REVIEW ARTICLE

Myocardial Bridging: A Review with Emphasis on Electrocardiographic Findings

Daniele Rovai, M.D.,* Gianluca Di Bella, M.D., Ph.D.,†
Alessandro Pingitore, M.D., Ph.D.,* and Michele Coceani, M.D.‡

From the *CNR, Institute of Clinical Physiology, Pisa, Italy; †Department of Clinical and Experimental Medicine, University of Messina, Messina, Italy; and ‡G. Monasterio Foundation, Pisa, Italy

Background: Myocardial bridging (MB) occurs when a segment of an epicardial coronary artery takes an intra-myocardial course, thus leading to systolic compression. Most myocardial bridges involve the left anterior descending artery and are observed in 14–35% of patients. Different pathophysiological mechanisms can induce symptoms secondary to myocardial ischemia: systolic coronary compression, diastolic dysfunction associated with aging and coronary atherosclerosis, LV hypertrophy, vasospasm, microvascular and endothelial dysfunction, plaque development proximal to the bridge.

Methods: We performed a literature review of MB, with a particular emphasis on electrocardiographic manifestations.

Results: Stable angina-like chest pain is the usual presentation and MB should be suspected in patients at low risk for coronary atherosclerosis which refer this symptom or which present myocardial ischemia at instrumental examinations. ECG changes are not specific for MB and resting ECG is often normal or presents ST segment anomalies. Exercise stress test often shows non specific signs of ischemia, conduction disturbances or arrhythmias which do not allow the distinction between myocardial bridging and other causes of myocardial ischemia; angina often appears during exercise, even in the absence of ECG changes. Myocardial perfusion deficits at scintigraphy are neither obligatory nor specific. Although the clinical significance of MB is still debated, MB has been associated with acute coronary syndrome, coronary vasospasm, and even sudden cardiac death.

Conclusion: Although MB may lead to myocardial ischemia during stress, its clinical presentation and electrocardiographic findings are not specific.

Ann Noninvasive Electrocardiol 2015;20(2):103–107

coronary artery anomaly; myocardial bridging; myocardial ischemia

In addition to coronary atherothrombosis, several pathological conditions may generate myocardial ischemia and its clinical manifestations. These conditions include coronary artery spasm, hypercoagulability, endothelial or microvascular dysfunction, congenital anomalies of coronary arteries, and diseases which affect coronary hemodynamics, such as aortic stenosis and insufficiency or severe ventricular hypertrophy and myocardial bridging (MB). The aim of this study is to review the most important aspects related with emphasis in the ECG changes in the case of MB.

CONCEPT

MB, first mentioned in 1737,¹ occurs when a segment of an epicardial coronary artery takes an intramyocardial course and is covered by a bridge of myocardium.² MB leads to systolic compression of the tunneled segment. Although this congenital coronary anomaly remains clinically silent in the majority of cases, clinical interest was triggered by an observed association of MB with myocardial ischemia.^{3–5}

Most myocardial bridges involve the left anterior descending artery. The depth and length of the

Address for correspondence: Daniele Rovai, M.D., F.E.S.C., CNR, Clinical Physiology Institute, Via Moruzzi 1, 56124 – Pisa, Italy. Fax: 0039-050-315 2166; E-mail: drovai@ifc.cnr.it

This study was supported by institutional grants of the CNR Institute of Clinical Physiology.

© 2014 Wiley Periodicals, Inc.
DOI: 10.1111/anec.12242

intramyocardial artery ranges from 1 to 10 mm and from 2 to 30 mm, respectively. During systole, coronary luminal diameter is reduced on average by 1 mm, generating a > 50% stenosis in one-third of patients.^{2,5} A minor degree of coronary atherosclerosis is sometimes present proximally to the bridge.

PREVALENCE

The prevalence of MB varies widely according to the methods used to detect such an anomaly. At *autopsy*, myocardial bridges were found in 15–80% of cases.^{6,7} These variations may be partly attributable to the care taken during preparation and selection of hearts. On average, myocardial bridges are present in about one-third of adults. At invasive *coronary angiography*, MB is diagnosed when a segment of a coronary artery is squeezed during systole with complete or partial release of the compression during diastole (Fig. 1).³ Using this criterion, myocardial bridges are reported in less than 2.5% of cases at routine coronary angiography. However, a dedicated study found MB in up to 16% of coronary angiograms.⁸ In small studies using *intravascular ultrasound*, the prevalence of MB increased to 23%. Using this technology, the myocardial bridge appears as a “half-moon”-like area that compresses the tunneled coronary artery during systole.⁹ Nowadays, MB is routinely diagnosed by coronary *computed tomographic angiography*, where the prevalence of this coronary anomaly ranges from 14% to 35%.¹⁰

Here, the myocardial bridge is seen as a shift of a coronary artery into the myocardium with a typical “step down-step up” phenomenon.

