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ACUTE MYOCARDIAL INFARCTION DUE TO MYOCARDIAL BRIDGE TREATED WITH SURGERY: A CASE REPORT

Myocardial bridging is congenital anomaly characterized by segment of epicardial coronary arteries passing through the myocardium. Various ischemic conditions are related with this pathology. We report a case of myocardial bridging that was complicated with acute anterior myocardial infarction and a review of the literature. The patient was treated successfully with coronary bypass graft surgery after unsuccessful percutaneous intervention.

Keywords Myocardial bridge; myocardial infarction; coronary; surgery

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Introduction

Myocardial bridging (MB) is a congenital variant in which a portion of an epicardial coronary artery, most frequently the middle segment of the left anterior descending (LAD) artery, takes an intramuscular course [1]. This arrangement of a “tunneled” segment of the artery under the “bridge” of overlying myocardium frequently results in compression of the artery during systole [2]. Myocardial ischemia due to bridging has been reported in the literature. The proximal part of the artery under the myocardial bridge is most susceptible to atherosclerosis. However, in this case, acute myocardial infarction and total occlusion of the artery under the myocardial bridge was present and could not be treated percutaneously due to the deep location and tortuosity. The patient underwent successful coronary artery bypass grafting (CABG).

Case Presentation

A 52-year-old female patient was admitted to the emergency department with chest pain radiating to the left shoulder and persisting for one hour. She had no history of coronary artery disease, hypertension, diabetes, dyslipidemia, or family history of coronary artery disease. She was an active smoker. The physical examination revealed no remarkable findings except blood pressure was of 150/80 mmHg. The ECG showed ST elevation in leads V₁–V₆, which was consistent with acute anterior myocardial infarction (Figure 1). The transthoracic echocardiogram showed mild impairment of left ventricular systolic function with antero-apical segmental hypokinesia and no visible thrombus.

The patient was immediately administered 300 mg acetylsalicylic acid and 180 mg ticagrelor. Urgent coronary angiography performed via the right femoral artery with guiding catheter 6F EBU3.5 (Medtronic, Santa Rosa, USA) revealed total occlusion of the LAD. The mid-part of the LAD was totally occluded, and atherosclerotic changes were found in the proximal part of the lesion (Figure 2).

First, a 0.014-inch Balance Middle Weight guidewire (BMW) (Abbott, Minnesota, USA) was advanced in a failed attempt to pass the occlusion. A 1.5×15 mm balloon (Invader, Alvimedica, Nanterre, France) was inserted to increase support, however, it was not possible to pass to the distal LAD. The guidewire and balloon were withdrawn, and the BMW was replaced with a PILOT 50 guidewire (Abbott, Minnesota, USA). This guidewire was successfully advanced to the distal portion of the LAD (Figure 3) along with the same 1.5×15 mm balloon. However, the balloon was not passed pass through the lesion.

