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Contents

Successful Combination of Interventional and Drug Treatment of Acute Myocardial Infarction Caused by Acute Occlusion of the Left Main Coronary Artery

D.G. Iosseliani[1], A.G. Koledinsky, V. L. Bashilov, D. V. Istrin, S. P. Semitko, I. Iu. Kostianov, N.V. Kuchkina
Moscow City Center of Interventional Cardioangiology, Moscow, Russia

Acute occlusion of the left main coronary artery (LCA) is considered to be fatal in the vast majority of cases (8). According to some authors, up to 99.5% of the patients with acute myocardial infarction caused by a lesion of the LCA die within the first 24 hours (4). Lesion of the LCA often results in "sudden" death.

According to our data based on assessment of selective coronary angiograms of more than nine thousands of patients with chronic forms of coronary artery disease (CAD) and three thousands of patients with acute myocardial infarction (AMI), chronic occlusion of the left main coronary artery occurred in 0.07% of cases, and acute occlusion occurred in 0.3% of cases. All patients with acute occlusion of the left main coronary artery were admitted to the Moscow City Center of Interventional Cardioangiology (hereafter MCCIC) in cardiogenic shock, which is known to be associated with high in-hospital mortality (5). Causes for such disappointing prognosis in patients with AMI with left main coronary artery lesion are critical failure of perfusion and necrosis of significant portion of the left ventricle, and, consequently, abrupt reduction in cardiac functional capacity. Treatment measures in these patients should be started as soon as possible, because any delay may be literally "deathlike". The measures must be directed towards restoration of failed coronary circulation using pharmaceutical, endovascular or surgical treatment, and towards maintenance of dramatically reduced cardiac pump function. For this purpose, apart from medical agents, indispensable are the methods of assisted circulation, in particular the method of intra-aortic balloon pump, which is virtually the method of choice in case of cardiogenic shock caused by acute left ventricle failure. An important condition for successful treatment of AMI patients with lesion of the LCA is prevention of reperfusion damage of the myocardium which may occur following myocardial revascularization procedures. In this regard, some authors (16), including us, pin their high hopes on the pharmacological protection of the myocardium using so called metabolic cardiocytoprotectors. The studies we performed showed that cardiocytoprotective drugs Neoton and Mexicor administered intracoronary directly after restoration of circulation in the infarction-responsible artery favorably affect limitation of zone of myocardial damage, and thereby improve the functional capacity of the left ventricle. Therefore, it can be assumed that combined application of early (within the first hours of the disease) myocardial reperfusion, assisted circulation (intra-aortic balloon pump) and pharmaceutical protection of ischemic myocardium from reperfusion damage, may help to improve the prognosis in AMI patients with lesion in the left main coronary artery.

The Moscow City Center of Interventional Cardioangiology has the experience with endovascular treatment of six patients with AMI and acute occlusion of the left main coronary artery. In four cases, despite successful restoration of antegrade blood flow using transluminal angioplasty and application of intra-aortic balloon pump (IABP) the attempt to prevent death was unsuccessful. All patients died from progressive left ventricular failure. In two cases the treatment consisted in a combination of a successful endovascular reperfusion procedure on the left ventricle myocardium, intra-aortic balloon pumping, and intracoronary administration of metabolic cardiocytoprotectors, which, in our opinion, allowed saving life to these patients.

As evidence of that, we suggest the following clinical examples for discussion.

Case No. 1

Patient S., 49 years old. Case record No. 895/2003, was brought to the department of cardiac resuscitation of MCCIC on March 06, 2003, at 1.00 p.m. by ambulance, with prolonged (3-hours long) angina attack and ECG pattern of the earliest stage of circular myocardial infarction.

Diagnosis on admission: CAD. Acute Q-wave circular myocardial infarction of LV, March 06, 2003. Cardiogenic shock.

It is known from his medical history, that the patient has had arterial hypertension for 10 years with BP increasing up to 190/120 mm Hg. He has received no regular antihypertensive therapy. He has been smoking about 30 cigarettes a day, denies alcohol abuse, has no evidence of hereditary problems, does not have diabetes mellitus. Of concurrent diseases, he reports stomach ulcer in remission with last exacerbation 5 years ago. Angina attacks started about a year ago, occurring with moderate physical activity (walking 200–250 m). He did not report to doctors on this matter, did not receive continuous anti-anginal therapy, occasionally took

[1] Russia, 101000, Moscow, Sverchkov per., 5
Moscow City Center of Interventional Cardioangiology
Tel. (+7 495) 624 96 36
Fax. (+7 495) 624 67 33
e-mail davidgi@mail.ru
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nitrates to treat the angina attacks. In the morning of March 6, 2003, at 10.00 a.m. he developed an intensive angina attack not resolving after administration of nitrates, whereby the patient called an ambulance. On the arrival of the ambulance team the patient's status was severe, he had hypotension, and his BP was 60/20 mm Hg, the patient's skin was pale, on auscultation there were disseminated moist rales in the lower parts of lungs (up to the scapula angle on both sides). In order to stop the pain, opioid analgesics (Fentanyl 2.0, Morphine 1.0 IV) were administered without any substantial effect. At 12.55 a.m. the patient was brought to MCCIC continuing to receive infusion therapy with cardiotonics (dopamine 8 μg/kg/min).

On admission, the patient was in an extremely severe state. He had skin pallor, mottling, and cold sweat. On auscultation there were weak breathing sounds, moderate amount of moist rales bilaterally. The respiratory rate was 22 per minute. The heart sounds were muffled, no murmurs were heard, pulse at peripheral arteries was very weak. Using dopamine infusion 5-7 μg/kg/min hemodynamics was relatively stabilized, arterial pressure increased to 90/60 mm Hg, and heart rate increased to 88 beats per minute. The liver could be palpated at the rib margin. ECG showed sinus tachycardia. HR was 90 beats per minute. PQ interval was 0.12 seconds, QRS was 0.09 seconds. QT was 0.37 seconds. ECG also showed monophasic elevation of ST segment up to 9 mm in leads V1-V6, reciprocal depression of ST segment up to 3 mm in leads II, III, aVF.

In the intensive care department dopamine infusion was continued at a dose of 8 μg/kg/min. Platelet aggregation inhibitors and anticoagulants were administered according to the standard of care adopted at MCCIC (Thrombo-ASS 325 mg, heparin 100 U/kg bolus + IV drip until activated clotting time (ACT) is over 300 seconds). Taking into consideration prolonged medically intractable pain, early stage of myocardial infarction, and cardiogenic shock, an emergency diagnostic left ventriculography with selective coronary angiography (CAG) was performed in the patient. According to the left ventriculography data, there were extensive dyskinesia of the anterior-apical region of the left ventricle and hyperkinesis of the posterior-basal segment (Fig. 1). Left ventricular ejection fraction was 34%. Left-ventricular end-diastolic volume (LVEDV) was 145.4 mL, and left-ventricular end-systolic volume (LVESV) was 96.3 mL. According to the selective coronary angiography data, occlusion of the LCA was found, without signs of antegrade opacification of the left coronary artery distal to the occlusion site (Fig. 2A). The right coronary artery (RCA) was diffusely affected, with maximum stenosis in its distal third, up to 70% (Fig. 2B). The left anterior descending artery (LAD) and the left circumflex coronary artery (LCx) were contrasted in a retrograde manner with selective contrasting of the RCA.

We explain preservation of the functional capacity of the left ventricle with relatively satisfactory values by availability of good collateral communication between the right coronary artery and the left coronary artery distal to its occlusion (Fig. 2B). During the selective coronary angiography, as well as after it, despite continued active drug therapy, the patient tended to have unstable hemodynamics with hypotension (80/30 mm Hg), for this reason intra-aortic balloon pump was initiated in 1:2 mode. After that, stabilization of hemodynamics was observed, blood pressure successfully increased to 100/60 mm Hg, and HR was 82 beats per minute. After that, PTCA of the left main coronary artery was started. A guiding catheter JL 8F. was placed into the orifice of the LCA, and, using coronary wire and balloon catheter, mechanical recanalization and PTCA of the LCA were performed (Fig. 3, 4A). Right after the first dilatation and appearance of the antegrade blood flow in the LCA system, in order to prevent the reperfusion damage of the myocardium and to preserve viability of peri-infarction ischemic zone of the myocardium, intracoronary exogenic phosphocreatine (ALFA WASSERMANN) was administered to the patient at a total dose of 2 g. according to the method developed in our Center (8).

After that, predilatation of the left main coronary artery bifurcation and LAD orifice was performed, using "U-Pass" balloon, manufactured by Cordis, J&J, 3 mm x 13 mm, with subsequent implantation of the coronary stent "Multilink Tetra", manufactured by "Guidant", measured 3.5 mm x 8 mm under pressure of 14 atm (Fig. 4B).

The control coronary angiogram showed a good result of stenting of the LCA and the LAD orifice; however, it also demonstrated a stenosing

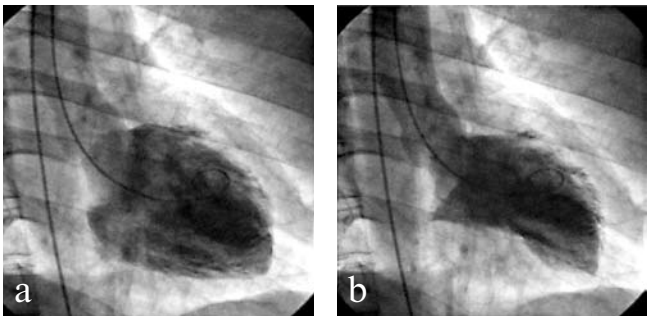

Figure 1. Ventriculography in patient S., 49 years old.
A — left ventricle in diastole; B — left ventricle in systole; akinesis of the anterior lateral and apical segments of the LV is clearly seen.

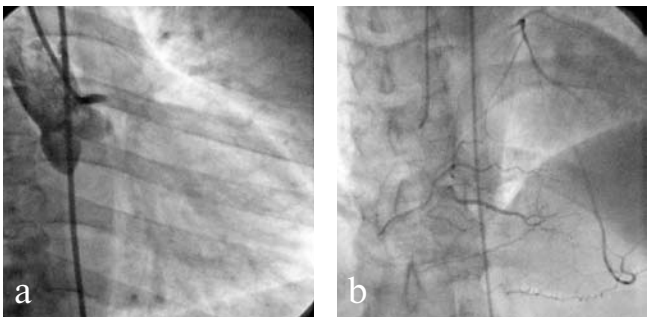

Figure 2. Ventriculography in patient S., 49 years old.
A – occlusion of the LCA; B – angiography of the RCA, good intersystem collaterals in the left coronary artery (LCA) are seen.

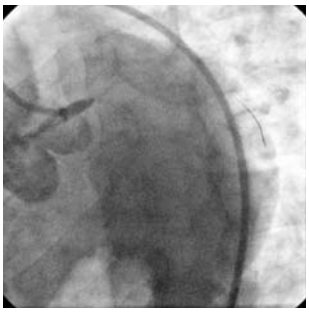

Figure 3. Mechanical recanalization of the LCA.

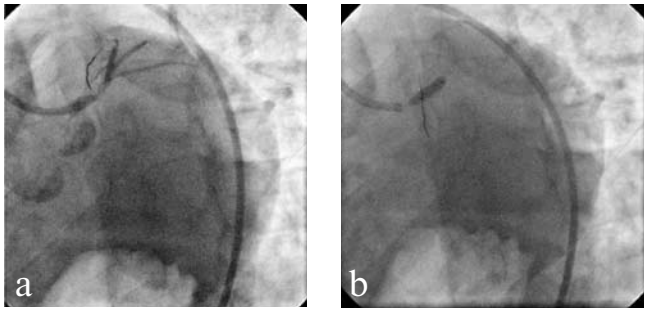

Figure 4. PTCA and stenting of the left main coronary artery.
A — recanalization and predilatation of the occluded segment of the left main coronary artery with "U-Pass" balloon manufactured by Cordis, J&J. Balloon diameter is 3.0 mm, length is 13 mm, dilation duration is 60 seconds;
B — stenting of the left main coronary artery with "Multilink Tetra" stent manufactured by "Guidant", the stent dimensions are 3.5 mm x 8 mm, dilation duration is 45 seconds.

lesion of LCx orifice (Fig. 5). A coronary wire was introduced through the lumen of the stent into the LCx, and PTCA of the LCx orifice was performed using a "U-Pass" balloon (manufactured by "Cordis") 3 mm x 13 mm, with good angiographic result (Fig. 6, 7).

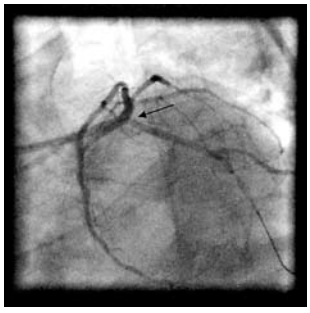

Figure 5. Control coronary angiogram after implantation of the stent into the LCA, the arrow points to the stenosis of LCx orifice.

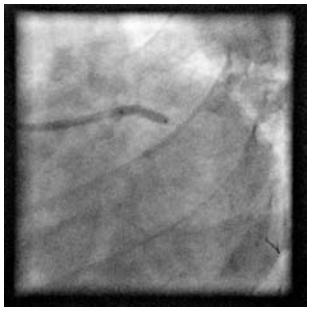

Figure 6. The wire and the balloon are introduced through the lumen of the stent into the LCA, PTCA of the LCx orifice is performed.

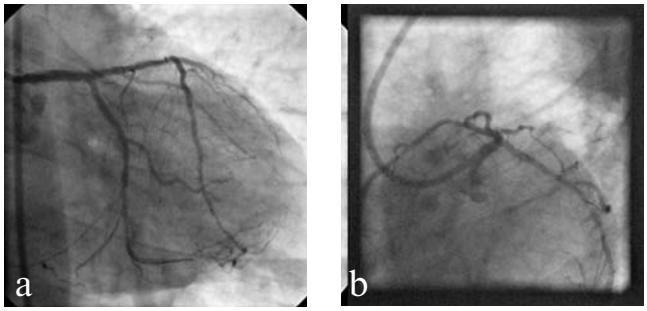

Figure 7. A, B — Control coronary angiography of the LCA in two views (before the end of the procedure).

After the endovascular procedure, the patient had the intra-aortic balloon pumping continued in 1:2 mode, and IV infusion of cardiotonic drugs (dopamine 8 μg/kg/min) was also performed. This being done, hemodynamics remained stable, BP remained at 95/62 mmHg, and HR was 92 beats per minute. The patient was transferred to the intensive care ward.

ECG showed sinus rhythm, obvious positive dynamics defined by reduction of the ST elevation in thoracic leads V1-V6 and reciprocal ST segment depression in leads II, III, aVF by over 50% of the baseline value.

Continuing the intra-aortic balloon pumping for over 18 hours, adequate hemodynamics was successfully maintained with dopamine infusion reduced to 4 μg/kg/min, and the signs of circulatory failure regressed. The patient also received platelet aggregation inhibitors (Ticlid, Thrombo-ASS), anticoagulants (heparin controlled by ACT), and diuretics (Furosemide, Aldactone).

In order to assess the results of the endovascular procedure, he had control selective coronary angiography performed on Day 5 of hospitalization (Fig. 8). It showed a good result of PTCA in all segments of the LCA, antegrade flow was TIMI 3, no angiographic signs of thrombosis or dissection were noted.

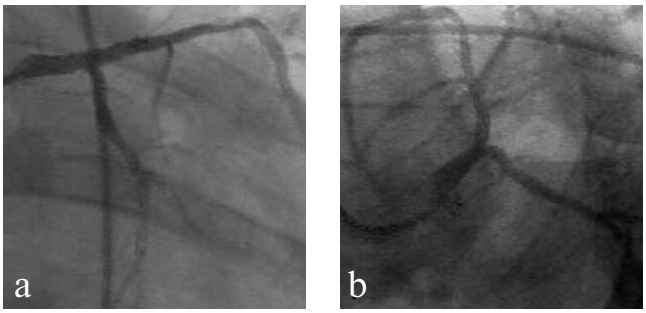

Figure 8. A, B — Data of control coronary angiography of the LCA on Day 5 post procedure.

On Day 5, the patient was transferred from the intensive care unit to the cardiology department in stable condition. On Day 6, the patient's physical activity regimen was extended and ambulation was allowed. Further, there was no recurrence of angina pain or signs of circulatory failure. By the time when the patient was discharged from the hospital, on Day 14, his state was fair, the skin and visible mucous membranes had normal color and moisture level. Vesicular breath sounds could be heard on lung auscultation, no rales were noted. Respiratory rate was 18 per minute. BP was 120/70 mm Hg. HR was 82 bpm. Heart sounds were mildly muffled, rhythmic, no murmurs were identified. The liver edge was at the rib margin. Neurological status was unremarkable.

ECG showed regular sinus rhythm. HR was 79 bpm. PQ interval was 0.13 seconds, QRS was 0.09 seconds. QT was 0.38 seconds. ST segment returned to the isoelectric line, T wave reversed in standard and thoracic leads. The echocardiography showed an improvement in global and segmental LV contrac-

tility, hypokinesis of the apex and the posterior wall of the left ventricle. Ejection fraction (EF) 53%, EDV 5.8 cm, ESV 4.0 cm, IVS thickness 11 mm, left ventricle posterior wall thickness (LV PWT) 12 mm. Cardiac valves were without apparent abnormalities. 24 hour ECG monitoring registered stable sinus rhythm with average rate of 72–94 bpm. ST segment was at the isoelectric line.

Patient recommendations included platelet aggregation inhibitors (Thrombo-ASS, Ticlid), anti-angina agents (Cardiket) and diuretics (Triampur), along with angiotensin converting enzyme (ACE) inhibitors (Perindopril at 4 mg/day), statins (Simvastatin), β-blockers (Carvedilol). On March 19, 2003, the patient was discharged from our clinic in stable state.

Case No. 2

Patient B., 56 years old. Case record No. 3997/2004, was brought to the department of cardiac resuscitation of MCCIC on October 17, 2004, at 1.05 p.m. by ambulance, with prolonged (9-hours long) anginal attack and ECG pattern of acute phase of transmural circular myocardial infarction.

Diagnosis on admission: CAD. Acute Q-wave anterior-septal-apical-lateral myocardial infarction, October 27 2004 (lasting for 9-hours). Essential hypertension, stage III. Circulatory insufficiency, class 2A. Pulmonary edema.

History: Patient noted having high blood pressure for a long time (more than 20 years), with maximum values up to 210/110 mm Hg, but received no regular treatment for his arterial hypertension. He noted no other diseases. Ten days before admission to the hospital he noted restrictive retrosternal pain for the first time in his life, but did not report to doctor. Present deterioration started early in the morning of October 27, 2004, when the patient felt intensive retrosternal pains, which achieved their peak at 10.30 a. m., and about that time he called an ambulance. On arrival of the ambulance team, the patient's status was severe, BP was 50/20 mmHg. Along with a significant angina state he had pale skin, on auscultation there were multiple moist rales in all parts of lungs. In order to stop pain, opioid analgesics were administered (morphine, 2.0 IV, with positive effect), and infusion cardiotonic therapy was initiated (dopamine 8 μg/kg/min), and this being done, the patient was admitted to the MCCIC at 1.05 p.m.

On admission, the patient's condition was judged as extremely severe. He had pale skin and cold sweat. On lung auscultation, the breathing sounds were weak, with a lot of coarse bubbling rales in all parts of lungs. Respiratory rate was 28 per minute. Heart sounds were muffled, no murmurs were heard, pulse at peripheral arteries was thready. While dopamine was being infused, HR was 108 beats per minute. BP was 105/70 mmHg. The liver was at the rib margin. Neurological status was unremarkable.

ECG revealed sinus tachycardia, normal position of the electrical axis of the heart. PQ interval was 0.12 seconds, QRS was 0.09 seconds. QT was 0.4 seconds. Monophasic elevation of ST segment up to 6 mm was seen in leads I, aVL, V1 V6, and reciprocal depression of ST segment up to 4 mm was seen in leads II, III, aVF.

In the intensive care department dopamine infusion was continued at a dose of 10 μg/kg/min. Platelet aggregation inhibitors and anticoagulants were administered (Thrombo-ASS 100 mg, heparin 100 mg/kg bolus with subsequent IV drip of this drug using ACT values for control). Taking into consideration the long duration of pain, early stage of the myocardial infarction, and clinical signs of cardiogenic shock, an emergency diagnostic left ventriculography with selective coronary angiography was performed.

Left ventriculography showed dyskinesia of the anteroapical region of the left ventricle and hypokinesis of the diaphragmatic and posterobasal segment (Fig. 9). Left ventricular ejection fraction was 24%. Left-ventricular end-diastolic volume was 141.8 mL, and left-ventricular end-systolic volume was 107.5 mL.

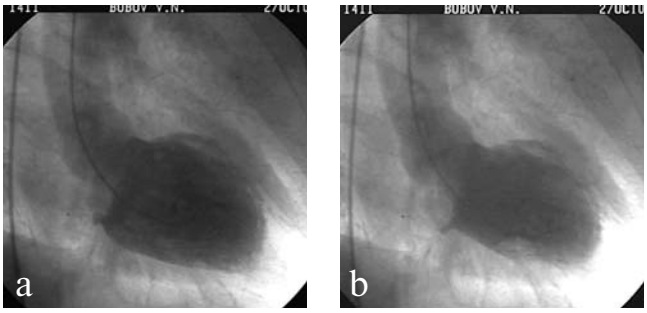

Figure 9. Left ventriculogram of patient B., 49 years old.
A — left ventricle in diastole;
B — left ventricle in systole, akinesis of the anterior lateral and apical segments of the LV is seen.

Selective coronary angiography : Left main coronary artery is occluded. (Fig. 10A). RCA has no hemodynamically significant stenosis (Fig. 10B).

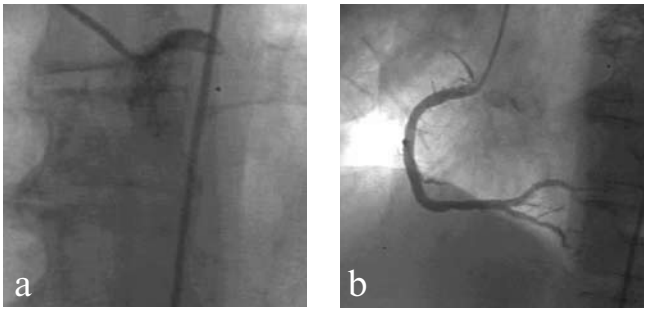

Figure 10. Selective coronary angiogram of patient B., 49 years old
A – occlusion of the left main coronary artery;
B – selective coronary angiogram of the RCA in the left oblique projection.

Immediately following the diagnostic coronary angiography , a guiding catheter JL 8F. was placed into the orifice of the LCA, and the mechanical recanalization procedure with PTCA of the left main coronary artery was performed (Fig. 11, 12A). Simultaneously to the recanalization, metabolic cytoprotector Mexicor (EcoFarmInvest) was administered intracoronary at a total dose of 200 mg, with

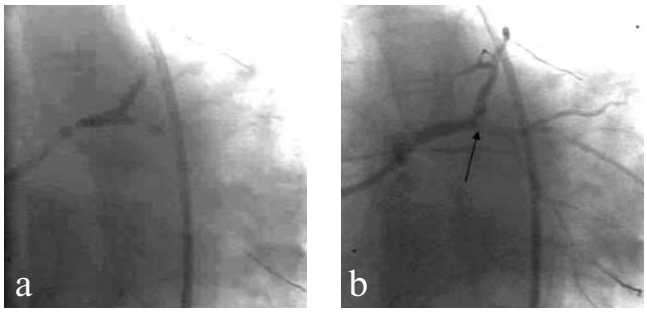

Figure 12. Balloon angioplasty of the left main coronary artery and the orifices of the LAD and LCx
A — transluminal angioplasty of the left main coronary artery and the orifices of LAD and LCx using kissing technique with two "U-PASS" balloons manufactured by "Cordis", 2.0 mm in diameter and 20 mm in length.
B – Result of the transluminal angioplasty. The arrow indicates the site of dissection of the LCx orifice.

subsequent IV infusion at 600 mg/24 hours for 5 days and then switched to oral administration of 300 mg/day for 2 months.

Following the balloon angioplasty of the bifurcation of the left main coronary artery and the orifices of LAD and LCx using "kissing" technique with balloons 2.0 mm x 20 mm, threatening dissection of the LCx orifice was noted (Fig. 12B). Therefore, an "R-stent evolution" coronary stent 3.5 mm x 28 mm was implanted into the LCx orifice, under pressure of 12 atm. Control coronary angiogram, showed a good result of stenting of the left main coronary artery and the LCx orifice (no residual stenosis, no dissection, antegrade blood flow TIMI 3). There was, however, a stenosis of the LAD orifice (Fig. 13). Therefore, a coronary wire was introduced through the lumen of the stent into the distal portion of LAD, and angioplasty of the LAD orifice was performed with a "U-Pass" balloon (manufactured by "Cordis") 3 mm × 20 mm, with good angiographic result.

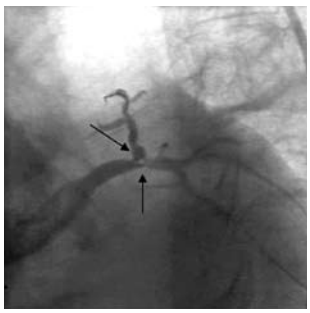

Figure 11. The figure shows stages of the mechanical recanalization and balloon predilation of the left main coronary artery. The arrows indicate the sites of residual stenosis in orifices of LAD and LCx.

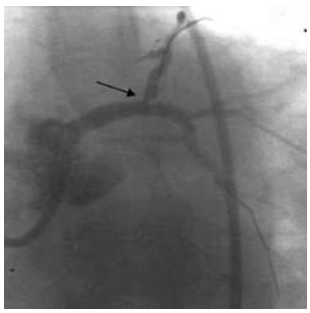

Figure 13. Result of LCx stent placement, the arrow indicates LAD orifice stenosis.

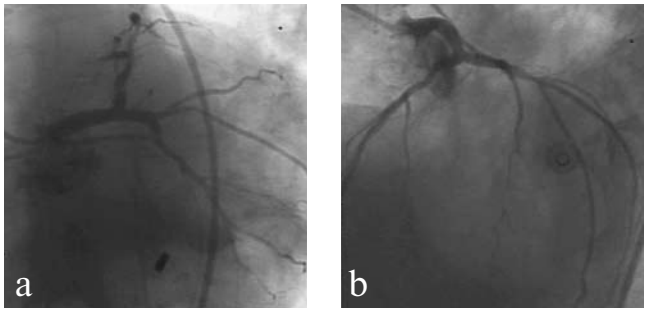

Figure 14. A, B — Control coronary angiography of the LCA in the left caudal and left oblique projections.

Despite good results of the endovascular procedure, complete restoration of the antegrade flow in the system of LCA, and infusion of cardiotonics (dopamine 20 μg/kg/min during the procedure), the patient had unstable hemodynamics with tendency to hypotension (80/30 mmHg), which was probably due to reduced cardiac output secondary to extensive damage of the myocardium. Thereby, after the endovascular procedure was completed, the patient had balloon pump placed into his aorta distal to the left subclavian artery under fluoroscopy guidance, and intra-aortic balloon pumping was started in 1:2 mode. After that, hemodynamics was stabilized, blood pressure increased to 90/60 mm Hg, and HR was 93 bpm. The patient was transferred to the intensive care unit.

ECG showed sinus rhythm, obvious positive dynamics defined by reduction of the ST elevation in leads I, aVL, V1-V6 and reciprocal ST segment depression in leads II, III, aVF by over 50% of the baseline value.

Within 24 hours dopamine infusion was successfully reduced to 4 μg/kg/min, and signs of circulatory failure regressed. In the intensive care unit, the patient received drug therapy with platelet aggregation inhibitors (Ticlid, Thrombo-ASS), anticoagulants (heparin controlled by ACT), diuretics (Furosemide, Aldactone).

On Day 9, the patient was transferred from the intensive care unit to the cardiology department in stable condition. On Day 11, the patient's physical activity regimen was extended and ambulation was allowed. Further, there was no recurrence of angina pain or signs of circulatory failure. By the time when the patient was discharged from the hospital, his state was fair, the skin and visible mucous membranes had normal color and moisture level. Harsh breath sounds could be heard on lung auscultation, no rales were noted. Respiratory rate was 16 per minute. BP was 128/76 mmHg. HR was 80 bpm. Heart sounds were mildly muffled, rhythmic, no murmurs were identified. The liver edge was at the rib margin. Neurological status was unremarkable.

ECG revealed sinus rhythm. HR was 80 bpm. PQ interval was 0.12 seconds, QRS was 0.09 seconds. QT was 0.41 seconds. Positive dynamics was seen as follows: ST segment returned to the isoelectric line, T wave reversed in standard and thoracic leads. Echocardiography revealed moderate dilation of the heart cavities, end-diastolic dimension was 6.4 cm, end-systolic dimension was 5.0 cm. End-diastolic volume was 211 cm3, and end systolic volume was 121 cm3. There was an improvement in global and segmental LV contractility: hypokinesis of the apex, akinesis of the anterior wall, akinesis of the lateral wall in the middle and apical thirds, left ventricle ejection fraction was 42%. Mitral valve: the cusps

moved in different directions, Doppler echocardiography revealed grade 1-2 mitral regurgitation into the left atrium.

