ATP stress MCE in evaluating coronary microvascular spasm: a case report

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Abstract

We report a case of coronary microvascular spasm assessed by ATP stress MCE (myocardial contrast electrocardiography). The patient had chest pain, but the coronary angiography was normal. There was apical ventricular septal perfusion delay before ATP stress, and the perfusion was significantly improved at peak stress, which was similar to the radionuclide myocardial perfusion characteristics of coronary microvascular spasm, In the recovery period, the flow spectrum resistance of the distal coronary artery of the left anterior descending artery increased compared with that before stress, which further confirmed that local coronary microvascular spasm was induced after vasodilation.

Introduction

There are many reasons for chest pain and tightness. In addition to coronary stenosis, coronary spasm and microcirculation lesions are also common causes of chest pain [1-2]. Because there is no "gold standard" for the detection of myocardial microcirculation lesions, especially the diagnosis of myocardial microvascular spasm is relatively difficult, some patients are often missed and misdiagnosed. We report a case of "reverse redistribution" of ultrasound ATP stress MCE (myocardial contrast electrocardiography) and increased spectral resistance of distal coronary blood flow induced during ATP stress recovery. It is considered to diagnose "coronary microvascular spasm".

Case introduction

A 35-year-old male patient had no obvious inducement for recurrent precordial pain 4 months ago. The pain lasted for tens of seconds and could be relieved by himself. Times of pains increased one week ago. His blood pressure was 120 / 77 mmHg and his heart rate was 71 beats / min. His physical examination did not hear heart murmur and friction sound, and no obvious abnormal signs were found. No obvious history of heart disease, no history of hypertension, diabetes, long smoking history for about 10 years, 15 cigarettes / days, drinking for 7 years, 100g/ days. Laboratory examination showed that ECG showed that T wave of v4-v6 lead was inverted(Supplementary Figure 1). CK-MB, troponin level, brain natriuretic peptide level and echocardiography showed no obvious abnormalities. Coronary angiography: the wall of the middle segment of the left anterior descending artery and the distal segment of the circumflex artery was irregular, and no obvious stenosis was found(Supplementary Figure 2). With the consent of the patient and the approval of the hospital ethics committee, ATP stress MCE was used to detect the microvascular function of the patient.

ATP 140ug / min / kg was injected through forearm cephalic vein for six minutes. The ventricular wall motion, MCE and distal blood flow spectrum of anterior descending branch were observed before ATP stress and 2-3 minutes after stress, and the ventricular wall motion and distal blood flow spectrum of anterior descending branch were observed during ATP stress recovery.

MCE showed that the myocardial perfusion in the apical/mid ventricular ventricular septum and anterior wall had a delayed perfusion defect before ATP stress (After "flash" microbubbles for 5 cardiac cycles) (*Figure. 1A;* Supplementary Figure 3A;Video1); At ATP peak stress, MCE showed a significant improvement in the same area (within "flash" microbubbles for two cardiac cycles) (*Figure. 1B;* ***Supplementary Figure 3B;Video2***); The flow reserve decreased slightly, CFR = Mean diastolic coronary flow velocity before ATP stress / Mean diastolic coronary flow velocity at ATP peak stress, which was 2.5. Compared with the recovery period, the resistance of coronary flow spectrum in diastolic period increased significantly, showing a "thumb" spectrum (*Figure. 2A and B;*Supplementary Figure 4). The velocity integral of blood flow spectrum changed from 17.16cm before ATP stress to 11.93cm during recovery period. There was no obvious abnormality in left ventricular wall motion before and after ATP stress and during recovery.

