Silent Culprit: NSTEMI-Associated Myocardial Bridging in a Healthy Adult

Sean Young, M.D.*, Srishti Taneja, M.D.*, Alyssa Kang, D.O.*

*Internal Medicine, Jefferson Einstein Montgomery Hospital, East Norriton, PA

Introduction

Myocardial bridging (MB), a commonly-occurring anatomic abnormality, is defined as a segment of a major epicardial coronary artery that passes intramurally, or 'tunnels', through the myocardium beneath a muscle bridge. [1-2] During left ventricular systole, this segment is compressed, a condition known as "milking".[3]

While this is typically a benign process that is found incidentally on imaging or post-mortem analysis, in rare cases it has been linked to myocardial ischemia, coronary thrombosis, cardiomyopathy, and even acute myocardial infarction and sudden cardiac death. [1-3]

Case Presentation

A 57-year-old Vietnamese male with no significant past medical history presented to the hospital with left-sided exertional chest pain radiating to his left shoulder. This pain had started intermittently 2 months ago but acutely worsened while driving 24 hours before presentation. The patient denied any known family history of sudden cardiac death, had no history of smoking or alcohol use, and reported his only medication was a daily 81mg aspirin. Vital signs were notable for slight tachypnea and hypertension, but the remainder of the physical exam was unremarkable. While EKG performed in the ED showed normal sinus rhythm, the patient's initial Troponin-I level was 8322, concerning for NSTEMI. A repeat EKG was performed on transfer to the Telemetry unit, showing new T-Wave inversions in leads V2-V4, consistent with Wellens Syndrome (Critical stenosis of the Left-Anterior Descending Artery) and prompting urgent cardiac catheterization.

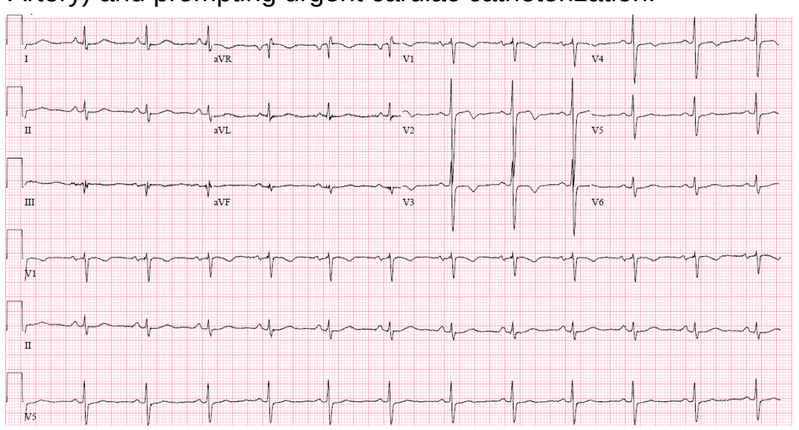

Fig. 1 Repeat EKG showing T-wave inversions in V2-V4, consistent with Wellens Syndrome.

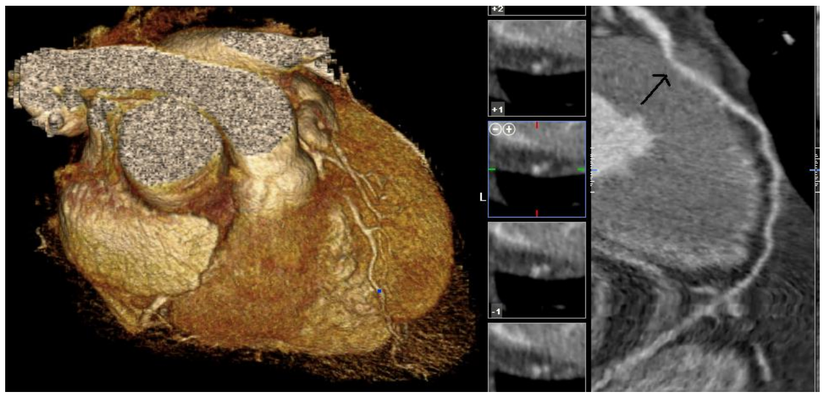

Fig. 2 Example of Myocardial bridging of the mid-LAD on curved planar reformatting CT Angiography. Arrow indicates systolic compression of the coronary lumen.[4]

Coronary angiography revealed a large-caliber Left-Anterior Descending artery with subtotal 99% and diffuse 80% proximal stenosis with TIMI I flow, and a prominent mid-LAD myocardial bridge with near systolic occlusion of the vessel. Balloon angioplasty and stenting was performed, reducing stenosis to 0% with TIMI 3 flow, however the prominent MB remained present at the end of the procedure. The patient remained symptom-free after the procedure and was discharged on antiplatelet therapy, a beta-blocker, calcium channel blocker, and high-intensity statin.