Twenty-four hour ECG monitoring registered stable sinus rhythm with average rate of 72–94 bpm. ST segment was stable.

Pharmaceutical treatment included platelet aggregation inhibitors (Thrombo-ASS, Ticlid), anti-angina agents (Cardiket), diuretics (Triampur), ACE inhibitors (Perindopril 2 mg/day), statins (Simvastatin), B-blockers (Carvedilol). On Day 18 from admission into MCCIC (November 15, 2004), the patient was discharged from our clinic in stable state.

In conclusion, these examples show that despite severe state and unfavorable prognosis in patients with acute occlusion of the LCA (6, 7), combined use of endovascular procedures of myocardial reperfusion, intra-aortic balloon pumping and intracoronary pharmacological myocardial protection may be effective. In our opinion, intracoronary administration of metabolic cytoprotectors contributed to reduction of reperfusion damage to cardiomyocytes.

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Novel Device for Recanalization of Chronic Total Occlusions

*A.M. Babunashvili[1], A, D.P. Dundua, Z.A. Kavteladze, D.S. Kartashov, V.V. Korobov**
Center of Endosurgery and Lithotripsy, Moscow, Russia
** Republic Clinical Hospital №2, Ministry of Public Health of Tatarstan Republic, Kazan*

Introduction

Chronic total occlusions (CTO) of coronary arteries (CA) still are considered as ones of the most complicated atherosclerotic lesions for percutaneous interventions . In spite of some progress in the development of new devices and experience accumulation, the success rate of endovascular procedures for occlusive lesions of CA does not exceed 80–85%. At the same time the rate of successful percutaneous interventions in cases of atherosclerotic stenoses reaches 99–100%. The main reasons for the failure of recanalization in CTO are as follows:

a) Occlusion cannot be passed with metallic and/or hydrophilic guidewires .

b) Guiding catheter can not be adequately engaged at the orifice of recanalized CA.

c) Guidewire sometimes passes subintimally and can not be redirected into a distal part of true artery lumen beyond the occluded site.

d) Balloon catheter for predilatation of the occluded segment can not be passed even after successful guidewire recanalization.

It is necessary to visualize collateral blood circulation and the lumen of occluded coronary artery distal of the occlusion segment to make sure that the tip of a quidewire is inside the true artery lumen beyond the occluded segment. Though the second arterial access and catheterization of both coronary arteries are often required.

We introduced a novel device that may solve some of the abovementioned problems, increase safety and controllability of the endovascular recanalization of CTO.

The device description

The device is created on the base of a monorail dilatation balloon catheter. Of note, the balloon segment of any catheter enlarges the diameter (profile) of its distal end and in some cases even the balloon catheter with minimal profile (1.5F) can not be passed through the occluded site. For purpose of decreasing the catheter profile we have removed its balloon segment carefully with a scalpel. The resulting dual-lumen catheter had one individual lumen for inserting over a wire guide and the other

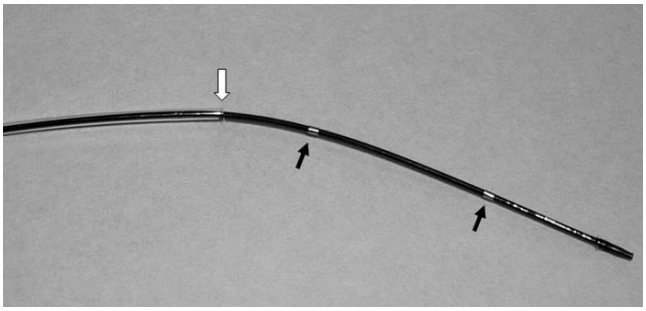

Figure 1. General view of the distal segment of the device. After removal of a balloon segment the channel for filling up a balloon remains open (white arrow) and contrast medium can be injected through it. At a distal segment beyond that point a long and thin part (~1.5F) with a V-shaped spout forms, facilitating better penetration of occluding material. Markers of the balloon segment are pointed with black arrows.

(balloon) lumen connected with catheter pavilion and remained open. This enabled injecting contrast medium and thereby controlling a position of the device tip in an artery lumen (Fig.1). Usually the profile of a distal part beyond a balloon segment of the majority of modern catheters is 0.018–0.020 inches (0.4–0.5 mm), which is comparable with a diameter of metallic coronary guidewires used for recanalization. After removal of a catheter balloon part this segment lengthened for 12–20 mm. Hereby, we have obtained ultra-low profile, relatively long (20–30mm) active part of a catheter for better penetration of a compact occluding material. Besides, it is important that it became possible to opacify a created tunnel

Table 1. Clinical and angiographic data of the patients

Sex	Age	Artery	Duration of the occlusion, months**	Length of the occlusion, mm*	FC by CCS	Diabetes	EF <0.5
M	65	RCA	24	64	II	–	–
M	61	RCA	18	80	II	–	–
M	51	OMB	8	34	III	+	–
F	54	RCA	36	72	III	+	+
F	52	RCA	54	84	II	–	–
M	60	LAD	12	42	III	+	+
M	52	LAD	10	46	II	–	–
M	52	RCA	22	36	III	–	–
M	64	RCA	4	17	III	–	+
Mean value			20.8 ± 6.9	54.9 ± 3.8	–		

Note: * measured by digital computer angiography (DCA);
** approximate evaluation based on the medical history;
FC by CCS — functional class of angina pectoris according to the Canadian Cardiac Society classification;
EF — ejection fraction; RCA — right coronary artery;
OMB CA — obtuse margin branch of circumflex artery;
LAD — left anterior descending artery.

[1]A.M. Babunashvili
Center of Endosurgery and Lithotripsy
Department of cardiovascular surgery.
Moscow, 111 123, Shosse Entuziastov, 62
Tel. (495) 305 34 04
Fax(495) 305 69 35
e-mail: vlesten@gmail.com
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and verify catheter and guidewire insertion into a true lumen of recanalized artery.

Clinical cases

We used the novel device for recanalization of chronic CA occlusions in 9 patients (Table 1). A good visualization of a distal segment of the occluded CA by intercoronary collaterals have been noticed in all cases.

In all 9 cases the recanalization of the occlusion was first attempted by using a metallic guidewire (hydrophilic guidewires Shinobi, Pilot or Asahi family), but if passage of the low profile balloon catheter failed we tried to succeed by new device. Using it we managed to verify definitely that the catheter was located inside the true artery lumen and to create primary channel for subsequent passage of balloon catheters or stents in order to complete the procedure (Fig.2). In the majority of cases it was not technically difficult to pass the catheter through the occluded zone due to low profile of its distal segment. Only in two cases we had to use guiding catheter deep intubation into the CA lumen to secure adequate passage of the catheter.

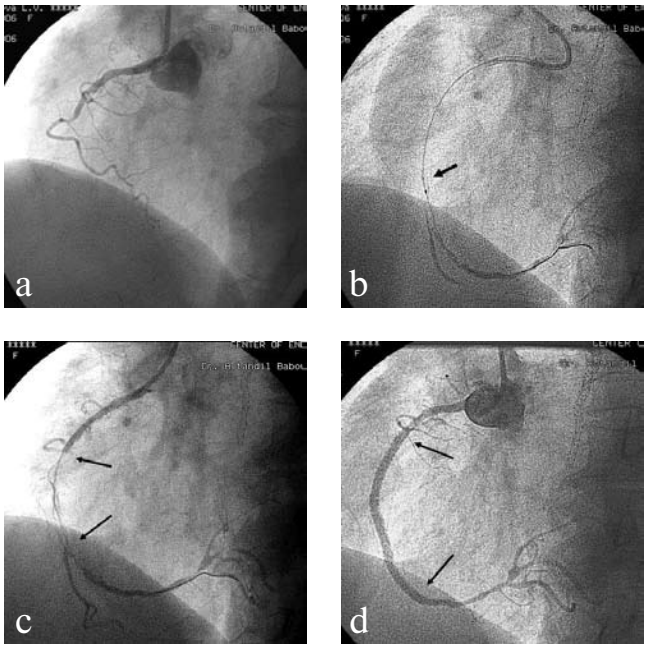

Figure 2. Coronary angiograms of a female patient aged 62:
A – Status before recanalization. Occlusion in the middle segment of the RCA. Distal lumen can be filled only through intercoronary collaterals;
B – After penetration of the occlusion through the channel for a balloon segment (pointed with arrow) with the recanalization device the angiogram of the post-occlusion artery segment has been made. It shows that the catheter tip is inside the true lumen.
C – Coronary angiogram after extraction of the device has revealed formation of a thin channel (pointed with arrows) in the occluded RCA segment with antegrade filling of the distal lumen of the artery;
D — Three drug-eluting stents were passed and implanted into the occlusion zone by "stent-in-stent" method with good immediate angiographic result. No additional dilatation was required. Stented segment is pointed with arrows.

Discussion

The suggested device is easy to make and does not require additional expenses. The device is based on "routine" single-rail balloon catheters manufactured by well-known brands. The described construction provides safe manipulation for a recanalization of occluded CA. It is known that in 10–15% of recanalization failures guidewire goes into a "false" lumen of an artery distal segment beyond the occlusion site (into a subintimal space, and in the worst case a perforation of an artery wall can occur).

Only three ways to verify threading a catheter through a true artery lumen are known for today:

1. Visualization of a distal segment of the occluded artery by opacification through collaterals;
2. Inserting a thin microcatheter (Transit type or perfusion catheter 3F) over a long (300cm) guidewire beyond the occlusion zone and visualization of a distal segment by injecting contrast medium through catheter lumen;
3. Intracoronary ultrasonic examination to detect the position of a quidewire and a catheter in the occluded and/or more distal segment.

It is important to note that the position of subintimally passed guidewire under fluoroscopic control coincides with suggested anatomic path of the distal segment of recanalized artery beyond the occlusion site (Fig.3). For this reason, to make sure that the tip of a guidewire is inside the true artery lumen it is necessary to catheterize the orifice of another CA (if intercoronary collaterals exist), that makes the procedure longer and much more complicated. This requires puncture and catheterization of another access artery (femoral or radial), therefore increasing the risk of bleeding and other complications associated with catheterization of peripheral artery.

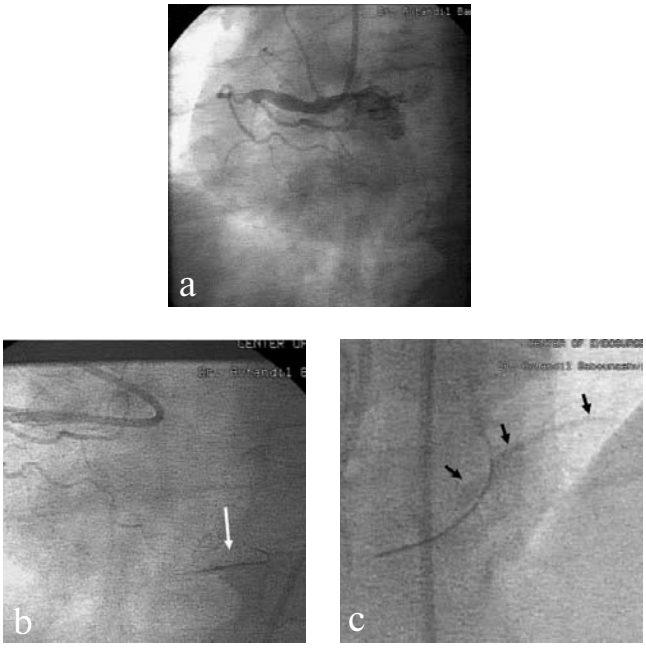

Figure 3. Coronary angiograms of a female patient aged 72:
A — RCA occlusion in the proximal segment with a guiding stump;
B – Hydrophilic guidewire is passed through the long occlusion. The location of the radio-opaque tip of the guidewire (pointed with arrow) coincides with suggested angiographic picture of the distal lumen;
C – Additional angiography in another view distinctly shows subintimal position of the guidewire tip. True lumen is pointed with arrows.

Intracoronary ultrasound control of the guidewire and catheters passage through the true channel

of the occluded segment proved to be sufficiently effective in Colombo and Tobias studies. However, intracoronary ultrasound examination makes recanalization procedure significantly more complicated and expensive, and is technically applicable only in a limited number of cases.

For the same purposes over-the-wire balloon catheter (over the wire) can be used. After advancing the catheter tip to a distal segment beyond the occlusion site the guidewire should be removed from the central lumen and contrast medium injected. But first of all, a profile of dual-lumen balloon catheters is always larger then that of single-rail ones, and secondly, the body of monorail catheter unlike dual-lumen catheter is made out of a thin hollow metallic tube, which increases its rigidity and radial stability. This is especially important when passing through "old", compact occluding masses. Thus, besides the opportunity to control the position of the catheter tip in a true lumen of artery, the new device provides adequate support while passing through chronic occlusive lesions. Furthermore, manipulations with monorail catheters require just a short guidewire, they are much easier to perform and usually can be done by one operator, whereas operations with dual-lumen balloon catheters (or other long thin catheters of Transit type) require a long guidewire and an assistant. Naturally, single operator is able to provide better coordination of manipulation when using a monorail catheter.

In 3 of 9 cases successful application of this device resulted in forming a tunnel about 1 mm in diameter in occluding material (Fig.4), that enabled stenting without preliminary balloon dilatation and saving an expensive ultra mini-profile balloon catheter. Finally, making these devices out of hydrophilic balloon catheters (like that manufactured by Acrostak Company, Switzerland, or Arashi catheter, manufactured by Terumo Company) will improve the penetration ability of the device.

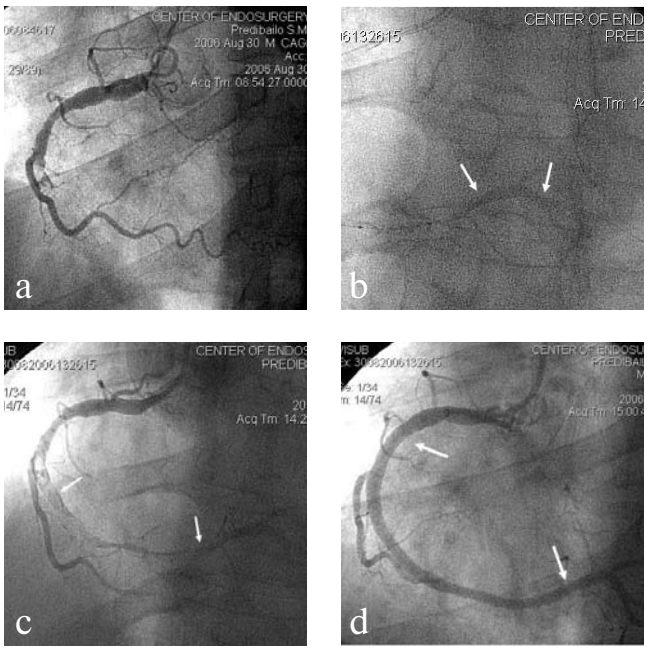

Figure 4. Coronary angiograms of a male patient aged 52:
A – Chronic RCA occlusion in the middle segment with filling of distal lumen only through intercoronary collaterals;
B – After penetration of the occluded vessel segment with the device. On the coronary angiogram distal RCA lumen (pointed with arrows) is clearly visualized through the open balloon channel. This confirms that the catheter tip is inside the true lumen;
C – After the extraction of the recanalization device a thin tunnel (pointed with arrows) in the occluded segment has formed. This tunnel facilitates passage of balloon catheters for additional predilatation.
D – After the balloon predilatation three drug-coated stents (pointed with arrows) have been implanted into the occluded RCA segment with good immediate angiographic result.

Late Percutaneous Coronary Interventions for Left Ventricular Postinfarction Aneurysms

V.I. Ganyukov[1], R.S. Tarasov, N.I. Susoyev, I.N. Shigantsov, A.A. Shilov, E.A. Levchenko, M.V. Demina, I.Yu. Bravve

Novosibirsk Cardiological Clinic, Novosibirsk, Russia

Keywords: late percutaneous coronary intervention, left ventricular aneurysm, myocardial viability, left ventricular remodeling.

Abbreviations:

LVA – left ventricular aneurysm
CAD – coronary artery disease
MI – myocardial infarction
EDV – end-diastolic volume
ESV – end-systolic volume
CABG – coronary artery bypass grafting
LAD – left anterior descending artery
SV – stroke volume
EF – ejection fraction
CHF – congestive heart failure
PCI – percutaneous coronary intervention

Introduction

Postinfarction aneurysm is one of the most severe and common types of left ventricular (LV) remodeling. As a rule, patients after Q-wave myocardial infarction (MI) causing left ventricular aneurysm (LVA) have hemodynamically significant coronary lesions associated with an increased risk of repeated MI and arrhythmic death. An optimal way to prevent or reduce LV remodeling is the reperfusion of the infarct-related artery during acute-stage MI, which preserves the LV function and improves the survival (5). Late reperfusion of the infarct-related artery is also crucial, as it decreases the severe ventricular remodeling, however, as compared to early reperfusion, it results in delayed restoration of ventricular function and geometry (9).

Myocardial region perfused from the infarct-related artery can have mosaic structure and include viable myocardium within the area of transient ischemia, scar changes, stunned myocardium and hybernating myocardium (15). Even the late reperfusion may improve the cardiac hemodynamics provided that there is substantial volume of hybernating myocardium within the necrotic or scar area.

Treatment of patients with LVA is believed to be the prerogative of open-heart surgery, however, an optimal timing for it is thought to be 4-6 months after MI (4). Late open surgery is a reasonable method, as it allows for LVA repair to be performed on the well-developed scar area and prevents perioperative complications associated with the acute-stage MI (2). One should also consider the fact, that the coronary artery bypass grafting (CABG) has significantly lower rate of hospital mortality as compared to CABG performed during acute-stage or subacute-stage MI. Nevertheless, in certain cases the myocardial reperfusion can not be postponed due to the high risk of unfavorable events. In this situation the PCI as a minimally invasive procedure, which doesn't necessitate cardiopulmonary bypass and pulmonary ventilation, can be performed earlier and is associated with lower risk of perioperative complications, apparently providing lower rate of unfavorable events before the aneurysmectomy is conducted.

Despite the extensive use of endovascular surgery for the treatment of CAD, the aspects of indications, eligibility, efficacy, safety and prognosis of late PCI for LVPA have been virtually disregarded in the literature and have indisputable research and practical importance.

The purpose of the study was to determine the role of late percutaneous coronary interventions (PCI) performed 4-8 weeks after myocardial infarction (MI) in patients with MI causing left ventricular aneurysms (LVA).

Materials and methods

Immediate and long-term outcome of the late PCI performed 4.8 ± 1.7 weeks (median 4 weeks) after MI with implantation of bare metallic stents into the LAD were analyzed in 10 patients with Q-wave anterior MI complicated by LVPA (Group I). The control group comprised 7 LVPA patients 8.0 weeks after MI, who received medical therapy (Group II). All subjects were men of similar age, with similar clinical data (see Table 1). The study groups didn't include diabetic patients. One patient in each group had second MI (10% and 14% in Groups I and II, respectively), whereas the majority of MI patients with LVA had their first MI. Hypertension was found in 8 patients in Group I (80%) vs 3 patients in the control group (43%). All patients had CHF of NYHA class II–III.

Postinfarction angina was the only clinical sign, which was significantly different between the study groups at baseline. Postinfarction angina was found in 7 patients (70%) in Group I vs 1 patient (14%) in Group II ($p < 0.05$). The angina suggested the presence of viable ischemic myocardium (1). Along with the angiographic data, this sign was taken into account when considering late PCI on the infarct-related artery.

Coronary angiography showed hemodynamically significant (> 70%) LAD stenosis in all patients with

[1]V.I. Ganyukov
8, Bld.6, ul. Zalesskogo,
Novosibirsk 630047, Russia
Tel.(383) 216-55-37
Fax (383) 226-29-71,
e-mail ganyukov@mail.ru
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Table 1. Clinical data of patients.

	Group I (n = 10)		Group II (n = 7)		
Clinical values	n	%	n	%	p
Men	10	100	7	100	NS
Age	54.3±8.32		51.28±11.04		NS
History of MI	1	10	1	14	NS
Hypertension	8	80	3	43	NS
Diabetes	0	0	0	0	NS
NYHA II–III CHF	10	100	7	100	NS
Postinfarction angina	7	70	1	14	< 0.05

half of Group I patients (n = 5) having LAD occlusion (see Table 2). Single-artery coronary lesions were found in 80% of cases (n = 8) (see Table 2). LAD occlusion was found in 57% of patients in Group II (n = 4) with single-artery lesions found in 57% (n = 4). The patients were distributed according to the level of LAD lesion as follows: half of the Group I patients had proximal stenosis, the remaining 50% (n = 5) had stenosis of the LAD middle portion. In control group proximal lesions were found in 57% of patients (n = 4), whereas three patients (43%) had middle portion stenosis.

Table 2. Angiographic findings in patients.

	Group I (n = 10)		Group II (n = 7)		
Angiographic data	n	%	n	%	p
Single-vessel disease	8	80	4	57	NS
> 70% LAD stenosis	10	100	7	100	NS
LAD occlusion	5	50	4	56	NS
Proximal LAD stenosis	5	50	4	57	NS
Middle LAD portion	5	50	3	43	NS

At baseline left ventriculography showed signs of postinfarction LVA and decreased LV contractile function with mean LVEF of 34.2±7.2% in Group I vs 35±10.8% in Group II (p>0.05) (see Table 3). The groups had similar baseline values of the end-diastolic volume (EDV), end-systolic volume (ESV) and LV stroke volume (p>0.05).

Table 2. Angiographic findings in patients.

	Group I (n=10)		Group II (n=7)		
Volumetric values	n	%	n	%	p
LVEF		34.2±7.2		35±10.8	NS
LV EDV	186.3±33.9 ml		254±64.5 ml		NS
LV ESV	123.7±32.7 ml		201.4±84 ml		NS
LV SV	62±12.55 ml		78.6±22.7ml		NS
LV aneurysm	10	100	7	100	NS

Coronary angiography was performed according to Judkins procedure (8) using PHILIPS — Integris BH 3000 two-aspect cardiovascular angiography unit. Evaluation of the hemodynamic values and Holter ECG was performed using Midas 5000 physiological station (HELLIGE). Angiomat infusor was used during ventriculography. The data were archived on CD using CD-MEDICAL archiving system (PHILIPS).

All Group I patients underwent late PCI with implantation of bare metallic stents into the infarct-related artery (LAD). Heparin administration during PCI was adjusted by activated thrombin time measured with the use of an automatic coagulation timer.

Immediate outcome of the PCI was assessed on the basis of the immediate success rate (target vessel reperfusion with < 10% residual stenosis) and hospital complications, such as death, repeated MI or CABG.

The long-term results were assessed on the basis of clinical findings, as well as of the results of ventricular and coronary angiography performed 7.9 ± 4.5 months after PCI (median 6.0 months). We analyzed the clinical state of patients, cardiac hemodynamics, signs of in-stent restenosis and further surgical strategy in these patients.

Statistical analysis was performed using ANOVA, Pirson's test was used to detect correlation (STATISTICA for Windows, Stat. Soft, Inc., 1984–2001).

Results and discussion

Late PCI after MI were successful in 100% of Group I patients. In 80% of them the revascularization was complete. There were no deaths, repeated MI or the need for CABG during hospitalization.

In the long-term period after the late PCI (at 6.2 ± 1.68 months) Group I patients had clinical improvement. At follow-up assessment 90% of patients (n = 9) had stable clinical course of CAD with grade 0–I angina of effort (Canadian classification) suggesting successful reperfusion and the presence of viable myocardium within the territory of the infarct-related artery at baseline. Only a single patient had signs of unstable angina due to the in-stent restenosis. Six (6) patients (86%) in Group II had grade II–III exertional angina at the follow-up assessment, 1 patient (14%) had progressive angina.

There were no deaths or repeated MI in the long-term period in both study groups (see Table 4). The severity of congestive heart failure (NYHA) at the follow-up assessment corresponded to NYHA II in 100% of Group II patients vs 86% in control group. One patient in Group II had severe heart failure corresponding to NYHA IV.

One of the most important general parameters describing LV functional state is the total LVEF having high prognostic value in such patients both for natural course of the disease and for the risk of surgery (1). In the long-term period after the late PCI In the long-term period after the late PCI Group I patients had an increase in the global LV contractile func-

Table 4. Clinical findings in the long-term period.

	Group I (n = 10)		Group II (n = 7)		
Clinical findings	n	%	n	%	p
Grade 0-I exertional angina	9	90	0	0	NS
Grade II-III exertional angina	0	0	6	86	NS
Progressive angina	1	10	1	14	NS
Repeated MI	0	0	0	0	NS
Death	0	0	0	0	NS
NYHA I-II CHF	10	100	6	86	NS
NYHA III-IV CHF	0	0	1	14	NS

tion (see Table 5). Thus, the mean LVEF measured by ventriculography increased from 34.2% ± 7.2 to 45.5% ± 11.6 (p = 0.003). Patients with EF value approximating 50% are known to have no additional risk factors associated with decreased myocardial contractile function (1). Patient on medical therapy after MI (Group II) had global LV contractile function well below the normal range both at baseline and at the follow-up assessment (35 ± 10.8 and 37 ± 14.7%, respectively). Apparently, the process of LV remodeling is not completed 4.8 ± 1.7 weeks after MI and event in the long-term period (provided that there's viable myocardium within the territory of the infarct-related artery) the LV function can be improved. Published data suggest, that the period of restoration can take time necessary for tissue regeneration and elimination of cellular structure damage (14). Therefore, the more severe is the myocardial dysfunction, the longer is the restoration period (12).

Table 5. Long-term angiographic outcome.

	Group I (n = 10)			Group II (n = 7)		
Parameter	at baseline	control	p	at baseline	control	p
LVEF (%)	34.2 ± 7.2	45.5 ± 11.6	0.003	35 ± 10.8	37 ± 14.7	NS
LV EDV (ml)	186.3 ± 33.9	174.6 ± 37.1	NS	254 ± 64.5	220.6 ± 56.9	NS
LV ESV (ml)	123.7 ± 32.74	97.2 ± 40.36	0.017	201.4 ± 84	167.3 ± 76.3	NS
LV SV (ml)	62 ± 12.55	76.6 ± 16.6	0.03	78.6 ± 22.7	77.88 ± 24.2	NS

The use of quantitative left ventriculography have provided evidence, that the risk of death increased parallel to the increase of LV diameter (6). Published data indicate, that the angiographically confirmed LV dilatation after MI is an unfavorable finding as regards to the survival prognosis. Patients with dilatation of the cardiac chambers after MI are found to have three times higher mortality (10). The results of ventriculography indicated an improvement in a number of cardiac hemodynamic parameters in patients after the late PCI as compared to the control group. In the long-term period after the late PCI (at 6.2 ± 1.68 months) there was a trend towards the decrease of LV EDV from 186.3 ± 33.9 (median 180.5) to 174.6 ± 37.1 ml (median 160.5) (p > 0.05). In Group II patients the values of LV EDV remained high at baseline and at the follow-up assessment (254 ± 64.5 and 220.6 ± 56.9 ml, respectively) (p > 0.05). LV EDV is also one of the most important prognostic criterion in patients within 1 years after MI (1). In the long-term period patients in Group I had findings suggesting significant decrease of the LV EDV. The LV EDV decreased from 123.7 ± 32.74 ml to 97.2 ± 40.36 ml at the follow-up assessment (p = 0.017). In Group II patients there were no significant difference between the baseline and the follow-up LV EDV values (201.4 ± 84 vs 167.3 ± 76.3 ml, p > 0.05). The mean LV SV in patients after the late PCI increased from 62 ± 12.55 to 76.6 ± 16.6 ml (p = 0.03), whereas in control patients the LV SV changed insignificantly (78.6 ± 22.7 vs 77.88 ± 24.2 ml, p > 0.05). These findings suggest positive effect of the late myocardial reperfusion on the cardiac hemodynamic values.

Late reperfusion of a single, infarct-related artery, provides an increase in LV global contractile function and improves other parameters of cardiac hemodynamics, which is apparently due to the partial restoration of the viable myocardium function within the LAD territory, as well as the functional improvement in remote myocardial segments. This can be associated to the fact, that the process of remodeling involves not only the dysfunctional, but also intact myocardial segments, which suffer chronic increase of the preload or cardiac chamber dilatation (13). In case of a multi-vessel disease, reperfusion of the infarct-related artery may provide an increase in the LV contractile function due to intersystem collateral pathways.

The rate of restenosis at the site of bare metallic stent implantation measured at follow-up coronary angiography was 20% (n = 2) with clinical signs being evident in one patient. One patient underwent CABG with LV aneurysm resection, the other one underwent PCI with repeated tenting of the target lesion.

In what concerns further treatment strategy in patients after late PCI (Group I), four of them (40%) were considered candidates for CABG and LV aneurysm resection, one underwent repeated procedure for restenosis, whereas 50% of patients (n = 5) had no direct indications for cardiac surgery or re-intervention (see Table 6). One should also consider the fact, that all Group I patients at baseline were candidates for an open myocardial revascularization with 80% of them (n = 8) being candidates for LVA repair. Apparently all Group II patients had underwent CABG by the follow-up assessment, in 71% of cases the CABG was accompanied by LVA resection.

These findings suggest, that even the late PCI in a number of patients with PLVA may stabilize the clinical course of CAD, decreasing the grade of angina and the heart failure. Our data have shown, that the late PCI in patients after MI with PLVA might improve the cardiac hemodynamic values, prevent severe LV remodeling and change the proportion of

Table 6. Further treatment strategy in patients after the late PCI.

