Editorial

Myocardial Bridge and the Progression of Atherosclerotic Plaque in the Proximal Segment

Sho Torii, Renu Virmani, Aloke Finn

Myocardial bridge (MB) is occasionally recognized, especially of the left anterior descending artery (LAD) during cardiac catheterization when a physician notes systolic compression of a coronary artery. Pathologically MB occurs when a segment of the artery takes a short intramyocardial path rather than an epicardial course.[1] Careful studies of MB document that both length and depth may be important, and these vary depending on the modalities used to assess, that is, angiography, computed tomography, or autopsy. We think the most accurate method of assessment of MB is computed tomography and the incidence of MB is 43% as reported by Liu et al,[2] with highest presence involving the LAD (80%: proximal, 3%; mid, 58%; distal, 19%), followed by diagonal (12%), obtuse marginal (7%), and right coronary artery (2%). They also showed a good correlation with systolic compression and MB depth but not with length.

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There is a higher incidence of cardiovascular events in patients with MB.[1] The reasons have remained uncertain, but recent data suggest an important role for the bridge in the development of atherosclerosis. Segment proximal to the MB, blood flow reversal at the entrance of MB because of systolic compression may lead to low wall shear stress which is proatherogenic affecting endothelial cell activation and transport of lipids into the arterial wall.[1] Although the intramural segments of the MB are protected from atherosclerotic disease, the segment proximal to the MB seems to have a predilection for the development of atherosclerosis (Figure). In this issue of *ATVB*, a study by Akishima-Fukasawa et al,[3] makes an important insight which helps to substantiate this theory.

Akishima-Fukasawa et al[3] evaluated 150 autopsied hearts, free from any cardiovascular disease and investigated the influence of MB on atherosclerosis development in the LAD. MB was identified in 93 hearts. Luminal stenosis ratio (LSR; similar to percent area stenosis) was significantly higher proximal to MB than in the segments under the MB and distal to the MB, suggesting important anatomic relationships between MB and atherosclerosis by location with respect to the bridge. The importance of traditional risk factors (RF) in 67 cases with LSR ≥50% was further analyzed. The authors defined the RF group as the patients with at least 1 RF among 3 factors (hypertension, hypercholesterolemia, and diabetes mellitus). Multiple comparison test showed that the LSR in the MB(+)RF(+), MB(+)RF(−), MB(−)RF(+), and MB(−)RF(−) group were significantly different. Furthermore, the site of a greatest stenosis in the MB(+)RF(+) group was 2.5 cm proximal to the MB. The results are similar to a previous article evaluated in autopsied MB(+) hearts with myocardial infarction that reports the greatest stenosis was located 2.0 cm proximal to the MB.[4]

Multivariate analysis was attempted to explore the role of an MB, RFs, gender, and age for cases with an LSR ≥50%, 60%, and 70%, respectively. The results indicated that age was an independent determinant of LSR ≥50% and 60%, but when LSR was ≥70%, MB(+)RF(+) was the only independent determinant. There were no differences between the RF(+) and RF(−) groups when an MB was absent, which is contrary to the well-known Framingham heart studies performed over many decades for the assessment of the influence of RF on coronary atherosclerosis.[5]

Another limitation of the study is that pathology cannot measure the hemodynamic significance of a bridge. The definition at MB was the presence of myocardial tissue covering LAD. As noted in Table 1, there was variability not only in the thickness of the bridge (1217±1058 μm) but also in the length of the bridge (1.99±1.08 cm). The hypothesis of an increase in atherosclerosis is based on systolic compression of flow reversal as the underlying mechanism, because the authors could not determine its existence at autopsy, therefore, the results will remain observational in nature.

Moreover, other unaccounted confounders also exist. The most glaring of these is the presence of bifurcations, which have been shown to have a dramatic effect on the location and progression of atherosclerosis. Bifurcations are known to play an important role in the development of atherosclerosis because of flow disturbances. In their analysis, Akishima-Fukasawa provide no information pertaining to the branching of the LAD, and thus the confounding effects of bifurcations on the relationship between MB and atherosclerosis remain unknown. Despite these limitations, this study suggests that there may be a potential role of MB as important contributors to the development of severe atherosclerosis (>70%). These data suggest that more nuanced understanding is required and that physicians should consider MB as having a possible role in the acceleration of atherosclerosis.

