Not all ST Elevations are Infarctions: Myocardial Bridging in Dextrocardia

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Introduction

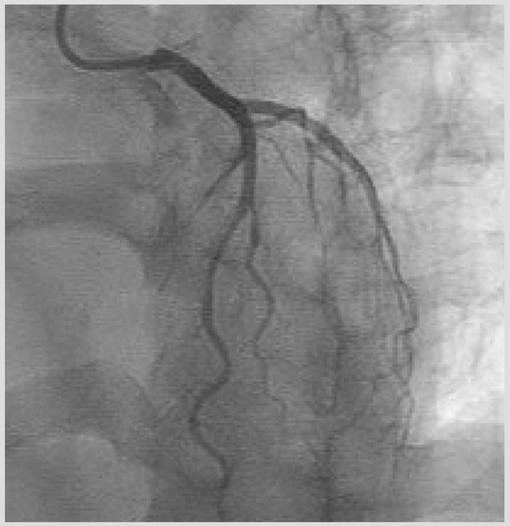
Myocardial bridging (MB)—most commonly involving the mid–left anterior descending artery (LAD)—can cause dynamic systolic compression, impaired diastolic filling, and demand–supply mismatch that clinically mimics acute coronary syndrome (ACS). Vasospasm and tachycardia may exacerbate ischemia in susceptible patients. Current reviews recommend beta-blockers or non-dihydropyridine calcium channel blockers as first-line therapy, with revascularization reserved for refractory cases. Nitrates should be used cautiously as they may worsen systolic compression.

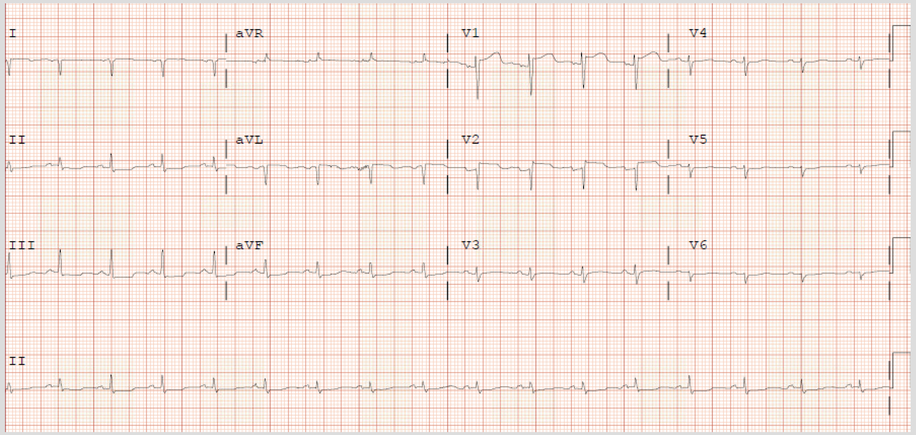
Case Description

A 48-year-old man with possible untreated type 2 diabetes, dextrocardia, and asthma presented with continuous emesis followed by hours of non-radiating substernal “lump-like” chest pain and mild dyspnea. He was hemodynamically stable; initial high-sensitivity troponin was <6 ng/L. Electrocardiography showed anterior ST-segment elevation (V1–V2) and reciprocal ST depression in inferior leads, poor R wave progression, right axis deviation. He was transferred for emergent coronary angiography.

Investigations

Left heart catheterization revealed a 40% mid-LAD occlusion representing myocardial bridge with dynamic systolic compression and no obstructive coronary disease. Chest pain resolved post-procedure, and serial troponins remained negative. Aspirin was continued, and prophylactic anticoagulation for venous thromboembolism was initiated. Further ischemia assessment using coronary CT angiography (CCTA) with or without CT-derived fractional flow reserve (CT-FFR) or functional stress testing to quantify ischemic burden and guide long-term management was recommended.





Discussion

This case highlights a STEMI mimic due to MB without fixed coronary stenosis, likely potentiated by transient coronary spasm. Prior reports document MB-associated ST- elevation and even myocardial infarction despite angiographically normal coronaries, underscoring the contribution of vasomotor dysfunction.
Dextrocardia complicates ECG interpretation; mirror-image right-sided precordial lead placement (V1–V6) is essential to obtain accurate tracings and avoid misclassification of anterior changes.
Management of symptomatic MB after exclusion of ACS centers on heart-rate reduction with beta-blockers or non-dihydropyridine calcium channel blockers, modification of cardiovascular risk factors, and reserving invasive or surgical strategies (e.g., unroofing, stenting) for objectively demonstrated, refractory ischemia.

Conclusion

In patients presenting with ST-elevation but angiographically normal coronaries, myocardial bridging with superimposed vasospasm should be considered. Resolution of symptoms
with persistently negative biomarkers strengthens this diagnosis.
Accurate ECG interpretation in dextrocardia requires correct lead placement, and downstream functional imaging (CCTA ± CT-FFR or stress testing) is critical for quantifying ischemia, guiding therapy, and avoiding unnecessary revascularization.

References

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