Myocardial Bridge Complicated by Acute Myocardial Infarction

Guram G. Imnadze, MD, PhD,[1,2] Andro G. Kacharava, MD, PhD[3,4]
[1]Department of Cardiology, Schuechtermann Clinic, Bad Rothenfelde, Germany; [2]University Witten/Herdecke, Witten, Germany; [3]Department of Medicine, Division of Cardiology, Emory University School of Medicine, Atlanta, GA; [4]Division of Cardiology, Atlanta Veterans Affairs Medical Center, Decatur, GA

Typically, myocardial bridging (MB) is considered a relatively benign condition; however, serious complications such as angina pectoris, myocardial infarction (MI), and sudden cardiac death may occur. The diagnosis and appropriate treatment of this pathology are important. We report a case of acute anterior wall ST-elevation MI occurring as a complication of MB involving the mid segment of the left anterior descending artery in a young, otherwise healthy woman who underwent a primary stenting procedure. Pathophysiologic mechanisms underlying the process leading from MB to acute MI vary, and so should the therapy for those patients. Coronary angiography, intravascular ultrasound, and, potentially, optical coherence tomography should be used to assist in establishing an accurate diagnosis in these complex patients and should guide the therapeutic decision in acute settings.

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KEY WORDS

Myocardial bridge • Myocardial infarction • Sudden cardiac death • ST-elevation myocardial infarction

Upon review, in 15% to 80% of autopsy cases, one or more large epicardial coronary arteries had a congenitally anomalous course, with the varying (10-50 mm) arterial segment passing intramurally before reappearing on an epicardial surface.[1-3] The myocardium overlying such a tunneled arterial segment is called the myocardial bridge (MB). Despite the fact that the MB can be observed over any epicardial artery, it is generally confined to the mid segment of the

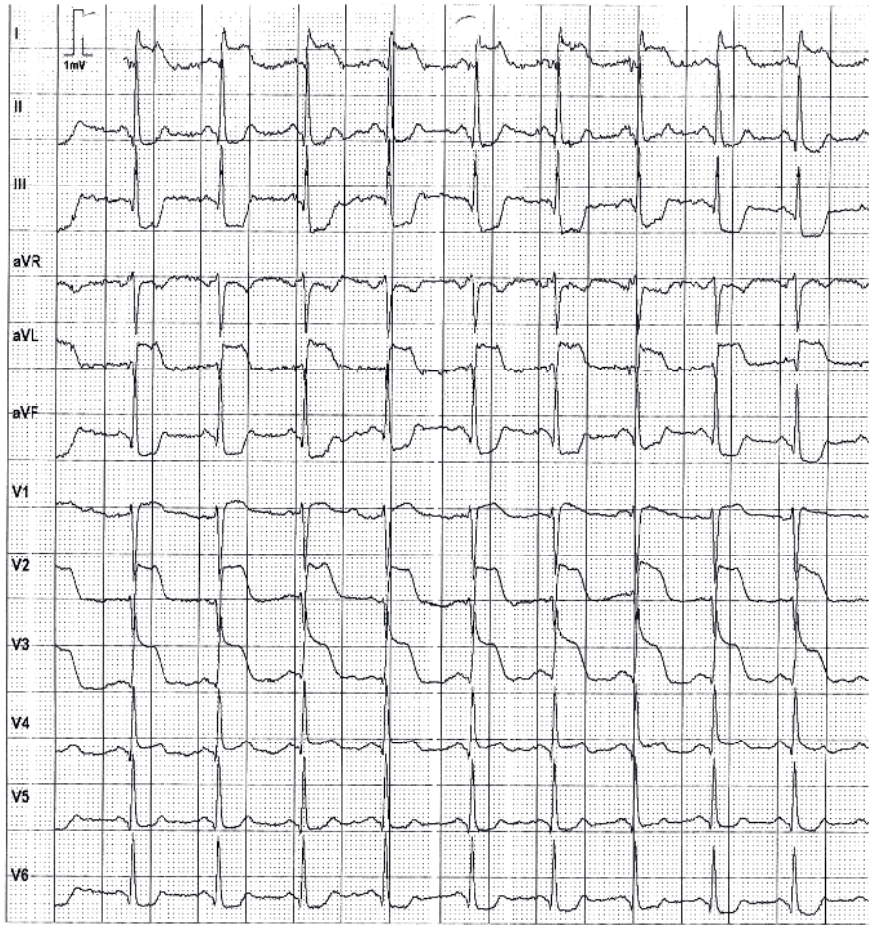
left anterior descending coronary artery (LAD).[4] The MB first was discovered in an autopsy by Reyman almost 300 years ago.[5] Geiringer, in his paper from 1951, provided an in depth review of the subject[6]; subsequently Portmann and Iwig, in 1960, provided the first angiographic images of systolic compression of the affected coronary artery.[7] Although MB is considered a relatively benign condition, serious complications such as angina pectoris,[8] myocardial infarction (MI),[9] and sudden cardiac death[10] have been reported. Therefore, the early diagnosis and treatment of this pathology are important. We present a case of an acute anterior wall ST-elevation MI (STEMI) occurring as a complication of MB involving the mid segment of the LAD in a young, otherwise healthy woman.

