Acute Myocardial Infarction Mimicking Takotsubo Cardiomyopathy in A Patient with Myocardial Bridging - A Case Report and Literature Review

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Abstract

It was reported that 1-5% of acute ST segment elevation myocardial infarction (ASTEMI) had no concrete atherosclerotic plaque and thrombus in relevant coronary artery. Takotsubo cardiomyopathy (TTC) and coronary spasm are two main causes of this situation. TTC have possessed these features: chest pain induced by emotional stress; prominent ST segment elevation in extensive precordial leads, but disproportionately mild elevation of myocardial marker in peripheral blood; no concrete occlusion or stenosis lesion in coronary arteries. Furthermore TTC is usually spontaneously reversible, and heart function returns to normal after a few weeks, therefore absence of irreversible myocardial damage is considered as one of criteria to the diagnosis of TTC. The pathophysiology of TTC is not well understood [1,2], and TTC may be due to multi-vessel coronary artery spasm, coronary microvascular dysfunction, myocarditis, catecholamine toxicity or even an apical-basal gradient in β(2) adrenergic receptors. Myocardial bridging (MB) of coronary artery can also cause myocardial injury. Furthermore, MB was proposed as a potential substrate in the pathogenesis of TTC by some cases [3,4]. Here we reported acute myocardial infarction mimicking TTC in the patient with intermittent exacerbation of MB in coronary angiography simultaneously, suggesting that the MB might be associated with the acute ST segment elevation myocardial infarction but the case is similar to the features of TTC, strongly supporting the association between TTC and MB.

Keywords: Takotsubo cardiomyopathy, Myocardial bridging, Acute myocardial infarction

Introduction

The patient was admitted in August 2010 for evaluating her recurrent chest discomfort. She was an active smoker, and had no history of hypertension or diabetes mellitus. Her 12-lead Electrocardiogram (EKG) was normal (Figure 1A), serum levels of Creatine Kinase Isoenzyme MB (CK-MB) and Cardiac Troponin I (cTnI) were within normal ranges. Coronary angiography (CAG) showed absence of significant lesion on all her coronary arteries (Figure 2A). Her medication included aspirin, and statin in the subsequent follow-up. Her chest discomfort still recurred, but with less frequency. The patient was re-admitted in May 2012 on account of severe and persistent chest pain and dyspnea for 5 hours after emotional stress. 12-lead EKG on this presentation showed sinus tachycardia with prominent ST segment elevation in leads I, avL,V 1-6 (Figure 1B). CK-MB and cTnI were mildly increased at 5th hour from the onset of her symptom. Her cTnI level peaked at 0.59 ng/mL in the second hospitalization. CAG was performed at 6th hour from the onset. All the coronary arteries were patent without any concrete stenosis or occlusion, but long and robust MB involved in almost total middle part of LAD (Figure 2B). This attack of myocardial infarction was thought to be caused by the MB. Echocardiogram (UCG) at the 3rd day of hospitalization showed that the left ventricle ejection fraction (LVEF) of 0.37 and her left ventricle was enlarged with an extensive hypocontractile area around the apex, but a hypercontractile base, which suggested the diagnosis of TTC. EKG on 6th hospital day showed that ST segment elevations were partially resolved and T waves were inverted in leads V1-6, I, avL. In addition, pathological Q waves were located in leads V1-3, and I, avL (Figure 1C). Her medication included aspirin, metoprolol, benazepril, and fluvastatin in the follow-up. On the 12th month of the follow-up, EKG and UCG were performed again. Pathological Q
Figure 1: (A) EKG during the first hospitalization in 2010; (B) EKG at 6th hour after onset during the second hospitalization in 2012; (C) EKG on 6th hospital day during the second hospitalization in 2012; (D) EKG in the subsequent 3-year follow-up; (E) EKG performed in 2016.

Figure 2: (A) 1st CAG performed in 2010 showed normal LAD with little wrinkle in the mid-LAD in systolic phase; (B) 2nd CAG performed in 2012 showed severe compression of the mid-LAD in systolic phase by a long MB.
waves in leads I, avL, V2-3 and thinning and bulging out of apical wall of left ventricle still existed (Figure 1D and Figure 3A).

She was re-admitted for evaluating her chest discomfort on exertion in April 2015. During the third hospitalization, EKG had showed that rs waves in leads I, avL, V3, QS waves in leads V2 and T waves were inverted in leads V2-S. Furthermore, her LVEF had rised to 0.4, and thinning and bulging out of apical wall of left ventricle still existed in UCG (Figure 1D). The 3rd CAG (the image of CAG was not available due to treat in other hospital) showed she had a mild lesion in the proximal of LAD and Left Circumflex Artery (LCX). But, the MB involved in almost total middle part of LAD could not be detected again. Her medication included aspirin, benazepril, metoprolol and fluvastatin in the subsequent follow-up. On the 10th month of the follow-up, EKG and UCG were performed again. Pathological Q waves in leads I, avL, V2-3 and T waves were inverted in leads V1-5. Her LVEF increased to 0.53, and thinning and bulging out of apical wall of left ventricle still existed in UCG. But compared to three years, the akinetic area was shrunk and located to the apex (Figure 1E and Figure 3B).

