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The Impact of Myocardial Bridging on Coronary Artery Spasm in Female Patient without Significant Atherosclerotic Stenosis: A Case Report

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Abstract: ***Background: Chest pain due to ACS is frequently a diagnostic concern of emergency physicians. However, this symptom can also present due to compression of the coronary artery by myocardial bridging (MB). MB is a frequent but often forgotten cause of angina. It has been accepted that MB might affect the cardiovascular system and sudden cardiac death have been reported. Case Illustration: A 43 year old woman with no significant past medical history, presented to the emergency departement with a sudden achy radiating pain in her left chest. No physical abnormalities were detected by clinical examination. Laboratory workup revealed high values for total cholesterol & cardiac troponin I. We did serial ECG and it exhibited any acute ischemic changes less than 20 minutes. We established the diagnosis of NSTEMI & the patient was managed conservatively. Following her presentation, ECG findings compatible with deeply inverted T waves in anterior lead. Therefore, we suggested patient to do coronary angiography & it showed dynamic obstruction of the LAD due to MB. Discussion: MB may cause chronic angina pectoris, ACS or coronary spasm. The mechanism behind these symptoms lies in the disturbance of blood flow through the tunneled artery. Our patient described sudden angina and her ECG showed a 2 to 3 mm ST elevation in anterior lead which returned to baseline quickly. This transient ST elevation is the key for the diagnosis coronary artery spasm (CAS). Coronary spasm frequently occur at MB segments because of endothelial or vascular dysfunction of the coronary artery at MB segments. Medication (beta-blockers & calcium channel blockers) is considered as first-line therapy and should be initiated only for symptomatic patients. Conclusions: MB is often forgotten cause of angina but can be present in up to 25% of the population. CAS frequently occur at MB segments. This condition coupled with any delay in management can lead to ischemia, infarction and sudden cardiac death.***

Keywords: myocardial bridging, coronary artery spasm, angina

1. Introduction

Chest pain is the most common principal complaint in patients presenting to the emergency departement. The presence of chest pain due to acute coronary syndrome (ACS) is frequently a diagnostic concern of emergency physicians. Symptoms of ACS are commonly caused by a ruptured or progressively enlarging atherosclerotic plaque. However, these symptoms can also present due to compression of the coronary artery by myocardial bridging (MB). Myocardial bridging is a frequent but often forgotten cause of angina and can be present in up to 25% of the population.

Myocardial bridging is characterised by a segment of a major epicardial coronary artery tunnelling through the myocardium, which is a frequent congenital anomaly, detected in 0,5 – 33% of angiographic studies.[1,2] Symptomatic patients are most often middle-aged men with typical or atypical chest pain, either related or unrelated to exercise. Myocardial bridging usually has a benign prognosis, but some cases associated with sudden death.[3]

However, the underlying pathogenesis of acute myocardial infarction caused by MB is not fully understood. Myocardial bridging without critical stenosis is generally considered as a harmless clinical anomaly in coronary angiography, but in some cases like myocardial ischemia, infarction, vasospasm, cardiac arrhythmias, ventricular rupture, and sudden cardiac death have been reported.[1,2]It is possible that coronary spasm frequently occur at MB segments because of endothelial dysfunction and/or vascular dysfunction of the coronary artery at MB segments. It has been accepted that MB might affect the cardiovascular system.[2,4] We report a case of angina pectoris due to MB related to coronary artery spasm, which was confirmed in coronary angiography.

2. Case Presentation

A 43 year old woman with no significant past medical history, presented to the emergency departement with a sudden achy radiating pain in her left chest. It occurred suddenly and lasted approximately 30 minutes before arrived in hospital. She had associated left hand pain and tingling sensation with nausea but did not experience diaphoresis, dyspnea, lightheadedness, or headaches. She had no history of hypertension, diabetes, dyslipidemia, smoking and had no family history of coronary artery disease. Her initial vital signs were blood pressure 140/100 mm Hg, respiratory rate 20 breaths per minute, heart rate 80 beats per minute, temperature 36˚C, and peripheral oxygen saturation 100% in ambiental air. Her body mass index was 24,2 kg/m^2. No physical abnormalities were detected by clinical examination. Lung sounds were normal with no audible cardiac murmurs.

