



Case Report

Double Whammy-A Case Report of Acute Myocardial Infarction Combined with Coronary Microvascular Dysfunction

Chunmei Qi*, Xiaodan Fan, Qingdui Zhang, Chengmeng Zhang

Department of Cardiology, the Second Affiliated Hospital of Xuzhou Medical University, No. 32 Meijian Road, Xuzhou, Jiangsu Province, China

*Corresponding author: Chunmei Qi, Department of Cardiology, the Second Affiliated Hospital of Xuzhou Medical University, 32 Meijian Road, Xuzhou, Jiangsu Province, China

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Abstract

Coronary artery disease incidence increases annually, while the average age of patients suffering from acute coronary syndromes is decreasing. Patients without obstructive coronary artery disease have also been proven to have a high risk of angina, meanwhile, patients with acute myocardial infarction are also complicated with perfusion disorder even after revascularization of coronary arteries and major branches. Which coronary microvascular dysfunction takes great responsibility of? We presented a 25-year-old man with a 17-year history of smoking and a 10-year history of drinking who underwent persistent severe chest pain for 1 hour. After immediate electrocardiogram and serum myocardial enzymes assay in emergency room, this young man was finally diagnosed as acute myocardial infarction. Emergency percutaneous coronary intervention was ready to perform through the green channel of chest pain center. Coronary angiogram showed total occlusion in the proximal left anterior descending coronary artery, distal left circumflex coronary artery and right coronary artery with TIMI 0 flow. After repeated balloon dilatation and intracoronary administration, the patient was then sent to CCU for further rehabilitation with pharmacotherapy. Coronary angiography was taken again 8 days later to reappraised the left anterior descending coronary artery, distal left circumflex coronary artery and right coronary artery as no blocking with TIMI 2 flow suggesting coronary microvascular dysfunction. Follow-up examination also confirmed myocardial infarction with coronary microvascular dysfunction. Afterwards, prescriptions including dietary and therapeutic lifestyle strategies were given for long-term rehabilitation. Follow-up indicated total remission of symptom and gradual improvement of cardiac function. Without hereditary factor, age factor or chronic disease factors, this young patient was attacked by acute myocardial infarction due to the over-consumption of his health. The more important thing was that acute myocardial infarction combined with coronary microvascular dysfunction suggested a poor prognosis which need earlier and more effective interventions urgently.

Keywords: Acute myocardial infarction; Coronary microvascular dysfunction; TIMI flow

Introduction

With the rapid development of socioeconomic status and the prevalence of unhealthy lifestyles, the incidence of coronary artery disease (CAD) increases annually, while the mean age of the patients suffering from acute coronary syndromes (ACS)

is decreasing, making CAD one of the most important diseases threatening public health. Studies have confirmed that the degree of subepicardial coronary artery stenosis and the nature of plaque are the main factors leading to cardiovascular events, while the structure and function of coronary microvessels have not received enough attention. The treatment of patients with acute myocardial infarction (AMI) aims to open subepicardial infarct-related blood vessels. However, even the successful opening of blood vessels

does not always result in the recovery of acute myocardial perfusion because of coronary microvascular dysfunction (CMD), one endotype of nonobstructive coronary artery disease, which suggests poor prognosis [1]. Approximately 70% of patients with CAD undergoing coronary angiogram (CAG) still have angina symptoms and evidence of clinical myocardial ischemia, which indicates that CMD exists in addition to CAD [2,3]. Oxidative stress and inflammatory reactions are important causes of ACS. CMD can interact with oxidative stress and inflammatory reactions with each promoting the other to form a vicious cycle leading to the progression and aggravation of ACS [4]. Moreover, CMD may occur after AMI and further aggravate myocardial injury [5]. We present the following case in accordance with the CARE reporting checklist (available at <http://dx.doi.org/10.21037/cdt-21-748>) (Table 1).

