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Case Report

Myotomy for Severe Left Anterior Descending Myocardial Bridging: A Case Report

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Abstract

Background: Myocardial Bridging (MB) is defined as a segment of a coronary artery that courses within the myocardium, which may result in systolic compression of the tunneled segment and compromise coronary blood flow. The Left Anterior Descending (LAD) artery is the most commonly affected vessel, with a reported incidence of 70-97%. This case report describes the incidental identification of MB in a patient scheduled for cardiac surgery and outlines the subsequent surgical management.

Case Illustration: A 53-year-old male was referred to our center with severe Mitral Regurgitation (MR). He had recurrent hospitalizations for symptoms of heart failure without any complaint of angina. Echocardiography revealed preserved Left Ventricular (LV) ejection fraction and severe MR. A positive ischemic response was observed on treadmill stress testing. Preoperative Coronary Angiography (CAG) demonstrated severe MB in the distal LAD, without evidence of coronary plaque. The patient underwent Mitral Valve (MV) repair with concomitant myotomy, resulting in improved LAD-MB without residual compression. Postoperative CAG confirmed complete resolution of the MB, with no evidence of a milking effect.

Conclusions: The management of MB remains challenging, and treatment strategies should be tailored to individual patient characteristics. Surgical myotomy is a viable treatment option, as demonstrated by the favorable outcome in this case.

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Introduction

Myocardial Bridging (MB) is a congenital condition in which a portion of the epicardial coronary artery is embedded within the myocardium. The muscle overlying the artery is referred to as the myocardial bridge, while the artery embedded within the muscle is termed a tunneled artery.[1] MB most commonly affects the Left Anterior Descending (LAD) artery, although other coronary arteries may also be involved.[2] The pathophysiological mechanisms underlying symptom development in MB remain unclear. However, current literature suggests that symptoms are most likely related to systolic narrowing of the bridged segment.[3] The management of MB presents a clinical challenge, with medical therapy typically considered the first-line treatment.[1] In the presence of concomitant conditions, alternative approaches may be required, and surgical myotomy may be considered in selected cases.[1-2]

We report a case of severe MB in a patient with concomitant severe Mitral Regurgitation (MR), in whom surgical myotomy resulted in successful resolution of the myocardial bridge.

Case Illustration

A 53-year-old male was known to have severe MR based on a preliminary evaluation by the referring physician. He was sent to our hospital for further investigations and management. He presented with a 2-month history of progressive dyspnea and recurrent hospitalizations for acute decompensated heart failure. There was no history of chest pain, stroke, syncope, myocardial infarction, or family history of heart disease. The only significant risk factor of Coronary Artery Disease (CAD) was a history of smoking. Physical examination revealed a grade 3/6 pansystolic murmur at the apex. Electrocardiography demonstrated atrial fibrillation with a normal ventricular response. Echocardiography revealed a preserved Left Ventricular (LV) ejection fraction (64%) and severe MR due to flail of the Posterior Mitral Leaflet (PML).

The patient underwent Coronary Angiography (CAG) to exclude atherosclerotic CAD. The CAG demonstrated markedly severe MB in distal LAD without evidence of atherosclerotic plaque. Pathognomonic signs of a milking effect and a step-

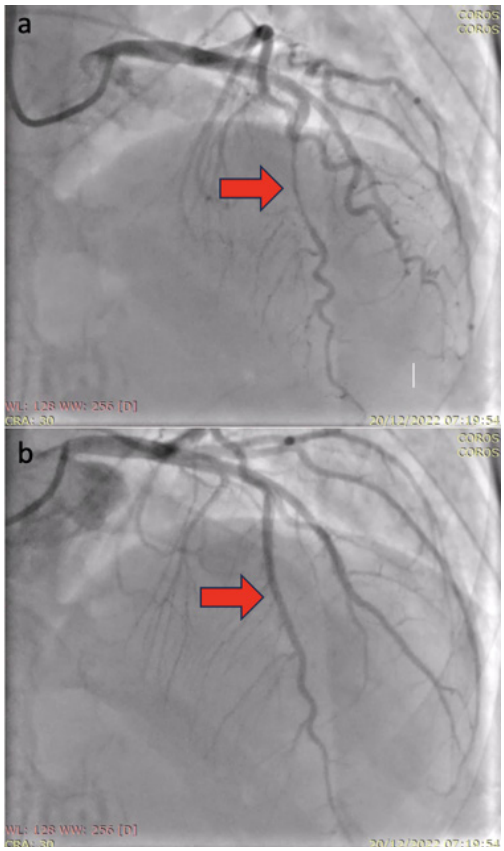

Figure 1. Pre-operative CAG showed a milking phenomenon and step down-step up appearance during systole and diastole in the LAD Coronary Artery. (a) Systolic phase showed severe LAD-MB. (b) Diastolic phase showed complete relief.

