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Narrative Review

Coronary Myocardial Bridge Updates: Anatomy, Pathophysiology, Clinical Manifestations, Diagnosis, and Treatment Options

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Abstract

Myocardial bridging is a frequent anomaly of the heart in humans and other animals. A myocardial bridge is typically characterized by the systolic narrowing seen with traditional catheter angiography, but this abnormality is not by itself a sign of ischemia or the need for intervention. In particular, transient spontaneous angina must be corroborated by reproducible narrowing during acetylcholine testing; this narrowing occurs during resting conditions and is responsive to nitroglycerin administration. Ischemia in myocardial bridging can result from acquired arterial wall disease (coronary artery atherosclerotic disease) or from instances of coronary spasm. Clinical evaluation should seek to identify baseline features such as myocardial bridge thickness (by using computerized axial tomography or intravascular ultrasonography) and the severity of systolic compression or reproducible spasticity (by administering acetylcholine). Nuclear myocardial scintigraphy is usually negative in patients with isolated myocardial bridging. Spastic coronary hyperactivity must be treated initially with antispasmodic medications, such as calcium channel blockers and nitrates, rather than by percutaneous stent placement or bypass surgery. Only exceptionally prolonged and critically severe spasm can induce intraluminal clotting and acute myocardial infarction. Recognizing the exceptionality and variability of ischemic presentations related to myocardial bridging is essential, as is establishing appropriate investigational methods for each of these facets of the condition.

Keywords: Myocardial bridging; coronary vessel anomalies; coronary artery stenosis; heart defects, congenital; cardiovascular abnormalities; myocardial ischemia; coronary vasospasm; coronary artery disease; ultrasonography, interventional; tomography, optical coherence

Introduction

Anatomically, myocardial bridging (MB) refers to a congenital coronary segment's abnormal passage beneath compact myocardium. Such coronary segments are subject to systolic compression in lateral or circular patterns of variable degree and length. Discussions of MB's influence require large, uniform patient populations with asymptomatic vs symptomatic baseline states and data on MB-relevant features (depth and length, determined by using traditional catheter angiography or computed tomographic angiography [CTA]) and other, potentially accompanying cardiovascular conditions, such as hypertrophic obstructive cardiomyopathy.[1] Appropriate stress testing and coronary vasomotor testing are required to rule out spontaneous spasm.[2,3]

The prevalence of MB is high—MB is frequently described as the most common congenital cardiac anomaly—but its clinical expression is minimal in terms of incidence and severity. A detailed description of clinical manifestations is the fundamental starting point in any given patient. An anomaly present in more than 1% of a large nonspecific population of individuals is usually considered a normal variant instead of an anomaly intrinsically capable of causing a disease state.[1,4]

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The first foundational MB investigation published in the modern age of medicine was written by Poláček,[5,6] who concluded that the reported incidence of MB in autopsy studies differed by the detection method employed: By gross external anatomic inspection, the incidence was 5%; by transversal longitudinal dissection, it was 17%; and by detailed microscopy (loop enlargement, probably ×10 original magnification), it was 60%. Similar disparities are also evident from traditional catheter angiography results (with or without nitroglycerin use), coronary CTA, intravascular ultrasonography (IVUS), and optical coherence tomography (OCT). The thinner the myocardial layer, the weaker its effect generally on the underlying coronary segment of systolic narrowing.

In humans, MB only occasionally produces adverse outcomes. In many animal species (eg, rabbits, hares, hamsters, squirrels, rats, birds), the intramyocardial course of most or all large arteries is frequent[5,7] and free of apparent ischemic manifestations. The severity of systolic luminal narrowing also depends on intramyocardial pressure, which is worse when hypertrophic obstructive cardiomyopathy is present, such that myocardial fiber disarray may contribute to it.[8] With each heartbeat, the coronary artery is mechanically stimulated by the MB effect.[9,10]

Key Points

- Coronary myocardial bridges are frequent in humans but usually benign.
- In symptomatic patients with MB, the main question is which testing is required to identify the causative mechanism for the presentation.
- Clinically significant baseline stenosis is rare in cases of MB; it may be transiently caused by spasm, which can be reproduced by acetylcholine testing; treatment will depend on the causative mechanism.

Abbreviations

CAD, coronary artery disease
CSA, cross-sectional area
CT, computed tomography
CTA, computed tomographic angiography
ECG, electrocardiogram or electrocardiography
FFR, fractional flow reserve
IVUS, intravascular ultrasonography
LAD, left anterior descending coronary artery
MB, myocardial bridging
MI, myocardial infarction
OCT, optical coherence tomography

Incidence and Severity in Humans

Poláček[5,6] reported that the incidence of MB varies according to the observation method employed and that MB is more frequent in the proximal left anterior descending coronary artery (LAD) tract (60% of all human MB). Associated coronary artery disease (CAD) seemed to be prevented at MB sites but aggravated proximally to the myocardial bridge. The U-sign (a short, deepening segment of an affected coronary artery in systole) is the angiographic sign of MB. In a traditional catheter angiography–based study of a large, nonspecific population, MB prevalence based on the U-sign of systolic narrowing was 14% to 20%.[11] The highest MB incidence (close to 100%) is associated with hypertrophic obstructive cardiomyopathy.[4,8]