	Group I (n = 10)		Group II (n = 7)		
Treatment strategy	n	%	n	%	p
No direct indications for revascularization or aneurysm repair	5	50	0	0	<0.05
Indications for CABG	0	0	2	29	NS
Indications for CABG and aneurysm repair	4	40	5	71	
Indications for re-intervention in the target lesion	1	10	0	0	

patients necessitating surgery for LVA and repeated myocardial revascularization, as well as the timing of surgery.

Therefore, the study has confirmed the possible favorable effect of late PCI with stenting of the infarct-related artery on the process of pathological myocardial remodeling in patient with PLVA, thus improving the prognosis of the disease and reducing the need for subsequent surgery.

Conclusions

1. Late PCI performed 4 weeks after an acute anterior MI is a safe method of myocardial reperfusion in patients with postinfarction left ventricular aneurysm.
2. Late PCI performed 4 weeks after an acute anterior MI in patients with postinfarction left ventricular aneurysm stabilizes the clinical course of CAD after MI.
3. Late reperfusion of the infarct-related artery (LAD, 4 weeks after an acute anterior MI) using PCI in patients with LV aneurysm may provide favorable effect on the cardiac hemodynamic values.
4. Late PCI performed 4 weeks after an acute anterior MI can change the proportion of patients necessitating surgical repair of the left ventricular aneurysm or coronary artery bypass grafting.

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Coronary Angioplasty in Acute Myocardial Infarction. A Review

A. L. Krylov[1]

Research Institute of Cardiology under Tomsk Research Center of the Russian Academy of Medical Sciences, Tomsk, Russia

The benefits of PTCA over thrombolytic therapy (streptokinase and plasminogen tissue activators) in myocardial infarction (MI) have been demonstrated in numerous randomized studies (1, 2, 3, 4, 5, 6, 7). This review, therefore, emphasizes the particularities of endovascular interventions in patients with cardiogenic shock, diabetes mellitus, elderly and females rather than the efficacy comparison between thrombolysis and angioplasty. This review evaluates the efficacy of PTCA versus stenting for reperfusion of the infarct-related artery. Particular emphasis is placed upon the problems of coronary angioplasty in MI.

Coronary angioplasty in patients with cardiogenic shock.

Myocardial infarction causing cardiogenic shock is known to have the most severe clinical course. First reports showing increased survival after PTCA in patients with cardiogenic shock were published in the late 1980-s and the early 1990-s (8, 9, 10). The endovascular procedures, however, were performed in relatively small groups of patients (24 to 45 patients per group) (8, 9, 10). In addition, as reported by P. Urban et al. in 1999 (11), PTCA failed to improve the prognosis in patients with cardiogenic shock. However, as the previous studies, this one enrolled relatively small number of subjects (n = 27) (11). Thus, in the 1990-s the need for a randomized study to assess the efficacy of endovascular procedures in cardiogenic shock became apparent. In 1999 the results of the two randomized studies concerning PTCA in cardiogenic shock were published (12, 13). The New-York study enrolled 152 patients after urgent reperfusion (PTCA or bypass grafting) and 150 patients on drug therapy only (12, 14). There was no significant difference in the mortality rate between the groups 30 days after MI (12). At 6 months after the cardiogenic shock the mortality in medical therapy group was 63% vs. 50% in revascularization group ($p = 0.027$) (12), at 1 year the mortality rate was 67% for medical therapy vs. 54% for revascularization ($p < 0.03$) (14). The study performed in Mayo Hospital (Rochester, USA) proved more promising (13). This study involved 1321 patients with cardiogenic shock, 578 of which underwent myocardial revascularization (PTCA or CABG), thrombolysis was conducted in 728 patients (13). At 1 year the survival was 92% in revascularization group vs. 85% in thrombolysis group ($p = 0.0003$) (13). Therefore, both randomized studies have shown, that revascularization significantly improved the survival rate in patients after cardiogenic shock. It remained unclear, however, why the 1-year mortality rate was as low as 8% in Mayo Hospital compared to the New-York patients, 54% of which died within 6 months. Most authors indicate, that the 1-year mortality in patients with cardiogenic shock after PTCA is around 50% (12, 14, 15, 16, 17, 18). The results of a Canadian randomized study published in 2003 (15) were largely similar to the data published by J.S. Hochman et al. (14). The Canadian study enrolled 82 patients with cardiogenic shock treated with PTCA (15). The mean time from the onset of symptoms to the endovascular procedures was 11 h (15). The 1-year mortality rate was 50% (15). Authors assessed correlation between the mortality rate in patients with cardiogenic shock and the efficacy of infarct-related artery reperfusion (15). The reperfusion efficacy was graded using TIMI scale (Thrombolysis In Myocardial Infarction) (15). The 1-year mortality was 39% in case of successful intervention vs. 85% in case of failure ($p < 0.001$) (15). The mortality rate was 38% for TIMI 3; 55% for TIMI 2; 100% for TIMI 0 or 1 ($p < 0.001$) (15). Thus, the authors demonstrated reverse correlation between the coronary flow velocity and the mortality rate: the lower was the blood flow, the higher was the mortality (15). In addition, the mortality rate was found to correlate with the age ($p < 0.001$) (15). There was a reverse correlation between the time of ischemia (the interval between the coronary thrombosis and reperfusion) and the mortality rate ($p = 0.019$) (15). The authors concluded, that PTCA was beneficial in cardiogenic shock if performed within 12 h after the onset of MI (15). This conclusion made by J.G. Webb et al. was largely similar to that of B.R. Brodie et al. (2003), who showed progression of the hospital mortality rate in patients with cardiogenic shock parallel to the time-to-reperfusion defined as the interval between the onset of symptoms and the balloon dilatation of the infarct-related artery (16). The mortality rate was 31% for the time-to-reperfusion below 3 h, 50% for 3 to 6 h, 62% for 6 h and longer ($p = 0,01$) (16). Consequently, PTCA in patients with cardiogenic shock should be performed within 3 h of MI, but no later than 6 h or more after coronary thrombosis onset. Interestingly, MI patients without shock had no correlation between the hospital mortality and the time to endovascular procedure (16). As mentioned above, the first randomized studies

[1]A.L. Krylov
634034 Tomsk,
151 Krasnoarmeyskaya str., Apt. 14.
Telephone (business hours): (3822) 26-21-74
Email: maslov@cardio.tsu.ru
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concerning emergency myocardial reperfusion in patients with cardiogenic shock enrolled subjects, who underwent PTCA or bypass surgery (12, 13, 14). The authors failed to compare the efficacy of PTCA and CABG. H.D. White et al. (2005) attempted to determine which method of myocardial reperfusion is the most effective in cardiogenic shock: open surgery or endovascular interventions (17). This study enrolled 128 patients with cardiogenic shock, 81 of which underwent PTCA (group I), whereas 47 patients underwent bypass grafting (group II) (17). The survival rate within 1 month of MI was 55% in group I vs. 57% in group II ($p = 0.86$), at 1 year the survival was 52% in group I vs. 47% in group II ($p = 0.71$) (17). Authors concluded, that there was no difference between the angioplasty and coronary artery bypass grafting as regards to survival after cardiogenic shock (17).

Therefore, the date reviewed above suggest, that PTCA is superior to thrombolysis for reperfusion of the infarct-related artery in patients with cardiogenic shock. Coronary artery bypass grafting has no apparent benefits over PTCA with respect to prognosis after cardiogenic shock. The high mortality rate in patients with cardiogenic shock still remains one of the challenges in cardiology.

PTCA in elderly patients with myocardial infarction

Elderly age is known to be an additional risk factor in MI. A study conducted in Holland enrolled 87 patients aged over 75 treated with thrombolytic therapy or PTCA (19). The 1-month follow-up has detected unfavorable events (death, stroke, recurrent MI) in 9% of patients after PTCA vs. 29% of patients after thrombolysis ($p < 0.01$) (19). At 1 year the rate of unfavorable events was 13% in group I vs. 44% in group II ($p < 0.001$) (19). The STEMI assessment (ST-elevation myocardial infarction) has shown, that the age can be an independent predictor of an unfavorable outcome after coronary angioplasty: the older is the MI patient, the higher is the risk of an unfavorable event after primary PTCA ($p < 0.001$) (20). A similar conclusion was made by G. Guagliumi et al (21), who found that the 1-year mortality rate after primary angioplasty increased parallel to age (1.6% in patients aged below 55 years, 2.1% in patients aged 55 to 65 years, 7.1% for patients aged 65 to 75, 11.1% for patients above 75 years of age; $p < 0.0001$) (21). The high risk of death after PTCA of the infarct-related artery in elderly patients is attributed to that fact, that they have lower reperfusion after endovascular interventions as compared to the middle age patients (20). Importantly, elderly patients have relatively high prevalence of endothelial dysfunction in peripheral arteries (22). In view of this fact G. De Luca et al. suggested, that a similar endothelial dysfunction is characteristic for coronary arteries in people aged above 65 (20). G. De Luca et al. (20) ascribed the low coronary perfusion parameters and poor long-term prognosis in elderly to coronary endothelial dysfunction. However, there have been no publications directly confirming this hypothesis. Stenting considerably improves the prognosis in elderly patients with MI. Thus, according to G. Guagliumi et al. (21), the need for repeated revascularization in patients aged over 65 is 17.6% at 1 year after balloon dilatation compared to as low as 7% after stenting ($p < 0.0001$) (21). At the same time, in elderly patients with MI the rate of death, stroke and recurrent MI doesn't depend on the method of endovascular intervention (21).

Primary PTCA therefore improves the prognosis in elderly patients with MI. Stenting of the infarct-related artery is the treatment of choice in such patients.

Coronary angioplasty in women with myocardial infarction

The mortality rate due to MI is known to be higher in women compared to men. However, till recently the effect of endovascular interventions on the mortality rate in women after MI hasn't been studied. The results of a randomized study performed in Munich were published in 2002. The study enrolled 502 women and 1435 men with MI treated by PTCA (23). Women were older as compared to men and had higher incidence of diabetes mellitus and hypertension (23). The 1-year follow-up showed no significant difference in mortality rate between the groups (23). After exclusion of elderly patients (aged above 75) the risk of death 1 year after PTCA of the infarct-related artery in women was lower, than in men ($p = 0.004$) (23). Thus, despite the fact, that women are associated with risk factors, such as diabetes, elderly age and hypertension, angioplasty eliminates the gender difference in mortality rate after MI.

Authors concluded that coronary angioplasty is more effective for myocardial reperfusion in women as compared to men (23). The reason for such high efficacy of PTCA in women remained unclear. Munich cardiologists assessed the difference in coronary perfusion after PTCA of the infarct-related artery between men ($n = 561$) and women ($n = 202$) (24). The low perfusion area was measured with 99mTc-MIBI (metoxyisobutyl isonitrile) perfusion scintigraphy before and 7 to 10 days following the primary angioplasty (24). The radioisotope tracer was accumulated in myocardial regions receiving blood, thus showing the ischemic area in CAD and MI patients (25). At baseline the ischemic area was 22% from the left ventricular area in women vs. 24% in men ($p = 0.26$) (24). After PTCA the perfusion defect was decreased to 6% in women vs. 10% in men ($p = 0.001$) (24). In addition, the authors calculated the so-called salvage index as the ratio between the myocardial area with restored perfusion and the area of initial perfusion defect (24). In women this value was 0.64 vs. 0.5 in men ($p < 0.001$) (24). The authors concluded, that PTCA was more effective for reperfusion of the infarction area in women as compared to men (24). The investigators faailed to provide an explanation for this gender difference.

An opposite conclusion was made in STEMI (ST-Elevation Myocardial Infarction) multi-center study (26), which enrolled 353 women and 1195 men with MI. The 1-year mortality rate after PTCA was 9.3% in women vs. 4.9% in men (p = 0.002). During hospital stay there were no differences between men and women in the area of myocardial necrosis and the coronary perfusion (26). Authors came to the conclusion, that primary PTCA didn't eliminate the difference between men and women in the mortality rate after MI (26). CADILLAC study (Controlled Abciximab and Device Investigation to Lower Late Angioplasty Complications), that involved 562 women with MI, demonstrated, that women had more favorable prognosis as compared to men (27). At 1 year after PTCA the mortality rate was 7.6% in women vs. 3.2% in men (p < 0.001) (27). Repeated revascularization was performed in 16.7% of women and 12% of men (p < 0.006) (27). During the first year the incidence of cardiac events was 24% in women and 15% in men (27). It was suggested, that the higher rate of unfavorable outcome after PTCA in women with MI was due to the fact, that their age was higher than in men with MI and they had higher incidence of diabetes and hypertension (26, 27). Primary stenting decreased the rate of unfavorable events in women from 28% (after PTCA) to 19% (p < 0.01), while the rate of repeat revascularization declined from 20% after PTCA to 11% after stenting (27).

Therefore, the published data concerning the effect of PTCA on the late prognosis of myocardial infarction in women are contradictory (23, 24, 26, 27). The reason for such contradiction was not discussed in the above publications. However, there's no contradiction as regards to the choice of infarct-related artery recanalization method in women. All investigators give preference to primary PTCA (23, 24, 26, 27), A.J Lansky et al. (27) suggested, that stenting is a method of choice for revascularization of the infarction area in women.

PTCA in patients with diabetes mellitus and myocardial infarction

A study performed in Holland enrolled 74 patients with MI and diabetes mellitus and a control group comprised of 391 patients with MI without diabetes (28). Recanalization of the infarct-related artery was performed by administration of streptokinase or PTCA (28). Diabetic patients proved to have lower left ventricular ejection fraction (LVEF) after coronary reperfusion as compared to other patients (p = 0.02), higher mortality rate (p < 0.001), higher incidence of severe heart failure (p = 0.004) (28). The authors provided an additional confirmation of the fact, that diabetes is an independent mortality risk factor in MI patients (28). The CADILLAC study involved 346 patients with diabetes and MI treated with thrombolysis or PTCA. The 1-year follow-up showed, that the risk of unfavorable events was 21.9%, vs. 16.8% in other patients (p < 0.02) (29). Within a year of MI the mortality rate in diabetics was higher (6.1%) compared to other patients (3.9%, p < 0.04) (29). The CADILLAC collaborators assessed the correlation between diabetes and coronary perfusion after recanalization of the infarct-related artery (30). Coronary perfusion was measured using MBG scale (myocardial blush grade) and STR-test (ST-segment elevation resolution) (30). Reperfusion of the infarct-related artery was performed using thrombolysis or an endovascular intervention (30). The values of MBG showed, that reperfusion of the ischemic area was achieved only in 44% of diabetic patients, which was significantly lower as compared to other patients (p = 0.01) (30). The ST-segment elevation persisted after recanalization in 20% of the diabetics vs. 8% in the control group (p = 0.002) (30). Similar data were obtained in a multi-center study under STEMI program, which involved 64 diabetic patients with MI, 322 patients comprised the control group (MI without diabetes) (31). In both groups the infarct-related artery revascularization was performed using endovascular approach (31). As measured by MBG, reperfusion of the infarction area was not achieved in 20% of the diabetics, whereas in the control group poor perfusion of the ischemic area was found in 10% of cases (p = 0.02) (31). The rate of incomplete ST-segment resolution was 55% in diabetic patients and 35% in other patients with MI (31). These facts have brought the authors (30, 31) to a conclusion, that the reason for lower efficacy of thrombolysis in diabetic patients compared to other patients is the poor coronary perfusion within the infarction area after recanalization of the infarct-related area. It is believed, that diabetic patients have higher rate of microcirculatory dysfunction within the infarction area and intravascular aggregation of the blood cells in the microcirculatory vasculature (31, 32) (i.e. the "no reflow" phenomenon) as compared to other patients. The point of this phenomenon is that reperfusion of large coronary arteries after 60-120 min occlusion doesn't result in complete restoration of the blood flow in the micro-vessels (33). The investigators attributed the poor efficacy of thrombolysis and PTCA of the infarct-related artery in diabetic patients to the "no reflow" phenomenon (31, 32).

The above facts suggest the importance of the search for an optimal reperfusion method in diabetic patients with MI. One of the first articles in this field was published by K. Thomas et al. in 1999 (34). Their study enrolled 16 patients after PTCA (group I) and 16 patients after thrombolysis (group II) (34). Reperfusion of the infarct-related artery was successful in 14 patients from group I vs. only 5 patients from group II (p < 0.001) (34). The LVEF value was 49% in group I compared to 36% in group II (p <0,05) (34). Serial LDH activity study showed, that the necrotic area in PTCA patients is 4 times smaller than in patients treated with thrombolysis (p < 0.05) (34). Higher LVEF value and smaller necrotic area in the group were apparently the result of a successful recanalization of the infarct-related artery (34). Unfortunately, due to the small sample size (n = 32)

authors failed to detect any benefits of PTCA over thrombolysis in diabetic patients with MI (34). In the CADILLAC study (346 diabetic patients with MI) (29) the 1-year rate of restenosis was 21% after stenting and 48% after successful PTCA of the coronary artery (p = 0,009). The 1-years rate of repeated revascularization was 10% in patients, who underwent stenting, vs. 22% in patients after PTCA (p = 0.004) (29). At the same time, G. De Luca et al. (35) demonstrated, that stenting (n = 84) didn't have any benefits over PTCA (n = 76) as regards to the 1-year mortality rate in diabetic patients with MI.

Consequently, stenting is optimal for revascularization of the infarction area in diabetic patients. Yet, implantation of bare metal stents in diabetic patients doesn't resolve the problem of high late mortality rate typical for this group. The wide clinical acceptance of stents covered with antiproliferative agents (Sirolimus or Paclitaxel) will probably solve the problem.

Comparison between balloon angioplasty (PTCA) and stenting of the infarct-related artery for myocardial revascularization

Further studies performed in the 21st century were dedicated to the search for the optimal revascularization method in MI. An important question was the need for stenting after MI as an alternative to balloon dilatation, which has a cost 2000 USD lower compared to stenting (36). The stent-PAMI study (Stent Primary Angioplasty in Myocardial Infarction) conducted in 448 MI patients treated with PTCA and 452 MI patients treated with stenting indicated, that the 1-year rate of repeated revascularization or hospitalization was significantly lower after stenting as compared to PTCA (36). The Holland study involved 112 patients (group I) treated with primary stenting of the infarct-related artery and 115 patients (group II) treated with PTCA (37). At 6 months the rate of recurrent MI was 1% in group I vs. 7% in group II (p = 0.036) (37). Repeated revascularization was performed in 4% of group I patients and 17% of group II patients within the same period (p < 0.0016) (37). The 1-year follow-up showed, that the rate of unfavorable outcomes (death, recurrent infarction) was 4% after stenting and 11% after PTCA (p = 0.04) (38). The 1-year rate of repeated revascularization was 13% in group I patients vs. 34% in group II patients (p < 0.001) (38). At 6 months after the endovascular intervention the angiographic restenosis was found in 12% of patients treated with stenting and in 34% of patients after PTCA (p < 0.001) (38). The results of CADILLAC study conducted between November 1997 and September 1999, which enrolled 2082 MI patients, were published in 2002 (1). The patients were divided between the four groups: (1) PTCA (n = 518); (2) PTCA + Abciximab (IIb/IIa glycoprotein receptor inhibitor) (n = 528); (3) stenting (n = 512); (4) stenting + Abciximab (n = 524) (1). The 6-month follow-up showed the rate of unfavorable events (death, recurrent MI, stroke, repeated hospitalization) to be highest in the first group — 20%, 16.5% in the second group, 11.5% in the third group (stenting), 10.2% in the fourth group (stenting + Abciximab) (1). Therefore, the rate of unfavorable events was 2 times higher (p < 0.001) in patients treated with stenting + Abciximab as compared to patients treated with PTCA only (1). There was no significant difference between the groups in the rate of recurrent MI, stroke or death, except for the rate of repeated revascularization, where the difference was significant (1). A similar conclusion was made in STEMI study, which enrolled 849 patients treated with stenting and 834 patients treated with PTCA of the infarct-related artery (39). The 1-year follow-up showed, that stenting doesn't have any benefits over PTCA with respect to the rate of death and recurrent MI (39). At the same time, within 6 months postoperatively repeated revascularization was performed in 15.7% of patients after PTCA and only in 5.2% of patients after stenting (p < 0.001) (1). Angiographic restenosis at 6 months postoperatively was found in 41% of patients after PTCA vs. 22% of patients after stenting (p < 0.001). Within this period the reocclusion rate was 11% in the first group vs. only 5.7% in the fourth group (p < 0.01) (1). The results of follow-up including these 2082 MI patients were published in 2003 (40). The follow-up was performed as part of the CADILLAC study (40). Clinical analysis suggested, that within the first 30 days following primary PTCA the rate of repeated angioplasty was 5.1% in patients after PTCA and 2.3% in patients after implantation of bare metal stents (p < 0.007) (40). Early thrombosis of the infarct-related coronary artery occurred in 1.4% of patients after PTCA and only in 0.5% of patients after stenting (41). The 1-year follow-up showed, that the rate of unfavorable events in patients after PTCA was 21.9% vs. 13.8% in patients after stenting (p < 0.001) (40). Results of the Munich study were published in 2004 (42). The study involved patients (n = 181), in whom recanalization of the infarct-related artery was unsuccessful within 24 h of hospitalization (42). Stenting was performed in 90 patients (group I), PTCA — in 91 (group II). Coronary perfusion was assessed with myocardial scintigraphy prior to and 7 to 10 days following the endovascular intervention (42). It was established, that the salvage index was 0.35 in group I vs. 0.25 in group II (42). There was no difference between the groups with respect to mortality rate (42). These data were confirmed in a randomized study performed by the same authors in 611 patients after unsuccessful thrombolysis for MI (43). The investigators concluded, that stenting is superior to PTCA as a method of revascularization (42, 43).

Therefore, randomized studies have shown, that stenting is more effective for infarction area reperfusion as compared to PTCA, as it is associated with slower restenosis progression and, consequently, lower need for repeated reperfusion. At the same time, stenting doesn't have any apparent benefits over PTCA as regards to the rate of death, stroke

or myocardial infarction. Final conclusions on the advantages of stenting and balloon angioplasty for reperfusion of the myocardial infarction area necessitate new multi-center randomized study using bare metal stents and stents with antiproliferative coating (Sirolimus, Paclitaxel).

The first such study was performed in Holland (44) and involved 96 MI patients, of which 46 had single-vessel disease and 12 — cardiogenic shock (44). Follow-up was conducted during 6 months after admission to the hospital. The rate of unfavorable events (death, recurrent myocardial infarction, repeated revascularization) during this period was 8.4% (44). For the non-coated stents the rate of unfavorable events was 10.2% (1). At first glance, the difference between Sirolimus-coated and non-coated stents is non-significant. However, the unfavorable events associated with Sirolimus-eluting stents mostly occur during hospital stay (only a single patient died after the discharge) (44). For the non-coated stents the 6-month rate of repeated revascularization was 5.7% (1), while only a single patient necessitated repeated procedure after a successful implantation of a drug-eluting stent during hospitalization for MI (44). The rate of restenosis associated with bare metal stents was 22%, the rate of reocclusion was 5.7% (1). Following implantation of Sirolimus-eluting stents there were no cases of restenosis or reocclusion (44). The authors concluded, that Sirolimus-eluting stents were safe and effective in MI (44).

Therefore, the results of independent randomized studies demonstrate, that PTCA is currently the method of choice in myocardial infarction. Compared to bypass grafting and thrombolysis this method demands shorter hospital stay. Particularly apparent are the benefits of PTCA over thrombolysis in MI patients with cardiogenic shock, diabetes, elderly patients or women with MI. Stenting is more effective for infarction area reperfusion compared to PTCA, as it is associated with slower restenosis progression and, hence, lower need for repeated reperfusion. At the same time, stenting doesn't have any apparent benefits over PTCA as regards to the rate of death, stroke or myocardial infarction. In conclusion we decided to outline the problems, which are yet to be resolved. Despite the wide clinical acceptance of PTCA and stenting of the infarct-related artery, the mortality rate in patients with cardiogenic shock is still around 50%. Stenting in diabetic patients doesn't resolve the problem of high mortality rate in the long-term period after MI. It is unclear whether stents with antiproliferative coating (Sirolimus, Paclitaxel) have significant advantages over bare metal stents for the treatment of myocardial infarction.

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Diagnostic Value of the High-Speed 64-Slice Multidetector Computed Tomography in assessment of Peripheral and Coronary Artery Disease

Z.A. Kavteladze , V.E. Glagolev
Center of Endosurgery and Lithotripsy, Moscow, Russia

The diagnosis of a cardiovascular disease until recently required hospitalization followed by minimally invasive interventions: angiography of coronary or peripheral arteries. These studies have become "the golden standard" of cardiovascular diagnostics due to their high diagnostic value. Today high-speed 64-slice multidetector computed tomography has became an alternative to diagnostic angiography of coronary and other arteries. This method has a number of benefits: the high speed of image acquisition, no need for hospitalization and minimally invasive procedures.

1. History of the method

Multislice computed tomography (MSCT) has been used for cardiovascular diagnostics since the 1990-s (1). In the beginning of its history the direct visualization of coronary arteries was impossible because of low resolution and high incidence of image artifacts, therefore, coronary atherosclerosis was evaluated by counting the content of the intravascular calcium (1). Quantification of coronary artery calcium is based on the coefficient of X-radiation absorption and the area of calcification sites (44). According to this scale, coronary calcification is defined as an area over 130 U in density (1). The Agatstone calcium index (CI) is the product of the calcification area and the density factor. Calcium index was reported to be a predictor of cardiovascular disease and correlates directly to the incidence of atherosclerosis: the risk of atherosclerosis increases proportionally to the index value (1,46). For example, low CI (10 U or below) is associated with 5–10% incidence of coronary artery disease. For moderate CI (11 to 100 U) the rate of 50% coronary artery stenosis is 20% or below, for CI between 101 U and 400 U the rate is 75%, i.e., there's a moderate risk of atherosclerosis. High CI (above 400 U) is characterized by approximately 90% risk of coronary atherosclerosis (46).

CI is a predictor of future cardiovascular accidents with their frequency being proportional to the index value (57).

[1]Z.A. Kavteladze,
Center of Endosurgery and Lithotripsy
Department of cardiovascular surgery.
Moscow, 111 123
Shosse Entuziastov, 62
Tel. (495) 305 34 04
Fax(495) 305 69 35
e-mail: vlesten@gmail.com
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The introduction of 4-slice in 1999 (43) and, later in 2001, 8-slice multidetector computed tomography (43) has provided an opportunity both to measure static objects as an indirect signs of atherosclerosis and to directly visualize the status of coronary bed (1). Unfortunately, high incidence of respiration-induced artifacts and errors due to heart excursions observed with conventional computed tomography (CT) units with few slices (3), this method was not widely adopted. The goal therefore was to create high-speed systems, able to provide images within a single cardiac cycle, i.e. less than 500 ms (gantry turnover time) in order to evaluate the coronary arteries with enough quality. The technology had rapidly evolved and, in the end of 2001, 16-slice (1,43) and, later, 32-slice and 40-slice multidetector CT systems were created. However, the objective was not finally completed. It was on the threshold of 2005 (5,46) when the non-invasive 64-MSCT occurred along the physician's arsenal providing faster image acquisition with 3-dimensional reconstruction of an object below 0.5×0.5×0.6 mm in size (5,43).

The documented superiority of 64-slice CT over 16-slice CT was shown as the higher imaging reliability and substantially lower incidence of motion artifacts (3,5,6,22,28). The reasons are as follows (3,20):

1. higher speed of gantry turnover: 330–420 vs 375-500 ms,
2. higher spatial resolution: 0.4–0.6 vs 0.75 mm, and
3. higher time resolution: 165–210 vs 188–250 ms.

In addition, the duration of 64-MSCT study is lower (6–13 s) as compared to the 16-MSCT (15–25 s) and requires lower amount of the contrast medium: 50–80 ml vs 70–100 ml, thus diminishing the risk of complications (3).

Therefore, along with the invasive methods, 64-MSCT has been most extensively used for the diagnosis of coronary and peripheral arterial diseases (3,43).

2. Diagnostic potential of the 64-MSCT

The 64-MSCT is used for cardiovascular assessment in the following situations (2,7,9,15):

1. Coronary artery disease (CAD),
2. Aortic lesions (coarctation, aneurysm, aortic dissection, etc.),
3. Peripheral arterial disease (lower extremities, carotid lesions, etc.),
4. Myocarditis,
5. Pericarditis,

6. Infective endocarditis,
7. Pulmonary embolism,
8. Congenital cardiovascular defects,
9. Acquired heart disease (e.g., aortic valve calcification causing aortic stenosis or insufficiency, etc.),
10. Arrhythmias.

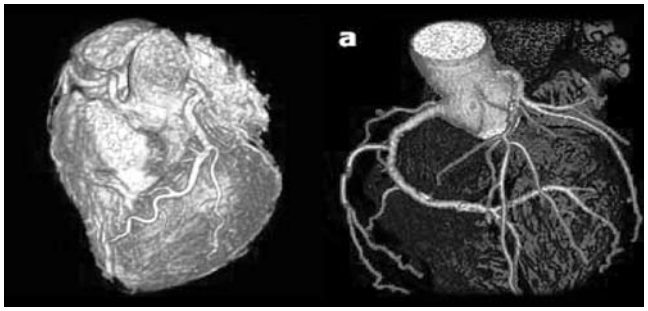


Figure 1. 3-D MSCT reconstruction of coronary arteries showing the entire length of the left main coronary artery, LAD, RCA.