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) IgM antibody serologies were negative uppon arrival at the emergency departement. Laboratory workup revealed high white blood cells (16.3x10^3/uL), high values

for total cholesterol (231 mg/dL), LDL-cholesterol (151 mg/dL), and tryglicerides (192 mg/dL), high cardiac troponin I (6,91 ng/mL) and high uric acid (6,79 mg/dL). Routine kidney function bloos tests, electrolit, fasting glucose and HBA1c were within normal range (serum creatinine 0.54 mg/dL, blood urea nitrogen 7.57 mg/dL, potassium 3.94 mmol/l, sodium 143 mmol/l, fasting glucose 103 mg/dL, HBA1c 5,4%).

We did serial electrocardiogram (ECG) and ECG exhibit any acute ischemic changes less than 20 minutes. The first resting ECG revealed sinus rhythm 79 bpm with ST elevation in procordial leads V1 to V6 (Figure 1). Second ECG showed sinus rhythm 85 bpm with inverted T waves in precordial leads V1 to V5 (Figure 2). Chest radiography showed clear lungs without pleural effussions or consolidations. The heart was normal in shape and size, no pericardial effusion or pulmonary vascular congestion were noted. A Transthoracal Echocardiography (TTE) demonstrated a preserved left ventricular function with an ejection fraction of 57%, mild mitral regurgitation, without any regional wall motion abnormality.

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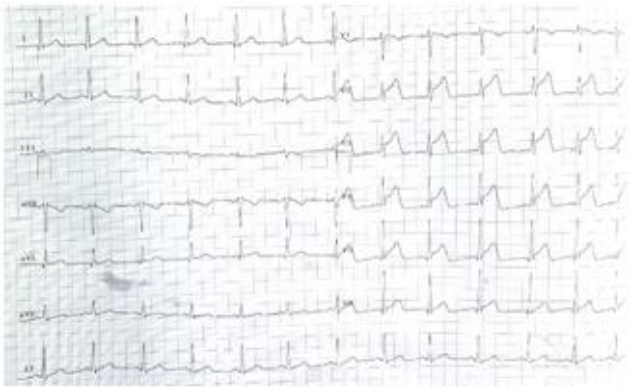

Figure 1: First ECG showed sinus rhythm and ST segmen elevation in lead V1 to V6

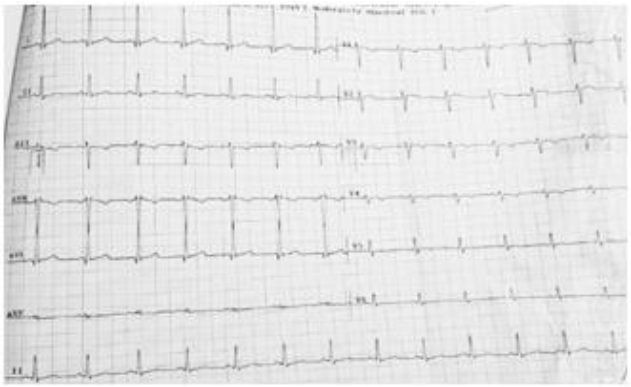

Figure 2: Second ECG showed sinus rhythm and inverted T waves in lead V1 to V5

We established the diagnosis of Non ST-segmen elevation acute coronary syndrome and the patient was admitted to intensive care unit. Following her presentation, the patient was immediately started on aspilet 160 mg loading dose followed by 80 mg OD orally, clopidogrel 300 mg loading dose followed by 75 mg OD orally, atorvastatin 40 mg OD orally, bisoprolol 2,5 mg OD orally, diazepam 5 mg OD orally, lactulosa 15 ml TID orally, low molecular weigh heparin (LMWH) 0,6 cc BID subcutaneous, and isosorbide dinitrate (ISDN) drip 2 mg per hour. The patients was managed conservatively. On follow up day 1, her ECG showed sinus rhythm 71 bpm and inverted T waves in lead V1 to V5 (Figure 3). The next day, T wave was more deeply in lead V1 to V6 and also showed in lead II, III, aVF (Figure 4). Her hospital course was uneventful and she was discharged on pharmacological therapy (aspilet, clopidogrel, atorvastatin, bisoprolol, ramipril, ISDN, and trimetazidine) with well controlled symptoms on follow up.

