Case Report

REGRESSIVE MYOCARDIAL INFARCTION WITH ST ELEVATION (STEMI) TREATED WITH CONSERVATIVE MEDICINAL THERAPY

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Summary

The case presented is that of a young man with atypical pathogenesis of myocardial infarction with ST-elevation (STEMI) after physical stress. It was provoked by partial thrombosis of the distal segment of the left main coronary artery (LM) at the bifurcation of the left anterior descending artery. After the emergency treatment, the disease process underwent reverse development without any consequences for left ventricular kinetics.
Key words: Acute myocardial infarction, STEMI, chest pain, coronary thrombosis, optical coherence tomography (OCT)

Introduction

We present a case of an acute coronary syndrome with ST elevation that was treated after conservative therapeutic scheme after clinical assessment of the patient. The coronary network status was also assessed using advanced technical methods. A team of cardiologists discussed the case. This led to proper decision-making for the patient's medical care.

Notwithstanding the tendencies for such cases to be managed by coronary interventional treatments, this case illustrates the importance of collaborative decision-making for individualized and personalized treatment strategies that rely on practice guidelines and recommendations.

The case description

We report a 31-years-old male patient. In the beginning, he was admitted to the hospital complaining of persistent constrictive chest pain with duration of 90 minutes. The pain appeared one hour after he had played football. He did not mention any past illnesses. The patient had no personal previous medical history. There was a risk factor- a family history of cardiovascular disease. At the age of 50, his father had myocardial infarction. On the maternal side, there were an aunt and an uncle who had suffered from ischemic coronary disease before age 60.

On hospital admission, the electrocardiogram

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showed ST-elevation of the lower and lateral leads, and ST-depression of the anterior leads (Figure 1).

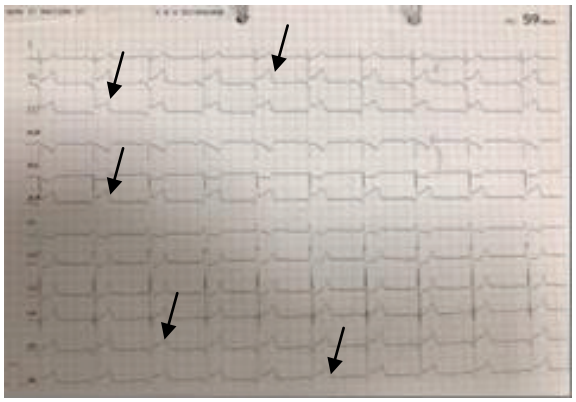

Figure 1. First ECG: ST-elevation of the lower and lateral leads and ST-depression of the anterior leads

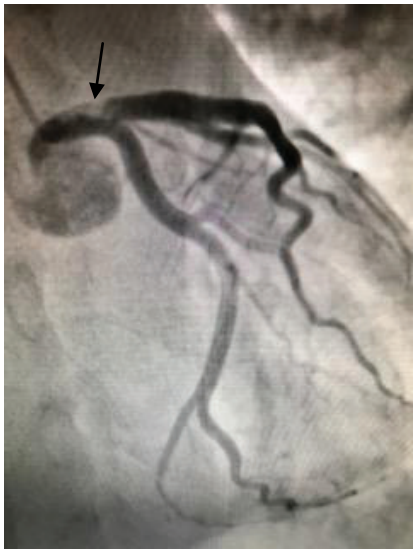

Figure 2. Coronarography: non-obstructive left main coronary artery distal segmental thrombosis, bifurcation with the left anterior interventricular artery

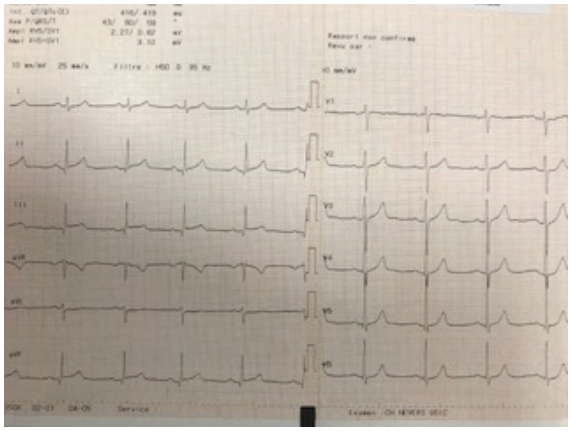

Figure 3. Second ECG: quasi-total regression of ST elevation

The patient demonstrated an increase in myocardial necrosis markers (troponin Ic).

A coronarography test was done 2 hours after the symptom onset and after administration of 250 mg of acetylsalicylic acid IV, heparin 5000 IU IV and 60 mg of prasugrel PO. It showed non-obstructive left main coronary artery (LM) distal segmental thrombosis, at the bifurcation of the left anterior descending artery (Figure 2).

At this point, the chest pain and the above-described electrocardiographic abnormalities disappeared (Figure 3). Therefore, the decision was to continue the dual anti-aggregate therapy, anticoagulation therapy with subcutaneous fondaparinux (arixtra) and intravenous Anti-GP II / III A (tirofiban) for 24 hours.

A second coronarography was done on the 3rd day. It showed a persistent minimal heterogeneous aspect of the distal part of the left main coronary artery and the ostium of the left anterior descending artery (LAD) and, therefore, an essential regression of thrombosis (Figure 4).

