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Hidden culprit: Unveiling recurrent chest pain masked by myocardial bridge

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ABSTRACT

A 52-year-old female presented to us in the emergency medicine department with a history of recurrent left precordial and retrosternal chest pain of dull aching type but not radiating to the left arm or neck, sometimes accompanied by sweating. These episodes had been occurring for 3 years, with each episode lasting for more than 1 h. The patient had a history of diabetes and hypertension and was on medications. She was evaluated outside; oesophago-gastro-duodenoscopy was done. It showed normal. She was on analgesics and on proton pump inhibitor. Our patient had persistent chest pain and was admitted for the same symptoms. After serial electrocardiogram (ECG) and serial troponin levels were noted to be normal. Hence, coronary angiogram was done. It showed a myocardial bridge lesion in the left anterior descending artery. It is a rare cause of chest pain, often we tend to narrow diagnosis once ECG and trop t negative. Significant obstruction can lead to myocardial ischemia, arrhythmia, and even sudden cardiac death. Early consideration in the working diagnosis enables early detection and treatment. This case report aims to enhance clinical decision making, improve patient outcome, and stimulate further research in this intriguing field.

Key words: Myocardial bridge; Chest pain; Coronary angiogram

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INTRODUCTION

Acute chest pain presentation in the emergency medicine room is often followed by serial electrocardiogram (ECG), serial serum troponin levels, and stratification of risk of myocardial infarction.[1] The process often overlooks vasospastic coronary syndromes presenting as recurrent chest pains with normal ECGs and normal serum troponin levels. This delays the diagnosis of coronary vasospasm particularly due to myocardial bridge lesion. Myocardial bridge lesion is an anatomical aberration in individuals where a segment of the coronary artery is buried in the myocardium causing episodic coronary vasospasm during myocardial contraction.[2] Coronary spasm, or coronary artery vasospasm, is a transient constriction of the coronary arteries, leading to reduced blood flow to the heart muscle. The exact mechanism involves several factors. Endothelial dysfunction, where the inner lining of blood vessels fails to produce enough vasodilatory substances, may contribute to increased vasoconstriction. Hyperreactivity of smooth muscle cells in the artery walls can cause sudden and intense constriction. An imbalance in the autonomic nervous system, which controls blood vessel tone, may trigger excessive vasoconstriction. Certain substances released within the artery walls, such as endothelin-1 and thromboxane A2, possess vasoconstrictive properties. Hypersensitivity of the coronary arteries to various stimuli, such as stress or certain drugs, may also induce spasms. The prevalence of Myocardial Bridge varies between 5% among those who undergo coronary angiogram (CAG) and more than 80% during autopsy.[3] This case report intends to show the need for a lower threshold for CAG in those with acute chest pain with non-specific or normal ECG and normal troponin. Early detection of myocardial bridges enables better prognosis and risk management.

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CASE REPORT

A female patient aged 52 years presented to us with left precordial chest pain not radiating to the left arm or neck, sometimes accompanied by sweating. It was non-exertional and not related to emotional factors. These episodes were experienced for more than hour per episode over the previous 3 years. There were no aggravating and relieving factors. It is not associated with breathlessness, palpitations, or giddiness. Episodes were present during sleep and rest. There was no history of nausea, vomiting, or reflux symptoms. She is known case of diabetes mellitus since 4 years and hypertension since 3 years and on oral medications and Insulin. During the recent episode of precordial chest pain, she experienced sweating. The Patient was seen by a primary physician immediately and sent home after ECG and 1st Serum Troponin being normal. She was treated with Proton pump Inhibitors. However, the patient continued to suffer the symptoms frequently once in 3 months. Hence, patient was admitted and evaluated. On examination, the patient was conscious and oriented. Pulse rate was 98 beats/min, blood pressure 130/80 mmHg, Jugular venous pulsation normal. All systemic examinations were normal.

