CASE REPORT

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Acute coronary syndrome in a young patient with ECG presentation of acute inferior myocardial infarction and acute thrombosis of left main stem coronary artery

Akutni koronarni sindrom kod mladog bolesnika sa EKG prezentacijom akutnog infarkta donjeg zida miokarda i akutnom trombozom glavnog stabla leve koronarne arterije

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Abstract

Introduction. The left main stem (MS) coronary artery (CA) (MSCA) thrombosis is a rare but potentially lethal manifestation of acute coronary syndrome. The standard approach in treating such patients is the primary percutaneous coronary intervention (pPCI) or CA bypass graft surgery. In some cases, depending on the morphological appearance of the thrombus, findings and flow rates assessed on coronary angiography (CAn), clinical conditions, and cardiologist's experiences, another possible method of treatment can be the conservative approach using antithrombotic therapy. **Case report.** A 37-year-old male was admitted to the emergency room with symptoms of an acute myocardial infarction with an ST elevation in diaphragmal localization. Using an emergency CAn, we have visualized a thrombus at the ostial and proximal part of the left MSCA, with no complete obstruction of the blood flow. Initially, dual antithrombotic therapy (ticagrelor and acetylsalicylic acid) was applied, and in the further procedure, it was decided to introduce glycoprotein IIb/IIIa platelet receptor inhibitor (tirofiban) as an intracoronary bolus (0.3 μg/kg) and later as a continuous infusion (0.1 μg/kg/min). Four days later, a control CAn and intravascular echocardiography were performed, and it was decided to continue the treatment using conservative therapy without a pPCI procedure. The patient was discharged in good condition with no signs of illness on the eighth day after hospital admission for home recovery, with planned frequent follow-ups in the future. **Conclusion.** In the case of non-obstructive thrombotic masses without significant atherosclerotic stenotic lesions, conservative treatment modality with the use of aggressive antithrombotic therapy may be considered.

Key words:
coronary artery disease; coronary vessels; myocardial infarction; platelet aggregation inhibitors; treatment outcome.

Apstrakt

Uvod. Tromboza glavnog stabla leve koronarne arterije (GSLKA) predstavlja retku ali potencijalno smrtonosnu manifestaciju akutnog koronarnog sindroma. Standardni pristup u lečenju takvih bolesnika jeste primarna perkutana koronarna intervencija (pPKI) ili hirurška revaskularizacija miokarda (*coronary artery bypass graft surgery*). U određenim slučajevima, na osnovu morfološkog izgleda tromba, nalaza i protoka dobijenih metodom koronarne angiografije (KAn), kliničke slike, kao i iskustva kardiologa, jedan od načina lečenja može biti i konzervativni pristup intenzivnom antitrombocitnom terapijom. **Prikaz bolesnika.** Muškarac star 37 godina primljen je u jedinicu za hitne slučajeve jer je pokazivao znake akutnog infarkta miokarda sa ST elevacijom dijafragmalne lokalizacije. Urađena je hitna KAn tokom koje je pronađen tromb u proksimalnoj trećini GSLKA, bez potpune opstrukcije protoka krvi. Inicijalno, primenjena je dvojna antitrombocitna terapija (tikagrelor i acetilsalicilna kiselina), a u daljoj proceduri odlučeno je da se uvede inhibitor glikoproteinskog IIb/IIIa receptora trombocita (tirofiban) u

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vidu intrakoronarnog bolusa (0,3 μg/kg), a zatim kao kontinuirana infuzija (0,1 μg/kg/min). Četiri dana kasnije, urađeni su kontrolna KAn i intravaskularna ehokardiografija, nakon čega je odlučeno da se nastavi samo sa konzervativnom terapijom, bez procedure pPKI. Bolesnik je bez tegoba, u dobrom stanju, otpušten na kućno lečenje osmog dana nakon prijema u bolnicu, uz planirano intenzivno praćenje u daljem toku oporavka. **Zaključak**. U slučaju neopstruktivnih trombocitnih masa bez značajne aterosklerozne stenoze, može se razmotriti modalitet konzervativnog lečenja upotrebom agresivne antitrombocitne terapije.