Case Report

A 45-year-old healthy woman came to the emergency department complaining of severe precordial chest pain for the past 2 hours, associated with diaphoresis and weakness. Cardiac examination revealed an S4 gallop. Past medical and family histories were unremarkable. The patient was a smoker with 20 pack/year history. She denied any medication or drug use. Chest radiograph results were normal. Electrocardiogram (ECG) revealed normal sinus rhythm with ST elevation in leads I, aVL, and V2 to V4, with reciprocal ST depression in leads II, III, aVF, and V6 (Figure 1). On admission, blood tests revealed elevated serum levels of troponin I (1.8 ng/mL; normal = 0.0-0.028 ng/mL) and creatine kinase MB (56.0 U/L; normal = 0-24 U/L). Transthoracic echocardiography (TTE) revealed moderate to severe hypokinesis of the apical region, and left ventricular ejection fraction (LVEF) was estimated to be 40% to 45%. The clinical presentation, biochemical markers, ECG, and echocardiographic changes were indicative of STEMI with the LAD as a culprit. She was treated with oral aspirin, clopidogrel, intravenous nitroglycerin, and heparin, and transported to the cardiac catheterization laboratory.

Coronary angiography revealed MB over the mid LAD with a thrombus at the proximal site of the arterial segment covered by MB (Figure 2A and Figure 2B). No other changes were noted. The thrombus was successfully aspirated using an aspiration catheter (Figure 2C and Figure 2D). The cine angiogram obtained after thrombus aspiration revealed severe (> 90% in diameter) systolic compression of the mid LAD (Figure 2C and Figure 2D). Due to continuous chest pain, a decision was made to stent the site with a drug-eluting stent (DES) (Figure 2E). Chest pain resolved with the stenting and the ST segment returned toward baseline. No peri- or postprocedural complications occurred. The patient did well during the hospitalization and, 3 days after the procedure, was discharged to a rehabilitation facility. Three weeks after the hospitalization, repeat TTE showed mild apical hypokinesis and an LVEF of 55%; 12 months postprocedure, the patient was free of symptoms. The results of 12-lead ECG, TTE, and an exercise stress test all were normal.

Figure 1. 12-lead electrocardiogram of a patient with myocardial bridging on admission.