down-step-up appearance were prominent during the systolic phase (Figure 1). A Treadmill Stress Test (TMT) was also performed and demonstrated a positive ischemic response. Accordingly, he was prepared for elective Mitral Valve (MV) repair with a concomitant myotomy procedure. During surgery, the tunneled segment of the LAD artery was unroofed from the overlying myocardial bridge by sharp dissection until the anterior wall of the artery was fully exposed (Figure 2). MV repair was completed during the same procedure. The postoperative course was uneventful, and the patient was discharged after 6 days of hospitalization. The cardiac rhythm converted to a sinus rhythm. Six months after surgery, postoperative CAG demonstrated no residual systolic narrowing of the LAD (Figure 3). The patient was scheduled for an annual routine check-up at the hospital. He is currently in good functional capacity and exercises regularly without limitation. His cardiac rhythm has remained sinus during long-term follow-up. Echocardiographic evaluation revealed preserved LV and Right Ventricular (RV) function with no residual MR.

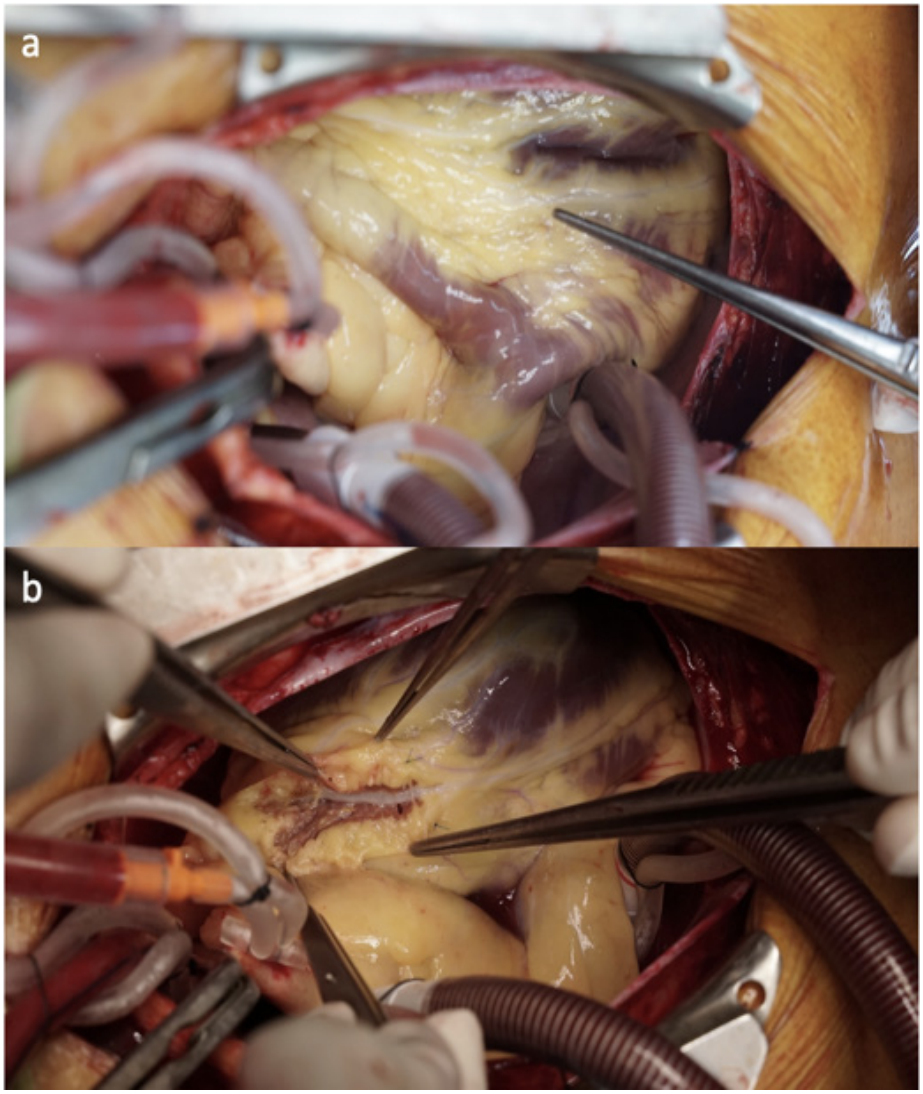

Figure 2. Intra-operative findings showed LAD-MB tunneled into the myocardium. (a) Before myotomy, (b) After myotomy.