Discussion

Coronary microvessels with spasm trend are often in a slight contraction state, and radionuclide myocardial perfusion is delayed. When vasodilators are used, myocardial perfusion is significantly improved. This phenomenon is called "reverse redistribution". Although "reverse redistribution" is related to coronary microvascular spasm, it also exists in other diseases, such as stunned myocardium and scar myocardium[3-4]. This case reports for the first time that the "reverse redistribution" of myocardial perfusion during ATP stress MCE may be related to coronary microvascular spasm. The decrease of coronary blood flow reserve function in the distal segment of the anterior descending artery also indicates the dysfunction of myocardial microcirculation in this region [5], and the blood flow spectrum resistance in the distal segment of the left anterior descending artery during the recovery period was significantly increased and the blood flow velocity integral was reduced, It further showed that the microvessels in the distal part of the left anterior descending branch induce spasm and contraction after vasodilation, resulting in the increase of blood flow spectrum resistance and the decrease of blood flow volume in the distal part of the coronary artery. It had been reported that ATP stress induced coronary vasospasm, but the exact mechanism of ATP induced coronary microvascular spasm was not clear, which might be related to the damage of vascular endothelial cells, the decrease of endothelium-dependent relaxation and the enhancement of reactive contraction [6-7]. The factors leading to vasospasm mainly included smoking, drinking, damage to vascular endothelium, excessive contraction of smooth muscle cells, autonomic nerve regulation disorder and inflammation [8-9]. This patient also had risk factors of smoking and drinking. In the past, ATP induced coronary spasm was considered as a side effect of ATP. In fact, ATP induced coronary spasm was not the spasm of the whole coronary artery [10], and this patient was only limited to the delayed defect myocardial perfusion of some segments of the left ventricular wall. Therefore, it could be inferred that there were certain lesions or dysfunction in myocardial coronary microvessels in areas with abnormal perfusion. When these coronary microvessels were stimulated by their own risk factors, they could induce coronary microvascular spasm and cause chest pain. This case needed to be distinguished from left anterior descending coronary artery spasm. In this case, there was no slight contraction of large coronary artery in resting coronary angiography, the area of delayed myocardial perfusion was inconsistent with the distribution area of coronary artery, and there was no significant acceleration of coronary blood flow. Therefore, it was considered that it was not caused by large coronary artery spasm.

There were few cases of ATP induced coronary microvascular spasm previously reported. On the one hand, the main reasons included there were no characteristic changes in ECG, two-dimensional ultrasound, CT and coronary angiography. On the other hand, during the process of ATP stress cardiac detection, the continuous observation of the distal coronary flow spectrum during the recovery period was ignored, and the diagnosis of coronary microvascular spasm was omitted.

Conclusion The "reverse redistribution" in ATP stress MCE myocardial perfusion and the increased spectral resistance of distal coronary blood flow during recovery further confirmed that the abnormal perfusion area was related to coronary microvascular spasm, which could provide a valuable reference for clinicians to find the cause of chest pain and chest tightness.

Reference

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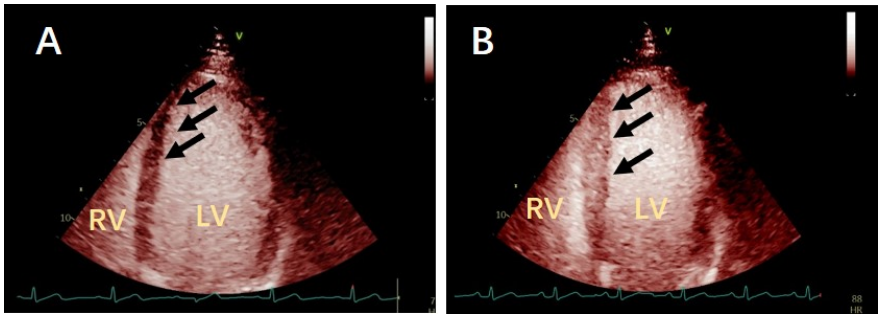

Figure 1 Transthoracic apical 4-chamber view,MCE showed "abnormal reperfusion" at the apex/mid of the ventricular septum, that is, there was a delayed perfusion defect in the apical/mid ventricular septum before ATP stress (A) (After "flash" for 5 cardiac cycles) (black arrow). At the ATP peak stress (A), myocardial perfusion was significantly improved at the same position (Within "flash" for 2 cardiac cycles) (black arrow).

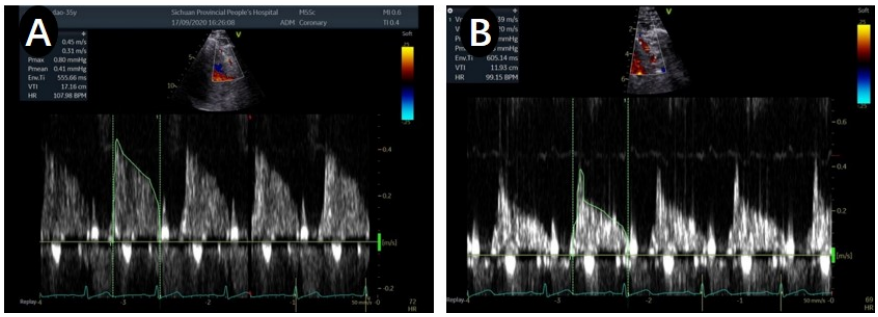

Figure 2 Changes of blood flow spectrum in the distal segment of left anterior descending coronary artery by transthoracic ultrasound. In the recovery period(B), the coronary flow spectrum resistance increased significantly and the blood flow velocity integral decreased compared with that before ATP stress(A).