The value of ATP stress MCE in evaluating myocardial microvascular spasm – a case report 1

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Abstract

We reported a patient with chest pain, but the coronary angiography was normal. ATP stress myocardial contrast electrocardiography(MCE) was performed. There was apical ventricular septal perfusion delay before ATP stress, and the perfusion delay areas were significantly reduced at the peak period, which was similar to the "reverse redistribution" perfusion characteristics of nuclear myocardium in coronary vasospasm, The areas of delayed perfusion in the recovery period were larger than that before stress, the increase of blood flow spectrum resistance in the distal segment of left anterior descending coronary artery and the occurrence of chest pain all showed that ATP induced myocardial microvascular spasm. The MCE perfusion characteristics and the changes of coronary spectrum had certain clinical value in the diagnosis of myocardial microvascular spasm.

Introduction

There were many causes of chest pain in patients. In addition to coronary stenosis, coronary spasm and microcirculation lesions were also common causes of chest pain [1]. Because there was no "gold standard" for the detection of myocardial microcirculation lesions, especially for the diagnosis of myocardial microvascular spasm, some patients were often missed and misdiagnosed. We reported a case of ATP-induced microvascular spasm and the diagnostic value of echocardiography.

Case introduction

A 35-year-old male patient presented with recurrent precardiac pain without obvious cause 4 months ago. The pain lasted for tens of seconds and was relieved spontaneously. The frequency of pain increased 1 week ago. His blood pressure(BP) was 120/77 mmHg, heart rate 71 beats per minute. His physical examination did not hear heart murmur and friction sound, and no obvious abnormal signs were found. There was no obvious history of heart disease , hypertension and diabetes. However, there was a long-term smoking history of about 10 years, 15 cigarettes /one day, and drinking for 7 years, 100g/ one day. Laboratory examination showed that CK-MB, troponin and brain natriuretic peptide levels were not significantly abnormal. No obvious abnormality was found by echocardiography. ECG showed that the T wave of lead V4-V6 was inverted. Coronary angiography exhibited 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. With the consent of the patient and the approval of the hospital ethics committee, ATP stress myocardial contrast electrocardiography (MCE) was used to detect the myocardial microvascular function of the patient.

ATP was injected into the fore arm cephalic vein at 140ug/min/Kg for 6 minutes. Ventricular wall motion, myocardial perfusion, blood flow spectrum of distal anterior descending branch, clinical symptoms, ECG and BP were observed before stress, 2-3 minutes after administration and during recovery period.

During MCE, the amount and speed of ultrasound microbubbles injected each time were the same. The results showed a slight delay in myocardial perfusion in the ventricular septal region at the apex of the heart

before stress (at the end of the diastolic period of five cardiac cycles after "flashing") (Fig. 1A). At peak period, MCE showed perfusion delay areas decreased (diastolic end of two cardiac cycles after "flashing") (Fig. 1B). Perfusion delay areas showed significantly larger than that before stress in the recovery phase (diastolic end of five cardiac cycles after "flashing") (Fig.1C). Coronary blood flow reserve CFR= mean diastolic coronary blood flow velocity before loading/mean diastolic coronary blood flow velocity during peak stress, which was 2.2. The diastolic blood flow spectrum resistance of coronary artery in recovery period was significantly higher than that before stress, showing a "thumb" spectrum (Fig. 2A and B). The blood flow spectrum velocity integral changed from 17.16cm before stress to 11.93cm in recovery period. There was no obvious abnormality in left ventricular wall motion before stress, peak period and recovery period, and there was no significant change in ECG and BP. The patient had no obvious symptoms before stress and at the peak period, and had chest pain for about 30 seconds in the recovery period, which was similar to ordinary chest pain. No chest pain occurred after clinical treatment with Diltiazem hydrochloride tablets.

