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CASE REPORT

Case report on surgical deroofing of left anterior descending artery in middle aged symptomatic patient and literature review

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Abstract

Background: Myocardial bridging, previously considered benign, has been associated with cardiovascular complications. We present a case of severe left anterior descending (LAD) bridging in a 40-year-old male with refractory chest pain.
Case Presentation: The patient experienced atypical chest pain despite treatment with antihypertensive medication. Invasive coronary angiography revealed severe LAD bridging. Surgical deroofing of the LAD was performed, resulting in complete resolution of symptoms.
Management & Results: Diagnostic modalities such as angiography and computed tomography angiography (CTA) aid in detecting myocardial bridging. Medical therapy involving beta blockers and calcium channel blockers is the first-line approach. Surgical intervention may be considered in refractory cases. Successful deroofing of the LAD in our patient resulted in symptom resolution.
Conclusion: Myocardial bridging should be considered as a potential cause of chest pain, and surgical intervention can be effective in relieving symptoms. Further research is needed to optimize diagnostic strategies and treatment approaches for myocardial bridging.

Keywords

Myocardial Bridging, Chest Pain, Left Anterior Descending Artery, Surgical Deroofing, Refractory Symptoms.

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Introduction

Myocardial bridge is a congenital anomaly commonly observed in coronary arteries. It refers to a condition where a band of myocardium overlies an epicardial coronary artery, causing compression during the systolic phase of the cardiac cycle[1]. Initially considered a benign variant, myocardial bridging has been increasingly associated with serious conditions such as arrhythmia, myocardial infarction, ischemia, and sudden cardiac death[2]. Treatment aims to reduce heart rate, thereby prolonging the diastolic phase of the cardiac cycle. Surgical intervention becomes necessary when medical therapy fails to control symptoms.

Case Presentation

We present the case of a 40-year-old male referred to us from a tertiary care center with a diagnosed severe left anterior descending (LAD) bridging. The patient had been experiencing atypical chest pain for the past 3 years, which was unrelated to exercise. The pain was localized on the left side of the chest, radiating to the left arm. He had a history of hypertension for the past four years and was initially treated with valsartan. As the chest pain episodes persisted, he was switched to amlodipine by a local practitioner. While his blood pressure was well controlled on amlodipine, there was no improvement in chest pain. Initial investigations, including electrocardiogram (Figure 1), showed no significant ischemic changes. Echocardiogram revealed normal chamber sizes and function without regional wall motion abnormalities.

The patient continued to experience multiple episodes of chest pain daily, both at rest and during exercise. These episodes lasted for several minutes and resolved spontaneously. Seeking further medical advice, he consulted a cardiologist at a tertiary medical center, where he was prescribed glyceryl trinitrate and diltiazem. However, the patient reported no relief from his symptoms. Invasive coronary angiography was subsequently performed, revealing severe LAD bridging (Figure 2 & 3). The patient was informed about the prognosis of the disease and nitrates were discontinued. He was continued on higher doses of diltiazem with close surveillance. However, there was no improvement in symptoms with medical management, leading to the decision for surgical intervention.

Managements & Results

The patient underwent surgical deroofing of the LAD, successfully resecting the muscle bridge from the mid to distal LAD (Figure 4). Subsequent follow-up visits demonstrated complete resolution of symptoms. A follow-up CT angiogram confirmed the patency of the LAD with no overlying muscle fibers (Figure 5). However, the patient later developed atypical localized chest pain, causing increased anxiety. To address his concerns, an invasive coronary angiography was performed, revealing the release of the LAD segment from the myocardium with proper filling (Figure 6). The patient was prescribed analgesics and continued to be followed in the clinic, with complete resolution of pain.

The surgical intervention resulted in the successful resolution of symptoms in the patient. Follow-up evaluations, including echocardiograms and CT angiograms, showed the patency of the LAD without any overlying muscle fibers. The patient demonstrated complete relief from chest pain and continued to be followed in the clinic without any recurrent symptoms.

Please note that the revised section provided above is a suggestion, and you can further modify or adjust it according to your specific requirements.

