Myocardial Bridging in a Man With Non–ST-Elevation Myocardial Infarction

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A 75-year-old man with hyperlipidemia was admitted with substernal chest tightness associated with an elevated cardiac-specific troponin. He had experienced symptoms of an upper respiratory tract infection with fever, cough, and decreased oral intake over the previous 2 weeks. He presented ≈4 hours after symptom onset with an initial troponin-T concentration of 0.29 ng/mL, eventually peaking at 0.31 ng/mL before downtrending. The ECG showed T-wave flattening in aVL with no previous ECG for comparison. Appropriate medical therapy for non–ST-elevation myocardial infarction was initiated, and the patient was taken to the cardiac catheterization laboratory.

Coronary angiography was performed from the right radial artery approach. There were no significant lesions in the right coronary or left circumflex arteries. There was a 40% lesion in the midportion of the left anterior descending artery associated with myocardial bridging (Figure 1 and Movie I in the online-only Data Supplement), which became more prominent with the intracoronary administration of 200 μg of nitroglycerin. A coronary pressure wire (RADI Medical Systems, Uppsala, Sweden) was introduced across the segment of myocardial bridging. The distal-to-proximal pressure ratio at baseline was 0.86, and the fractional flow reserve (FFR) obtained after the intravenous administration of adenosine at 180 μg·kg⁻¹·min⁻¹ was 0.76 (Figure 2).

Myocardial bridging refers to an intramyocardial course of an epicardial coronary artery. The depth and length of the tunneled segment may vary, ranging from short, superficial overlying segments of myocardium to tunneling up to 10 mm below the epicardial surface. Although an epicardial coronary artery course is typical among primates, lower mammals commonly manifest embedded coronary arteries. Myocardial bridging in humans is largely considered a congenital evolutionary remnant.

The prevalence of angiographically detected myocardial bridging is generally reported to be <5%. The finding of myocardium overlying the epicardial coronary arteries is much more common at autopsy, being observed in 30% to 85% of adults.¹ Prevalence is particularly high among heart transplant recipients and patients with hypertrophic cardiomyopathy, with de novo myocardial bridging reported in the former group. The midportion of the left anterior descending artery is most commonly involved.

Although myocardial bridging is typically benign, numerous clinical presentations have been attributed to myocardial bridging, including ischemia, infarction, ventricular tachycardia, and sudden cardiac death. Vessel compression is classically considered to occur primarily during systole, although numerous studies have shown the encroachment of compression into mid- or even late-diastole with decreased diastolic epicardial, and coronary sinus blood flow, as well.²⁻³ Perhaps compounded in some patients by left ventricular diastolic dysfunction, microvascular disease, or vasospasm, this diminished flow results in a myocardial supply/demand mismatch and elevated coronary sinus lactate concentration.¹⁻² This mismatch may be intensified by tachycardia or high adrenergic tone.

Myocardial bridging generally comes to clinical attention with coronary angiography, which is the gold standard for diagnosis. The administration of intracoronary nitroglycerin classically provokes more severe compression of the tunneled segment. Bridging may also be detected with intravascular ultrasound, optical coherence tomography, intracoronary Doppler, cardiac MRI, and coronary computed tomography. The hemodynamic importance of a bridging segment may be assessed with mean FFR measurement during intravenous adenosine administration, although the addition of diastolic FFR obtained during dobutamine infusion may be more sensitive for physiologically relevant lesions.⁴ Atherosclerotic plaques are commonly observed in the coronary segment immediately proximal to the myocardial bridge but typically not within the tunneled segment itself, a finding which has been attributed to flow dynamics and endothelial shear stress.¹⁻²

Medical management with negative inotropic and negative chronotropic agents is considered first-line therapy. Although...
percutaneous intervention for myocardial bridging has been reported, there is concern for poor treatment durability with in-stent restenosis rates of 36% to 75% with bare metal stents and 18% to 25% with drug-eluting stents. There may also be increased risk for coronary perforation, stent fracture, and in-stent thrombosis. Surgical unroofing or bypass are further options for patients with refractory symptoms and amenable anatomy.

The patient reported here had no further symptoms with the resolution of his infection-related tachycardia and the addition of metoprolol succinate 50 mg daily. The FFR measurement obtained was of intermediate significance, but, given the elevated complication rate and poor durability of interventional therapy, medical management would have remained the preferred initial strategy even in the setting of a definitely significant FFR. One month after this event, the patient was able to perform 10.1 METS on a Bruce protocol with a blunted maximal heart rate of 115 beats per minute on metoprolol and no perfusion defect by single-photon emission computed tomography.

Disclosures
Dr Bhatt discloses the following relationships: Advisory Board: Cardax, Elsevier Practice Update Cardiology, Medscape Cardiology, Regado Biosciences; Board of Directors: Boston VA Research Institute, Society of Cardiovascular Patient Care; Chair: American Heart Association Get With The Guidelines Steering Committee; Data Monitoring Committees: Duke Clinical Research Institute, Harvard Clinical Research Institute, Mayo Clinic, Population Health Research Institute; Honoraria: American College of Cardiology (Senior Associate Editor, Clinical Trials and News, ACC.org), Belvoir Publications (Editor in Chief, Harvard Heart Letter), Duke Clinical Research Institute (clinical trial steering committees), Harvard Clinical Research Institute (clinical trial steering committee), HMP Communications (Editor in Chief, Journal of Invasive Cardiology), Journal of the American College of Cardiology (Associate Editor; Section Editor, Pharmacology), Population Health Research Institute (clinical trial steering committee), Slack Publications (Chief Medical Editor, Cardiology Today’s Intervention), WebMD (CME steering committees); Other: Clinical Cardiology (Deputy Editor); Research Funding: Amarin, AstraZeneca, Bristol-Myers Squibb, Eisai, Ethicon, Forest Laboratories, Ischemix, Medtronic, Pfizer, Roche, Sanofi Aventis, The Medicines Company; Unfunded Research: FlowCo, PLx Pharma, Takeda. The other authors report no conflicts.

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