Bridge that disconnects?

The intramural course of an epicardial coronary artery was first described anatomically (autopsy) by Reyman[2] in 1737 and angiographically in 1960,[3] while Polacek in 1961 was the first to use the term “myocardial bridge” for the entity. Autopsy prevalence for the bridge has always been higher (5.4% to 85.7%) compared to imaging (0.5% to 1.6%), possibly because the segment of a coronary artery tunneled intramuscularly may not always be compressed in systole to be visible angiographically.[4] Like the varied prevalence rates it has always been associated with varied manifestations ranging from angina, acute coronary syndrome, left ventricular dysfunction, arrhythmias, heart blocks and even sudden death. Atherosclerosis preferentially develops immediately proximal to the bridged segment, likely due to alterations in shear stress, while the compressed segment itself is often spared. Based on symptoms and the Schwartz classification, treatment ranges from only reassurance from the treating team (which helps avoid stress, anxiety and depression among the patient and his/her family) to beta-blockers/calciump channel blockers, surgical myotomy, intracoronary stenting or coronary artery bypass graft surgery.

Wellens et al.,[4] were the first to report atrioventricular (AV block) associated with myocardial bridge. They described a 35-year-old otherwise healthy young man with paroxysmal infra-Hisian AV block induced by exercise. This patient had a myocardial bridge affecting the proximal left anterior descending (LAD) and major septal arteries with a reversible radionuclide hypoperfusion of the septum during exercise. It was postulated that ischemia of the conduction system due to systolic milking of LAD artery was responsible for the paroxysmal AV block in this patient.

The blood supply to the infranodal conduction system is primarily through the penetrating branches of the LAD artery. This artery usually perfuses the left anterior fascicle and distal right bundle. The left posterior fascicle has a dual supply from the right coronary and LAD arteries. Occlusion of the LAD artery proximally therefore may be associated with right bundle branch block and/or left anterior fascicular block commonly. The finding at angiography of a myocardial muscle bridge causing systolic milking of the coronary artery and an exercise-induced septal defect on thallium stress testing led to the speculation that the muscle bridge produces septal ischemia, possibly resulting in the patient’s intermittent heart block. However, the role of myocardial muscle bridges as a cause of ischemia remains controversial and the stress induced perfusion defects could sometimes just be related to baseline left bundle branch block (LBBB).

Lin et al.,[5] in the current issue of this journal report a 51-year-old man presenting with a Mobitz type I (Wenckebach type) AV block coexistent with a myocardial bridge in the mid-portion of the LAD artery. Although the causal association of bridge with presence of Mobitz type I block is supported by its disappearance after bypass surgery, some questions remain in a curious reader’s mind. The AV node is the site of Wenckebach type of block and this is supplied by the right coronary artery, hence the ischemia caused by transient LAD artery occlusion, that too in the mid portion, cannot affect AV nodal conduction. However, rarely intra-His Wenckebach has been reported,[6] and this could be the mechanism in the present case. The possibility of age-related intermittent degenerative co-existing conduction disease also needs to be considered. Additionally, the precipitation of symptoms with alcohol intake makes one wonder if the AV block is related to this, as has been documented previously.[7] Also unexplained is the elevated serum troponin I (TnI) at presentation without any obvious infarction. Finally, grafting the distal LAD artery only cannot be expected to reperfuse the septal artery which itself is affected by the bridge.

Myocardial bridges continue to leave us speculate and wonder about their cause, course, manifestations and treatment. In line with Leonardo da Vinci’s words, “the greatest deception men suffer is from their own opinions”, we continue to form and reform our opinions about the myocardial bridge.

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