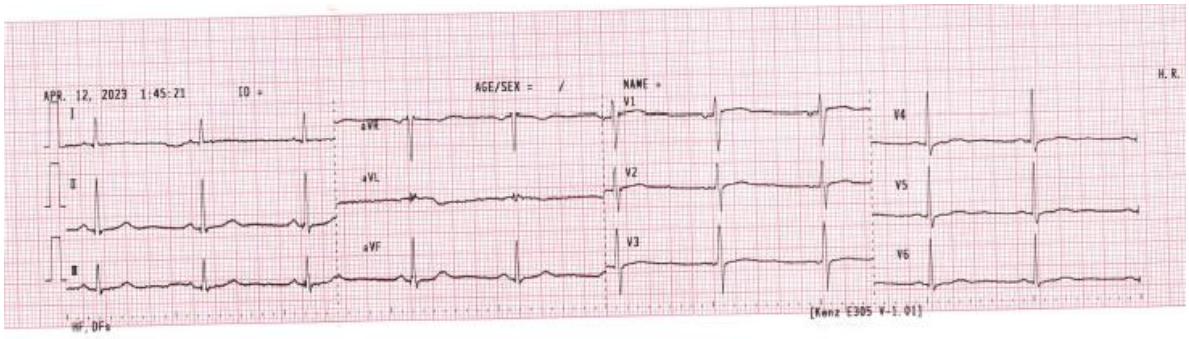

Figure 1: preoperative ECG

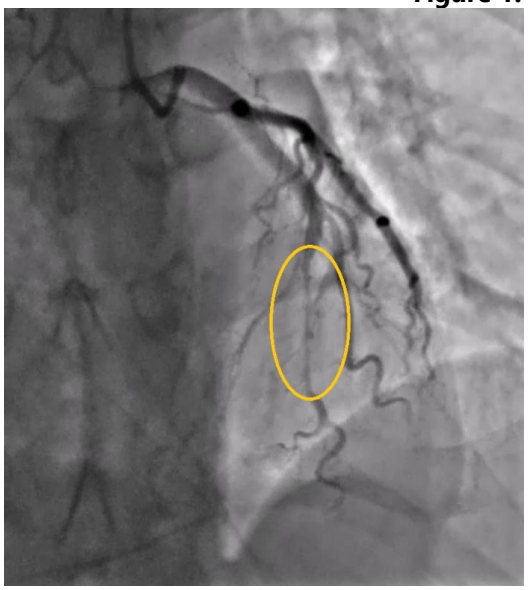

Figure 2: Severe systolic collapse of LAD

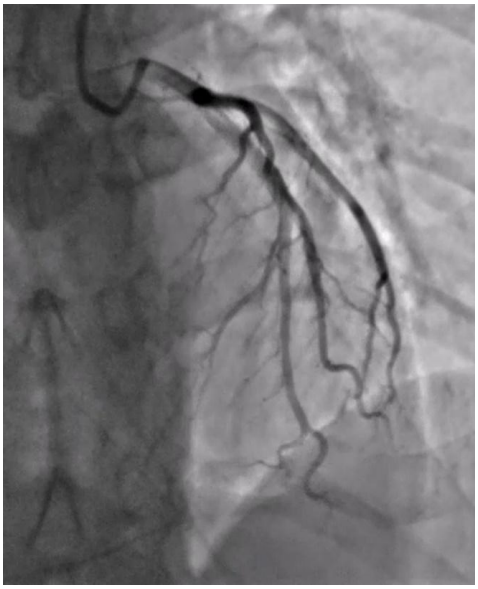

Figure 3: diastolic decompression of LAD

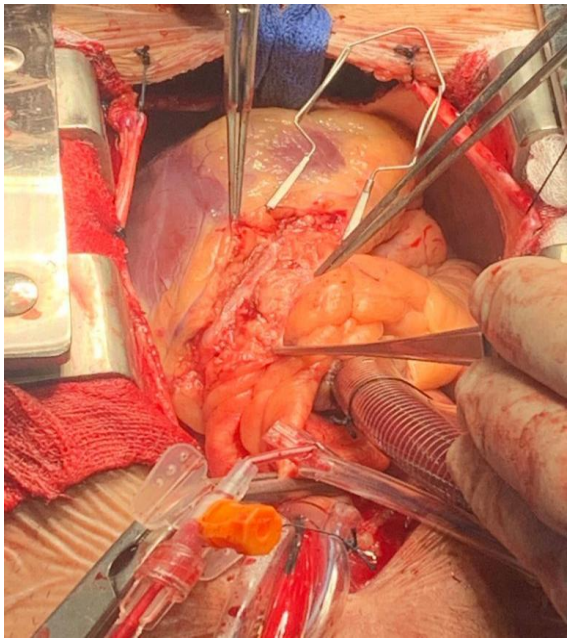

Figure 4: release of left anterior descending artery after resection of muscle bridge

