Double myocardial bridge causing acute myocardial infarction in a premenopausal woman

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Abstract
Myocardial bridging is a congenital abnormality which is characterized by an intramuscular course of a segment of the coronary artery. The presence of myocardial bridging influences coronary vasculature due to mechanical compression and systolic milking. The symptoms and complications of myocardial bridging depend on the number and depth of the tunneled artery segments, the grade of systolic narrowing and the heart rate, and include myocardial ischemia, coronary spasm or acute coronary syndrome. Dual myocardial bridges are rare. We present a case of an acute myocardial infarction in a premenopausal woman due to double myocardial bridging which was identified on coronary angiography. Myocardial perfusion single photon emission tomography (SPECT) was performed for the evaluation of infarct size and residual ischemia.

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Introduction

Myocardial bridging is a congenital coronary artery abnormality in which the vessel, instead of running a normal epicardial course, tunnels intramuscularly [1, 2]. In autopsy studies, the frequency of myocardial bridging ranges between 5.4% and 80%, while on cardiac computed tomography (cCT) and coronary angiography (CA), which is considered the gold-standard method for the diagnosis of myocardial bridging, the frequency ranges between 25% to 30.2% and 0.5% to 16.0%, respectively [1, 2]. Left anterior descending artery (LAD) is the vessel where myocardial bridging is located in the majority of cases (67% to 98%), although cases of left circumflex and right coronary artery location have also been reported [2-5].

Myocardial bridging does not affect significantly the myocardium in most cases, since blood flow in coronary arteries occurs mainly during the diastolic phase. However, artery compression during the systolic phase, which is depicted as "milking effect" on CA, may result in hemodynamic changes and complications, causing myocardial ischemia, angina, acute coronary syndrome, left-ventricular dysfunction, myocardial infarction and sudden cardiac deaths in several cases [6]. Hemodynamic changes and the consequent symptoms and complications depend on the grade of systolic artery compression and the number and depth of the tunneled artery segments, as well as on the heart rate [7].

Double myocardial bridging has been rarely reported in the literature [8, 9]. We present a case of an acute myocardial infarction in a premenopausal woman due to double myocardial bridging and in the absence of other risk factors.

Case presentation

A 44-year-oldpremenopausal woman (body weight 65kg and height 1.72m) was admitted to the hospital due to chest pain with back reflection, sweating and tendency to vomit for the last 1-2 hours. She had no risk factors or history of coronary artery disease (CAD). Electrocardiogram (ECG) showed ST elevation of the anterior wall and transthoracic 2D echocardiography revealed hypokinesia in the septal and apical walls and ejection fraction (EF)=45%-50%. She was initially treated with antiplatelets, anticoagu-

alant and antianginal drugs, followed by thrombolysis with tenecteplase due to persistent chest pain and ST elevation. Moreover, on the day following admission, she was transferred to our hospital for coronary angiography, which revealed two myocardial bridges in the course of left anterior descending artery, located at the distal and the mid segment of the vessel, causing partial systolic suppression-milking effect, as well as mild atheromatosis at the same site (Figure 1A). No stenoses were detected in the remaining vessels. The patient's condition remained stable, and she was discharged from the hospital four days later with the suggested medication which included clopidogrel-acetylsalicylic acid, ivabradine, ramipril, nisoldipine, atorvastatin and pantoprazole. Three months later, the patientunderwent a technetium-99m (^{99m}Tc)-tetrofosmin myocardial gated single photone-mission computed tomography (SPECT) for the assessment of any residual myocardial ischemia and evaluation of necrosis. She was submitted to a Bruce exercise stress test, without discontinuation of treatment (duration of test 7:54 min: sec, METS:10.00, 87% of the maximum predicted value achieved). She didn't exhibitany clinical or ECG signs of ischemia. Gated SPECT showed a small hypokinetic transmural myocardial perfusion defect at the apex and limited periinfarction ischemia in the apical segments of anteroseptal wall and septum, as well (Figure1B).

Discussion

Myocardial bridging refers to a segmental intramyocardial course of a coronary artery instead of epicardial surface running and is an abnormality of congenital origin. Usually, myocardial bridging is a benign situation without significant symptoms when no severe systolic compression occurs.

