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INTERMEDIATE

CASE REPORT: CLINICAL CASE

Myocardial Infarction by a Myocardial Bridge in the LAD Combined With Atrioventricular Re-Entrant Supraventricular Tachycardia

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ABSTRACT

We present the case of a patient who presented with palpitations and was found to have atrioventricular re-entrant tachycardia with unusually elevated cardiac biomarkers. A coronary computed tomographic angiography showed a myocardial left anterior descending artery bridge; an accessory pathway was ablated, and cardiac magnetic resonance revealed anteroseptal myocardial infarction resulting from hypoperfusion during tachycardia caused by the left anterior descending artery myocardial bridge. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2022;4:1115–1118)

HISTORY OF PRESENTATION

An 18-year-old patient without any past medical history presented to our emergency department with regular palpitations and light-headedness for approximately 1 hour. The patient had no comorbidities but had felt these palpitations several times before—each time directly after physical exercise but for only a short duration—and had never felt any chest pain during these episodes.

LEARNING OBJECTIVES

- To understand the significance and role of myocardial bridges as mostly benign anomalies that can lead to hypoperfusion in certain scenarios.
- To understand the importance of the collaboration and connection between different subspecialities of cardiology—interventional cardiology, rhythmology, and imaging—for the best diagnostic and therapeutic strategy for each patient.

INVESTIGATIONS

The emergency triage nurse felt a radial pulse of over 200 beats/min, but when the 12-lead electrocardiogram (ECG) was performed, the patient spontaneously converted to sinus rhythm. The first ECG showed normal sinus rhythm but signs of myocardial ischemia with a significant ST-segment elevation in aVR and significant ST-segment depression in leads II, III, aVF, and V_4 to V_6; the PR interval was normal, without evidence of pre-excitation (**Figure 1A**). The

