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Case Report
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# Myocardial Infarction in a Young Asymptomatic Athlete due to a Myocardial Bridge

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## Abstract

**Background:** Coronary artery anomalies are among the causes of sudden cardiac death in young athletes and should always be considered in the setting of abnormal paraclinical medical examinations, even if no symptoms are present.

**Case Summary:** We report a case of a 16-year-old asymptomatic male patient, in whom an abnormal ECG during pre-sports participation screening triggered further investigation. Multimodality imaging with coronary CT angiography and stress perfusion cardiac magnetic resonance led to the diagnosis of an extensive myocardial ischemic scar due to a long and deep myocardial bridge of the left anterior descending artery. The patient was initially advised to abstain from competitive exercise due to the presence of peri-infarct myocardial ischemia. Because of the extent of the ischemia and the patient's desire to exercise, surgical unroofing was recommended.

**Conclusion:** Although commonly benign, long, and deep myocardial bridges may occasionally be associated with large ischemic scars. Young athletes may be totally asymptomatic due to high coronary flow reserve. Multimodality imaging has a role in the thorough evaluation of such cases.

**Keywords: Case report; Pre-sports participation screening; Myocardial ischemia; Myocardial bridge**

## Abbreviations

ACE: Angiotensin Converting Enzyme; CMR: Cardiovascular Magnetic Resonance; CCTA: Coronary Computed Tomography Angiography; ECG: Electrocardiogram; LGE: Late Gadolinium Enhancement; MB: Myocardial Bridge

## Introduction

Although myocardial bridges are a relatively common condition, they often stay undetected due to the absence of symptoms and a low clinical suspicion. Patients with myocardial bridging can range from asymptomatic to highly symptomatic. The symptoms arise from the systolic compression of the intramyocardial segment which leads to myocardial ischemia, and may include exertional angina, shortness of breath, fatigue, or palpitations [1]. The case presented herein demonstrates the importance of a thorough evaluation during pre-sports participation screening of young athletes. Abnormal ECG findings, such as pathological T-wave inversions, should not always be associated with underlying cardiomyopathies, but coronary artery anatomy needs to be evaluated too. This case highlights that myocardial bridges may be the cause of an ischemic scar even in the absence of symptoms and reflects the value of multimodality cardiac imaging by Cardiac Magnetic Resonance (CMR) and Coronary CT Angiography (CCTA) in the diagnostic work-up of these patients.

## Case Presentation

A 16-year-old male semi-professional footballer presented to our Unit for pre-sports participation screening. He was asymptomatic, reporting no episodes of presyncope/syncope, palpitations, chest pain or shortness of breath on exertion. On physical examination the patient had a good body habitus, no heart murmurs, and no abnormal findings from chest auscultation. The ECG showed T-wave inversion in leads II, III, aVF, and V4-V6, and pathological Q-waves in leads I and aVL (Figure 1). The patient's transthoracic echocardiogram was normal and could not explain the abnormal ECG.

Past medical history was unremarkable. There was family history of premature cardiovascular disease, i.e., death due to acute myocardial infarction of the maternal grandfather at the age of 45.

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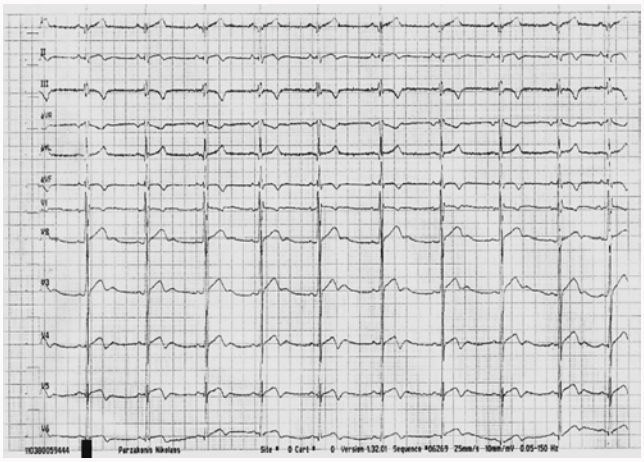

**Figure 1:** ECG. Electrocardiogram showing T-wave inversion in leads II, III, aVF, V4-V6, and pathological Q-waves in leads I, aVL.

