Myocardial Bridging
A Congenital Variant as an Anatomic Risk Factor for Myocardial Infarction?

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According to postmortem studies, coronary atherosclerosis can be substantial and reach a high prevalence of advanced lesions, including atheroma and fibroatheroma in young adults.1,2 Risk factors associated with coronary atherosclerosis such as dyslipidemia, smoking, diabetes mellitus, and hypertension have been identified. The development of atherosclerosis seems to be related to the magnitude and duration of exposure.3 Although research concentrates on identifying genetic markers in genome-wide association studies, there is still a lack of knowledge about the individual risk of developing coronary atherosclerosis.4 When atherosclerosis presents as acute coronary syndromes, mortality may be high.5 Plaque rupture and erosion have been identified as major underlying pathological-anatomic characteristics,6 but some suspect other mechanisms.7 In this issue of Circulation, Ishikawa et al8 present evidence that myocardial bridging may play a role as a congenital anatomic risk factor for coronary atherosclerosis and myocardial infarction.

Myocardial bridging results when myocardial tissue covers part of the left anterior descending coronary artery, resulting in a tunneled arterial segment, which can be regarded as a congenital variant.9–11 The prevalence is reported to be >50% at autopsy.12 Clinically, the diagnosis of myocardial bridging is established by coronary angiography demonstrating a systolic compression, described as a “milking effect,”13 but it was present in only <5% of cases recently in a large number of Chinese patients.14 When nitroglycerin was used as a provocation test to unmask the myocardial bridging,15 an incidence of 16.1% was described in another large study of >5000 patients.16 The incidence as determined by coronary angiography may be as high as 40% when positive inotropic agents are used in addition to nitroglycerin.17

Myocardial bridging also has been studied with intracoronary ultrasound.18–21 In addition to systolic compression, a new characteristic, the half-moon phenomenon, has been described and is attributed to adipose tissue partly surrounding the tunneled segment. For the first time, the physician can directly demonstrate that the step-down and step-up angiographic appearance of the bridged segment is related to the fact that the artery not only is covered by myocardial tissue but also may be imbedded deep in the septal myocardium, reaching the subendocardial surface of the right ventricle.12

For a long time, myocardial bridging was regarded as a variant without any hemodynamic or physiological relevance because left coronary flow is maximal during diastole and not systole. But, frame-by-frame analysis of intracoronary ultrasound images has revealed delayed relaxation during early diastole, the period of highest coronary blood flow.18 The considerable delay in blood flow and reduced distal coronary pressure are presumed to impaire coronary vasodilator reserve.22 This assumption is strengthened by studies with intracoronary Doppler in patients with myocardial bridging. In early diastole, a peak of high flow velocity was detected, followed by a plateau of low velocity, which could be related to high pressure gradients obtained proximal to, within, and distal to bridged segments.21,23 This represents a typical sign of myocardial bridging: the “fingertip” phenomenon (“peak-plateau” phenomenon). In addition, an abnormal coronary flow reserve (>3.0 regarded as normal) distal to the bridged segment was described.21,23–25 As proof of concept, the abnormal flow pattern, high intracoronary pressure gradients, and reduced flow reserve disappeared after coronary stent placement.21,25

The intracoronary ultrasound studies also revealed that the tunneled segment is free of disease, a most stimulating aspect for research. We hypothesized that this may also be related to the compression of the tunneled segment during systole, enhancing the lymph drainage of the vessel wall that is important for the prevention of lipid accumulation and disease development. However, extended atherosclerotic plaques were detected proximal to the bridged segment, even in patients with normal-appearing coronary angiograms, whereas the bridged segments were free of disease,20 confirming previous pathoanatomic studies.26–28 An enhanced endothelial cell permeability, accumulation of apolipoprotein B, and proliferating cell nuclear antigens in smooth muscle cells proximal to the bridged segment but not in the tunneled segment were characteristic features.29 In addition, hemodynamic forces may explain the possibly enhanced atherosclerotic process. The high pressure gradients may increase the local wall tension and stretch and induce endothelial injury and plaque fissuring with subsequent thrombus formation, which is supported by autopsy and clinical stud-
ties. However, it remained unclear whether the amount and distribution of atherosclerosis in the proximal segment are different from that observed in a control population because myocardial bridging is a common condition and may coincide with another frequent disease, coronary atherosclerosis, found in 85% of men and 55% of women in the general population when coronary calcification is regarded as a typical sign of the disease.

Ishikawa et al present verification of the supposed relevance of myocardial bridging for the development or enhancement of the atherosclerotic process in the proximal part of the left anterior descending coronary artery. They analyzed the extent and distribution of coronary atherosclerosis in 100 consecutive autopsy hearts from patients with myocardial infarction and found that nearly half had a myocardial bridge. In addition, they examined 200 normal hearts, 100 with bridging and 100 without bridging. They found that coronary atherosclerosis was more pronounced and extended upward toward the coronary ostium, augmenting the natural history of the disease predisposing to myocardial infarction. The authors thereby report for the first time pathological-anatomic evidence of the myocardial bridging phenomenon as a novel anatomic risk factor for coronary atherosclerosis and myocardial infarction. Future studies must demonstrate not only the presence of bridging but also the disease stage and the extent and distribution of coronary atherosclerosis in those with and without myocardial bridges after adrenergic stimulation and decreasing afterload.

A particular value of this work lies in the translation of these findings into clinical practice by the recently described ability to noninvasively detect myocardial bridging with intravascular ultrasound, a sensitivity of 93% and specificity of 82% for distinguishing myocardial bridging from coronary calcium by cardiac computed tomography. With cardiac computed tomography studies will need to evaluate the natural course of myocardial bridging and to demonstrate whether the congenital variant is clearly a congenital anomaly rather than a congenital anomaly. Semin Ultrasound CT MR. 2008;29:195–203.

References


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