CASE REPORT

THROUGH THE LENS OF UNIQUENESS: AN ATYPICAL PRESENTATION OF MYOCARDIAL BRIDGE IN THE CORONARY TRIFURCATION

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Myocardial bridges are commonly encountered findings during coronary angiography, typically occurring over the mid or distal segments of the left anterior descending artery (LAD). Coronary trifurcation represents another variant of normal coronary anatomy. However, muscle bridges rarely involve a trifurcation branch. Symptomatic patients with muscle bridges often present with angina or dyspnea and may even experience acute coronary syndromes. Management typically involves medical intervention, with percutaneous coronary intervention (PCI) or cardiac surgery reserved for severe cases. Here, we present a unique case of a young female who presented with acute coronary syndrome due to a muscle bridge occurring over a trifurcation branch.

Keywords: Muscle Bridge, Coronary Trifurcation, Acute Coronary Syndrome

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INTRODUCTION

The major coronary arteries, including the left anterior descending (LAD), left circumflex (LCx), and right coronary artery (RCA), are typically epicardial, meaning they reside on the surface of the heart muscle. Among the various coronary anomalies, a muscle bridge is a notable condition where a band of heart muscle (myocardium) overlies a segment of an epicardial coronary artery. This anomaly is observed in approximately 20% of patients undergoing coronary angiography.[1] Muscle bridges are primarily detected over the LAD and can lead to significant systolic compression of the affected artery segment during the contraction phase of the cardiac cycle (systole). Despite this systolic narrowing, the myocardium, which predominantly receives its blood supply during the relaxation phase (diastole), may still be adequately perfused.

Although muscle bridges are often asymptomatic and considered benign, they can occasionally be linked to clinical symptoms such as angina (chest pain) and, in rare cases, acute coronary syndrome (ACS). The pathophysiological mechanism involves the transient reduction of blood flow due to the compression of the artery during systole, which can lead to myocardial ischemia and subsequent symptoms.

Another notable coronary anomaly is coronary trifurcation, where an additional major coronary artery branches off from the left main stem along with the LAD and LCx. This anomaly has an incidence of 24% in the Pakistani population and can present unique diagnostic and therapeutic challenges.[2] In this case report, we present a 54-year-old woman who developed ACS due to a muscle bridge over a trifurcation branch. We detail her presentation, diagnostic evaluation, and management plan, emphasizing the interplay between these coronary anomalies and the clinical approach to such a rare and complex condition.

CASE REPORT

Patient Information: A 54-year-old woman with a six-year history of Type-II diabetes mellitus and a four-year history of hypertension presented with chest pain that began two days prior. She also reported a three-month history of angina CCS-II and dyspnea NHYA-II. The chest pain was sudden in onset, moderate in intensity, radiating to the back, and associated with nausea and vomiting. She initially sought care at a nearby medical center, where her ECG showed ST segment depression in leads V4-V6, and a Troponin-T test using the kit method was weakly positive. She was managed medically, resulting in improvement of her chest pain, and was then referred

to our facility for coronary angiography and further management.

Clinical Findings: Upon presentation, the patient was vitally stable with a normal general physical examination. ECG showed persistent ST segment depression. Blood chemistry was within normal limits. A 2D echocardiography revealed an ejection fraction of 60%, with a structurally normal heart and no regional wall motion abnormalities.