| Time | Events |
|-----------------------|---|
| 19:49 11 October 2018 | First electrocardiogram and serum myocardial enzymes assay in emergency room (Figure 1,2) |
| 20:13 11 October 2018 | Emergency PCI was ready to perform through the green channel of chest pain center. |
| 20:53 11 October 2018 | First balloon dilatation (Video 1, 2) |
| 21:26 11 October 201 | Intracoronary administration (Video 3) |
| 21:26 11 October 201 | Serum myocardial enzymes reexamination after admitting to CCU (Figure 2) |
| 22:38 11 October 2018 | Echocardiography, serum myocardial enzymes reexamination (Figure 2) |
| 17 October 2018 | CAG reexamination (Video 4) |
| 19 October 2018 | Discharge |
| 20 October 2018 | Adenosine stress perfusion imaging via SPECT |
| 19 November 2018 | Echocardiography reexamination |
| 22 November 2018 | Adenosine stress perfusion imaging via SPECT and echocardiography reexamination |
| 9 May 2019 | Echocardiography reexamination |

Table 1: Timeline.

Case Presentation

A 25-year-old man presented with severe resting chest pain and dyspnea for 1 hour without related primary diseases or family medical history. Immediate electrocardiogram was taken and showed typical ST-segment elevation in lead of II, III, AVF and V2-V6 (Figure 1) after his arrival at emergency room, which suggested acute myocardial infarction (AMI). The significantly raised serum myocardial enzymes (Figure 2) indicated that this young man had been suffering from AMI for more than an hour which was long before the occurrence of clinical symptoms. After entering the green channel of chest pain center, emergency coronary angiography was performed and showed total occlusion in proximal segment of left anterior descending artery (LAD), distal segment of left circumflex branch (LCX) (Video 1) and right coronary artery (RCA) (Video 2) with TIMI 0 flow. After repeated balloon dilatation, the second diagonal branch was reperfused, while proximal segment of LAD and LCX remained no perfusion even after intracoronary administration of tirofiban and sodium nitroprusside (Video 3). The young man was admitted to CCU after

operation for rehabilitation followed by regular electrocardiogram which exhibited ST segment coming down in lead of II, III and AVF, but r wave amplitude decreasing in lead of V2-V6 (Figure 3). Tirofiban was continuously intravenous pumped for 48 hours after percutaneous transluminal coronary intervention (PCI), with oral medication of aspirin, ticagrelor, atorvastatin and metoprolol. When the serum myocardial enzymes were nearly normal, coronary angiography re-examination performed 8 days after patient's AMI displayed no stenosis in LAD, LCX or RCA, whereas the visual scoring of LAD and LCX was TIMI 2 flow indicating CMD combination (Video 4,5). Without other interventional treatment, nifedipine was added to restore the disturbance of coronary microcirculation. After discharge, the patient was instructed to take prescribed medication, low fat diet, physical activity, regular schedule and other healthy modifications. The review of electrocardiograms after discharge indicates that the patient's condition was significantly improved. Therefore, such patients should be identified early and given the correct intervention, which can significantly reduce the risk of clinical events.

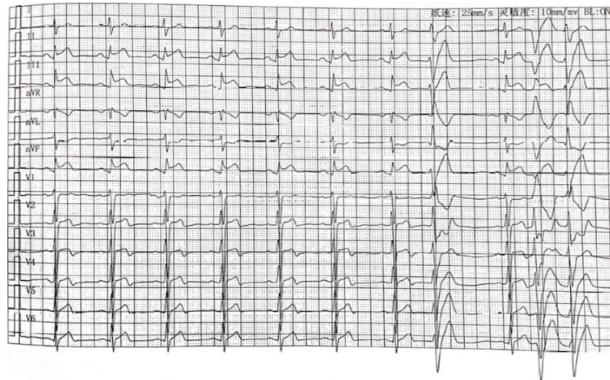


Figure 1: Electrocardiogram after arriving at emergency room showed ST-segment elevation in lead of II, III, AVF and V2-V6. Ventricular extrasystole occurred singly and in pair.

