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MULTICOMPLICATED MYOCARDIAL INFARCTION

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The clinical course of myocardial infarction frequently burdened by a variety of complications, which largely determine its scenario and prognosis. Despite advances in the management of this disease, complications can and do occur. These dangers arise from two principal sources: on the one hand, from the local effects of the lesion and the circulatory depression that follows, and, on the other, from the hazards that may attend during active treatment.

On example of clinical case is demonstrated severe course of myocardial infarction with development of multiple complications at different stages, but, ultimately, with a favorable outcome. Early, aggressive, and judicious treatment of these complications may substantially decrease the morbidity and mortality associated with this disease.

KEY WORDS: myocardial infarction, complications of myocardial infarction

ИНФАРКТ МИОКАРДУ З БЕЗЛІЧЧЮ УСКЛАДНЕНЬ

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Клінічний перебіг інфаркту міокарда часто обтяжується різними ускладненнями, які значною мірою визначають його сценарій і прогноз. Незважаючи на успіхи в лікуванні цього захворювання, ускладнення можуть траплятися і трапляються. Ці загрози виникають з двох основних джерел: з одного боку – це локальні ефекти вогнища ураження і подальша декомпенсація кровообігу, з іншого – це ризики, пов'язані з активним лікуванням.

На прикладі клінічного випадку розглядається важкий перебіг інфаркту міокарда з розвитком безліч ускладнень на різних етапах, проте, у кінцевому підсумку, зі сприятливим кінцем. Раннє, агресивне і розумне лікування цих ускладнень може істотно знизити важкість перебігу і смертність, пов'язані з цим захворюванням.

КЛЮЧОВІ СЛОВА: інфаркт міокарду, ускладнення інфаркту міокарда

ИНФАРКТ МИОКАРДА С МНОЖЕСТВОМ ОСЛОЖНЕНИЙ

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Клиническое течение инфаркта миокарда зачастую отягощается множеством осложнений, которые, в свою очередь, определяют его сценарий и прогноз. Несмотря на достижения в лечении данной патологии, осложнения могут и продолжают встречаться. Эти угрозы возникают из двух основных источников: с одной стороны – это локальные эффекты очага поражения и последующая декомпенсация кровообращения, с другой – это опасности, связанные с активным лечением.

На примере клинического случая рассматривается тяжелое течение инфаркта миокарда с развитием множества осложнений на разных этапах, однако, в конечном итоге, с благоприятным исходом. Раннее, агрессивное и разумное лечение этих осложнений может существенно снизить тяжесть течения и смертность, связанные с этим заболеванием.

КЛЮЧЕВЫЕ СЛОВА: инфаркт миокарда, осложнения инфаркта миокарда

INTRODUCTION

The clinical course of myocardial infarction frequently burdened by a variety of complica-

tions, which largely determine its scenario and prognosis [1]. Complications are different and life threatening. Despite advances in the

management of this disease, complications can and do occur.

Acute myocardial infarction frequently takes life in the first hour after a heart attack, before qualified medical aid is provided. If a person survives the sudden loss part of the ventricular muscle due to ischemic necrosis, there follows a period in which special dangers threaten each of the physical systems and the personality itself [2]. These dangers arise from two principal sources: on the one hand, from the local effects of the lesion and the circulatory depression that follows, and, on the other, from the hazards that may attend during active treatment [2]. Development of complications are determined not only by the size of lesion, but also a combination of conditions (first of all, the state of the myocardium on the background of atherosclerosis of the coronary arteries, prior myocardial diseases, presence of electrolyte abnormalities) [1]. The onset of each of these complications usually results in explicit symptoms and physical manifestations. Thus, a basic knowledge of the complications that occur in the postinfarction period and the clinical syndromes associated with each, will allow the physician to evaluate and treat the complication appropriately. Prompt diagnosis and therapy are life-saving. Outcome of patients with myocardial infarction is determined by the complications that develop in the early and late stages of the disease.

Main purpose of the doctor to create conditions for uncomplicated healing of lesion, prevent inadequate stress response – distress [3]. The mechanism of complications is always the same: desynchronization of necrotic and reparative processes [3].