I. Coronary arteries

High resolution of the 64-MSCT has the following benefits:

A) reliable imaging of coronary arteries (2, 3, 4, 18) showing the location of atherosclerotic lesions and revealing the congenital coronary malformations (see Figures 1, 5, 17);

B) evaluation of patency of aortocoronary bypass grafts and endografts (stents) (7, 8, 17, 29, 33) (see Fig. 4);

C) CI calculation (1, 2, 3, 12) in order to make the disease prognosis more precise;

D) detection of perfusion disorders and decreased myocardial viability early after myocardial infarction and in the long term (2, 13, 24, 45, 50);

E) assessment of the cardiac contractile function (2, 11, 13, 21, 25);

F) evaluation of the pericardium and the heart valves (2, 10) (see Figures 18, 23, 29–30).

Visualization of the coronary atherosclerotic lesions using MSCT is an alternative to the invasive coronary angiography (CAG) (see Fig. 5) and is used in both confirmed (2, 3, 12, 40) and suspected CAD, as well as in asymptomatic patients (2, 14, 67) for diagnostic purposes, determination of the risk groups and their prognosis.

Indications to the study include (2, 3, 40, 42):

1. atypical chest pain,
2. risk factors:
   - — hypertension,
   - — dislipoproteidemia,
   - — obesity,
   - — diabetes mellitus,
   - — smoking,
   - — high CI value,
   - — family history of coronary artery disease, sudden death, peripheral artery disease.
3. acute coronary syndrome, myocardial infarction without ST segment elevation for the assessment of coronary lesions (16). MSCT detects possible complications of myocardial infarction, like interventricular septal rupture (see Fig. 2) (56) or left ventricular aneurysm rupture (see Fig. 3).
4. changes following coronary artery bypass grafting (CABG) or percutaneous transluminal angioplasty (PTCA) with stent placement in order to assess the patency of grafts and stents (7, 8) (see Fig. 4).

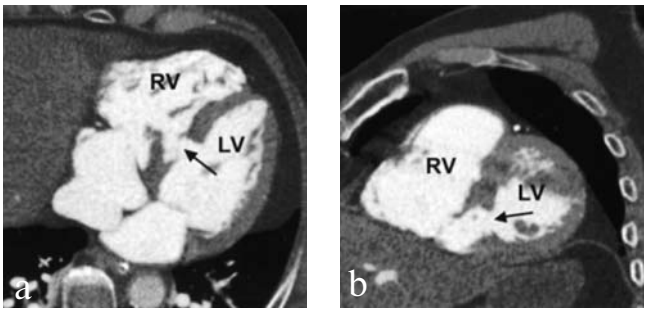


Figure 2. MSCT of the heart. Acute myocardial infarction causing interventricular septal rupture (arrow).

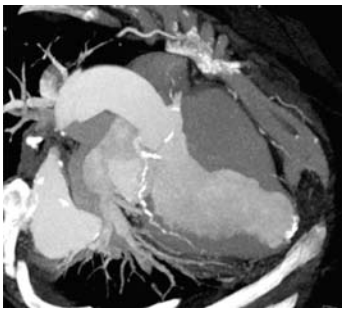


Figure 3. MSCT of the heart. Apical LV aneurysm (arrow), coronary artery calcification.

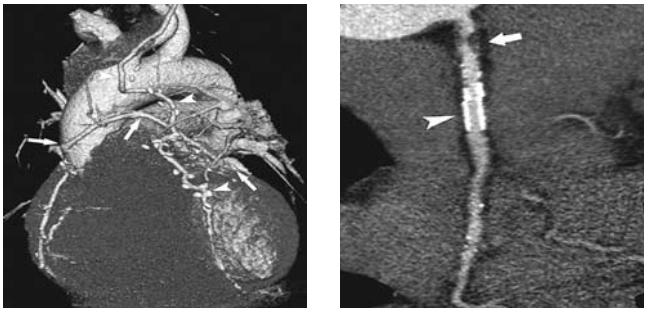


Figure 4. 3-D reconstruction in a patient after CABG and PTCA with stent implantation.
Left picture: arrows showing coronary bypass grafts.
Right picture: proximal coronary artery stenosis with a patent stent located below.

II. Extra- and intracranial vessels

MSCT of the extra- and intracranial vessels is performed in cerebrovascular atherosclerosis, cerebrovascular accidents, which act as the indications to the assessment of the intracranial and cervical vascular anatomy in order to rule out congenital malformations, including the arterial malformations (see Fig. 6,7), as well as to exclude atherosclerotic lesions of the extra- and intracranial arteries in patients after stroke or TIA (see Fig. 8) (2,3). Together with the arteries, the brain anatomy is reconstructed showing the finest lesions (tumors, haematomas, etc.) 0.4-0.6 mm in diameter. Precise diagnosis provided by MSCT allows to determine the cause of the disease and the location of lesions, which is crucial for further treatment strategy.

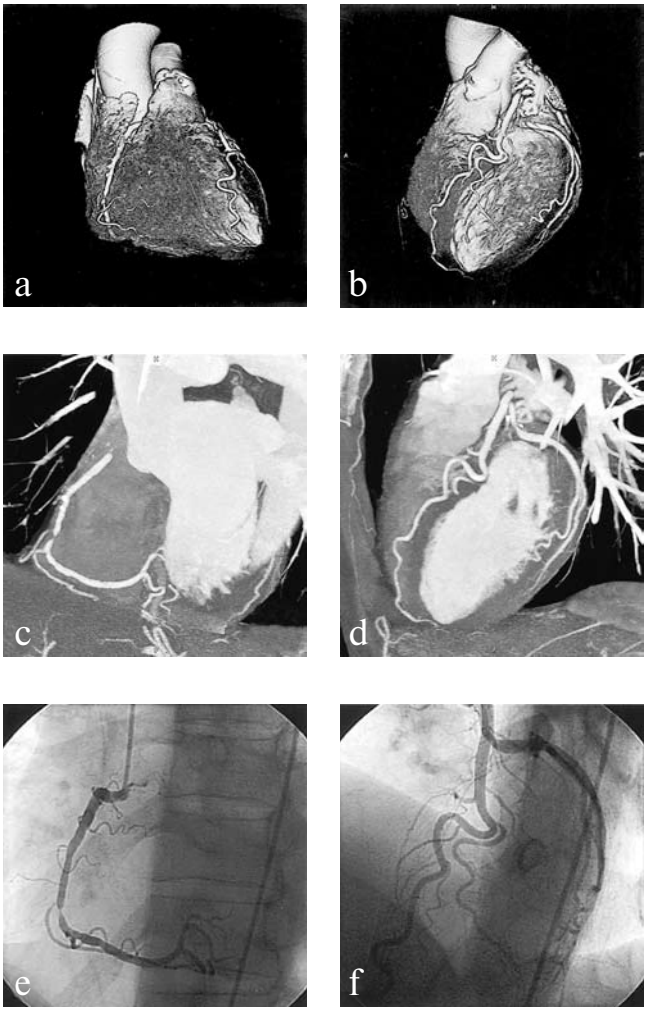


Figure 5. CAG (E, F) and coronary MSCT (A, B, C, D). Flow-limiting RCA stenosis (arrows).

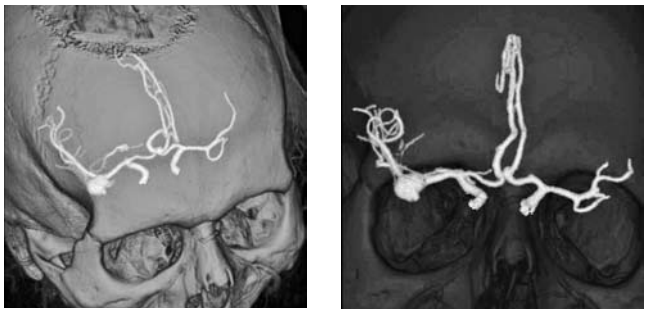


Figure 6, 7. Arterial aneurysm detected on intracranial MSCT (arrows).

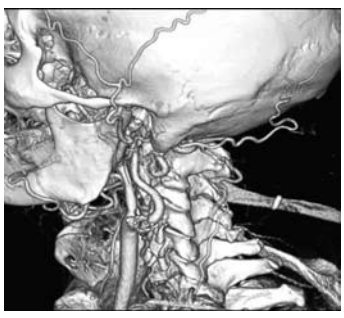


Figure 8. MSCT 3-D reconstruction of the carotid arteries showing stenosis of the left internal carotid artery (arrow).

III Aortic arch, thoracic and abdominal aorta

Confirmed or suspected malformations of the aorta and its branches (see Figures 10,11), aortitis of various origin, aortic atherosclerosis causing the aneurysm formation or occlusion — this list is far from the complete number of conditions requiring the 64-MSCT, which has a diagnostic potential significantly exceeding that of other methods. It was reported, that all types of aortic dissection can be visualized with this method (2,3,52).

Leschka S. et al (2005) (66) published an interesting case report describing a patient, who developed back pain during endovascular repair of an aortic coarctation. Urgent 64-MSCT diagnosed acute aortic dissection and the patient underwent open repair.

The opportunity to perform high-precision anatomic diagnosis, reliability and the speed of image acquisition determine the need to refer patients with such conditions to MSCT.

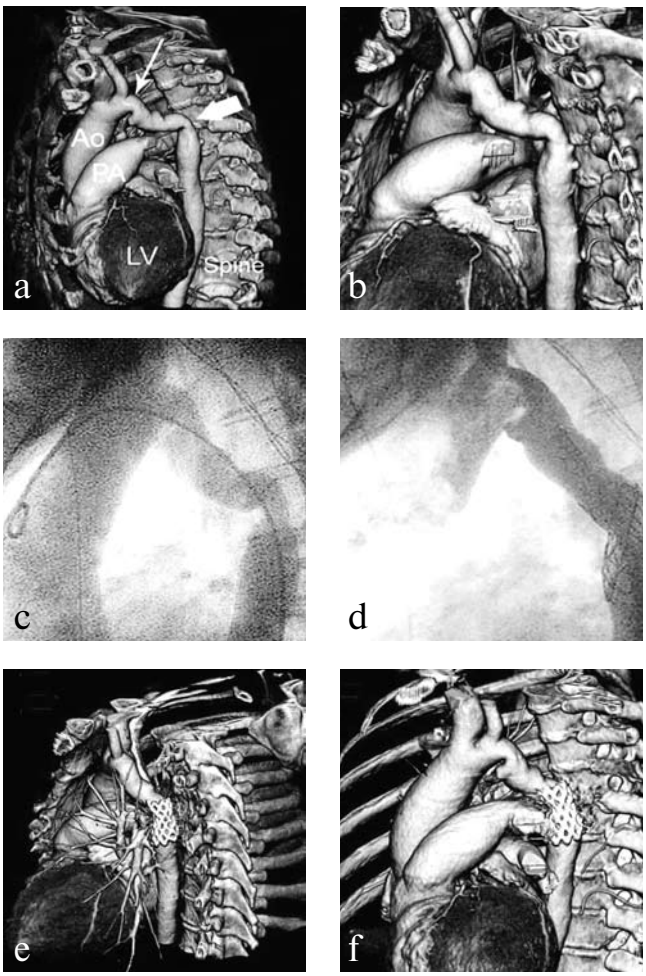


Figure 9. MSCT in a patient with aortic coarctation (A,B) (arrows). Aortography (C,D). Completion study after endovascular repair, follow-up assessment (E,F).

MSCT is also used as a completion study after surgical repair of aortic coarctations (see Fig. 9) (55).

The next indication to the assessment of the abdominal aorta and its branches is the search for renal artery stenosis in medically resistant hypertension (2, 3) (see Fig. 12).

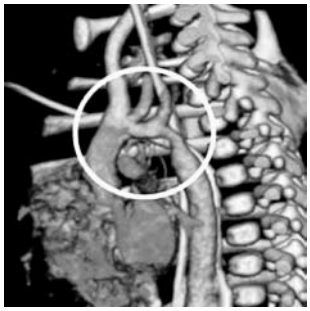


Figure 10. MSCT in a patient with aortic coarctation.

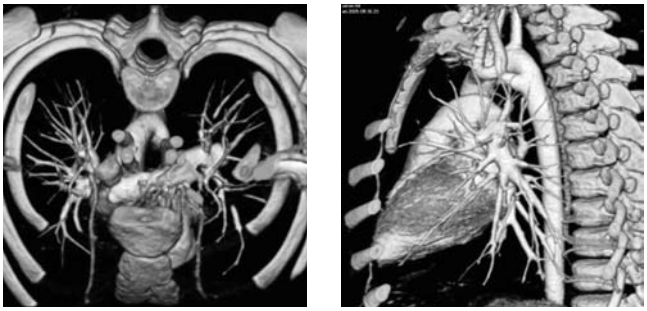


Figure 11. Aortic malformation (double aortic arch) (arrows).

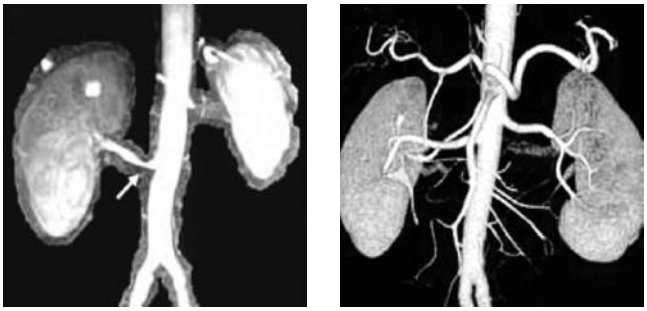


Figure 12. MSCT in a patient with renovascular hypertension showing right renal artery stenosis (arrow).

Clinical manifestations of an aortic branch stenosis requires detailed assessment and the patient might undergo invasive aortic angiography (AAG) or MSCT, which is more convenient (see Figures 13, 14).

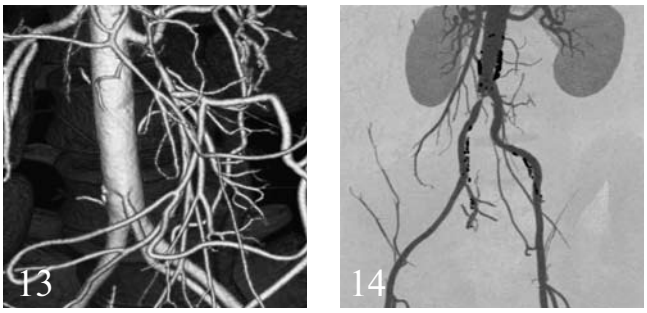


Figure 13, 14. MSCT 3-D reconstruction of the abdominal aorta and peripheral arteries. Right picture: stenosis of the upper mesenteric artery (arrow). Left picture: occlusion of the right iliac artery (arrow).

IV. Peripheral arteries of the upper and the lower extremities

Symptoms of peripheral arterial disease (Fig. 15,16) comprise another indication to MSCT. The study results can avail the doctor in his choice of treatment strategy among the range of minimally invasive endovascular repair or open reconstruction.

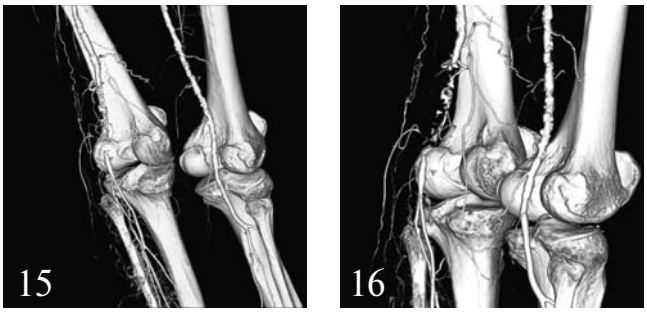


Figure 15, 16. 15,16. MSCT 3-D reconstruction of peripheral arteries showing occlusion of the left popliteal artery (arrow). Multiple atherosclerotic lesions of the right superficial femoral and popliteal arteries.

V. MSCT of the heart

Along with direct visualization of the coronary arteries 64-MSCT allows for simultaneous noninvasive assessment of various malformations of the heart and the adjacent vessels, including valvular disease (10,15,26). In this situation the method is used for more precise anatomic diagnosis needed to determine further treatment strategy, thus providing a single substitute for multiple studies. For example, a patient with degenerative aortic valve stenosis and ascending aorta aneurysm prior to the grafting of valve and aorta requires the assessment of coronary reserve in view of further simultaneous grafting and CABG. 64-MSCT would substitute diagnostic CAG, AAG and echocardiography, thus substantially reducing the time of the patient's assessment without detriment to the diagnostic value.

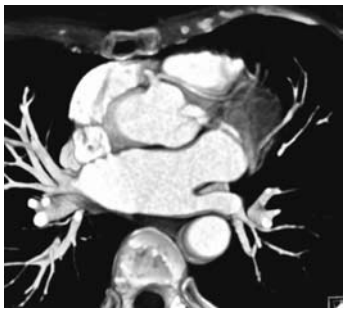
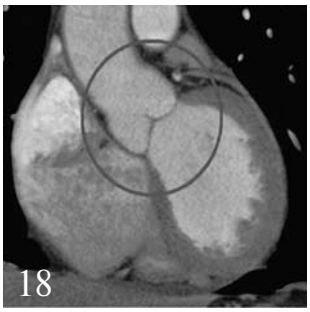


Figure 17. Coronary artery malformation. LCA origins from the RCA (arrow).

The study speed and accuracy are important in many clinical situations, for example, Valsalva sinus rupture in acute myocardial infarction was reported to be diagnosed using the 64-MSCT, interestingly, echocardiography failed to revealed this situation (51). Rapid and accurate diagnosis using 64-MSCT will point to proper treatment strategy without loosing time.

MSCT was reported to detect the bicuspid aortic valve (see Fig. 20,21), aortic valve vegetations in infective endocarditis (43) (see Fig. 24).

In patients with bicuspid aortic valve at high risk of ascending aorta aneurysm dissection, the precise diagnosis is crucial for the up-to-date surgical repair of the situation.



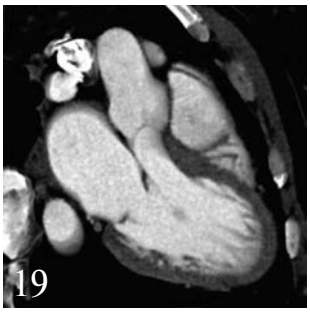
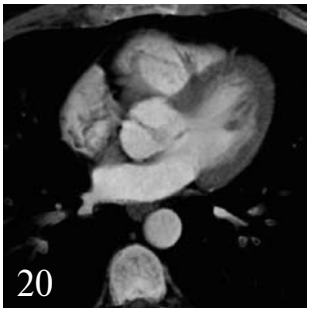


Figure 18, 19. MSCT showing normal aortic valve (left), mitral and aortic valves (right).



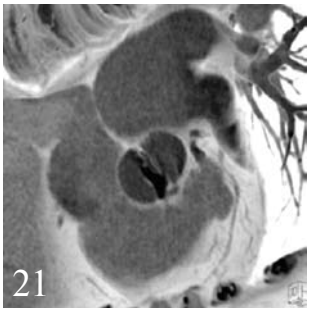
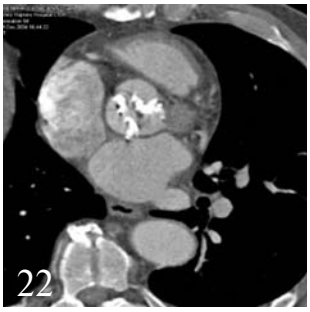


Figure 20, 21. Left picture: bicuspid aortic valve (arrow). Right picture: calcified bicuspid aortic valve (arrow).



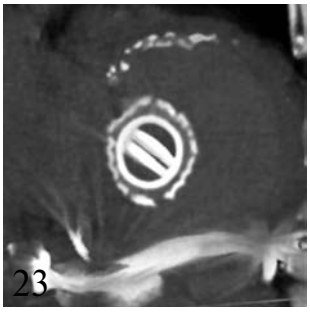
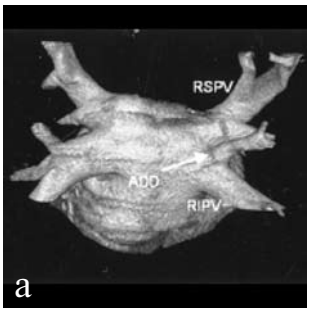
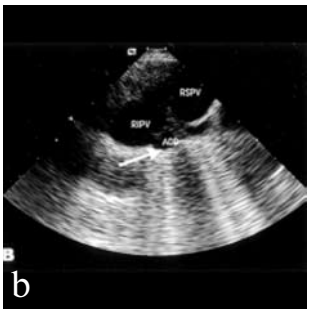
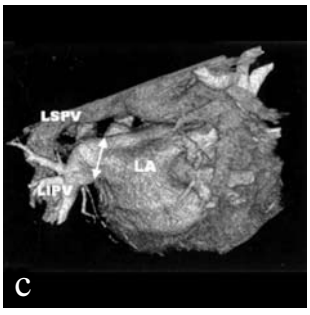


Figure 22, 23. Aortic valve calcification (arrow). Right image showing aortic valve graft.







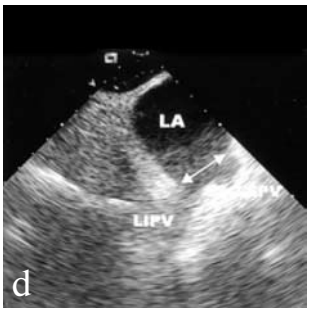
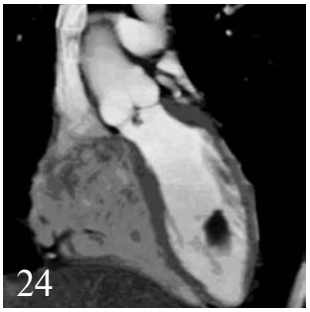


Figure 26. Additional right pulmonary vein (arrow) as shown by MSCT (A) and transthoracic echocardiography (B). Common origin (double arrow) of the left pulmonary vein as shown by MSCT (C) and transthoracic echocardiography (D).



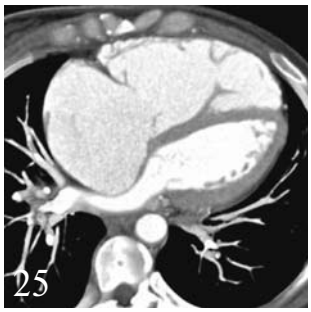
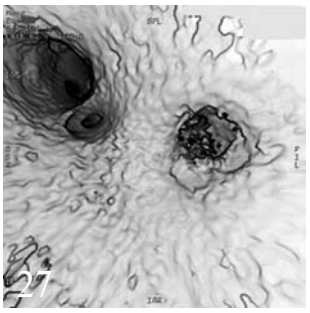


Figure 24. Infective endocarditis. Aortic valve vegetations (arrow).
Figure 25. MSCT of the heart showing enlarged right ventricle (arrow).

Considering the high image accuracy of the method, MSCT can be extensively used in patients with various heart rhythm disorders (2, 27, 54), particularly in the following situations:

1. Suspected arrhythmogenic dysplasia of the right ventricle, assessment of the right ventricular structures (Fig. 25).
2. Assessment of the heart chambers in patients with atrial fibrillation or other rhythm disorders.
3. Studies prior to or following radiofrequency ablation to assess possible changes of the pulmonary veins and heart anatomy (65) (Figures 26, 27)
4. Identification of left atrial appendage thrombus. In patients with paroxysms of atrial fibrillation the presence thrombus in the left atrial appendage thrombus must be determined. Conventional method is transesophageal echocardiography. A more convenient alternative to the echocardiography is the MSCT.



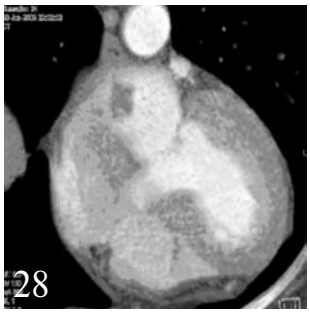
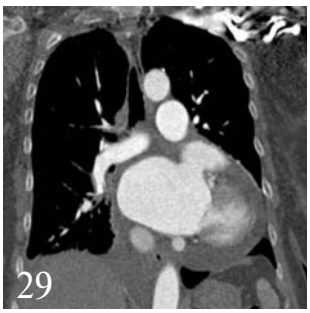


Figure 27. 64-MSCT of the pulmonary vein origin.
Figure 28. MSCT of the heart showing left atrial myxoma (arrow).



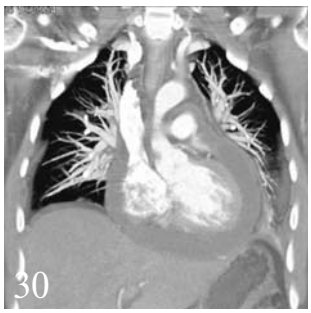


Figure 29, 30. Exudative pericarditis (exudate) (arrows).

Another application of the 64-MSCT is the diagnosis of cardiac tumors, myocardial infiltration due to amyloidosis, sarcoidosis (31, 53) (see Fig. 28). Within few minutes the MSCT will specify the location of a heart lesion and provide additional information

on coronary circulation with the assessment of pericardial structures and valves.

Exudative or restrictive pericarditis, as well as suspected pericardial diseases (including those due to myocardial inflammatory diseases) are also among the fields of application of the 64-MSCT (2) (see Figures 29, 30).

VI. Pulmonary vessels

Along with the heart, MSCT allows for visualization of other organs and systems, therefore, additional visualization of the pulmonary artery and the lungs (see Fig. 40) facilitates the diagnosis of a pulmonary embolism (PE) (19,52). The high speed and image accuracy provided by the 64-MSCT will help the doctor start the required therapy in proper time.

Figure 40. MSCT imaging of the heart, lungs, pulmonary veins and arteries.

1. Study procedure

MSCT angiography is based on the scanning technique during arterial phase (quick passage of the contrast medium). There's no special preparation to the study. MSCT can be performed on the outpatient basis. The patient lies supine (Fig. 41). After preliminary scanning for precise location of the target organs within the area of interest, the patient is administered IV iodine contrast and, after a short delay, ECG-controlled scanning is performed (slightly above 10 s in duration) with further computerized data processing. Complete study duration is approximately 15–20 minutes. Final data will then be available for analysis and 4-dimensional reconstruction.

Development of the method was facilitated by the following factors:

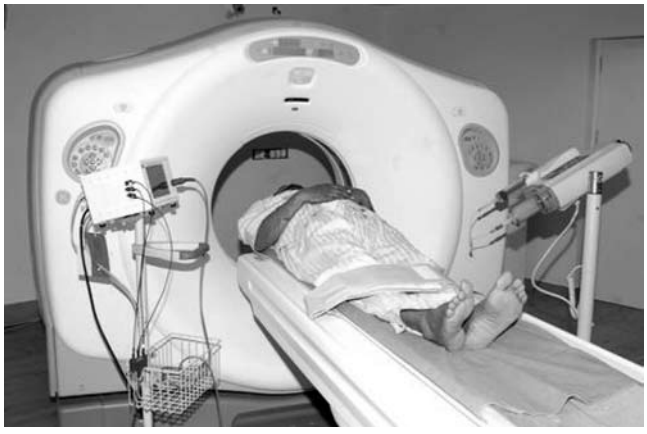


Figure 41. Study procedure.

1. high diagnostic value,
2. relatively simple procedure,
5. speed of image acquisition
4. patient convenience (no need for premedication, laboratory examination, etc.).
5. the fact that MSCT is the first noninvasive coronary imaging method, which doesn't require hospitalization and doesn't pose the risk of perioperative complications.

4. Comparison with other imaging methods

The following imaging methods are currently used to study heart and vessels in medical practice:

1. ultrasound (echocardiography, Doppler study, intravascular ultrasound study),
2. magnetic resonance tomography (MRT),
3. invasive angiography,
4. myocardial radionuclide imaging and
5. computed tomography (8-16-32-MSCT).

Most common CAD imaging methods are the stress tests. Direct comparison of the stress test and 16-MSCT performed by Dewey M. et al. (2006) in 80 patients showed significant benefit of the latter as regards to detection of coronary artery disease by invasive angiography: sensitivity 73% vs 91%, specificity 31% vs 83% (p = 0.039) (CAG was performed at study completion to provide detailed verification of CAD) (67). In conclusion, even the 16-MSCT has higher diagnostic value as compared to the stress tests for registration of coronary lesions.

Invasive AAG and CAG are known to be "the golden standards" of arterial imaging, therefore, introduction of the coronary MSCT has created another problem for the investigators — comparison of its results with invasive imaging methods (2, 5, 20, 47, 48).

Direct comparison of the 16-slice multidetector CT of coronary arteries to CAG showed, that the sensitivity, specificity, positive and negative predictive values were 85-89%, 98%, 90-91%, 96-98%, respectively (36, 37, 38, 41) with the incidence of motion artifacts being significantly higher than in 64-MSCT (22). These data suggested lower diagnostic value of the 16-MSCT as compared to the 64-MSCT (6,22,32). Garcia M.J. et al (2006) studied 187 patients with coronary CI over 600 and found, that the sensitivity of the 16-MSCT was 89% to 94% and the specificity 51% to 67%. The authors concluded, that the 16-MSCT wasn't an alternative to diagnostic CAG due to the high number of undiagnosed lesions (60).

