Myocardial bridging and prognosis: more evidence but jury still out

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Myocardial bridging (MB) is a congenital variant of coronary anatomy in which a segment of a major epicardial coronary artery that normally has an epicardial course runs intramurally through the myocardium beneath the muscle bridge. It occurs most frequently in the mid-portion of the left anterior descending (Figure 1). It is also called ‘tunneled artery’. Geiringer1 first presented an in-depth analysis of autopsy samples in 1951, but clinical interest and systematic research were triggered by observed association of MB with myocardial ischaemia. In this regard, although traditionally considered a benign condition, MB may cause clinical symptoms including angina, myocardial infarction, life-threatening arrhythmias, and sudden death.2–4 It is widely accepted that MB might cause these ischaemic complications5,6 either from direct compression of the coronary artery during systolic contraction or by enhancement of the natural progression of atherosclerosis in the coronary segment. Both mechanisms are closely associated with changes in haemodynamic stress driven by the force of the MB compression through a combination of anatomical properties, such as the location, length, and thickness of the MB.7

The true prevalence of MB is not fully known because it is largely underdiagnosed by conventional angiography, cardiac computed tomography (CCT) should be the preferred non-invasive imaging modality because of its multiplanar and three-dimensional capabilities; it can visualize not only the coronary lumen, but also the vessel wall and the surrounding tissues, including the myocardium. MB can also be identified by CCT even when no significant changes in haemodynamic stress driven by the force of the MB compression through a combination of anatomical properties, such as the location, length, and thickness of the MB.7

The frequency of MB detection at autopsy than at CCT may be due to differences between these methods, because an MB thinner than 200 μm can only be detected by autopsy.

The study of Rubinshtein et al.9 in this issue of the journal reports on prognosis among patients clinically referred for CCT who were found to have MB but were without prior coronary heart disease or obstructive disease on CCT. Patients were followed for a mean of ~6 years for the outcome of cardiovascular death or non-fatal myocardial infarction ascertained using chart review and a national death registry. Out of 648 chest pain patients referred for 64-slice CCT in a 1-year screening period, 334 patients met the criteria for primary analyses. No angiographic control is present. One hundred and seventeen patients (35%) had MB on CCT, that was most frequently found in male, in case of left coronary dominance and at the level of the mid-left descending artery (71% of the patients). The event rate over the follow-up period was low (3.8%, 0.6%/year) in total population even though 52% of the patients had MDCT evidence of subclinical atherosclerosis, without statistically significant difference between patients with or without MB (5.1 vs. 3.2%, respectively).

Several important points warrant comment. First, the prevalence of MB reported in the current study, as acknowledged by the authors, may be higher than in general population, as patients with more symptoms (or inconclusive stress test results) are more likely to be referred for CCT. However, the contrary cannot be excluded. For instance, the reported rate of MB may be lower than that of a population with typical chest pain without obstructive coronary artery disease, which is under-represented in the current study. Secondly, it is a retrospective analysis of a small, low-risk population with MB that comprised the cohort (117 patients), which limits broad generalizations and conclusions. Results of the current study are underpowered to assess the
impact of MB on hard clinical endpoint, with a power of only 16%,
although the outcome data confirm the generally good long-term
prognosis in patients with isolated MB reported by others. To
this regards, the MB group shows an at least numerically disturbing
37% increase in term of relative risk for cardiovascular death or
myocardial infarction when compared with no MB patients; that
trend would likely become significant with a larger sample size
(1729 patients per group). Another caveat that limits broad gener-
alization of the study conclusions is the impossibility to assess the
impact of unmeasured confounders unbalance on the final study
outcome results. For instance, considering that half of the patients
have subclinical atherosclerosis that mandates aggressive medical
therapy, an imbalance in the prevalence of aspirin, statins, beta-
blockers may bias the results. Thirdly, out of 117 patients with
MB, 27% had what is described by the authors as ‘superficial
variant of MB (partially covered with myocardium),’ an entity
that is not and cannot be well depicted in the current study.
Our knowledge on this point is very limited. This subset may
not be described as having a bridge (and may not have potential
for systolic compression), although it has been shown that
dynamic compression of the vessel may also occur in segments
without fully overlying muscle by the entrapment of MB within
the interventricular gorge with possible transient occlusion of
prominent septal branches arising from or near the involved
segment may occur.10,11