Discussion

Acute coronary syndrome (ACS) including ASTEMI with normal CAG is not a rare clinical situation [5]. Lesions of coronary arteries per se including aborted thrombosis or coronary spasm must be suspected in this situation. We did not perform intravascular ultrasound (IVUS) for this patient, but the vessel wall of LAD was smooth, and the vessel lumen was well perfused without any haziness. Furthermore in patients with aborted thrombosis in coronary artery, ST-segment elevation will be often resolved after the culprit artery is opened. But in this case, the ST-segment elevation of precordial leads still evolved, when the LAD was proved to be patent by CAG. So we believed that aborted coronary thrombus or spasm should be a less possible cause for her.

Long and potent MB on LAD is unique positive finding of CAG in this case. And MB is the most common coronary artery variant. The current gold standard for diagnosis of MB by CAG is the typical “milking effect”, which compromises the sensitivity of MB finding by CAG. MB is generally considered as a benign entity without significant effect on coronary flow [6, 7]. However, there are emerging evidences that certain MB characteristics may be associated with cardiovascular morbidity. It was reported that MB in the mid LAD is associated with myocardial dyssynchrony and those with more severe bridging tend to have an increased incidence of dyssynchrony [8]. Cases of acute myocardial infarction caused by MB were also reported previously [9-13]. Such as, Rujic et al. [14] reported nitroglycerine induced AMI in a patient with MB. In our case, the intermittent exacerbation of MB can be found which might be attributed to the changes of stimulating factor, such as emotional factor induced tachycardia (compromising diastolic filling of coronary arteries), increase the myocardial contractility, resulting into ASTEMI with no concrete atherosclerotic plaque and thrombus.

More interestingly, the clinical course of this particular case also possessed the features of TTC. Though cardiac function is better than before, her apical ballooning did not completely disappear even in the following four year. Similar to our case, Lee, et al [15] reported a case of persistent TTC with apical mural thrombus. In their case QRS complexes in leads V1-3 presented with QS type at admission, and evolved to rs waves at 3months later, the follow-up ECG showed persistent akinesia of the LV apex with slightly improved contractility of the mid-ventricular wall segment. Naruse, et al. [16] reported 3 patients with TTC who had remained Late Gadolinium Enhancement (LGE) in the chronic phase, suggesting small and focal myocardial abnormalities. In addition, TTC has been identified in 1-2% of admissions for acute myocardial infarction [17,18], but its mechanism is still unclear. Delgado, et al. [19] performed CAG and intravascular ultrasound (IVUS) evaluation in 11 patients with TTC, and demonstrated that atherosclerotic coronary lesions in the LAD causing an aborted myocardial infarction could not be the primary underlying cause of TTC.

To begin with the first case about TTC and MB, several examples also were discussed in this aspect up to now. Such as Migliore, et al. [4] and Peters, et al. [20] reported an underling MB in the LAD could represent a pathophysiological substrate of the TTC. But, the underlying mechanism is still obscure. Increased adrenergic activity is accepted as the main mechanism of TTC, which may lead to tachycardia, decreased diastoles, and increased myocardial contractility, which exacerbating the effects of MB on LAD, thus exclusively result in a apical injury. In addition, combined action between cardiac insufficiency and left ventricular outflow tract obstruction can increase the Left Ventricular End-Diastolic Pressure (LVEDP) which can result in the significant reducing of coronary perfusion pressure and myocardial damage.
But in this case, the old MI features in EKG and UCG did not completely disappear even in the following four years, which could also be attributed to damage caused by the severe and extensive MB. The effect of MB to coronary flow is changeable, myocardial contractility is believed to be one of important determents, as well as the length and depth of intramural artery [21]. Coronary flow normally occurs just 15% during systolic phase [22], whereas mainly during diastolic phase. As the systolic compression of MB on intramural coronary artery is relieved in diastolic phase, the vessel will restore coronary flow obviously. It would lead to persistent myocardium stunning so that taking shapes the ASTEMI if the systolic compression of MB was sustaining. In addition, A recent study shown that the anatomical properties of MB muscle, for example thickness, length and location, were associated with a shift of coronary intimal lesion more proximally, an effect that may increase the risk of ASTEMI [23]. Due to the retrograde blood flow toward proximal LAD by the MB compression force, the atherosclerotic lesions can be made in the LAD intima proximal to MB. Hence, the presence of the MB is regard as getting command of the distribution of atherosclerotic lesions with the affected LAD [24,25]. More interestingly, Derkacz, et al and Bonnemeier, et al. [26,27] reported ASTEMI, lesions with the affected LAD [24,25]. More interestingly, Derkacz, et al and Bonnemeier, et al. [26,27] reported ASTEMI, resulting from the patients with MB, had disproportionately a mild elevation of myocardial marker (CTnI) in peripheral blood as same as the properties of TTC. Therefore, our case supports the notion that acute myocardial infarction mimicking Takotsubo cardiomyopathy in a patient with myocardial bridging.

Conclusively, we observed acute myocardial infarction mimicking TTC in the patient with intermittent exacerbation of MB in coronary angiography simultaneously, suggesting that the MB might be associated with the acute ST segment elevation myocardial infarction. In addition, this case also suggests a possible association between TTC and MB. Whether MB could be an important pathogenesis or just an aggravating factor for TTC needs further investigation.

References


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