As stated above, the diagnostic potential of the 64-MSCT is higher as compared to the 16-MSCT, therefore, the creation of an improved unit has put the question of whether the 64-MSCT is an alternative to CAG. Numerous studies dedicated to this problem have shown, that the 64-MSCT of coronary arteries has the following benefits over CAG in the diagnosis of hemodynamically significant stenosis (over 50%): sensitivity 94%–100%, specificity 95%–97%, positive predictive value 87%–97%, negative

predictive value 99%–100% (3, 20, 23, 30, 35, 47, 48). These results confirm the identity of MSCT and CAG for the diagnosis of coronary artery atherosclerosis (see Figures 5,42) and point to the possibility to use the high-speed CT instead of the invasive diagnostic study.

Modern 64-MSCT is not inferior to invasive methods, which are regarded as the "golden standards" (CAG and AAG) for coronary and peripheral arterial assessment, exhibiting a number of benefits over the selective CAG:

1. relatively simple diagnostic procedure,
2. absence of perioperative complications,
3. low study duration and information acquisition,
4. no need for hospitalization and
5. premedication, which eventually determines the patient convenience.

Along with the above mentioned benefits over CAG, MSCT gives an additional characteristic of an atherosclerotic plaque (detection of soft plaques, calcification degree, etc.) (2), determines the cardiac contractile function (by the values of diastolic and systolic volumes of the left and the right ventricles, as well as the precise calculation of the ejection fraction), reveals the areas of LV dyskinesis (during the 4-D reconstruction) with additional assessment of the heart anatomy and vessels. And, what is important for the prognosis, MSCT allows for determination of perfusion degree and the myocardial viability (2, 13, 24, 45, 50), which is particularly important for patients after myocardial infarction and patients with heart failure.

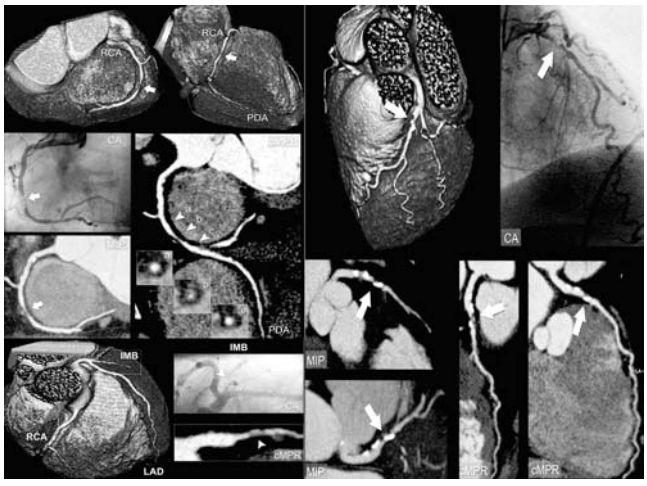


Figure 42. Direct comparison of selective CAG and 64-MSCT. Right picture: stenosis of the RCA middle portion (arrows), left picture: stenosis of the proximal LAD portion (large arrows), calcium inclusions within the lesion.

The next "golden" standard for the diagnosis of global or regional LV contractile function, is the MRT. Studies directly comparing the high-resolution MRT and the 64-MSCT have shown, that the results of heart assessment in patient after MI were statistically similar (13). The work by Baks T. has shown, that the 64-MSCT significantly determines the area of myocardial necrosis in comparison to the 1.5 T MRT.

Comparison of the two methods: ventriculography and the 64-MSCT used to assess the global LV function showed the statistical similarity between their results (21).

The comparison between the 64-MSCT and the intravascular ultrasound study in the internal carotid artery stenosis revealed significant benefit of MSCT as regards to plaque type verification (58).

Khare K.R.(2006) directly assessed the two methods (stress-echocardiography and 64-MSCT) demonstrating high efficacy of the latter for the diagnosis of chest pain origin (49).

Gerber T.C. et al (2005) found identity between the results of transthoracic echocardiography and the 16-MSCT for visualization of posterior mitral valve prolapse (61) (see Fig. 43), Alkadhi H. et al (2006) (62) and Gudrun M. et al (2006) (63) — for the assessment of the aortic valve. Authors show 100% sensitivity and 97.3% specificity of the MSCT for the diagnosis of valvular disease.

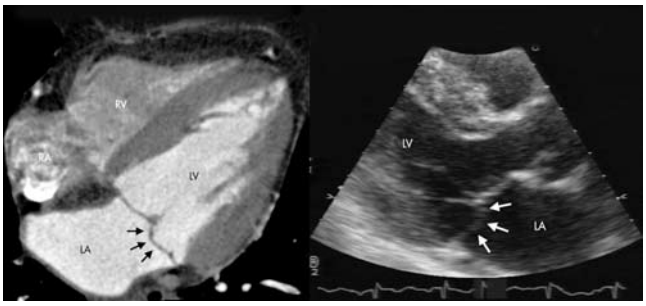


Figure 43. MSCT of the heart (left picture showing longitudinal slice) and echocardiography (parasternal longitudinal aspect, right picture). LA-left atrium; LV-left ventricle; RA-right atrium; RV-right ventricle. Prolapse of the posterior mitral valve cusp (arrows).

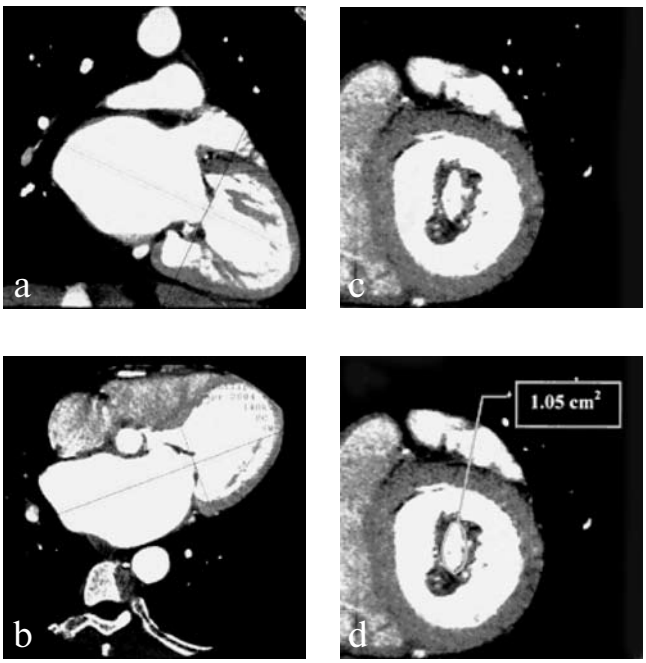


Figure 44. MSCT with contrast enhancement (white and black colors reversed to provide similarity with sonographic appearance). Adequate positioning of the longitudinal 2-chamber aspect (A), 4-chamber aspect (B), mitral valve aspect (C), mitral valve opening area (D).

Messika-Zeitoun D. et al (2006) compared the potential of transthoracic echocardiography and 16-MSCT to measure the stenotic mitral valve area and showed MSCT to be superior to echocardiography as

regards to measurement and anatomic imaging (see Fig. 44). The authors suggested to use this method in patients with compromised ultrasound window or prior to valve grafting (64).

Similar results were presented by Gilard M. et al (206) (59), who proposed to use MSCT in all patients prior to aortic valve grafting thus providing simultaneous assessment of the coronary reserve and the anatomic appearance of the aortic valve.

Monique R.M. et al. (2005) published the results of direct comparison made between the echocardiography and the MSCT in 42 patients prior to ablation of pulmonary veins: the measured diameters of the pulmonary veins at the origin were comparable, however, MSCT more frequently revealed additional branches of the pulmonary arteries (65).

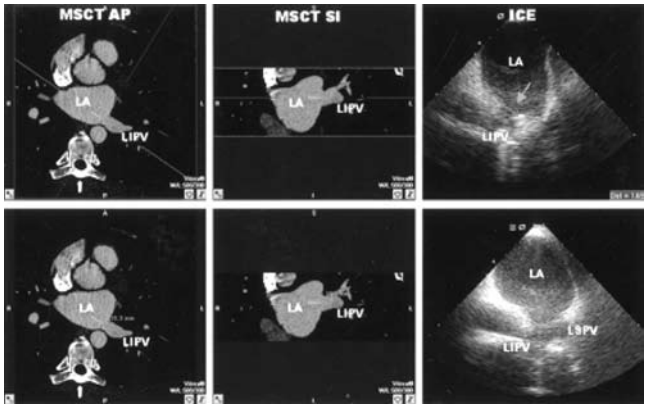


Figure 45. Changes of the left inferior pulmonary vein (LIPV) on the MSCT (left picture) and the transthoracic echocardiography (right picture). LA-left atrium. LSPV –left superior pulmonary vein.

Therefore, the MSCT is not inferior to the ultrasound study in the assessment of valvular disease and other cardiac structures, at the same time providing higher image quality and the possibility of simultaneous assessment of coronary arteries with plaque type differentiation and detection of myocardial perfusion disorders.

The potential of 16-MSCT in the assessment of intracranial aneurysms was demonstrated by Wintermark M. et al. Comparison with AAG revealed the sensitivity, specificity and accuracy of the 16-MSCT to be 94.8%, 95.2% and 94.9%, respectively (74).

Fraioli F. et al (2006) evaluated 50 patients with renal artery stenosis confirmed by angiography showing high sensitivity, specificity, accuracy, positive and negative predictive values of the MSCT (100%, 98.6%, 96.9%, 98.2% and 97.8%, respectively) (73). Authors recommend more extensive use of the MSCT for the assessment of this condition.

Some authors compared the efficacy of the 16-MSCT with conventional AAGF in PAD of the lower extremities (69, 70, 71, 72). Bui T.D. et al. (2006) demonstrated, that the sensitivity and specificity for all arterial segments were 86% and 90% for <50% stenosis, 79% and 89% for 50–99% stenosis, 85% and 98% for occlusion, respectively (69). Xiao-dan Zhang et al (2006) assessed arterial segments with > 50% stenosis and showed the sensitivity to be 94.3%, specificity — 98.4%, positive predictive value — 92.7%, negative predictive value — 98.7% (70). Thus, the comparability of 16-MSCT and AAG in PAD was advocated by many investigators (69,70,71,72), who also noted significantly shorter duration of the procedure: 2.5 +/– 0.3 min for 16-MSCT vs 37.5 +/– 5.2 for AAG, p = 0.006 (71).

Table 1 shows the results of comparison made between the diagnostic potentials of the 64-MSCT and other imaging methods (2):

Table 1. Comparison between the diagnostic potential of 64-MSCT and other imaging methods.

	Echocardiography with Doppler study	CAG	64-MSCT	MRT
Coronary anatomy	–	++++	+++	DM
Systolic function	++	Ventriculography	++	+++
Dyskinetic zones	++	–	++	NS
Cardiovascular anatomy	++	–	+++	+++
Plaque characterization	IVUS only	–	++	PM
Myocardial perfusion	–	–	++	+++
Myocardial viability	++	–	DM	+++

DM – further development of the method

As shown in the table, the 64-MSCT implies the cumulative potential of all other imaging methods, currently being the most universal diagnostic modality having preserved high safety, informativity and convenience for the patient.

5. Limitations of the MSCT

MSCT has a number of limitations similar to those of CAG and AAG. The contraindications are as follows (3, 17):

Absolute:

1. severe of moderate allergy to iodine.

Relative:

2. severe renal failure (serum creatinine over 1.5 μmol/l).
3. pregnancy
2. poor condition of the patient, including severe CHF.
5. patient's inability to lie in supine position.
6. patient's inability to hold the breath for 15 s.
7. severe obesity with body mass over 130 kg.
8. multiple myeloma
9. decompensated hypothyroidism
10. pheochromocytoma
11. chronic atrial fibrillation.
12. history of thromboembolism.

6. Discussion

Cardiovascular 64-MSCT includes new revolutionary options for all clinicians, providing noninvasive outpatient few-minute assessment of vessels 0.1 to 0.3 mm in any vascular territory (43) with maximum convenience for the patient.

In addition to the anatomic accuracy, the 64-

MSCT evaluates myocardial function, thus helping the physician in the choice of a treatment strategy and disease prognosis.

The introduction of 64-MSCT has decreased the need for diagnostic CAG and AAG for the assessment of coronary and peripheral arteries, therefore, the amount of invasive diagnostic procedures will substantially decline.

64-MSCT extends the diagnostic options for all clinicians, because many specialists refuse to refer patients to invasive studies fearing complications and discomfort associated with endovascular manipulations. In patients, who are ineligible for stress testing or show ambiguous results, the 64-MSCT would provide exact assessment of coronary artery tree, thus ruling out a large number of chest pain syndromes, which mimic the CAD (67). At the same time, the atherosclerotic changes revealed in the cardiovascular system allow for effective medical and, where necessary, surgical therapy (angioplasty or CABG). The MSCT results facilitate the choice of surgical strategy prior to coronary angioplasty, thus rendering great assistance to the interventional radiologists.

Modern 64-slice multidetector CT is not inferior to the invasive methods (CAG and AAG) in the assessment of peripheral and coronary arteries, besides, it has a number of obvious benefits, such as:

1. noninvasiveness,
2. no need for hospitalization,
3. the possibility of anatomic and functional assessment,
4. high speed of image acquisition,
5. patient convenience.

This vascular imaging method doesn't exclude the time-proved invasive methods, which comprise the diagnostic "golden standard", but acts as an adequate alternative. The 64-MSCT is the new assistant for a physician, including the potential of other diagnostic options: ultrasound study, MRT or angiography.

In addition to cardiovascular assessment, the 64-MSCT ensures adequate evaluation of other systems, providing an opportunity of hollow organ reconstruction, i.e. visual noninvasive colonoscopy or bronchoscopy. However, as compared to the 16- and 32-MSCT, which have lower speed, it shows no substantial superiority.

Further development of the innovative technology will result in the creation of 128 and 256-MSCT with higher diagnostic value (4), however, higher number of slices will greatly increase the patient radiation load.

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Complex Methods of Vascular Imaging to Determine the Strategy of Endovascular Coronary Procedures

Ye.P. Kokhan, V.A. Ivanov , M.Yu. Movsesiants[1], A.B. Shames, Yu.A. Bobkov, S.A. Terekhin, I.V. Trunin, V.L. Smirnov, A.V. Ivanov

Vishnevsky Central Military Hospital, Krasnogorsk, Russia

Keywords: coronary angiography, quantitative coronary angiography, intravascular ultrasound study, intravascular manometry, fraction flow reserve.

Abbreviations

CABG – coronary artery bypass grafting
IVM – intravascular manometry
IVUS – intarvascular ultrasound study
CAD – coronary artery disease
CAG – coronary angiography
MALA – minimum arterial lumen area
LCx – left circumflex artery
RCA – right coronary artery
LAD – left anterior descending artery
QCAG – quantitative coronary angiography
FFR – fraction flow reserve
PCI – percutaneous coronary intervention

Introduction

Coronary artery disease (CAD) is the most common cardiovascular disease in all developed countries. CAD accounts for over half of cardiovascular mortality rate. Russia has one of the highest CAD prevalence and mortality in Europe. All these factors determine the importance of modern CAD assessment (1).

Since the 1950-s coronary angiography (CAG) has become the "golden standard" of imaging and still has its high significance. However, even angiography fails to give the complete picture of coronary atherosclerosis.

Intravascular methods, including intravascular ultrasound study (IVUS) and intravascular manometry (IVM), comprise the modern options of non-radiological imaging and allow to obtain a novel level of data as compared to coronary angiography.

IVUS has a number of benefits over CAG: tomographic vs planimetric (i.e. silhouette) image; direct visualization of the lumen vs lumen shadow imaging; possibility to study all structures of the arterial wall with high resolution; no need for a contrast medium; direct measurement of the lumen diameter/area; direct vs. indirect visualization of the plaque, plaque type differentiation; potential to reveal intimal dissection with higher sensivity (2, 3). Toshihiko Nishioka et al. compared the results of IVUS with those of myocardial perfusion scintigraphy and showed, that the minimum arterial lumen area (MALA) predicting reversible perfusion disorders on myocardial perfusion scintigraphy is 4 mm2 (4).

Despite all benefits of IVUS, its use is limited by the fact, that this study provides only anatomic information. The IVM provides data on the function of vasculature and determines the hemodynamic significance of stenosis.

A special guidewire is used to measure the intra-arterial pressure distal to the site of stenosis. As a result, the fraction flow reserve is calculated (the ratio of the mean coronary pressure to the mean proximal coronary pressure determined using measurement guidewire at maximum hyperemia, i.e. 20 s following intracoronary administration of papaverin). In an intact coronary artery the fraction flow reserve (FFR) is 1.0 (5). Studies performed by Nico H.J. Pijls et al. comparing the results of stress testing to IVM have determined the threshold value of FFR to be 0.75. If the value is decreased below 0.75, the stenosis is defined as hemodynamically significant (6,7). Debates are ongoing as regards to the indications to FFR assessment and its diagnostic value.

Purpose of the study: complex assessment of 50–70% coronary artery stenoses to determine the strategy of endovascular intervention in patients with coronary artery disease.

Material and methods

A total of 73 CAD patients were assessed at Vishnevsky Central Military Clinical Hospital #3, including 65 men and 8 women with a mean age of 49 (± 5.5) years. Patient selection criteria were the presence of grade II–III stable angina (Canadian Cardiological Association, 1976) (1), 50–70% coronary stenosis on CAG and adequate medical therapy. The study didn't include patients with acute coronary syndrome. A total of 38 patients (52.1%) had a history of myocardial infarction with persisting grade II–III angina of effort, 35 patients (47.9%) had grade II–III angina of effort without a history of myocardial infarction.

Quantitative coronary angiography, intravascular ultrasound study and intravascular manometry

Catheterization of the coronary arteries was performed through standard femoral approach. In the beginning of the procedure all patients received a bolus of 5000 IU heparin. Additional heparin administration was done if the procedure duration was over

[1]Ivanov V. A., Movsesiants M. Yu.
Moscow Region 143420, Krasnogorsky district,
Arkhangelskoye post office, Vishnevsky Central Military Hospital,
Tel. (495) 564-63-76,
Fax (495) 561-93-25
e-mail: ivanov-angio@yandex.ru, mov-maik@rambler.ru
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90 minutes. Nitroglycerin (100 μg) was injected prior to coronary angiography and at 30-min intervals thereafter.

The results of quantitative coronary angiography (QCAG) were evaluated using automatic contour analysis system. A guidewire catheter was used as the calibration standard. Measurements were performed in an aspect optimally showing the stenosis and the reference segment of the artery. Minimum diameter at the site of stenosis, minimum and maximum distance between the lumen edge of stenosis and the external border of the artery (thus allowing to calculate the stenosis eccentricity index — EI) were measured. Angiographic length of stenosis was defined as the distance between its proximal and distal edges. Calcification was defined as the additional shadowing within the stenosis.

Coronary angiography was followed by IVUS procedure. A 0.014'' coronary guidewire was advanced through the stenosis and used to introduce the IVUS probe. The intravascular ultrasound imaging was performed while pulling back the probe (at a speed of 0.5 or 1 mm/s) automatically or manually. Measurements were conducted using integrated IVUS software. The lumen area was measured as the area within the intima borders (see Fig. 1), the vessel area — as the area circumscribed by the external elastic lamina (EEL) (8).

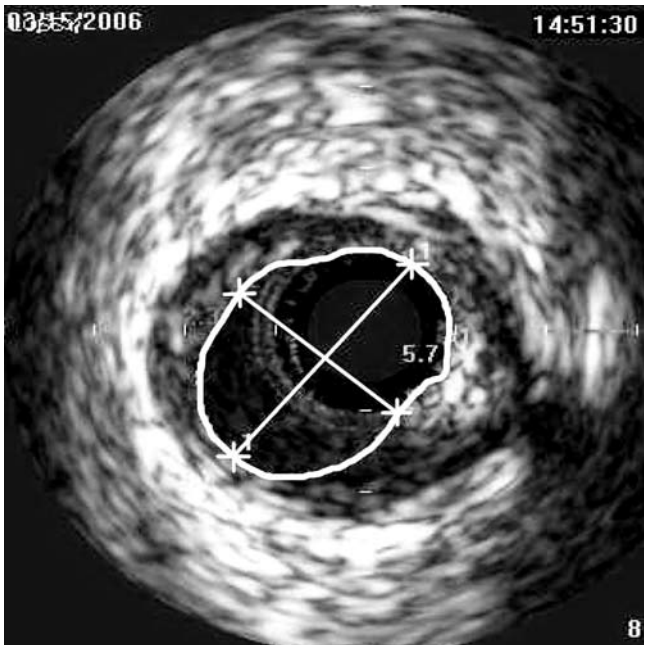

Figure 1. IVUS: contour showing the lumen area.

Aortic pressure was measured using 6F or 7F guidewire. The blood flow velocity was assessed in an angiographically unchanged or the reference vessel and distal to the target vessel stenosis using an original 0.014'' guidewire combining a standard Doppler probe on the tip and a standard pressure probe located 3 cm proximal to the tip of the measurement catheter. Sensors were positioned in a single location for all measurements during the procedure. Aortic pressure (Pa), coronary pressure (Pd) and the fraction flow reserve (FFR = Pd/Pa), as well as the ECG were continuously recorded at rest and during hyperemia after bolus intracoronary administration of papaverin (12 mg into the left coronary artery, 8 mg into the right coronary artery) (6) (see Fig. 2).

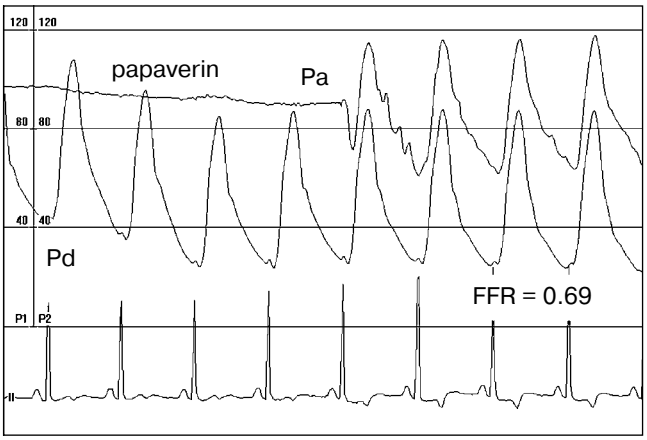

Figure 2. Aortic pressure curve (Pa), coronary pressure curve (Pd) and FFR value after intracoronary administration of papaverin.

CAG and IVUS were performed in all patients. Group I included 47 (64.4%) patients with a minimum lumen area below 4.0 mm2.

The remaining 26 patients with a minimum lumen area above 4.0 mm2 additionally underwent fraction flow reserve measurement.

Group II included 9 (12.3%) patients with FFR below 0.75.

Group III included 17 (23.3%) patients with FFR above 0.75.

As shown by CAG and IVUS, most commonly the stenoses were located in LAD (53.7%), RCA (26.6%) and LCx 19.7 %. The majority of stenoses were tubular (40.8%) (10 to 20 mm), diffuse stenoses (above 20 mm) accounted for 30.1%, discrete stenoses (below 10 mm) — for 29.1%.

Results

In Group I the minimum lumen area as measured by IVUS was below 4.0 mm^2. All patients in the group underwent PTCA with stenting under IVUS guidance.

In Group II the minimum lumen area as measured by IVUS was over 4.0 mm^2, whereas the FFR value was below 0.75. In this group patients also underwent PTCA with stenting under IVUS guidance with completion FFR measurement.

In Group III PTCA with stenting was not considered, because the minimum lumen area was high enough to ensure adequate flow velocity. Patients from the group received medical therapy and had been followed-up by a cardiologist.

The endovascular procedure results were assessed using CAG, IVUS or IVM (see Table 1).

Positive angiographic, ultrasound and manometry results were obtained in 100% of cases.

Clinical success was defined as an increase in exercise tolerance by 2 or more grades, complete

Table 1.

Criteria	Positive	Negative
Angiographic	– ≤20% residual stenosis – TIMI III blood flow – no signs of dissection	– >20% residual stenosis – blood flow < TIMI III – D–F dissections
IVUS MUSIC criteria	– stent implantation along the entire lesion – adequate in–stent lumen area – symmetrical stent expansion – *Minimum LD/maximum LD≥0.7	– stent implantation across partial length of the stenosis – insufficient in–stent lumen area – non–symmetrical stent expansion – Minimum LD/maximum LD <0.7
Manometry (FFR)	FFR over 0.9	FFR below 0.9

*LD – lumen diameter

reduction of angina and/or signs of ischemia. The treatment was defined as failed if there was no increase in exercise tolerance, as well as in cases of angina recurrence during in-hospital stay (see Table 2).

Table 2.

Groups I and II	Patients	
	n	(%)
Increase by ≥2 grades	52	(93 %)
Increase by 1 grade	3	(5.3 %)
Unchanged grade of angina	1	(1.7 %)

In Group III the assessment was performed within 6 months after surgery according to the presence of major or minor cardiac events, including recurrent ischemia, acute myocardial infarction, repeated PTCA or CABG, death (see Table 3).

Table 3.

Values during the 6-month period	Patients	
	n	(%)
Unchanged	16	(94 %)
Recurrent ischemia	1	(5.9 %)
Acute myocardial infarction	0	0
Repeated PTCA	0	0
CABG	0	0
Death	0	0

Among the 17 patients in Group III one had angina recurrence at 4 months postoperatively. Follow-up CAG, IVUS and IVM revealed the following findings (see Table 4).

Table 4.

	Baseline	at 4 months
QCAG (%)	LAD stenosis 55 %	LAD stenosis 65 %
IVUS (MALA, mm^2)	4.4	4.1
FFR	0.81	0.74

The patient underwent PTCA with stenting guided by IVUS and FFR measurement. The result was positive on angiography, IVUS and manometry. During hospital stay the exercise tolerance increased by 2 grades.

Discussion

Patients with 50–70% coronary artery stenosis were analyzed according to the results of the complex assessment. We believe that the angiographic appearance of such lesions is most ambiguous for the subjective assessment by an interventional radiologist (5). This can result in diagnostic errors and, subsequently, unreasonable stenting. Therefore, CAG should be accompanied by more reliable methods of vascular imaging, which provide more detailed anatomic and functional characterization of the coronary lesions.

Based on the results of multi-center trials (COMIUS, MUSIC, SIPS, CRUISE) and projects performed in Vishnevsky Central Military Clinical Hospital we conclude, that IVUS determines the true coronary lumen area, anatomic structure of the plaque, residual lumen area and the lesion length. This method allows for selection of an adequate stent diameter and length during the procedure, assessment of the stent expansion and the in-stent lumen area, additional angioplasty under high pressure in case of insufficient in-stent lumen area. Comparison of the long term results shows, that these criteria significantly decrease the rate of restenosis (8, 9, 10,11).

One of the basic criteria for patient selection in our study was the minimum arterial lumen area as measured on IVUS. Minimum lumen area below 4 mm^2 is an unambiguous indication to PCI to prevent irreversible myocardial changes (4). Minimum arterial lumen area above 4 mm^2 seems to require additional characterization of the stenosis significance.

The major indications to FFR measurements are the so-called «intermediate» coronary stenoses (30 to 70%), multi-vessel disease, stenosis at the artery origin and diffuse stenoses. The FFR doesn't depend on the heart rate and the blood pressure. Its normal value is 1 and the well-known threshold for clinically significant stenosis is 0.75 (6,7). After PCI the FFR value might be 0.9 or above (12). A prospective multi-center study (DEFER) showed, that there's no improvement of the long-term prognosis after PTCA with stenting in patients with FFR above 0.75 (13).

Functional assessment of stenosis was performed in groups II and III to detect the IVUS threshold values of the arterial lumen area. In 9 cases (12.3%) this assessment allowed for detection of patients necessitating PTCA with stenting and in 16 cases allowed to decline the intervention (22%).

Conclusions

1.The complex assessment of coronary arteries — angiography, intravascular ultrasound study and intravascular manometry — is a highly informative method of anatomic and functional evaluation of coronary vasculature currently defined as the modern diagnostic standard in CAD patients.

2. In case of ambiguous angiographic findings the ultrasound study can be used to evaluate coronary lesions.

3. Measurement of the fraction flow reserve in

case of threshold values on IVUS to provide more accurate assessment of the coronary pool.

4. Complex approach provides more precise determination of indications to stenting and prevents inadvertent stenting.

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Echocardiography in Cardiac Tamponade

I.E. Manykin, G.E. Belozerov[1], Yu.A. Radchenko
Department of Endovascular Methods of Diagnostics and Treatment,
N.V.Sklifosovsky Research Institute of Emergency Medicine, Moscow, Russia

Cardiac tamponade is a major hemodynamic disorder associated with the rapid development of clinical signs of acute cardiovascular failure. Compression of cardiac chambers by gas or liquid accumulated in the pericardial cavity is believed to cause cardiac tamponade (1, 2, 4).

Unless adequately treated cardiac tamponade is fatal. Therefore, timely diagnosis of cardiac tamponade is critical in emergency medicine. This is achieved by a range of clinical and diagnostic techniques, with echocardiography playing an important role (3). However, few clinical studies have focused on ultrasonic evaluation of intracardiac circulation in cardiac tamponade.

The objective of this study was to evaluate the changes of intracardiac circulation in patients with cardiac tamponade of different origin.

Material and methods

10 patients were evaluated by echocardiography. Two patients were admitted to N.V. Sklifosovsky Research Institute of Emergency Medicine with suspected dissecting aneurysm of the ascending aorta. Three patients had incised and stab wounds of the left chest. Two patients were hospitalized because of blunt thoracic trauma. One case presented with a cauterant poisoning. Two patients had mediastinitis. Based on clinical findings, physical examination and X-ray studies cardiac tamponade was suspected in each of these patients.

