Journal of the American College of Cardiology
© 2014 by the American College of Cardiology Foundation
Published by Elsevier Inc.

Vol. 63, No. 22, 2014
ISSN 0735-1097/$36.00
http://dx.doi.org/10.1016/j.jacc.2014.01.049

Myocardial Bridging

Contemporary Understanding of Pathophysiology With Implications for Diagnostic and Therapeutic Strategies

Michel T. Corban, MD,* Olivia Y. Hung, MD, PhD,* Parham Eshtehardi, MD,* Emad Rasoul-Arzrumly, MD,* Michael McDaniel, MD,* Girum Mekonnen, MD, MPH,* Lucas H. Timmins, PhD,† Jerre Lutz, MD,* Robert A. Guyton, MD,‡ Habib Samady, MD*

Atlanta, Georgia

Patients with myocardial bridging are often asymptomatic, but this anomaly may be associated with exertional angina, acute coronary syndromes, cardiac arrhythmias, syncope, or even sudden cardiac death. This review presents our understanding of the pathophysiology of myocardial bridging and describes prevailing diagnostic modalities and therapeutic options for this challenging clinical entity. (J Am Coll Cardiol 2014;63:2346–55)
© 2014 by the American College of Cardiology Foundation

Coronary arteries that tunnel through the myocardium are seen in as many as 40% to 80% of cases on autopsy; however, functional myocardial bridging is less commonly observed on angiography (0.5% to 16.0%) and can range from 4 to 80 mm in length (1–4). Although myocardial bridges can be found in any epicardial artery, 67% to 98% occur in the left anterior descending coronary artery (LAD) (5,6). Bridges have been described as superficial or deep on the basis of 3 observations: 1) they range from 0.3 to 28 mm in depth (4,5); 2) anatomically they consist of either superficial myocardial fibers that traverse over the LAD or deep fibers that encircle the LAD (5,7); and 3) bridges >5 mm deep are less amenable to surgical myotomy (8). The hemodynamic impact of myocardial bridging depends on the thickness and length of the bridge, the orientation of the bridge relative to myocardial fibers, and the presence of loose connective or adipose tissue around the bridged segment.

From the *Andreas Gruentzig Cardiovascular Center, Division of Cardiology, Department of Medicine, Emory University School of Medicine, Atlanta, Georgia; †Wallace H. Coulter Department of Biomedical Engineering, Georgia Institute of Technology and Emory University, Atlanta, Georgia; and the ‡Division of Cardiothoracic Surgery, Department of Surgery, Emory University School of Medicine, Atlanta, Georgia. Dr. Hung is supported by the Ruth L. Kirschstein National Research Service Awards training grant (5T32HL007745). Dr. Mekonnen is supported by a grant from the National Heart, Lung, and Blood Institute of the National Institutes of Health (P01HL101398). Dr. Timmins is supported by an American Heart Association Postdoctoral Fellowship (11POST7210012). Dr. Guyton is a consultant for Medtronic, Inc. Dr. Samady receives research funding from Volcano Corp., St. Jude Medical, Toshiba, and Forest Pharmaceuticals. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose. Drs. Corban and Hung contributed equally to this work.

Manuscript received October 24, 2013; revised manuscript received January 24, 2014, accepted January 28, 2014.

Pathophysiology

Autopsy and intravascular ultrasound studies have shown that the intramural and distal segments of bridged vessels remain free from atherosclerotic disease while the proximal segment of the vessel is prone to developing atherosclerosis (9,10). Biomechanical forces may explain these observations. At the entrance of a myocardial bridge, fluid mechanics play an important role in plaque formation because disturbed near-wall blood flow patterns are a central factor in the spatial distribution of atherosclerosis (11,12). Low and oscillatory wall shear stress (WSS) are associated with increased expression of vascular cell adhesion molecule 1 (11,13) and reactive oxygen species production (14) as well as the development of a proatherogenic endothelial cell phenotype (12). Indeed, autopsy studies have shown that coronary segments immediately proximal to myocardial bridges, where WSS is low, have structurally dysfunctional, flat and polygonal endothelial cells, whereas endothelial cells lining bridged segments, where WSS is physiological or high, are structurally intact (15). Clinical studies in patients with mild atherosclerosis but without bridging have shown greater plaque progression in segments with low WSS compared with physiological or high WSS (16). In a case-control series comparing patients who had bridging with control patients (17), the wall shear rate, which is the velocity gradient perpendicular to the wall, was found to be lower proximal to the bridge compared with within the bridge.

Figure 1 shows a computational fluid dynamics model at end-systole of the LAD in a patient with a symptomatic myocardial bridge revealing an area of relatively low WSS proximal and distal to the bridge and high WSS within the bridge. Enhanced myocardial compression at the bridge

entrance also results in abrupt breakage of the propagating antegrade systolic wave, disrupting blood flow patterns, exacerbating the low WSS, and intensifying endothelial injury and the stimuli for plaque formation (18). Another proposed mechanism of plaque formation proximal to a myocardial bridge involves solid mechanical forces that result from the motion and deformation of the coronary tree and myocardial material properties. Specifically, compression within the bridge and severe vessel angulation at the junction of the bridge result in a heterogeneous stress field in the proximal segment. The induced stresses are hypothesized to be conducive to plaque development and possible fissuring in the proximal segments (18).

