

Received: 2020.01.25

Accepted: 2020.04.30

Available online: 2020.05.20

Published: 2020.07.19

# Myocardial Bridging: A Case Presentation of Atypical Chest Pain Syndrome in a Young Woman

## Authors' Contribution:

Study Design A

Data Collection B

Statistical Analysis C

Data Interpretation D

Manuscript Preparation E

Literature Search F

Funds Collection G

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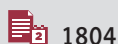
**Patient:** Female, 29-year-old  
**Final Diagnosis:** Myocardial bridging  
**Symptoms:** Chest pain  
**Medication:** —  
**Clinical Procedure:** Coronary angiography • CT scan • echocardiography  
**Specialty:** Cardiology • General and Internal Medicine

**Objective:** Rare disease

**Background:** Myocardial bridging, although frequent, is often a forgotten cause of angina. It is a congenital anomaly in which the coronary artery tunnels through the myocardium with the overlying muscle, termed a myocardial bridge. The tunneled artery is prone to increased myocardial compression, mechanical load, endothelial damage, and vascular remodeling. During myocardial systole, the tunneled artery undergoes narrowing caused by myocardial compression, which leads to disruption of blood flow, thereby precipitating angina, arrhythmias, infarction, and sudden cardiac death.

**Case Report:** Here, we present a case of a 29-year-old white woman who presented with atypical left-sided achy chest pain, occurring primarily at rest. Further evaluation showed mildly elevated troponins, with normal electrocardiogram, chest x-ray, and CTA chest. She subsequently underwent coronary angiography and was found to have myocardial bridging of her left anterior descending artery, with compression of up to 40% during systole. She was initially treated with diltiazem, but due to adverse effects was transitioned to metoprolol succinate, which she has tolerated well.

**Conclusions:** Myocardial bridging, although benign in nature, carries a vast array of complications requiring these patients to undergo prompt diagnosis and treatment. Vascular spasm, wall stress of the tunneled artery, and intensity of systolic constriction coupled with any delay in management can lead to ischemia, infarction, dysrhythmias, and death. Therefore, 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.

**MeSH Keywords:** Coronary Angiography • Coronary Vessel Anomalies • Myocardial BridgingFull-text PDF: <https://www.amjcaserep.com/abstract/index/idArt/923075>

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## Background

Myocardial bridging is a congenital variant of the coronary artery vasculature in which the vessel tunnels through the myocardium rather than running a typical epicardial course. The prevalence ranges anywhere from 0.5% to 85%, noted mostly on coronary angiography and autopsy [1]. It is primarily believed to be a benign finding, but it has been reported to cause myocardial ischemia, infarction, arrhythmias, and even sudden cardiac death [2]. These complications arise when the tunneled artery becomes compressed during systole and the stress and damage the vessel undergoes from the overlying muscle fibers and increased flow velocity. Treatment is therefore targeted towards decreasing the length and force of the contraction these arteries undergo. First-line therapy includes negative inotropic and chronotropic agents such as beta-blockers and calcium-channel blockers. Ivabradine and anti-platelet therapy could also be used if needed. If medical therapy fails to control symptoms, surgical intervention is warranted.