The ECG (Figure 1) shows normal sinus rhythm with Q waves in lead III, aVF, and poor R wave progression. Serum troponin was negative. 2D Echocardiography showed no regional wall motion abnormality, mild MR, trivial TR, sclerotic AV trivial TR, concentric left ventricular hypertrophy, ejection fraction -60%, tricuspid regurgitation peak gradient-27 mmHg, No Clot, No Pericardial Effusion. On the advice of the cardiologist, a CAG (Figure 2) showed a bridge lesion in the left anterior descending (LAD) branch and it was presented with diastolic and systolic contrast. Fasting blood glucose-251 mg/dL, postprandial blood glucose-291 mg/dL, Hemoglobin A1C-10.1%, serum calcium-9.6 mg/dL, total cholesterol-121 mg/dL, triglycerides-221 mg/dL, high-density lipoprotein (HDL) cholesterol-43.0 mg/dL, low-density lipoprotein (LDL) cholesterol-69 mg/dL, very low-density lipoprotein cholesterol-44.2 mg/dL, total cholesterol/HDL cholesterol-2.8, total triiodothyronine electrochemiluminescence immunoassay (ECLIA)-0.96 ng/mL, total thyroxine (ECLIA)-9.11 µg/dL, thyroid stimulating hormone (ECLIA)-3.92 µIU/mL, ultrasound abdomen and pelvis-normal study.

The patient received initial anticoagulants, beta blockers, antiplatelets, and statin combination. This combination was later optimized with single antiplatelet, hydroxymethylglutaryl-CoA (HMG-CoA) reductase inhibitors (statins), beta-blockers, antianginal medications along with proton pump inhibitors. Blood sugars were

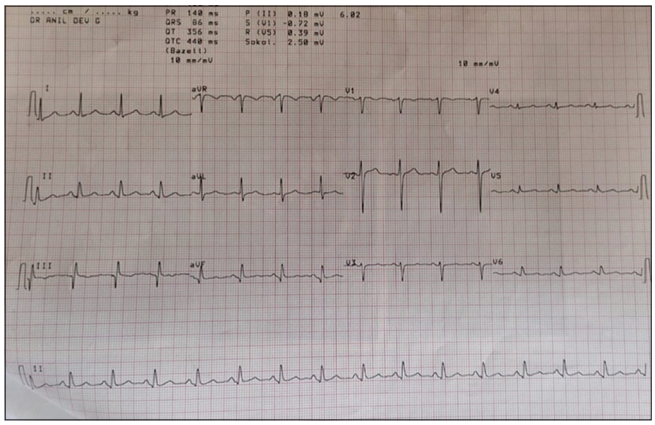

Figure 1: Electrocardiogram-normal sinus rhythm and Q waves in Lead III, aVF with poor R wave progression

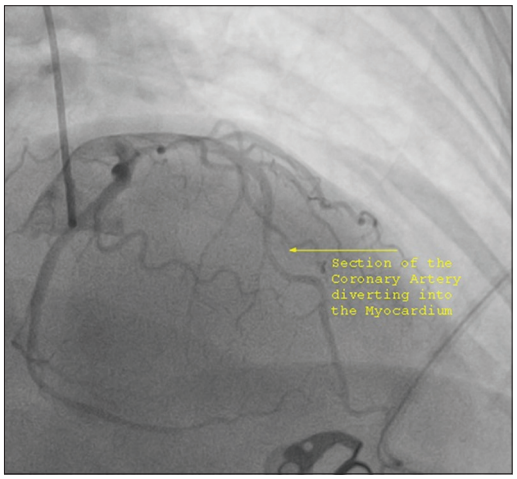

Figure 2: Coronary angiogram image showing the myocardial bridge lesion in the left anterior descending branch

controlled with Insulin. After CAG, the patient was followed up for 8 weeks. Patient was advised to continue beta-blockers, antiplatelets, and statins. In subsequent follow-up, the patient made a recovery from symptoms and regained her confidence in her own well-being.

