Myocardial bridge and atherosclerosis

Miyokardiyal köprüleme ve ateroskleroz

There is an evidence from pathophysiologic and intravascular ultrasound studies that the arterial segment proximal to the myocardial bridge (MB) has a higher frequency of atherosclerosis, whereas the tunneled segment is relatively spared. The reason for atherosclerosis being confined mainly to the part of the vessel proximal to the bridge is unclear, but may be related to local wall stress, flow and shear stress conditions, and subsequent injury to the vessel wall (1, 2). It has been shown that areas of low mean shear stress and areas where blood flow departs from a laminar unidirectional pattern, including areas of oscillatory flow and flow reversal, seem to be prone to the development of atherosclerotic plaques, preceded by the development of endothelial dysfunction (3-5). Myocardial bridge may initiate similar dynamic and endothelial alterations in the proximal segment of the artery. Thus, low shear stress may contribute to atherosclerotic plaque formation proximal to the bridge, whereas high shear stress may have a protective role within the bridged segment (6, 7).

In the study by Duygu et al (8), published in the current issue of the Anadolu Kardiyoloji Dergisi, 6272 coronary angiography recordings had been retrospectively reviewed for the presence of MB, and then classified according to the presence or absence of concomitant angiographically evident atherosclerosis. The study provides important data by means of clinical, demographic and anatomic predictors of atherosclerosis development due to MB. Although the statistical methods inadequate to derive a precise decision about the predictors of development of atherosclerosis secondary to myocardial bridging, the results suggest that older age, multiple risk factors and more importantly the ratio of systolic compression of the bridging segment may be related to atherosclerosis. The degree of systolic compression was higher in patients with atherosclerosis, and accordingly a significant correlation between the degree of systolic narrowing and clinical presentation probably might be detected. Interestingly a recent study showed positive correlation between the degree of systolic narrowing and the ratio of atherosclerotic stenosis consistent with the findings of the current study (9). It would be very interesting if the authors have made a detailed statistical analysis about the relationship between the "magnitude of systolic compression" and atherosclerosis, angiographic characteristics, clinical presentation (unstable-stable angina).

The MB + atherosclerosis group should have been constructed with only the patients who have atherosclerosis on bridging artery, and patients with single vessel disease on the artery other than bridging coronary should have been excluded. However, the authors have included nine patients who have atherosclerotic lesions on the artery unrelated to the MB. This approach affects the anatomic cause-result relationship between MB and atherosclerosis. Similarly, single-vessel disease has been detected in 45% of the patients and multi-vessel disease in the remaining 55%. Consequently, 55% of the patients might have atherosclerotic disease possibly independent to MB. This condition again prevents to derive a precise cause-result relationship. According to the aim of the study, MB + atherosclerosis group should be constituted with patients who have atherosclerotic lesions exclusively in the bridging artery. Furthermore, angiographic imaging may underestimate the presence of atherosclerosis in patients who have MB but have not "angiographically visible" coronary artery disease. Certainly, a significant part of the patients without angiographic evidence of atherosclerosis in the proximal segment may have early stages of coronary atherosclerosis or positive remodeling revealed by intravascular ultrasound. It has been shown that approximately 90% of patients with MB have atherosclerosis proximal to the bridge demonstrated by intravascular ultrasound (10). However, the angiographic evaluation, which was done by the authors of the current study (8), certainly more important than intravascular study to seek out the "clinical significance" of angiographically evident atherosclerotic disease associated with MB.

As a conclusion, this study is providing noteworthy clinical and demographic data different to previous studies those focused on the hemodynamic features of MB and hemodynamic causes of atherosclerosis development secondary to MB. The readers actually are eager to have some definite results about the clinical, demographic, and angiographic predictors of the development of atherosclerosis secondary to MB. Unfortunately, the statistical method, partly due to limited number of patients, was not strong enough for defining the "predictors of atherosclerosis development secondary to MB" which the readers would be more curious about. On the other hand, several important conclusions can be derived from this study: 1. The probability of isolated MB is more common in young patients having lower number of risk factors, 2. Older age and multiple risk factors may initiate or accelerate atherosclerosis in proximal segment of the vessel under hemodynamic stress, 3. Higher degree of systolic compression may be related to pathogenesis of atherosclerosis. 4. Combination of atherosclerotic obstructions and MB may also cause more frequently unstable angina pectoris, and require more interventional therapy as expected.

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Regarding the findings of ultrasound studies and the results of the current study it is possible to speculate that almost all MBs are associated with some extent of atherosclerosis proximal to the bridging segment where the hemodynamic stress most prominent. Traditional concomitant risk factors, age, and high degree of systolic compression may accelerate atherogenesis and cause ischemic syndromes as reported in this study.

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