

Myocardial bridging with left ventricular hypertrophy presenting as Wellens pattern

Ahmad Abuarqoub, Maria Naranjo, Fayez Shamoon

Department of Cardiology, Saint Joseph's Regional Medical Center, Paterson, NJ, USA

Correspondence to: Ahmad Abuarqoub. 90 Arcadia Road Apt B, Hackensack, NJ 07601, USA. Email: ahmada6@hotmail.com.

Abstract: The course of epicardial coronary arteries into a muscular tunnel under a bridge of myocardium is known as myocardial bridging (MB). This could be a benign anomaly, nevertheless, it could have a great impact on the quality of life in the setting of severe anginal symptoms. The clinical presentation and diagnosis could be challenging in those patients. The treatment options start from simple medical therapy to surgical intervention in refractory cases, the role of percutaneous coronary intervention (PCI) is limited in MB. We are describing a case of severe MB presenting as Wellens pattern with underlying left ventricular hypertrophy (LVH).

Keywords: Myocardial bridging (MB); hypertrophic cardiomyopathy (HCM); Wellens syndrome; sudden cardiac death (SCD)

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Introduction

The course of epicardial coronary arteries into a muscular tunnel under a bridge of myocardium is known as myocardial bridging (MB). This could be a benign anomaly, nevertheless, it could have a great impact on the quality of life in the setting of severe anginal symptoms. The clinical presentation and diagnosis could be challenging in those patients. The treatment options start from simple medical therapy to surgical intervention in refractory cases, the role of percutaneous coronary intervention (PCI) is limited in MB. We are describing a case of severe MB presenting as Wellens pattern with underlying left ventricular hypertrophy (LVH).

Case presentation

A 45-year-old Hispanic male presented to the emergency department for new onset of chest pain. The pain was described as an intermittent retrosternal chest heaviness, 7 out of 10 in severity, radiating to left arm, which started 20 minutes prior to arrival while the patient was watching TV, was relieved by nitroglycerine and rest, and was

associated with shortness of breath. There was no associated orthopnea, paroxysmal nocturnal dyspnoea (PND) or lower limb edema, palpitations, change in mental status, nausea, vomiting or back pain. The patient stated that he had a similar episode 3 days earlier when he was shoveling the snow but was associated with sweating and shortness of breath which resolved at rest. He has been treated for essential hypertension during the last 2 years with losartan 50 mg, hydrochlorothiazide (HCTZ) 12.5 mg, atenolol 100 mg and statins for dyslipidemia. He is a nonsmoker and denied any illicit drug use including cocaine. His grandfather sustained an acute myocardial infarction (MI) in his 50s. On clinical examination was unremarkable except for elevated blood pressure of 160/90 in both arms and body mass index (BMI) of 42. A chest X-ray was normal and 12 leads electrocardiogram (EKG) obtained shows deep asymmetric T wave inversion involving the anterolateral and inferior leads which had biphasic pattern on prior EKG (*Figures 1,2*). Cardiac marker first set were negative. Echocardiogram (*Figure 3*) showed no regional wall motion abnormalities, with preserved left ventricular (LV) ejection fraction in addition to moderate

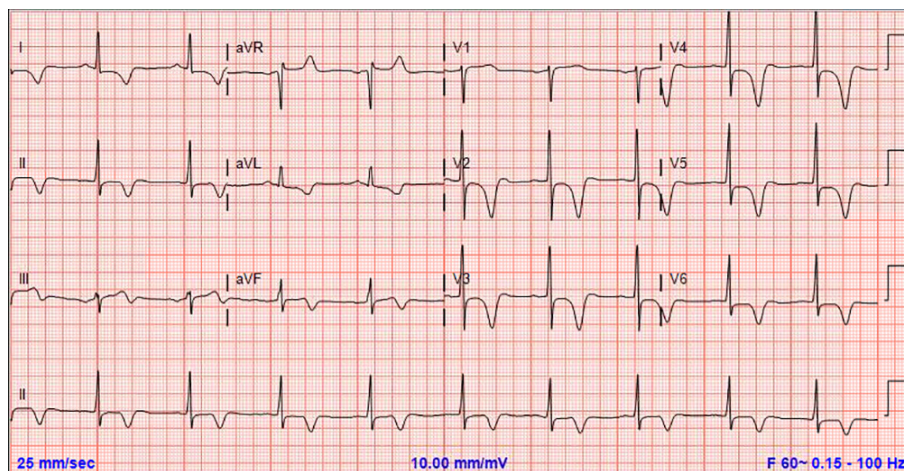


Figure 1 Electrocardiogram (EKG) showing deep asymmetric T wave inversion involving the anterolateral and inferior leads.

