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Myocardial bridges: Overview of diagnosis and management

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Abstract

A myocardial bridge is a segment of a coronary artery that travels into the myocardium instead of the normal epicardial course. Although it is general perception that myocardial bridges are normal variants, patients with myocardial bridges can present with symptoms, such as exertional chest pain, that cannot be explained by a secondary etiology. Such patients may benefit from individualized medical/surgical therapy. This article describes the prevalence, clinical presentation, classification, evaluation, and management of children and adults with symptomatic myocardial bridges.

KEYWORDS

intramyocardial coronary artery, myocardial bridge, myocardial ischemia, sudden cardiac death

A myocardial bridge (MB) is defined as an intramyocardial segment of an epicardial coronary artery. Perhaps first described by Reyman in 1737, one of the first descriptions of an MB in the modern literature was published by Geiringer in 1951.[1] Geiringer noted that, although this "trivial and slight deviation from normal" had been largely overlooked as a potential etiology for pathology, this altered anatomic course might influence the development of atherosclerosis. In an unselected human autopsy series of 100 patients, he identified myocardial bridging in 23 hearts, and detailed the myocardial fiber orientation and relative relationships among the coronary arteries, epicardial fat, and myocardium observed in these patients.

The case examples reported in this series reflect observations made in subsequent reports, namely that MBs can range greatly in length as well as depth, that a coronary artery may have more than one bridged segment, and that the overlying fiber orientation may impact the functional significance of a bridge. Ferreira et al. reported a necropsy series of 90 consecutive hearts and found two distinctive patterns of fiber orientation.[2] In the more common superficial orientation, the myocardial fibers cross the artery transversely toward the apex of the heart at an acute angle or perpendicularly. In the deeper orientation, the fibers crossed the left anterior descending (LAD) coronary artery and surrounded it by a muscle bundle that arose from the right ventricular apical trabeculae and crossed the artery transversely, obliquely, or helically before terminating in the interventricular septum. They postulated that the deeper orientation could twist the vessel and compromise diastolic flow, resulting in ischemia.

It is understood that MBs are common, however, the estimations of the prevalence of MBs vary. It is important to consider that estimations will vary at least in part as a result of several key variables, including the means of identification (eg, computed tomography (CT), intravascular ultrasound (IVUS), or autopsy), which vessels are examined, and which definition of a bridge is applied (eg, only a "deep" bridge vs both "superficial" and "deep" bridges). Perhaps the most fundamental variable is whether an MB is even considered. Unlike hypertrophic cardiomyopathy, which will usually be obvious to the pathologist, MBs can be easily obscured by epicardial and pericardial fat. As such, autopsy series have estimated the prevalence between 5% and 86%.[3,4] The largest autopsy report, which included 1056 subjects, found a prevalence of 26%, 88% of which involved the LAD.[5] One population-based study with CT estimated a prevalence of 22.5%.[6] As a result of these study and others, an estimated prevalence of approximately 25% is generally accepted.

Given the common prevalence of MBs, many physicians have concluded that MBs are benign "normal variants" that are without the possibility of pathologic consequence. While the vast majority of MBs are likely asymptomatic, numerous case reports and series have documented the association between MBs and angina or anginal-equivalent symptoms, including exertional chest pain and exertional dyspnea.[7–9] In addition to symptoms, there are also a number of reports in the literature that have associated MBs with ventricular arrhythmia, myocardial infarction, syncope, and sudden cardiac death.[10–14]

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In clinical practice at Stanford, our group has similarly observed a common clinical presentation of symptomatic MBs with exertional chest pain and/or exertional dyspnea.[15,16] Our evaluation process ensures exclusion of more commonly considered etiologies (eg, obstructive coronary artery disease [CAD], asthma) as well as review of diagnoses that have been previously attributed to a given patient's symptoms. As an example, a not uncommon presentation in our practice has been exertional dyspnea previously empirically diagnosed as asthma, yet no improvement in symptoms with asthma therapy and no prior confirmation of the asthma diagnosis with pulmonary function testing. The age of onset of symptoms can vary widely from childhood through late adulthood; the variation in some part secondary to concomitant endothelial dysfunction, clinical recognition, and other factors. Although more commonly described in the adult literature, our observation of symptomatic MBs in children has been similarly observed by other groups.[17–19]