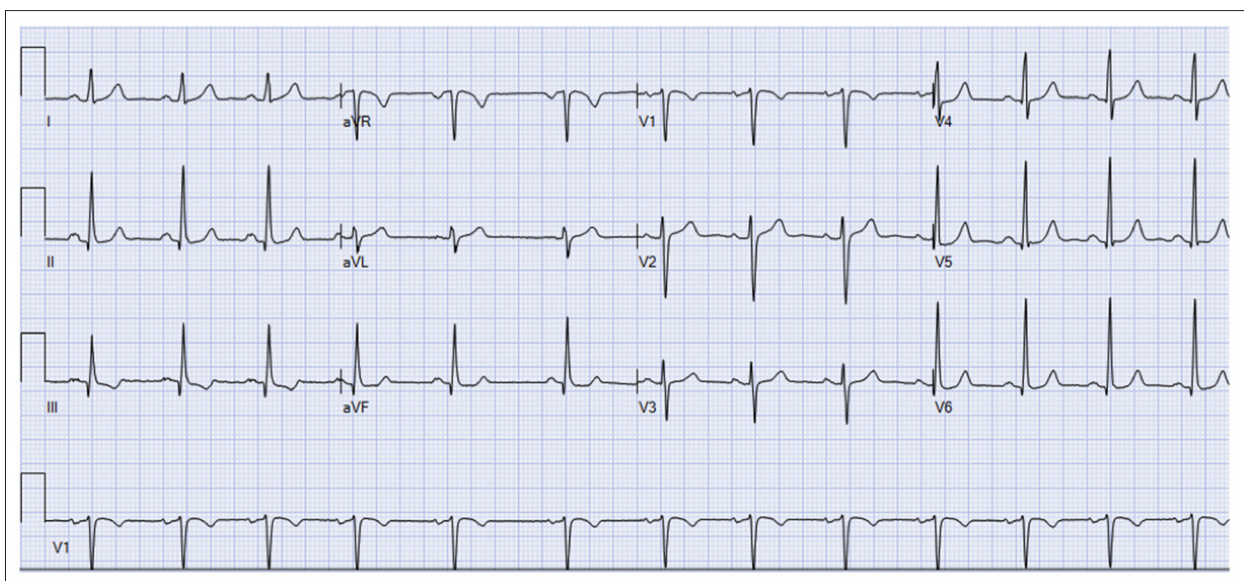
## Case Report

We present the case of a 29-year-old white woman with no significant past medical history who developed a sudden achy non-radiating pain in her left chest. It 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.

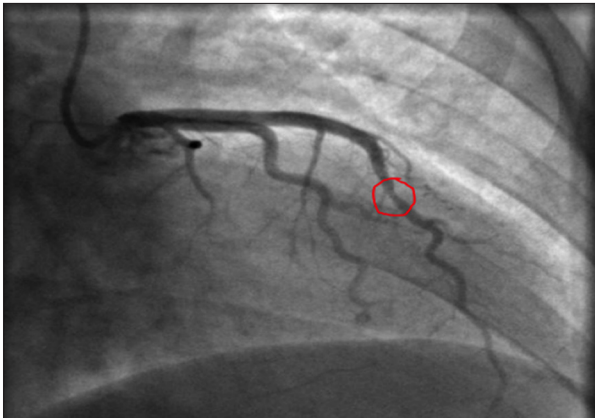
She had a Mirena IUD in place and had no known drug allergies. She had smoked one-half to one pack per day for 11 years but quit intermittently during her pregnancies. She drank alcohol occasionally but does not use any illicit drugs. Her family history was significant for a father with early coronary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

Upon admission, she was found to have an elevated troponin of 0.36, which remained relatively stable throughout her hospital stay. A urine drug screen was negative, minimizing the suspicion of drug-induced ischemia. D-dimer was obtained out of concern for a pulmonary embolus, but it was negative. Vasculitic pathology was considered; therefore, ESR and ANA were obtained, both of which were negative. CK and CK-MMB were both within normal limits, at 64 and 0.9, respectively, and a lipid panel was unremarkable. She was observed on a telemetry unit during her stay, without any major arrhythmias identified or worsening of symptoms.

EKG revealed normal sinus rhythm and did not exhibit any acute ischemic changes (Figure 1). Chest radiography showed clear lungs without pleural effusions or consolidations. A CTA chest was obtained, which revealed clear lungs and no pulmonary embolus. The 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, which revealed 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.



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



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

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

Myocardial bridging is a congenital anomaly described as tunneling of a coronary artery through the myocardium rather than running a typical epicardial course. Its prevalence ranges from 0.5% to 29.4% when detected by angiographic procedures; however, by autopsy, occurrences are noted to range from 5.4% to 85% [1]. Myocardial bridging most commonly involves the left anterior descending artery, as demonstrated in our patient, but there have been a few case reports of involvement of the right coronary artery. Typically a benign feature, symptoms usually do not appear until the third decade of life. Symptoms may include anginal equivalents, arrhythmias, and sudden cardiac death. The mechanism behind these symptoms lies in the disturbance of blood flow through the tunneled artery. There is enhanced myocardial compression in which the vessel enters into the myocardium, leading to a disturbance of blood flow to the rest of the myocardium. This disturbance mainly occurs during systole and is resolved in the diastolic phase. This transient hypoperfusion is thought to be the cause of presenting symptoms, myocardial ischemia, infarction, and sudden cardiac death. Our patient only described symptoms during rest; however, they can also occur with exertion.

During rest, there is vasospasm of the tunneled artery, as well as mechanical trauma to the vessel wall. The inherent nature of the tunneled artery, coursing through the myocardium, causes the arterial wall to undergo an increased amount of oscillatory wall shear stress, prompting increased vascular cell adhesion

molecule expression, reactive oxygen species production, and the development of pro-atherogenic endothelial cells. Together, the overexpression of these molecules and cells stimulate endothelial injury and plaque formation, leading to chest pain syndromes, dysrhythmias, ischemia, and death [3]. During exercise, ischemia is mediated by effort-induced tachycardia, which increases myocardial oxygen demand, contractility, and flow velocity, and reduces the coronary flow [4,5]. Symptoms and complications of myocardial bridging are associated with the degree of systolic narrowing, depth of tunneled artery, number of tunneled segments, and high heart rate [5,6].