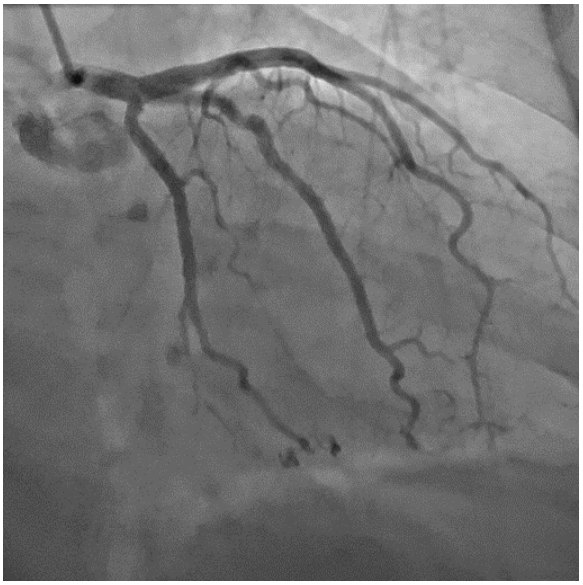

Figure 1: Coronary Angiogram in diastolic phase

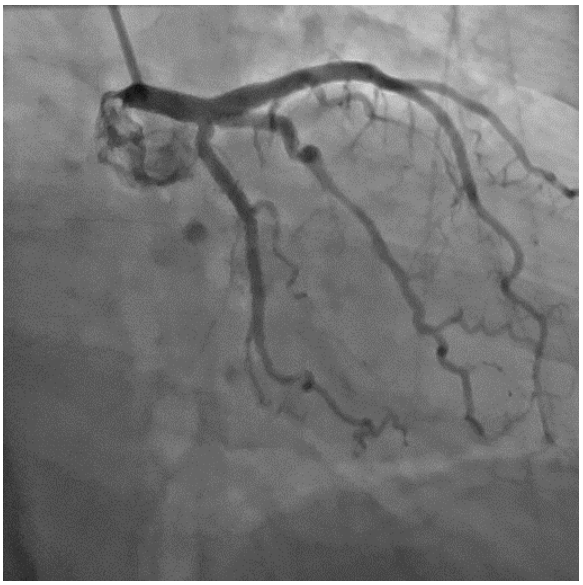

Figure 2: Coronary angiogram showing significant systolic squeeze in Trifurcation branch

Timeline

- **Two days prior to presentation:** Onset of chest pain.
- **Three months prior:** History of angina CCS-II and dyspnea NHYA-II.
- **Initial visit:** ECG showed ST segment depression in leads V4-V6; Troponin-T was weakly positive.
- **Current presentation:** Persistent ST segment depression; referred for coronary angiography.

Diagnostic Assessment: Coronary angiography was performed using a right radial approach. It revealed a trifurcating left main stem with a dominant right coronary artery. Both left and right coronary systems were free of atherosclerotic disease. However, a muscle bridge was noted over the trifurcation branch, causing significant systolic squeeze, as shown in Figures 1 and 2.

Therapeutic Intervention: The patient was started on beta-blockers to manage the muscle bridge. She was discharged the same day with instructions to continue the medication.

Follow-up and Outcomes: Three months later, the patient reported being asymptomatic and pain-free on the medical management. She remains on beta-blocker therapy with no further interventions planned unless symptoms reoccur.

DISCUSSION

The major coronary vessels, which are visualized during routine angiography, are epicardial, meaning they lie on the surface of the heart muscle. Occasionally, a band of cardiac muscle crosses one or more epicardial coronary arteries, a condition known as muscle bridging. Muscle bridging is a common anatomical variation, found in about 25% of postmortem studies.[3] Despite being epicardial, coronary vessels experience systolic compression, with only about 18% of the total coronary blood flow occurring during systole. These vessels receive the maximum blood supply during the diastolic phase.[4] Muscle bridges can cause delayed diastolic relaxation of the underlying artery, which is usually asymptomatic but can lead to ischemia under conditions of stress and increased myocardial oxygen demand. Additionally, muscle bridges may act as sites for atherosclerosis, compounding the risk of ischemia.

