chest pain, as well as bring awareness to complications that may arise as a result of myocardial bridging.

Case Report

Our case presents a 49-year-old female with a past medical history of hypertension and hypothyroidism who presented to the emergency department complaining of a constant, excruciating, pressured, substernal chest pain with left shoulder radiation. The pain began 3 days prior to presentation and did not alleviate despite treatment with oxycodone-acetaminophen. The patient also reported associated symptoms of fever, chills, fatigue, nausea, and vomiting.

Upon admission, her blood pressure was 163/96mmHg, but her vital signs were otherwise unremarkable. Physical examination revealed left shoulder tenderness, with no abnormalities on cardiac auscultation. Initial work up revealed high-sensitivity cardiac troponins of 1.757ng/mL and an ECG consistent with normal sinus rhythm without ischemic changes. Transthoracic echocardiogram showed no wall motion abnormalities with preserved left ventricular ejection fraction of 65%. Aspirin, sublingual nitroglycerin, metoprolol, and heparin drip were initiated due to clinical findings concerning for NSTEMI, with symptom improvement. Troponin I level continued to increase during the following 24 hours and peaked at 22.200ng/mL.

Based on her symptomatology and increasing troponin levels, left heart catheterization was indicated and demonstrated a 50-60% segmental eccentric stenosis in the middle segment of the LAD that was aggravated by moderate myocardial mid-LAD bridge.

The patient was started on a calcium channel blocker for the myocardial bridge but continued to have intermittent chest pain. It was at this point that it was decided that an unroofing procedure would be necessary. Her symptoms resolved after an unroofing procedure where the LAD was immobilized, and the cardiothoracic surgeon was able to free approximately 1.5 cm of overlying muscle on the LAD. The LAD was then visualized proximally and the procedure of unroofing the muscle showed the continued pathway of the LAD with no overlying muscle. Subsequently, the patient's symptoms improved. She returned one month later with chest pain and was diagnosed as having post-cardiac injury syndrome (formerly Dressler syndrome), which is defined as pericarditis with or without a pericardial effusion resulting from injury of the pericardium. She was treated with ASA/colchicine and her symptoms resolved shortly thereafter.

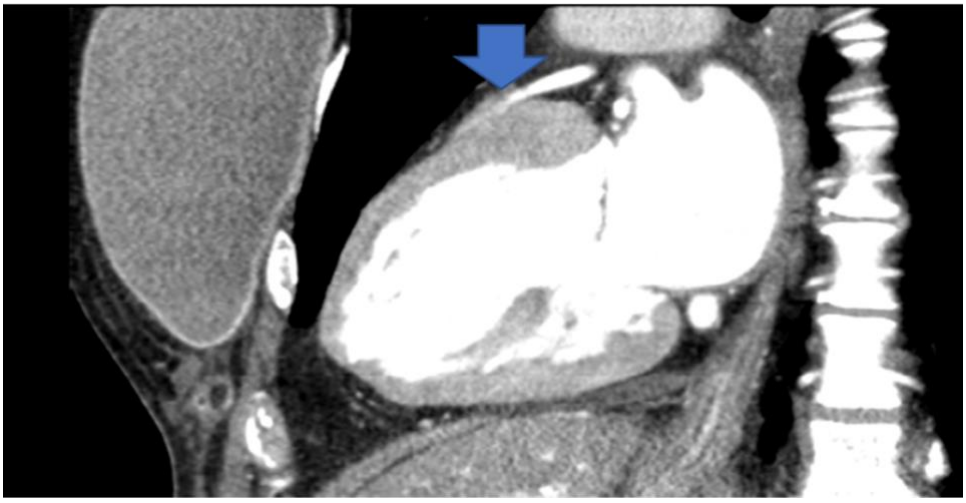

Figure 1: Cardiac CT showing invagination of LAD into the myocardium (Blue arrow).