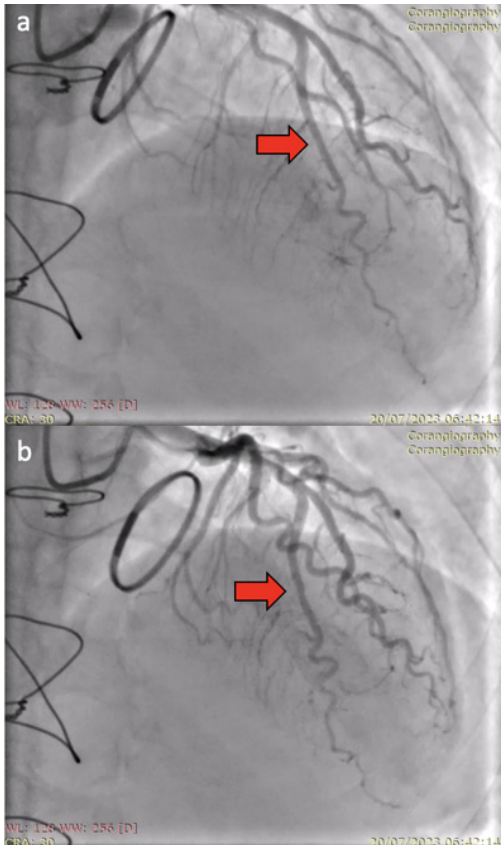

Figure 3. Post-operative CAG showed no MB.
(a) Systolic phase, (b) Diastolic phase.

Discussion

The reported prevalence of MB varies widely depending on the diagnostic modality used. Given the variety of diagnostic methods available, a single prevalence estimate is difficult to determine. The most commonly involved artery is the LAD.[2] By invasive CAG, MB has been reported in approximately 0.5-12%.[4] Other studies have reported higher prevalence rates using Coronary Computed Tomography Angiography (CCTA), ranging from approximately 19–22%.[5] Nevertheless, necropsy and autopsy findings are considered the gold standard for identification of MB, and around 33-42% were reported.[4] In our case, CAG was performed to exclude atherosclerotic CAD in a patient with severe MR. Interestingly, a markedly severe MB was identified in the distal LAD. No additional diagnostic modalities (intracoronary imaging tools) were considered, as the presence of MB was not anticipated at the outset of the patient's evaluation.

Anatomically, the MB is characterized by a segment of a coronary artery coursing beneath the myocardium. The depth of the tunneled artery correlates with the degree of systolic compression and the delayed increase in luminal diameter during diastole.[2] Based on depth, MB can be classified as superficial, deep, or very deep. These categories are defined by an overlying myocardial thickness of 1–2 mm, >2 mm, and >5 mm, respectively.[2,6] Because myocardial perfusion occurs predominantly during diastole (approximately 85%), only a minority of coronary blood flow is affected by systolic compression in MB.[1] Frequently, patients with MB are asymptomatic. The location, depth, length, and degree of systolic compression are key determinants of the clinical presentation of MB. Additional factors associated with symptomatic MB include atherosclerotic coronary disease, concomitant vasospasm, increased heart rate, low systemic arterial pressure, and other comorbid conditions.[1] These mechanisms contribute to reduced myocardial perfusion through shortening of diastolic perfusion time and increased epicardial vasoconstriction.[7-8] Accordingly, patients with symptomatic MB may present with ischemic features.[1] In our patient, treadmill stress testing demonstrated a positive ischemic response, initially

raising concern for ischemic heart disease. However, the patient did not report anginal symptoms. In the context of volume overload in severe MR, the observed ischemic response on TMT may represent a false-positive finding for CAD, attributable to ventricular strain and supply/demand mismatch. Consequently, the positive TMT result in this patient is likely a secondary manifestation of the underlying valvular pathology and LV strain, rather than indicative of primary CAD. In the absence of anginal symptoms, the valvular pathology provides a sufficient explanation for the patient's symptoms of dyspnea and heart failure.

Several diagnostic modalities have been commonly used to assess the morphological and functional significance of MB. CCTA is associated with high spatial and contrast resolution, allowing detailed visualization of the coronary lumen, vessel wall, and surrounding myocardium. This enables accurate characterization of MB, particularly the depth of the tunneled artery.[9] Preoperative anatomical assessment of MB is highly recommended to inform surgical planning. However, in the present case, CCTA was not performed due to financial constraints, precluding comprehensive preoperative anatomical evaluation of the MB. On CAG, a key diagnostic feature of MB is systolic compression of the vessel, referred to as the "milking effect". A significant milking phenomenon is defined as a ≥70% reduction in the minimal luminal diameter during systole, with a persistent ≥35% narrowing during mid-to-late diastole.[1-2] Another angiographic feature is the "step-up–step-down" appearance, reflecting complete or partial decompression of the affected segment during diastole.[10] Consistent with the literature, these pathognomonic findings were observed on CAG in our patient (Figure 1).