Resting electrocardiograms rarely show any abnormalities in these patients; therefore, other modalities are needed to identify myocardial bridging. Coronary angiography remains the criterion standard of diagnosing the presence of myocardial bridging. The systolic compression of the tunneled artery is portrayed as a “milking effect” on coronary angiography, demonstrated as retrograde blood flow and subsequent antegrade flow with expansion of the vessel diameter during diastole. Other invasive studies such as intravascular ultrasound and intracoronary Doppler sonography can be used for diagnosis. Intravascular ultrasound characterizes the length, thickness, and location of the myocardial bridge. It is seen as a “half-moon” sign, demonstrated by an echolucent area between the bridged artery and the epicardial tissue that persists throughout the cardiac cycle [3]. Intracoronary Doppler demonstrates a diastolic fingertip phenomenon specific for myocardial bridging, characterized by rapid diastolic blood flow followed by a plateau in the bridged segment [7].

Noninvasive studies such as cardiac CT, cardiac MRI, and transthoracic echocardiography can also be used to diagnose myocardial bridging, but these are not the modalities of choice. Cardiac CT is a currently evolving method as an alternative to diagnosing coronary anomalies. It provides information regarding the lumen and walls of the coronary arteries and is effective in evaluating coronary anomalies, as well as atherosclerosis, stent patency, bypass grafts, and myocardial irregularities [8]. It offers the advantage of being noninvasive, using lower doses of radiation, and usefulness in treatment planning. Although is not the criterion standard for diagnosis, cardiac CT would have been a great option to diagnose myocardial bridging in our patient, given the low pretest probability of discovering significant coronary atherosclerosis on angiography as the cause of her presentation in light of her young age and minimal risk factors.

Our patient, without any significant cardiac history or risk factors, was found to have a 40% lesion of the mid-LAD from myocardial bridging, likely due to the effects of shear stress of the tunneled artery from repetitive compression. It is unclear if her family history of early coronary disease had an impact

on her coronary anomaly. Cerit found that patients with myocardial bridging have increased levels of inflammatory markers, neutrophil-to-lymphocyte ratio, and platelet activity [9]. It is well known that increased platelet activity and increased neutrophils play a key role in the development and destabilization of atherosclerotic plaques in cardiovascular disease. Although she did not exhibit any atherosclerosis on angiography, there may have been a genetic component of platelet and neutrophil activity contributing to her symptoms, especially given the history of early cardiovascular disease in her father.

Given the pathophysiology underlying myocardial bridging, treatment is focused on decreasing the compression of intramyocardial arteries via decreasing systolic contraction and prolonging the diastolic phase. First-line therapy includes negative inotropic and chronotropic agents such as beta-blockers and calcium-channel blockers, both of which were used in our patient. These agents lower transmural pressures, reducing the compression of the tunneled artery and prolonging diastole, thus mitigating symptoms. They have the added benefit of reducing heart rates, thus decreasing the frequency of systolic compression and increasing the diastolic period. Ivabradine has also been shown to have an effect on myocardial bridges by inhibiting the pacemaker current, thus decreasing heart rates and subsequently prolonging diastole [1]. If atherosclerosis is present, anti-platelet therapy can be used as an 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]. Intracoronary injection of dobutamine, epinephrine, and isoproterenol was also shown to exacerbate vessel narrowing during systole and delay diastolic relaxation [5]. If symptoms persist despite medical therapy, intracoronary stenting or surgical myotomy can be considered, as these measures also counteract transmural pressures [10]. Minimally invasive coronary artery bypass grafting has also been reported as an option for the management of myocardial bridges [11].

In evaluating the cause of atypical chest pain syndromes in patients with low risk for atherosclerosis, as in this case, it is also important to consider other nonatherosclerotic causes of

myocardial injury such as MINOCA (myocardial infarction with nonobstructive coronary arteries), microvascular angina, vascular spasm, coronary dissection, and coagulation disorders, as they can present similarly. MINOCA occurs in patients presenting with an acute myocardial infarction but in the absence of obstructing coronary disease, with no lesion greater than 50% and without another explanation for the presentation [12]. It is mostly seen in younger female patients presenting with NSTEMI, similar to our patient [13]. MINOCA was considered in this case, but with the angiographic findings of the existing myocardial bridge, we postulated that our patient's presentation was due to the bridge. Microvascular angina, vascular spasms, and coronary dissection could also be considered; however, with no change in electrocardiography and the findings of myocardial bridging on angiography, our suspicion for other causes remained low. Nonetheless, it is imperative to perform complete imaging studies for diagnosing coronary anomalies in patients that have low pretest probability of atherosclerosis but who are presenting with anginal equivalents, as these anomalies can cause significant complications.

## Conclusions

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 a variety of symptoms, including angina and dysrhythmias, but 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. Therefore, it is imperative that patients for whom there is 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.

## Conflicts of interest

None.

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