Approximately 90% of muscle bridges involve the left anterior descending artery (LAD),[5] with the left circumflex (LCx) and right coronary arteries (RCA) being less commonly affected. According to a study

by Rana et al., 98% of muscle bridges involve the LAD, with only 1-2% affecting the trifurcation branch.[6] In our case, the trifurcation exhibited significant systolic squeeze, a rare presentation. Muscle bridging is more prevalent in females, and while patients can present at any age, they are typically younger.[7] The risk factors commonly associated with ischemic heart disease, such as diabetes, hypertension, and stroke, are less prevalent in these patients.[8]

Most patients with muscle bridging are asymptomatic, but some present with angina or dyspnea.[9,10] Triantafyllis et al. reported a case of Takotsubo syndrome triggered by an LAD muscle bridge.[11] A study by Shah et al. found that 46% of Pakistani patients with muscle bridges also had concomitant atherosclerotic disease.[12] There are frequent case reports implicating muscle bridges as a cause of acute coronary syndrome. Matta et al. reported that 14.5% of patients with myocardial infarction with non-obstructive coronary arteries (MINOCA) had muscle bridges as a significant finding, with 85% presenting as non-ST-elevation myocardial infarction (NSTEMI).[13] Our patient also presented with NSTEMI, underscoring that significant myocardial bridging can cause cardiac ischemia and infarction. In this case, the absence of atherosclerotic disease suggests that the significant systolic squeeze of the trifurcation branch was the primary cause of the NSTEMI.

ECG findings in patients with muscle bridges are variable. Typically, there are no significant findings, though early repolarization and non-specific ST-T changes can be seen.[14] ST-elevation myocardial infarction (STEMI) has also been reported. 2D echocardiography may show regional wall motion abnormalities if myocardial infarction occurs due to a muscle bridge. Stress testing may be employed, but most cases are diagnosed by cardiac computed tomography angiography (CCTA), which not only confirms the diagnosis but also guides management. CCTA-derived fractional flow reserve (FFR) allows functional assessment.[15] Invasive coronary angiography, with its characteristic "milking" effect or systolic squeeze of the artery, often confirms muscle bridging. The "half-moon" sign visualized during slow IVUS pullback can also aid in diagnosis. Cardiac magnetic resonance imaging (CMR) is another diagnostic tool that can assess both the presence and functional significance of muscle bridges.[16]

Pharmacological treatment is the first-line therapy for most symptomatic patients with muscle bridges. Beta-blockers and calcium-channel blockers are commonly used, while nitrates should be used cautiously as they may exacerbate symptoms by increasing systolic squeeze. Our patient responded well to medical management with beta-blockers. For patients who remain symptomatic despite optimal medical therapy, or those presenting with STEMI, percutaneous coronary intervention (PCI) is considered[17]. However, no randomized data support the routine use of stents in muscle bridges.[18] While early reports indicated high in-stent restenosis rates, the use of drug-eluting stents has reduced this risk. Rare complications such as stent fracture, perforation, and thrombosis have been reported.[19] Surgical options, including coronary artery bypass grafting or "unroofing" with myomectomy, are alternatives for severe cases. Most patients with myocardial bridging have an excellent long-term prognosis.

CONCLUSION

Muscle bridges are a common anatomical finding, typically benign but capable of causing significant clinical symptoms, including angina and myocardial infarction, in severe cases. This condition should be considered in younger patients presenting with angina. Medical management is effective for most patients, while invasive procedures are reserved for those with severe or refractory symptoms. Early diagnosis and appropriate management are crucial for preventing adverse outcomes.

This case illustrates the rare occurrence of acute coronary syndrome (ACS) due to a muscle bridge over a trifurcation branch. Despite the potential for significant systolic compression, medical management with beta-blockers has proven effective, leading to an excellent outcome for the patient. Continued follow-up is necessary to monitor for any recurrence of symptoms and ensure long-term management success.

AUTHORS' CONTRIBUTION

AUHQ, WM, MR, WAL, MWH, and AM: Concept and design, data acquisition, interpretation, drafting, final approval, and agree to be accountable for all aspects of the work. AUHQ, WM, MR, WAL, MWH, and AM: Data acquisition, interpretation, drafting, final approval and agree to be accountable for all aspects of the work.

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