Myocardial Bridge: Harmless or Harmful

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The coronary arteries and their major branches usually run on the surface of the heart in the subepicardial tissue. Sometimes, a portion of the artery runs under a “bridge” of superficial myocardial fibers for a short distance. This condition has been given various terms “myocardial bridge”, “intramural coronary artery”, “mural coronary artery”, “coronary artery overbridging” and “myocardial loop” (1). The incidence of myocardial bridge has been reported to vary between 15% and 85% in autopsy, and 0.5% to 2.5% in angiographic series (2). The length varies widely from 4 to 40 mm, the thickness also varies from 1 to 4 mm (2). This is found most often at the mid portion of the left anterior descending coronary artery, and sometimes at the right coronary artery and left circumflex artery. Myocardial bridges are recognized primarily because of the “systolic narrowing” or “milking effect” as seen on coronary angiography. Angelini et al (1) have concluded that at least 5% of the general population is estimated to have angiographic systolic narrowing, and myocardial bridges are not likely causes of myocardial ischemia.

Since the first angiographic description of myocardial bridge in vivo by Porstmann and Iwig (3) in 1960, many ischemic events possibly caused by myocardial bridge have been reported, including many reports from Japan (4–6). Accumulating evidence throughout the world has indicated that myocardial bridge may cause angina pectoris (7, 8), coronary spasms (9, 10), myocardial infarction (10–14), ventricular septal perforation (15), life threatening cardiac arrhythmias (6, 16) and sudden cardiac death (2). Many case reports from Japan contributed much to the understanding of the pathophysiological role of myocardial bridge (4–6, 9, 17) including a case report by Kurisu et al (18) in this issue of the Journal.

As coronary narrowing takes place during systole and because a major portion of the flow occurs during diastole, it is intriguing to consider how myocardial bridge causes myocardial ischemia. Bourassa et al (2) have clearly answered this question. In a frame-by-frame analysis of cineangiograms, they demonstrated that the milking effect of the coronary artery had an extension of the obstruction into diastole, which averaged 136 ms or 26% of diastole. Early diastolic diameter gain was delayed with a persistent mid-diastolic diameter reduction by the myocardial bridge. Quantitative analysis of the Doppler flow profile shows a highly characteristic pattern in approximately 90% of the patients with an abrupt early diastolic flow acceleration, which has been termed as the “finger-tip” phenomenon, a rapid mid-diastolic deceleration and mid-to-late diastolic plateau. The abrupt early diastolic flow acceleration is due to the persistent diastolic diameter reduction. This is followed by a rapid diastolic lumen gain leading to a plateau during late diastole. This characteristic flow pattern has been well described by Bourassa et al (2). It is not hard to believe that a myocardial bridge may cause flow limitation leading to myocardial ischemia in such patients with a rapid heart rate where the diastolic phase is relatively shortened or in cases with maximal coronary arterial dilation in which flow velocity is maximal and any minor resistance against flow could cause flow reduction. In fact, coronary flow reserve defined as the ratio of mean flow velocity achieved at peak hyperemia to mean resting flow velocity obtained after intracoronary injection of papaverine was reduced from 3.0 in normal subjects to 2.0 or 2.6 in those with a myocardial bridge (2). $^{201}$Tl single photon emission computed tomography (SPECT) immediately following treadmill in patients with myocardial bridge showed a reversible perfusion defect (7). Dobutamine stress echocardiography also showed wall motion abnormalities in these patients (8). On the other hand, intracoronary stent placement to a myocardial bridge was successful to normalize coronary fractional flow reserve (19) and to abolish clinical symptoms (20).

However, how can we explain many cases with coronary spasms (9, 10) or myocardial infarction (10–14) in patients with a myocardial bridge? In this issue of the Journal, Kurisu et al (18) report a 28-year-old Japanese male with myocardial infarction. This patient was admitted 2 hours after the onset of chest pain, and emergency coronary angiogram revealed a massive thrombus at the proximal portion of the left anterior descending coronary artery. Follow-up coronary angiogram obtained 8 days after onset demonstrated an obvious myocardial bridge with systolic compression. Intravascular ultrasound revealed an atherosclerotic plaque in the segment immediately proximal to the myocardial bridge. They concluded that coronary spasm was elicited at the myocardial bridge followed by thrombus formation and myocardial infarction (18).

See also p 1157.
There are numerous case reports of myocardial infarction related to myocardial bridge (10–14). Intravascular ultrasound study by Ge et al (21) revealed atherosclerotic plaques in the segments proximal to the bridge in 12 (86%) of 14 patients. Their follow-up study in 1999 revealed atherosclerotic involvement of the proximal segment in 61 (88%) of the 69 patients, and no plaques were found in the bridge or distal segments (22). The present report in this issue of the Journal (18) is consistent with these observations (21, 22).

Teragawa et al (9) have suggested a link between myocardial bridge and coronary spasm. They performed a spasm-provocation test in 114 Japanese patients with chest pain by infusing acetylcholine into the left coronary artery. Patients with myocardial bridge (MB) experienced coronary spasm more frequently than patients without MB (MB+: 73%; MB−: 40%, p=0.0006). They considered vascular dysfunction at level of the endothelium and/or smooth muscle as a possible cause of spasm at the myocardial bridge. Endothelial dysfunction (23) and/or dysfunction of smooth muscle cells (24) is thought to be involved in the genesis of coronary spasm. Ishii et al (25) have demonstrated that endothelial cells and smooth muscle cells are preserved beneath the myocardial bridge, but not in the segment proximal and distal to the myocardial bridge.

Summarizing these reports, it can be said that the myocardial bridge is no longer harmless, and it can be harmful. Mechanical compression which persists during early diastole can cause myocardial ischemia and chest pain. The proximal portion of the myocardial bridge can be a favorite site of atherosclerotic lesions. Furthermore, vascular dysfunction at the myocardial bridge may cause vasospasm which can lead to lethal ischemic events, arrhythmias, myocardial infarction and sudden cardiac death.

Fortunately, patients with myocardial bridge and systolic compression of the left anterior descending coronary artery have been described as having a good or an excellent long-term prognosis. In their state-of-the-art paper, Bourassa et al (2) summarized the demographic and clinical characteristics of patients with symptomatic myocardial bridges, based on several clinical observations. Among them, observation based on only 69 cases was the greatest number of patients (22). We need a greater number of patients followed up for a sufficient time in order to conclude that the myocardial bridge is harmless.

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References