



CAUSE OF PERSISTENT CHEST PAIN: TWO MYOCARDIAL BRIDGES IN A YOUNG WOMAN

 Zeynep Rana Yıldız¹,  Servet Altay²

¹Trakya University School of Medicine, Edirne, TÜRKİYE

²Trakya University School of Medicine, Department of Cardiology, Edirne, TÜRKİYE

ABSTRACT

Myocardial bridging is a congenital anatomical anomaly characterized by a coronary artery with an epicardial course that dips into the myocardial tissue before resurfacing on the heart's surface. While it is commonly observed in the left anterior descending coronary artery, it typically occurs in a single coronary artery or a segment thereof. Myocardial bridging is generally asymptomatic and benign; however, in some cases, it can present with symptoms such as chest pain, arrhythmias, myocardial infarction, coronary vasospasm, syncope, and even sudden death. In our case, a patient admitted to our hospital with persistent chest pain for 6 months had myocardial bridges involving two different segments of the left anterior descending artery. Therefore, myocardial bridges should be considered in young patients presenting with angina pectoris.

Keywords: Angina pectoris, coronary artery, myocardial bridging

INTRODUCTION

Coronary arteries typically run through the outer layer of the heart known as the epicardium. However, in some cases, branches of the coronary arteries traverse the myocardial muscle tissue before returning to the heart's surface. This congenital situation is referred to as myocardial bridging (1).

Myocardial bridging can occur in any coronary artery, including the right coronary artery (RCA), left anterior descending artery (LAD), circumflex artery, and their branches, but it is mostly present in LAD (2, 3).

Although it is usually benign, patients can present with serious complications such as ischemia, syncope, ventricular tachycardia, arrhythmia, cardiac arrest, and even sudden death (4-7).

In this article, we discuss a 36-year-old female patient presenting with exercise-induced angina pectoris.

CASE REPORT

A 36-year-old female experiencing exertional chest pain for the past six months, classified as class II according to the

Canadian Cardiovascular Society angina severity classification, was admitted to our hospital. She had no history of smoking, hypertension, diabetes mellitus, hyperlipidemia, or family history of cardiovascular diseases. The patient had been started on metoprolol 50 mg, acetylsalicylic acid (ASA) 100 mg, and atorvastatin 20 mg at an external center due to her symptoms.

On admission, her blood pressure was 120/82 mmHg, and her heart rate was 86 beats per minute. Physical examination of the heart and respiratory system revealed normal findings, with no cardiac murmurs and pulmonary edema. The baseline electrocardiogram documented a sinus rhythm at 80 beats per minute, with no atrioventricular conduction irregularities or repolarization abnormalities. A transthoracic echocardiogram showed normal results.

Her levels of troponin, hemoglobin, thyroid function, lipid panel, and serum creatinine were all normal. An exercise stress test was performed, which revealed a 0.5 mm ST segment depression in the infero-lateral leads, and the patient reported non-specific chest pain. Considering the mismatch between the symptoms and the exercise stress test findings, a myocardial perfusion scintigraphy was performed.



Address for Correspondence: Zeynep Rana Yıldız, Trakya University School of Medicine, Edirne, TÜRKİYE

e-mail: zeynep.yildiz99@yahoo.com

ORCID iD of the authors: ZRY: 0000-0003-4488-7451; SA: 0000-0001-7112-3970.

Received: 29.09.2023 Accepted: 12.06.2024

Cite this article as: Yıldız ZR, Altay S. Cause of persistent chest pain: two myocardial bridges in a young woman. Turk Med Stud J. 2024;11(2):59-61.



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Following the detection of 10% ischemia in the anteroseptal wall via myocardial perfusion scintigraphy, coronary angiography was conducted. The angiography revealed two myocardial bridges located in the proximal and middle segments of the LAD artery. These bridges caused a critical 95% stenosis during systole, a phenomenon known as the milking effect (Figure 1a). Interestingly, during diastole, there was a significant improvement in the stenosis of the tunneled coronary artery segment (Figure 1b). Notably, the pre-stenotic proximal and distal segments had no pathological findings. The lengths of the myocardial bridge segments were measured at 22 mm in the proximal segment and 15 mm in the middle segment.

Subsequently, we discontinued statin and ASA therapy and adjusted the metoprolol dosage based on her blood pressure and heart rate. During the follow-up appointments, a decrease in angina was observed.

DISCUSSION

Myocardial bridging is a congenital anomaly of the coronary arteries. It occurs when a segment of the coronary artery traverses the myocardial tissue, forming a "bridge" of muscle (3). The historical background of myocardial bridging reveals early observations, with the first descriptions dating back to 1737 and 1805. In 1951, the first examination of myocardial bridges in autopsy was performed, followed by radiological descriptions in 1960 (8).

The prevalence of myocardial bridging varies in different studies. Autopsy series have reported frequencies ranging from 15% to 85%, while angiographic procedures show lower rates, ranging from 0.5% to 16% (1, 9, 10).

In retrospective studies conducted by Akyol et al. (11) and Oylumlu et al. (12) in Türkiye, myocardial bridging was found to be more common in men than in women, with mean ages of 59.3 and 57.1, respectively. However, our patient's age was below these mean ages, which is unusual. Male predominance has been reported in autopsy and angiographic studies (6, 8).

Myocardial bridges can also be categorized based on their depth in the myocardial tissue, with the superficial type being more prevalent, accounting for approximately 75% of cases (2, 13).