Echocardiography was performed using HP Sonos 100 CF, GE 500 PRO, ESAOTE Idea and Megas (Italy) scanners. In addition to evaluating the pericardial cavity the examination included assessment of left ventricular end-diastolic and end-systolic volumes (LV-EDV and LV-ESV, ml), overall contractility of the left ventricle (LV-EF, %), stroke index (SI, ml/m^2) and cardiac index (CI, L/min/m^2). Apical view was used to assess right ventricular end-systolic and end-diastolic areas (RV-EDA, cm^2 and RV-ESA, cm^2), and to calculate the right ventricular fractional area change (RV-FAC, %). Left parasternal view was used to evaluate the right ventricular end-diastolic dimension (RV-EDD, cm) and the pulmonary annulus diameter (PAD, cm). Pulsed Doppler study was used to measure the pulmonary artery stroke index (PA-SI, ml/m^2) and pulmonary artery flow (minute volume) (PAF-MV, L/min/m^2).

[1]G.E.Belozerov
Dept. of Endovascular Methods of Diagnostics and Treatment,
N.V. Sklifosovsky Research Institute of Emergency Medicine,
Russia, 129010, Moscow, B. Sukhrevskaya square, 3.
Phone. (007 495) 280 45 79
Fax (007 495) 921-02-02
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The volume of fluid in pericardial cavity was measured by bi-dimensional echocardiography from the 4-chamber apical view.

All patients underwent surgery. In one case urgent thoracotomy was performed. In two cases an anterior mediastinal drain was placed. In seven cases a pericardial drain and a pericardial catheter were placed.

Diagnostic pericardiocenthesis with subsequent catheterization was performed according to Brock procedure (5th intercostal space on the left midclavicular line). A 6F Pig-tail catheter with several side holes was used. The location of the catheter tip was verified by injection of a water-soluble contrast agent.

Results

The study revealed inconsistencies in clinical findings, X-ray findings and echocardiography results. In each case significant enlargement of cardiac margins and decreased cardiac pulsation were revealed on X-ray.

Clinical symptoms of cardiac tamponade (enlarged cardiac dullness, muffled heart tones, tachycardia, hypotension and distension of the neck veins) were seen only in 5 cases (2 patients with mediastinitis, two patients with blunt chest trauma and one patient with an incised and stab wound of the chest).

Echocardiography revealed a separation of the pericardial leaflets ranging from 10 to 40 mm in 8 patients. The volume of pericardial fluid ranged from 200 to 1400 ml. In two cases there was no fluid in the pericardial cavity, but there were echocardiographic signs of a mass in the anterior mediastinum. Severe impairment of intracardiac circulation was seen in only 4 cases (one patient with chest wound, two patients with external compression of the right ventricle and one patient with chest trauma). The rest of the patients presented with satisfactory intracardiac circulation despite the fluid found in the pericardium.

One of the causes of tamponade was intrapericardial bleeding due to heart trauma and heart injury. Clinical signs of blood tamponade were observed within the first hours of trauma or injury.

Similar clinical situation was seen in patient P., 25 years old, who was admitted to the ICU of the institute two hours after a motorcycle accident. Clinical findings included signs of acute cardiac failure: tachycardia, hypotension, dyspnea and skin paleness. ECG showed decreased R-wave amplitude in V1 through V6. X-ray showed increased heart size with smoothened waist of the heart. Urgent admission echocardiography revealed separated pericardial leaflets of up to 10 mm along the ventricular contours,

with this separation corresponding to 150–200 ml of liquid (Fig. 1).

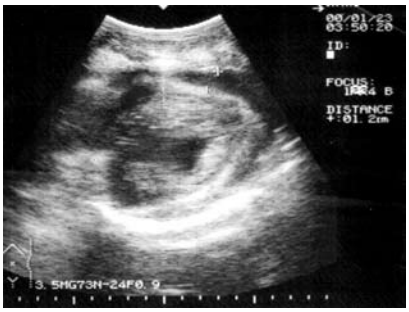

Figure 1. Echocardiogram showing hemopericardium.

The distance between the pericardial leaflets increased as the examination proceeded. The examination revealed a decrease in size of both ventricles (LV-EDV – 79 ml, RV-EDD – 1.8 cm), a decrease in stroke index and cardiac index, and a decrease in pulmonary artery volume flow. The patient maintained high myocardial contractility of both ventricles (LV-EF – 75 %, RV-FAC – 47 %). Urgent drainage of the pericardial cavity was performed. 200 ml of fresh blood were removed. Following the elimination of blood tamponade the patient's intracardiac circulation improved significantly (Table 1).

Table1. Parameters of circulation of patient P. before and after suction of 200 ml of blood from the pericardial cavity.

	Control	I	II
HR per, min	70.0 ± 4.9	100	101
LV-EDV, mL	124.2 ± 4.3	79	120
LV-EF, %	65.2 ± 2.7	75	70
SI, ml/m^2	41.4 ± 6.0	30	42.5
CI, L/min/m^2	3.1 ± 0.5	3.7	4.2
RV-EDD, cm	2.4 ± 0.02	1.8	2.6
RV-EDA, cm^2	17.5 ± 1.2	18.4	24.7
RV-FAC, %	45.0 ± 3.7	47	40
PA-SI mL/m^2	39.1 ± 1.8	20	29
PAF-MV, L/min/m^2	2.7 ± 0.4	2.0	2.9
E/A	1.9 ± 0.07	1.1	1.4

I — Before drainage.
II — After pericardial drainage.

The severity of impairment of circulation depended on both the rate of increase in pericardial fluid volume and the type of this fluid. Figure 2 presents an echocardiogram of patient Sh., 22 years, old who was admitted to the Institute with an incised and stab wound to the left chest. On admission the patient's was fair. Blood pressure and heart rate (HR) were normal. X-ray showed signs of left hemothorax. Admission echocardiography revealed no separation of the pericardial leaflets. Intracardiac hemodynamic parameters were normal. Primary surgical management of the wound was performed and a left pleural drain was placed.

Later the patient's condition suddenly worsened. Clinical signs of acute cardiac failure appeared.

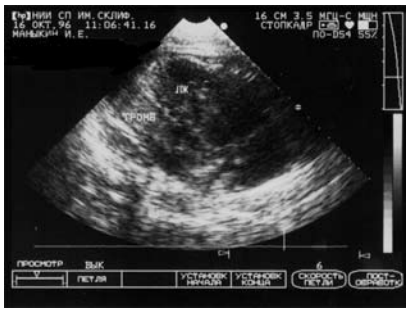

Figure 2. Blood clot in the pericardial cavity.

Repeat echocardiography showed a massive blood clot in the pericardial cavity which caused deformation of the ventricles. Diastolic diameter of the right ventricle decreased from 4.9 cm to 2.7 cm. Diastolic area of the right ventricle decreased from 28.4 to 14.7 cm^2. Among other findings there were a decrease in pulmonary artery volume flow, marked impairment of filling of the left ventricle and a decrease in diastolic volume of the left ventricle from 130 ml to 78.6 ml. Stroke index and cardiac index also decreased. Like in the first case, myocardial contractility of both ventricles remained unchanged and even increased.

Table 2. Parameters of circulation of Patient Sh. on admission and before surgery.

	Control	I	II
HR per, min	70.0 ± 4.9	100	102
LV-EDV, mL	124.2 ± 4.3	130	78.6
LV-EF, %	65.2 ± 2.7	79	79
SI, ml/m^2	41.4 ± 6.0	53.0	31.1
CI, L/min/m^2	3.1 ± 0.5	5.3	3.3
RV-EDD, cm	2.4 ± 0.02	4.9	2.7
RV-EDA, cm^2	17.5 ± 1.2	28.4	14.7
RV-FAC, %	45.0 ± 3.7	47	48
PA-SI mL/m^2	39.1 ± 1.8	48	29
PAF-MV, L/min/m^2	2.7 ± 0.4	4.8	2.3
E/A	1.9 ± 0.07	1.5	0.5

I — On admission.
II — Before surgery.

Intraoperatively, a 150 ml blood clot was found in the pericardial cavity. The blood tamponade was caused by a penetrating wound of the right ventricle. The wound was successfully closed. Table 2 shows changes in the hemodynamic parameters of patient Sh. on admission and immediately before surgery.

In two cases, clinical signs of cardiac failure were caused by external heart compression. Figure 3 shows an echocardiogram of patient F., 45 years old, with an incised and stab wound of the left lung. The post-operative period was complicated by an abscess of the anterior mediastinum. The mass in

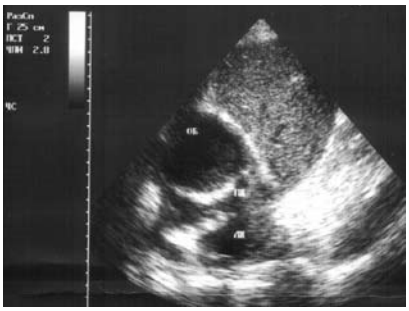

Figure 3. Anterior mediastinal abscess.

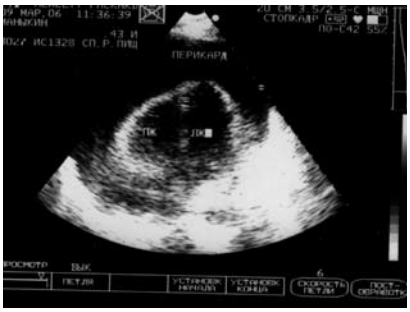

Figure 4. Echocardiogram in pericarditis.

the anterior mediastinum caused deformation of the right ventricular cavity and was accompanied by typical hemodynamic changes seen in cardiac tamponade (Table 3). 250 ml of pus were drained from the anterior mediastinum. Following the drainage, hemodynamic parameters improved significantly and clinical signs of acute cardiac failure disappeared.

Table 3. Parameters of circulation of patient F. before and after suction of 250 ml of pus from the anterior mediastinum.

	Control	I	II
HR per, min	70.0 ± 4.9	100	101
LV-EDV, mL	124.2 ± 4.3	70.8	117.9
LV-EF, %	65.2 ± 2.7	72	79
SI, ml/m^2	41.4 ± 6.0	25.4	46.6
CI, L/min/m^2	3.1 ± 0.5	2.4	4.7
RV-EDD, cm	2.4 ± 0.02	1.7	2.6
RV-EDA, cm^2	17.5 ± 1.2	13.3	24.3
RV-FAC, %	45.0 ± 3.7	43	45
PA-SI mL/m^2	39.1 ± 1.8	8.3	18
PAF-MV, L/min/m^2	2.7 ± 0.4	0.8	1.9
E/A	1.9 ± 0.07	1.1	1.5

I — Before drainage.
II — After abscess drainage.

On repeated examination a significant increase in the size of both right and left ventricular cavities and marked elevation of stroke index, cardiac index and pulmonary artery volume flow were seen.

Although fluid or gas in the pericardial cavity is considered to be one of the major echocardiographic symptoms of tamponade, even considerable separation of the pericardial leaflets may not be associated with severe hemodynamic disorders.

Figure 4 presents an echocardiogram of patient K., 43 years old, with mediastinitis complicated by purulent pericarditis with a gradual increase in the amount of fluid in the pericardial cavity.

Echocardiography revealed 1400 ml of fluid with particles of moderate echogenicity in the pericardial cavity. The size and contractility of the right ventricle were normal. Left ventricular function parameters and volume flow through the pulmonary artery were quite high. Clinical signs of acute cardiac failure were absent (Table 4). Doppler study revealed impaired diastolic filling of the left ventricle shown by a decrease in E/A ratio to 1.09. Thus, slow accumulation of fluid in the pericardial cavity definitely affected cardiac function, but did not cause severe intracardiac hemodynamic disorders associated with low cardiac output syndrome.

Table 4. Parameters of circulation of patient K. before and after suction of 1400 ml of pus from the pericardial cavity.

	Control	I	II
HR per, min	70.0 ± 4.9	87	76
LV-EDV, mL	124.2 ± 4.3	78.6	118
LV-EF, %	65.2 ± 2.7	76.2	86.3
SI, ml/m^2	41.4 ± 6.0	27.4	46.2
CI, L/min/m^2	3.1 ± 0.5	2.4	3.9
RV-EDD, cm	2.4 ± 0.02	2.86	3.2
RV-EDA, cm^2	17.5 ± 1.2	19.5	28.3
RV-FAC, %	45.0 ± 3.7	51	38
PA-SI mL/m^2	39.1 ± 1.8	27.3	45.3
PAF-MV, L/min/m^2	2.7 ± 0.4	2.35	3.9
E/A	1.9 ± 0.07	1.09	1.37

I — Before drainage.
II — After drainage.

1400 ml of pus were drained from the pericardial cavity. Removal of this fluid led to an increase in aortic and pulmonary flow and to an improvement of left ventricular diastolic function with an increase in E/A ratio to 1.37.

Discussion

The study shows that the separation of pericardial leaflets is not a reliable sign of blood tamponade. Blood tamponade was associated with decreased sizes of cardiac chambers and certain hemodynamic signs such as decreased stroke index and cardiac index, decreased pulmonary volume flow, and altered diastolic filling of the left ventricle. The changes in these parameters lead to the development of clinical signs of acute cardiac failure. Early decrease of myocardial contractility in cardiac tamponade was absent. This type of intracardiac hemodynamic impairment was associated with a fast increase in

the amount of pericardial fluid or with a change in its physical properties (formation of a blood clot).

Similar hemodynamic changes were seen in mechanical external heart compression, i.e. in anterior mediastinal abscess.

Elimination of blood tamponade or external heart compression resulted in fast normalization of intracardiac hemodynamic parameters.

Conclusions

1. Cardiac tamponade is associated with a marked decrease in both systemic and pulmonary flow.
2. Myocardial contractility of the ventricles in the early period of cardiac tamponade remains normal.
3. The rate of fluid accumulation and formation of blood clots are factors that affect the pathogenesis of blood tamponade.
4. Timely surgical elimination of cardiac tamponade results in fast normalization of intracardiac hemodynamic parameters.

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Successful Urgent Endovascular Procedure in a Female Patient with Critical Stenosis of the Left Main Bifurcation: Case Report

D.G. Iosseliani, I.Yu. Kostianov[1], D.G. Gromov, A.V. Kononov, M.V. Diagileva, N.V. Kuchkina
City Center of Interventional Cardioangiology, Moscow, Russia

In 1912 J.Herric gave the first description of the left main coronary artery (LMCA) lesion detected during autopsy of a 55-year old man, who died from myocardial infarction causing cardiogenic shock (1). Left main stenosis (> 50% considered hemodynamically significant) is found in 3 to 5% of cases of coronary arteries catheterization for acute coronary syndrome, myocardial infarction. LMCA stenosis is an extremely unfavorable predictive factor in medically treated patients and is associated with high mid-term (within 1 year postoperatively) mortality rate (2).

Particularly disastrous form of the LMCA lesion is the occlusion, which, according to different authors, is found in 0.01 to 0.7% of all patients (3). In the majority of cases acute left main occlusion is fatal. Early reperfusion of the obstructed LMCA using endovascular procedures (angioplasty) and intraaortic balloon counterpulsation (IBC) is considered a "life-saving" operation for patients with acute Q-wave myocardial infarction commonly accompanied by cardiogenic shock (4).

Till recently LMCA lesions have been the direct indication to CABG (5). However, many authors report, that 11% to 19% of patients with LMCA stenosis (depending on the age group) are inoperable and are at extremely high risk of hospital mortality (6-8). Factors, such as comorbidities (diabetes mellitus, obesity, chronic systemic diseases, etc.), decreased myocardial contractile function (LVEF < 30%), grade 3–4 mitral regurgitation due to acute myocardial infarction, serum creatinine > 2.0 mg/dl, small or calcified coronary arteries substantially worsen the unfavorable prognosis of left main lesions. The 3-month mortality rate after the detection of the left main stenosis reaches 24.2% in this patient group with cardiac mortality accounting for 20.2% (10.8% deaths due to myocardial infarction, 9.4% deaths after CABG) (7-9).

Indications for endovascular treatment of "unprotected" stenotic left main coronary artery have been extended, endovascular procedures are being increasingly adopted in patients, who used to be regarded as absolute candidates for direct myocardial revascularization. Today stenting for LMCA stenosis is associated with good immediate success rate when performed for proper indications with hospital mortality rate being 0 to 2%.

Clinical implementation of the improved stents, particularly with antiproliferative and antithrombotic coating, etc., has substantially improved the quantitative and qualitative outcomes of the endovascular procedures performed in unprotected stenotic left main coronary artery (10-13). Studies SIRIUS and TAXUS II have revealed significantly decreased long-term restenosis rate after stenting of the unprotected LMCA as compared to other data, indicating 50 to 60% incidence of the long-term restenosis with regular stents (14).

Moscow City Center of Interventional Cardioangiology has an experience with the stenting of unprotected left main coronary artery in 16 patients, including 4 cases of successful urgent myocardial revascularization performed for acute LCMA occlusion and 12 cases of elective stenting for critical LCMA stenosis. Stenting of the unprotected LCMA was usually performed using matrix stents with angiographic success being 100%, all the procedures of unprotected LCMA stenting went on without complications.

All patients had clinical improvement during hospital stay. Mid-term follow-up assessment was performed in all patients after LCMA stenting with bare metal stents; the rate of LCMA restenosis was 50%. The long-term total and cardiac survival rate after unprotected left main stenting was 87.5%.

The article reports a case demonstrating successful stenting of the left main bifurcation stenosis in patients with acute coronary syndrome. Endovascular treatment was used in cases of drug-resistant unstable angina and in patients with high risk of complications associated with CABG (obesity, diabetes mellitus, neoplasm and prior right nephrectomy).

Patient R., female, aged 61, case history 1149, was urgently admitted to the Coronary care unit of the Moscow City Center of Interventional Cardioangiology on 24.03.06 at 9:35 p.m. and presented with severe chest pain. ECG showed circular subendocardial ischemia. Differential diagnosis on admission included non-Q-wave circular myocardial infarction and severe unstable angina.

History data: the patient gave a long history of hypertension (maximum BP values 285/145 mm Hg) irregularly treated with hypotensives. There was no history of MI or stroke. Chest pain episodes were first detected in 2002, since January 2006 these episodes became more frequent, prolonged

[1]I.Yu.Kostianov
Moscow City Center of Interventional Cardioangiology
Russia, 101000, Moscow, Sverchkov per., 5
Phone: 007 495 624 96 36
Fax: : 007 495 624 67 33
e-mail: davidgi@mail.ru
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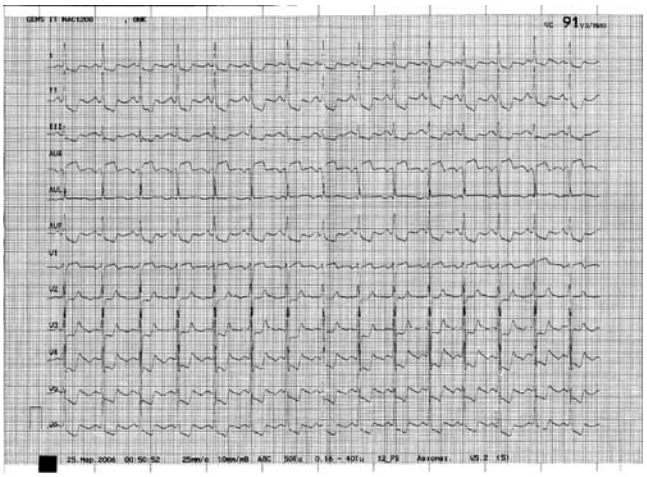
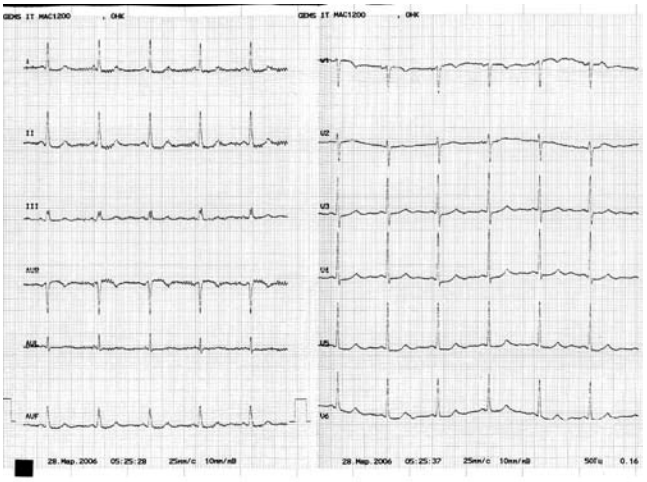


ECG 1



ECG 2

Figure 1.

and severe, the patient had decreased exercise tolerance, chest pain at rest, increased consumption of nitrates. Between February and March 2006 she was treated in the Municipal Hospital N55 for unstable angina. Despite the in-hospital and, subsequently, outpatient therapy with disaggregants, nitrates, beta-blockers, calcium channel blockers, ACE inhibitors and metabolic agents, the patient still had low-grade angina and episodes of chest pain at rest, there was a trend to progression. Chest pain occurred up to 8–10 times daily while additional oral nitromint-spray and nitroglycerin tablets gave short effect. The patient's condition was aggravated by comorbidities: primary multiple carcinoma of the right kidney (right nephrectomy in 2001) and the right breast (sectoral right mastectomy in 2003 followed by radiation therapy), moderate subcompensated type 2 diabetes mellitus. On 24.03.2006 she developed a prolonged chest pain episode not reduced by nitrates, for which she received emergency care. In order to reduce pain she was administered additional doses of nitromint-spray, combined anaglesics (IV bolus of 2.0 ml tramal). The patient was admitted to the Moscow City Center of Interventional Cardioangiology at 9:35 p.m.

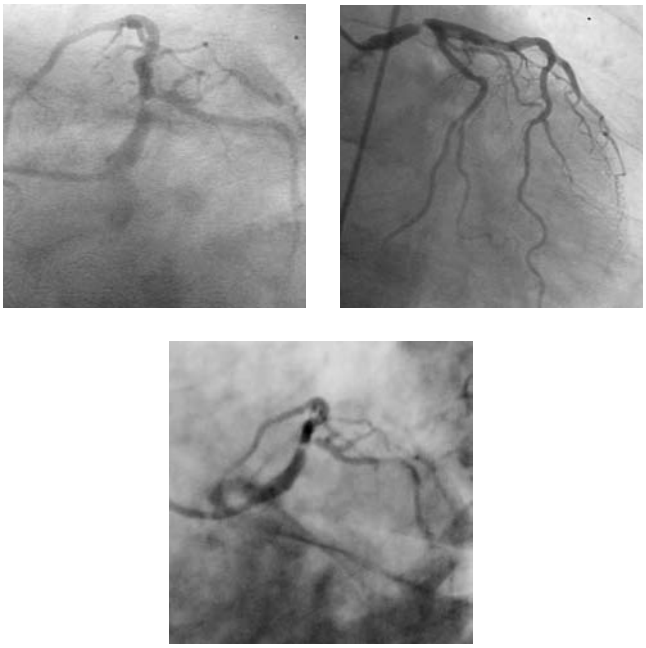


Figure 2.

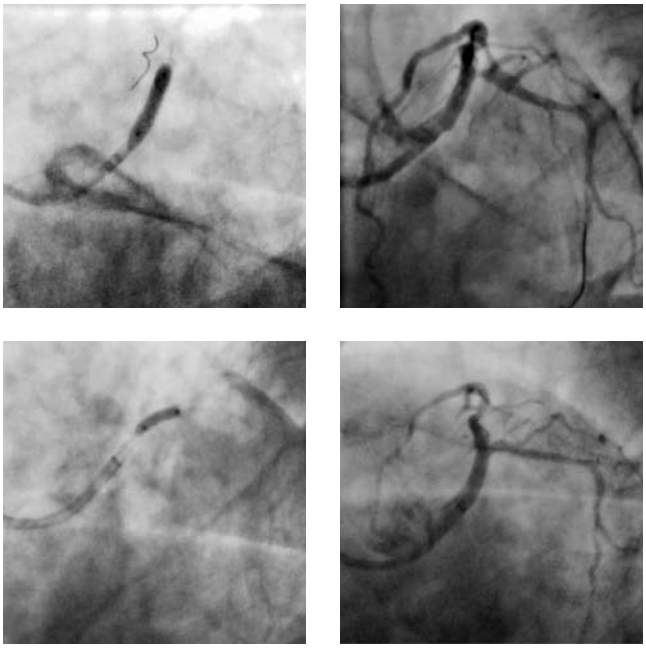


Figure 3.

State on admission: moderately severe. Skin appears pale, moist. Breath sounds harsh, without rales. Respiration rate 17 per min. Hear sounds muffled, regular, no murmur. Accentuated second tone over the aorta. HR 72 bpm. BP 130/80 mm Hg. Liver not extending beyond the costal margin. Neurological state unremarkable. ECG showed regular sinus rhythm with no axis deviation. HR 68 bpm. Horizontal ST depression up to 3 mm in leads I, II, III, AVF, V3–V6. Blood sample showed leukocytosis up 11.8×10^9 / l and elevated serum glucose (up to 18.2 mmol/l).

In the ICU the patient was initially treated with IV infusion of 1% nitroglycerin at 100 μg/min, 1500 U/h heparin, oral acetylsalicylic acid 100 mg daily, bisoprolol 10 mg daily, amlodipin 10 mg daily, ticlopidin 500 mg daily, insulin 16 U daily. Between

25.03.06 and 26.03.06 during IV infusion of nitrates the patient had multiple episodes of severe chest pain accompanied by unfavorable ECG changes, namely the progressive ST depression in leads I, II, III, AVF, V2–V6 (up to 7 mm). (see Fig. 1, ECG I). Nitroglycerin was increased to 250 μg/min, narcotic analgesics were administered twice (1.0 ml 2% promedol IV). Due to persisting chest pain resistant to the combined antianginal therapy, as well as for the evaluation of coronary circulation and determination of further treatment strategy, the patient underwent diagnostic selective coronary angiography on 26.03.06, which showed 90% eccentric left main stenosis involving the bifurcation, 95% stenosis of the LCx origin, diffuse hemodynamically non-significant lesions of LAD and RCA (see Fig.2, angiographic images 1–3).

On 27.03.06 at 9:15 a.m. the patient underwent a procedure in the Department of endovascular diagnostics and treatment. Direct stenting (Bx Sonic 4 × 18 mm) of the left main coronary artery extending to the LAD origin was performed followed by angioplasty and stenting (Bx Sonic 2.75 × 8 mm) of the LCx origin (through the mesh of the stent located in the left main and extending to LAD). The procedure gave good angiographic outcome (see Fig.3, angiographic images 4–7). Control study revealed TIMI 3 antegrade flow. There were no signs of residual stenosis, intimal dissection or distal embolization. Thus, this female patient underwent complete endovascular myocardial reperfusion.

For the purpose of follow-up and treatment she was reallocated to the CCU. After the endovascular procedure the patient showed favorable ECG changes, namely the normalization of ST in all leads, chest pain didn't recur (see Fig.1, ECG II). Therapy was changed to oral nitrates (isosorbide mononitrate 80 mg daily, low-dose bisoprolol 5 mg daily), heparin was discontinued. Physical activity regimen was extended and the patient reallocated to the Department of Cardiology on 30.03.06. There were no further episodes of chest pain, the patient had good exercise tolerance and normal hemodynamic values. While in the Department of Cardiology she underwent echocardiography and 24-hour monitoring. EchoCG revealed LVEF 77%, normal contractile function, end-systolic LV volume 32 cm2, end-diastolic LV volume 144 cm2, normal aorta and valves. 24-hour monitoring showed no ischemic changes of the ST segment.

On the base of echocardiographic findings, the absence of signs of elevated serum cardiospecific markers, and positive ECG changes, the following diagnosis was formulated: CAD with type II unstable angina, 90% left main stenosis, 95% LCx stenosis, prior stenting of the left main with PTCA and stenting of the LCx; stage 2 hypertension, moderate type II diabetes mellitus; primary multiple carcinoma; right mastectomy in 2001; right nephrectomy in 2003.

In the Department of Cardiology the patient continued through disaggregants, antianginal agents, hypotensives and hypoglycemic agents. During the treatment the chest pain episodes didn't recur, hemodynamic indices and laboratory findings remained normal. On 5.04.06 the patient was discharged in stable condition. A follow-up assessment was recommended at 6 months.

Therefore, this example demonstrates high efficacy of the endovascular treatment for LCMA stenosis in a female patient with severe comorbidities (obesity, diabetes mellitus, neoplasm and history of right nephrectomy), high risk of unfavorable immediate outcome of medical therapy and extremely high risk of open surgery. Successful myocardial reperfusion provided immediate stabilization of the clinical condition and minimization of medical therapy, which is crucial for a patient with a single kidney and metabolic disorders. We believe that endovascular interventions are currently a reasonable and, sometimes, the only alternative to direct myocardial revascularization in such patients.

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Successful Urgent Endovascular Procedure in a Patient with Subtotal Stenosis of the Unprotected Left Main Coronary Artery Associated with Subtotal Stenosis of the Right Coronary Artery

L.S. Barbarash, A.A. Azarov[1], M.A. Lobanov, B.L. Khaes

Research and Manufacturing Problem Laboratory of Reconstructive Cardiovascular Surgery of the Siberian Branch of the Russian Academy of Medical Sciences, Kemerovo, Russia

Endovascular procedures for the stenosis of the left main coronary artery (LMCA) are extremely difficult. The lesion of LMCA is associated with very unfavorable prognosis (1–5).

