

CASES OF MOBILE THROMBI IN CORONARY ARTERIES

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SUMMARY

Five cases of mobile thrombi in coronary arteries revealed by angiography are described; repeated examinations demonstrated lysis of clots during a course of therapy.

Keywords: coronary arteries, mobile thrombi, myocardial infarction.

AMI – acute myocardial infarction

CAG – coronary angiography

CPK – creatine phosphokinase

ECG – electrocardiography

LAD – left anterior descending artery

LCA – left coronary artery

RCA – right coronary artery

TIMI — thrombolysis in myocardial infarction

INTRODUCTION

In rare cases, coronary angiography (CAG) reveals masses in coronary arteries, visualized as oscillating contrast defects, not fully occluding the lumen of an artery. In the literature, such findings have been called “mobile” or “movable thrombus” [1-3]. In 3 cases found in the literature, there were men aged 33 and 44 and a 24-year-old woman hospitalized for acute myocardial infarction (AMI). Both men had multiple coronary artery thrombosis — occlusion of the right coronary artery (RCA) and the mobile thrombus in the proximal third of the left anterior descending artery (LAD) in one case [1], and simultaneous occlusion of LAD and RCA and the mobile thrombus in the circumflex artery in the other patient [3]. The woman had a mobile thrombus detected in the middle third of the LAD, but the other coronary arteries were unchanged [2]. The management consisted of RCA angioplasty and intracoronary infusion of tiskinase 6.400.000 IU in one case and installation of a balloon for intra-aortic counter pulsation and thromboaspiration of coronary arteries in the second patient. Repeated CAG performed in a month in one patient and intravascular ultrasound performed in a week in the

second patient showed a good treatment effect. The young woman underwent intracoronary thrombolysis with a good effect too.

Five hundred one thousand CAG procedures performed in N.V. Sklifosovsky Institute in 2009 — 2011 revealed mobile clots in the coronary arteries in 5 patients (0.01%): 4 men and 1 woman aged 32 to 73 years. CAG was performed within 24—48 hours after hospitalization in 3 patients and also 1 and 2 months after AMI in 2 patients.

CLINICAL OBSERVATIONS

A 50-year-old male patient V. was hospitalized 4 h 20 min after the intense chest pain had appeared. ECG showed heart rate 101 beats per minute, the horizontal position of heart's electrical axis, the picture of the acute phase myocardial infarction of the left ventricle with ST-segment elevation in II, III, aVF, V6 leads up to 1.5 mm. Hyperfermentation (CPK 669 IU/L, CK-MB 56 IU/L, troponin 9.76 ng/mL) was revealed. Echocardiography showed dilation of the left atrium (40 mm, 70 mL), moderate myocardial hypertrophy of the left ventricle (inter-ventricular septal thickness 12 mm, left ventricular posterior wall thickness 11 mm), no asynergy zones and ejection fraction 57%. The patient had a bad habit as smoking one pack of cigarettes per day. CAG was performed 48 hours after admission as the patient initially refused to be tested. Results: right-dominant coronary circulation, an oval non-occlusive thrombus of 3.3x2.6 mm with "blurred" contours and small vibrational motions in the distal part of the LCA with the spread to its bifurcation was revealed. No other changes of coronary arteries were found.

Therapy: Heparin 25,000 units IV infusion, then 5000 units SC 4 times per 24 hours; Plavix 300 mg, then continued at 75 mg daily and followed by Zillt 75 mg; Arixtra 2.5 mg SC; Aspirin 125 mg per 24 hours.

CAG repeated 14 days later showed the complete lysis of the thrombus and no hemodynamically significant stenoses of coronary arteries.