Discussion

This case was the first to report the "reverse redistribution" of local myocardial perfusion during ATP stress MCE. Previous reports showed that coronary vessels with spastic trend were often in a slight contraction state, and radionuclide myocardial perfusion was delayed. When vasodilators were used, myocardial perfusion was significantly improved. This phenomenon was called "reverse redistribution", which was related to coronary vasospasm, but it also existed in other diseases, such as stunned myocardium, scar myocardium, etc. [2-3]. The decrease of coronary flow reserve also indicates the dysfunction of myocardial microcirculation in these areas [4]. Chest pain, expansion of delayed myocardial perfusion areas, increase of distal coronary flow spectrum resistance and decrease of blood flow volume all showed local myocardial microvascular contraction during recovery period. Cases of ATP-induced coronary vasospasm have been reported, but the exact mechanism of ATP-induced coronary vasospasm was not clear, which could be related to the damage of vascular endothelial cells, the decrease of endothelium-dependent relaxation and the enhancement of reactive contraction [5-6]. The factors leading to vasospasm mainly included smoking, drinking, damage to vascular endothelium, excessive contraction of smooth muscle cells, autonomic nerve regulation disorder , inflammation and so on[7-8]. 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, and this patient was only limited to the delayed myocardial perfusion of some segments of the left ventricular wall. Therefore, it could be inferred that there were certain lesions or dysfunction in myocardial micro vessels in areas with delayed perfusion. When these regional myocardial micro vessels were stimulated by risk factors, they could induce spasm and chest pain. This case needed to be distinguished from the large coronary spasm of the left anterior descending artery. In the resting state, the coronary angiography did not find a slight contraction of the large coronary artery, the delayed areas of myocardial perfusion were inconsistent with the distribution areas of the coronary artery, and the coronary blood flow did not accelerate significantly. Therefore, it was considered that it was not caused by the large coronary spasm.

There were few cases of ATP-induced coronary micro vasospasm reported in the past. The main reasons were as follows. On the one hand, myocardial micro vasospasm had no characteristic changes in ECG, 2D ultrasound, CT and coronary angiography. On the other hand, the characteristics of myocardial perfusion and coronary flow spectrum were ignored in recovery period.

Conclusion The "reverse redistribution" areas of ATP stress MCE were the prone areas of myocardial microvascular spasm, and the areas of delayed myocardial perfusion in the recovery period were the areas of ATP-induced myocardial microvascular spasm. Therefore, ATP stress MCE could provide a valuable reference for clinicians to seek chest pain caused by myocardial microvascular spasm.

Abbreviation

ATP Adenosine triphosphate

MCE myocardial contrast electrocardiography

BP blood pressure

ECG electrocardiogram

Reference

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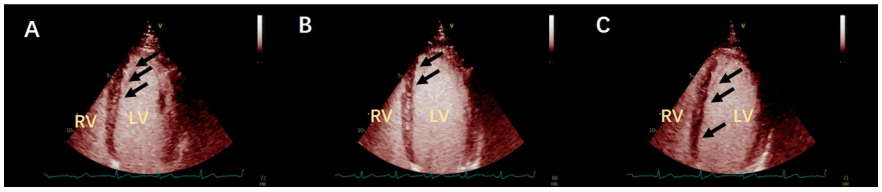

Figure 1 Image of four-chamber cross-section myocardial perfusion angiography.

A there was delayed perfusion at the apex of ventricular septum before load (end of diastole of 5 cardiac cycles after "Flash", arrow). B the delayed myocardial perfusion areas were reduced at peak period (end of diastole of 2 cardiac cycles after "Flash", arrow) ,this was " reverse reperfusion" .C the delayed areas of myocardial perfusion were significantly increased during recovery period.

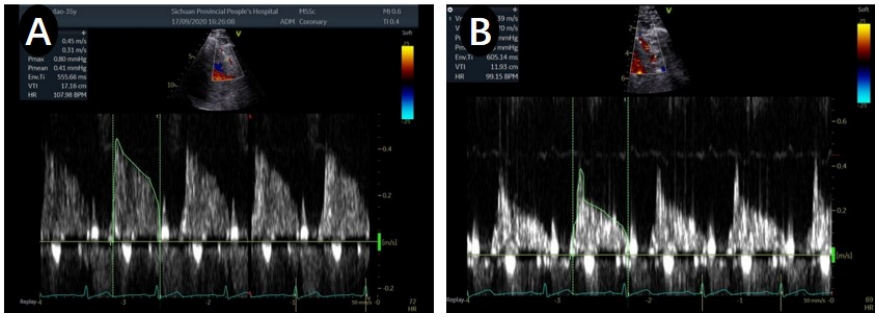

Figure 2 Blood flow spectrum of the distal segment of the left anterior descending coronary artery. In the recovery period (B), the coronary flow spectrum resistance increased significantly and the blood flow velocity integral decreased compared with that before stress(A).