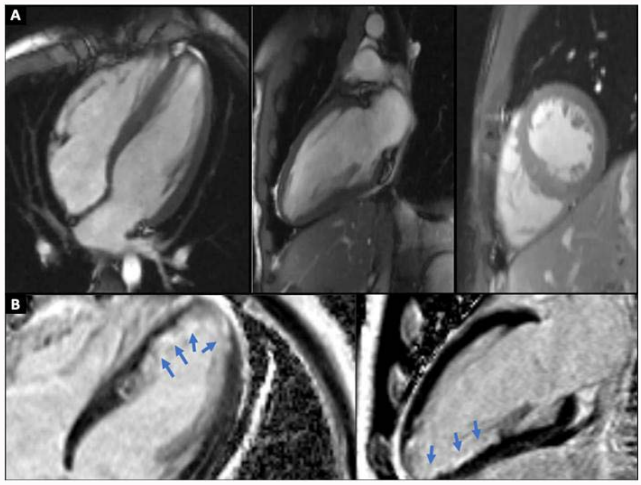


**Figure 2:** CMR. A) CMR images showing no left ventricular hypertrophy, normal wall tapering and no crypts. B) LGE images showed an ischaemic scar (blue arrows) in the mid-apical interventricular septum, the apical inferior wall, and the true apex.
CMR: Cardiac Magnetic Resonance; LGE: Late Gadolinium Enhancement

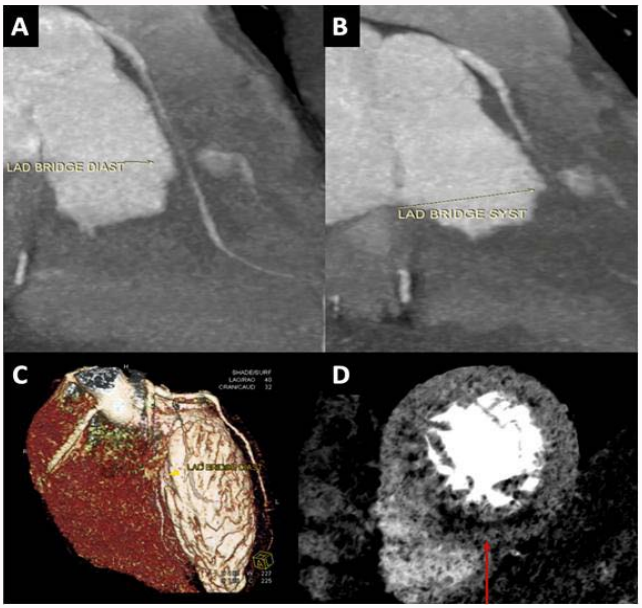


**Figure 3:** CCTA. A) CCTA image showing the myocardial bridge in the LAD and B) LAD obliteration in systole. C) 3D volume rendered CCTA image showing the myocardial bridge in diastole. D) Minimum intensity projection CCTA image showing the myocardial infarction (red arrow).
CCTA: Coronary Computed Tomography Angiography; LAD: Left Anterior Descending Artery

No history of cardiomyopathy was reported.

Given the patient's ECG, transthoracic echocardiographic findings and the epidemiological profile, two main differential diagnoses were considered. A probable subclinical cardiomyopathy was suspected as the leading diagnosis. The alternative diagnosis was the presence of a coronary artery anomaly, and more specifically of a myocardial bridge, whose compression could lead to myocardial ischemia, thus interpreting the pathological T-wave inversion.