Discussion

MB-associated stenosis typically occurs proximal to the bridge location. While the underlying mechanism for this has not been fully elucidated, studies suggest that bridging causes alterations to the hemodynamic microenvironment, reducing proximal shear stress (SS) and increasing tensile stress (TS). This subsequently leads to increased local expression of adhesion molecules (ICAM/VCAM-1), inflammatory cytokines (TNF-α, IL-1), growth factors (PDGF), Endothelin-1 (ET-1), and matrix proteases (MMP) which further promote atherogenesis.[5-7]

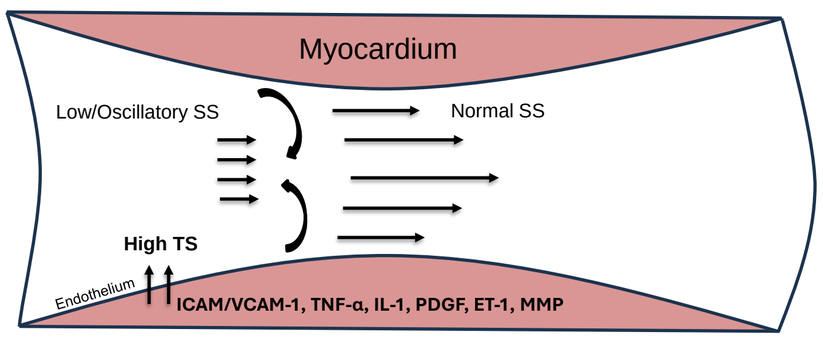

Fig. 3 Potential mechanisms for MB-proximal atherosclerosis. TS can be found using Laplace's law: TS= P(r/t) where P is blood pressure, r is radius of the vessel lumen, and t is vessel wall thickness.[5]

In addition to what is shown in figure 3, the formation of a proximal plaque is likely not the sole mechanism of myocardial ischemia. Intracoronary doppler flow and pressure measurements with coronary angiography have shown persistent decreases in vessel diameter as well as flow reserve in the early diastolic phase of symptomatic patients.[8] Therefore, tachycardia and increased contractility during stress/exercise may also contribute to impaired myocardial perfusion.

It is also important to note that, in cases of MB-associated ischemia, nitrates are contraindicated as they cause vasodilation of neighboring vessels, leading to perfusion steal and subsequently worsening the systolic narrowing of the affected vessel, further worsening symptoms.[8,9]

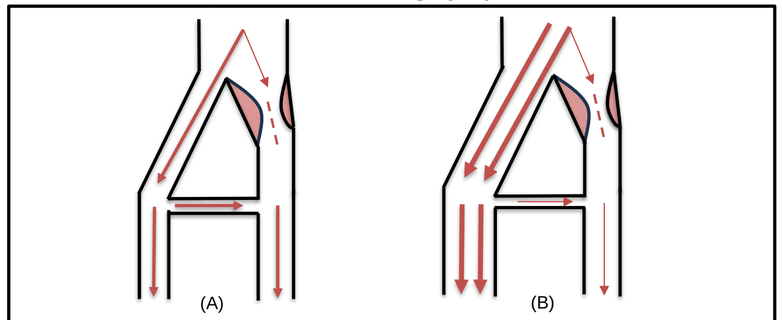

Fig. 4 Example of coronary perfusion steal. (A) Cardiac tissue distal to the MB receives adequate perfusion via collateral vessels. (B) After nitroglycerin administration, healthy vessel dilation prevents blood from crossing collateral vessels and provokes anginal pain.[9]

Conclusion

In closing, this case highlights the potential for myocardial bridging to contribute to acute coronary syndromes, such as NSTEMI, particularly when combined with proximal coronary arterial stenosis. While often asymptomatic, its presence can complicate the clinical course and management of coronary artery disease, emphasizing the need for careful evaluation and individualized treatment strategies. In patients receiving nitroglycerin for anginal symptoms whose pain is provoked rather than palliated, consider myocardial bridging as a potential underlying factor.

References

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