Myocardial Bridge

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A myocardial bridge (MB) is an anatomical variant in which the myocardial muscle partially covers the epicardial coronary arteries, in particular, the left anterior descending coronary artery (LAD). Although MB can be detected in any coronary artery, most involve the left anterior descending coronary artery (LAD). This variant has historically been regarded as benign, because contraction of the bridged muscles alters blood flow within the underlying LAD during systole, whereas coronary flow in the LAD occurs predominantly during diastole. However, an MB can lead to significant clinical issues, such as arrhythmia, myocardial ischemia, conduction disturbances, myocardial infarction and sudden death in a subset of patients. Autopsy and CT studies have identified MB in ~25% of patients, whereas only 10% of patients have angiographically detectable systolic compression. Intravascular imaging is more sensitive than angiography for detecting minor MB compression. Imaging and autopsy studies have shown a greater plaque burden in the LAD segment proximal to the MB than within the tunneled LAD segment, and this can be associated with a series of severe cardiovascular events. In general, symptomatic patients should be treated conservatively with medical management comprising beta-blockers and non-dihydropyridine calcium-channel blockers to reduce arterial compression by the muscular band and slow the heart rate, thereby increasing the diastolic period. Various strategies including surgery have been attempted to treat refractory symptoms, depending on the status of patients.

KEY WORDS: acute coronary syndrome, cardiac surgery, intracoronary imaging, medical management, myocardial bridging

I. Introduction

A myocardial bridge (MB) is an anatomical variant in which the myocardial muscle partially covers the epicardial coronary arteries. Although MB can be detected in any coronary artery, most involve the left anterior descending coronary artery (LAD). This variant has historically been regarded as benign, because contraction of the bridged muscles alters blood flow within the underlying LAD during systole, whereas coronary flow in the LAD occurs predominantly during diastole. However, an MB can lead to significant clinical issues, such as arrhythmia, myocardial ischemia, conduction disturbances, myocardial infarction and sudden death in a subset of patients.

II. Prevalence and diagnostic testing

The prevalence of MB varies widely according to the detection methods applied. The reported MB rates among numerous necropsy series (Fig. 1) range from 5% to 86% and an average of ~25% of adults have MB. The reported rates of MB are higher according to pathological series including thin MB or even myocardial strands with minimal hemodynamic consequences, than those determined by coronary angiography, which typically detects systolic compression as a “milking effect” (Fig. 2). Intracoronary angiography is the most popular means of diagnosing MB in the clinical setting, with detection rates ranging from 0.5% to 12% at rest and up to 40% upon provocation or after intracoronary nitroglycerin injection. Numerous factors have been presumed to account for the reported mismatch between the rates of “tunneled arteries” that run intramurally through the myocardium compared with angiographic findings. These include MB thickness and length, the reciprocal orientation of the coronary artery and myocardial fibers, loose connective or adipose tissue around the bridged segment, aortic outflow tract obstruction, in which the systolic tension that develops in the MB overcomes the intracoronary artery pressure, the intrinsic tone of the wall of the coronary artery, a proximal coronary fixed obstruction that causes a decrease in distal intracoronary pressure, and the status of myocardial contractility. Intravascular ultrasound (IVUS) can clearly visualize eccentric or concentric systolic compression in the tunneled segment of an artery that persists into diastole, accompanied by a highly specific echolucent “half-moon” appearance throughout the cardiac cycle (Fig. 3). Vessel compression can be detected by IVUS under coronary provocation even in the absence of angiographically significant milking. The prevalence of MB determined by IVUS, which is more sensitive than angiography for detecting minor compression, is 23%. Optical coherence tomography (OCT) can also detect MB with a homogeneous specific “band” appearance outside the adventitia (Fig. 4).
The recent introduction of cardiac computed tomography (CCT), with multiplanar and three-dimensional capabilities, has significantly improved the rate of MB detection (Fig. 5), even when the milking effect and/or changes in vessel course are absent or mild on conventional angiograms. Hence, the CCT-based prevalence of MB ranges from 5% to 76%, depending on the intrinsic heterogeneity of the study population, imaging modality, and type of MB.

### III. Effects of myocardial bridge on atherosclerotic plaque formation

Autopsy and clinical studies have found a greater plaque burden in the LAD segment proximal to the MB compared with the tunneled LAD segment. This enhanced location of atherosclerotic plaque associated with an MB is partially attributed to hemodynamic disturbances caused by MB muscle contraction. In particular, the distribution of wall shear stress...
relative to the tunneled segment might be involved, as lower shear stress might confer a predisposition to enhanced lipid transfer across the endothelium and more atherosclerosis. One study that used a computational fluid dynamics model of the LAD in a patient with a symptomatic MB identified areas of relatively low wall shear stress proximal and distal to the MB, and high wall shear stress within the tunneled segment. Likewise, a case-control study of patients with MB also showed that wall shear rate, which is the velocity gradient perpendicular to the wall that relates to wall shear stress according to blood viscosity, was lower in the segment proximal to the MB than within the tunneled segment.

