Big Hearts, Little Reserve: Coronary Flow Velocity Reserve After Bypass Grafting in Patients With Left Ventricular Hypertrophy

Zaid Abdelsattar, MD, MS, and Juan A. Crestanello, MD

In this issue of *Seminars*, Honda et al[1] evaluate myocardial microvascular function in patients with severe left ventricular hypertrophy (LVH) after myocardial revascularization with CABG. The authors used coronary flow velocity reserve (CFVR), measured by transthoracic echocardiography Doppler of the LAD, to assess microvascular function before and after LIMA to LAD CABG. CFVR represents the ratio between maximal (stimulated) coronary blood flow, induced by a coronary vasodilator, and baseline (resting) blood flow. As an inexpensive, radiation-free and noninvasive modality, CFVR is useful to confirm graft patency and evaluate for functional changes of myocardial ischemia. The authors found that although CFVR improves postoperatively in all patients, it does not reach expected normal values in patients with LVH even with a patent LIMA graft. The findings highlight the effects of microvascular dysfunction.

Coronary microvascular dysfunction is increasingly recognized as a source of persistent ischemia in patients with ischemic heart disease and is associated with poor prognosis.[2–4] It can result in an impaired ability to increase coronary blood flow in response to increased oxygen demand (equivalent to flow limiting epicardial coronary stenosis), or it can result in microvascular spasm. In patients with LVH, the increased oxygen demand is further hindered by adverse hemodynamic changes including high left ventricular pressures, relative low coronary perfusion pressure compared to intracavitary pressures, and increased extravascular compressive forces.

The paper by Honda et al has several limitations that affect the external validity and reliability of the findings. The number of patients with LVH is small and only represents a minority of the study population. Second, the postoperative CFVR measurement was only performed once and at varying time points. Whether CFVR improves with time after improved epicardial coronary perfusion is not known. Lastly, the clinical effects of the reduced CFVR are not known in those patients. It would be interesting to know whether these patients have microvascular angina despite patent grafts.

Notwithstanding these limitations, the paper sheds light on microvascular dysfunction in patients with LVH after CABG. As we gain a better understanding of the mechanisms behind microvascular dysfunction, ongoing ischemia, and adverse outcomes, we have to keep in mind the three-compartment model of the coronary vasculature (epicardial coronary arteries, prearterioles, and the coronary microvasculature). Epicardial revascularization in the face of microvascular dysfunction may not be the only treatment for these patients. After all, you can lead a horse to water, but you can't make it drink.

Drs Abdelsattar and Crestanello.

Central Message

Microvascular dysfunction associated with left ventricular hypertrophy may limit the beneficial effects of coronary revascularization.

REFERENCES

1. Honda K, Yuzaki M, Kaneko M, et al: Functional evaluation of the myocardial ischemia after coronary artery bypass surgery using coronary flow velocity reserve in left ventricular hypertrophy. Semin Thorac Cardiovasc Surg 31:166–173, 2019
2. Levy D, Garrison R, Savage D, et al: Prognostic implications of echocardiographically determined left ventricular mass in the Framingham heart study. N Engl J Med 322:1561–1566, 1990. https://doi.org/10.1056/NEJM199005313222203
3. Camici P, Crea F: Coronary microvascular dysfunction. N Engl J Med 356:830–840, 2007. https://doi.org/10.1056/NEJMra061889
4. Kaski J, Crea F, Gersh B, et al: Reappraisal of ischemic heart disease. Circulation 138:1463–1480, 2018. https://doi.org/10.1161/CIRCULATIONAHA.118.031373

Department of Cardiovascular Surgery, Mayo Clinic, Rochester, Minnesota

Disclosures: The authors have no conflicts of interest related to the content of this article.

Address reprint requests to Juan A. Crestanello, MD, Department of Cardiovascular Surgery, Mayo Clinic, 200 First St SW, Rochester, MN 55905. E-mail: crestanello.juan@mayo.edu

DOI of original article: http://dx.doi.org/10.1053/j.semtcvs.2018.09.012.

https://doi.org/10.1053/j.semtcvs.2019.02.013