A variety of noninvasive and invasive modalities have been utilized to anatomically identify and functionally assess MBs. Given the widespread use of single-photon emission computerized tomography (SPECT) myocardial perfusion imaging (MPI) for the detection of myocardial ischemia secondary to obstructive CAD in the practice of adult cardiology, SPECT MPI has been considered as a modality for functional assessment of MBs. However, SPECT MPI tends to demonstrate a lower sensitivity for the detection of ischemia from MBs, preferentially detecting the most critically compressed LAD segments.[20] Unlike the fixed obstruction of CAD, which persists throughout the cardiac cycle and often affects a moderate or greater territory of myocardium, the ischemia from MBs is dynamic (occurring in late systole to early diastole) and typically affects only a small territory of myocardium in the distribution of septal perforators within the bridged segment. As such, the mechanism of this ischemia appears to not be optimally imaged by SPECT.

Our group at Stanford recently described the first report to suggest that stress echocardiography may serve as an effective noninvasive modality for the identification of MBs.[15] This finding has been subsequently replicated by at least one other group.[21] We described a unique pattern of focal end-systolic to early-diastolic buckling in the septum with apical sparing on multi-beat poststress images, which correlated well with invasive assessment of ischemia in bridged LAD segments. These findings also supported our theory that the dynamic mechanism of ischemia from MBs may be mediated through the Venturi effect. Although our subsequent anecdotal experience has been that stress echocardiography can demonstrate high sensitivity and specificity for the detection of MBs, formal analysis to determine the sensitivity and specificity of this modality, as well as its relationship to ischemia, is ongoing.

Distinct from the functional assessment potentially provided by stress echocardiography, cardiac CT can provide a valuable noninvasive anatomic assessment for MBs, as it is unique in its ability to directly visualize the coronary arteries, the myocardium, and the relationship between these structures. The sensitivity and specificity of CT interpretation reflect the continuum of MBs; namely that the depth of bridging of a coronary artery can span from fine superficial bridging to deep bridging. Given that superficial bridging can represent overlying muscle fibers at or below the current spatial resolution of CT (commonly 0.5–0.75 mm), the interpreting physician must use care to recognize superficial bridges, also referred to as "partial bridging" or "partial encasement," as a segment of the LAD in direct contact with the left ventricular myocardium in the interventricular groove, as illustrated by Kim et al.[22] In contrast, deep MBs, also referred to as "complete bridging" or "full encasement," represent a segment of coronary that is surrounded by myocardium, with varying depth of overlying myocardium (Figure 1). Computed tomography also permits exclusion of concomitant obstructive CAD and ostial coronary anomalies, as well as demonstrates the relationship between the bridged segment and adjacent diagonal and larger septal branches, which can be particularly useful in surgical planning.

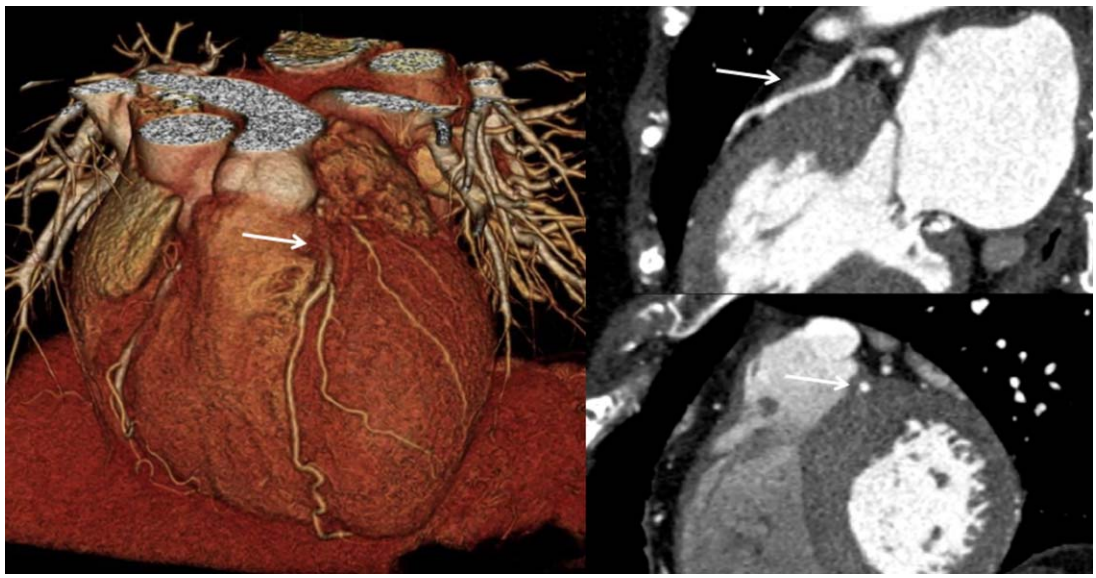

FIGURE 1 Cardiac CT demonstrating a deep myocardial bridge in the proximal LAD (white arrow) on three-dimensional (3D) and corresponding long and short axis two-dimensional (2D) images