On example of clinical case is demonstrated severe course of myocardial infarction with development of multiple complications: on the one hand, from the focal necrosis of the heart muscle and the pump dysfunction that ensue, and, on the other, from the side effects that accompany active treatment. In this incident the severity of complications exceeded the severity of myocardial infarction itself. It needed the set of interventions to take out the patient from such grave condition.

OBJECTIVE

The purpose of this article is to represent the complications that may arise in the course of acute MI and demonstrate that various

therapeutic modalities, both medical and surgical, should be able to improve not only symptoms but also survival of the patient.

CLINICAL CASE

65 years old retired male, resident of urban area.

COMPLAINTS

Patient complain of burning central chest pain, more than 60 min duration, without any radiation, nitroglycerine intake doesn't relief pain, abrupt onset, severity 7; and dyspnea at rest, exacerbated by minimal physical exertion

PRESENT ILLNESS

Central chest pain and worsening symptoms of dyspnea had been appeared abruptly, when the patient was at home and carried out household chores. Patient took nitroglycerine three times, but it did not relief pain. After 30 min patient had called to emergency. ECG had been recorded. Signs of STEMI of the posterior wall had been found and patient had been delivered to cardiologic emergency department.

PAST MEDICAL HISTORY

Over 20 years patient suffer from essential hypertension. Patient said that prescribed by cardiologist medications (ACE inhibitors, diuretics and b-blockers) had been taken regularly, but BP level had not been controlled properly (it occurred rising of BP to 160–200/100 mm Hg).

In 1999 patient underwent left internal carotid endarterectomy was carried out because of carotid artery atherosclerosis.

Since 2003 year bother persistent atrial fibrillation tachysystolic form, which on repeated occasions was successfully converted to sinus rhythm by pharmacologic cardioversion (IV amiodarone)?

25.02.2010 patient had STEMI inferior wall complicated by cardiogenic shock, Dressler's syndrome.

06.12.2010 patient had ischemic stroke in the circle of Willis.

DRUG HISTORY

Patient intake following medicines, prescribed by cardiologist: nebivolol 5 mg per day, losartan 50 mg per day, hydrochlorothiazide 25 mg per day, aspirin 75 mg per

day, clopidogrel 75 mg per day, atorvastatin 20 mg pd.

FAMILY HISTORY

Patient's mother and brother suffer from essential hypertension.

ALCOHOL AND SMOKING

Patient intake about 1,5 L of normal beer per day, equivalent 42 units of alcohol per week.

Patient smoke 1,5–2 packs of cigarettes per day during 40 years, which equals 60–80 pack-years.

INSPECTION

Vital signs:

Body temperature – 36,8 °C

PS – 40 bpm

BP – 110/60 mm Hg

Respiratory rate – 17 pm

High – 188 cm

Weight – 105 kg

BMI – 30,2 kg/m²

Elderly male, has correct orientation in space and surroundings, mild depressed. The posture is orthopnea (the patient uses 3 pillows). Skin is pale, mild cyanosis of the lips, fingers and toes, rashes and hemorrhages are absent. Turgor and elasticity of the skin is decreased. Subcutaneous fat tissue is increased, predominantly in abdominal zone (central obesity, waist circumflex 138 cm). Nails are without any abnormalities. Mucous membranes are pale and wet. Tongue is clear and wet. Severe edema of the low extremities (3+). Lymph nodes are not palpable. Joints are normal, active and passive movements are painless. The head and neck examination is normal. Carotid pulsation. JVP 6,5 cm above the sternal angle. The chest is normal shape. Decrease breath sounds and bibasilar coarse crackles of the lungs to auscultation. The point of apex beat is diffuse, 3 cm in diameter, shifted to the left (palpated 1,5–2 cm to the left from midclavicular line in the 5th intercostal space). S1 and S2 are soft. Holosystolic murmur best heard at the tricuspid valve. Abdomen is soft and nontender. Hepatomegaly (+ 4 cm), liver palpation is tender. The kidneys are not palpable. Stool is normal. Urination is normal.

