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Late Gadolinium Enhancement on Cardiac Magnetic Resonance Imaging in Acute Myocardial Infarction Due to Myocardial Bridging: A Clinical Dilemma

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Authors' contributions

This work was carried out in collaboration between all authors. Author MLZ performed data collection and wrote the initial manuscript draft. Author HRA, TCB, and AMS performed data collection, assisted with literature search, and assisted with manuscript revision. Author DMT collaborated with MLZ on the initial manuscript draft and wrote the subsequent revisions. All authors read and approved the final manuscript.

Case Study

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ABSTRACT

A 51-year-old male with a family history of premature coronary artery disease (CAD) presented with acute myocardial infarction (AMI) with coronary angiography demonstrating no angiographic disease and a mid-left anterior descending (LAD) myocardial bridging (MB) segment with 71% mean lumen diameter (MLD) compression. Due to continually rising biomarkers and recurrent angina, cardiac magnetic resonance imaging(CMR) was obtained demonstrating late gadolinium enhancement (LGE) involving the mid-distal LAD territory. Patient subsequently underwent successful percutaneous coronary intervention (PCI) with drug-eluting stent (DES) to the MB segment with resolution of symptoms, which persisted over a year. MBis defined as an intramuscular segment resulting in overlying bands of myocardium, also called "tunneled" artery. Once thought benign, MB has been reported to cause unstable angina, AMI, life-threatening arrhythmias, and sudden cardiac death. PCI has been reported to relieve symptoms balanced against rates of in-stent restenosis and target lesion revascularization as high as 19% with DES. This case illustrates the utility of CMR in the setting of AMI to guide decision to purse PCI in symptomatic MB.

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1. INTRODUCTION

Coronary artery disease (CAD) is the most common cause of death amongst adults leading to the use of rapid diagnostic protocols for the evaluation of potential acute coronary syndromes. Patients with ischemic EKG changes, chest pain, and/or elevated cardiac biomarkers are typically stratified for urgent invasive coronary angiography; however, when this modality fails to identify a culprit lesion in the setting of laboratory evidence of myonecrosis further work-up may be indicated. This case highlights the potential utility of cardiac magnetic resonance imaging (CMR) as an adjunctive diagnostic test in patients with acute coronary syndromes and non-obstructive coronary artery disease to determine extent and location of myonecrosis as a decision tool for revascularization in the setting of myocardial bridging (MB).

2. CASE PRESENTATION

A 51-year-old male with family history of premature CAD as only cardiac risk factor presented to a tertiary care center with complaints of recurrent exertional chest discomfort that radiated to his left jaw and shoulder and relieved with rest. His chest discomfort was associated with dyspnea on exertion upon climbing stairs as well as during his regular treadmill runs. On previous admission 2 weeks prior to presentation, he was discharged home after he underwent a treadmill stress test that was electrically and clinically negative. His physical exam was largely benign, with no pertinent cardiac, respiratory or abdominal findings. His initial work-up in the emergency department included serial electrocardiograms with non-specific ST abnormalities that were not dynamic, chest X-ray with normal findings, and a comprehensive metabolic panel with cardiac biomarkers, pro-BNP and D-Dimer that returned normal with the exception of an initial troponin of 0.16ng/mL (0-0.1ng/mL) and a potassium level of 3.2mmol/L (3.3-4.5mmol/L). Of note, urine toxicology screen was unrevealing. Patient was initially loaded on dual anti platelet therapy (aspirin 325mg and clopidogrel 600mg, orally), fond aparinux 2.5mg subcutaneously daily, lisinopril 10mg daily, metoprolol tartrate 25mg twice daily, and atorvastatin 80mg daily. In the setting of recurrent chest discomfort and elevated cardiac biomarkers, an invasive strategy was favored and patient underwent urgent invasive coronary angiography, which revealed no angiographic evidence of coronary atherosclerosis or vasospasm. A myocardial bridging (MB) segment in the mid left anterior descending (LAD) was seen with a 71% mean lumen diameter (MLD) systolic compression Fig. 1A. Transthoracic echocardiogram obtained following coronary angiography on hospital day 1 (HD1) was notable for mild concentric left ventricular hypertrophy (LVH) and a regional wall motion abnormality consistent with LAD territory. Patient subsequently underwent CMR on HD1, demonstrating similar wall motion abnormalities and late gadolinium enhancement (LGE) involving 50-75% myocardial thickness in the mid-distal anteroseptal and apical segments consistent with a LAD myocardial infarction Fig. 2A-C. Patient continued to have episodic anginal chest pain refractory to maximal tolerated medical therapy including the addition of amlodipine 5mg daily prompting referral for percutaneous coronary intervention (PCI) with placement of two overlapping everolimus-eluting stents with a total stented segment length of 39mm Fig. 1B. Since PCI, he had an isolated re-evaluation for recurrent chest pain evaluated with cardiac CT angiography (CCTA) demonstrating a widely patent LAD stent. The patient has otherwise been chest pain free and asymptomatic for over a year.