Figure 1. ECG showing segment elevation in chest leads

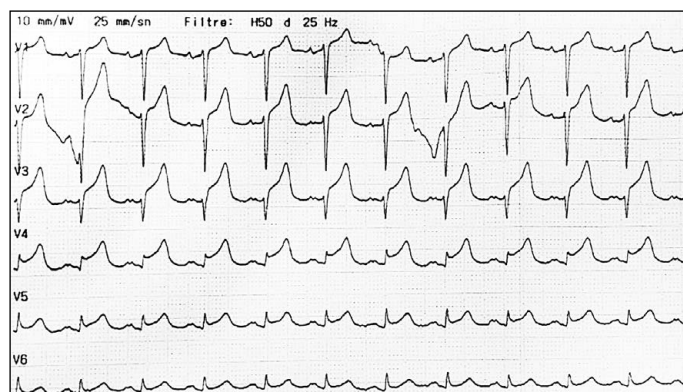
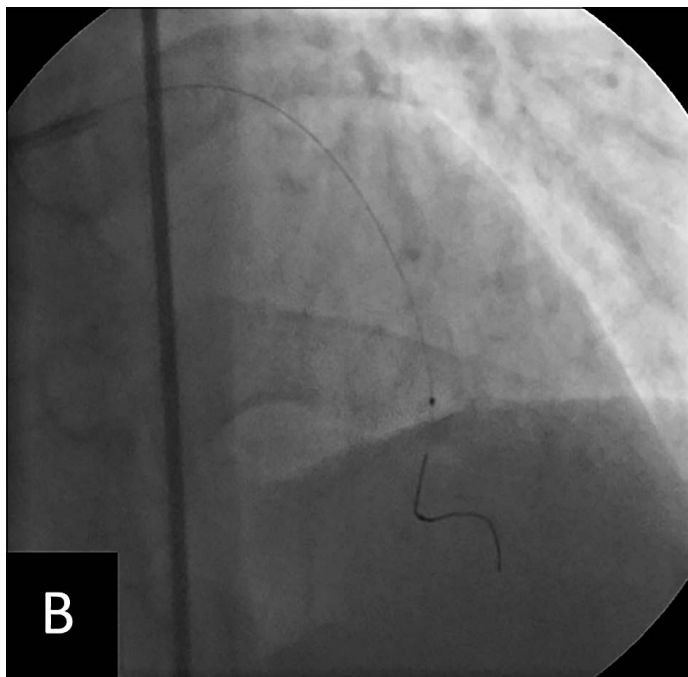
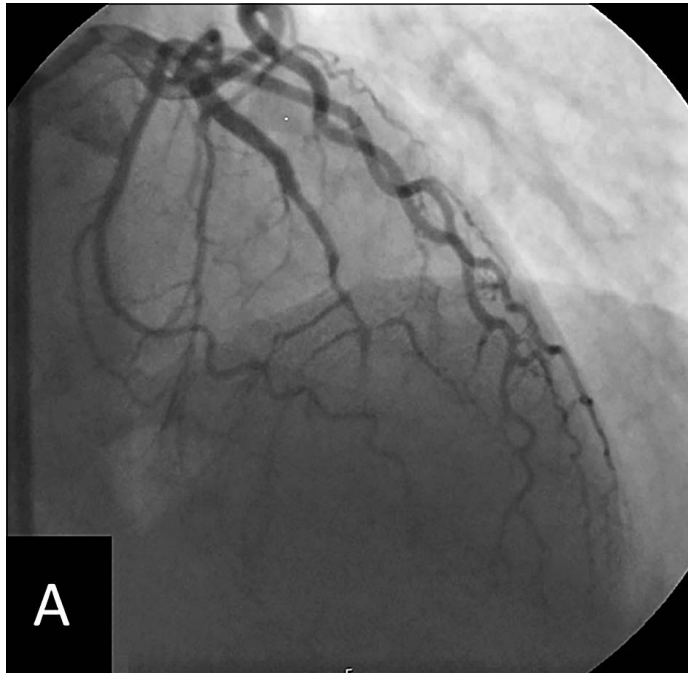


Figure 2. A. Coronary angiography showing total occlusion of the LAD. **B.** Coronary angiography showing the intramyocardial course of the LAD during partial wiring with a BMW guidewire and unsuccessful balloon advancement



Subsequently, to provide blood flow, dilatation was performed just proximal to the obstructed segment. There was thrombolysis in the region of myocardial infarction, and grade III flow after balloon inflation. It was seen that the main lesion located inside the myocardium was compressed during every systolic contraction. Due to the plaque rupture, deep myocardial bridge, and tortuosity of the artery, it was decided to perform emergency CABG. A left internal mammary artery-LAD bypass graft was performed after bridging segment.

During the surgery, the intramyocardial course of the LAD and small bleeding foci around the vessel were seen, possibly due to guidewire advancement in the lesion area (Figure 4). The patient was discharged five days following surgery with no problems.

Discussion

The association between MB and myocardial infarction is usually related to myocardial contraction over the bridging segment or to atherosclerotic changes proximal to bridge. This case was unique due to the anatomic

Figure 3. A. Coronary angiography showing successful wiring with PILOT 50 guidewire and kinking of the guidewire and its intramyocardial course. **B.** TIMI -III flow after percutaneous intervention