A 32-year-old male patient S. was hospitalized with symptoms of acute popliteal artery thrombosis. ECG showed focal changes of the left ventricular anterior wall. Echocardiography revealed signs of local failures of the left ventricular myocardium contractility in the anterolateral and septal walls, hypokinesis of apical segments; troponin was 0.276 ng/mL. After collecting medical history and examination of the patient, small-focal anteroseptal AMI spreading on the apex and the lateral wall of the left ventricle was suspected. Typical pain had happened a month before admission. Bad habits: smoking. CAG showed 65% stenosis of the middle third of the LAD and an oscillating contrast defect of a round shape with clear smooth contours more distally. The other coronary arteries were not changed.

Therapy: Warfarin 5 mg and Heparin 2,500 IU 6 times per 24 hours. CAG performed 9 days later showed the complete clot lysis and unevenness of artery contours without hemodynamically significant stenoses. Taking into account the systemic arterial thrombosis (coronary arteries, arteries of the lower extremities) and the young age of the patient, thrombophilia cannot be excluded.

A 37-year-old female patient K. experienced initial heart pain during fitness classes. The patient was transported to another hospital by ambulance. ECG gave signs of an acute stage Q-wave AMI of the anterior wall, septum and apex spreading to the lateral wall of the left ventricle. Thrombolytic therapy with Actilyse gave no signs of blood flow restoration in the infarct-related artery. After recurrence of retrosternal pain the patient was transported to N. V. Sklifosovsky Institute. ECG showed sinus rhythm, heart rate 85 beats per minute, QS with V1-4, ST-segment elevation in V2-4 up to 2 mm. From medical history: familial hypercholesterolemia, smoking a pack of cigarettes per day.

CAG performed 23 hours after the onset of the disease: right-dominant coronary circulation, the main LCA without hemodynamically significant stenoses, stenosis of the middle third of the LAD and oscillating round shaped thrombotic mass with “blurred” contours more distally, narrowing 90% of the lumen. Blood flow TIMI 3. Intermediate artery, 2nd and 3rd diagonal arterial branches were 70 — 80% narrowed, the RCA had diffuse changes, and the posterolateral artery had 50% stenosis.

Therapy: Plavix 300 mg, then 75 mg per 24 hours; Fraxiparine 0.6 SC 2 times per 24 hours; Aspirin 125 mg per 24hours.

CAG performed 9 days later demonstrated the complete lysis of the thrombus and also sequential 70% and 75% stenoses of the middle third of the LAD. The 38-mm-long drug-eluting stent “Taxus” of 3 mm diameter was implanted and provided a good hemodynamic effect. Blood flow TIMI 3.

A 62-year-old male patient G. had complaints of retrosternal pain, weakness and hyperhidrosis upon arrival. The pain first appeared two days earlier, lasted 7 hours, then recurred. ECG showed sinus rhythm, heart rate 115 beats per minute, pathological Q and ST elevation in V1-4; CPK 283 IU/L, CK-MB 39 IU/L, glucose 9.0 mmol/L. The patient smoked 10 cigarettes per day at that time.

CAG was conducted 48 hours after the onset of the decease (2 hours after hospitalization): right-dominant coronary circulation, the main LCA without any stenoses, occlusion of the LAD in the middle third, 50% stenosis of a large branch of the obtuse margin artery. The RCA was dilated with 50% stenoses, its distal third had three oscillating enlighten segments of a round and an oval shape with a maximum length of 3.8 mm (Fig. 1 a, b).

Mechanical recanalization, balloon dilatation, stenting of the middle third of the LAD and implantation of a bare-metal stent «Liberte» (3 mm diameter, 20 mm length) were performed. Blood flow TIMI 3.

Therapy: Plavix 300 mg, then 75 mg per 24 hours, Aspirin 125 mg per 24 hours. The repeated CAG had not been performed (the patient left the Institute for the further treatment at the place of residence).