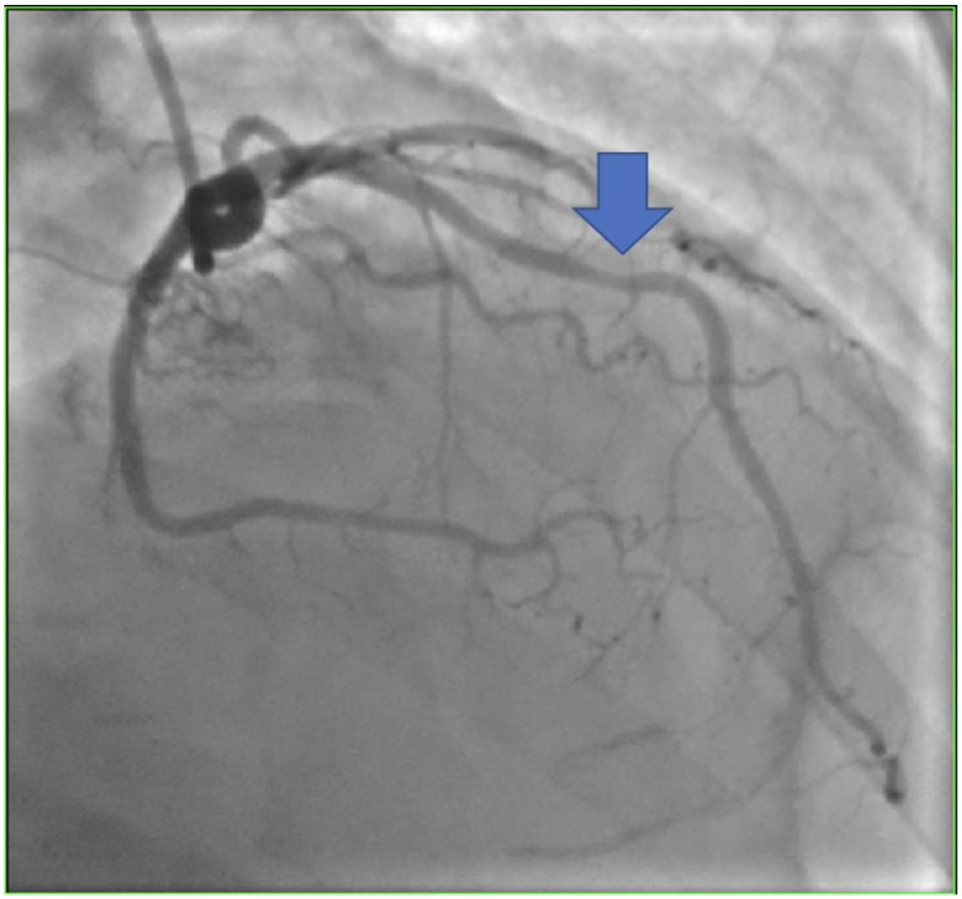

Figure 2: Coronary angiography demonstrating myocardial mid LAD bridge (Blue arrow).

Discussion

In myocardial bridging, compression of the coronary artery occurs during systole while most of the blood flow and oxygen supply to the myocardium happens during diastole. This pathophysiology leads to the asymptomatic presentation seen in most patients with myocardial bridging due to the absence of myocardial ischemia [3]. In some cases, compression of coronary arteries has been shown to persist during diastole in some patients, which causes decreased myocardial perfusion. When this occurs, these patients may present with a variety of clinical manifestations, which may include asymptomatic to acute coronary syndrome, coronary spasm, exercise-induced dysrhythmias, atrioventricular conduction block, myocardial stunning, transient ventricular dysfunction, syncope, or sudden death [4]. This array in presentation has no correlation to the length or depth of the tunneled segment, nor the degree of systolic compression [5]. Patients may also develop coronary artery disease due to the expedited development of atherosclerosis and endothelial dysfunction from the disruption of blood flow through the narrow myocardial bridging segment [3].

The preferred method of diagnosis for myocardial bridging is coronary angiography [6]. As the myocardium contracts during systole, the lumen of the artery is narrowed and returns to normal diameter during diastole. Intracoronary ultrasound is another diagnostic technique, and it provides additional information including the length, depth, and position of the tunneled arterial segment.

