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Case Report

Myocardial bridging manifested as acute coronary syndrome: an unusual presentation of the so-called benign coronary anatomy variant

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ABSTRACT

Myocardial bridging is an anatomical variant characterized by a coronary artery that tunneled through the myocardium. Often asymptomatic, myocardial bridging could also go to a further extent, presenting as myocardial ischemia in the form of stable angina pectoris, acute coronary syndrome, arrhythmias, or even sudden cardiac death. Although these are uncommon manifestations, it is still a challenge for physicians when encountering such cases. Additional imaging techniques are often required to diagnose myocardial bridging. This report is about a unique case of a 72-year-old man presenting with acute coronary syndrome (ACS) in the form of unstable angina pectoris (UAP), which was later found to be caused by myocardial bridging on coronary angiography. Following administration of beta-blocker, antiplatelet, and statin, the angina symptoms, as well as ischemic sign on electrocardiography (ECG), were completely resolved.

Keywords: Acute coronary syndrome, Coronary angiography, Myocardial bridge, Myocardial bridging, Unstable angina pectoris

INTRODUCTION

Myocardial bridging (MB) is a congenital anatomical variation of coronary artery that tunneled through the myocardium. Myocardial bridges can be found in any epicardial artery, but mostly (67-98%) occur in left anterior descending (LAD) coronary artery, primarily in the middle segment.[1]

MB is often incidentally found in symptomatic patients presenting with angina, suggesting myocardial ischemia. MB was divided into superficial and deep muscle types. Deep muscle types could compress the coronary artery, reduce the flow, and inducing myocardial ischemia.[2]

MB patients might also present with more complicated cardiac events including acute coronary syndrome, coronary vasospasm, exercise-induced dysrhythmias, ventricular arrhythmias, or even sudden cardiac death.[3] The degree of hemodynamic impact depends on the thickness and length of the bridge, orientation of the bridge relative to myocardial fibers, and presence of loose connective tissue or adipose tissue around the bridged segment.[1]

MB mostly diagnosed on MSCT or coronary angiography, with milking effect as a pathognomonic sign showing coronary compression by myocardium on systole and release of coronary artery during diastole.[1,2] Medical treatment with beta blockers and non-dihydropiridine calcium channel blockers remain the frontline therapy of symptomatic MB.[1,4] Surgical myotomy, PCI, and CABG are reserved for patient refractory to medical therapy. Risk factors modification and antiplatelet should be considered as MB are at increased risk of developing atherosclerosis.[4]

CASE REPORT

A 72-year-old man came to emergency department, complaining of chest pain 4 hours prior to admission. It

occurred in the morning after his morning prayer ritual. The chest pain was described as heaviness that felt from the center of his chest through his back and lasted for more than 10 minutes. The pain was first relieved by rest, but it appeared again after some activities.

He has already felt this kind of chest pain in quite some time, probably in the past months. He already visited cardiology policlinic 5 days before with occasional chest pain and anterior ischemic on ECG. Echocardiography suggested normal left ventricular ejection fraction (LVEF), and he was advised to take either MSCT or coronary angiography for further examination. He was prescribed ASA 80 mg od, atorvastatin 20 mg od, nitroglycerin 2.5 mg od, and ISDN 5 mg only when the chest pain arose. He opted for coronary angiography, but that day, his chest pain worsened before his scheduled date for elective coronary angiography. History of another comorbidity was denied. He is a non-smoker.

On examination, he was compos mentis, afebrile, blood pressure (BP) 194/105 mmHg, heart rate (HR) 115 bpm, normal respiratory rate, and SpO_2 99% on room air. Other physical examinations were unremarkable. ECG revealed a sinus rhythm with rate 95 bpm and atypical changes in anterior leads (Figure 1). High-sensitivity troponin did not increase, with value 12.2 pg/ml (referral range: 0-14 pg/ml). Other laboratory examinations were unremarkable. Chest radiography revealed cardiomegaly (Figure 2).

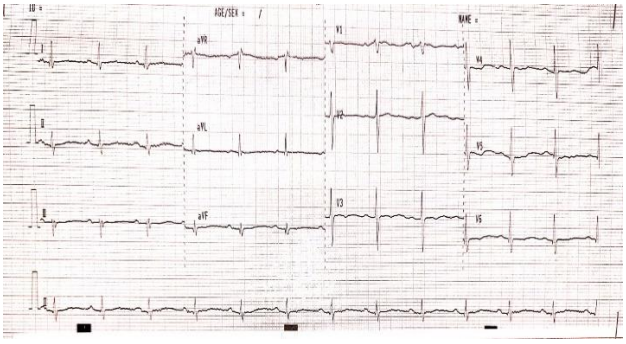

Figure 1: ECG at presentation: ischemic in anterior leads.