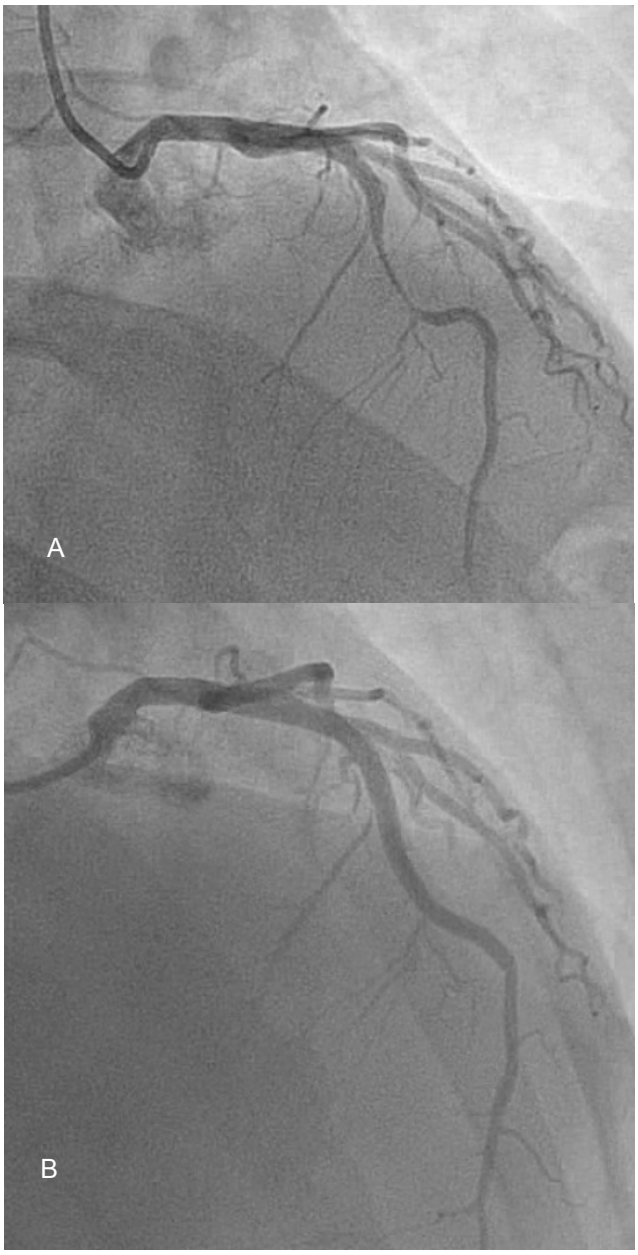

Fig. 1. Coronary angiography in the LAO cranial projection demonstrating >70% systolic compression of the mid-LAD MB segment (A) and in the same projection following PCI (B)

