CLINICAL VIGNETTE

Myocardial Bridging – A Cause for Atypical Chest Pain

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A 43-year-old male with anxiety and hyperlipidemia presents to the emergency department with chest pain. He describes the pain as a sensation of left chest tightness, which is intermittent, non-radiating, and worse at night when he is trying to asleep. At night, he feels the chest tightness with blood "pounding in his neck", associated with heart racing. The pain has been recurrent for the last month and does not appear to worsen with activity. He reports decreased activity since the onset of his chest pain, with decreased exercise. While at work, he began feeling the same sensations and an electrocardiogram showed T wave abnormalities. He was advised to go to the ED. He has family history of cardiac disease: grandfather suffered a heart attack at age 60 and his father has coronary artery aneurysm status post Bentall procedure.

On examination, the vital signs were normal: afebrile, blood pressure 126/79, pulse 59, respiratory rate 16, and O2 saturation 100% on room air. He appeared fatigue, but not in acute distress. Chest pain was 1/10. Respirations were normal and lungs clear on auscultation. Heart was bradycardic to the mid-50s, with regular rhythm and no murmurs, rubs or gallops. Normal jugular venous pressure pulsation and no lower extremity edema. Abdomen and skin exam were normal.

Labs included CBC and chemistries were normal. Coagulation panels normal and troponins were negative x 2 and BNP was normal. Covid/flu pcr negative. Initial electrocardiogram showed normal sinus rhythm with T wave inversion in the inferior leads. Chest radiograph was normal. Cardiology consulted and obtained echocardiogram with Definity as well as CT coronary angiogram for risk stratification.

He was placed on telemetry while awaiting studies. Echocardiogram showed normal ejection fraction of 65-70%, without wall motion abnormalities, or significant valvular dysfunction. Although the CT coronary angiogram did not show plaque formation, it noted deep bridging of the middle segment of the left anterior descending (LAD) coronary artery with depth of 2mm and length of 13 mm. During systole, the bridge segment narrowed by 50% of the diastolic diameter.

Myocardial bridging is a congenital anomaly where a segment of the coronary artery has a "tunneled" intramuscular course under a "bridge" of myocardium.[1] This was first reported in 1737 by Reyman.[2] During systole, there is compression of the artery which is normally benign, however, if severe, may be associated with angina, myocardial ischemia, acute coronary syndrome, left ventricular dysfunction, arrhythmias, and death.[1] The prevalence of this phenomenon is frequent, found in about one third of adults.[3] Although any of the coronary arteries may bridge, the majority occurs with the LAD.[3] CT coronary angiography is the diagnostic gold standard. Clinical presentation of myocardial bridging has been associated with angina, myocardial infarction, left ventricular dysfunction, myocardial stunning, paroxysmal AV blockade, as well as exercise-induced ventricular tachycardia and sudden cardiac deaths.[2] However, despite the high prevalence of myocardial bridging, these complications are uncommon. Given the low prevalence of clinical findings, it has been a challenge to correlate data from diagnostic testing to attribute clinical ischemic symptoms directly to myocardial bridging, or indirectly to concomitant vasospasm, atherosclerosis or no findings.[4]

Complications of myocardial bridging include coronary spasms, thrombosis, coronary dissection, or atherosclerosis formation proximal to the myocardial bridge. The degree of systolic compression is thought to be associated with these complications. Myocardial bridging is otherwise benign.

First line medical therapy for patients experiencing symptoms include beta blockers and or calcium channel blockers to reduce heart rate.[1] Nitrates are contraindicated as they can to worsen systolic compression of the bridged segments.[1] For severe cases that do not respond to medical therapy, surgical myotomy and coronary artery bypass graft surgery have been considered. Risks from surgical intervention include dissection and death.[4] There are no accepted anatomic or function classification of myocardial bridging that provide basis for specific treatment.[4]

After discussing treatment options with cardiology, the patient opted not to initiate beta blocker therapy as his heart rate remained bradycardic during his hospital stay. Nitroglycerin was given during his CT coronary which led to a brief episode of atrial flutter with the patient briefly feeling worse after the medication. He returned to sinus rhythm without other interventions. Cardiology also recommended an outpatient stress test as well as cardiac monitoring and follow up. The patient was surprised with the diagnosis of myocardial bridging. He will monitor for symptoms before considering more invasive treatments.

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