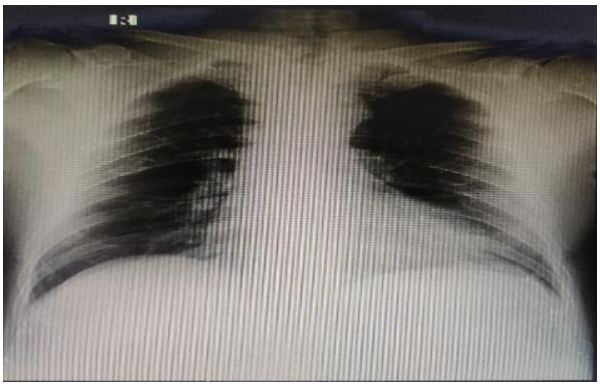

Figure 2: Chest radiography.

He was assessed with unstable angina pectoris. A loading dose of ticagrelor 180 mg and ASA 320 mg was given, continued with maintenance dose of ticagrelor 90 mg od and ASA 80 mg od.

Enoxaparin was given at 1 mg/kg every 12 hours. Nitrogylcerin was increased to 5 mg t.i.d. Administration of atorvastatin 20 mg od was continued, in addition to bisoprolol 2.5 mg od. He was planned for a coronary angiography the day after.

Coronary angiography revealed 30% narrowing of middle left anterior descending (LAD) during systole period and an increase vessel lumen diameter during diastole, suggesting a milking phenomenon caused by myocardial bridging (Figure 3). The other epicardial coronary branch was normal.

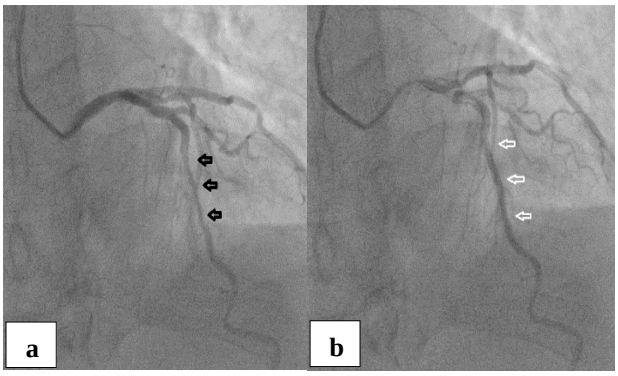

Figure 3: (a) Narrowing mid-LAD during systole and (b) increased mid-LAD diameter during diastole.

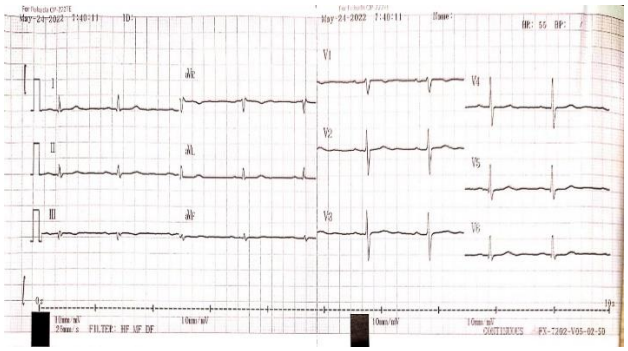

Figure 4: ECG two days after MB diagnosis.

Ticagrelor and enoxaparin were discontinued after diagnosis of myocardial bridging. Optimal medical treatment and lifestyle was advised, with clopidogrel 75 mg, ASA 100 mg od, atorvastatin 20 mg od, and bisoprolol 2.5 mg od. Nitroglycerin was stopped. He got better with symptoms and his BP returned to normal throughout the hospital stay. The atypical ischemia sign on ECG was resolved (Figure 4). The patient was also advised to regularly measure his own blood pressure outside monthly hospital blood pressure monitoring. He was discharged on the fourth day of hospitalization.