CLINICAL DATA

At the time of admission to the hospital complete blood count detected neutrophilic leukocytosis (WBC 11.8 10⁹/L, NE 9.1

10⁹/L, 77,8 %); urine analysis – mild proteinuria (0.068 g/L); biochemical blood profile revealed normal ranges of serum glucose (4.1 mmol/L) and bilirubin (9.98 mkmol/L), hyperfermentemia – increased levels of AST (103.4 U/L) and ALT (64.2 U/L), raised levels of creatinine (424.97mkmol/L) and urea (32.6 mmol/L), hypokalemia (3.1 mmol/L), hypoproteinemia (48.4 g/L); cardiac biomarkers were increased – CK-NAC 364.3 U/l, CK-MB 68.83 U/l.

ECG on admission: bradycardia, heart rate 40 bpm, junctional rhythm, LBBB (QRS 0.14 ms), acute circular MI (ST elevation > 2 mm III, AVF, V1-V5, ST depression 1 mm I, AVL).

Transthoracic echocardiogram data: Signs of total heart failure with hypertrophy and dilation of heart chambers, valvular regurgitation, LV contractility reduction. Development of pulmonary hypertension. Hydropericardium. Hypokinesia of the LV posterior wall, which is affected by infarction.

Data of the abdomen ultrasound: diffuse alteration of liver and pancreatic parenchyma; hepatomegaly; venous liver congestion; bilateral hydrothorax.

CLINICAL DIAGNOSIS

Main disease

Coronary artery disease. Acute repeated ST elevated circular myocardial infarction (28.08.2015). Postinfarction inferior wall cardiosclerosis (2006). Killip IV.

Essential hypertension III stage, 2 grade.

Persistent atrial fibrillation, tachysystolic form.

Risk score 4 (very high).

Chronic congestive heart failure II NYHA with the reduction of LV contractility.

Complication

Junctional rhythm, bradycardia

Acute prerenal failure

Concomitant diagnosis

Alcoholic liver disease

Obesity class I

COURSE OF DISEASE

On the third day of the disease early in the morning in arose complains such as black colored stools, fatigue, and dizziness.

During inspection: body T – 36,5° C, Pulse – 34 bpm, BP – 90/60 mm Hg, respiratory rate – 20 pm. Patient was drowsy and sluggish. Skin and mucous membranes were pale and dry.

Bronchial breath sounds of the lungs to auscultation. Decrease breath sounds in bases. Rhonchi and crackles were not auscultated. Pulse was regular, soft and small (pulsus filiformis). Soft S1 heart sound to auscultation. Abdomen was soft and tender in epigastric region. Hepatomegaly (+4 cm), palpation of liver was tender. The kidneys were not palpable.

Complete blood count revealed rapid significant decline level of RBC (2.39 $10^{12}/L$), Hb (76 g/L), and HCT (23.1 %); persisting neutrophilic leukocytosis (WBC $12.5 \cdot 10^9/L$, NE $9.5 \cdot 10^9/L$, 76.1 %).

Fibroscope was carried out to the patient: several acute ulcers 0,5–0,8 cm in diameter, covered by fibrin were found in antrum of the stomach.

The same day in the evening in patient took place respiratory arrest and cardiac arrest: absence of the breathing and pulsation of the main arteries, pupil dilation, and loss of consciousness.

ECG-monitor: isoline.

In patient developed clinical death. Emergency measures: IV epinephrine, indirect cardiac massage. After 3 min, emergency measures were successful: cardiac activity and respiration were restored – PS 80 bpm, BP 150/100 mmHg, RR 18 per min.

In this case the most prominent atherosclerotic plaque was localized in the right coronary artery. The right coronary artery distributes blood to right ventricle, right atrium, posterior portion of the interventricular septum, posterior wall of the left ventricle and the heart conduction system (including sinoatrial node). Ischemia of SA node may lead to its dysfunction (bradycardia, SA arrest, etc.)

Considering the severity of the patient's condition, refractory bradycardia (HR 40 bpm), developed acute renal failure and GIT bleeding, clinical death, to improve patient's condition, temporal pacemaker was implanted.

Temporal pacemaker implantation improved organ perfusion: renal failure abated (creatinine 74.77 $\mu\text{mol}/L$, urea 9.8 mmol/L), also occurred rising of total protein level (50.1 g/L) and rising level of potassium (5.0 mmol/L) to normal ranges.