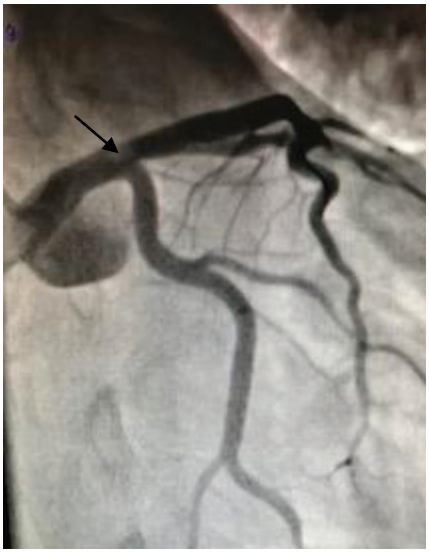

Figure 4. Coronarography on day 3: partial regression of the left main coronary artery distal segmental thrombosis, bifurcation with the left anterior interventricular artery

Optical coherence tomography (OCT) confirmed a plaque rupture without significant stenosis (Figure 5).

Echocardiography showed preserved systolic function of the left ventricle (LVEF=60%) and homogeneous left ventricular kinetics. No valvulopathy was detected, and the size and capacity of the right ventricular cavity were normal.

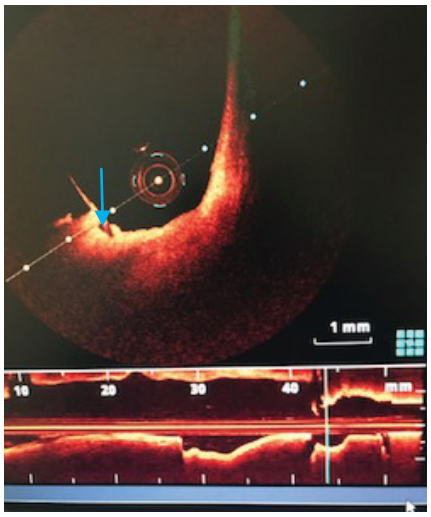

Figure 5. Optical coherence tomography confirmed a plaque rupture without significant stenosis

The myocardial necrosis markers were as follows: troponin Ic – 0.25 mcg/l on admission, and the peak value of troponin on the hour 7 was 15.07 mcg/l

The patient was discharged from hospital with regular therapy for 12 months with prasugrel 10 mg PO QD, acetylsalicylic acid 75 mg PO QD, atorvastatin 40 mg PO QD, bisoprolol 2.5 mg PO QD, pantoprazole 40 mg PO QD and finally – one month of fondaparinux (arixtra) 7.5 mg SC QD.

Control coronary angiography done after two months showed a small atheromatous plaque of the left main coronary artery in the distal segment without significant stenosis with neither thrombosis nor plaque rupture (Figure 6).

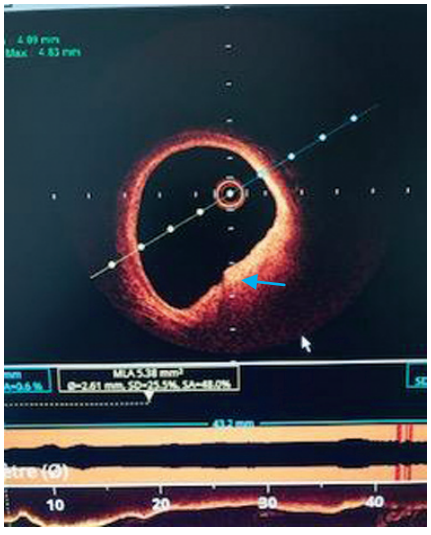

Figure 6. Control coronary angiography after two months: a small atheromatous plaque of the left stem coronary artery in the distal segment without significant stenosis

The thrombophilia profile test result was normal. The homocysteine, anti-thrombin III, protein C and Protein S values were normal, as were the coagulation factors II, V, VII, X. The activated protein C resistance test was normal. No anti-cardiolipin antibodies, anti-β2 glycoprotein 1 antibodies and lupus anticoagulant were detected.

Discussion

The case reported refers to a regressive myocardial infarction with ST-elevation (STEMI) without signs of aggravation, treated in 2015. The thrombus did not obstruct the left main coronary artery at the bifurcation of the left interventricular artery and resulted from a post-exercise plaque rupture [1].

We chose a medical conservative therapeutic strategy taking into consideration that the ST elevation regressed spontaneously and the chest pain subsided (signs of reperfusion). The coronary flow was TIMI III. Therefore, neither for fibrinolysis therapy nor surgical revascularization could be appropriate [2]. More often, the primary angioplasty in this severe thrombotic environment may increase the risk of coronary flow alteration [3].

Many studies and publications support the strategy of deferred angioplasty [4-6] which avoids coronary stenting in certain patients and allows selection of conservative medical treatment with anti-thrombotic glycoprotein IIb/IIIa inhibitors, double antiaggregant and anticoagulant [7, 8]. In fact, N. Amabile et al. (2015) showed the feasibility and the safety of thrombus analysis in STEMI acute phase and its regression later on using OCT [8]. All prescribed medications for the initial medical treatment in the STEMI acute phase and in the discharge prescription meet the recommendations of the ESC [9] except for prasugrel, which should be substituted by ticagrelor.

Conclusions

Partial thrombosis of the left main coronary artery at the bifurcation of the left interventricular artery, due to a small plaque rupture resulting in a temporary ST-elevation regresses after urgent medical treatment. There are no consequences

in left ventricular kinetics. An OCT test has confirmed the disappearance of thrombosis after two months.

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