JSCAI
The official journal of the Society for Cardiovascular Angiography & Interventions

Editorial

The Challenge of Myocardial Bridging

Samit M. Shah, MD, PhD [a,b], Odayme Quesada, MD [c,d], Timothy D. Henry, MD [d,*]

[a] Section of Cardiovascular Medicine, Department of Medicine, Yale University School of Medicine, New Haven, Connecticut; [b] Department of Medicine, VA Connecticut Healthcare System, West Haven, Connecticut; [c] Women's Heart Center, The Christ Hospital Heart and Vascular Institute, Cincinnati, Ohio; [d] The Carl and Edyth Lindner Center for Research and Education at The Christ Hospital, Cincinnati, Ohio

Coronary artery myocardial bridging is common and, in most cases, a bystander that is incidentally recognized at the time of invasive coronary angiography or cardiovascular computed tomography angiography (CCTA). The prevalence of myocardial bridges has been reported in multiple series and ranges from 2% to 7% with invasive coronary angiography but is specifically higher with CCTA (19%-22%) and at autopsy (as high as 45%).[1-4]

Although most observed myocardial bridges are clinically benign, there are numerous case reports of ischemia or even infarction that have been attributed to dynamic compression in bridge segments.[5] The features of myocardial bridges that are associated with clinical significance are an intramyocardial depth of >2 to 4 mm and a length of 10 to 30 mm.[6] Notably, systolic compression alone is unlikely to cause clinically significant ischemia. The correlation between intravascular imaging, Doppler flow assessment, and quantitative coronary angiography has demonstrated delayed luminal expansion in early diastole and abrupt early diastolic flow acceleration in constricted lumens.[7,8] This results in a decrement in the coronary perfusion pressure across the bridge segment, which can be measured using either a full-cycle fractional flow reserve or nonhyperemic ratio such as instantaneous wave-free ratio or resting full-cycle ratio.[7] Hemodynamic perturbations in bridge segments result in altered endothelial cell morphology at the entrance to tunneled intramyocardial segments related to low wall shear stress and changes in the expression of vasoactive agents such as endothelial nitric oxide synthase, endothelin-1, and angiotensin-converting enzyme.[8,9] As a result, the presence of myocardial bridging is also associated with both epicardial and microvascular endothelial dysfunction.[10,11]

In many cases, the treatment of symptomatic myocardial bridging involves pharmacologic therapy to increase diastolic filling time and reduce myocardial contractility.[4,11] This is most commonly achieved with beta-blockers or nondihydropyridine calcium-channel blockers; however, in refractory cases, surgical management may be considered.[12] Surgical operations for relief from physiologically significant bridging can include surgical myotomy of the myocardial bridge ("unroofing") or coronary artery bypass graft surgery. The long-term success of these operations has not been well described, and single-center registries have reported a rate of recurrent symptoms of up to 40%.[13] Percutaneous coronary intervention (PCI) with stent placement can effectively resolve the hemodynamic pressure gradient across bridge segments; however, this has not become routine practice because of high rates of in-stent restenosis (75% for bare metal stents and 25% for drug-eluting stents) and concerns for stent fracture or perforation at the time of intervention.[4]

In this issue of *JSCAI*, Sawhney et al[14] report a case of a patient who underwent surgical unroofing for the management of a symptomatic myocardial bridge but developed recurrent symptoms within 2 years and successfully underwent PCI with stenting, leading to symptom relief and resolution of a provocable pressure gradient across the bridge with dobutamine. It is important to note that a pharmacologic positron emission tomography scan did not show evidence of ischemia, and post-PCI follow-up was performed only at 3 months.

To illustrate the challenge of diagnosis and treatment in patients with myocardial bridges, we briefly present a similar case. A 59-year-old White woman with a history of hyperlipidemia, hypertension, and chronic pain due to a prior motor vehicle accident had a history of several years of exertional chest pressure and dyspnea, consistent with Canadian Cardiovascular Society class III symptoms. She presented to the emergency department with an increase in the intensity of symptoms, leading to CCTA, which demonstrated no obstructive coronary artery disease and no specific mention of a myocardial bridge. An echocardiogram demonstrated normal cardiac function, with no significant abnormalities. In addition to her exertional symptoms, she reported episodic resting chest pain, which responded to nitroglycerin. Given the progressive symptoms, she was referred for coronary functional angiography (CFA) for the evaluation of the presence of coronary microvascular dysfunction and/or coronary artery vasospasm. Angiography revealed mild coronary artery disease (<30%), with evidence of a mild, midleft, anterior descending (LAD) artery myocardial bridge. CFA using a Doppler flow

DOI of original article: https://doi.org/10.1016/j.jscai.2022.100559.
Keywords: coronary artery; myocardial bridging; percutaneous coronary intervention; physiology.
* Corresponding author: Tim.Henry@thechristhospital.com (T.D. Henry).

https://doi.org/10.1016/j.jscai.2022.100545
Received 4 November 2022; Accepted 7 November 2022
Available online 4 January 2023

wire revealed a markedly abnormal coronary flow reserve of 1.5 in the LAD, consistent with severe endothelial-independent coronary microvascular dysfunction. Intracoronary infusion of middose acetylcholine (36.4 μg) resulted in vasoconstriction in the mid-to-distal LAD, consistent with endothelial-dependent microvascular dysfunction. With high-dose (108 μg) acetylcholine infusion, she developed 70% epicardial coronary artery vasospasm at the distal portion of the myocardial bridge. With intracoronary nitroglycerin, she had more prominent systolic compression of the myocardial bridge. Therefore, her diagnosis was a symptomatic myocardial bridge with concomitant endothelial dysfunction and endothelium-dependent microvascular dysfunction, and mechanical treatment of the bridge alone would be unlikely to relieve her symptoms. She was treated with a statin, angiotensin-receptor blocker, high-dose beta-blocker, dihydropyridine calcium-channel blocker, and cardiac rehabilitation, with improvement to stable Canadian Cardiovascular Society class II symptoms.