Within the bridge, increased mechanical loads likely contribute to constrictive vascular remodeling as an attempt to restore loads to homeostatic levels (19). These mechanisms are amplified with diastolic dysfunction that occurs with left ventricular hypertrophy. In addition, separation of the bridged segment from perivascular adipose tissue in the epicardium that is associated with proinflammatory cytokines and adipokines may be a protective mechanism against the development of atherosclerosis (20). These factors likely contribute to plaque formation proximal to myocardial bridges and exert an atheroprotective role within the bridge. The relative lack of atherosclerosis observed distal to myocardial bridges despite the presence of low WSS is not well understood. Clearly, complex and dynamic biomechanical factors influence the blood flow within and at the exit of the bridge that in aggregate appear to attenuate the proatherosclerotic stimulus of low WSS observed distal to the bridge.

Additional pathophysiological changes can induce symptoms of myocardial ischemia in previously asymptomatic patients (Fig. 2). 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

Abbreviations and Acronyms

BMS = bare-metal stent(s)
CABG = coronary artery bypass grafting
DES = drug-eluting stent(s)
FFR = fractional flow reserve
LAD = left anterior descending coronary artery
MSCT = multiple-slice computed tomography
PCI = percutaneous coronary intervention
TVR = target vessel revascularization
WSS = wall shear stress

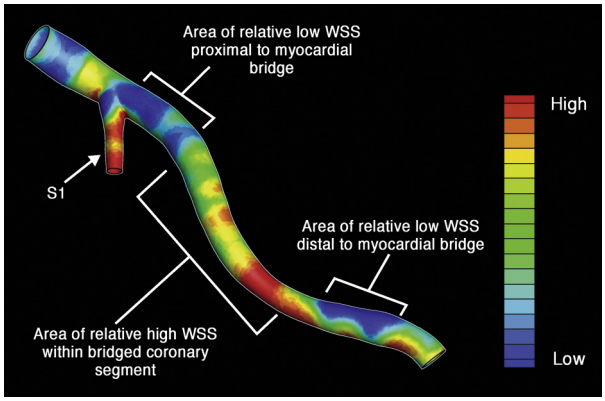

Figure 1 **Relative WSS Profile of the LAD in the Context of Myocardial Bridging**

Relative WSS profile of a 3-dimensional angiographically reconstructed LAD during systole from a patient with myocardial bridging. Coronary segments proximal and distal to the myocardial bridge show relatively low WSS compared with the bridged segment. LAD = left anterior descending coronary artery; S1 = first septal branch; WSS = wall shear stress. Image created by Craig Skaggs.

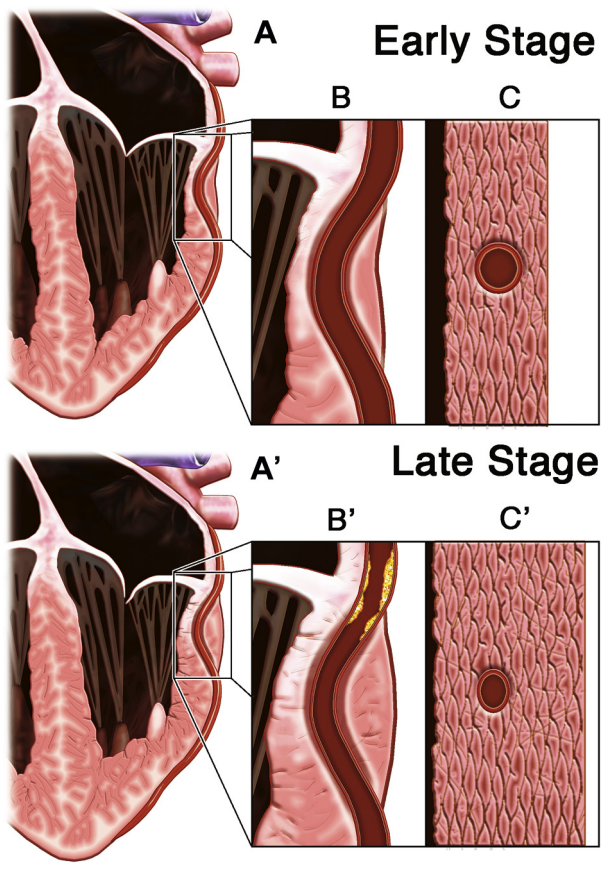

Figure 2 **Schematic Diagram of the Effects of Aging on the Myocardial Bridge**

(A) Heart with myocardial bridging, early stage. **(B)** Longitudinal view of the bridged vessel. **(C)** Cross-sectional view of the vessel in the middle of the myocardial bridge. **(A′)** Heart with myocardial bridging, late stage, with ventricular hypertrophy and diastolic dysfunction. **(B′)** Longitudinal view of the bridged vessel, with hypertrophied muscle and plaque progression proximal to the bridge. **(C′)** Cross-sectional view of the vessel in the middle of the myocardial bridge showing hypertrophied muscle and negative remodeling of the vessel with decreased lumen diameter. Images created by Clare Wang and Craig Skaggs.

Download English Version:

https://daneshyari.com/en/article/2945377

Download Persian Version:

https://daneshyari.com/article/2945377

Daneshyari.com