DISCUSSION

Generally, MB is often considered a benign condition, probably because most coronary bridge constrictions only occur during the systolic period, while coronary flow returns during diastole.[1] Many studies suggested a potential hemodynamic significance of MB, and some implied a possible correlation between MB and various cardiovascular pathologies including myocardial ischemia, acute coronary syndrome, ventricular wall rupture, life-threatening arrhythmias, HCM, and apical ballooning syndrome or sudden death.[5] In this case, we will discuss the findings of myocardial bridging in the patient with ACS symptoms in the form of unstable angina pectoris.

There are two main pathological findings in autopsy and intravascular ultrasound (IVUS) in myocardial bridging, which are the absence of atherosclerosis at the intramural and distal segments of the MB, and the presence of developing atherosclerosis at the proximal segment of the MB, in which related biomechanical forces may account for these findings.[6] At the site of the myocardial bridge opening, fluid mechanics could influence and stimulate plaque formation of atherosclerosis, with disturbed blood flow patterns near the wall. Low and oscillatory wall shear stress (WSS) are associated with increased vascular cell adhesion molecule-1 expression, production of reactive oxygen species (ROS), and development of endothelial cells that is pro-atherogenic.[1,2] Therefore, coronary segments that are located immediately proximal to myocardial bridges, where the WSS is low, portray some structurally dysfunctional endothelial cells. On the contrary, structurally intact endothelial linings are found in the bridged segments where the WSS is high and physiologic.[1,4] For further reference, in the study of coronary artery in patients with mild atherosclerosis without myocardial bridging, higher plaque progression was also observed in the segments where WSS is low.[1]

Diagnosis

In asymptomatic patients, angina and symptoms of myocardial ischemia could be triggered by several mechanisms. Patients with LV diastolic dysfunction caused by aging, hypertrophy, and atherosclerosis could worsen the imbalance between supply and demand imposed by the bridge.[7] The presence of hypertrophy itself also results in compression and reducing coronary microvascular reserve. On the other hand, the process of prolonged vasoconstriction in MB during the systole period leads to endothelial damage and thus stimulates aggregation of platelet, along with spasm mechanism, inducing myocardial ischemia.[2] The development of plaque in the proximal segments of the bridge and endothelial remodeling within the bridge could aggravate the degree of coronary obstruction and reduce myocardial flow.[1] Each of these factors contributes to a different degree of symptoms development in MB patients.

Systolic coronary narrowing in myocardial bridging is further divided into 3 classes: class 1 with <50% narrowing, class 2 with 50-75% narrowing, and class 3 with >75% narrowing.[2] Blood flow reserve will significantly decrease if the constriction of the vessel is >50% or even >70%. This condition is often responsible for adverse complications in many patients, from angina on exertion, resting angina, or in more advanced cases to acute myocardial infarction and sudden death.[8]

In the presented case, the patient may indicate coronary artery disease in his first visit with ischemia findings on ECG but later came up with acute coronary syndrome although there is no further ischemic change compared to previous ECG. The patient was first assessed as unstable angina pectoris. Management of ACS in this patient was based on following presentation and established guideline by ESC with ASA and ticagrelor loading dose, along with nitroglycerin, enoxaparin, and coronary angiography.[10]

Anatomically, MB is mostly found in the middle segment of the LAD coronary artery. In the other case, it can also be found in the diagonal and marginal branches with a percentage of 18% and 40% of cases, respectively.[1] MB can occur in a single vessel or multiple vessels and can be found in the same or different branches. Based on the depth, MB is divided into superficial or deep muscle types. Usually, there is no disruption of coronary artery flow in the superficial type, while the deep type will experience suppression by the myocardium, causing coronary flow reduction and inducing myocardial ischemia.[4]

The mechanism of myocardial ischemia in MB patients involves two hypotheses. First, myocardial ischemia is caused by dynamic compression of myocardial fibers forming the tunnel. Second, it is related to atherosclerosis formed in the proximal segments of the MB. The extent and duration of compressed segments are clinically important because the compression may induce exertional chest pain, which may mimic the ACS manifestation.[3]

Several modalities are available for assessing MB, whether non-invasive or invasive modalities. The non-invasive including multi-slice computed tomography (MSCT). The invasive includes coronary angiography (CAG), intravascular ultrasound (IVUS), and fractional flow reserve (FFR).[1,2,4]