Seven days after pacemaker implantation fever had been developed, body temperature was in ranges 37.7–38.2°C, also persist neutrophilic leukocytosis (WBC $10.0 \cdot 10^9/L$,

NE $7.9 \cdot 10^9/L$, 79.7 %) and increased ESR (18 mm/h).

Transthoracic echocardiogram was repeated: development of bacterial vegetations on the right coronary cusp of aortic valve were revealed; signs of total heart failure with hypertrophy and dilation of heart chambers, valvular regurgitation, LV contractility reduction, pulmonary hypertension, hydropericardium, hypokinesia of the LV posterior wall were preserved.

Chest X-Ray was carried out: absence of pulmonary seeding, pulmonary congestion, bilateral hydrothorax, cardiomegaly.

Blood culture samples were negative.

FINAL DIAGNOSIS

Main disease

Coronary artery disease. Repeated ST elevated circular myocardial infarction (28.08.2015). Postinfarction inferior wall cardiosclerosis (2006). Killip IV

Essential hypertension III stage, 2 grade

Persistent atrial fibrillation, tachysystolic form

Chronic heart failure with systolic dysfunction of left ventricle

Risk score 4 (very high).

Complication

Junctional rhythm, bradycardia (40 bpm)

Acute prerenal failure

Clinical death

Temporal pacemaker implantation

Possible nasocomial active device-related (temporal pacemaker) infective endocarditis

Acute gastric stress ulcers, GIT bleeding

Anemia of blood loss, moderate

Concomitant diagnosis

Alcoholic liver disease, alcoholic hepatitis

Obesity class I

MANAGEMENT

1. Life style modification: diet: low in saturated fat and high in omega-3 fat, low carbohydrates, low sodium (3g/d); limit alcohol consumption; body weight control: goal BMI 18.5–24.99; smoking cessation.

2. Acute coronary syndrome treatment: IV Morphine sulfate; low molecular weight heparin (enoxaparin 80 mg/2d); aspirin 75 mg/d; clopidogrel 75 mg/d; atorvastatin 80 mg/d; eplerenonum 25 mg/d; ramipril 2,5 mg/d; pantoprazole 40 mg/d [4].

Additional recommendations: thrombolytic therapy (IV alteplase) or PCI [5].

3. Bradycardia treatment: atropine IV/IM; temporal pacemaker implantation [6].

Recommendations: pacemaker implantation change to transcutaneous pacing to avoid infective endocarditis development [7].

4. Infective endocarditis treatment: pacemaker removal; IV Cefepime 1,0 g 2 times/d 14 days; IV Vancomycin 2 g 1 times/d 7 days [8].

5. Acute renal failure treatment: reduction of cardiac output due to myocardial infarction and bradycardia due to sinoatrial ischemia, hypovolemia due to GI bleeding lead to prerenal acute kidney injury. Temporal pacemaker implantation improves cardiac output and kidney perfusion. IV solutions to increase blood volume are not indicated, because of increasing heart preload and heart demands [9–10].

6. GIT bleeding treatment: withhold of low molecular weight heparin, aspirin, and clopidogrel. Prescribe: hemostatic therapy (IV ε-aminocaproic acid 100,0 2 times/d, IV etamsilat 12,5 % 4,0 3 times/d, IV menadione 1 % 1,0 3 times/d); antisecretory drugs (IV

famotidine 20mg 2 times/d, IV pantoprazole 40 mg 1 time/d) [11].

7. Anemia treatment: diet (red meat, beetroot, spinach, pomegranates, soy beans, whole grain bread, peaches, prunes and raisins); ferrous sulfate 60 mg/d per os for 3 months; folic acid 400 mcg/day per os for 3 months [12].

8. Alcoholic liver disease treatment: Essential phospholipids 300 mg/ 2d for 3 months; Argininum 1.5 g/ 2d for 3 months.

CONCLUSIONS

The case report demonstrates complicated myocardial infarction, and reminds clinicians that prompt recognition and management are critical in this uncommon grave clinical case. Clinicians must keep potentially lethal complications in mind when evaluating these unstable patients. Early, aggressive, and judicious treatment of these complications may substantially decrease the morbidity and mortality associated with acute myocardial infarction.

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