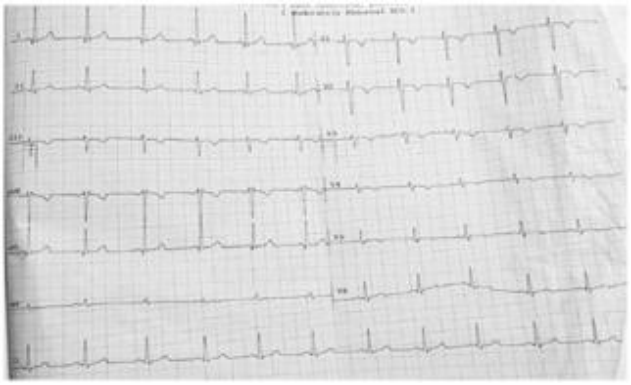

Figure 3: Follow up day 1, ECG showed sinus rhythm and inverted T wavesin lead V1 to V5

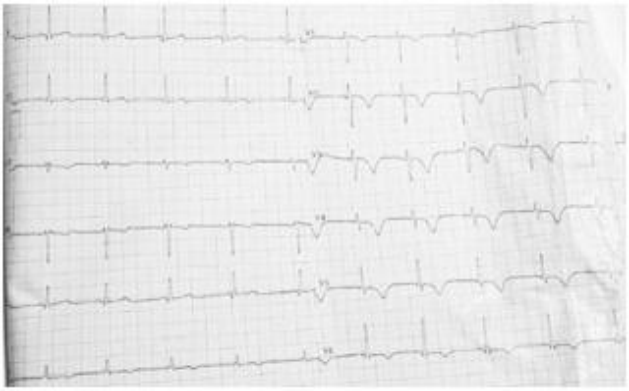

Figure 4: Follow up day 2, ECG showed sinus rhythm, inverted T waves more deeply in lead V1 to V6 and also in lead II, III, aVF

Keeping in mind that our patient presented ECG findings compatible with ischemic changes (deeply inverted T waves) in anterior lead (V1 to V6), which is highly spesific for critical Left anterior descending (LAD) artery stenosis. Therefore, we suggested patient to do coronary angiography. The patient underwent cardiac catheterization after discharged from hospital. Coronary angiography showed non significant lession at LAD and dynamic obstruction of the LAD due to myocardial bridging (MB) (Figure 5). The right coronary artery, left main coronary artery, and left cicumflex coronary artery were normal. The patient received extensive counseling with respect to her diagnosis and opted for non-invasive medical management.

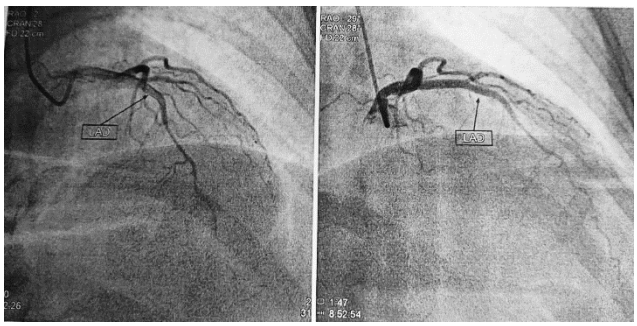

Figure 5: Coronary angiography showed myocardial bridging at LAD

3. Discussion

Myocardial bridging is a congenital anomaly described as tunneling of a coronary artery through the myocardium rather than running a typical epicardial course. In MB, a coronary artery that runs typically on the epicardium presents a transient intramyocardial course and is exposed to externally mechanical compression during ventricular systole. Its prevalence ranges from 0.5% to 29.4% when detected by angiographic procedures.[5,6]

Myocardial bridging mostly asymptomatic, it may cause chronic angina pectoris, acute coronary syndrome, coronary spasm, ventricular septal rupture, arrhythmias, exercise-induced atrioventricular conduction blocks, transient ventricular dysfunction, and sudden death.[3,6,7] The mechanism behind these symptoms lies in the disturbance of blood flow through the tunneled artery. There is enhanced myocardial compression in which the vessel enters into the myocardium, leading to a disturbance of blood flow to the rest of the myocardium. This disturbance mainly occurs during systole and is resolved in the diastolic phase. This transient hypoperfusion is though to be the cause of presenting symptoms, myocardial ischemia, infarction, and sudden cardiac death.[5,6,7]