A CMR scan was performed to further assess for possible underlying cardiomyopathy. CMR showed normal left ventricular function and volumes, and the presence of a subendocardial scar on Late Gadolinium Enhancement (LGE) images (Figure 2). The LGE pattern was in keeping with an ischemic scar, which was an unexpected and atypical finding given the young age of the patient and the absence of any anginal symptoms and traditional cardiovascular risk factors.

To rule out coronary atherosclerosis or any coronary artery anomalies that could explain the ischemic scar, a CCTA scan was performed. The CCTA revealed a myocardial bridge, i.e., a deep (8 mm) and long (60 mm) intramyocardial course of the mid left anterior descending artery (Figure 3), which was most likely the cause of the infarct. Additionally, there was a second myocardial bridge in the obtuse marginal branch of the left circumflex artery.

To further stratify the arrhythmic risk of the patient, a 24-h ECG Holter monitoring was performed, which did not demonstrate any arrhythmogenicity; exercise treadmill test was also normal without any evidence of ischemia, symptoms, or malignant ventricular arrhythmias.

The patient was advised to abstain from exercise until ruling out active myocardial ischemia. Beta-blockers and Angiotensin-Converting-Enzyme (ACE) inhibitors were initiated. The possibility of cardiac surgery for the unroofing of the myocardial bridge was mentioned. A discussion with the patient and the patient's family in the context of shared decision-making was proposed. However, the patient strongly wished to continue exercising and avoided any contact for the next 12 months.

At reevaluation, resting ECG and echocardiography were unchanged, and a follow-up CMR scan was ordered, to assess for left ventricular remodeling, or changes in systolic function and the extent of the scar. The follow-up CMR scan showed no significant changes in biventricular volumes or function; the ischemic scar was also unchanged. However, stress perfusion CMR (with regadenoson) showed evidence of inducible peri-infarct myocardial ischemia (Figure 4).

Given that patients with myocardial ischemia should not be allowed to engage in competitive sports, the patient was advised to abstain from competitive exercise. The patient was referred to a specialized cardiothoracic surgery center for surgical unroofing. However, the patient refused to proceed with the operation. He

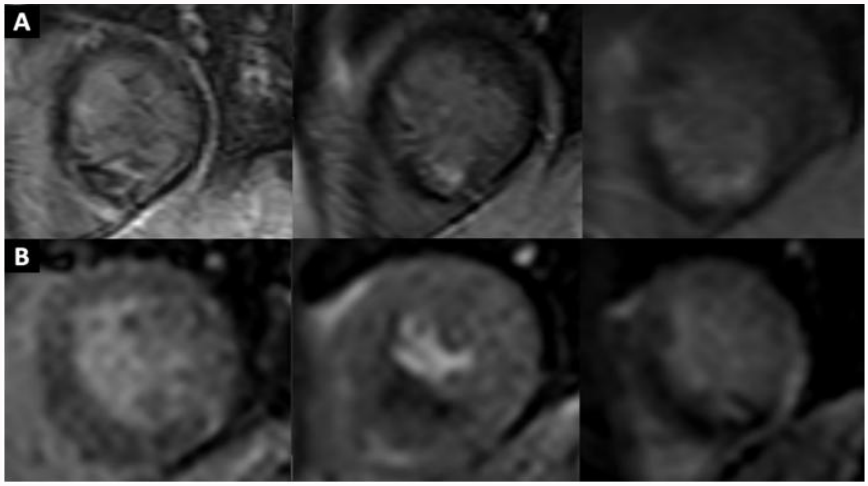


**Figure 4:** Follow-up CMR. A) LGE images showing no changes in the ischaemic scar. B) Stress perfusion images showing evidence of inducible peri-infarct ischemia.
CMR: Cardiac Magnetic Resonance; LGE: Late Gadolinium Enhancement

abstained from competitive exercise and continued receiving the prescribed pharmacological treatment.