The purpose of this paper consists in the demonstration of the feasibility of urgent endovascular myocardial revascularization for subtotal lesion of LMCA and the right coronary artery (RCA).

Our Research and Manufacturing Problem Laboratory of Reconstructive Cardiovascular Surgery of the Siberian Branch of the Russian Academy of Medical Sciences has an experience with five successful procedures of percutaneous transluminal angioplasty (PTCA) on the protected LMCA.

The patient K., male, aged 67 (case history N990), was admitted to the Department of endovascular diagnostics and treatment on April 27, 2006, for coronary angiography with the diagnosis: Coronary heart disease. Angina pectoris, NYHA class III. Postinfarction cardiosclerosis (MI in 1995). Chronic heart failure I. Functional class II. Arterial hypertension, stage III, risk 4.

History data: arterial hypertension of more than 10 years duration, with AP elevations up to 220/120 mm Hg. The patient did not receive regular hypotensive therapy. The familial history is not burdened, the patient has no diabetes mellitus. In 1995, without prior angina pectoris, the patient had a Q-wave postero-inferior, postero-basal MI. After MI he developed angina of the NYHA class III. At admission his state was satisfactory. Lung auscultation revealed respiration without rales, 18/min. Heart sounds were rhythmic, muffled, AP — 135/80 mm Hg, HR — 75 bpm.

ECG revealed sinus rhythm, HR — 64 bpm, scar changes of posterior and inferior segments of the left ventricle.

Echocardiography showed that heart cavities were not enlarged. LV EF — 63%. Valvular apparatus without marked pathology.

Selective coronary angiography revealed: right type of coronary circulation, subtotal stenosis of the LMCA in the middle and distal segments, arterial diameter 4,0 mm at a length of about 20 mm (fig.1); occlusion of the circumflex artery (CxA) in the middle/3, with satisfactory filling of the distal bed through collaterals arising from the right coronary artery system; < 75% stenosis in the middle/3 of the left anterior descending branch (LAD); subtotal stenosis in the middle/3 of the RCA (fig. 2) of more than 15 mm length.

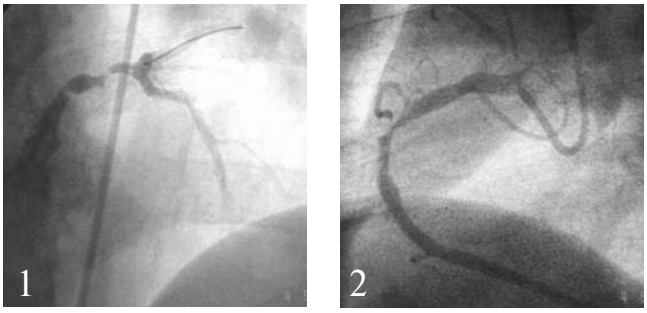

Figure 1. Subtotal stenosis of the LMCA. A coronary guide wire is inserted in the obtuse margin branch.
Figure 2. Subtotal stenosis of the RCA. A coronary guide wire is inserted.

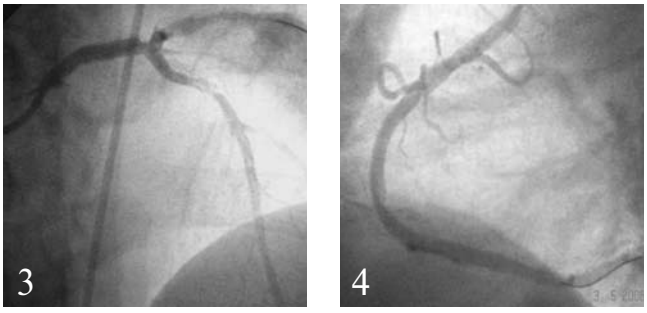

Figure 3. Control coronary angiogram shows complete restoration of the geometry of the LMCA.
Figure 4. Complete restoration of the blood flow in the RCA after stenting.

In the morning of April 28, 2006, at 8.20 a.m., the patient developed an episode of consciousness loss with convulsions. After 30 seconds the patient returned to consciousness, subsequently retrosternal anginal pains developed. No ECG changes were revealed. This state was interpreted as acute coronary syndrome. The patient was brought to the cathlab with the clinical picture of progressive angina pectoris. A balloon for intra-aortic counterpulsation (IABC) was inserted under fluoroscopic control distal to the left subclavian artery, IABC was started in 1:1 regimen. Stabilization of hemodynamics was achieved with AP 100/60 mm Hg, HR 71 bpm.

After preliminary insertion of a coronary guide wire (fig. 1 predilatation of subtotal stenosis of the LMCA

[1]Azarov A.A.,
azaraa@cardio.kem.ru
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was performed with Sprinter balloon (2,5 × 18,0 mm). After that coronary stent DRIVER (4,0 × 23,0 mm) was implanted under 12 atm. Pressure. The dilatation lasted 15 sec. Control coronary angiogram showed total restoration of the LCMA geometry (fig. 3). During the second stage of the procedure direct stenting of the subtotal stenosis of the middle segment of the RCA was performed (DRIVER stent, 4,0 × 18,0 mm, implantation pressure 11 atm., dilatation duration 20 sec.), with complete restoration of the arterial lumen (fig. 4). The hemodynamics remained stable through the whole procedure. The patient was transferred to the coronary care unit. IABC was removed at Day 3, after AP stabilization at a level of 120/80 mm Hg.

It was decided to perform selective restoration of the LDA and the CxB lumens in three months.

The patient was discharged on May 16, 2006. With the applied drug therapy anginal attacks did not recur, no rhythm disturbances were noted, hemodynamics remained stable. The patient was able to tolerate the loads of the 3rd regimen (walking up to 500 meters per day, going up the stairs to the second floor) with adequate reactions.

Thus, despite unfavorable prognosis of urgent endovascular interventions in patients with the lesion of unprotected LMCA and subototal stenosis of the RCA, PTCA with stenting is effective and justified, preferably with IABK support.

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State of Blood Inhibitory Systems, Coagulation, Fibrinolysis, and Kinin Formation in Patients with Unstable Angina Undergoing Coronary Angioplasty with Stenting

V.A. Ivanov[1], V.V. Krashutsky, A.N. Pyriev, A.B.Shames, A.A. Vishnevsky
3rd Central Military Hospital, Krasnogorsk, Russia

LIST OF ABBREVIATIONS

ACS — acute coronary syndrome
ADP — adenosine-5'-diphosphate
AMI — acute myocardial infarction
APTT — activated partial thromboplastin time
ART — activated recalcification time
ATIII — antithrombin III
CA —coronary angioplasty
CAD — coronary artery disease
CKF — coefficient of kallikrein formation
CS — coronary stenting
CTR — coefficient of thrombosis risk
DIC — disseminated intravascular coagulation
DIFHT — differentiation index for fibrinolytic and heparin therapy
ECG — electrocardiogram
EchoCG — echocardiography
ET — ethanol test
FA — fibrinolytic activity
FG — fibrinogen
fXIII — fibrinase, fibrin stabilizing factor
HFS — Hageman factor system
HT — heparin time
IHTA — index of heparin therapy adequacy
KKS — kallikrein-kinin system
LAD — left anterior descending artery
MI — myocardial infarction
PCI — percutaneous coronary intervention
PEA — progressive exertional angina
PK — plasma kallikrein
PPK — plasma prekallikrein
PR — prothrombin ratio
PST — protamine sulfate test
PTCA — percutaneous transluminal (balloon) coronary angioplasty
RCA — right coronary artery
SFMC — soluble fibrin monomer complexes
TT — thrombin time
UA — unstable angina

Introduction

Vigorous implementation of interventional methods in diagnostics and therapy of CAD is a revolutionary achievement of modern cardiology (20, 38, 46–49). That especially concerns the percutaneous transluminal coronary balloon angioplasty (PTCA) (23–25, 33, 41) and coronary stenting (29, 30, 42, 43, 51), which allowed for significant reduction of risk of acute coronary complications and restenosis incidence (35, 36, 44). In the USA and Europe, more than 50% of primary PTCA is finished by stent implantation (2). Currently, in the USA only, there are approximately 500,000 percutaneous interventions in coronary arteries performed annually. Worldwide there are more than 1.2 mln interventions per year (1, 20, 21, 16), whereas in the Russian Federation the number of percutaneous coronary interventions, while having a certain trend to increase (3895 procedures in 2001, 6996 in 2003) is clearly insufficient (5).

In early phases of coronary stenting technique development acute and subacute stent thrombosis was reported in 15–24% of cases (27, 31, 52). Acute thrombosis of stented artery is associated with very high risk of "major cardiac complications". Particularly, death is reported in 7–19%, AMI in 57–85% and urgent CABG is performed in 30–44% of cases (3, 4, 34). Unstable angina (6, 11, 12, 19) and recent AMI (up to 2-3 weeks ago) (39, 40) are commonly recognized as risk factors for stent thrombosis. Certain baseline variables of patient hemostasis were established to have an important role in stent thrombosis (37, 50), however, in real settings, by no means can measurements of such variables as GP IIB/IIIA platelet receptors activity or prothrombin fragments F 1 + 2 be performed in every patient. Some suggest (17, 32) that, after simplification and reduction of costs of such tests, a detailed analysis of hemostasis status will become a mandatory element of coronary angioplasty protocol. Development of potent anti-platelet drugs, which reliably decrease the risks of thrombotic complications of coronary angioplasty (CA), has facilitated the improvement in results of CA (17, 18, 22, 26, 28, 45). However, thrombotic complications of coronary angioplasty remain the issue of the day in interventional cardiology. Hemostasis-associated risk factors of CS-related MI, the status of blood inhibitory systems, coagulation, fibrinolysis and kinin formation in CA-related MI, and the status of these systems of limited proteolysis in uncomplicated CAD after PCI have not been adequately studied (11, 14, 15).

Purpose of the study

Optimization of hemostasis-based prediction and diagnostics of acute myocardial infarction related to coronary angioplasty with stenting in patients with unstable angina.

[1]V.A. Ivanov
Russia, 143420, Krasnogorsk,
Vishnevsky Central Military Hospital N3
e-mail: ivanov-angio@yandex.ru
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Objectives of the study:

1. To study the dynamics of variables of blood inhibitory systems, coagulation, fibrinolysis, and kinin formation in patients with unstable angina, who underwent coronary angioplasty with stenting without thrombotic complications

2. To study the dynamics of variables of blood inhibitory systems, coagulation, fibrinolysis, and kinin formation in patients with unstable angina, who underwent coronary angioplasty with stenting complicated by acute MI

3. To establish the hemostasis-based risk factors and AMI criteria in the setting of coronary angioplasty with stenting.

Material and methods

From 1991 to 2004 inclusive, in A. A. Vishnevsky Central Military Clinical Hospital #3 there were 2042 PTCA performed in 1236 patients with CAD (1.65 balloon dilatation s per patient) and 1037 stenting procedures in 758 patients (1.35 stents per patient), with general mortality of 0.73% for this whole period. The most significant CA complications included acute MI (21 — 1.7% of postoperative patients), stent thrombosis confirmed by angiography (19 — 1.8% of stenting procedures) and bleeding events (9 — 0.7% of postoperative patients). The incidence of unsatisfactory results of CA was higher in the initial phase of PTCA implementation. For instance, during 1991–1996, PTCA was performed in 252 patients with CAD, with mortality rate of 1.6%, while in 1997 to 2004 there were 984 patients who underwent the procedure, including 92 patients (9.3%) with acute coronary syndrome, with mortality rate of 0.3%. For the last 5 years mortality rate related to CA is 0.2–0.3%.

The study enrolled patients with unstable angina (n = 55), who underwent PTCA with stenting after clinical investigation, which included laboratory assessment of interrelated systems of blood coagulation, fibrinolysis and kinin formation, lipid metabolism, electrocardiography (ECG), echocardiography (EchoCG), incremental cycle ergometry (CEM), myocardial perfusion scintigraphy with Thallium-201 or Tc-99m-Technetril, coronary angiography (CAG) at the Radiosurgical Diagnostics and Treatment Center of A. A. Vishnevsky Central Military Clinical Hospital.

Before the procedure, routine clinical tests of blood, urine, stool, and biochemical panel (AST, ALT, bilirubin, creatinine, urea nitrogen, sugar, cholesterol, total protein, potassium and sodium ions) were performed, using autoanalyzers SMA 12/60 (Technicon, USA) or “SPECTRUM” (ABBOT, USA), as well as blood osmolarity, phenotyping of dyslipoproteinemia, acid-base balance (ABB), blood gases, blood coagulation tests.

Instrumental diagnostic methods included assessment of central hemodynamics by tetrapolar rheography (Kubichek, 1973) using “Bioset 8000–01” device (Germany), stress tests with bycycle ergometer (Ergofit-777, USA) or treadmill (Technogim Run-X-T, Italy). When these methods were unavailable due to any causes, 24-hour (Holter) ECG monitoring (ROSIN, USA), two-dimensional echocardiography of cardiac chambers and major vessels using ultrasound equipment “ASPEN (by Acuson, USA), chest X-ray to identify coronary calcification, and to assess the cardiac chambers and status of pulmonary circulation (especially in the group of patients with history of MI).

Coronary angiography was performed via standard Judkins femoral approach for purposes of diagnosis clarification, identification of the pattern and grade of atherosclerotic impairment of coronary circulation, and assessment of state of left ventricle myocardium. URCI, CORDIS, ACS (USA) and COOK (Denmark) catheters were used (Rabkin IKh et al. 1987). Angiography equipment employed was: “Advantx DLX”, GE (USA) or “Angioscop D33” with “Digitron 3V” add-on device, Siemens (Germany). State of cardiomyocytes and myocardial microcirculation were assessed by myocardial perfusion scintigraphy with Thallium-201 or Tc-99m-Technetril, using gamma camera Starcam 4000i, General Electric (USA) or by ECG-synchronized myocardial perfusion tomoscintigraphy with Tc-99m-Technetril using gamma camera E.cam, Siemens (USA). 3D and 4D-MSPECT software was used. Evaluated variables included central hemodynamics parameters and ejection fraction of right and left heart ventricles.

Total cholesterol (TC), triglyceride (TG), low density lipoprotein cholesterol (LDL-C), very low density lipoprotein cholesterol (VLDL-C), and high density lipoprotein cholesterol (HDL-C) concentrations were measured using biochemistry analyzer “Abbot” with biochemistry set “Spectrum”, USA.

Before procedure and on Days 1, 3, 7, and 14 after CS, variables of Hageman factor system (HFS) were evaluated in accordance with “Instruction on application of standardized clinical laboratory tests” (7), USSR Ministry of Health Guidelines “Assessment of humoral mechanisms of bio- and pharmacological drugs action in regulation of blood inhibitory systems, coagulation, fibrinolysis and kinin formation” (13) and GIUV MO RF (Russian Federation Ministry of Defense State Institute of Postgraduate Medical Training) Guidance Manual (9).

Kallikrein, coagulation and fibrinolysis variables were evaluated using venous blood stabilized with trisubstituted sodium citrate 3.8% solution (9:1). In order to prevent contact activation, blood was drawn from the median cubital vein using siliconized needle with wide lumen by gravity flow without tourniquet application into siliconized test tubes. Blood and laboratory ware handling, siliconizing, reagent preparation for coagulation and fibrinolysis measurements were performed in accordance with the regulatory documents mentioned above. Platelet rich and platelet poor plasma were obtained by blood centrifugation at 1000 rpm (300 g) (platelet rich) for 5 minutes and at 3000 rpm (1000 g) (platelet poor) for 10 minutes at room temperature.

Platelet rich plasma was used for assessments of: activated plasma recalcification time (APRT) (kaolin time) using technique introduced by Bergerhof, Roka (1954) with modifications by Interclinical Coagulation Laboratory of I. M. Sechenov Medical Academy, Moscow.

Platelet poor plasma was used for assessments of: plasma kallikrein (PK) and prekallikrein (PPK) (using technique by Paskhina and Krinskaya 1977), activated partial thromboplastin time (APTT) (Proctor, Rapaport, 1961 as modified by Smolianitskii et al. 1982); prothrombin ratio (PR) (Quick 1943); fibrinogen (FG) (Rutberg, 1961); fibrin stabilizing factor (fXIII, fibrinase) (Sigg and Duckert as modified by Baluda et al. 1965); thrombin time (TT) (Sirmai 1957); heparin time (HT) (Sirmai 1957); soluble fibrin monomer complexes (SFMC), measured by means of: a) ethanol test (ET) (Godal, Abildgaard 1966); b) protamine sulfate test (PST) (Lipinski, Worowski (1968); plasma fibrinolytic activity (FA) by euglobulin lysis method (Kowalski et al. 1959); antithrombin III (ATIII) activity (Krashutskii) (9, 10, 13).

Note that both activity of kallikrein/its precursor prekallikrein, and activity of the main inhibitor of activated coagulation factors ATIII were evaluated. Intrinsic pathway of coagulation was evaluated by APTT and APRT, extrinsic pathway was assessed by prothrombin ratio, final phase of coagulation — by fibrinogen concentration, fXIII activity and SFMC presence (via ethanol and protamine sulfate tests). State of the fibrinolytic system was assessed by euglobulin lysis time (as an indirect measure of palsminogen activators).

To evaluate the Hageman factor system characteristics and monitor the effectiveness of antithrombotic measures, a number of indices and coefficients were calculated (9, 10, 13):

— Coefficient of thrombosis risk (CTR): (fXIII/ATIII)*1.778 (normal value 1.0±0.07).

– Index of heparin therapy adequacy (IHTA) (APTT/PR)*2 (normal value 1.0 ± 0.05; adequate range 1.5–2.5).

— Differentiation index for fibrinolytic and heparin therapy (DIFHT): (ATIII/APTT)*0.45 (normal value 1.0±0.06).

– Coefficient of kallikrein formation (CKF): (PK/PPK)*33 (normal value 1.0 ± 0.2).

The units of measurement of parameters used for calculation were: PK, PPK values — in μmol/min*L; ATIII, PR — %; fXIII — seconds

The results were compared with data obtained in evaluation of 57 healthy subjects. DIC syndrome forms and stages were determined according to Krashutsky method (8).

Depending on the outcomes of coronary angioplasty, patients were assigned into 2 groups.

The first group (n = 40) included patients with UA who did not experience MI after coronary angioplasty. The second group included 15 UA patients with MI after CA.

The mean age in the first group was 53.6 ± 1.3 years, there were 39 males and one female. Nine patients (22.5%) had essential hypertension, 1 patient (2.5%) had diabetes mellitus, and 11 (27.5%) had history of MI. CAD duration was 3.8 ± 0.8 years, duration of the period of instability before admission to the hospital was 9.7 ± 1.3 days. Eleven procedures (27.5%) were performed for new-onset angina (the time from onset was 23.8 ± 5.3 days), 18 (45.0%) for progressive exertional angina, 11 (27.5%) for post-MI angina, on average in 41.9 ± 6.1 days after myocardial infarction. Hemodynamically significant one-vessel coronary artery disease was seen in 21 cases (52.5%), two-vessel lesions in 14 cases (35.0%) and three-vessel lesion was observed in 5 cases (12.5%). PTCA was performed after UA patient was stabilized, on average in 18.07 ± 1.5 days after admission. Thirty three patients (82.5%) underwent one artery angioplasty, 7 patients (17.5%) had two arteries repaired. In 25 cases (62.5%) there was no recurrence of angina after PTCA procedure. In 7 cases (17.5%) recurrence of angina occurred in postprocedural phase on average on day 2.4 ± 0.7 after angioplasty ("early recurrence" of angina). In 8 cases (20.0%) angina recurred after patient discharge, on average 3.4 ± 1.1 months post procedure (6 patients underwent re-PTCA, 1 patient had direct myocardial revascularization procedure). In 2 patients (5.0%), post PTCA ECG showed transient myocardial ischemia. In 1 (2.5%) case (2.5%) the procedure was complicated by ventricular fibrillation. In 1 (2.5%) case there was paroxysm of atrial fibrillation. In 2 cases (5.0%) procedure was complicated by acute atrioventricular and intraventricular conduction disturbances. Two patients (5.0%) developed profuse bleeding at the femoral puncture site, which required hemotransfusion, and 1 patient needed urgent surgery for suturing the femoral artery defect.

Fifteen patients of the second group had MI (9 cases of transmural MI and 6 subendocardial MI) in the basin of the coronary artery which was subjected to PTCA (LAD — 9 cases, RCA — 6 cases) in the first 24-hour period post procedure, two patients (13.3%) died. The predisposing factors for AMI after PTCA were: 1) subintimal mechanical damage to the atheromatous plaque in 6 patients; 2) stent thrombosis in 3 patients; 3) technical difficulties while PTCA (difficult anatomy of coronary vessels) in 3 patients.

Results and discussion

HFS parameters dynamics in patients with UA without thrombotic complications of transluminal coronary angioplasty with stenting is shown in Table 1, which demonstrates 2.4-fold increase in plasma kallikrein activity, 3.9 fold increase in coefficient of kallikrein formation (t = 1.7989), 1.7 fold increase in index of heparin therapy adequacy and increase in antithrombin III consumption by 9% from baseline on Day1.

On Day 3, there were significant signs of structural hypercoagulation: increase in fXIII activity, thrombin and SFMC formation according to ET with AT III consumption (by 25.5% from the baseline

Table 1. Kallikrein, coagulation and fibrinolysis parameters dynamics in unstable angina patients following uncomplicated transluminal coronary angioplasty.

No.	Parameters of the aggregative state of blood	Norm	Baseline pre-PTCA data	Day 1 post PTCA	Day 3 post PTCA	Day 7 post PTCA	Day 14 post PTCA
1	(PR) (%)	92 ± 3,9	99.72 ± 0.94	97.1 ± 3.85	94.0 ± 3.36	98.3 ± 1.05	96.5 ± 2.87
2	ART (sec)	63,15 ± 1,67	60.08 ± 0.34	77.7 ± 9.36	62.33 ± 2.04	54.8 ± 2.49 (P1 < 0,05)	61.5 ± 6.02
3	APTT (sec)	46,55 ± 1,22	45.55 ± 0.27	76.0 ± 6.39 (P1 < 0,05) (P2 < 0,05)	40.42 ± 2.12 (P1 < 0,05) (P2 < 0,05)	41.5 ± 1.8 (P1 < 0,05) (P2 < 0,05)	43.5 ± 2.72
4	FG (g/L)	2,2 ± 0,36	3.55 ± 0.14 (P1 < 0,05)	3.3 ± 0.27 (P1 < 0,05)	3.69 ± 0.25 (P1 < 0,05)	4.17 ± 0.17 (P1 < 0,05) (P2 < 0,05)	3.37 ± 0.52 (P1 < 0,05)
5	fXIII (sec)	58,27 ± 3,86	60.0 ± 6.24	55.16 ± 1.35	74.0 ± 3.45 (P1 < 0,05)	62.5 ± 2.5	58.3 ± 3.4
6	ET (+)	0,2 ± 0,07	0.45 ± 0.16	0.29 ± 0.19	1.14 ± 0.16 (P1 < 0,05) (P2 < 0,05)	0.29 ± 0.11	0.35 ± 0.18
7	PSTW (optical density units)	0,22 ± 0,013	0.43 ± 0.023 (P1 < 0,05)	0.495 ± 0.068 (P1 < 0,05)	0.48 ± 0.078 (P1 < 0,05)	0.49 ± 0.084 (P1 < 0,05)	0.4 ± 0.042 (P1 < 0,05)
8	AT III (%)	103,6 ± 4,1	96.87 ± 4.5	87.8 ± 7.25	71.4 ± 7.35 (P1 < 0,05)	75.2 ± 13.7	92.0 ± 9.6
9	CTR (a.u.)	1,0 ± 0,07	1.1 ± 0.12	1.34 ± 0.14	2.41 ± 0.48 (P1 < 0,05) (P2 < 0,05)	1.55 ± 0.27	1.35 ± 0.3
10	(FA) (min)	245 ± 11,7	245.45 ± 12.0	261.7 ± 27.19	296.3 ± 23.0	275.8 ± 10.28 (P1 < 0,05)	262.6 ± 18.56
11	(PK) (μmol/L)	11,39 ± 2,33	31.6 ± 3.15 (P1 < 0,05)	75.0 ± 9.4 (P1 < 0,05) (P2 < 0,05)	76.75 ± 10.6 (P1 < 0,05) (P2 < 0,05)	70.0 ± 7.2 (P1 < 0,05) (P2 < 0,05)	43.67 ± 6.75 (P1 < 0,05)
12	(PPK) (μmol/L)	369,9 ± 39,37	393.62 ± 20.62	236.1 ± 91.5	356.9 ± 54.49	406.6 ± 79.8	514.4 ± 73.5
13	CKF (a.u.)	1,0 ± 0,2	2.65 ± 0.27 (P1 < 0,05)	10.4 ± 4.3 (P1 < 0,05)	7.08 ± 1.2 (P1 < 0,05) (P2 < 0,05)	5.57 ± 1.19 (P1 < 0,05) (P2 < 0,05)	2.8 ± 0.6 (P1 < 0,05)
14	IHTA (a.u.)	1 ± 0,05	0.91 ± 0.03	1.56 ± 0.16 (P1 < 0,05) (P2 < 0,05)	0.96 ± 0.05	0.94 ± 0.04	0.9 ± 0.06
15	DIFHT (a.u.)	1 ± 0,06	0.96 ± 0.048	0.43 ± 0.043 (P1 < 0,05) (P2 < 0,05)	0.6 ± 0.09 (P1 < 0,05) (P2 < 0,05)	0.78 ± 0.16	0.95 ± 0.1

(P1<0,05) – Statistically significant difference from the norm.
(P2<0,05)- Statistically significant difference from baseline.

level), 2.4 fold increase in plasma kallikrein, 2.6 increase in coefficient of kallikrein formation and 2.2 fold increase in coefficient of thrombosis risk compared to baseline values, coupled with depression of fibrinolysis (increased euglobulin lysis time). On Day 7 post PTCA, the following changes from baseline are seen: APTT decrease, fibrinogen increase and AT III decrease coupled with increased kallikrein activity and coefficient of kallikrein formation. Two weeks after balloon dilatation of coronary arteries with stenting, HFS parameters returned to baseline.

HFS parameters dynamics in UA patients who underwent percutaneous transluminal coronary angioplasty complicated by acute MI is shown in Table 2. This table demonstrates the changes from pre-PTCA baseline on Day 1 of CA-related MI as follows: 1.9 fold increase in APTT; 4.4 fold decrease in DIFHT; 1.37 decrease in FG content; 1.2 fold decrease in fXIII activity; 2.3 fold decrease in AT III; 2.2 fold increase in CTR; FA decrease demonstrated by 1.5 fold increase in euglobulin clot lysis time; 2.4 fold increase in plasma kallikrein activity; 2.8 fold increase in coefficient of kallikrein formation.

Table 2. Kallikrein, coagulation and fibrinolysis parameters dynamics in unstable angina patients following coronary angioplasty complicated by MI.