PATHOPHYSIOLOGY

A new light on the pathogenic mechanisms leading to ischemia in patients with MB has recently been shed by a study that combined stress echocardiography, intravascular ultrasound, and fractional flow reserve.¹¹ An early relaxation of the interventricular septum with apical sparing has been found at peak exercise in patients with isolated MB of the left anterior descending artery. Eighteen patients with angina-like chest pain and the above echocardiographic pattern were studied by coronary angiography, intravascular ultrasound of the left anterior descending artery, and

intracoronary pressure and Doppler measurements at rest and during dobutamine stress. All patients showed MB of the left anterior descending artery at intravascular ultrasound. Of note, all these patients showed a pathologic fractional flow reserve (≤ 0.75) in the left anterior descending artery during dobutamine stress. Furthermore, peak Doppler flow velocity inside the bridge more than doubled during dobutamine infusion. At the level of the bridge, intracoronary pressure increased during systole (due to external compression) while it declined during diastole well below aortic pressure (Fig. 2). These data suggest a new type of myocardial ischemia, different from the usual forms of ischemia caused by obstructive coronary artery disease. In patients with MB, ischemia is caused by systolic coronary compression that affects coronary perfusion pressure during diastole. However, the exact relationship between systolic compression and decline in diastolic pressure is not yet fully understood.

In another study, 18 patients with lone MB were studied by fractional flow reserve before and after dobutamine infusion.¹² At baseline, only one lesion was functionally significant (FFR < 0.80). After dobutamine infusion, diastolic FFR became severely reduced and two additional lesions became functionally significant. There are different pathophysiological changes that can induce symptoms of myocardial ischemia in previously asymptomatic patients.¹³ First, increasing left ventricular diastolic dysfunction associated with aging, hypertension, and coronary atherosclerosis can exacerbate the supply-demand mismatch imposed by the bridge. Second, development of left ventricular hypertrophy can increase compression and reduce the coronary microvascular reserve. Third, coronary vasospasm, microvascular dysfunction, or endothelial dysfunction related to cardiovascular risk factors combined with the bridge can result in myocardial ischemia. Fourth, plaque development proximal to the bridge can augment coronary obstruction by the bridge. Fifth, the negative remodeling within the bridge can reduce myocardial flow. Thus, myocardial ischemia is not purely related to systolic vascular compression. Systolic and diastolic flow impairment contributes to myocardial supply-demand mismatch in patients with MB.

Pathologic studies have demonstrated that MB is a frequent component of the hypertrophic cardiomyopathy phenotype. In a morphological

Figure 1. A 46-year-old man was admitted for suspect acute coronary syndrome. The patient underwent coronary angiography which excluded the presence of significant stenosis, while also unveiling a myocardial bridge of the mid left anterior descending coronary artery with evident systolic compression (yellow arrows, left-hand panel) and normal caliber during diastole (white arrows, right-hand panel).

Figure 2. Intracoronary pressure and Doppler flow velocity traces during a dobutamine stress test in a patient with myocardial bridging. Reproduced with permission from Lin et al.¹¹

analysis of more than 250 hearts, myocardial bridges were found in 41% of specimens and more frequently than in a control group of hearts.¹⁴ However, no systematic association with hypertrophic cardiomyopathy-related sudden death was evident. MB of the left anterior descending artery is also a frequent finding in patients with apical ballooning syndrome.¹⁵

These studies consistently demonstrate that myocardial bridges are not innocent bystanders and that they are able to induce myocardial

ischemia during stress, as in the case of dobutamine infusion in the cath laboratory or strenuous exercise in daily life.

CLINICAL PRESENTATION

Stable angina-like chest pain is the usual presentation of MB. Myocardial infarction, exercise-induced ventricular arrhythmias, and sudden cardiac death are rare.²

Figure 3. A. 55-year-old man without coronary risk factors come to our attention for typical effort angina. Resting ECG was normal (top panel). During the exercise stress test angina appeared after 6 minutes in the absence of electrocardiographic changes (lower panel). The patients were affected by myocardial bridging on the left anterior descending artery with systolic compression.

From the point of view of the clinical cardiologist, MB should be suspected when a patient at low risk for coronary atherosclerosis refers angina-like chest pain or presents myocardial ischemia at instrumental examinations. Frequently in presence of exercise angina the ECG remains normal (Fig. 2).

Myocardial infarction, left ventricular dysfunction, myocardial stunning, paroxysmal AV

blockade, as well as sudden cardiac may occur in MB. However, considering the prevalence of MB, these complications are rare. Patients may present with atypical or angina-like chest pain with no consistent association between symptom severity and the length or depth of the tunneled segment or the degree of systolic compression.

ECG TECHNIQUES

Resting electrocardiogram is generally normal in patients with MB, as frequently occurs in patients with stable angina, although some patients may present ST segment anomalies, especially in presence of left ventricular hypertrophy. Exercise stress tests often show nonspecific signs of ischemia, conduction disturbances or arrhythmias which do not allow the distinction between MB and other forms of ischemia.² Of note, in our experience, angina appears very often during the exercise stress test in patients with MB even in the absence of ECG changes (Fig. 3).