## Discussion

Myocardial bridging is a relatively common anatomical variation [1], which is considered a benign finding that is not associated with excess cardiovascular risk [2]. However, not every case of myocardial bridging carries the same risk; in the case of long and deep myocardial bridges systolic obliteration and milking of the intramyocardial segment of the epicardial coronary artery may cause symptoms due to myocardial ischemia and more rarely an infarct [2,3]. During exercise, the inducible ischemia caused by the compression of the myocardial bridge is more prominent. The normal increase in coronary blood flow during the systolic phase that is observed with the elevation of heart rate in healthy individuals cannot be achieved, due to the myocardial bridge's compression from the cardiac muscle [4].

The atherosclerotic burden of individuals with myocardial bridging, and particularly of the coronary arteries segment proximal to the myocardial bridge, should always be evaluated. In the presence of atherosclerotic lesions, the individual should be treated as patient with coronary artery disease, even if the myocardial bridge causes no symptoms [5].

Myocardial bridges are classified into two types: Superficial and deep. Importantly, there is not definitive classification of the bridging based on their depth [6]. According to the typical criteria, superficial myocardial bridges have a depth between 1 mm and 2 mm, while deep myocardial bridges have a depth greater than 2 mm [2]. Superficial myocardial bridges are significantly more common than deep ones, accounting for approximately 2/3 of the cases [4,6]. Deep myocardial bridges, however, are more frequently associated with complications, and mainly with myocardial ischemia due to excessive compression of the affected coronary artery [3]. Apart from the depth of the myocardial bridge, another important factor that determines the functional impact to the heart is the length of the coronary arteries tunneled segment. This length typically ranges from 4 mm to 80 mm. Bridges longer than 25 mm are considered long and are more likely to cause myocardial ischemia [7]. In our case, the bridge was measured to be 8 mm deep and 60 mm long. These anatomical characteristics mark a high probability of severe functional impact to the myocardium and interpret the patient's persistent ischemia and the existence of a myocardial infarction.

Interestingly, our patient had suffered a large infarct without any symptoms. This is a rare finding, considering that asymptomatic patients with myocardial bridging usually have minimum systolic obliteration and limited-to-no ischemia. However, development of ischemic scarring with no accompanying symptoms can be explained by the high coronary flow reserve capacity that these trained individuals have [8].

Asymptomatic individuals with no evidence of myocardial ischemia and/or infarction usually do not need to receive treatment for this condition. Patients with symptoms and/or evidence of ischemia may be offered beta-blockers or calcium channel blockers; these drugs, however, can affect athletes' performance and are often poorly tolerated in such individuals [9]; aspirin is indicated only in the presence of atheromatous plaques, while ACE inhibitors can be given in the presence of a myocardial infarction [5]. When a patient has symptoms refractory to pharmacological treatment, then surgical unroofing or revascularization can be performed [10]. PCI carries the risk of potential complications including stent restenosis, fracture, or even coronary perforation, and so has only been performed in anecdotal cases [3,5]. Continuation of sports could be considered in asymptomatic individuals with no evidence of ischemia or complex cardiac arrhythmias during stress testing, while competitive exercise is contraindicated when persistent ischemia and/or ventricular arrhythmias are present [5].

## Conclusion

In conclusion, myocardial bridging is a common condition ranging from a benign asymptomatic finding to a potentially severe disease that may be the cause of myocardial ischemia, ischemic scar and/or arrhythmias. This case demonstrates the need for thorough evaluation of all individuals, even asymptomatic ones, in the presence of compelling indications such as an abnormal ECG. Although the absence of symptoms usually indicates a more benign course, patients with certain anatomical characteristics may have myocardial-bridge-related complications, including a myocardial infarction. Moreover,

physicians should be vigilant for coronary artery anomalies which could be the cause of an abnormal ECG during pre-sports participation screening and utilize multimodality imaging with CMR and CCTA in the diagnostic work-up of these patients.

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