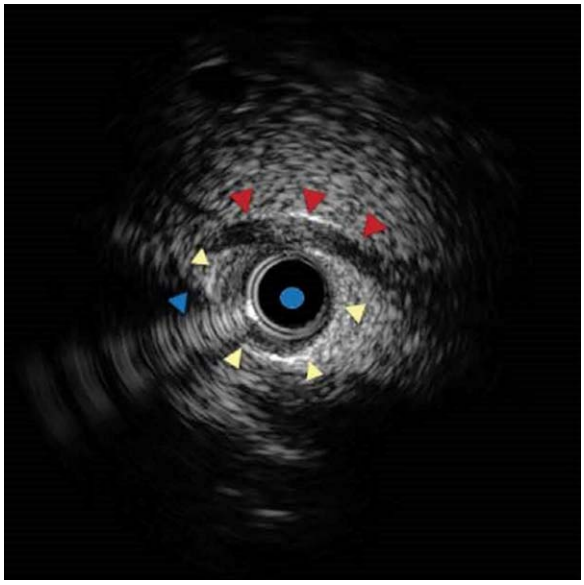

FIGURE 2 Intravascular ultrasound (IVUS) picture of an echo-lucent half-moon sign. The red arrows outline the echo-lucent half-moon sign. The yellow arrows outline the vessel wall. The blue round circle is positioned in the center of the IVUS catheter and the blue arrow points to an artifact from the catheter. (Used with permission from Lin et al. *J Am Heart Assoc*. 2013)

Although invasive coronary angiography (ICA) might seem a useful modality for anatomic assessment, the sensitivity of ICA for detection of myocardial bridging is low, generally estimated as approximately 5%, with ranges in prior studies between 0.5% and 12%.[23,24] As such, ICA alone cannot exclude the presence of a MB. Intracoronary nitroglycerin may dilate nonbridged segments, potentially increasing sensitivity for detection of bridged segments.[25] However, IVUS is generally regarded as a more definitive invasive modality for the anatomic detection of MBs, which are characterized on IVUS as a echolucent "half-moon" area (Figure 2) immediately adjacent to the lumen throughout the cardiac cycle.[26] Although there has been uncertainty as to the nature of the "half-moon" echolucency, Yamada et al. demonstrated in a post-mortem heart utilizing IVUS, pathologic assessment, and histologic assessment, that the echolucency identified by IVUS did indeed represent overlying muscle band.[27]

Intravascular ultrasound can also demonstrate the degree of systolic compression within a bridged segment, as well as the extent of plaque progression proximal to the bridged segment, which might predispose patients with an MB to myocardial infarction.[13] In addition to the anatomic assessment provided by IVUS, pressure and flow can be assessed proximal to, within, and distal to a bridged segment prior to and during invasive dobutamine stress with a simultaneous Doppler flow and pressure wire to confirm the physiologic significance of an MB (Figure 3). With Doppler assessment, physiologically significant