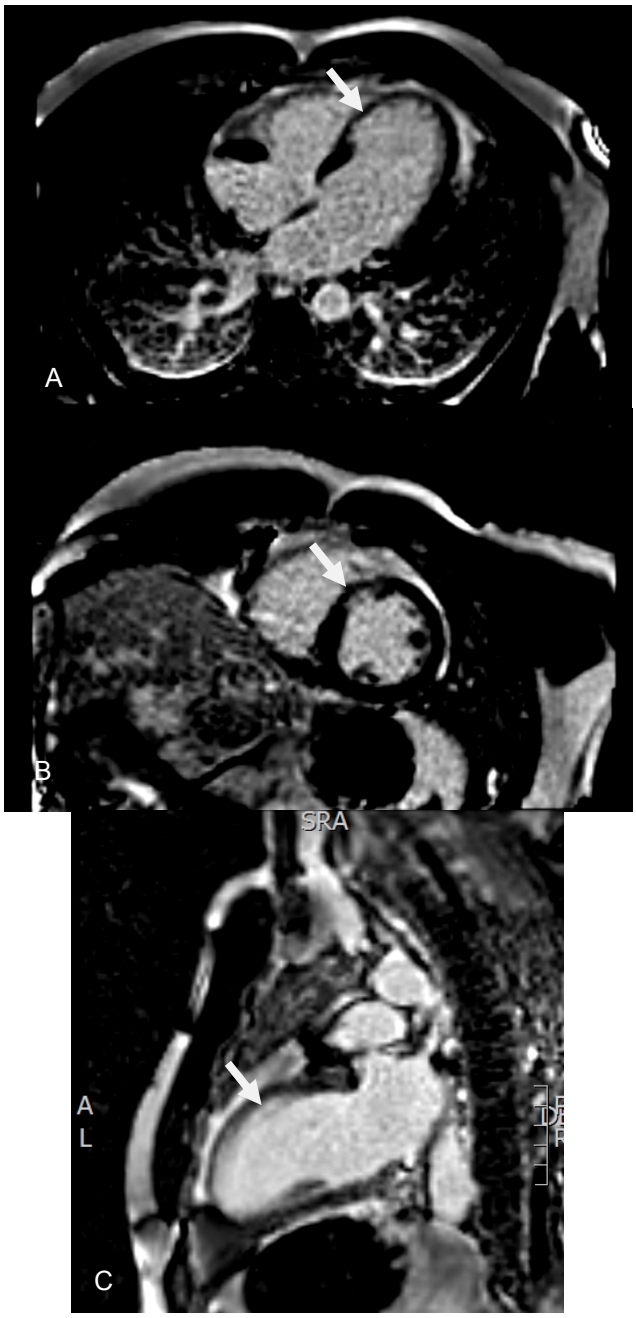

Fig. 2. Cardiac MRI demonstrating late-gadolinium enhancement and wall thinning in the mid to apical anteroseptal wall (white arrows) visualized in the 4-chamber (A), short axis (B), and 2-chamber (C) projections

3. DISCUSSION

MB refers to a major epicardial coronary artery, most commonly the left anterior descending, which contains an intramuscular segment resulting in overlying bands of myocardium which can cause various degrees of compression, also called “tunneled” artery. Systolic compression, or milking effect, was first described in 1976 in 0.5% of patients referred for coronary angiography [1]. The reported incidence of MB varies widely depending on the population studied, ranging from 15-85% [2-6] in autopsy studies, 0.5-2.5% [1,7-9] in invasive coronary angiography series, and 30.5% based on coronary CT angiography [10]. Additionally, newer cardiac imaging modalities such as CCTA and CMR have allowed for the diagnosis of co-existent structural abnormalities associated with MB segments, such as atrial diverticula, which were frequently very difficult to evaluate [11]. Early publications suggested a benign, predominantly asymptomatic course, however MB has more recently been implicated in clinical syndromes ranging from unstable angina, AMI, life-threatening ventricular arrhythmias, and sudden cardiac death [7-9,12]. Early suggestion of benign progress was based on fact that coronary flow occurs predominantly during diastole while MB segment compression occurs in systole. More recent data suggest that symptoms are more common with >50% MLD reduction and that severe MLD reduction (>70%) results in a persistent narrowing up to 40% into early-mid diastole. Additionally, flow velocities within the MB segment are more than twice as high as those found in segments both proximally and distally further impairing coronary flow reserve [13]. This effect is exaggerated in the setting of tachycardia as evidenced by prior studies using atrial pacing [14-15]. Anatomic factors that seem to increase the likelihood of symptoms include longer length, increased thickness, and more proximal location as well as the presence of LVH [16].

Initial management of symptomatic patients involves maximal tolerated doses of beta-blockers, calcium channel blockers, and antiplatelet agents with a goal to reduce chronotropy and contractility, and prevent platelet aggregation at sites of endothelial disruption. Nitrates may worsen symptoms by reducing distal coronary pressure, decreasing systemic arterial pressure and thus coronary perfusion pressure, and reflex tachycardia [17]. Prior to the advent of coronary stenting, surgical myomectomy was the only option for patients with refractory symptoms. The higher risk of peri-procedural complications and unpredictable intramural course can lead to extensive myocardial damage with associated risk for ventricular aneurysm formation [18-20]. This has resulted in PCI as a more attractive initial management strategy with internal mammary grafting reserved for patients who fail percutaneous treatment. Several small case series (up to 16 patients) have reported successful relief of refractory anginal symptoms in MB patients treated with PCI. The rates of in-stent restenosis (ISR) and target lesion revascularization (TLR) are as high as 36% with bare-metal stenting and 19% with drug-eluting stents [21-22].

4. CONCLUSION

This case highlights the utility of multiple imaging modalities to include CMR to determine the etiology of myonecrosis in patients with no angiographic evidence of coronary atherosclerosis and myocardial bridging. This is the first case illustrating the use of CMR to confirm MB as the causative lesion in the setting of AMI followed by successful PCI and long-term follow-up demonstrating good symptomatic relief.

CONSENT

Not applicable.

ETHICAL APPROVAL

All authors hereby declare that all experiments have been examined and approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki. This project has been cleared for publication by the department of clinical investigation, Brooke Army Medical Center.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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