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ABBREVIATIONS AND ACRONYMS

CCTA = coronary computed tomographic angiography

CMR = cardiac magnetic resonance

ECG = electrocardiogram

LAD = left anterior descending artery

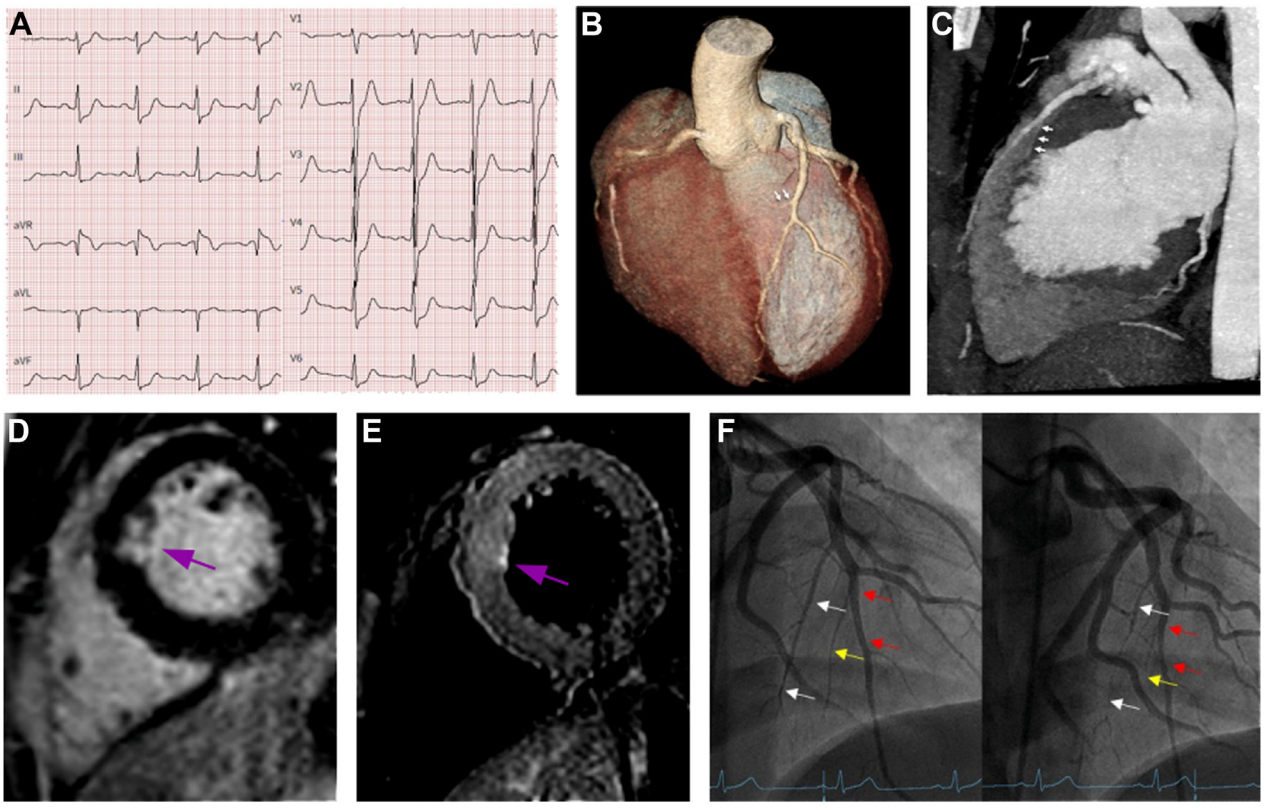
ECG changes resolved completely within 3 hours of observation. Laboratory testing showed elevated cardiac enzymes, with a peak high-sensitivity troponin T of 654 ng/L and creatine kinase of 1,323 U/L. Because the patient was young without any cardiovascular risk factors and did not feel any explicit chest pain, we performed coronary computed tomographic angiography (CCTA) to investigate the ECG changes and elevated cardiac enzymes. The CCTA showed no signs of coronary artery disease, dissection, or thrombus; however, it did reveal a complete myocardial bridge in the mid left anterior descending artery (LAD), which means that it is completely covered by myocardium (Figures 1B and 1C). The emergency transthoracic echocardiography showed a mild thickening of the basal interventricular septum of 13 mm, with normal biventricular and valve function. The cause of left ventricular thickening was unclear because the patient had no history of arterial hypertension and performed physical activity at the recreational level only.

MANAGEMENT

An electrophysiologic study was performed the following day. Wolff-Parkinson-White syndrome with a left laterally localized accessory pathway and only retrograde conduction was identified and treated with radiofrequency ablation. The induced atrioventricular re-entrant tachycardia during the electrophysiologic study had a heart rate of 220 beats/min.

Subsequently, we performed a cardiac magnetic resonance (CMR) scan to investigate the cause of the disproportionally elevated cardiac biomarkers. The

FIGURE 1 Initial Electrocardiogram in the Emergency Department After Conversion to Sinus Rhythm



(A) Coronary computed tomographic angiography showing extensive myocardial bridging of the left anterior descending artery and 2 septal branches **(white arrows)** supplying the anteroseptal segments in **(B)** 3D reconstruction and **(C)** maximum-intensity projection. Cardiac magnetic resonance revealed focal myocardial infarction **(purple arrow)** by visualizing **(D)** subendocardial late gadolinium enhancement (T1-weighted image acquired 10 minutes after gadolinium contrast agent administration) and **(E)** edema (T2-weighted spin echo image, **purple arrow**) in the anteroseptal basal and midventricular segments. **(F)** Coronary angiography in diastole **(left)** and systole **(right)** demonstrated clear "milking" of the first **(white arrow)** and second **(yellow arrow)** septal branches and the myocardial bridge of the LAD **(red arrow).**

examination revealed focal anteroseptal subendocardial late gadolinium enhancement with surrounding edema (**Figures 1D and 1E**), explaining the cause of localized mild left ventricular thickening shown in transthoracic echocardiography, indicating subacute myocardial infarction. Based on the result of the CMR, we proceeded to invasive coronary angiography with optical coherence tomography. We did not identify any kind of coronary stenosis or dissection on optical coherence tomography, but we could visualize the large myocardial bridge that stretched over 2 septal branches of the LAD, which correlated well with the region of myocardial infarction (**Figure 1F,** Video 1, Supplemental Figure 1).