Vascular tone seems to affect the degree of visible systolic compression, as indicated by the increased severity of myocardial bridge narrowing after nitroglycerin administration.[4,11] In contrast, CTA imaging typically suggests that the main correlate for MB anatomical severity is the thickness of the overlying myocardial bands. Computed tomographic angiographic imaging is clinically obtained only at end-diastole, when MB effect is trivial. Imaging modalities such as IVUS and OCT identify MB geometrically on the basis of muscle thickness and systolic luminal cross-sectional area (CSA) compared with proximal or distal reference CSAs.[12] Phasic stenosis is not an exact physiological or clinical marker of MB functional severity (the stenotic effect affects only 30%-40% of the cardiac cycle in resting conditions) but could be important during exercise, when systole can increase to more than 70% of the total cardiac cycle time at a heart rate of 170/min to 200/min. Hemodynamic measurement of stenosis by using pressure wires or flow wires also has limitations because of the presence of a potentially spasmogenic foreign body and variable phasic flow.[2,3,10,12-15]

Mild hypoplasia of the MB segment (5%-10% smaller CSA with respect to the reference CSA in end-diastole) is consistently identified by IVUS and OCT.[16] The important message from hemodynamic measurement is that isolated MB (no CAD, no spasm) does not substantially impede blood flow and generally does not justify intervention. Conversely, during a positive acetylcholine test of endothelial dysfunction, induced spastic narrowing especially affects diastolic time,

whereas nitroglycerin dilates the end-diastolic CSA while intensifying systolic narrowing.[17]

Both IVUS and OCT can exactly measure a potentially complicating atherosclerotic intimal thickening or stenosis. The "half-moon sign" frequently seen on IVUS imaging indicates the presence of an MB effect but not its severity.

Clinical Manifestations and Pathophysiology

In 99% of cases, isolated MB does not affect either mortality or the incidence of myocardial infarction (MI) or effort-induced angina, nor does it relate to positive stress testing for ischemia.[8,11,18] Acute MI and resting angina with electrocardiographic (ECG) changes are rarely reported in MB and are mainly the result of sustained spastic narrowing at the site, which may be complicated by luminal secondary clotting.[19]

Cardiologic interest in MB has focused mainly on the mechanisms and factors affecting clinical expression of an MB-related stenotic effect. Most patients with MB are studied for chest pain, abnormal stress test results, or clinical events by some combination of traditional catheter angiography, CTA, and IVUS, which consistently shows mild or no CAD-related intimal thickening at the MB site.[17] Mild, diffuse, smooth luminal narrowing is usually the product of baseline MB-related hypoplasia and is not CAD based,[15,20] which simultaneously suggests that MB protects against CAD development.

Especially in patients with precordial chest pain at rest that is sensitive to nitroglycerin administration and not reproducible with exertion, one should first consider intermittent coronary spasmodic events. In isolated MB indicated by traditional catheter angiography, stress testing of any kind is not usually positive for reversible ischemia in the dependent territory. Although impractical to obtain, the best evidence would be a 12-lead ECG during angina at rest. Even without fixed CAD stenosis (negative stress testing), patients with MB can have confusing resting or effort-induced angina. Vascular tone testing with either acetylcholine or ergonovine is therefore required.[2,3,9,10,21] The potential existence of microvascular dysfunction (with spasm as a result of small vessel disease) has been debated without definitive conclusions on what it would exactly constitute or how frequent it is.[10]

In an autopsy study of male and female hearts, Ferreira et al[22] proposed a classification method for distinguishing between superficial and deep myocardial bridges. The superficial bridges were those bridges in which the LAD ran along the interventricular groove, with the overlying muscle bridge crossing perpendicularly or at an acute angle. In contrast, the deep myocardial bridges were those in which the LAD was situated deeply in the interventricular septum; deviated toward the right ventricle; and was crossed by longitudinal muscle fibers in an oblique, helical, or transverse fashion. Some investigators[22-24] have speculated that the sheathlike orientation of these lengthy muscle fibers is associated with the compression and depression of the LAD and adjacent arteries, as suggested by evidence of fibrosis. As a result, such deep myocardial bridges are associated with myocardial ischemia and can lead to angina, MI, or sudden cardiac death.

Although in vivo imaging methods have been used to inspect the morphology of myocardial bridges, a study by Möhlenkamp and colleagues[25] suggests that the severity of symptoms that accompany MB is not associated with the length or depth of the deep, tunneled segment. A retrospective analysis of intramyocardial segment length and depth could therefore lead to a more reliable method for assessing the severity of myocardial bridges. In vivo imaging, especially around the ventricular septum,[23] may also provide insight into the severity of the myocardial bridge by assessing the degree of cell injury and tissue fibrosis in that region.