Ključne reči:
koronarna bolest; koronarni krvni sudovi; infarkt miokarda; antiagregaciona sredstva; lečenje, ishod.

Introduction

Acute coronary syndrome is a medical condition involving acute ST-elevation myocardial infarction (MI) (STEMI), non-ST-elevation MI, sudden MI caused by myocardial ischemia and unstable angina pectoris. According to the most recent division of MIs, Type 1 acute MI is caused by rupture or erosion of unstable atherosclerotic plaque in epicardial coronary arteries (CA). Platelet adhesion and aggregation occur when the prothrombogenic material is released from plaque. That is accompanied by the activation of coagulation and the forming of a thrombus that can completely occlude the so-called infarcted artery [1].

Sometimes, a thrombus can be formed in the proximal segment of the coronary artery, and its fragments can embolize the distal segments of the artery and cause a "distant" infarction.

Plaque rupture and occlusive thrombosis in the left main stem CA (MSCA) are usually associated with sudden death or major shock MI. Occlusive thrombi and thrombi that cause critical myocardial ischemia must be resolved urgently either by percutaneous coronary angioplasty or bypass surgery [2]. If the mentioned procedures cannot be done urgently, one of the remaining options would be fibrinolytic therapy. However, this therapy has poor results in patients who are in a state of shock, which is often the case here. If percutaneous or surgical reperfusion is performed, the treatment is continued with dual antiplatelet therapy (DAPT) in order to ensure the permeability of stents or grafts. Rarely, a non-occlusive thrombus in which thrombogenic material has been embolized can be seen in the left MSCA, as a result of which a minor MI is registered [3]. The aim of this case report was to present a patient with a thrombus in the left MSCA and embolization of the distal segment of the left anterior descending (LAD) CA caused by thrombus migration, which also resulted in a smaller occluded posterior descending artery. Therefore, according to its localization, STEMI is manifested as an MI with ST-elevation in the inferior leads on the electrocardiogram (ECG). That raises the question of whether a heart attack lesion is actually a thrombus in the left MSCA or an occluded distal segment of the LAD CA. Questions are also raised regarding the treatment of non-occlusive thrombus mass in the MSCA that does not lead to myocardial ischemia itself. Dual anti-aggregation therapy in combination with glycoprotein (GP) inhibitors, which inhibit the aggregation by blocking the IIb/IIIa receptors on platelets, may be one of the more successful approaches in treating these patients [4, 5].

Case report

The patient was a 37-year-old male admitted to the coronary intensive care unit with chest pain lasting for two hours, clinically and hemodynamically stable, with a blood pressure of 120/80 mmHg and a pulse rate of 60 beats/min. ECG on arrival showed ST-segment elevation in leads II, III, and augmented vector foot (AVF) and tall T waves on the anterior wall (Figure 1). The patient was diagnosed with STEMI of the inferior myocardial wall. He received DAPT (180 mg of ticagrelor and 300 mg of acetylsalicylic acid) and