Discussion

The diagnosis of MB made by coronary angiography ranges from

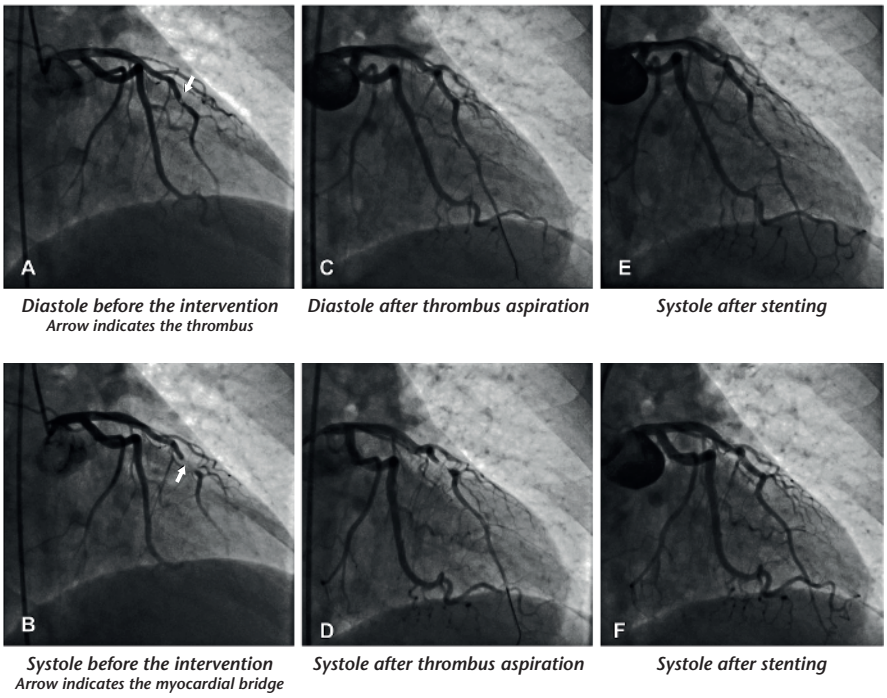


Figure 2. Coronary angiography of the left coronary artery in right anterior oblique projection. LCA with clot at the bridged segment (*arrow*) of the mid LAD (**A, B**). LCA after the aspiration of the clot from the LAD (**C, D**). LCA after the stent placement in mid LAD (**E, F**). LAD, left anterior descending artery; LCA, left coronary artery.

0.5% to 16%,[1-3] and can be classified as superficial or deep based on the depth and anatomic type of the myocardial fibers.[4,11] The superficial type, which represents the majority of MB cases, consists of fibers running over the coronary artery perpendicular to or in acute angle toward the cardiac longitudinal axis. Deep MBs consist of myocardial fibers encircling coronary arteries. MI can be defined as necrosis of a region of the myocardium caused mainly by an acute insufficient supply of blood to the heart muscle. There are myriad causes[12,13] that can lead to development of MI, but the majority of those causes are related to atherosclerotic plaque rupture, fissure, or erosion with occlusive thrombus formation.[13] Ramos and colleagues described ruptured atherosclerotic plaque with associated occlusive thrombus located at the entrance of the coronary vessel into the MB.[14]

The association between MB and MI has been described previously.[9] Relatively less has been written on the underlying mechanisms of a possible connection between those two entities.[15] Several pathology and intravenous ultrasound (IVUS)[16,17] studies have been conducted confirming the initial observation[6] that, despite presence of endothelial dysfunction within the bridged segment,[18] the intramural segments and distal edges of a coronary artery covered by an MB are, in most cases, free from significant atherosclerosis. This protective effect can be at least partially explained by the presence of high or normal wall sheer stress and sheer stress rate within the MB-covered segment.[19] Conversely, a high incidence of coronary atherosclerotic plaque was found within the 20-mm segment of the artery from its entrance to the MB.[20] Low and oscillatory wall sheer stress has been proposed as a cause of the development of a proatherogenic phenotype of endothelial cells covering the vessel wall in those proximal to MB vascular segments.[21] It has been well appreciated that the segments of arterial tree exposed to low sheer stress are predisposed to atherosclerotic plaque generation and its rapid progression.[22] The other potential protective cause of the intramural segment of the coronary artery may be its separation from the proinflammatory milieu of the perivascular epicardial adipose tissue.[23] Although the aforementioned factors most likely play a role, they cannot explain the absence of atherosclerosis in the distal to MB segment of the vessel. The factors protecting the distal segment from plaque formation appear to be complex and require further investigation.

Deep MBs consist of myocardial fibers encircling coronary arteries.