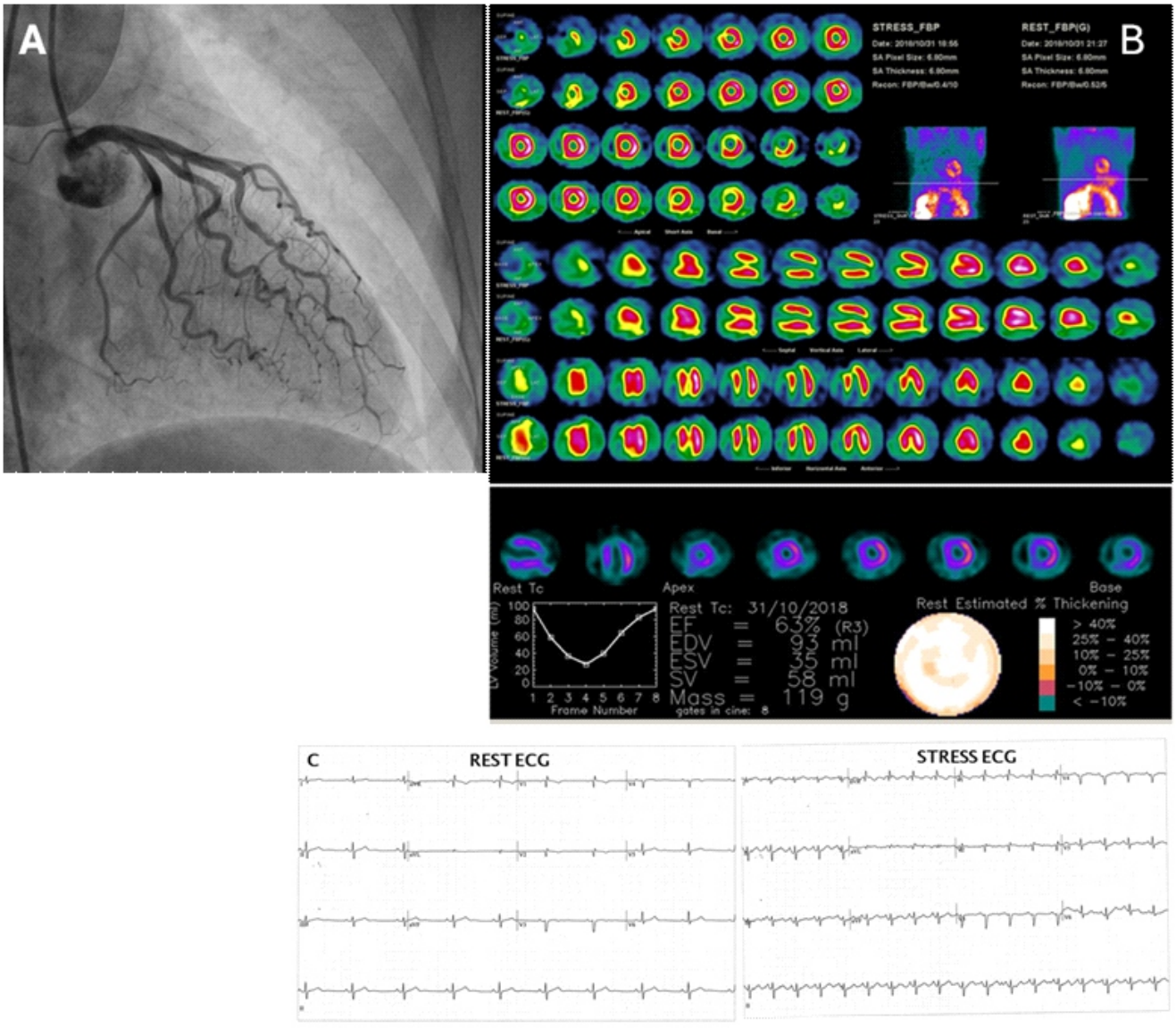

Figure 1. A). Coronary artery angiography showing two myocardial bridges at the mid and the distal segment of left anterior descending artery, causing partial systolic suppression ("milking effect"), as well as mild atheromatosis at the same sites. B). Gated SPECT myocardial perfusion stress-rest imaging with ^{99m}Tc-tetrofosmin showing a small hypokinetic transmural myocardial perfusion defect at the apex and limited periinfarction ischemia in the apical segments of anteroseptal wall and septum. C). Electrocardiogram at rest and during the stress phase of SPECT myocardial perfusion study.

