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EDITORIAL COMMENT

Intramural Course of Coronary Arteries

A Bridge Too Far No More*

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In this issue of *iJACC*, Dimitriu-Leen et al. (1) present an important analysis of 1,000 patients who underwent diagnostic coronary computed tomography for evaluation of chest pain among patients without obstructive coronary artery disease (CAD). No fewer than 210 (22%) of patients had a documented intramural course of a coronary artery, which was usually the ramus intermedius or the left anterior descending artery. Follow-up was >95% complete at 5 years, and exceedingly low event rates were found, with a 5-year case fatality rate of 2.4%, an incidence of myocardial infarction of 0.7%, and hospitalization for unstable angina of 1.4%. Cumulative event rates were almost 5% or approximately 1% per year. There was no significant difference in composite event rates between patients with or without an intramural course (4.5% vs. 4.6%) by 5 years. The investigators appropriately concluded that incidentally detected myocardial bridging was a benign anomaly when found in a low-risk population without obstructive CAD and was not associated with an increased risk of coronary events or cardiac death.

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It has long been known that normally epicardial coronary arteries can "dip" into the myocardium for variable lengths of their course, a phenomenon colloquially known as myocardial bridging. First recognized at autopsy by Reyman in 1737 (2), this phenomenon was also observed in the earliest days of coronary angiography (3) and was mentioned in the authoritative review by Angelini on coronary artery anomalies (4). Not surprisingly, the prevalence of myocardial bridging varies with diagnostic techniques, being noted in <15% of patients assessed angiographically, but it is as high as 80% in some autopsy series. Clinicians have been vexed about conflicting data on the association of bridging with myocardial ischemia and infarction. On the one hand, post-mortem examinations have revealed evidence of chronic myocardial ischemia and fibrosis in myocardial segments subserved by the bridged artery (5), and numerous case reports have documented an association among intramural coronary arteries with myocardial ischemia, infarction, and even sudden cardiac death. Not surprisingly, such associations are sufficient to cause worry and concern on the behalf of patients and their clinicians.

On the other hand, mechanistic and translational work has suggested a lower prevalence of obstructive atherosclerosis among bridged segments. This hypothesis has been supported by direct pathologic observation, and demonstration of diminished expression of endothelial nitric oxide synthase and Endothelin-1 in bridged segments at autopsy (6). In an attempt to resolve this apparent contradiction, invasive imaging and physiologic tools such as intravascular ultrasonography and fractional flow reserve have been used to better understand the pathophysiology of myocardial bridging. Because most coronary blood flow occurs during diastole, it has long been assumed that intermittent systolic compression will not result in clinically meaningful myocardial ischemia. Invasive studies with fractional flow reserve, with and without inotropic stimulation and induction of tachycardia, have demonstrated this is not always the case. Individual patients may have diminished coronary blood flow under conditions of physiologic (and clinical) stress. Similar findings were noted by Angelini in anomalous right coronary arteries arising from the left coronary sinus when subjected to saline-atropine-dobutamine infusion.

It should be recognized that an intramural coronary course is a specific type of coronary anomaly, one that is 6 times more common than the more

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malignant anomalous left coronary artery originating from the right sinus. Thus, intramural coronary arteries often represent a challenge to the clinician, especially when patients have no or atypical symptoms. Intravascular ultrasound has been proposed as a potential investigational technique to evaluate the reduction in cross-sectional area of the coronary artery before and after inotropic stimulation. A significant reduction in cross-sectional area may be indicative of potential hemodynamic compromise with exercise (6). Treatment of patients with anatomical bridging and physiological evidence of diminished anterograde coronary blood flow has been empirical and may include medical therapy, off-label coronary stenting, and surgical unroofing, all of which have been used with variable degrees of success.

Because of the frequency with which myocardial bridging over coronary arteries is observed, the greater question has always been what to recommend to patients with incidentally found intramural coronary artery courses. Previous series have reported small numbers of patients (7–10) that had a generally good prognosis. Thus, the present study with its 210 incidentally found patients, coupled with a similar number in various angiographic series, serve to confirm the impressions of most cardiologists that in most cases, myocardial bridging is a benign incidental finding. Most patients can be reassured that this anomaly will not have an adverse impact on their risk of coronary events or sudden cardiac death. Having said that, clinicians should remain attuned to the rare patient with angina pectoris and provocable myocardial ischemia who may be the exception to the rule. The investigators should be congratulated on an important and practical contribution to the literature.

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REFERENCES

1. Dimitriu-Leen AC, van Rosendael AR, Smit JM, et al. Long-term prognosis of patients with intramural course of coronary arteries assessed with CT angiography. J Am Coll Cardiol Img 2017;10:1451–8.

2. Reyman HC. Diss. De vasis cordis propriis. Bibl Anat 1737;2:359–79.

3. Portmann WC, Iwig J. Die intramurale koronarie im angiogram. Fortschr Rontegenstr 1960;92:129–32.

4. Angelini P, Uribe C, Monge J, Tobis JM, Elayda MA, Willerson JT. Origin of the right coronary artery from the opposite sinus of Valsalva in adults: characterization by intravascular ultrasonography at baseline and after stent angioplasty. Catheter Cardiovasc Interv 2015;86:199–208.

5. Morales AR, Romanelli R, Tate LG, Boucek RJ, de Marchena E. Intramural left anterior descending coronary artery: significance of the depth of the muscular tunnel. Hum Pathol 1993;24:693–701.

6. 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;193:408–14.

7. Harikrishnan S, Sunder KR, Tharakan J, et al. Clinical and angiographic profile and follow-up of myocardial bridges: a study of 21 cases. Indian Heart J 1999;51:503–7.

8. Yano K, Yoshino H, Taniuchi M, et al. Myocardial bridging of the left anterior descending coronary artery in acute inferior wall myocardial infarction. Clin Cardiol 2001;24:202–8.

9. Kitazume H, Kramer JR, Krathamer D, El Tobi S, Proudfit WL, Sones FM. Myocardial bridges in obstructive hypertrophic cardiomyopathy. Am Heart J 1983;106:131–5.

10. Navarro-Lopez F, Soler J, Magrina J, et al. Systolic compression of coronary artery in hypertrophic cardiomyopathy. Int J Cardiol 1986;12:309–20.

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