MSCT has some advantages for the evaluation of MB since it is a non-invasive imaging modality compared to coronary angiography. It allows the assessment of the coronary artery walls, lumen, and surrounding myocardium, along with the length and depth of the bridge, as well as the location of atherosclerosis associated with MB and identification of coronary vulnerable plaques.[1] Several clinical features are associated with plaque vulnerability including a positive sign for remodelling, low attenuation, and the presence of spotty calcification.[10]

Coronary angiography is the gold standard and the most common modality for diagnosing MB.[8] The typical finding is the presence of systolic narrowing or the so-called "milking effect" of the epicardial artery, described as "step-down" and "step-up" that delimits the affected area.[4] The presence of myocardial bridging in angiography depends on the diameter change between systole and diastole in the bridged coronary segment. The milking effect will significantly appear when there is >70% narrowing in minimal luminaire diameter (MLD) during systole and persistent >35% reduction in MLD during mid to late diastole.[1]

IVUS, when readily available, would be helpful when the angiography could not be performed or if the result remains uncertain. The typical finding of IVUS is the half-moon sign, an echo-lucent area that exists only between the MB segment and the epicardial tissue that persists throughout the heart cycle.[4,6] Intracoronary Doppler will show a fingertip phenomenon that is distinctive for MB, characterized by rapid diastolic blood flow followed by a plateau in the bridging segment.[11]

FFR is often used to assess the presence of physiological organic stenosis, using pressure wire and intravenous injection of adenosine or dobutamine. Patient with evidence of MB with FFR <0.75 is likely to have MB-related myocardial ischemia.[6]

Treatment

First-line pharmacological treatment in symptomatic MB is beta-blockers and calcium channel blockers.[12] The negative chronotropic and inotropic effects of beta-blockers may reduce heart rate and contractility and decrease the frequency of systolic and early diastolic compression, thereby increasing the diastolic filling time.[13] Administration of non-dihydropyridine calcium channel blockers might be useful in patients with concomitant vasospasm.[14] In patients with clinical evidence of atherosclerosis, the use of antiplatelet should be considered. Nitrate administration should be avoided in patients with MB as it may increase the narrowing of systolic compression and dilating segments proximal to the bridge, causing exacerbation of retrograde flow, reducing the myocardial ischemic threshold, and worsening symptoms.[15]

For patients with severe symptoms who are refractory to conservative medical treatment, other treatment options such as coronary stents, supra-arterial myotomy/surgical myotomy, and CABG may be considered.

PCI might be considered in patients who experience refractory symptoms with optimal medical therapy. Significant stent restenosis can occur in a small number of patients. Besides, there are many other concerns for MB patients such as stent fractures, thrombosis, and an increased risk of perforation during deployment.[16]

Coronary artery bypass grafting (CABG) and surgical myotomy are the two most common surgical approaches. CABG is mostly beneficial in myocardial bridges >25 mm length or >5 mm depth to avoid the risk of graft failure or when the bridged coronary segment fails to decompress completely in diastole.[7] Surgical myotomy is considered for short and superficial MB. Myotomy surgery involves the resection of muscle fibers that compress the MB and has been shown to improve coronary blood flow and relieve symptoms.[14] On the other hand, myotomy can cause several complications including wall perforation, the formation of ventricular aneurysms, and postoperative bleeding.[7]

In this presented case, treatment with oral medication has resulted in a good outcome, with diminished symptoms and complete ECG resolution, and no cardiac event throughout the hospital stay. MB generally has a good long-term prognosis, depending on the coronary compression degree. Patients could have occasional angina symptoms but mostly do well with medical therapy, especially with beta-blockers.[17] Percutaneous and surgical interventions should be reserved only for patients with severe symptoms who cannot be treated satisfactorily with oral medication.[18]

CONCLUSION

Myocardial bridging is an anatomical variant of the coronary artery that tunneled through the myocardium. Myocardial compression during the systolic period to the bridged segments may cause myocardial ischemia and the formation of atherosclerosis proximal to the bridge. Oral medication with beta-blockers, calcium-channel blockers, antiplatelet, and avoidance of nitrates remain the frontline therapy. PCI, CABG, or surgical myotomy are reserved for cases refractory to medical therapy. Although considered to be benign, myocardial bridging might also present with more advanced cases such as acute coronary syndrome, arrhythmia, or even sudden cardiac death, all owing to ischemia or even infarction. Physicians should always consider this mechanism of supply-demand mismatch caused by myocardial bridging when managing chronic and acute coronary syndrome.

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