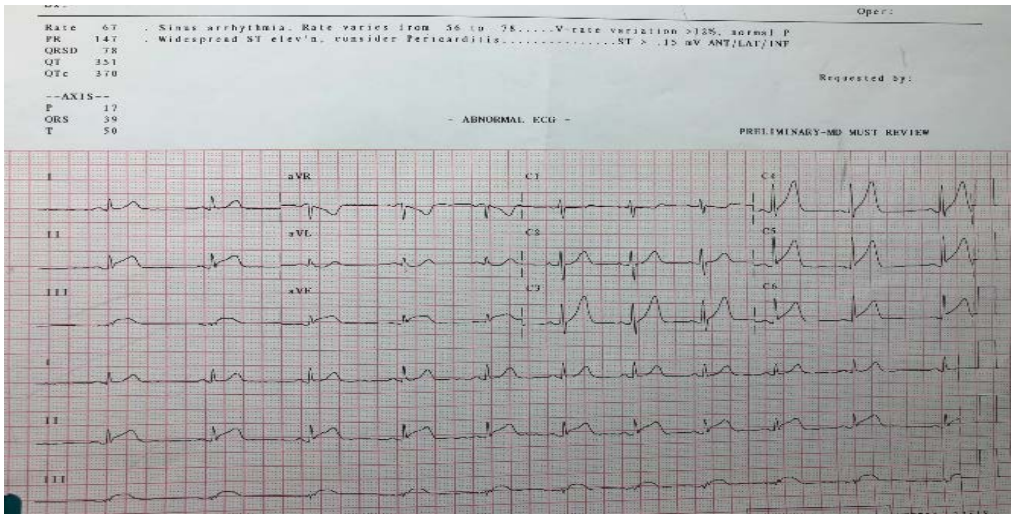

Fig. 1 – Electrocardiogram on admission to the coronary unit. ST-segment elevation in leads II, III, and augmented vector foot and tall T waves on the anterior wall.

was immediately admitted to the cardiac catheterization room for emergency coronary angiography (CAn). CAn was performed using a radial approach. Prior to the review of the coronarographic findings, the patient intravenously received 7,000 units of unfractionated heparin in a bolus. CAn showed a thrombus on the ostial and proximal part of the left MSCA without flow obstruction (Figure 2A). The right CA was without significant changes (Figure 2B). Occlusion of the distal LAD artery in front of the apex of the heart has also been observed, where the artery was less than 2 mm in diameter (Figure 2C). Due to the risk of distal embolization and further iatrogenic microcirculatory injury associated with the use of thrombus aspiration, the clinical decision of the cardiologist performing the intervention was to introduce GP IIb/IIIa platelet receptor inhibitor (tirofiban) as an intracoronary bolus (0.3 μg/kg). The patient was transferred to the coronary intensive care unit where the treatment was continued with tirofiban for 24 hrs as an intravenous infusion (0.1 μg/kg/min) and after that with low-molecular-weight heparin in addition to acetylsalicylic acid, ticagrelor, and atorvastatin. Bedside transthoracic echocardiogram showed normal left ventricular size with an ejection fraction of 60%, with hypokinesis of the distal anterior wall and apex and no pericardial effusion.

In the following days, the patient received DAPT and anticoagulant therapy with enoxaparin subcutaneously in therapeutic doses twice a day, and he was absolutely clinically stable.

Intravascular ultrasound (IVUS) was performed seven days after admission, with atherosclerotic plaque found, narrowing the ostium and proximal segment of the left MSCA by 48%. The minimum lumen area of the left MSCA was 8.12 mm (Figure 3).

There was also an opening of the distal, occluded LAD CA segment (Figure 4) and resolution of ST-segment

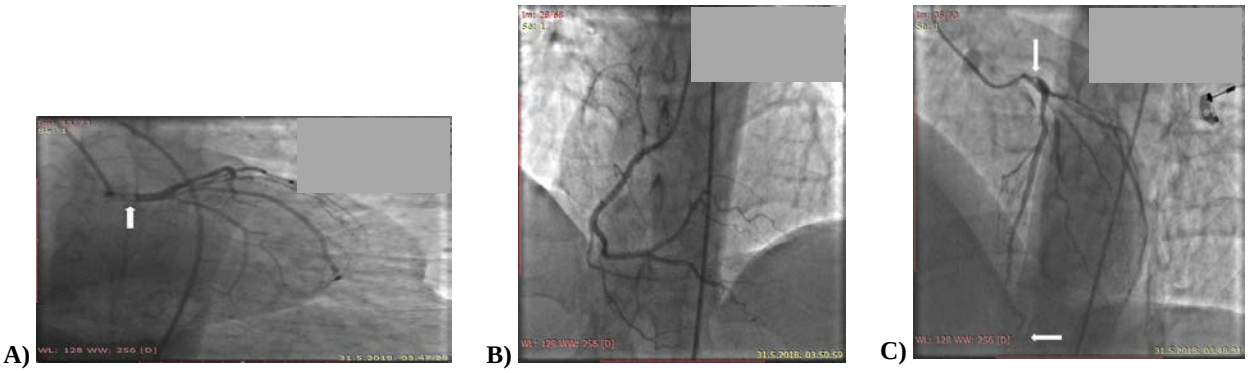

Fig. 2 – Coronary angiography image of: the fluctuating thrombus in the left main stem coronary artery (arrow) (A); the right coronary artery with no obstructions (B); the left coronary artery with distal embolization (arrows)(C).

