Myocardial bridging/Angiographic prevalence of myocardial bridging

Miyokardiyal köprüleşme/Miyokardiyal bandın anjiyografik prevalansı

Muscle overlying an epicardial segment of a coronary artery, first mentioned by Reyman in 1737 (1), is termed a myocardial bridge, and the artery coursing within the myocardium is called a tunneled artery. It is characterized by systolic compression of the tunneled segment, which remains clinically silent in the vast majority of cases. An in-depth analysis of autopsy samples was first presented by Geiringer et al. in 1951 (2), but clinical interest and systematic research was triggered by an observed association of myocardial bridging with myocardial ischemia (3-5).

In the March 2006 issue of the Journal Çay et al. (6) reported the prevalence of myocardial bridging among almost 26.000 Turkish patients aged 56±11 years including 18% women undergoing coronary angiography (6). Among the 1.2% of subjects with myocardial bridging, 46% of subjects had coexisting coronary artery disease and 42% had systolic compression >50%. As expected in this age group undergoing coronary angiography, risk factors were frequently present ranging from 12.7% with diabetes mellitus to 42.7% of subjects with hyperlipidemia. Interestingly, in patients with systolic compression <50%, diabetes was more frequent than in subjects with >50% compression. It can only be speculated that this finding is related to a higher rate of associated coronary artery disease in diabetics, where myocardial bridging may not have been the main cause of chest discomfort, angina, ischemia or any other reason that has led to angiographic workup. This information is, however, not provided, which is unfortunate as one would have expected a higher rate of relevant systolic compression in diabetic patients compared to non-diabetic subjects. Impairment in endothelium-dependent vasorelaxation, as it may occur in diabetic patients, has recently been identified as a potential mechanism contributing to increased, but not decreased, vasoconstriction beneath the myocardial bridge (7,8).

Most bridges (96.5%) were located in the left anterior descending artery (LAD) (6). Involvement of two vessels was an exception (n=2) (6). This is consistent with previous angiographic and pathologic studies (9). While myocardial bridging is seen at careful autopsy in about 1/3 of cases, angiographic prevalence is much lower, ranging from 0.5% to 40% in selected subgroups (9). In their work, Çay et al. (6) for the first time present such prevalence data for a Turkish population. Interestingly, the proximal and distal segments were equally affected, which may have been due to definition of segments. Whether ethnicity can contribute to such an observation is thus far not clear.

The characteristic angiographic feature of myocardial bridging is systolic compression of the epicardial vessel, usually the left anterior descending artery (LAD), with the angiographic "milking effect". As Çay et al. (6) have pointed out, modern imaging techniques, such as intravascular ultrasound (IVUS), intracoronary Doppler-ultrasound (ICD) and intracoronary pressure-wires, have contributed significantly to our understanding of the pathophysiologic consequence of this entity (9-12). While previously considered a clinically insignificant variant, ICD recordings demonstrated an increased flow velocity in the tunneled segment. Frame-by-frame analysis on IVUS revealed a delayed relaxation after systolic compression, which may extent significantly into diastole (13). This contributes to the impaired coronary flow reserve and also to ischemia. On IVUS images, a circular or eccentric rhythmic compression of the vessel is visible, which may be partial or complete, and is called "half-moon-phenomenon" (11). Recently, contrast-enhanced computed tomography technology (13-15) and also magnetic resonance imaging (16) has been used to visualize systolic compression non-invasively. Transthoracic intravascular Doppler-echocardiography may be successful in depicting the characteristic typical finger-tip phenomenon (13).

As acknowledged by Çay et al. (6), the rate of myocardial bridging in the Turkish population undergoing coronary angiography would have likely been higher, if the investigators had routinely injected nitroglycerine into the coronary artery. Administration of nitroglycerin not only dilates segments adjacent to the tunneled segment but also reduces pressure in the tunneled segment - mechanisms that both contribute to increased compression and an improved angiographic detection rate. Especially in symptomatic subjects with otherwise normal angiograms but myocardial bridges, such provocation tests should be used to assess the functional significance of the bridge (13,17). In the past, nitroglycerine, orciprenaline, dobutamine or atrial stimulation has been used for this purpose.