These cases demonstrate the challenge of managing patients with symptomatic myocardial bridging and the need for complete evaluation of coronary physiology, including CFA, for the presence of concomitant coronary microvascular dysfunction and coronary vasoconstriction. Surgical therapies, such as unroofing or coronary artery bypass, may be therapeutic in some cases; however, many patients develop recurrent symptoms or complications, including pericarditis. There may be a role of PCI in the management of patients with symptomatic and physiologically significant bridging as a standalone therapy or for those with recurrent symptoms after surgical therapy; however, before proceeding with mechanical relief of the bridge, either surgically or percutaneously, complete evaluation and treatment of coronary microvascular dysfunction and vasospasm must be considered given the frequent prevalence of vasoconstriction and microvascular dysfunction at the site of myocardial bridging. More long-term data are direly needed regarding the technical success and long-term outcomes if PCI is to be considered for the management of myocardial bridging.

Declaration of competing interest

Odayme Quesada is funded in part by NIH K23 HL151867. Samit M. Shah receives research support from Women's Health Research at Yale, Abbott Vascular Inc, and the US Food and Drug Administration to support projects within the Yale-Mayo Clinic Center of Excellence in Regulatory Science and Innovation (CERSI, U01FD005938). Timothy D. Henry reported no financial interests.

Funding sources

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

References

1. Irvin RG. The angiographic prevalence of myocardial bridging in man. *Chest*. 1982; 81(2):198–202. https://doi.org/10.1378/chest.81.2.198
2. Hostiuc S, Negoi I, Rusu MC, Hostiuc M. Myocardial bridging: a meta-analysis of prevalence. *J Forensic Sci*. 2018;63(4):1176–1185. https://doi.org/10.1111/1556-4029.13665
3. Nakanishi R, Rajani R, Ishikawa Y, Ishii T, Berman DS. Myocardial bridging on coronary CTA: an innocent bystander or a culprit in myocardial infarction? *J Cardiovasc Comput Tomogr*. 2012;6(1):3–13. https://doi.org/10.1016/j.jcct.2011.10.015
4. Sternheim D, Power DA, Samtani R, Kini A, Fuster V, Sharma S. Myocardial bridging: diagnosis, functional assessment, and management: JACC state-of-the-art review. *J Am Coll Cardiol*. 2021;78(22):2196–2212. https://doi.org/10.1016/j.jacc.2021.09.859
5. Tauth J, Sullebarger T. Myocardial infarction associated with myocardial bridging: case history and review of the literature. *Cathet Cardiovasc Diagn*. 1997;40(4): 364–367. https://doi.org/10.1002/(sici)1097-0304(199704)40:4<364::aid-ccd9>3.0.co;2-7
6. Virmani R, Farb A, Burke AP. Ischemia from myocardial coronary bridging: fact or fancy? *Hum Pathol*. 1993;24(7):687–688. https://doi.org/10.1016/0046-8177(93)90001-w
7. Escaned J, Cortés J, Flores A, et al. Importance of diastolic fractional flow reserve and dobutamine challenge in physiologic assessment of myocardial bridging. *J Am Coll Cardiol*. 2003;42(2):226–233. https://doi.org/10.1016/s0735-1097(03)00588-6
8. Bourassa MG, Butnaru A, Lespérance J, Tardif JC. Symptomatic myocardial bridges: overview of ischemic mechanisms and current diagnostic and treatment strategies. *J Am Coll Cardiol*. 2003;41(3):351–359. https://doi.org/10.1016/s0735-1097(02)02768-7
9. Masuda T, Ishikawa Y, Akasaka Y, Itoh K, Kiguchi H, Ishii T. The effect of myocardial bridging of the coronary artery on vasoactive agents and atherosclerosis localization. *J Pathol*. 2001;193(3):408–414. https://doi.org/10.1002/1096-9896(2000)9999:9999<::AID-PATH792>3.0.CO;2-R
10. Sara JD, Corban MT, Prasad M, et al. Prevalence of myocardial bridging associated with coronary endothelial dysfunction in patients with chest pain and non-obstructive coronary artery disease. *EuroIntervention*. 2020;15(14):1262–1268. https://doi.org/10.4244/EIJ-D-18-00920
11. Corban MT, Hung OY, Eshtehardi P, et al. Myocardial bridging: contemporary understanding of pathophysiology with implications for diagnostic and therapeutic strategies. *J Am Coll Cardiol*. 2014;63(22):2346–2355. https://doi.org/10.1016/j.jacc.2014.01.049
12. Möhlenkamp S, Hort W, Ge J, Erbel R. Update on myocardial bridging. *Circulation*. 2002;106(20):2616–2622. https://doi.org/10.1161/01.cir.0000038420.14867.7a
13. Hemmati P, Schaff HV, Dearani JA, Daly RC, Lahr BD, Lerman A. Clinical outcomes of surgical unroofing of myocardial bridging in symptomatic patients. *Ann Thorac Surg*. 2020;109(2):452–457. https://doi.org/10.1016/j.athoracsur.2019.07.005
14. Sawhney R, Christensen J, Schaffer J, Al-Aziz K. Percuataneous coronary intervention in myocardial bridging. *J Soc Cardiovasc Angiogr Interv*. 2023;2(2):100563.