Our patient described anginawhich occurred suddenly. In emergency unit, her ECG showed a 2 to 3 mm ST elevation in lead V1 to V6 which returned to baseline quickly. Severe chest pain, usually without physical effort and with a concurrent ECG showing transient ST elevation, is the key for the diagnosis coronary artery spasm (CAS).[8] It is possible that coronary spasm frequently occur at MB segments because of endothelial dysfunction and/or vascular dysfunction of the coronary artery at MB segments.[2]There are multiple proposed effects of MB on endothelial function. It has been proposed that the milking of MB segments causes increased shear stress. Increased shear stress associated with MB appears to reduce the production of vasoactive agents such as endothelial nitric oxide synthase, endothelin-1 and angiotensin-converting enzyme within the bridging segment. Consequently, the increased shear stress and high intravascular pressure in MB appears to negatively affect endothelial function and significantly affects endothelium-dependent vasorelaxation. These effects could potentially predispose patients with MB to coronary vasospasm.[9]

Myocardial bridging most commonly involves the left anterior descending artery, as demonstrated in our patient, but there have been a few case reports of involvement of the right coronary artery. Resting ECG rarely show any abnormalities, therefore other modalities are needed to identify myocardial bridging. Coronary angiography remains the criterion standard of diagnosing the presence of myocardial bridging. Angiography reveals that patients exhibiting more than 75% narrowing of the LAD from MB compression during cardiac systole experience severe myocardial ischemia.[1,10] The systolic compression of the tunneled artery is portrayed as a milking effect on coronary angiography, demonstrated as retrograde blood flow and subsequent antegrade flow with with expansion of the vessel diameter during diastole. Noninvasive studies such as

cardiac CT, cardiac MRI, and transthoracic echocardiography can also be used to diagnose myocardial bridging, but these are not the modalities of choice.[5,7,11]

Medication is considered first-line therapy and should be initiated only for symptomatic patients to improve their quality of life. It is reported that beta-blockers and calcium channel blockers showed favourable outcomes on most symptoms. Beta blocker therapy is the first choice of treatment for symptomatic patients. These drugs reduce the degree of systolic coronary artery narrowing and lengthen the diastolic via their negative inotropic and chronotropic properties. The negative inotropic effect of beta blockers increases coronary perfusion and alleviates symptoms evoked by the MB. Calcium channel blockers are effective in the treatment of MB for their good negative inotropic and chronotropic effects and the reduction in vascular resistance with an improvement in blood flow. The administration of diltiazem was the first choice pharmacological treatment. Calcium channel blockers with diltiazem plus nicorandil was used to treat the coronary spasm, which was the main pathology in the present case. The use of nitrates which is very effective for relieving coronary spasm, may exacerbate the systolic narrowing of the MB segments. They intensify systolic compression of the tunneled segment, leading to retrograde flow and thus worsening symptoms. Intracoronary injection of dobutamine, epinephrine, and isoprotenerol was also shown to exacerbate vessel narrowing during systole and delay diastolic relaxation.[1,2,5,12,13] Aggressive risk factor modification is recommended because of the inherent risk of the MB including atherosclerosis. Antiplatelet and statin therapy should be considered when subclinical atherosclerosis is detected.[14]

If symptoms persist despite medical therapy, coronary artery angioplasty with stent implantation, coronary artery bypass grafting (CABG) and surgical myotomy of the bridge can be considered. It was reported that percutaneous coronary intervention is useful to relieve chest symptoms in patients with MB, however it was also reported that incidence restenosis is relatively high. Therefore, pharmalogical treatment should be used in patients with MB.[1,2,5]

As is known already, clinical diagnosis of MB is impossible. Therefore, young individuals consulted to the hospital with chest pain and similar cardiac complaints should be examined in detail and more carefully as for the presence of MB. Severe MB was associated with high incidence of coronary artery spasm and MB patient with coronary artery spasm were likely to have higher incidence of recurrent angina. Intensive medical therapy and close clinical follow up are needed for a better clinical outcomes in MB with coronary artery spasm.

4. Conclusions

Myocardial bridging is characterised by a segment of a major epicardial coronary artery tunnelling through the myocardium which is a frequent congenital anomaly. This case often forgotten cause of angina but can be present in up to 25% of the population. Patients can present with a variety of symptoms including angina. Coronary artery spasm frequently occur at MB segments because of endothelial dysfunction and/or vascular dysfunction. This condition coupled with any delay in management can lead to ischemia, infarction and sudden cardiac death. Therefore, it is imperative that patients for whom there is low clinical suspicion for atherosclerosis but who are presenting with angina equivalents should undergo coronary angiography to assess for MB and receive immediate treatment.

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