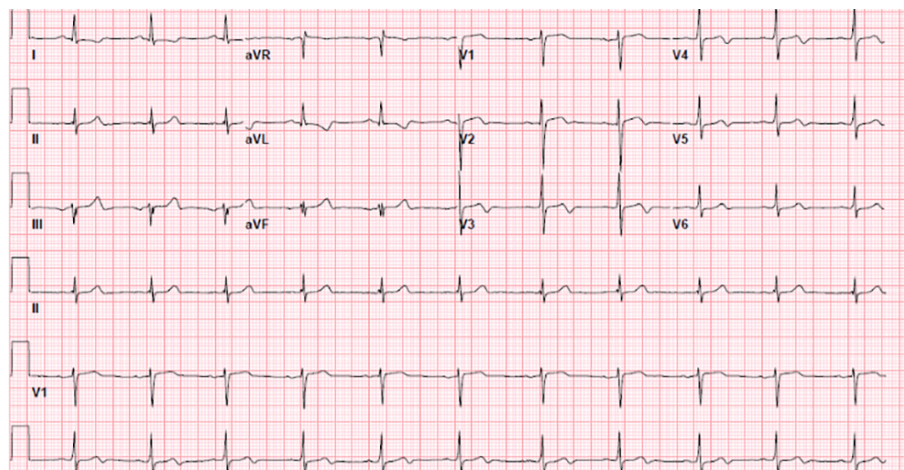


Figure 2 Old electrocardiogram (EKG) showing biphasic T waves on anterior leads.

LVH. In the setting of typical presenting symptoms and EKG changes concerning of Wellens syndrome, coronary angiogram was done which showed severe bridging of mid left anterior descending (LAD) in addition to ectasia of right coronary artery (RCA) and left circumflex (LCX) (Figures 4,5). The decision was made to proceed with medical treatment including aggressive blood pressure control and risk factor modification, in addition to beta blockers, avoidance of nitrates, and antiplatelet therapy. The patient was discharged from the hospital without complications, repeat EKG showed resolution of ST-T changes, the plan was for outpatient follow-up for possible

need of surgical intervention if symptoms are refractory.

Discussion

MB is a relatively frequent finding in postmortem ranging from 15% to 85% of cases (1). However, functional MB is less observed on angiography (0.5% to 2.5%) (2), although detection can increase up to 16% if provocation tests are used (3,4).

MB is defined as a segment of a coronary artery that takes a “tunneled” intramuscular course under a bridge of overlying myocardium, resulting in systolic compression

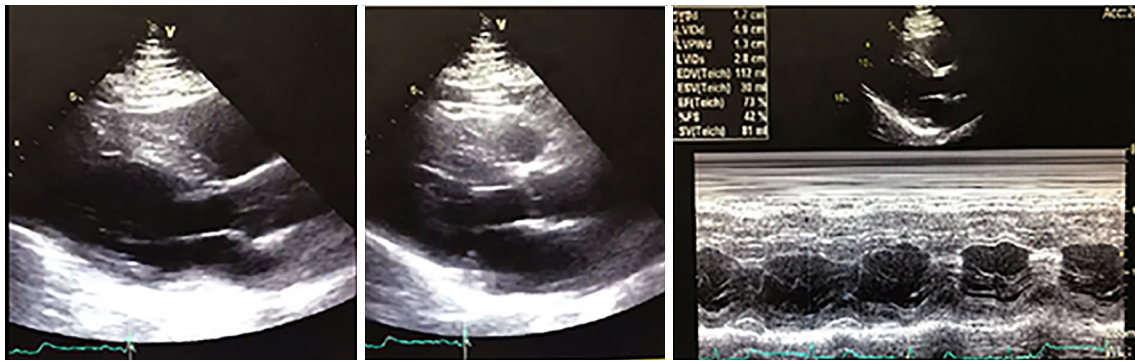


Figure 3 Parasternal long axis and M-Mode of transthoracic echocardiogram (TTE) showing preserved left ventricular (LV) function with moderate concentric left ventricular hypertrophy (LVH).

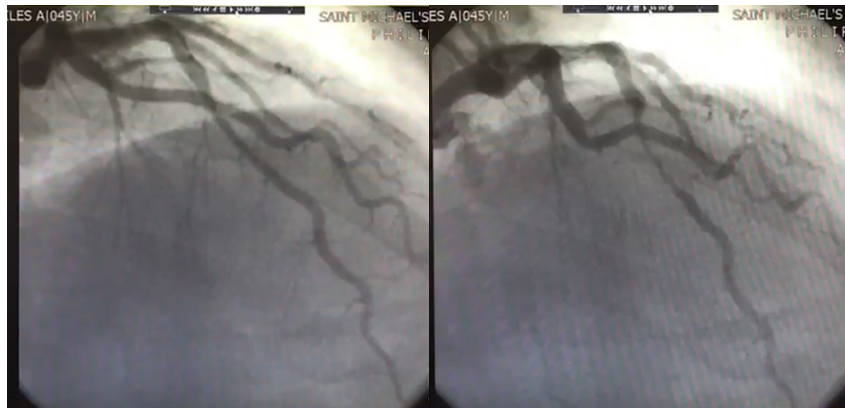


Figure 4 Right anterior oblique (RAO) cranial views of coronary angiogram after nitroglycerine injection showing severe bridging mid left anterior descending (LAD) during systole.