Another aspect that might have been treated more in depth by
the Authors, concern the selected endpoints. Mortality and acute
myocardial infarction are hard events that show major prognostic
outcome. However, it should be considered the option to include
softer events as endpoints. MB is under investigation and we know
it may cause ischaemia; therefore, softer endpoints such as recur-
rent angina/chest pain, re-hospitalization for chest pain, repeated
stress tests, and so forth. This information might not improve prog-
osis but it may improve treatment strategies and quality of life of
some of these patients.

From the technical standpoint, one could also argue that the
definition and classification of MB on CCT is still not widely
accepted and recognized. In fact, in the study from Rubinshtein
more attention has been paid to the so-called complete/deep
intra-myocardial course of the coronary artery. As stated above,
the prevalence of superficial intra-myocardial course is expected
to be much higher. Some questions have to be answered, yet,
concerning the reporting methodology. Systolic imaging can be
performed on CCT; it could be asked whether measurements
(i.e. depth, length) of the MB should be obtained also in this
phase of the cardiac cycle.

Even with all the reported limitations that call for caution in
interpreting the results of the current study, the authors need to
be congratulated to have conducted an important analysis on the
prognostic role of isolated MB as assessed by CCT disease that
confirms a general good prognosis of these patients. The study
highlights the need new imaging technology to match the unmet
clinical need of improving our knowledge of the anatomical
and functional characteristics of MB whether or not isolated.

Figure 1 Example of myocardial bridging (MB) of the middle tract of left anterior descending (LAD) coronary artery as displayed on Cardiac CT (A—3D volume rendering—arrowhead). The longitudinal curved multiplanar views in orthogonal planes (B and C) show the location and length of the MB. Axial cross-section of the LAD performed proximal (D), distal (F) and within the MB (E) show the typical pattern of deep intra-myocardial course. The deep or complete intra-myocardial course consists of a 360° myocardium surrounded vessel (E—arrowhead) as also displayed in pathology sample (E’—arrowhead).
The perspective is avoid to carry on as we do considering this entity simplistically as black or white picture—culprit or innocent bystander’. Since then, large multicentre clinical databases, based on standardized definition, are required to identify criteria that justify the link between clinical sign and/or symptoms and a given pattern of MB as the primary culprit and which move beyond the current empirical approach to the clinical management of this frequent coronary anomaly.9,10

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References

Transcatheter closure of paravalvular leak secondary to left ventricular peri-annular pseudoaneurysm

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Left ventricular (LV) pseudoaneurysm is a clinically rare condition, often difficult to diagnose; one-third of the occurrences result from a surgical procedure, mostly mitral valve replacement (MVR). The detailed anatomy of sac and its communication can be delineated with a transoesophageal echocardiogram (TOE), cardiac MRI, or contrast CT scan.

We report a case of a 77-year-old woman, who underwent an MVR with a mechanical prosthesis. After 3 years, she developed infective endocarditis. A TOE showed a wide posteromedial paravalvular leak, determining a severe regurgitant jet into the left atrium (LA) (Panel A); furthermore an LV recess was detected just below the origin of the jet confirmed by a contrast CT-scan (Panel C, arrow). The rupture was located in the posterior atrio-ventricular groove, creating a LV pseudoaneurysm communicating with the LA (Panel D, arrow). Because of a high surgical risk, a transcatheter closure of the defect was planned. The leakage was crossed with a retrograde approach. The Mullins catheter was advanced in the LV and an extra-stiff Amplatz wire was placed in the apex for positioning the 9F guiding catheter (Panel D), used for the implantation of an AMPLATZER™ Duct Occluder 10/12 mm (S Judie, Inc., USA) (Panel E). Finally, successful leak closure of the defect was achieved (Panel F, asterisk). The patient was discharged after 5 days.

A catheter-based closure approach for a paravalvular leak and LV pseudoaneurysm has been described before. We report an uncommon case of transcatheter treatment of an LV peri-annular pseudoaneurysm ruptured in the LA, in a patient with a previous MVR and endocarditis.