In the synopsis, our multimodality imaging-based pathophysiologic explanation for the myocardial infarction in this young, healthy individual was a reduced filling and hypoperfusion caused by the combination of the large myocardial bridge of the mid LAD, including the septal branches, and the sustained extreme tachycardia during his atrioventricular reentrant tachycardia with concealed Wolff-Parkinson-White syndrome.

The patient was discharged with a treatment of aspirin 100 mg/day for a duration of 1 month after the left-sided ablation and the recommendation to perform only low-intensity physical exercise until the follow-up consultation. We decided against beta blocker therapy at discharge because we suspected that the patient would not physiologically reach a heart rate above 200 beats/min during his recreational exercise activity of football and jogging, which was confirmed by exercise testing later on.

DISCUSSION

Myocardial bridges are a frequent finding, with a mean prevalence of 25% reported in autopsy series and similar rates documented using CCTA.[1] They are mostly considered a benign anomaly and are typically located in the mid LAD.[1] The significance of myocardial bridges regarding myocardial ischemia has been discussed controversially for decades. They have been associated with stable angina, acute coronary syndromes, takotsubo cardiomyopathy,[2] and malignant arrhythmias. Several studies examined a possible “milking effect” with narrowing and reduced flow during systole caused by attributable to compression by thickening myocardium.[3] Some patients with a strong “milking effect” can also experience chest pain and repolarization changes on ECG during tachycardia.[3] Beta-blockers are considered as first-line therapy because they promote diastolic filling and decrease systolic compression of the artery and maximal heart rate. Patients experiencing from chest pain with proven ischemia (during physical, not pharmacologic, stress testing) despite optimal medical therapy can be treated with surgical myocardial unroofing or coronary bypass graft surgery in the case of long and deep symptomatic bridges.[4] Percutaneous coronary intervention of myocardial bridges is disputed because of a high rate of target lesion failure.

FOLLOW-UP

At the follow-up consultation 3 weeks later, the patient was doing well and had not experienced any chest pain or palpitations since discharge. We performed 2 exercise tests with different protocols to achieve the maximum heart rate. During the first exercise test, with a slowly progressing speed on a cycle ergometer, the patient achieved 253 W and a maximum heart rate of 184 beats/min. Subsequently, a second exercise test with rapidly progressing resistance and speed was performed. Here, the patient reached a maximum heart rate of 190 beats/min for only a few seconds. At the highest heart rate, we could see slight ST-segment elevation in aVR, which was only discreet and not comparable to the ECG changes from the initial ECG. The patient did not feel any chest pain during the exercise tests, and no rhythm disorders were noted. We cleared the patient for sports participation at any intensity because the maximum physiologically achievable heart rate was below the threshold that induces myocardial ischemia. We also decided against treatment with beta blockers and any invasive procedure.

CONCLUSIONS

Even though myocardial bridges are generally considered benign, it is important to identify clinical scenarios in which they have the potential to induce myocardial ischemia. The most important trigger is sustained tachycardia caused by arrhythmias or extensive exercise in competitive athletes. In addition, there are also some reports in the literature of myocardial ischemia caused by coronary spasm in combination with a myocardial bridge.[5] Multimodality imaging is essential for a comprehensive investigation of symptomatic patients with myocardial bridges. CCTA is a useful tool in the first assessment because it can provide information on the location and extent of myocardial bridges. In ambiguous cases, CMR can differentiate myocardial infarction caused by myocardial bridges from other pathologies that may coincide with tachycardia and

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elevated cardiac biomarkers such as perimyocarditis or arrhythmogenic cardiomyopathies. Eventually, coronary angiography is the reference method to visualize the systolic narrowing or "milking effect" of the coronary artery. An invasive investigation allows intravascular imaging and fractional flow reserve measurements for a detailed understanding of the coronary anatomy and the physiologic impact of myocardial bridges.

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KEY WORDS myocardial infarction, MR sequences, supraventricular arrhythmias

APPENDIX For a supplemental figure and video, please see the online version of this paper.