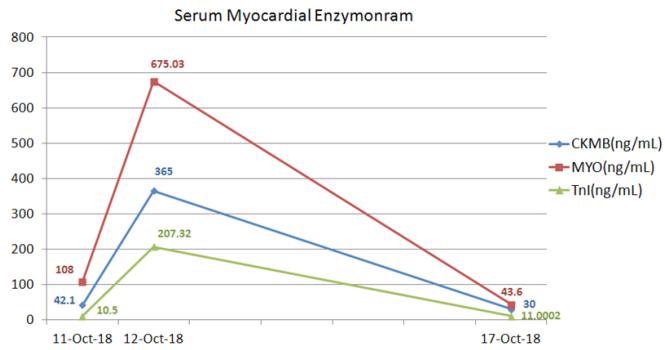


Figure 2: Serum myocardial enzymes raised after patient's arriving at emergency room, significantly increased within 24 hours and fell back 6 days later. *CKMB: Creatine Kinase-MB; MYO: Myoglobin; TnI: Troponin I.

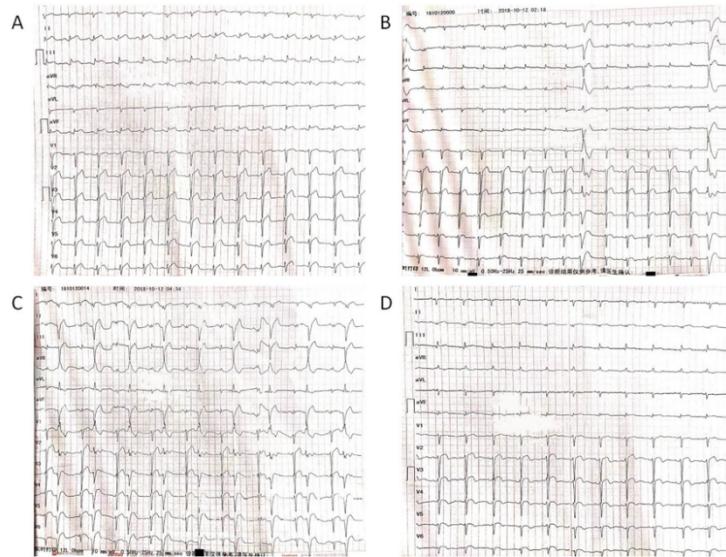


Figure 3: Regular reexamination of electrocardiogram showed ST segment coming down in lead of II, III and AVF, but r wave amplitude decreasing in lead of V2-V6 as rS type. A was taken at 23:00 11 October 2018. B was taken at 02:18 12 October 2018. C was taken at 04:34 12 October 2018. D was taken at 10:51 12 October 2018.