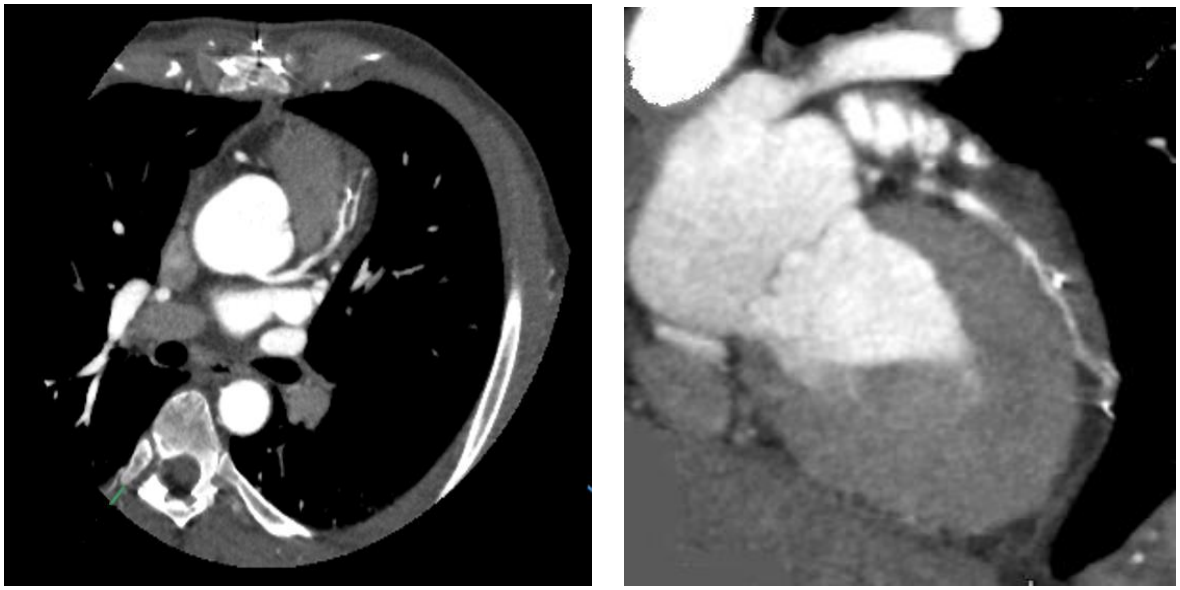

Figure 5(a & b): postopertive CT angiogram showing release of LAD

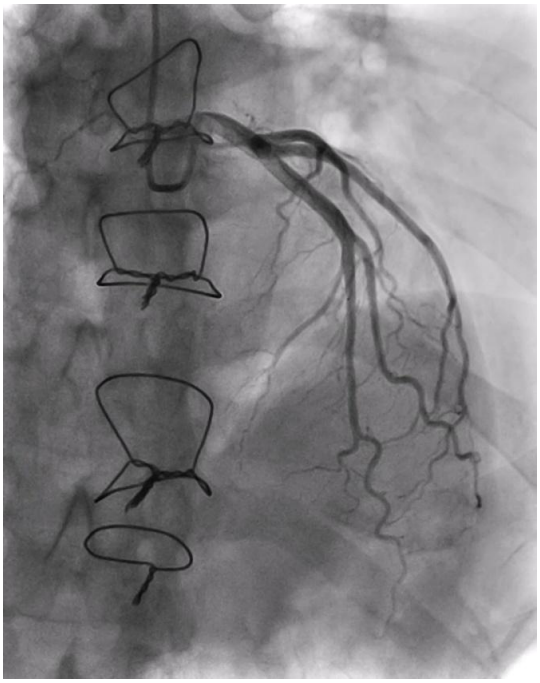

Figure 6: Release of compressed LAD segment after myotomy

Discussion

The prevalence of myocardial bridging varies considerably across different studies, largely influenced by the diagnostic modality employed. A meta-analysis reported an average prevalence of 18.9% using CCTA, which is higher than the prevalence observed with conventional angiography (1.9%)[1]. It is more commonly found in males and predominantly affects the left anterior descending artery. Notably, there is a significant association between myocardial bridging and hypertrophic obstructive cardiomyopathy (HOCM).