Beta-blockers are first choice therapy. Intracoronary administration of a short-acting beta-blocker attenuated systolic compression and early-diastolic flow could be reduced. The diastolic/systolic flow ratio was normalized and symptoms induced by atrial stimulation could be improved (18). In cases of contraindication or additional vasospasm, calcium antagonists have been helpful. In severe cases, strenuous exercise should be limited, to avoid the negative consequences of tachycardia.

Coronary stenting was first described in 1995 by Stables et al. (19). However, the rate of restenosis and/or complications has been too high to generally recommend this strategy even in severe myocardial bridging (9). Whether drug-eluting stents yield a better outcome remains to be shown. In severe cases with symptoms refractory to medication and with signs of ischemia in non-invasive tests, surgery should be considered after careful appraisal of the benefit-risk-ratio.

Long-term prognosis is usually good, i.e. event-free 5-year survival >95% (9). In children and in subjects with hypertrophic

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cardiomyopathy event-free prognosis may be reduced. Recently, Vaz et al. reported a higher rate of events among 174 subjects (0.7%) aged 49 years (30% women) with an isolated myocardial bridge and no other cardiac disease (20). During a follow-up period of 4.2 years, 3 deaths and 7 anterior myocardial infractions occurred in 9 subjects (5.2%). Aspirin- (hazard ratio (HR): 0.16, 95% CI: 0.03-0.86, p=0.03) and nitrate usage (HR: 8.59, 95% CI: 1.5-49.1, p=0.01) were independently associated with outcome. However, it is not clear why so many subjects with an isolated myocardial bridge have received nitroglycerine, which is known to augment systolic compression, and which may therefore account for the unexpected high event rate.

In summary, myocardial bridging is a frequent entity, which can be observed in up to 40% of patients with normal coronary angiograms using provocation tests and sophisticated diagnostic testing. Çay et al. (6) for the first time reported prevalence data in a Turkish population. Their findings are in line with previous reports from other countries. Such data are important as they contribute to an increased awareness of a frequent entity and provide the scientific ground for ethnic similarities and differences in disease, which is of increasing importance in a globalizing world.

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Author`s reply

Dear Sir,

We would like to thank to the author for his/her comments about our manuscript entitled 'Angiographic prevalence of myocardial bridging'.

In our study, there was a statistically significant difference between Group A (<50% of systolic compression) and Group B (≥50% of systolic compression) according to the presence of diabetes as mentioned by the author and it can only be speculated that this finding is related to a higher rate of associated coronary artery disease in diabetics, where myocardial bridging may not have been the main cause of chest discomfort, angina, ischemia or any other reason that has led to angiographic work-up. However, we found that there was also a significant difference between Group 1 (<50% of systolic compression) and Group 2 (≥50% of systolic compression) according to the presence of diabetes. These groups include patients having myocardial bridging without coronary heart disease angiographically.

For left anterior descending artery (LAD) myocardial bridges the pathology is generally found in the middle portion of the LAD coronary artery. However we found that, bridges of LAD coronary artery were distributed almost equally between middle and distal segments. Interestingly, no myocardial bridging was present in the proximal segment. This equality was probably due to segment definition: the segment between first diagonal and second diagonal coronary arteries was termed middle segment and the segment after second diagonal branch was termed distal one in our study. As mentioned by the author whether ethnicity can contribute to such an observation is thus far not clear.

As the author pointed out, bridging is more likely to be noted after intra-coronary nitroglycerin. However, the prevalence is likely underestimated because nitroglycerine was routinely not given in our study.

We would like to thank the author of the letter to the editor for his/her important comments again.

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