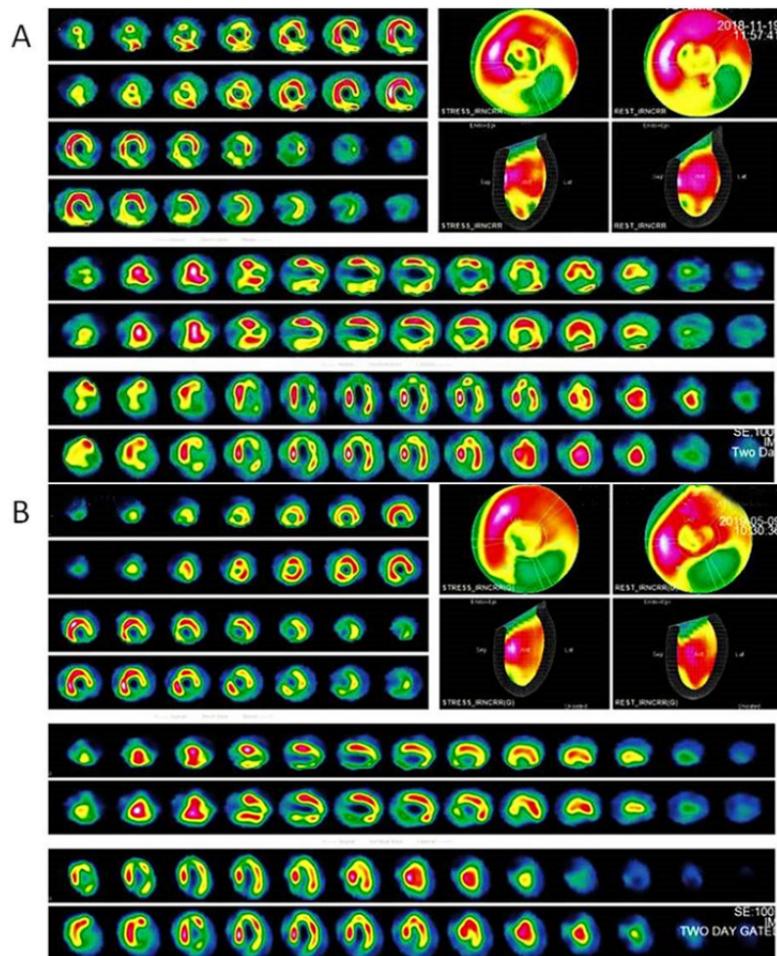


Figure 4: Adenosine stress perfusion imaging via SPECT: Myocardial imaging of the left ventricle was still clear, and the radioactive distribution of partial myocardium in the anterior wall, anterior apex, inferior posterior wall and lateral wall was obviously sparse, while the reflective distribution of the rest of the ventricle was not obviously abnormal. In the resting state, the distribution of the above sparse radioactive areas was partially improved, showing reverse redistribution, and there was no obvious abnormality in the reflection distribution of other chamber walls. A was taken in 19 November 2018. B was taken in 9 May 2019.

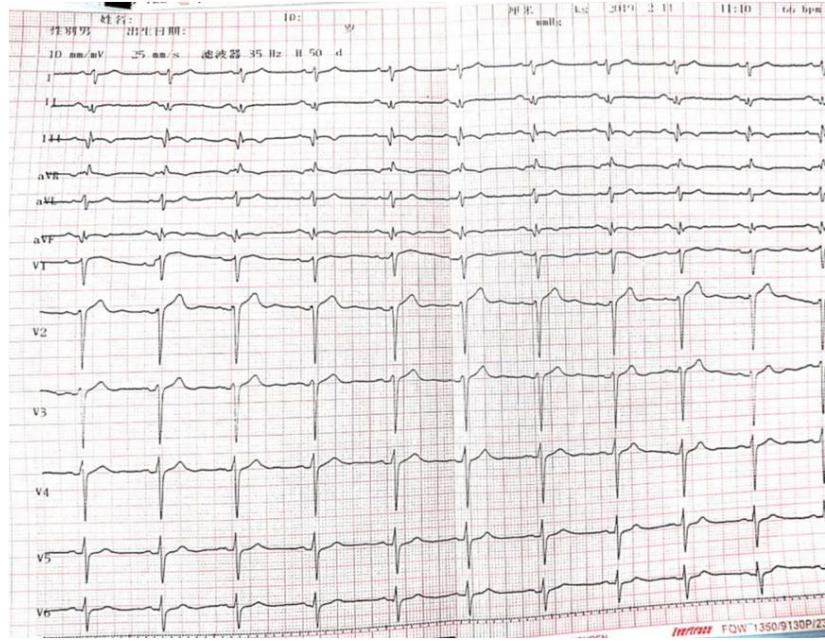


Figure 5: Electrocardiogram was almost normal at 4-month follow-up.

Video 1: Coronary angiography showed a total occlusion in the proximal of LAD, distal LCX.

Video 2: Coronary angiography showed a total occlusion in distal RCA.

Video 3: Coronary angiography showed no reperfusion after intracoronary administration of tirofiban and sodium nitroprusside in LAD and LCX.

Video 4: Coronary angiography reexamination 8 days later showed no stenosis in LAD and LCX, but TIMI 2 flow.

Video 5: Coronary angiography reexamination 8 days later showed no stenosis in RCA, but TIMI 2 flow.

Follow-up

Color doppler ultrasonography on the seventh day showed weakened myocardial motion of left ventricular involving inferior wall, posterior wall and lateral wall with reduced left ventricular ejection fraction (LVEF) as 48%. Adenosine stress perfusion imaging via SPECT confirmed myocardial injury (Figure 4) at the 1-month and 4-month follow-up. Electrocardiogram was almost normal at the 4-month follow-up (Figure 5), and ultrasonography suggested slightly moderated myocardial motion of left ventricular covering with inferior wall, posterior wall and apex with LVEF as 53% and 54% respectively at the 1-month and 6-month follow-up. When recently reexamined in clinic, more than 2 years' taking medicine as directed after AMI occurrence, ultrasonography was almost normal indicating a good prognosis.