The modality of choice for the diagnosis of myocardial bridging and the evaluation of "milking effect" is CA. In cases of reduction of minimal luminal diameter over 70% during the systolic phase and persistence of a greater than 35% reduction during the mid to late diastolic phase, the "milking effect" may be significant and cause various hemodynamic changes and cardiac events, even in the absence of coronary artery stenoses [6, 7, 10].

Hemodynamic changes are attributed to mechanical compression of the coronary artery due to "milking effect" during the systolic phase which results in myocardial ischemia. The coexistence of atherosclerosis at the proximal part of myocardial bridging may enhance the hemodynamic changes. Moreover, the systolic "milking effect" causes disturbance of blood flow and may lead itself to the development of atherosclerosis at the entrance of myocardial bridging, while the intramuscular and distant segments are spared [11,12].

The formation of thrombus is another complication of myocardial bridging, partly due to atherosclerotic plaque rupture at the proximal segment induced by the systolic vascular compression. Thrombi formation results in the development of acute coronary syndromes. Additionally, myocardial bridging and turbulent flow may cause endothelial vascular dysfunction, abnormal vasoreactivity and coronary spasm [4].

Hemodynamic changes also depend on the depth and the number of tunneled arteries, the length of myocardial bridge, the orientation of myocardial bridge in relation to the myocardial fibers, the age of the patient and the presence of other risk factors, as well as the presence of myocardial hypertrophy and the heart rate [3, 5].

Double myocardial bridging is rare. To our knowledge, limited cases have been reported in the literature [8, 9]. We consider that in our case, the presence of dual myocardial bridges was the cause of coronary spasm and acute myocardial infarction, in the absence of other cardiovascular risk factors.

In conclusion, myocardial bridging, although is generally a benign congenital coronary artery abnormality, however in several cases may cause severe and life-threatening cardiac events. Double myocardial bridging is rare and may be the main reason for acute myocardial infarction even in premenopausal women, in the absence of other risk factors.

The authors declare that they have no conflicts of interest

Bibliography

1. Corban MT, Hung OY, Eshtehardi P et al. Myocardial bridging: contemporary understanding of pathophysiology with implications for diagnostic and therapeutic strategies. *J Am Coll Cardiol* 2014; 63(22): 2346-55.
2. Lee MS, Chen CH. Myocardial Bridging: An Up-to-Date Review. *J Invasive Cardiol* 2015; 27: 521-8.
3. Yukio I, Yoko K, Ehiichi K, Toshiharu I. (1) coronary events caused by myocardial bridge. *Ann Vasc Dis* 2009; 2(2): 79-94.
4. Ishii T, Ishikawa Y, Akasaka Y. Myocardial bridge as a structure of "double-edged sword" for the coronary artery. *Ann Vasc Dis* 2014; 7(2): 99-108.
5. Ishikawa Y, Akasaka Y, Suzuki K et al. Anatomic properties of myocardial bridge predisposing to myocardial infarction. *Circulation* 2009; 120(5): 376-83.
6. Teragawa H, Oshita C, Ueda T. The Myocardial Bridge: Potential Influences on the Coronary Artery Vasculature. *Clin Med Insights Cardio* 2019; 13: 1179546819846493.
7. Hostiuc S, Rusu MC, Hostiuc M et al. Cardiovascular consequences of myocardial bridging: A meta-analysis and meta-regression. *Sci Rep* 2017; 7(1): 14644.
8. Izgi A, Tanalp AC, SağlamM et al. Dual myocardial bridges successfully treated with intracoronary stent placement. *Anadolu Kardiyol Derg* 2006; 6(2): 205.
9. Ibarrola M. Multiple myocardial bridges associated with left-ventricular dysfunction, intermittent left bundle branch block, and cardiac memory: A case report. *Ann Noninvasive Electrocardiol* 2018; 24(2): e12594.
10. Duymun S, Misodi E. Myocardial Bridging: A Case Presentation of Atypical Chest Pain Syndrome in a Young Woman. *Am J Case Rep* 2020; 21: 1-5.
11. Torii S, Virmani R, Finn A. Myocardial Bridge and the Progression of Atherosclerotic Plaque in the Proximal Segment. *Arterioscler Thromb Vasc Biol* 2018; 38(6): 1250-1.
12. Akishima-Fukasawa Y, Ishikawa Y, Mikami T et al. Settlement of Stenotic Site and Enhancement of Risk Factor Load for Atherosclerosis in Left Anterior Descending Coronary Artery by Myocardial Bridge. *Arterioscler Thromb Vasc Biol* 2018; 38(6): 1407-14.