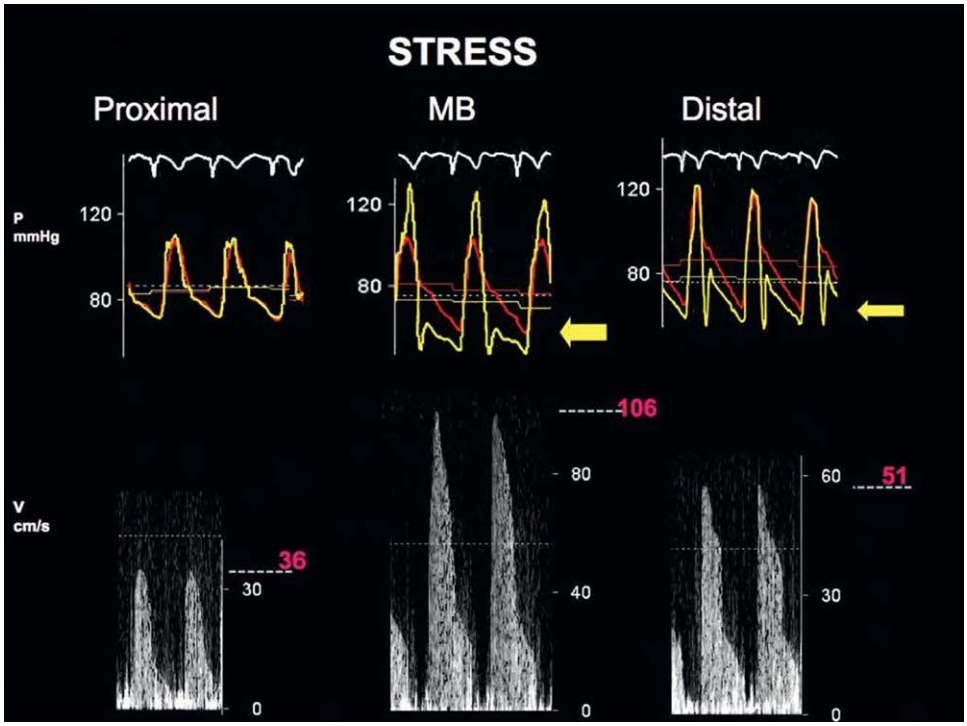

FIGURE 3 Representative readings of flow velocity and pressure. With peak stress, there is no significant change in the 2 pressure lines proximally. Within the myocardial bridge, there is systolic overshoot (yellow line) and diastolic decline in pressure, resulting in a diastolic fractional flow reserve (dFFR) of 0.74 (large yellow arrow). Distal to the bridge, there is diastolic pressure recovery with a dFFR of 0.88 (small yellow arrow). Stress Doppler peak velocities are 36, 106, and 51 cm/s, respectively. (Used with permission from Lin et al. *J Am Heart Assoc*. 2013)

bridges demonstrate accelerated flow velocity in early diastole and retrograde flow in the proximal vessel.[28] As described by Escaned et al., dobutamine stress is instrumental to the physiological assessment of MBs, with the diastolic fractional flow reserve serving as the metric for evaluation of ischemia from the bridge.[29] It is important to note that the use of the diastolic FFR for evaluation of this dynamic mechanism of ischemia is distinct from the mean FFR that is used for the evaluation of ischemia from fixed obstructive CAD. Moreover, invasive evaluation of this dynamic ischemia requires the chronotropic mechanism of dobutamine stress. Vasodilator agents, such as adenosine, that are often used for the evaluation of ischemia from fixed obstructive CAD, are not effective in the assessment of ischemia from MBs.

No randomized clinical trial data exists in the literature regarding options for medical or surgical management of myocardial bridging. Beta-blocker therapy is generally regarded as first line medical therapy for symptomatic patients with the goal of decreasing heart rate and contractility, thereby reducing coronary compression. Evidence for this therapy was demonstrated by Schwarz et al.[30] who demonstrated a reduction in compression and maximal flow velocity following the administration of esmolol during invasive assessment. We preferentially utilize nebivolol, a highly selective beta blocker thought in preclinical studies to produce endothelium-dependent vasodilation by increasing nitric oxide release,[31,32] given the concomitant endothelial dysfunction that we have observed in many of our patients with an MB. Diltiazem and dihydropyridine calcium channel blockers, such as nifedipine, can similarly be helpful in reducing symptoms via a reduction in compression and improvement in endothelial function.

When symptoms are recalcitrant to medical therapy, unroofing (myotomy) is the preferred surgical treatment strategy in the absence of concomitant obstructive CAD in the LAD.[33] We regard percutaneous stent placement for treatment of MB-associated symptoms as relatively contraindicated, given the potential risk of stent fracture from unabated compression[34–37] and increased risk of restenosis. Coronary artery bypass graft (CABG) surgery has been utilized by other centers as a surgical treatment strategy, however, CABG is associated with the risk of graft closure secondary to competitive flow given that the bridged native LAD is dynamically compressed yet remains patent.[38] Moreover, given the persistent compression, the local ischemia is not addressed by CABG. As such, failure of CABG to improve symptoms has been associated with the need for subsequent unroofing.[39] Unroofing definitively corrects the anatomic defect, thereby improving flow and relieving the source of myocardial ischemia,[40] and can be performed via sternotomy on or off cardiopulmonary bypass or via a minimally invasive approach.[41–45]

CONFLICT OF INTEREST

None.

DISCLOSURES

None.

AUTHOR CONTRIBUTIONS

The author contributed in the concept/design, drafting, critical revision and approval of the article.

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