Moreover, the image produced by the Doppler ultrasound can show a sharp acceleration of flow in early diastole followed by an immediate marked deceleration and a mid-diastolic pressure plateau [7]. The most commonly involved artery is left anterior descending artery (LAD) with a prevalence of 67-98% [8].

The pharmacologic treatment of symptomatic myocardial bridging includes beta blockers and nondihydropyridine calcium channel blockers. Through their negative chronotropic effects, these agents can increase the diastolic coronary filling period and decrease the compression of the involved arteries. Antiplatelet therapy may be added if atherosclerosis is detected.

If these treatments are unsuccessful, as they were in this patient, different surgical procedures may be considered to provide therapeutic relief. These surgical procedures include unroofing with myotomy of the myocardium, coronary artery bypass graft (CABG), or stenting of the coronary arteries [9,10]. The unroofing procedure can be performed safely with almost no cardiac-related deaths and 63% resolution of chest pain. However, as was the case with this patient, patients may continue to have nonischemic chest pain, in which case medical therapy must be continued after surgery [11]. Compared to other options, unroofing surgery eliminates the risk of restenosis, stent thrombosis, and perforation during stent deployment from stenting procedures, and eliminates the risk of graft failure from CABG surgeries [9]. Moreover, this procedure has been shown to reverse local myocardial ischemia and increase coronary blood flow [10].

Due to the risk of myocardial ischemia associated with myocardial bridging, patients may develop post-cardiac injury syndrome, commonly known as Dressler Syndrome, due to the inflammation of the pericardium in the first 8 weeks following the ischemic incident [14]. The narrowing of the coronary blood vessels due to muscular compression produces the same immune-mediated responses to ischemia seen in atherosclerotic myocardial infarction. It is therefore important to be aware of the risk of this complication in patients with symptomatic myocardial bridging. Blood cultures may be obtained to differentiate between inflammatory and infectious etiologies of post-cardiac injury syndrome which may be precipitated by incidental viral infection or post-surgical infection. Treatment is initiated with NSAIDs for 4-6 weeks in mild cases and, if refractory, corticosteroids [15]. Severe cases with evidence of pericardial effusion may require pericardiocentesis with subsequent catheter drainage.

Conclusion

Myocardial bridging is a congenital heart defect in which a segment of a coronary artery tunnels underneath the myocardium. Although myocardial bridging is often benign, it is also a rare and often overlooked cause of angina. The most involved artery is the mid-segment of the left anterior descending artery (LAD) with a prevalence of 67-98%. While medical management with negative inotropic/chronotropic agents such as beta blockers and calcium channel blockers are considered first-line therapy, surgical interventions including myocardial unroofing procedures can be safely performed and are associated with a 63% reduction in chest pain.

A 49-year-old female with a past medical history of hypertension and hypothyroidism presented to the emergency department complaining of a constant, excruciating, pressure like substernal chest pain with left shoulder radiation. Treatment was initiated with conventional therapeutics (list them out) without success (describe why “without success”. What made you think it was unsuccessful? Were symptoms the same, worsening, was she experiencing new symptoms, were certain test showing that she was not improving?) At this point, the treatment was discontinued, and the patient was prepped for an unroofing procedure (at what point was it discovered that she had myocardial bridging? Include this in the timeline wherever it occurred so that it is understood why someone might think an unroofing procedure might be necessary). During this procedure, the LAD was immobilized, and the patient experienced immediate improvement in her symptoms (describe how she improved).

This case illustrates…? If conventional therapeutic treatments are unsuccessful in resolving angina in patients with myocardial bridging, different surgical procedures may be considered to provide relief. These surgical procedures include unroofing with myotomy of the myocardium, coronary artery bypass graft (CABG), or stenting of the coronary arteries. The unroofing procedure can be performed safely with almost no cardiac-related deaths and 63% resolution of chest pain. Compared to other options, unroofing surgery eliminates the risk of restenosis, stent thrombosis, and perforation during stent deployment from stenting procedures, and eliminates the risk of graft failure from CABG surgeries. Moreover, this procedure has been shown to reverse local myocardial ischemia and increase coronary blood flow.

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