Bridge over troubled coronary artery/ Muscular bridge causing non-ST-segment elevation myocardial infarction

Tehlikedeki koroner arterin köprüsü/ ST-elevasyonsuz miyokard infarktüsüne neden olan miyokardiyal köprüleme

Dear Editor,

Myocardial bridging is a congenital condition, defined as the intramyocardial course of a portion of the coronary artery. It was first described by Reyman in 1737 (1) and the artery coursing within the myocardium is called the "tunneled artery" (2). The left anterior descending is the most common artery affected. The coronary arteries are not necessarily epicardial in all the mammals. For example the rodents do have intramyocardial vessels and the chimpanzees have intramural coronary arteries (2).

The significance of the presence of the bridging is also controversial. There is a significant discrepancy between the autopsy series and angiographic series in terms of the incidence of myocardial bridging. Some autopsy series have reported the incidence to be as high as 85%, whereas the angiographic incidence remains only in the 1.5% to 16% range (3, 4). Some have advocated that, by careful review of the coronary angiograms, for the specific purpose of identifying the muscle bridge, there would be a higher incidence of myocardial bridging (5). Nevertheless, the true significance of the myocardial bridging is debatable since only 15% of the coronary blood flow occurs during systole. There are numerous reports of patients with myocardial bridges with unstable angina (6), myocardial infarction (7-9), dysrhythmias (10, 11) and sudden death (12, 13) but in general this abnormality is considered to be clinically harmless.

Intravascular ultrasound (IVUS) and Doppler evaluation of the myocardial bridges have provided valuable insight to the problem. Ge et al. described the highly specific echolucent half moon phenomenon over the bridge segment that existed throughout the cardiac cycle (3). Ge et al. also showed a high incidence of atherosclerotic plaque proximal to the area of the muscle bridge (3). Thus, the hemodynamic impairment of the coronary blood flow by the bridging can be accentuated by the formation of atherosclerosis at the proximal segment of the artery and may explain some of the presentations of angina, dysrhythmias and myocardial infarction. When intracoronary Doppler was used, the characteristic early diastolic fingertip phenomenon was observed (3). This fingertip phenomenon represents a sudden increase in the blood flow velocity due to an increase in the blood flow with early diastole due to the relaxation of myocardial tension and microcirculation but persistent decrease in the vessel diameter. Both the Doppler findings and the systolic compression of the coronary artery can be accentuated by the use of nitroglycerine (14) and rapid pacing (15). This has important implications for the treatment of myocardial bridging. Beta-blockers are the mainstay of treatment and avoidance of nitrates might be necessary.

In the December issue of your Journal Aytan et al. (16) describe a patient with a myocardial bridging of the left anterior descending artery presenting with a non ST segment elevation myocardial infarction. Since this patient had already had a coronary angiogram 4 years before the presentation and no report of a bridge was given, one possibility is that the use of antianginal agents presumably including nitroglycerine might have contributed to the augmentation of the angiographic appearance of the bridging. The other mechanism would be the formation of thrombus proximal to the area of bridging. Whatever the mechanism was in this patient it would have been interesting to have a follow-up SPECT imaging to see if the anterior ischemia reported in the previous study had resolved.

Myocardial bridging of the coronary arteries, although mostly asymptomatic, might not be so innocent after all. Coronary angiograms should be carefully scrutinized to identify this abnormality.

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Author`s reply

Dear Editor,

We appreciate the comments of our colleague regarding our report, as well as the additional data that had been provided. The possible mechanisms for the detection failure of the myocardial bridging by angiography that had been done 4 years ago were very well outlined in the letter; use of antianginal agents presumably including nitroglycerine might contribute in augmenting the angiographic appearance of the bridging and secondly the formation of thrombus may be proximal to the area of bridging. The first angiography was done in another center and the patient had not been followed up. As a result we could not have any idea of why in the first angiography no report of a bridge was given. With his admission to the emergency department of our center antianginal therapy was started. At the 3rd day of the onset of angina cardiac catheterization was performed. Coronary angiography revealed suspicion of thrombosis and 95 % luminal narrowing by systolic compression in the mid segment of the left anterior descending coronary artery at left anterior oblique cranial position. The gold standard diagnostic tool for diagnosing myocardial bridge is selective coronary artery angiography. But the new imaging techniques like intravascular ultrasound, intracoronary Doppler ultrasound, and intracoronary pressure devices as invasive techniques (1, 2) and electron beam tomography, multislice computed tomography, magnetic resonance tomography, or transthoracic Doppler echocardiography as noninvasive imaging techniques (3) can be used for diagnosis of functional and morphological status of bridges.

Single photon emission computed tomography (SPECT) has been proposed for further investigation in such cases (4) and at the 7th day of the onset of symptoms a SPECT was performed. Moderately extended, mildly severe (+1) reversible ischemic defect was reported.

It is prudent to follow these patients with a SPECT imaging as it has been suggested by our colleague. It was planned to follow-up the patient with another SPECT imaging during his follow-ups to see if the anterior ischemia had resolved; however, the patient did not come to his follow-up examination.

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