Discussion

The patient could not undergo FFR to evaluate microvascular damage because of his poor financial condition. It was also regrettable that our hospital had not carried out CMR or OCT for the evaluation of CMD, which had been pushing forward by our team these years. So we defined this patient as AMI combined with CMD via the examination of CAG and SPECT. What makes the present case interesting is that CMD combined with AMI is partly due to poor living habits including smoking, drinking, staying up late and lack of exercise. This patient had smoked one pack a day for 17 years, drank 100 g a day for 10 years and stayed up late for 8 years, seemed to be without other ascertainable macrovascular risk factors. But the serum lipid level was significantly abnormal after hospital examination, which was one of major risk factors for coronary heart disease. This occurrence of AMI was in a young man. We ended the operation with PTCA alone and intensive drug treatment. Eight days postoperatively, angiogram showed no narrowing at the coronary vessels, confirming that the patient was complicated with CMD. CMD is one of the mechanisms leading to myocardial ischemia. Initial studies suggested that these patients had a good prognosis. Unfortunately, this young man experienced obstructive CAD. CMD refers to the clinical syndrome of exertional angina or myocardial ischemia caused by the abnormal structure and/or function of coronary anterior arterioles and arterioles under the influence of multiple pathogenic factors. CMD can occur in four separate clinical settings: (1) in the absence of obstructive epicardial CAD and structural heart disease, (2) in the presence of structural heart disease, (3) in the presence of obstructive epicardial CAD, and (4) secondary to iatrogenic causes. CMD diagnostic factors include clinical symptoms and auxiliary examination. The current technology cannot directly observe microvessels in the human body; they can only be evaluated by measuring myocardial blood flow (PET, myocardial contrast echocardiography, MR), coronary circulation blood flow (coronary artery invasive/noninvasive techniques such as internal doppler flow guidewire), TIMI score and coronary flow reserve (microcirculation resistance index, coronary flow reserve) to reflect coronary microvascular function [4]. These auxiliary examinations have advantages and disadvantages in the evaluation of coronary microvascular function, and they need comprehensive development. The use of traditional antianginal and antiatherosclerotic medications and some novel agents may be beneficial; however, clinical trials are needed to assess the efficacy of pharmacologic and nonpharmacologic therapeutic modalities. In addition, studies with longer-term follow-up are needed to determine the prognostic benefits of these agents. Current treatments for CMD include management of risk factors, application of drugs to reduce postcardiac load and heart rate, and application of ATP-sensitive potassium channel openers to regulate coronary microcirculation; for CMD with obstructive coronary disease, attention needs to be paid attention to membrane

coronary artery and CMD lesions at both levels, enhanced risk factor control, revascularization intervention, and coronary microcirculation regulation. Smoking was proven to be in strongly associated with CMD [4]. CMD carries an increased risk for adverse cardiac events, thus, it should be aggressively managed. A healthy lifestyle and rational use of drugs can better improve myocardial microcirculation reperfusion, reduce the occurrence of slow and no reflow blood flow, and reduce the occurrence of adverse cardiovascular events.

Conclusion

Risk factors for CMD are similar to traditional cardiovascular disease risk factors, including smoking and drinking. Treatment of CMD starts with lifestyle modification and risk factor control. Invasive functional coronary angiography can be considered if diagnosis is difficult. With the application of cardiac magnetic resonance imaging, optical coherence tomography and intracoronary physiology examinations these years, diagnosis of coronary microvascular dysfunction is no longer puzzling, but there is a long way to go to search for special therapy.

Reporting Checklist: The authors have completed the CARE reporting checklist (available at <http://dx.doi.org/10.21037/cdt-21-748>).

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. Written informed consent was obtained from the patient for use data for academic publication purposes.

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