Children with hypertrophic obstructive cardiomyopathy and myocardial bridges may have an increased QTc dispersion and a higher rate of monomorphic ventricular tachycardia on Holter ECG compared with subjects without myocardial bridges.¹⁴

Myocardial perfusion deficits at stress myocardial perfusion scintigraphy are neither obligatory nor specific. A retrospective analysis of 42 patients with isolated myocardial bridges and no evidence of coronary atherosclerosis that underwent stress/rest gated SPECT showed perfusion abnormalities in 43% of patients with > 50% coronary narrowing caused by bridging.¹⁰ These perfusion abnormalities were mild and not specific.

Prognostic Impact

The clinical significance of MB is still debated. The fact that only a small subset of patients present severe anginal symptoms and that coronary compression occurs during systole (while coronary blood flow is higher during diastole) leads us to conclude that MB is a benign entity. On the other hand, MB has been associated, in rare occasions, with acute coronary syndromes, coronary vasospasm, and sudden cardiac death, although data are derived from small series of selected patients.^{4, 5, 13, 16, 17}

Treatment

The treatment depends on the presence of objective signs of ischemia.¹⁸ If MB is an incidental finding on angiography without signs of ischemia, treatment is not necessary. In the presence of objective signs of ischemia (exercise testing or altered coronary hemodynamics), beta-blockers and/or calcium channel blockers are recommended. Patients refractory to medical therapy may be considered

for percutaneous coronary interventions or surgical treatment.¹⁹

REFERENCES

1. Reyman HC. Disertatio de vasis cordis propriis. Med Diss Univ Göttingen 1737:1-32.
2. Möhlenkamp S, Hort W, Ge J, et al. Update on myocardial bridging. *Circulation* 2002;106:2616-2622.
3. Noble J, Bourassa MG, Petitclerc R, et al. Myocardial bridging and milking effect of the left anterior descending coronary artery: Normal variant or obstruction? *Am J Cardiol* 1976;37:993-999.
4. Angelini P, Tivellato M, Donis J, et al. Myocardial bridges: A review. *Prog Cardiovasc Dis* 1983;26:75-88.
5. Alegria JR, Herrmann J, Holmes DR, et al. Myocardial bridging. *Eur Heart J* 2005;26:1159-1167.
6. Poláček P, Kralovec H. Relation of myocardial bridges and loops on the coronary arteries to coronary occlusions. *Am Heart J* 1961;61:44-52.
7. Penther P, Blanc JJ, Bosch J, et al. L'artère interventriculaire antérieure intramurale: étude anatomique. *Arch Mal Coeur* 1977;70:1075-1079.
8. Qian JY, Zhang F, Dong M, et al. Prevalence and characteristics of myocardial bridging in coronary angiogram—data from consecutive 5525 patients. *Chin Med J* 2009;122:632-635.
9. Ge J, Jeremias A, Rupp A, et al. New signs characteristic of myocardial bridging demonstrated by intracoronary ultrasound and Doppler. *Eur Heart J* 1999;20:1707-1716.
10. Ma E, Ma G, Yu H, et al. Assessment of myocardial bridge and mural coronary artery using ECG- Gated 256-slice CT angiography: A retrospective study. *Sci World J* 2013; article ID 947876, 6 pages.
11. Lin S, Tremmel JA, Yamada R, et al. A novel stress echocardiography pattern for myocardial bridge with invasive structural and hemodynamic correlation. *J Am Heart Assoc* 2013;2:e000097.
12. Park K, Youn T-J, Park K-W, et al. Physiologic evaluation of myocardial bridging: A new analysis for an old disease. *Can J Cardiol* 2011;27:596-600.
13. Corban MT1, Hung OY1, Eshtehardi P1, et al. Myocardial bridging: Contemporary understanding of pathophysiology with implications for diagnostic and therapeutic strategies. *J Am Coll Cardiol* 2014;63:2346-2355.
14. Basso C, Thiene G, Mackey-Bojack S, et al. Myocardial bridging, a frequent component of the hypertrophic cardiomyopathy phenotype, lacks systematic association with sudden cardiac death. *Eur Heart J* 2009;30:1627-1634.
15. Migliore F, Maffei E, Perazzolo MM, et al. LAD coronary artery myocardial bridging and apical ballooning syndrome. *JACC Cardiovasc Imaging* 2013;6:32-41.
16. Rubinshtein R, Gaspar T, Lewis BS, et al. Long-term prognosis and outcome in patients with a chest pain syndrome and myocardial bridging: A 64-slice coronary computed tomography angiography study. *Eur Heart J Cardiovasc Imaging* 2013;14:579-585.
17. Kim S, Jeong MH, Kim HK, et al. Long-term clinical course of patients with isolated myocardial bridge. *Circ J* 2010;74:538-543.
18. Schwarz ER, Gupta R, Haager PK, et al. Myocardial bridging in absence of coronary artery disease: Proposal of a new classification based on clinical-angiographic data and long-term follow-up. *Cardiology* 2009;112:13-21.
19. Bockeria LA, Sukhanov SG, Orekhova EN, et al. Results of coronary artery bypass grafting in myocardial bridging of left anterior descending artery. *J Card Surg* 2013;28:218-221.