Disclosures

None.

From the CVPath Institute, Gaithersburg, MD.

Correspondence to Renu Virmani, MD, CVPath Institute, 19 Firstfield Rd, Gaithersburg, MD 20878. E-mail rvirmani@cvpath.org

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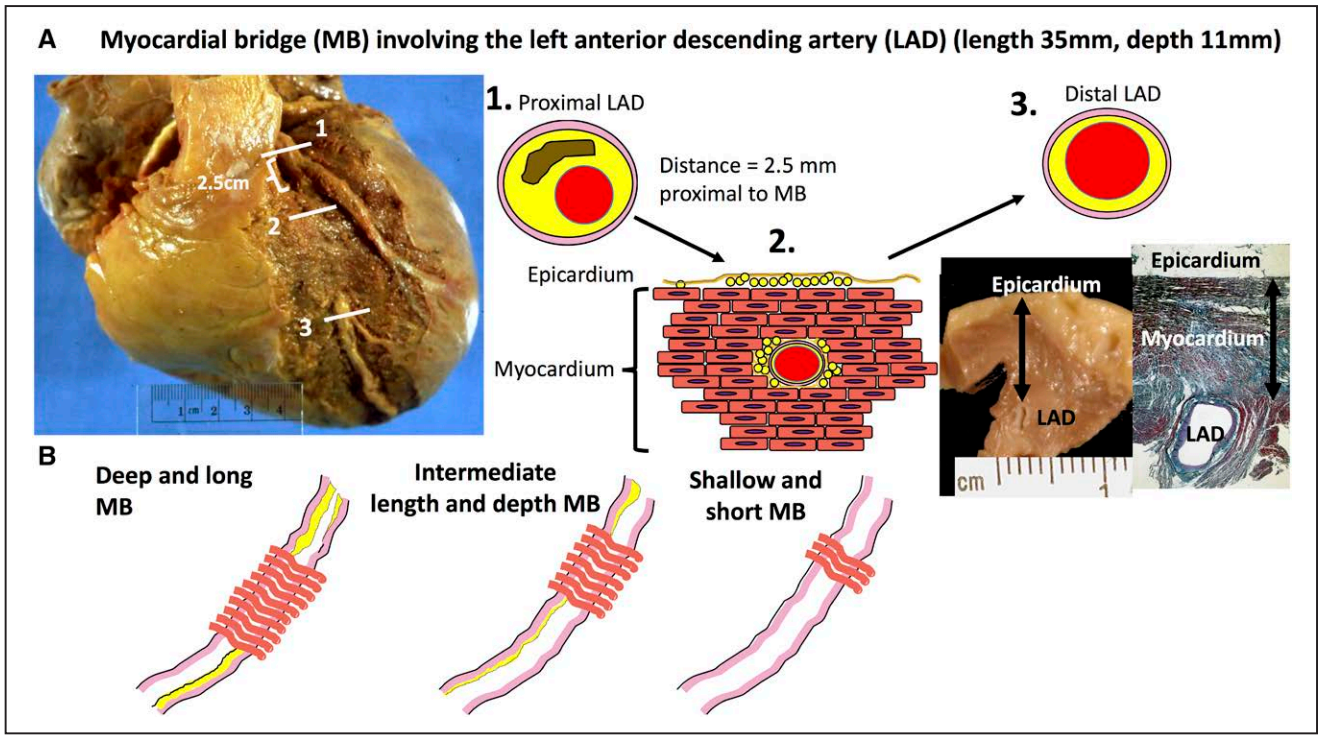

Figure. A, Gross and schematic images of the myocardial bridge (MB) involving the left anterior descending artery (LAD). Note the LAD proximal to the MB has a large first diagonal branch that would also influence the location and severity of atherosclerosis. Note gross and histological section showing the deep location of the LAD in a case of MB. **B**, Morphological variance in MB: length and depth of bridged segment should influence the extent of flow disturbance and atherosclerosis.

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