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Editorial

When Is It a Bridge Too Far?

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Myocardial bridge (MB) is a congenital anomaly of the coronary arteries in which a major epicardial coronary artery has a segment that courses intramurally through the myocardium.[1] The prevalence of MBs varies based on the method used to assess the coronary anatomy—it has been reported in up to 30% of patients diagnosed with cardiac computed tomography angiography,[1] between 5% and 86% in autopsy studies, and between 0.5% and 33% with coronary angiography.[2] Historically, MBs in the general population have been considered a benign entity; however, in patients with MB presenting with angina, the evaluation and management can be challenging. It is imperative to evaluate whether the MB is hemodynamically significant and whether other mechanisms such as coronary microvascular dysfunction (CMD) or epicardial spasm may be contributing to the anginal symptoms.

In this issue of *JSCAI*, Allan et al[3] evaluated 30 patients with angina and no obstructive coronary artery disease (ANOCA) that was initially attributed to MB and therefore referred to University of Chicago Medical Center for evaluation by a robotic surgical unroofing procedure. Patients with significant coronary disease (defined as stenosis >50% or fractional flow reserve [FFR] ≤0.8) or valvular disease were excluded. The authors defined MB as hemodynamically significant when resting flow reserve (RFR) was ≤0.76 after administration of escalating doses of dobutamine (maximum, 40 mcg/kg/min). In addition to testing for hemodynamic MB, all patients underwent invasive coronary functional testing utilizing the thermodilution method to evaluate for the prevalence of endothelium-independent CMD (defined as coronary flow reserve [CFR] <2.0 or index of microvascular resistance [IMR] ≥25 in response to 140 mcg/kg/min adenosine), microvascular spasm (defined as the presence of chest pain and ischemic ECG changes in response to 40 mcg [low dose] acetylcholine [Ach] in the absence of an ischemic FFR ≤0.80) and epicardial spasm (defined as angiographic spasm ≥90% or ischemic FFR ≤0.80 in response to 100 mcg of Ach).

The authors avoided a wire exchange between adenosine and Ach testing, which resulted in the assessment of epicardial and microvascular spasms with a wire in place. Ach CFR and IMR were recorded and considered abnormal if CFR was <1.5 or IMR >31, although they were not used to define microvascular spasm. The authors concluded that only 47% of patients (n = 14/30) with angina and MB actually had a hemodynamically significant MB using the RFR cutoff ≤0.76. Interestingly, when FFR was utilized for the detection of hemodynamically significant MB using a cutoff of ≤0.80, the FFR failed to detect 11 out of the 14 patients previously diagnosed using RFR. In the majority of patients, the presence of MB alone did not explain the anginal symptoms. There was a high prevalence of coronary functional abnormalities in patients with MB, with up to 77% of patients (n = 23/30) demonstrating either endothelium-independent CMD (60%, n = 18/30), microvascular spasm (30%, n = 9/30), or epicardial spasm (37%, n = 11/30). Only 7 out of 30 patients (23%) with MB and anginal symptoms did not demonstrate a functional abnormality, including 2 patients who demonstrated mild vasoconstriction (20%-89%) with Ach in the absence of chest pain or ischemic ECG changes. Moreover, the authors did not observe a relationship between presence of coronary functional abnormalities and hemodynamically significant MB.

There are a number of important implications of this study. The high prevalence of coronary functional abnormalities in patients with ANOCA and MB (77%) referred for an MB unroofing procedure supports the fact that anginal symptoms in patients with MB are usually multifactorial. In fact, surgical or invasive MB procedures have been associated with increased rates of complications such as high rates of graft failure due to competitive flow[4] and high rates of in-stent restenosis.[5] Furthermore, MB unroofing procedures have a high incidence of recurrent chest pain (up to 60%) at 3-year follow-up.[6] Based on the findings in this study, it is likely that patients with MB may have had underlying coronary functional abnormalities resulting in ongoing symptoms. Therefore, a complete evaluation in MB patients presenting with angina should include coronary physiology testing prior to referral for a surgical unroofing procedure for MB. Diagnosis of

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coronary functional abnormality can also guide targeted medical therapy based on abnormal pathways, such as β-blockers in endothelium-independent CMD and calcium channel blockers and nitrates in coronary spasms.[7] This adds to the current literature, which supports that MB is a potential mechanism of endothelial dysfunction for patients with ANOCA.[8,9] Although the underlying pathophysiology between the 2 entities is not well understood, regional alterations in vasoactive substances such as nitric oxide and endothelin-1, and high intravascular pressure and shear stress conditions within the bridged segment might be the basis for the development of pathologic vasoconstriction and endothelial dysfunction in patients with MB.[8] Finally, it is worth noting that there is also considerable variability in both technique and definitions that impact the diagnosis of alternative mechanisms for chest pain as well. For example, it has been reported that CFR may be overestimated using thermodilution compared with Doppler wire techniques; hence, using CFR <2.0 may even underestimate the percentage of patients with CMD.[10]

Therefore, how do these results impact the already challenging diagnosis and management of symptomatic MB? Given that in more than half of the referred patients for a surgical unroofing procedure the MB was deemed hemodynamically insignificant and that alternative causes of chest pain were detected in 77% of patients, coronary functional testing in patients with MB is critically important to have a more complete understanding of the cause(s) of a patient's chest pain. Future research should focus on the pathophysiologic links between MB, CMD, and spasm along with the impact of medical and invasive therapeutic approaches in this patient cohort.

Declaration of competing interest

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