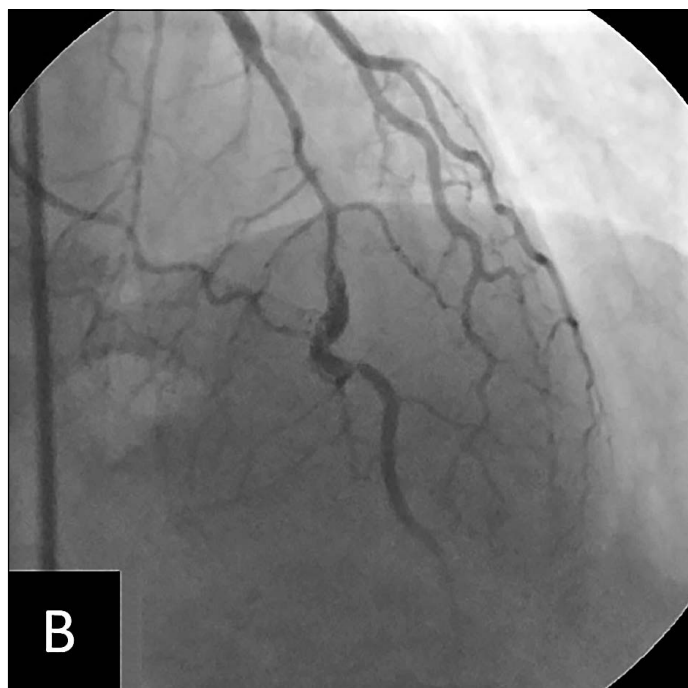
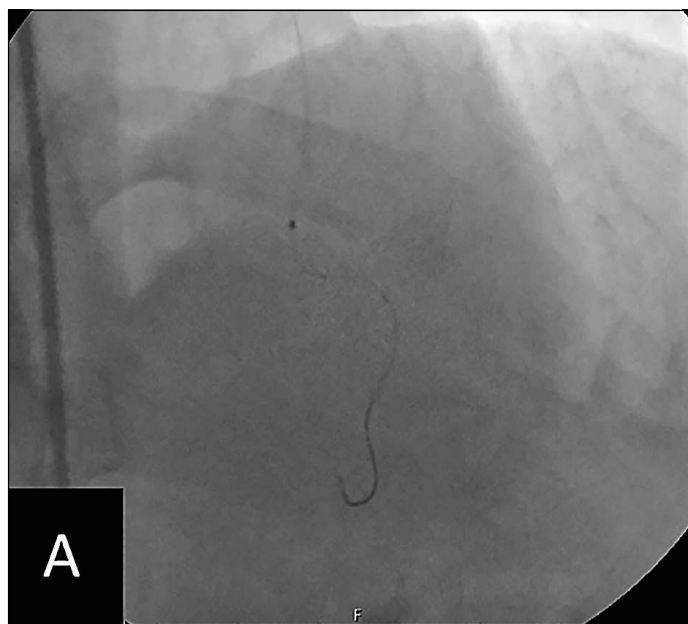
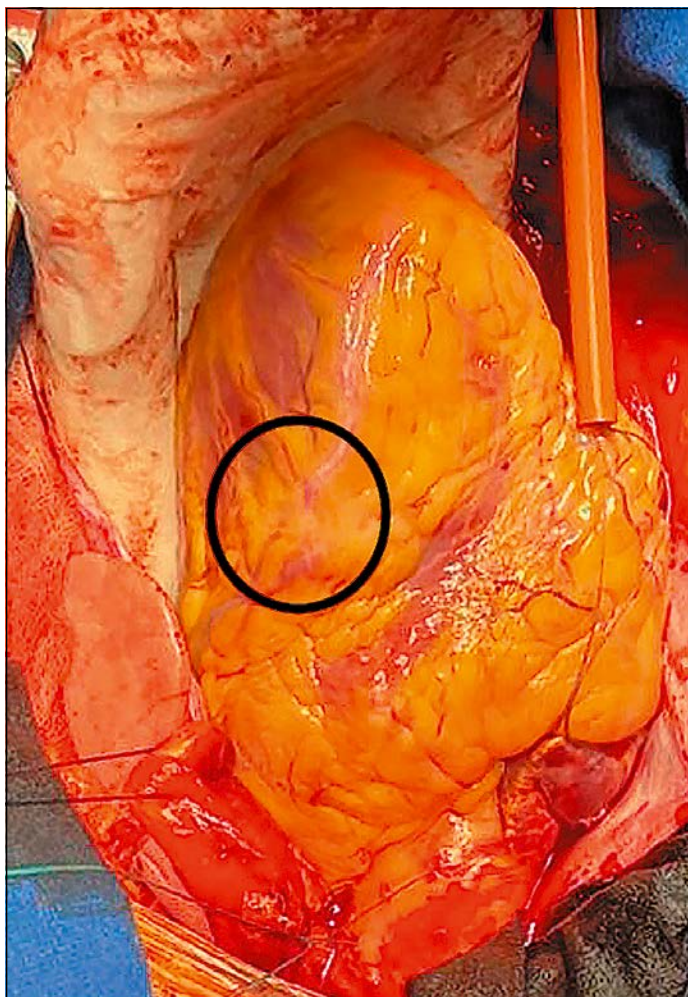


Figure 4. The deep, intramyocardial course of the LAD and small bleeding foci around the vessel (circle) as seen during surgery



location, challenges for percutaneous intervention, and, finally, recovery following emergency surgery.

MB is a common congenital coronary anomaly that was detected in 1.5–16% of cases during coronary angiography and up to 80% in some necropsy series [3–5]. The pathophysiology of MB is insufficiently understood. The proximal part of the bridging segment tends to be associated with atherosclerosis rather than the MB segment itself [6]. Hemodynamic and structural changes, such as blood flow disturbance, elastic damages, and deposition of lipids and mucopolysaccharides could be responsible for the atherosclerotic changes [7]. In the present case, we observed atherosclerotic changes proximal to the bridging segment, however, the total occlusion was within the myocardium. This was an unusual presentation for MB. There may have been spontaneous coronary dissection in this case since the bridging area does not generally include atherosclerotic stenosis or thrombus.