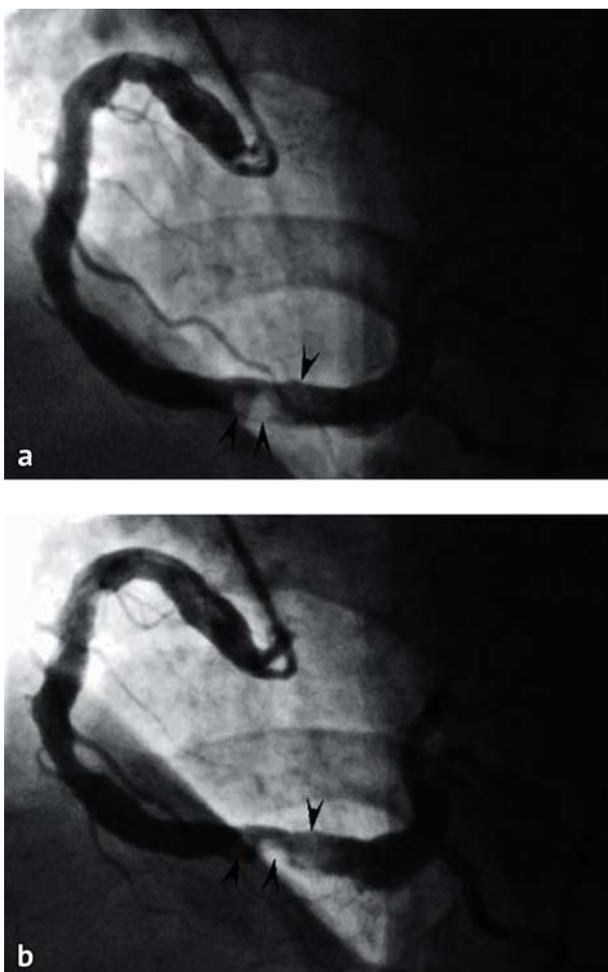


Fig. 1. Coronary angiogram. The right coronary artery is altered with diffuse 50 % stenoses in the mouth and in the middle third. There are three mobile floating thrombi (arrows) in the distal third of the artery.

A 73-year-old male patient P. was hospitalized with a cardiac-cerebral variant of hypertensive crisis complaining of retrosternal pain. The patient had a small-focal lower AMI and was treated in the other medical institution. CAG had not been performed.

CAG (N.V. Sklifosovsky Institute): right-dominant coronary circulation, the main LCA was without any hemodynamically significant stenoses, the middle third of the LAD had two stenoses of 75% and 70%. The circumflex artery was underdeveloped starting from the place where a large obtuse marginal branch arose. The obtuse marginal branch had 90% stenosis

starting from the ostium of the artery. There was a rotating round shaped contrast defect of 1.5 mm diameter distally to the stenosis. The RCA had a stenosis in the proximal third of up to 50% and two 90% stenoses in its distal third. The posterolateral artery had a 50 % stenosis. The therapy included Thrombo ASS 100 mg. The patient refused of the further therapy for family reasons and was discharged.

CONCLUSION

Thrombotic masses forming in coronary arteries are friable and incompact. Lysis may happen spontaneously as well as due to the therapy. CAG may be performed when the lysis of the edges of thrombotic masses is taking place, while the thrombus acquires spherical or oval shape and the "core" of the thrombus still remains unchanged. In 2 cases out of 5 when AMI lasted 24—48 hours, contours of the intracoronary thrombi were indistinct and "blurred", and oscillatory motions occurred with a small amplitude. In 2 cases where the thrombi had existed for 1 and 2 months, and the unknown amount of time in another case, blood clots had clear and smooth contours; they were not attached to the arterial wall and moved with large amplitude, even performing rotational motions.

The repeated CAG was performed in 3 patients. Thrombolytic and antiplatelet therapy was successful in 1 patient, and exclusively antiplatelet treatment – in 2 patients. Even a one-month-existing thrombus lysed under the therapy.

There is an uncertain question of therapy for long-existing intracoronary thrombi. In one patient the thrombus had existed more than two months, and in another case the time of mobile thrombi existence was unknown. These thrombi may possibly cause further complications, requiring constant antiplatelet therapy.

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