Acetylcholine Testing

Acetylcholine testing of endothelial dysfunction aims to show controlled reproduction of spontaneous symptoms during angiographic monitoring of coronary luminal size and ECG monitoring of chest pain (or equivalents). Complication rates are similar to rates observed in expert centers using simple traditional catheter angiography.[21,26] Although noninvasive testing by systemic administration of ergonovine[27] with ECG and echocardiographic monitoring seems an attractive alternative, results are uncertain, given their low yield in the presence of low coronary serum concentrations of ergonovine and when an immediate antidote is lacking; intracoronary nitroglycerin administration is considered essential for documenting reversible spasm to prevent severe and prolonged ischemic response and to ensure safety.[26] Years of clinical experience in acetylcholine

testing in patients with MB have confirmed the correlation between acetylcholine and spontaneous resting angina patterns.[21,28,29] During testing, patients with typical symptoms have almost 90% probability of chest pain and spasm reproduction at MB sites with ECG changes; the probability of these events in atypical chest pain (with different clinical contexts, variable angina location) is much lower.[29]

Recent pediatric cardiology discussions seem questionable when they suggest that the congenital ectopic anomaly of the left coronary artery originating from the right sinus of Valsalva, with an intraseptal, intramyocardial, or infundibular course, is the cause of chest pain, MI, or sudden death. These suggestions lack ultimate support from acetylcholine testing to rule out alternative causes.[30,31] Using pressure wires to validate the presumption of stenosis in tortuous vessels (without using nitroglycerin administration or angiography to rule out artifactual spasm[32]) seems especially dubious. In this unusual pathology, angiographic evidence of baseline stenosis has never been reported. As a result, surgery is being advised solely on the basis of speculative gradients in rare congenital coronary arterial anomalies.[30,31]

Alternative testing modalities have recently been reported, particularly computed tomography (CT)–derived fractional flow reserve (FFR) computational fluid dynamics to calculate flow and CT-derived FFR using dynamic (phasic, in systole vs diastole) myocardial perfusion imaging. Hecht[33] suggested that CT-derived FFR can be used for coronary evaluation in certain conditions, including chronic hemodynamic angina (focusing on gradually progressive CAD and effort-induced angina), acute-onset unstable angina with CAD plaque erosion or ulceration (unstable CAD angina at rest), and spasm at the MB site (although CT-derived FFR cannot detect the onset of spontaneous spastic angina), with insufficient initial results.

Although reviewing a continuous series of 104 patients with variable clinical presentations and angiographic evidence of MB, Zhou et al[34] analyzed the use of CT-derived FFR to correlate CTA-based MB "severity subclassification" (depth and length, systolic angiographic stenosis of 30%-90%) with clinical outcomes. The authors concluded that 46% of patients had an FFR less than 0.80. The study was nevertheless inadequate to assess MB severity, and long-term major adverse cardiac events were not assessed. More in-depth evaluations are required.

Yu et al[35] recently reported the use of dynamic CT-derived FFR myocardial perfusion imaging in 498 patients with MB and multiple potential confounding factors such as hypertrophic cardiomyopathy, sudden cardiac death at rest, and unstable angina. They calculated absolute myocardial blood flow (assuming that 100 mL/100 g of myocardium is normal and that an FFR <0.80 would be proof of resting ischemia) and proposed that systolic imaging may be the most sensitive and specific method for identifying ischemia. They concluded that more studies are required to achieve clarity.

Treatment Alternatives

By itself, MB is not an intrinsic mechanism of clinically severe stenosis and prognosis, especially not one that is generally capable of causing chest pain or ischemia at rest, MI, or death. Medical therapy to prevent atherosclerotic progression is the only useful prophylactic treatment for preventing CAD; b-blockers have no demonstrated value. Percutaneous coronary intervention,[12,13,36-40] unroofing, or bypass grafting is not generally required in the absence of severe CAD.[14,41] If calcium channel blockers and nitrates are insufficient palliation for angina pain in patients without CAD stenosis, stent angioplasty could be useful, despite the likelihood of less-than-ideal results (including greater risk for restenosis from stent crushing under repetitive systolic compression).

A question remains: In the presence of isolated MB, are stent angioplasty, bypass coronary surgery, and coronary unroofing potentially more advantageous than medical treatment? Avoiding MI and death is not the primary objective in treating MB, but palliating angina could be. Medical treatment for MB with spastic angina usually involves vasodilators such as calcium channel blockers and long-term or short-term nitrates; antilipid medication and angiotensin-converting enzyme inhibitors are not generally helpful for preventing coronary arterial spastic pathology, though they can be quite useful when hypertension or hyperlipidemia is out of control.

The existence of an enormous population with MB should discourage a scattershot "vaccination approach" (ie, widespread intervention). In studies following patients with MB for many years, cardiologists have noticed a consistent trend that includes periods of active spastic manifestation alternating with an asymptomatic state. Most carriers of these congenital defects start reporting symptoms at middle or advanced age, and they

do not die of cardiac causes when associated cardiac diseases are absent. A conservative approach is generally the first and most effective choice in similar cases.

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