Coronary cineangiography remains the most common technique for diagnosing MB. The typical depiction of bridging on angiography involves a systolic narrowing, or “milking”, of an epicardial artery, with a “step-down” and

“step-up” demarcating the impacted area. Newer diagnostic modalities such as coronary computed tomographic angiography (CCTA) [8], intravascular ultrasound (IVUS) [9], intracoronary doppler [10], and fractional flow reserve (FFR) [11] have enabled more complete analysis of the anatomic and hemodynamic consequences of the systolic compression, including pathological effects on coronary flow.

Based on angiographic findings, the MB has long been considered benign, and patients with a LAD MB were reported to have a good long-term prognosis [12]. In contrast, the potential hemodynamic significance of a MB has been noted [13]. Some studies, usually case reports, have implied a possible correlation between a MB and various cardiovascular pathologies, including acute myocardial infarction [14, 15], ventricular rupture [16], life-threatening arrhythmias [17], hypertrophic cardiomyopathy [18], takotsubo cardiomyopathy [19], and sudden death [20].

Zhou et al. reported a patient with MB who was admitted with myocardial infarction. However, there were no atherosclerotic changes or thrombus in the bridging segment [14]. In another study reported by Akdemir et al., a patient with coronary thrombus and MB was followed conservatively [15]. Apart from these cases, atherosclerotic changes and total occlusion have been found before the bridging segment.

The first step in the treatment of MB consists of pharmacological therapy [21]. First-line therapy for patients thought to be experiencing symptoms secondary to MB consists of beta-blockers [22] and non-dihydropyridine calcium-channel blockers (CCB) [23]. These drugs lengthen diastole and, by their negative inotropic effects, increase coronary perfusion. CCB are used mostly when MB is associated with coronary vasospasm [14]. However, percutaneous coronary intervention (PCI), myotomy, or CABG might be considered for selected patients refractory to medical therapy.

Stent implantation can be option for symptoms resistant to pharmacological therapy. However, concerns regarding perforation during stent deployment [24], stent fracture [25], in-stent restenosis [26, 27], and stent thrombosis [28] have limited stent use in this condition. Tandar et al. reported two cases of stent fracture following stenting of a myocardial bridge [25]. In one patient, they decided to proceed with surgery, and in the second patient, they employed a second stent to the fracture segment. In a retrospective study, Kursaklioglu et al. found a very high rate of restenosis after stenting of the MB segment compared to stenting in pre-bridge LAD atherosclerotic lesions [26]. Ernst et al. reported five-year angiographic

and clinical follow-up data of patients with drug-eluting stent implantation for symptomatic MB [24]. They found that in-stent restenosis occurred in 18.7% of the patients after 12 mos. This was lower than in MB patients treated with bare metal stents [29]. In the study of Ernst et al., symptoms disappeared in almost all patients, and coronary perforation as a complication of the procedure was seen in only one patient, who was treated with graft-stent implantation [24]. In contrast to symptomatic relief, the stenosis rate was relatively higher. In the present case, stent implantation was first attempted. However, the balloon advancement failed. Even if we had inserted a balloon, we still might not have been able to have implanted a stent in the MB due to the depth and tortuosity.

Surgery is another option to treat MB refractory to medical therapy. Surgery is applied either as supra-arterial myotomy and de-roofing of the muscle bands, or as CABG. Most of the studies reported no mortality during or after surgery, however, surgical myotomy carries the risk of entering the ventricle, bleeding, and aneurysm formation when it is deep and extensive [30].

CABG is superior to myotomy in the short- and mid-term and should be the treatment of choice in patients with refractory symptoms unresponsive to medical treatment, and particularly in patients with extensive and

deep bridges [30]. Specifically, CABG is indicated for the patients with extensive (>25 mm) or deep (>5 mm) myocardial bridging, or when the tunneled coronary segment is unlikely to be decompressed completely in diastole [21]. Surgical relief of myocardial ischemia due to MB can be accomplished with low operative risk and excellent mid-term result [31]. In our case, we preferred CABG to myotomy. The LAD was totally occluded, so only myotomy would not have been satisfactory. This case was different from other cases due to the lesion location and its intramyocardial course. This was a pathophysiologically unexpected situation. In this case, our approach was primarily to ensure blood flow, and this was accomplished by emergency surgery.

Conclusion

MB is a common coronary anomaly and often has a good prognosis. It can also cause myocardial infarction under the myocardial layer. Percutaneous coronary intervention should be made cautiously, and surgery should not be delayed when necessary.

No conflict of interest is reported.

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