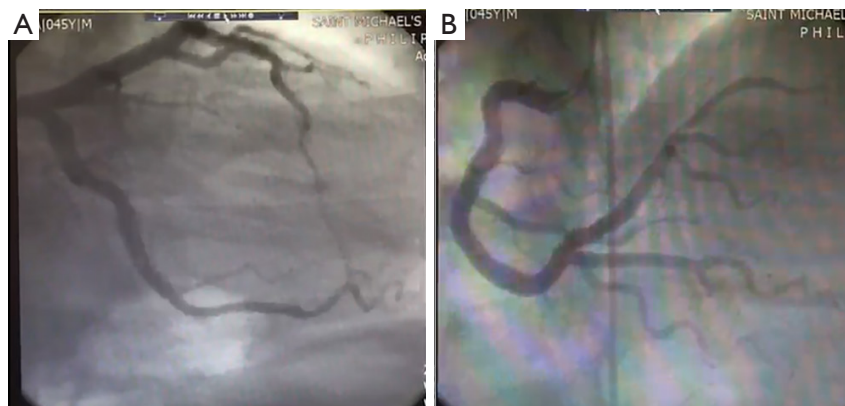


Figure 5 Right anterior oblique (RAO) caudal view. (A) Ectasia of proximal left circumflex (LCX) in addition to bridging of LAD; (B) ectasia of proximal to mid right coronary artery (RCA).

of a coronary artery. The large discrepancy in reported prevalence clearly maybe due to the lack of true gold standard test for the diagnosis of bridging.

Certain population, namely hypertrophic cardiomyopathy (HCM) patients has higher prevalence than general population with rates up to 30% (5). MB has been associated with increased risk of sudden cardiac death (SCD) in pediatric population with HCM. Although Sorajja *et al.* demonstrated not increased risk of cardiac death or SCD in adult patients with HCM (6).

The most frequent coronary artery involved is the LAD at the middle segment on coronary angiography. In other coronary computed tomography angiography (CTA) case series concluded that the RCA and LCX artery involvement at similar rates (7). In addition, secondary arteries such as diagonal (18%) and marginal (40%) branches are also commonly involved (8). There is difference in prevalence of bridging by gender which is higher in male (7,8).

MB presents with a variety of symptoms varying from angina, atypical chest pain, arrhythmia to sudden death, thus there is no consensus for the clinical diagnosis of this entity without visualization of the coronary arteries.

Myocardial blood flow (MBF) occurs predominantly during diastole while coronary compression of the myocardial bridge is at systole. However, clinical invasive and noninvasive diagnostic tools such as CTA, positron emission tomography (PET) intravascular ultrasound and intracoronary Doppler and percutaneous coronary angiography have contributed greatly to our understanding of the anatomic, hemodynamic, and pathophysiological consequences of systolic compression, which extends to mid/late diastole causing persistent diastolic luminal narrowing with increases both the blood flow velocities and retrograde flow, and a reduced flow reserve. Also, it has been postulated that accelerated atherosclerosis of the proximal segment of coronary artery before the bridged segment contribute to myocardial supply-demand mismatch in patients with MB (8-10).

Although Konen *et al.* conducted a prospective study with a total sample of 100 patients with intermediate pretest probability of coronary artery disease, and MB identified using CTA, with combination of PET and invasive coronary angiography to identify the systolic compression of coronary artery and measure atherosclerotic burden in coronary artery. Plaque morphology proximal to MB was comparable to other vessels. Therefore, according to this finding, MB is not associated with more advanced atherosclerotic

disease (11).

Medical management should be considered in patient like our patient presenting with angina and ischemia induced on stress test or demonstrated systolic vasoconstriction of the intramural coronary artery more than 70% or diastolic vasoconstriction of more than 35% on coronary angiography (12), beta-blockers still first-line therapy which will decrease the heart rate, that in turn increases the diastolic coronary filling period, and decreasing contractility, although there has not been any randomized clinical evaluating the long-term efficacy of beta blockers (13,14). Calcium channel blockers can be used as a second-line (15) or in combination with beta-blockers that may have vasodilator effect segments proximal to the tunneled segment. Nitrates are contraindicated; it has been shown to accentuate systolic compression of bridged segments (16).

Nevertheless, if medical treatment is unsuccessful stent implantation or surgery are therapeutic alternatives however, case reports on stent insertion reported raising the concerns of perforation during stent deployment (17-20), in-stent restenosis (20), stent fracture (21,22), aneurysm formation (22), and stent thrombosis (18) thus avoiding there use in MB is preferable. On the other hand, surgical options for MB include surgical myotomy and coronary artery bypass graft surgery with anastomosis of the left internal mammary artery to the LAD artery can be contemplated for refractory cases. Further research is required to better define the patient population that would derive the greatest benefit from surgical and percutaneous intervention (23,24).

Conclusions

The clinical presentation of MB is variable, thus an appropriate diagnostic test should be chosen with coronary angiography being the gold standard. The cornerstone of treatment of MB is medical with beta blockers and calcium channel blockers being the regimen of choice. The role of PCI is very limited in MB due to the adverse outcome reported in literature and the surgery is reserved for cases refractory to medical therapy, MB is more commonly described in HCM patients and presentation as Wellens pattern is rare in medical literature.

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None.

Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

Informed Consent: Written informed consent was obtained from the patient for publication of this manuscript and any accompanying images.

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