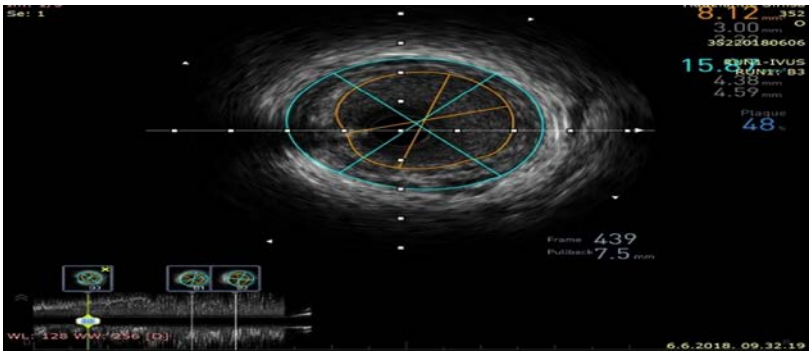

Fig. 3 – Intravascular ultrasound shows 48% stenosis in the left main stem coronary artery.

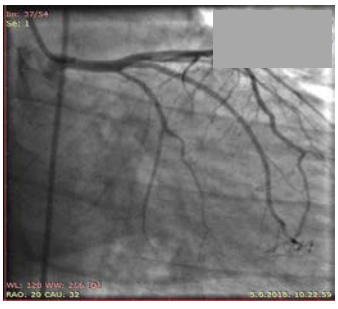

Fig. 4 – Coronary angiography shows an opening of the distal occluded left anterior descending coronary artery segment.

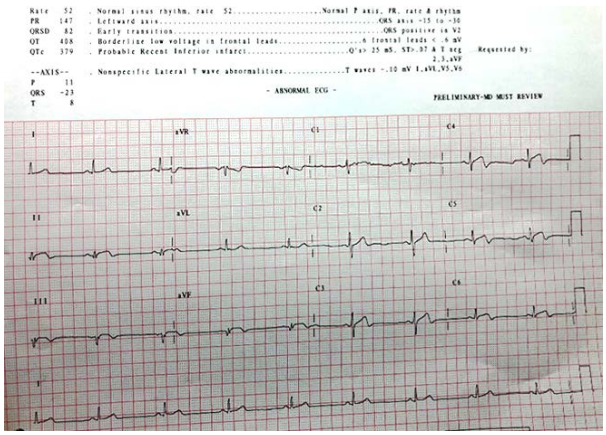

Fig. 5 – Electrocardiogram shows resolution of ST-segment elevation in inferior leads.

elevation in inferior leads (Figure 5). The patient was discharged after eight days and treated with dual antithrombotic therapy for up to 12 months. A follow-up exercise stress test was performed one month later, and the test result was normal. No adverse events occurred during the 12-month follow-up period.

Discussion

As mentioned above, the thrombosis of the left MSCA may result in extensive heart muscle necrosis, cardiogenic shock, or sudden cardiac death [6]. Considering this, the challenge for every cardiologist is an immediate diagnosis and the problem solution. In addition to the acute thrombosis of the MSCA, it is also necessary to think about other potential causes of the clinical condition and ECG changes by the type of necrosis that accompanies MSCA thrombosis, such as aortic dissection, embolic events, CA dissection, vasculitis, or spasm [7–10]. Etiological disorders may include other causes that lead to increased thrombogenic potential, such as taking cocaine (cocaine-induced spasm or rupture of plaque), as well as other reasons [11–17].