The rupture/fissuring of an atherosclerotic plaque located in a proximal coronary segment is not the only described cause of acute MI in the presence of MB. Prolonged focal coronary spasm[24] secondary to underlying endothelial dysfunction,[18] with or without subsequent thrombus formation,[1] spontaneous coronary dissection,[25] and severe and long-lasting systolic compression extending and affecting diastolic relaxation with residual diastolic narrowing of the vessel[26] all have been associated with MI in the presence of MB. Hemodynamic stability in patients with acute inferior wall MI may potentially be jeopardized by the MB over the LAD.[27] The possible explanation for this can be an acute hyperadrenergic state during STEMI; this may lead to a compensatory increase in contractility of an anterior wall, including the MB segment, with subsequent

severe and long-lasting systolic compression of the LAD extending into a diastolic phase, causing an ischemia of the anterior wall.[27]

The different pathophysiologic mechanisms leading to an acute MI, or to a hemodynamic deterioration in the presence of acute MI, require timely diagnosis and differential therapeutic approach to patients with MB. Patients with STEMI due to plaque rupture with focal thrombosis,[14] and patients with spontaneous coronary artery dissection at the MB site,[25] are best served with immediate percutaneous or surgical revascularization. Similarly, urgent revascularization of the MI culprit vessel,[27] not affected by MB, may unload MB-contained myocardial segment and resolve MB-related vessel compression.[27] On the other hand, in patients in whom the cause of MI is a focal spasm at the MB site, or a severe and long-lasting systolic compression without significant plaque burden, medical therapy may be tried initially,[28,29] and stenting considered as a rescue treatment. Nitrates should be avoided in cases in which systolic or systole-diastolic compression of the vessel leads to ischemia, because they cause reduction of the intrinsic coronary wall tension, augment the MB contractility due to reflex sympathetic stimulation, and potentially provoke a horizontal "steal" effect by dilating other epicardial coronary vessels.[3]

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The decision regarding what type of stent to use in acute MI, and how to position it in a patient's vessel with MB, is still not clear. With regard to stenting, most of the currently available elective percutaneous intervention (PCI) studies have concluded that bare-metal stents have higher rates of target vessel revascularization (TVR) than DES[30]; and DES restenosis rates are still higher compared with restenosis rates in standard atherosclerotic lesions not associated with MB.[30] Stent fracture in DES[31-33] is likely due to metal fatigue from repetitive stress from myocardial contractions of MB fibers, which may be a major contributive factor to increased rates of restenosis and TVR. The other issue is stent positioning. According to a small study conducted on 70 patients with MB who had PCI predominantly with DES, TVR at 1-year follow-up was 24% among patients who had stents extended into the segment containing the MB, in comparison with 3% among patients whose stents covered precisely the epicardial obstructive lesion.[34] In light of those results, the role of IVUS in guiding accurate positioning of the stent during PCI may be critical in decreasing future restenosis and TVR.

In order to assess the relationship between MB and major adverse cardiac events, a meta-analysis of the available prognostic studies was conducted.[35] The study has suggested that the relevance between MB and MI appears to be real, but it was unable to confirm that the presence of MB is an independent risk factor associated with MI. According to the study, further research is required to determine the anatomic and functional characteristics of MB associated with MI.

Conclusions

MB is a well-known congenital condition that can be diagnosed on coronary angiography and on autopsy. Generally considered a benign anomaly, it can present as an acute coronary syndrome, as it did in our patient. Pathophysiologic mechanisms underlying the process leading from MB to acute MI vary. Coronary angiography, IVUS, and optical coherence tomography,[36] if available, assist in establishing a diagnosis and should guide the therapeutic decision in complex patients. Additional studies are required in order to optimally select the specific diagnostic and therapeutic approaches in patients with MB complicated by acute MI. ■

The authors declare no real or apparent conflicts of interest. The patient signed an informed consent agreement.

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MAIN POINTS

- Myocardial bridging (MB) is considered a relatively benign condition; however, serious complications such as angina pectoris, myocardial infarction (MI), and sudden cardiac death may occur.
- Superficial MBs, which represents the majority of MB cases, consists of fibers running over the coronary artery perpendicular to or in acute angle toward the cardiac longitudinal axis. Deep MBs consist of myocardial fibers encircling coronary arteries.
- Coronary angiography, intravascular ultrasound, and, potentially, optical coherence tomography should be used to assist in establishing an accurate diagnosis.
- The different pathophysiologic mechanisms leading to an acute MI, or to a hemodynamic deterioration in the presence of acute MI, require timely diagnosis and a differential therapeutic approach to patients with MB. Patients with ST-elevation MI due to plaque rupture with focal thrombosis, and patients with spontaneous coronary artery dissection at the MB site, are best served with immediate percutaneous or surgical revascularization.