DISCUSSION

Acute chest pain is a common complaint in the emergency room and can have various causes. Some of the life-threatening conditions that need to be ruled out are acute coronary syndrome, pulmonary embolism, aortic dissection, pneumothorax, cardiac tamponade, and esophageal rupture. A differential diagnosis should be based on the patient's history, physical examination, ECG, and cardiac biomarkers.

A myocardial bridge occurs when a segment of a coronary artery tunnels through the heart muscle instead of resting on its surface.[3,4] This can cause compression of the artery

during systole, potentially leading to symptoms like angina. Patients may present with recurrent chest pain, which can mimic those of coronary artery disease, leading to repeated emergency room visits. Diagnosis is based on CAG which is the gold standard for diagnosing myocardial bridges. It reveals the characteristic "milking effect" or systolic compression of artery. The LAD coronary artery and the junction of the proximal and middle thirds of the artery is the most common site reported for myocardial bridging[2] This is a congenital aberrance that results from the formation of a heart muscle over the segment of epicardial coronary arterial segment which when contracted would interrupt the blood supply through the affected epicardial coronary arterial segment. This interruption of blood supply would cause the symptoms of ischemic chest pain. Management includes medical therapy. First line treatment often includes beta-blockers or calcium channel blockers to reduce heart rate and myocardial contractility, thereby decreasing the compression of the artery. Lifestyle modifications include encourage patients to avoid triggers such as heavy physical exertion and stress. Surgical intervention in rare cases where medical therapy is ineffective, surgical options like myotomy or coronary artery bypass grafting may be considered. Prognosis, most of the patients with uncomplicated myocardial bridges have a good prognosis with appropriate medical management. Regular follow-up is essential to monitor symptoms and adjust treatment as needed. Educate patients about the nature of their condition and the importance of adhering to prescribed medications and lifestyle changes. Inform them about the signs of worsening symptoms that would necessitate immediate medical attention.

The prevalence of myocardial bridge in the population is noted to be approximately 5%. The ends of the myocardial bridge are at risk of narrowing due to atherosclerotic plaque or thrombus in circulation.[4] Thus risk management involves measures to reduce the risk for atherosclerotic plaque formation at the ends of the myocardial bridge lesion and also to avoid free thrombus formation in the circulation through antiplatelets and HMG-CoA reductase inhibitors. Intravascular ultrasound and flow fraction reserve have been used in determining need for stent placement in intermediate myocardial bridge lesion.[5] Myocardial bridge lesions are affected by mural coronary artery systolic stenosis, myocardial bridge thickness, and systolic and diastolic compressions of the heart.[6] Patients with a low risk for atherosclerosis or other cardiovascular diseases, if present with typical or atypical angina, myocardial bridge should be suspected as it can be caused by the extra pressure on the blood vessel under the bridge when the heart muscle contracts. Thus, myocardial bridge is considered a risk factor for myocardial infarction with non-obstructive coronary arteries.[7,8]

CONCLUSION

Myocardial bridges are generally considered benign congenital anomaly, but it is important to identify clinical scenarios in which they have the potential to cause myocardial ischemia and infarction. This case highlights the importance of expanding the differential diagnosis to myocardial bridging in the work-up for the cause of chest pain. Myocardial bridging is a frequent but often forgotten cause of chest pain. Patients can present with a variety of symptoms, including angina, dysrhythmias, and sudden cardiac death. Hence, these patients should undergo prompt coronary angiography.

Therefore, it is of vital importance that patients for whom there is clinical suspicion and presenting with atypical chest pain should undergo coronary angiography to assess for myocardial bridging and receive immediate treatment for this coronary anomaly to prevent morbidity and mortality.

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ADG- Concept and design of the study; prepared the first draft of manuscript, interpreted the results; **MTR-** Reviewed the literature and manuscript preparation; **MTR-** Concept, coordination, review of literature and manuscript preparation; **ADG-** Interpreted, preparation of manuscript and revision of the manuscript.

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