In this case report, we describe a patient with acute coronary syndrome but with an ECG presentation of STEMI at a diaphragmal location. Unexpectedly, a thrombosis of the left MSCA was found using CAn, yet no obstructions of the right system were present. An interesting finding was the CA visualization of ostial stenosis and thrombus in the proximal 1/3 of the left MSCA but without complete left MSCA luminal obstruction, which explains the hemodynamic stability of the patient. The infarction in our patient was obviously the consequence of a thrombus fragment causing embolization of the distal segment of the LAD, which passes to the diaphragmal side into the small posterior descendent artery that supplies blood to one part of the lower myocardial wall. We had several treatment options available. The first option was primary percutaneous intervention (pPCI) on the left MSCA, with stent implantation, with or without thrombus aspiration. Another option was the tirofiban therapy for 2–3 days, which would be followed by a bypass surgery. However, as no symptoms were shown during CAn, and the ECG showed a minor lower wall infarction with preserved R wave in inferior standard leads with thrombolysis in myocardial infarction (TIMI)3 flow through all branches of the left CA (LCA) except the distal LAD, which was about 2 mm in diameter, we opted for the third solution. The patient was young and had a low risk of hemorrhagic complications. He had already received a loading dose of ticagrelor and acetylsalicylic acid, and he also received an intracoronary bolus of the GP IIb/IIIa inhibitor, followed by the intravenous infusion of the GP IIb/IIIa inhibitor in the following 24 hrs. Selective CAn was repeated in IVUS after a few days when the atherosclerotic plaque was detected in the proximal segment of the left MSCA with a slight reduction in the circulating lumen of the MSCA, but with no thrombus observed there and with the distal LAD that was passable. We considered that pPCI carried a high risk of infarction extension and thrombus re-embolization. Emergency bypass surgery carried a high risk of hemorrhagic complications because the patient had already received 180 mg of ticagrelor and 300 mg of acetylsalicylic acid. In addition, apart from the thrombus on the ostium of the LCA, no other hemodynamically significant atherosclerotic changes were observed in the branches of the LCA. The triple antiplatelet therapy with therapeutic doses of low molecular weight heparin significantly reduced the chance of growth of thrombus mass and embolization. On the other hand, there was a dethrombosis effect observed with GP inhibitors. Namely, in fresh arterial thrombi, there were a lot of incompletely activated platelets completely inhibited by GP inhibitor, which led to a weakening of the structure and disintegration of the thrombus because activated platelets are thrombus nodes.

Among existing recommendations, there are no particular ones when it comes to the thrombus of the left MSCA. Anyway, it is recommended that the blood flow is restituted to the embolized coronary circulation segregation as soon as possible [18, 19]. The standard for treating such patients is CA bypass graft surgery or pPCI intervention [19]. Nevertheless, in the light of a better understanding of these methods of treatment and greater use of antithrombotic therapy, we have defined a group of patients that have fully recovered after only being treated with the conservative method – the antithrom-

botic therapy [20–23]. As in the above-mentioned case, hemodynamically stable patients are evaluated clinically, using CAn (for non-obstructive thrombus), as well as using additional morphological methods recommended for the left MSCA, such as IVUS, and, after that, a decision can be made to apply the method of treatment with antithrombotic therapy [24].

Conclusion

This case is interesting from two aspects. First of all, thrombus embolization of the left MSCA led to the development of STEMI of inferior myocardial wall localization as a consequence of occlusion of the distal LAD. In addition, and more importantly, the patient was clinically stable with TIMI3 flow through all LCA branches, except the distal LAD artery, with the development of low ischemia of the lower myocardial wall. Due to the high risk of infarction extension by thrombus migration during pPCI and the high risk of bleeding in the case of bypass surgery, we successfully applied triple antiplatelet therapy with anticoagulant therapy while doing subsequent angiographic control using IVUS. In this case, conservative treatment led to a complete lysis of the thrombus mass, and the patient was discharged to home treatment with minimal heart tissue necrosis. In the case of non-obstructive thrombotic masses without the presence of a significant atherosclerotic stenotic lesion, the use of aggressive antithrombotic therapy as a treatment modality may be considered.

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