



Myocardial Bridge and Proximal Atherosclerosis

A 45-year-old male was incidentally found to have a significant mid-LAD lesion on CT angiography. There was no history of diabetes, hypertension, smoking, or family history of coronary artery disease. He exercised regularly and ran marathons. His lipid profile consistently showed untreated LDL levels over 160 mg/dL since last 6 years.

Subsequent catheter angiography demonstrated a 90% stenosis in the mid-LAD, followed distally by a prominent myocardial bridge (MB) producing severe systolic compression of a short LAD segment (Video 1, Fig 1 & 2). Between the distal end of the stenotic lesion and the proximal end of the MB, there was a 9.6 mm long segment of normal LAD (Fig 3).

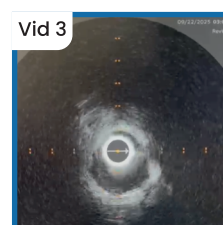
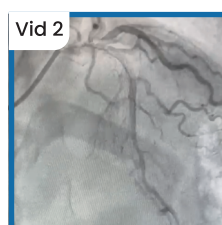
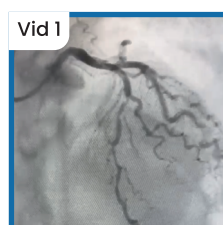
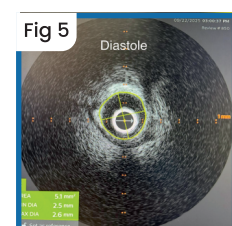
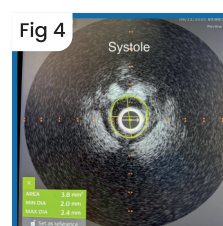
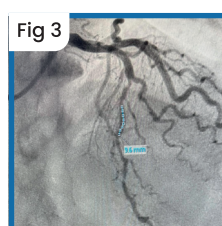
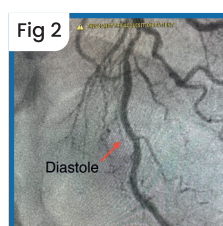
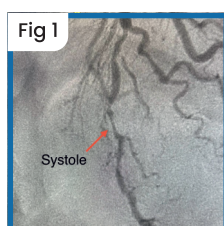
A nuclear perfusion scan revealed reversible ischemia involving the anterior wall and apical septum. IVUS-guided PCI of the LAD lesion was performed using a single stent, while the myocardial bridge was left untreated (Video 2). Pre-PCI IVUS showed a fibro-fatty obstructive plaque causing severe stenosis, followed by a bridged segment that was free of disease (Fig 4 & 5). Post-PCI IVUS confirmed optimal stent deployment, with a 9 mm gap between the distal stent edge and the proximal end of the MB (Video 3).

A myocardial bridge predisposes to development of atherosclerotic lesions, particularly proximal to the bridged segment (1,4).

Mechanism

The tunneled (bridged) segment itself is usually protected from atherosclerosis (5) (possibly due to higher shear stress during systole and different endothelial phenotype). However, the segment just proximal to the bridge is exposed to:

- Increased wall stress
- Disturbed shear stress and flow turbulence (oscillatory shear index higher proximal to the bridge)
- Endothelial injury → These changes promote lipid accumulation, inflammation, and plaque formation.



Clinical Evidence

- Angiographic and IVUS/OCT studies show higher prevalence of atherosclerotic plaque formation proximal to MBs, while the intramyocardial segment itself is usually free of plaque (1).
- Histopathologic studies confirm this distribution of atherosclerosis (2).
- Proximal lesions may become hemodynamically significant and can contribute to ischemia, in addition to the dynamic compression of the bridged segment (1).

Implications

MBs are not just a “benign” finding — the combination of fixed atherosclerotic stenosis proximal to the bridge + dynamic systolic compression within the bridge can markedly worsen ischemia (3). PCI can be performed to the proximal LAD stenosis, taking care to avoid extending the stent into the myocardial bridge. Stenting into the MB can lead to stent fracture and eventual restenosis. Intravascular imaging (IVUS/OCT) is highly recommended to define the exact landing zones. If the bridge is long or extends very close or into the stenosis, CABG (LIMA–LAD) is often the safer option.

References

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