While myocardial bridging can occur in any coronary artery, it is most frequently observed in the middle segment of the LAD (2). Various studies report different incidences; for instance, Yukio et al. (14) reported 68.1% of cases in the middle segment of the LAD, while Loukas et al. (8) found different percentages for various arteries (14-16). In a necropsy study Ferreira et al. (13) conducted, 50 hearts with myocardial bridges were examined, 35 of the 50 hearts had single myocardial bridge and only appeared on LAD, 10 of them had two myocardial bridges and 5 of them had three. Such variations may arise due to population differences and study methodologies. In our case, we identified two consecutive myocardial bridges causing sequential critical stenosis in the LAD coronary artery, which is a rare occurrence.

The RCA is the second most common vessel and has been reported between 1-18.5% (8, 11, 14).

There are various methods to make a diagnosis of myocardial bridging. Multidetector computed tomography coronary angiography, conventional coronary angiography, and intravascular ultrasound are methods used to diagnose myocardial bridging (9). Non-invasive techniques, such as nuclear stress tests and stress echocardiography, or invasive techniques, such as fractional flow reserve, can be used to evaluate whether myocardial bridges cause ischemia. In our case, we diagnosed the patient, who had presented to our hospital with a six-month history of exertional angina, using coronary angiography.

The proximal segment of the myocardial bridge can be associated with atherosclerosis due to mechanical and blood flow changes (16). Artery compression during systole leads to hemodynamic alterations, and substances like lipids and mucopolysaccharides conglomerate to the coronary artery segment proximal to the myocardial bridge. Additionally, higher expression of vasoactive agents was detected in the segment proximal to the myocardial bridge (16, 17).

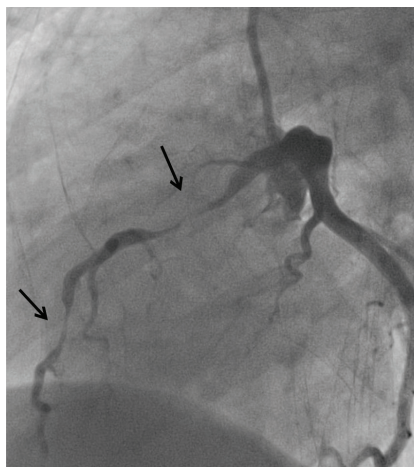


Figure 1a

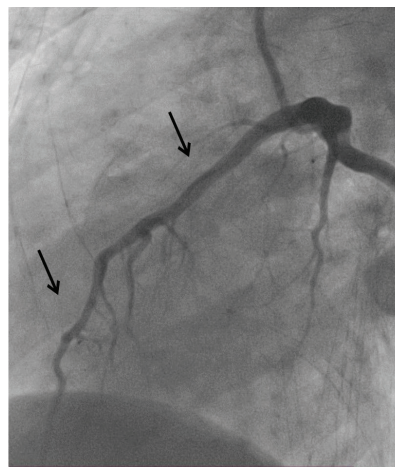


Figure 1b

Figure 1: Myocardial bridging was identified on coronary angiography.

Arrows: Myocardial bridging was stenosed about 95% during systole in proximal and middle segments and recovered during diastole.

Myocardial bridging has traditionally been considered a benign condition, but in some cases, compression during systole on a portion of the coronary artery can lead to blood flow obstruction, resulting in various complications. These complications may include myocardial infarction, arrhythmias, exertional angina, vasospastic angina, acute coronary syndrome, syncope, and even sudden death (2, 16-18). Other potential causes such as microvascular angina, myocardial infarction with non-obstructive coronary arteries, vascular spasm, spontaneous coronary dissection, and coagulation disorders should be considered in the differential diagnosis for individuals in this age group.

Pharmacological treatment options for symptomatic patients with myocardial bridging include beta-blockers as the first choice, calcium channel blockers, nitrates, ivabradine, and antiplatelet therapy. From the perspective of long-term prognosis, the effectiveness of stent implantation at the site of a myocardial bridge is not favorable, and thus, coronary stents are generally not preferred. Coronary artery bypass grafting and surgical myotomy may be the final treatment options for patients with refractory symptoms and signs (16, 19, 20).

The long-term prognosis for patients with myocardial bridging remains a subject of debate. Some studies suggest that patients with myocardial bridging, in the absence of other cardiovascular diseases have a good long-term prognosis while others indicate an elevated risk of non-fatal myocardial infarctions and adverse cardiac events associated with myocardial bridging (21, 22).

In conclusion, we have presented this case due to its unique characteristics, which are less commonly reported in the literature. These include the patient's young age, female gender, and the presence of two distinct myocardial bridges within the same artery. This case emphasizes the importance of considering atypical presentations and diagnostic challenges in the management of coronary artery disease, particularly in young and female patients. Further research and documentation of such cases may contribute to comprehend the spectrum of cardiovascular pathologies and their management strategies.

Ethics Committee Approval: N/A

Informed Consent: Informed consent was obtained from the patient.

Conflict of Interest: The authors declared no conflict of interest.

Author Contributions: Concept: S.A., Design: S.A., Data Collection or Processing: Z.R.Y., Analysis or Interpretation: S.A., Literature Search: Z.R.Y., Writing: Z.R.Y.

Financial Disclosure: The authors declared that this study received no financial support.

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