

### Introduction

- Myocardial bridging, though frequent, is often a forgotten cause of angina.
- It occurs when part of a coronary artery tunnels through the myocardium with the overlying muscle termed a myocardial bridge.
- The tunneled artery, by nature, is prone to enhanced myocardial compression, mechanical load, and vascular remodeling.
- During myocardial systole, the tunneled artery undergoes narrowing by way of myocardial compression leading to the classical presentation of angina.

### Case

We present a 29 year old Caucasian female with no significant past medical history who developed a sudden achy non-radiating pain in her left chest. It initially occurred while at rest and lasted approximately 30 minutes with resolution after aspirin administration. She had associated left hand pain and numbness but did not experience diaphoresis, dyspnea, nausea, lightheadedness, or headaches.

She has the Mirena IUD in place and has no known drug allergies. She smokes one-half to one pack per day for 11 years but has quit intermittently during her pregnancies. She drinks alcohol occasionally but does not use any illicit drugs. Family history is significant for a father with early coronary artery disease and stent placement in his early 40s.

Upon admission, she was found to have an elevated troponin to 0.36, which remained relatively stable throughout her stay. She was observed on a telemetry unit during her stay without any major arrhythmias identified or worsening of her symptoms.

Other relevant labs include:

D dimer <150  
CK: 64  
CK-MMB 0.9  
ANA negative

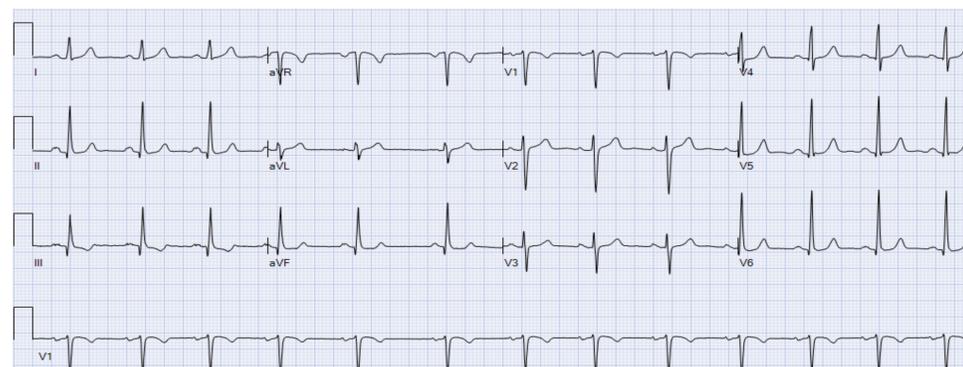


Figure 1: Electrocardiogram of our patient with myocardial bridging demonstrating normal sinus rhythm without any acute ischemic changes.

Chest radiography showed clear lungs without pleural effusions or consolidations.

CTA chest was obtained which revealed clear lungs and no pulmonary embolus. Heart was normal in shape and size. No pericardial effusion or pulmonary vascular congestion were noted.

An echocardiogram was performed revealing a hyperdynamic myocardium with an ejection fraction of 60-65%. Otherwise, echocardiography was unremarkable. She subsequently underwent a cardiac catheterization with images as shown. She was found to have a tubular zone of myocardial bridging of the left anterior descending artery with a mid vessel lesion of 40% (Figure 2). She was started on aspirin and diltiazem and discharged home. Upon outpatient follow-up with cardiology there was concern regarding diltiazem as she experienced episodes of lightheadedness and restless legs while taking the medication. Thus, she was switched to metoprolol succinate for therapy, which she has been tolerating well.

### Discussion

The finding of myocardial bridging in angiographic procedures ranges from 0.5% to 29.4%; however by autopsy the prevalence ranges from 5.4 to 85% [2].

- It most commonly involves the left anterior descending artery
- There is enhanced myocardial compression at the entrance of the myocardial bridge causing a disturbance of blood flow to the rest of the myocardium, which is resolved in the diastolic phase. This transient interruption of blood flow is thought to be the cause of myocardial ischemia and subsequent infarction and sudden cardiac death.

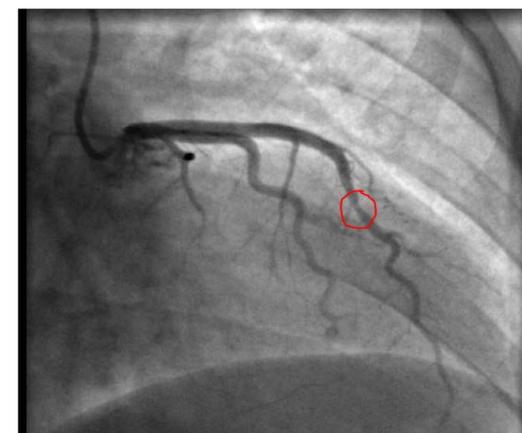


Figure 2: Coronary angiography of our patient demonstrating compression of the left anterior descending artery during systole (red circle).

- The underlying mechanism of endothelial injury and plaque formation of the tunneled artery includes
  - Increased oscillatory wall shear stress
  - increased vascular cell adhesion molecule expression
  - reactive oxygen species production
  - development of pro-atherogenic endothelial cells [3].
- Diagnostics
  - Resting EKGs rarely show any abnormalities
  - Coronary angiography remains gold standard
    - systolic compression is portrayed as a “milking effect with expansion of the vessel diameter during diastole
  - intravascular ultrasound: “half moon” sign, illustrated by an echolucent area between the bridged artery and the epicardial tissue [3].
  - cardiac CT, cardiac MRI, and TTE can be used, however the sensitivity and specificity of these studies are low

Symptoms and complications are associated with the degree of systolic narrowing rather than the length of the tunneled artery [4].

Treatment is focused on decreasing the compression of intramyocardial arteries via decreasing systolic contraction and prolonging the diastolic phase by way of negative inotropic and chronotropic agents such as beta-blockers and calcium-channel blockers.

- If atherosclerosis is present, anti-platelet therapy can be used in adjunct.
- Drugs to avoid include pure vasodilating agents such as nitrates as they intensify systolic compression of the tunneled segment leading to retrograde flow and thus worsening symptoms [3].
- If symptoms persist despite medical therapy, stenting of the vessel, surgical myotomy, or minimally invasive CABG are options [5] [6].

### Conclusion

- Myocardial bridging is a frequent but often forgotten cause of angina and can be present in up to 25% of the population.
- Patients can present with either typical or atypical chest pain syndromes however the vast array of complications arising from myocardial bridging require these patients to undergo prompt coronary angiography.
- Vascular spasm, wall stress of the tunneled artery, and intensity of systolic constriction coupled with any delay in management can lead to ischemia, infarction, and sudden cardiac death.
- To this end, it is imperative that patients who have low clinical suspicion for atherosclerosis but who are presenting with anginal equivalents undergo coronary angiography to assess for myocardial bridging and receive immediate treatment for this coronary anomaly.

### References

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