IV. Clinical significance of MB

Four ischemia-inducing mechanisms have been postulated to explain the coronary events and clinical symptoms of patients with MB. One is that the formation of atherosclerotic plaque in the proximal segment of the LAD upstream from the MB entrance due to collision between retrograde and anterograde flow causes trauma to the endothelium, as well as low wall shear stress.
stress in the proximal reference segment\(^\text{25}\). Low shear stress could lead to atherosclerosis by enabling the mass transfer of lipids across the arterial wall. Another is that external systolic compression and endothelial dysfunction in a tunneled artery could stimulate coronary vasospasm and platelet aggregation, resulting in acute coronary syndrome in some clinical settings\(^\text{24, 26, 27}\). Another is that MB should not cause ischemia because they primarily affect systolic, and not diastolic flow. However, at high heart rates, diastole shortens and the systolic contribution to coronary blood flow increases. Also, compression of an MB during systole into diastole would have to limit blood flow distal to the MB during diastole to cause ischemia has been assumed\(^\text{28}\). The fourth proposed mechanism is the “Venturi effect”, which is the reduction in fluid pressure that results when a fluid flows through a constricted section of a pipe. Such mechanisms might participate in decreasing arterial pressure in the MB section, and thus perfusion pressure to corresponding septal branch vessels, leading to focal ischemia\(^\text{3}\).

V. Management

A survey of treatment options described in the literature for patients with symptomatic MB indicates medical approaches, stent deployment in the MB segment, and surgery.

VI. Medications

Medical management consists of beta-blockers to reduce compression of the artery by the muscular band and slow the heart rate, thus increasing the diastolic period\(^\text{29}\). Calcium-channel blockers with negative chronotropic effects when endothelial dysfunction is significant within the MB\(^\text{30}\) (determined by intracoronary acetylcholine administration) can be supplements or alternatives. Vasodilators including nitroglycerin should be cautiously prescribed for patients with MB. Nitrates generally should be avoided because they increase the angiographic degree of systolic narrowing and can cause symptoms to worsen.

Intensive risk factor modification is recommended because of the potential risk of an MB inducing atherosclerosis. Thus, anti-platelet agents should be considered when subclinical atherosclerosis is evident.

VII. Stent placement

Haager et al. (2000) reported that stent deployment was useful for treating symptomatic patients with MB\(^\text{31}\). However, the results of MB stenting are conflicting, and often associated with in-stent restenosis (ISR), possibly because stent fracture or chronic stent recoil under an MB could arise due to mechanical compression by MB contraction during systole\(^\text{32-34}\). Drug-eluting stents (DES) have contributed to a remarkable reduction in ISR compared with bare metal stents (BMS); therefore, DES implantation in a bridged segment has been attempted to remedy ISR. A previous study with a short-term follow-up suggested that DES is a better choice BMS for treating MB. However, the incidence of ISR in patients with MB remains relatively high. The authors of these reports speculated that stent fracture and stent recoil are associated with ISR. On the other hand, a recent IVUS study of paclitaxel-eluting stents found that the dominant mechanism of ISR after MB stenting is neointimal growth rather than chronic stent recoil\(^\text{35}\).
VIII. Surgical strategies

Surgical options for MB include coronary artery bypass grafting (CABG) and surgical myotomy unroofing. Coronary artery bypass grafting is indicated for patients with long (> 25 mm) or thick (> 5 mm) MB or when the tunneled coronary segment is unlikely to be completely decompressed during diastole\(^{30}\). The success of CABG with both arterial and venous conduits is variable and limited\(^{37}\). The incidence of graft failure is high, more so in arterial conduits, presumably because of competitive flow. Surgical unroofing of a LAD MB directly addresses the pathology, rather than selecting a treatment modality initially designed for another disease process, for example, PCI or CABG. Surgical unroofing by an experienced surgeon can safely proceed either on-pump or off-pump without major morbidity or mortality\(^{18}\). The possible complications of surgical unroofing include wall perforation, ventricular aneurysm formation and postoperative bleeding\(^{30}\).

IX. Prognosis

Although MB have generally been considered benign, it is now regarded as a cause of angina-like chest pain, coronary spasm, myocardial ischemia (identified as changes on electrocardiography and by myocardial perfusion stress test findings), acute coronary syndromes, left ventricular dysfunction/stunning, arrhythmias (including supraventricular and ventricular tachycardia), and even sudden cardiac death. Serious events are rare, and whether they can be directly attributed to MB remains controversial. Ischemia is more closely associated with the degree of systolic compression than lesion length or bridge location\(^{30}\).

X. Conclusions

Although ~ 25% of patients have MB according to autopsy and CT findings, only <10% of patients have angiographically detectable systolic compression. Myocardial bridges can be associated with lethal cardiovascular events such as myocardial infarction, arrhythmia and sudden death. Symptomatic patients should be treated conservatively with beta-blockers and non-dihydropyridine calcium-channel blockers to reduce arterial compression by the muscular band, slow the heart rate and consequently increase the diastolic period. Several interventional strategies including surgery have been applied to treat refractory symptoms depending on the status of each patient.

Disclosures

The authors have nothing to disclose regarding this manuscript.

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