No.	Parameters of the aggregative state of blood	Norm	Baseline pre-PTCA data	Day 1 post PTCA	Day 3 post PTCA	Day 7 post PTCA	Day 14 post PTCA
1	(PR) (%)	92 ± 3.9	99.9 ± 2.32	95.1 ± 2.15	96.0 ± 2.14	97.4 ± 2.3	98.3 ± 3.7
2	ART (sec)	63.15 ± 1.67	56.1 ± 1.17 (P1 < 0.05)	53.3 ± 3.6 (P1 < 0.05)	52.8 ± 2.1 (P1 < 0.05)	55.3 ± 2.6 (P1 < 0.05)	54.7 ± 3.2 (P1 < 0.05)
3	APTT (sec)	2.2 ± 0.36	4.25 ± 0.23 (P1 < 0.05)	3.1 ± 0.31 (P1 < 0.05) (P2 < 0.05)	4.56 ± 0.32 (P1 < 0.05)	5.29 ± 0.35 (P1 < 0.05) (P2 < 0.05)	4.3 ± 0.29 (P1 < 0.05)
4	FG (g/L)	2.2 ± 0.36	3.55 ± 0.14 (P1 < 0.05)	3.3 ± 0.27 (P1 < 0.05)	3.69 ± 0.25 (P1 < 0.05)	4.17 ± 0.17 (P1 < 0.05) (P2 < 0.05)	3.37 ± 0.52 (P1 < 0.05)
5	fXIII (sec)	58.27 ± 3.86	78.5 ± 4.35 (P1 < 0.05)	65.2 ± 3.25 (P1 < 0.05)	86.4 ± 3.8 (P1 < 0.05)	93.5 ± 4.2 (P1 < 0.05) (P2 < 0.05)	69.3 ± 3.8 (P1 < 0.05)
6	ET (+)	0.2 ± 0.07	0.42 ± 0.19	0.9 ± 0.11 (P1 < 0.05) (P2 < 0.05)	2.0 ± 0.21 (P1 < 0.05) (P2 < 0.05)	2.6 ± 0.25 (P1 < 0.05) (P2 < 0.05)	0.53 ± 0.24
7	PST (optical density units)	0.22 ± 0.013	0.53 ± 0.04 (P1 < 0.05)	0.6 ± 0.06 (P1 < 0.05)	0.74 ± 0.08 (P1 < 0.05) (P2 < 0.05)	0.69 ± 0.09 (P1 < 0.05)	0.5 ± 0.05 (P1 < 0.05)
8	AT III (%)	103.6 ± 4.1	98.5 ± 3.5	43.0 ± 5.0 (P1 < 0.05) (P2 < 0.05)	44.3 ± 4.0 (P1 < 0.05) (P2 < 0.05)	76.5 ± 14.5	95.6 ± 8.2
9	CTR (a.u.)	1.0 ± 0.07	1.4 ± 0.1 (P1 < 0.05)	3.1 ± 0.39 (P1 < 0.05) (P2 < 0.05)	3.5 ± 0.31 (P1 < 0.05) (P2 < 0.05)	2.17 ± 0.43 (P1 < 0.05)	1.3 ± 0.09 (P1 < 0.05)
10	(FA) (min)	245 ± 11.7	180.3 ± 19.6 (P1 < 0.05)	274.4 ± 21.13 (P1 < 0.05)	310.0 ± 20.0 (P1 < 0.05) (P2 < 0.05)	300.0 ± 60.4	261.0 ± 24.5 (P2 < 0.05)
11	(PK) (μmol/L)	11.39 ± 2.33	38.9 ± 2.5 (P1 < 0.05)	93.1 ± 11.2 (P1 < 0.05) (P2 < 0.05)	77.8 ± 9.8 (P1 < 0.05) (P2 < 0.05)	75.0 ± 8.4 (P1 < 0.05) (P2 < 0.05)	44.6 ± 5.7 (P1 < 0.05)
12	(PPK) (μmol/L)	369.9 ± 39.37	424.6 ± 32.33	367.8 ± 61.5	256.9 ± 53.6 (P2 < 0.05)	324.3 ± 69.8	448.6 ± 82.9
13	CKF (a.u.)	1.0 ± 0.2	3.0 ± 0.36 (P1 < 0.05)	8.3 ± 1.39 (P1 < 0.05) (P2 < 0.05)	8.3 ± 1.39 (P1 < 0.05) (P2 < 0.05)	7.64 ± 1.7 (P1 < 0.05) (P2 < 0.05)	3.3 ± 0.65 (P1 < 0.05)
14	IHTA (a.u.)	1 ± 0,05	1.09 ± 0.04	2.15 ± 0.15 (P1 < 0.05) (P2 < 0.05)	1.36 ± 0.05 (P1 < 0.05) (P2 < 0.05)	1.24 ± 0.04 (P1 < 0.05) (P2 < 0.05)	0.88 ± 0.05 (P2 < 0.05)
15	DIFHT (a.u.)	1 ± 0.06	0.83 ± 0.04 (P1 < 0.05)	0.19 ± 0.03 (P1 < 0.05) (P2 < 0.05)	0.3 ± 0.012 (P1 < 0.05) (P2 < 0.05)	0.57 ± 0.11 (P1 < 0.05) (P2 < 0.05)	1.03 ± 0.08

(P1<0.05) Statistically significant difference from the norm.
(P2<0.05) Statistically significant difference from baseline.

Therefore, in UA patients who developed acute MI on Day 1 post-PTCA there is a partial coagulopathy in terms of consumption of inhibitor (ATIII) and blood coagulation factors (FG, fXIII). In general, the variables of homeostasis system are consistent with subacute DIC syndrome. On Day 3 post CA the changes in the variables of homeostasis system were the same as on Day 1, but there were higher ET and CTR values and less APTT, IHTA, and FA (i.e. increase in euglobulin lysis time) values. On Day 7 of MI following PTCA thrombin formation was not decreasing, there was an increase in ATIII activity, and kallikrein activation remains high. By Day 14, the variables of homeostasis system approached their baseline values.

Tables 1–3 allow to compare the baseline state of hemostasis system in uncomplicated UA patients who developed post-CA MI. The tables show that UA patients who developed MI after PTCA had significantly more pronounced activation of coagulation (higher levels of FG, fXIII, thrombin formation, SFMC as measured by PST, and CTR), fibrinolysis (lower euglobulin lysis time) and kinin formation (higher kal-

Table 3. Baseline values for kallikrein, coagulation and fibrinolysis parameters in unstable angina patients following coronary angioplasty for uncomplicated postprocedural phase vs. postprocedural acute MI.

No.	Parameters of the aggregative state of blood	Norm	Uncomplicated PTCA in UA patients (P1)	Post-PTCA MI in UA patients (P2)	Differences significance N-P1	Differences significance N-P2	Differences significance P1-P2
1	(PR) (%)	92 ± 3.9	99.72 ± 0.94	99.9 ± 2.32	> 0,05	> 0,05	> 0,05
2	ART (sec)	63.15 ± 1.67	60.09 ± 0.34	56.1 ± 1.17	> 0,05	> 0,05	> 0,05
3	APTT (sec)	46,55 ± 1,22	45.55 ± 0.27	54.2 ± 1.68	> 0,05	> 0,05	> 0,05
4	FG (g/L)	2.2 ± 0.36	3.55 ± 0.14	4.25 ± 0.23	> 0,05	> 0,05	> 0,05
5	fXIII (sec)	58.27 ± 3.86	60.0 ± 6.24	78.5 ± 4.35	> 0,05	> 0,05	> 0,05
6	ET (+)	0.2 ± 0.07	0.45 ± 0.16	0.42 ± 0.19	> 0,05	> 0,05	> 0,05
7	PST (optical density units)	0.22 ± 0.013	0.43 ± 0.023	0.53 ± 0.04	> 0,05	> 0,05	> 0,05
8	AT III (%)	103.6 ± 4.1	96.87 ± 4.75	98.5 ± 3.5	> 0,05	> 0,05	> 0,05
9	CTR (a.u.)	1.0 ± 0.07	1.1 ± 0.12	1.4 ± 0.1	> 0,05	> 0,05	> 0,05
10	(FA) (min)	245 ± 11.7	245.45 ± 13.03	180.3 ± 19.6	> 0,05	> 0,05	> 0,05
11	(PK) (μmol/L)	11.39 ± 2.33	31.6 ± 2.15	38.9 ± 2.5	> 0,05	> 0,05	> 0,05
12	(PPK) (μmol/L)	369.9 ± 39.37	393.62 ± 20.62	424.6 ± 32.33	> 0,05	> 0,05	> 0,05
13	CKF (a.u.)	1.0 ± 0.2	2.65 ± 0.27	3.0 ± 0.36	> 0,05	> 0,05	> 0,05
14	IHTA (a.u.)	1 ± 0,05	0.91 ± 0.03	1.09 ± 0.04	> 0,05	> 0,05	> 0,05
15	DIFHT (a.u.)	1 ± 0.06	0.96 ± 0.048	0.83 ± 0.04	> 0,05	> 0,05	> 0,05

likrein activity) when compared with the patients who had no coronary angioplasty complications.

This shows that at preprocedural baseline, UA patients without thrombotic complications of PTCA had moderate activation of coagulation (increases in FG level and thrombin formation shown via SFMC) and kinin formation (increase in PK and CKF) consistent with chronic DIC-syndrome, while patients who had MI on Day 1 post coronary angioplasty, showed activation of coagulation, fibrinolysis and kinin formation consistent with borderline state between chronic and subacute DIC syndrome just before the procedure.

Comparative characteristics of kallikrein, coagulation and fibrinolysis parameters in unstable angina patients following PTCA uncomplicated and complicated by MI are shown in Tables 4, 5 and Figures 1–4. These indicate that in UA patients with post-PTCA myocardial infarction there are significantly more pronounced increase in CTR (1.9 fold, vs. 1.2 fold in uncomplicated PTCA group, and more dramatic (2.3 fold) AT III consumption compared to baseline level (in uncomplicated CA group ATIII decreased 1.1 fold). In postprocedural MI there is also a more dramatic increase in thrombin formation markers, particularly ET, which is supported by increased consumption of FG and fXIII in patients with post-PTCA MI compared to uncomplicated CA on Day 1 post PTCA as compared to UA patients in uncomplicated CA group.

Table 4. Comparison of the dynamics of kallikrein, coagulation and fibrinolysis parameters in unstable angina patients following coronary angioplasty, uncomplicated vs. complicated by MI (Days 1–3 post PTCA).

No.	Parameters of the aggregative state of blood	Day 1 post PTCA		Day 3 post PTCA	
		Uncomplicated PTCA	post-PTCA MI	Uncomplicated PTCA	post-PTCA MI
1	(PR) (%)	97.1 ± 3.85	95.1 ± 2.15	94.0 ± 3.36	96.0 ± 2.14
2	ART (sec)	77.7 ± 9.36	53.3 ± 3.6 (P < 0,05)	62.33 ± 2.04	52.8 ± 2.1 (P < 0,05)
3	APTT (sec)	76.0 ± 6.39	102.8 ± 4.1 (P < 0,05)	40.42 ± 2.12	65.4 ± 2.15 (P < 0,05)
4	FG (g/L)	3.3 ± 0.27	3.1 ± 0.31	3.69 ± 0.25	4.56 ± 0.32 (P < 0,05)
5	fXIII (sec)	55.16 ± 1.35	65.2 ± 3.25 (P < 0,05)	74.0 ± 3.45	86.4 ± 3.8 (P < 0,05)
6	ET (+)	0.29 ± 0.19	0.9 ± 0.11 (P < 0,05)	1.14 ± 0.16	2.0 ± 0.21 (P < 0,05)
7	PST (optical density units)	0.495 ± 0.068	0.6 ± 0.06	0.48 ± 0.078	0.74 ± 0.08 (P < 0,05)
8	AT III (%)	87.8 ± 7.25	43.0 ± 5.0 (P < 0,05)	71.4 ± 7.35	44.3 ± 4.0 (P < 0,05)
9	CTR (a.u.)	1.34 ± 0.14	3.1 ± 0.39 (P < 0,05)	2.41 ± 0.48	3.5 ± 0.31 (P < 0,05)
10	(FA) (min)	261.7 ± 27.19	274.4 ± 21.13	296.3 ± 23.0	310.0 ± 20.0
11	(PK) (μmol/L)	75.0 ± 9.4	93.1 ± 11.2	76.75 ± 10.6	77.8 ± 9.8
12	(PPK) (μmol/L)	236.1 ± 91.5	367.8 ± 61.5	356.9 ± 54.49	256.9 ± 53.6
13	CKF (a.u.)	10.4 ± 4.3	8.3 ± 1.39	7.08 ± 1.2	10.0 ± 2.18
14	IHTA (a.u.)	1.56 ± 0.16	2.15 ± 0.15 (P < 0,05)	0.96 ± 0.05	1.36 ± 0.05 (P < 0,05)
15	DIFHT (a.u.)	0.43 ± 0.043	0.19 ± 0.03 (P < 0,05)	0.6 ± 0.09	0.3 ± 0.012 (P < 0,05)

(P<0,05) - Significant difference between patient groups

Table 5. Comparison of the dynamics of kallikrein, coagulation and fibrinolysis parameters in unstable angina patients following coronary angioplasty, uncomplicated vs. complicated by MI (Days 7–14 post PTCA).

No.	Parameters of the aggregative state of blood	Day 7 post PTCA		Day 14 post PTCA	
		Uncomplicated PTCA	post-PTCA MI	Uncomplicated PTCA	post-PTCA MI
1	(PR) (%)	98.3 ± 1.05	97.4 ± 2.3	96.5 ± 2.87	96.0 ± 2.14
2	ART (sec)	54.8 ± 2.49	55.3 ± 2.6	61.5 ± 6.02	54.7 ± 3.2
3	APTT (sec)	41.5 ± 1.8	60.4 ± 1.3 (P < 0,05)	43.5 ± 2.72	58.8 ± 1.7 (P < 0,05)
4	FG (g/L)	4.17 ± 0.17	5.29 ± 0.35 (P < 0,05)	3.37 ± 0.52	4.3 ± 0.29
5	fXIII (sec)	62.5 ± 2.5	93.5 ± 4.2 (P < 0,05)	58.3 ± 3.4	69.3 ± 3.8 (P < 0,05)
6	ET (+)	0.29 ± 0.11	2.6 ± 0.25 (P < 0,05)	0.35 ± 0.18	0.53 ± 0.24
7	PST (optical density units)	0.49 ± 0.084	0.69 ± 0.09	0.4 ± 0.042	0.5 ± 0.05
8	AT III (%)	75.2 ± 13.7	76.5 ± 14.5	92.0 ± 9.6	95.6 ± 8.2
9	CTR (a.u.)	1.55 ± 0.27	2.17 ± 0.43	1.35 ± 0.3	1.3 ± 0.09
10	(FA) (min)	275.8 ± 10.28	300.0 ± 60.4	262.6 ± 18.56	261.0 ± 24.5
11	(PK) (μmol/L)	70.0 ± 7.2	75.0 ± 8.4	43.67 ± 6.75	44.6 ± 5.7
12	(PPK) (μmol/L)	406.6 ± 79.8	324.3 ± 69.8	514.4 ± 73.5	448.6 ± 82.9
13	CKF (a.u.)	5.57 ± 1.19	7.64 ± 1.7	2.8 ± 0.6	3.3 ± 0.65
14	IHTA (a.u.)	0.94 ± 0.04	1.24 ± 0.04 (P < 0,05)	0.9 ± 0.06	0.88 ± 0.05
15	DIFHT (a.u.)	0.78 ± 0.16	0.57 ± 0.11	0.95 ± 0.1	1.03 ± 0.08

(P<0,05) - Significant difference between patient groups

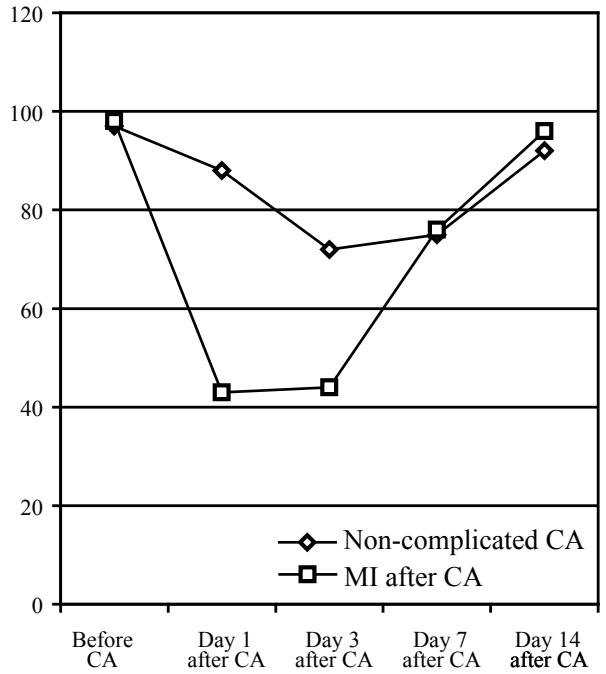

Figure 1. ATIII dynamics (%) in UA patients after coronary angioplasty, uncomplicated vs. complicated by MI.

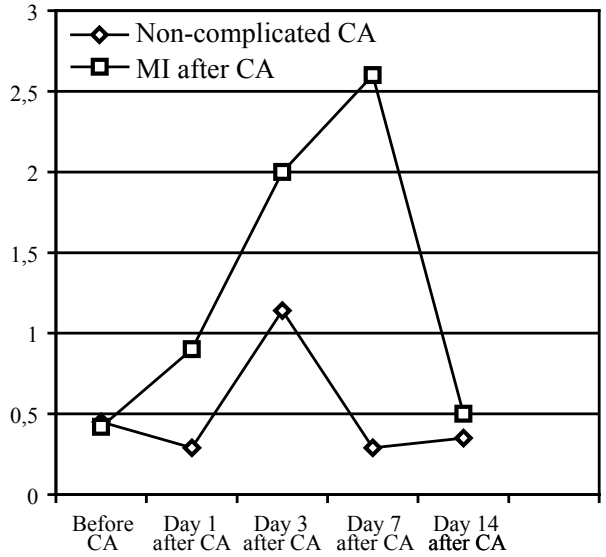

Figure 3. Ethanol test (ET, arbitrary units) dynamics in UA patients after coronary angioplasty, uncomplicated vs. complicated by MI.

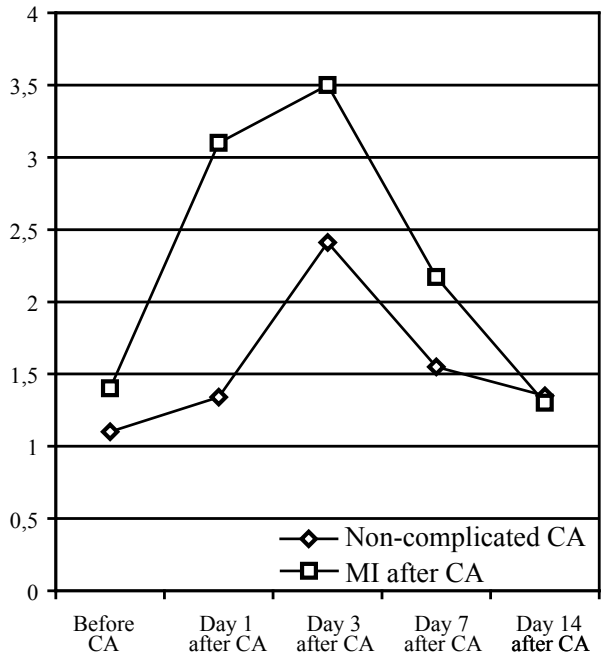

Figure 2. Coefficient of thrombosis risk (CTR, arbitrary units) dynamics in UA patients after coronary angioplasty, uncomplicated vs. complicated by MI.

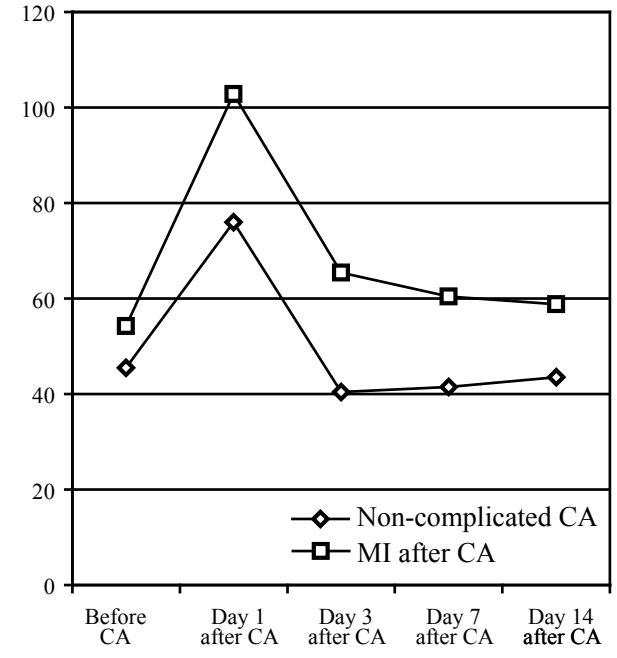

Figure 4. Fibrinogen (FG, g/l) content dynamics in UA patients after coronary angioplasty, uncomplicated vs. complicated by MI.

Conclusions

1. In UA patients following uncomplicated PTCA+stenting, baseline pattern of blood coagulation (increase in FG level and thrombin formation, shown via SFMC) and kinin formation (increase of PK and CKF) showed moderate activation consistent with chronic DIC. On Day 1 post CA there was adequate heparinization (increase in APTT and IHTA of 1.7-fold over baseline) without significant dynamics in FG, fXIII, ATIII, ET, PST, and CTR.

2. Hemostasis-based criteria for postprocedural MI prognosis at preprocedural baseline include activation of coagulation, fibrinolysis and kinin formation consistent with borderline state between chronic and subacute DIC.

3. Hemostasis-based criteria for MI on Day 1 post coronary angioplasty with stenting are disturbances in blood coagulation, fibrinolysis and kinin formation consistent with subacute DIC — pronounced thrombin formation, SFMC as shown by ethanol test, increase in coefficient of thrombosis risk 2 fold or more from baseline, partial consumption of FG, fXIII, intrinsic pathway factors (1.9 fold increase of APTT) and dramatic consumption of AT III, decreasing it 2 or more fold from baseline level.

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Common Femoral Artery Puncture, Aspects of the Approach

M.I. Bilan[1], K.N. Shokhov

Medical and Sanitary Unit of Magnitogorsk Administrative Authorities, Department of angiography and interventional radiology, Magnitogorsk, Russia

Femoral artery (FA) puncture is the most common approach for diagnostic and interventional endovascular procedures (1). Despite the minimally invasive tools and highly effective hemostatic technique used, the rate of puncture site complications remains rather high: 0.3–1% for diagnostic and 1–5% for interventional procedures (2). False aneurysm, arteriovenous fistula, intimal dissection, acute thrombosis of the lower limb's vessels are only few among possible complications of FA puncture.

The purpose of the article is to give more precise technical aspects of the femoral approach, which could make the procedure easier to perform. The report is intended for those, who are currently at the beginning of their career as interventional radiologists; however, it might also be of use for experienced colleagues.

Vascular approach is commonly performed at the level of the common femoral artery (CFA). More proximal (external iliac artery — EIA) or more distal (deep femoral artery — DFA, superficial femoral artery — SFA) has been associated with higher rate of complications (3; 4; 5; 2).

Importantly, the anatomic classification doesn't include the term common femoral artery. This term was introduced by vascular surgeons to define the FA portion between its origin (inguinal ligament) and the DFA origin. Femoral artery is a small caliber artery. Its mean diameter is 6.9 ± 1.4 mm (6.1 ± 1.1 mm in women, which is significantly smaller compared to men — 7.6 ± 1.2 mm) (6). The diameter was found to depend on the body weight, age, the presence of diabetes mellitus. (7). The CFA length is 4.3 to 5 cm (8, 6). The small length of the artery makes the proper puncture site selection crucial.

Early Russian publications give the following puncture reference points: "inguinal fold", "Poupart's ligament" and "inguinal ligament". The puncture site is located on this level or slightly lower (9, 10, 11, 12, 13, 14, 15, 16, 17). D. Grier et al. performed questioning of the interventional radiologists and found that the puncture reference points were: inguinal fold for 39.2% of the specialists, maximum femoral artery pulsation point for 24.7%, bony reference points for 13%, a combination of inglunal fold and maximum femoral artery pulsation for 13%, a combination of three points for 1% (18). The inguinal fold, however, was shown to be 6.7 cm below the inguinal ligament on average (19) and the DFA origin is in 71.9%–78% of cases above the inguinal fold (18, 20).

Analysis of the femoral approach procedures has shown, that the puncture of EIA, DFA and SFA was performed in 13%–35.9% of all cases (6, 2). Such a high rate of undesirable puncture has spurred the specialists to seek more precise reference points for CFA puncture.

Studies have shown that the CFA is always located on the level of the femoral head (FH) (21). DFA was reported to origin distal to the middle of FH in 96.5%–99.4% of cases (6, 20, 2). Therefore, the CFA segment between the middle of FH and its upper edge is considered optimal for puncture (6, 22) (see Fig.1). Some authors propose to perform arterial puncture under fluoroscopic guidance for more precise location of this site (23, 24).

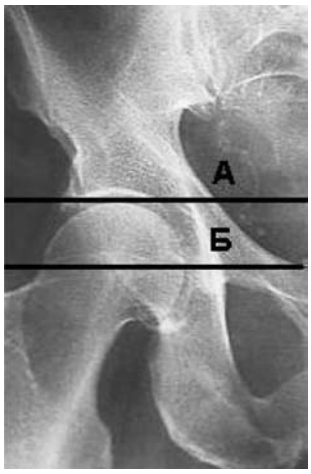

Figure 1. Right hip joint X-ray.
Line A is drawn at the upper edge of the FH, line B is drawn across the middle of the FH. The optimal level of FA puncture is between the lines A and B.

We introduced steps of catheterization intended for more precise determination of the skin puncture site. At the same time, the intervention doesn't require fluoroscopic guidance.

Step 1 is the definition of preliminary puncture site located at the crossing of FA pulse and the inguinal fold. This point should be marked with an X-ray positive instrument (puncture needle, forceps) (see Fig.2). Use fluoroscopic guidance (a single image is enough) to locate the preliminary puncture site and the middle of the FH (see Fig.3). Step 2 includes correction of the puncture site by moving it to the middle of the FH, which is usually 1–2 cm above (see Fig.4). New puncture site location is also controlled by fluoroscopy (see Fig.5).

[1]Bilan M.I.,
18 Naberezhnaya str., Cheliabinsk Region, Magnitogorsk 45500, Diagnostic center.
Tel.: 8(3519) 29-29-81,
e-mail: bilan@mmk.ru
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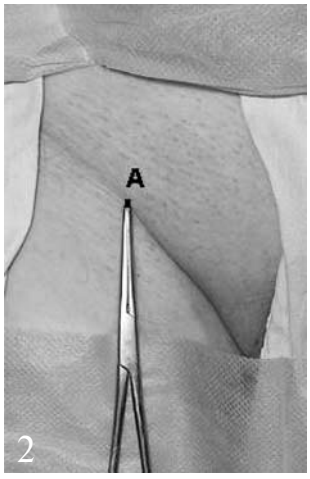


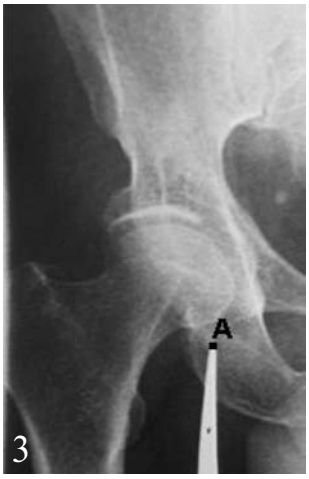
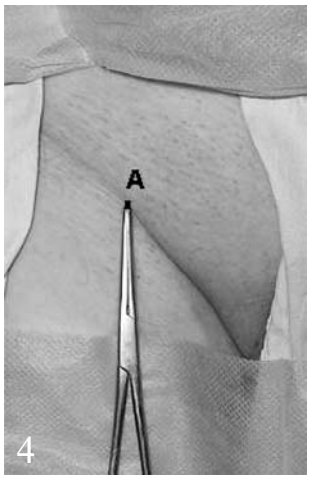


Figure 2. Step 1. Preliminary puncture site is located on the crossing of FA pulse and the inguinal fold.
Figure 3. Step 1. Fluoroscopic control of the preliminary puncture site.



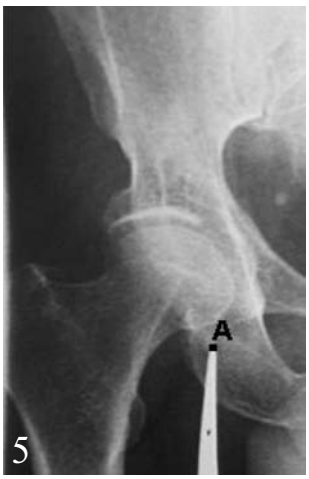
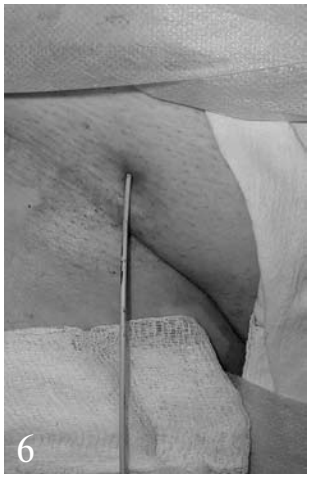


Figure 4. Step 2. Correction of the puncture site.
Figure 5. Step 2. New puncture site location.

In the majority of cases these two steps ensure the location of puncture site over the middle of the FH. Puncture performed at this point allows for catheterization of the CFA (see Figs. 6,7).



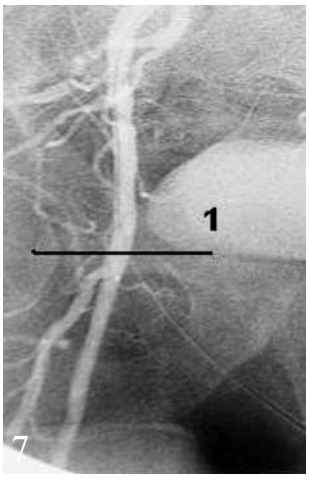


Figure 6. The site of FA puncture on the skin surface.
Figure 7. Femoral artery arteriography (right oblique aspect). The puncture site (line 1) corresponds to the CCA and is located over the middle of the FH.

The technique slightly prolongs the puncture, but ensures high probability of CFA catheterization, which is particularly important in patients at risk of puncture site complications. These groups of patients include:

— women,
— patients with abnormal body mass (obesity or cachexy),
— patients with severe hypertension, diabetes mellitus, coagulation disorders,
— elderly patients, patients with stenosis or severe calcification of the CFA,
— patients requiring thrombolysis or anticoagulation,
— patients requiring large-bore instruments (7F or above),
— patients with prolonged catheterization,
— patients requiring simultaneous catheterization of the femoral vein,
— patients necessitating repeated CFA puncture.

Technical maneuvers increasing the rate of CFA puncture might be inconvenient and prolong the intervention. Nevertheless, when used by the physician they ensure lower per cent of puncture site complications.

CONCLUSIONS

1. Small caliber and length of SFA make the puncture site selection particularly crucial.
2. Conventional criteria for the puncture site selection might not always be optimal.
3. Bony reference points (area between the middle of the FH and its upper edge) are most optimal for CFA catheterization.
4. The puncture site selection should be particularly careful in patients at risk of complications.

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The Editorial Council of the Journal considers necessary to declare its neutral attitude towards the authors' viewpoint.