Considerable variability in clinical presentation and concomitant conditions may independently influence the management of patients with MB.[1] Accordingly, no single standardized treatment approach exists.[8] Available management options include pharmacological therapy, Percutaneous Coronary Intervention (PCI), and surgical treatment.[1-2] Once the diagnosis is made, pharmacological treatment should be attempted first. Pharmacological therapy using negative inotropic and chronotropic agents, such as β-blockers and calcium-channel blockers, is considered first-line treatment.[1,8] These agents act by reducing sympathetic drive and mitigating hemodynamic disturbances and vasospasm. If symptoms persist despite medical therapy, alternative treatment options may be considered.[11] Another option is stenting of the bridged segment, which may reduce systolic compression and increase diastolic luminal diameter. However, a major limitation is the reported failure rate, which approaches 50%.[12] Given the patient's requirement for surgical intervention for severe MR, a concomitant surgical approach for the MB was deemed the most pragmatic and efficient strategy, thereby obviating the need for a trial of medical therapy. This approach allowed simultaneous correction of both the valvular pathology and the MB, optimizing overall cardiac function and minimizing potential future risks.

Current evidence suggests that surgery is the treatment of choice for patients with refractory MB-related symptoms.[2] Surgical approaches may include myotomy, Coronary Artery Bypass Surgery (CABG), or a combination of both.[1] The cardiopulmonary bypass machine can be on or off during the procedure. Myotomy refers to the resection of the overlying myocardial fibers to relieve compression of the tunneled artery. The artery is identified distal to the intramyocardial segment and carefully followed proximally, with unroofing performed for at least 1 cm on either side. Isolated myotomy is favored in the absence of proximal atherosclerotic lesions or excessive arterial tortuosity, in younger patients, and is best performed at high-volume centers.[2] Potential complications of myotomy include coronary wall perforation, ventricular aneurysm formation, and postoperative bleeding.[13] Alternatively, bypass surgery may be performed alone or in combination with myotomy. CABG is recommended in cases of extensive (>25 mm) or deep (>5 mm) MB, high risk of scarring after myotomy, proximal atherosclerotic disease, or when the diastolic caliber of the LAD is smaller than the proximal segment.[14] Most commonly, LAD-MB is bypassed using the left internal mammary artery.[2] In a cohort study by Hemmati et al, 71 subjects with isolated MB who underwent myotomy resulted in a successful outcome. There were no cardiac deaths, and 63% of patients reported no further symptoms during postoperative follow-up.[13] However, to the best of our knowledge, no studies have reported the incidence of MB in patients presenting with heart failure symptoms. Therefore, limitations to our findings should be acknowledged. At present, there are no established guidelines for the management of patients with concomitant severe MR and MB. Ultimately, myotomy was deemed the optimal surgical approach for this patient, given the superficial intramyocardial course of the LAD-MB,

concomitant valvular disease, and the institutional expertise in cardiac surgery. The procedure was successfully performed, and the postoperative course was uneventful. Follow-up CAG at 6 months demonstrated resolution of the MB, and the patient has remained asymptomatic. Comprehensive echocardiographic evaluation revealed no residual MR (MV area by velocity time integral 4.25 cm^2, mean MV gradient 1.31 mmHg) and a preserved LV ejection fraction of 70%. Consequently, no additional diagnostic or therapeutic interventions are required beyond ongoing pharmacological treatment.

Conclusion

Consideration of optimal LAD-MB treatment is necessary in clinical practice. Thorough identification and assessment to determine the anatomic characteristics of MB are also necessary before deciding the best management. Myotomy is a safe technique for favorable MB patients with severe MR without atherosclerotic lesions.

List of Abbreviations

CAD	Coronary Artery Disease
CAG	Coronary Angiography
CABG	Coronary Artery Bypass Surgery
CCTA	Coronary Computed Tomography Angiography
LAD	Left Anterior Descending
LV	Left Ventricular
MB	Myocardial Bridging
MR	Mitral Regurgitation
MV	Mitral Valve
PCI	Percutaneous Coronary Intervention
PML	Posterior Mitral Leaflet
RV	Right Ventricular
TMT	Treadmill Stress Test

Ethical Clearance

A case report is exempt from ethical approval in our institution.

Publication Approval

All authors consent to the publication of this manuscript.

Authors' Contributions

Conception and the framework of the manuscript were initiated by TMHP, WAW, ME, and AK. The manuscript was drafted by TMHP and ME. AK, WAW, JT, and ID critically revised the manuscript. All authors contributed to the article and approved the submitted version.

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Conflict of Interest

All authors declare no conflicts of interest.

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