While myocardial bridging was previously considered a benign condition, recent studies have demonstrated its association with myocardial ischemia, infarction, coronary spasm, and sudden cardiac death. However, establishing a causal relationship requires further investigation[2]. The compression of the tunnelled segment during systole is responsible for the symptoms associated with myocardial bridging. In states of tachycardia, the shortened diastolic phase exacerbates symptoms as coronary artery filling primarily occurs during diastole. Furthermore, myocardial bridging is highly associated with atherosclerosis, as the turbulent flow across the tunnelled segment leads to endothelial injury and subsequent plaque formation[3]. Symptomatic patients often exhibit longer segments and more severe compression.

There is no single modality considered as the gold standard for diagnosing myocardial bridging. Angiography is the most commonly used imaging tool in patients with chest pain and a high pre-test probability of atherosclerotic disease, with bridging typically being an incidental finding, as observed in our case. Intravascular ultrasound (IVUS) aids in enhancing detection rates and provides better characterization of the tunnelled segment. Intracoronary nitrates during angiography help reveal the tunnelled segment by dilating the normal segments[4].

Cardiac computed tomography angiography (CCTA) is another imaging modality that is more

sensitive in detecting myocardial bridging compared to invasive angiography, with a detection rate of approximately 58%[5]. This may be attributed to the increasing use of CCTA as a diagnostic modality for investigating chest pain. CCTA provides better visualization of the length and depth of the tunnelled segment by demonstrating the bulk of muscle fibers overlaying the artery, which is superior to invasive angiography. The diagnostic accuracy of CT angiography in assessing coronary artery stenosis, both preoperatively and postoperatively, is well-established[6]. Similarly, CT angiography can also be used for preoperative and postoperative assessment of myocardial bridging.

The dynamic nature of myocardial bridging limits the use of tests commonly employed to determine the functional significance of fixed obstructions[2,7]. A small study involving 20 patients with myocardial bridging demonstrated that instantaneous wave-free ratio (iFR) is a superior modality compared to fractional flow reserve (FFR) for assessing the functional significance of myocardial bridging[8]. iFR based on CCTA (iFRCT) has also shown better performance in detecting myocardial ischemia caused by myocardial bridging compared to invasive FFR and CT-based FFR[9]. Myocardial perfusion scans have limited sensitivity in assessing the functional significance of myocardial bridging[10].

The management of myocardial bridging largely depends on the severity of symptoms, functional significance, and associated conditions. Treatment is primarily focused on reducing heart rate (thus prolonging diastole and shortening systole) and contractility. Beta-blockers are the first-line agents, followed by rate-limiting calcium channel blockers, especially when myocardial bridging is associated with coronary spasm[5]. Currently, no randomized controlled trials (RCTs) are available to compare the roles of beta-blockers and calcium channel blockers. Ivabradine, though not specifically studied for myocardial bridging, can be used as an adjunct to beta-blockers and calcium channel blockers, especially when there is associated reduced ejection fraction and symptoms of heart failure11. Aggressive risk factor modification should be considered in these patients due to the inherent propensity of myocardial bridging to promote atherosclerosis, and antiplatelet therapy should be prescribed if plaques are present.

Nitrates are contraindicated in cases of myocardial bridging as they worsen symptoms and angiographically exacerbate systolic narrowing while dilating the proximal segment, thereby increasing retrograde flow[4,5,7]. However, it is not uncommon for patients to use nitrates for the treatment of angina. Another misconception is the association of coronary spasm with myocardial bridging, for which rate-limiting calcium channel blockers (e.g., diltiazem) are the most appropriate option. Nitrates induce reflex tachycardia, which nullifies their antispasmodic properties.

In cases where medical treatment fails to provide relief and persistent symptoms are present, interventional options should be considered[5-7]. Percutaneous coronary intervention (PCI) may alleviate symptoms; however, no study has demonstrated its ischemic benefit[8-12]. Moreover, PCI carries risks such as perforation, stent fracture, stent thrombosis, and in-stent restenosis, limiting its utility. Surgical options include myotomy and coronary artery bypass grafting (CABG). The choice of method depends on the available expertise. Myotomy, though technically challenging and associated with the risk of wall perforation, should be avoided in cases with deep and long segments of the tunnelled artery[13]. However, due to the high rate of graft failure, myotomy should be the preferred procedure in centers with available expertise. When CABG is considered, saphenous vein grafts should be preferred[14].

Conclusion

There are no major guidelines currently available for the diagnosis and treatment of myocardial bridging. Medical therapy should be considered as the first-line treatment, and in refractory cases, surgery is a viable option. Further research is necessary to establish the optimal management approach for myocardial bridging and to clarify its association with ischemic events and adverse cardiovascular outcomes.

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