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PARTICULARITIES OF THE TRICORONARIAN
ATHEROSCLEROTIC LESIONS OCCLUSION
BY ACUTE THROMBOSIS AT THE CX I
IN DIABETIC PATIENT

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Introduction

Numerous studies have been conducted on the link between atherosclerosis and atherothrombotic events. The role of endothelium is to maintain vascular health. The endothelium modulates vascular flow by controlling vasodilator tonus, inhibiting platelet aggregation and clotting factors, forming a barrier over the procoagulant subendothelial layer. Endothelium also acts as a barrier against infamy and has the ability to self-repair in case of injury.

Studies show that endothelial dysfunction is a predictor of the progression of atherosclerosis and acute coronary events in patients with or without known coronary disease. Patients with diabetes have disorders in the coagulation system, which causes a hypercoagulable status [5].

About 70% of cases of acute coronary thrombosis involve the dislocation of an atherosclerotic plaque and 30% involve superficial lesions of the intima at the site of thrombus formation. Superficial endothelial lesions, which cause coronary thrombosis, most commonly occur in women and diabetic patients with hypercholesterolemia. It is assumed that the cause of lesions would be the action of metalloproteinases in the subendothelial layer, which dislocates the endothelial cells from the basal lamina, causing desquamation. Up to 25% of endothelial erosions occur asymmetrically [4].

The systemic origin of endothelial dysfunction aggravates the process of atherosclerosis and consequently occurs with acute coronary syndrome or chronic ischemic coronary heart disease.

PCI in the first hours of STEMI can be divided into primary PCI, PCI combined with reperfusion pharmacological therapy (PCI) and rescue PCI after failure of pharmacological reperfusion. Primary PCI (balloon inflation) should be performed in all cases within the first two hours of first medical contact. Diabetic patients with angina pectoris symptoms should be screened by early coronary angiography, and primary PCI will be the preferred therapy in these patients.

Clinical case

Patient D., aged 58 years, was urgently hospitalized in the Cardiology Recovery Section on 07.03.2017.

Accusation at admission. Retrosternal pain of constrictive character, irradiation in the shoulder and the left hand, present at rest, moderate intensity dyspnea, headache, dizziness, general weakness.

The history of the disease. He is considered ill for many years with HTA. It's under the family doctor's record. Outpatient treatment is irregularly administered. In the last 3-4 days the state gradually worsens, presenting the above-mentioned accusations. On March 7, 2017, he requested the AMU service. He was urgently transported and hospitalized in the IMSP SCM Holy Trinity Hospital for the diagnosis and treatment tactics.

Objective data: overall status of mean severity. Pink-pale skin. Rash is missing. Peripheral edema absent. Auscultatively throughout the lung area there is a vesicular murmur, missing ralles, FR = 18/min. Apexian shock is determined in the intercostal space V with 1.5 cm lateral to the left medioclavicular line, 1.5 cm wide. Power and moderate resistance. Rhythmic, attenuated cardiac noises, FCC – 68 beats/minute, TA – 140/80 mmHg. The abdomen is enlarged because of mass of adipose tissue and have soft palpation. No change in liver and spleen. Current intestinal transit. No pain during micturition. Negative Giordano sign bilateral.

Paraclinic examination: hemoleucogram: hemoglobin – 166 g/l, erythrocyte – 5,1x10¹²/l, color index – 0,83, hematocrit – 43,4%, leukocyte – 10,2x10⁹/l, unshed – 8% – 3%, lymphocyte – 15%, monocyte – 8%, VSH – 24 mm/h.

Biochemical analysis: prothrombin – 119%, fibrinogen – 4.4 g/l, urea – 5.2 mmol/l, creatinine – 0.09 mmol/l, glucose – 7.2 mmol/l, ALT – 139, AST – 116 u/l, total cholesterol – 6.2 mmol/l, triglyceride – 2.30 mmol/l, potassium – 4.9 mmol/l, natrium – 147 mmol/l.

Echocardiographic examination: 13.03.2017 – Induction of ascending Aortic Wall, VAo, VM. The cavities of the heart are not dilated. Concentric hypertrophy of the VS myocardium. The apical segment hypokinesia of the PPVS myocardium, the apical and middle segment of the PLVS myocardium. The pump function of the VS myocardium is sufficient. FE Simpson – 56%. Echo-CS Doppler: V max – N. Insufficient VM gr. I-II, VT gr. I-II, VAP gr. I. Impairment of relaxation of the VS myocardium.

Coronary angioplasty: triconaric atherosclerotic lesions; occlusion through acute thrombosis on Cx I. Moderate stenosis on LAD II, RCA II.

Angioplasty Protocol. Acute thrombosis occlusion of Cx I is attempted. The common left coronary artery is intubated with a 3.5-6F XB catheter. The lesion was traversed by a *PILOT 50*, 0.014 to the distal portion of the vessel. Pre-treatment with a 2.5-20 mm SC swollen flask of 8 atm – 15 sec is practiced. Then implant the stent *DES PROMUS* 2.75-20 mm, inflated at 8 atm – 15 sec. The proximal segment of the stent was postdiluted with a balloon *NC3.0* – 15 mm, swollen at 14 atm 15 sec. There is a reduction in lesion score from 100% to 0 with TIMI III flow and good myocardial blush. Residual stenosis and dissections are not determined. Was't complication during the intervention.

Clinical diagnosis

Tricoronaric atherosclerotic lesions. Occlusion through acute thrombosis on aCx I. Moderate stenosis on LAD II, RCA II. Condition after PCI on aCx I (07.03.2017). Extremely high risk of high blood pressure. ICC II (NYHA), stage B ACC/AHA. Type II subcompensated diabetes mellitus. Dyslipidemia.

aCx I before and after angioplasty



Stationary treatment

Tab. Cardiomagnyl – 75 mg, Tab. Plavix – 75 mg, Sol. Cardimac, Sol. Pyracetam, Tab. Mildronat – 500 mg 2 daily.

Conclusions

Patient D., aged 58 years, accusing retrosternal pain of constrictive character, irradiation in the sho-

ulder and the left hand, present and rest, moderate intensity dyspnea, headache, dizziness, general weakness following a coronar angiography Established diagnosis: Tricoronaric atherosclerotic lesions. Occlusion through acute thrombosis on aCx I. Moderate stenosis on LAD II, RCA II. A stent was applied with revascularization of the respective region. The risk factors to which the patient is subjected, namely the presence of diabetes mellitus, HTA, irregular treatment of ambulatory treatment, and dyslipidemia with 6.2 mmol/l cholesterol were determined from the biochemical analysis. SCORE score of 6%, which indicates a high risk of cardiovascular death over the next 10 years.

The patient is recommended to be registered with the family doctor, cardiologist, with dynamic monitoring of TA, glucose, lipid profile. Respecting the proper diet and a healthy lifestyle.

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