CLINICAL CASE

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INTENSIVE THERAPY FOR SEVERE POST-OPERATIVE COMPLICATIONS IN A CAD PATIENT AFTER A BYPASS SURGERY

ABSTRACT

The article presents a clinical case of severe postoperative complications with a clinical death episode in a patient with coronary artery disease after a bypass surgery. On the second day after the surgery, the patient, who had underwent a mammary coronary bypass surgery of the anterior descending artery and an aortocoronary bypass surgery of the right coronary artery and circumflex artery with artificial circulation, developed acute kidney injury, requiring renal replacement therapy. In the following days, there was esophageal-gastric bleeding with the development of DIC-syndrome and hemorrhagic shock, resulting in clinical death registered for 2 minutes. After a successful cardiopulmonary resuscitation, a number of other serious complications were observed: acute respiratory distress syndrome and acute destructive calculous cholecystitis, treated with cholecystectomy.

A comprehensive intensive therapy, including vasopressor/inotropic hemodynamic support, aggressive respiratory therapy, efferent methods of detoxification, resulted in successful management of all the complications, stabilization of the patient's condition, restoration of the functions of organs and systems and recovery of the patient.

Keywords: coronary artery disease, coronary artery and mammary coronary bypass surgeries, acute kidney injury, esophageal-gastric bleeding, hemorrhagic shock, acute respiratory distress syndrome.

INTRODUCTION

Despite the high efficiency of bypass surgeries in the treatment of coronary artery disease (CAD), these operations are associated with a high risk of developing a number of complications, including thrombosis[8], bleeding[3], myocardial infarction [6], acute kidney injury (AKI) [5], acute respiratory distress syndrome (ARDS) [1], and infectious complications [7]. Complications in the perioperative period are most common in people suffering from diabetes, excessive body weight, chronic lung and kidney diseases [2]. The use of artificial circulation (AC) during the surgery, as well as antiplatelet and anticoagulant therapy, can initiate failures in the blood coagulation system with pronounced thrombocytopenia and the development of hemorrhagic syndrome.

The **objective of the research was** to analyze severe postoperative complications with a clinical death episode developed in a CAD patient after a coronary artery and mammary coronary bypass surgeries.

The material of the research was a clinical case of intensive therapy of a patient with the following diagnosis:

Principal: CAD. Stable angina of class 3. Postinfarction cardiosclerosis from 06 September 2007. Coronary sclerosis. Condition after stenting the anterior interventricular branch (AIB) of the left coronary artery (LCA) in 2007, transluminal balloon angioplasty (TBA) and right coronary artery (RCA) stenting in 2012.

Secondary: Diabetes mellitus, type 2 in the stage of decompensation. Diabetic retinopathy OU, nephropathy, polyneuropathy. Obesity of degree 2, body mass index (BMI) - 42, android type. Dislipidemia. Hypertensive disease of degree 3. Arterial hypertension of degree 3, cardiac risk of degree 4. Papillary adenocarcinoma of the left lobe of the thyroid gland $T3N^0M^0$. Condition after radical strumectomy in2012.

Clinical observation. Patient M., 58, was admitted to the Cardiac Surgery Unit RH No.1- NCM for an examination and further CAD treatment.

Complaints at admission: short breath, a burning sensation behind the sternum after little physical exertion (walking for a distance under 50 m, going up one floor), increase in arterial pressure, and blurry vision.

From the *history*, it is known that the patient has suffered from CAD since 2007, when first angina pains appeared after moderate physical exertion. In the same year, he suffered a myocardial infarction of the anterior wall of the left ventricle with the formation of the Q wave. During the examination, an occlusion was identified with stenosis of the coronary arteries, which was treated with stenting of the middle third of the AIB and RCA. In 2015, because of restenosis, stenting of the PCA was performed.

The concomitant diseases included hypertension with the maximum value of systolic blood pressure up to 230 mmHg (since 2010) –the administered antihypertensive therapy was not effective; chronic calculous cholecystitis.

Objective status: State of moderate severity. Alert.

The skin and visible mucous membranes were of normal color, t of body was 36.6 C^0. Excessive type of nutrition, hypersthenic, height 160 cm, weight - 105 kg (BMI - 42). The peripheral lymph nodes were not enlarged.

The lung breathing was vesicular, carried out in all sections, there was no wheezing, respiration rate was 18 per minute.

The heart sounds were muffled, rhythmic, heart rate was 68 per minute. The blood pressure was 140/82 mmHg.

The tongue was clean, moist. The abdomen was soft, painless, enlarged by subcutaneous fat. The liver lied along the costal arch edge. The stool had shape, without pathological impurities.

Diuresis was adequate. Peripheral edema was absent.

On 03 February 2016, with artificial circulation, pharmacological cold cardioplegia and under combined endotracheal anesthesia, the unit surgeons performed the coronary artery bypass surgery (CABS) of the RCA and circumflex artery (CA), as well as the mammary coronary bypass surgery (MCBS) of the anterior descending artery (ADA). The surgery lasted for 04 hours 35 minutes, duration of anesthesia – 05 hours 35 minutes.

The course of general anesthesia: inhalation low-flow anesthesia with Supran (0.2-4.0 % vol.) with central fentanyl analgesia (5-10 μg/kg), without any peculiarities. Antegrade cardioplegia with Custodiol solution 2000 ml, cooled to $+6^0C$, pressure in the aorta about 100 mmHg, asystole on the 2nd minute. The time of

AC – 02 hours 31 minutes, the time of aortic compression– 01 hour 39 minutes. The calculated volumetric perfusion rate – 5.28 ml/min, systemic BP during AC – 62/57 mmHg, the apparatus pressure in the arterial main line of the AC was 130-140 mmHg. Hypothermia down to 34.0^{0}C. During the AC, the partial pressure PaO_2, $PaCO_2$ and sVO_2, as well as the parameters of the acid-base state were within the limits of normal values.

At the end of the main stage of the surgery, after warming the body to 36.1^{0}C, the cardiac activity was restored, external stimulation of the heart was performed, after which there developed ventricular fibrillation. The rhythm was restored by defibrillation with a discharge of 50 J on the open heart. Antiarrhythmic therapy: Lidocaine 1 mg/kg, magnesium sulfate 250 mg. The patient's own rhythm was restored, in the form of sinus bradycardia, heart rate at 45 per minute. The temporary pacemaker (5A, frequency 78 per minute) was connected. The hemodynamics with inotropic support of Dopamine at 3 μg/kg/min, CVP in the 8-12 mmHg range.

The intraoperative blood loss was 150 ml due to the amount blood with tissue wastes and surgical material; the rest of the blood was reinfused with the help of drainage suction of the AC and CellSaver apparatus.

After the surgery, the patient was taken to the Intensive Care Unit. They continued ALV in CMV mode: respiratory volume 730 ml, RMV 9.5 l/ in, PEEP + 5 cm w.c., FiO_2 45%, Paw18-20 w.c., SpO_2 99%.

Days 1-3 (03-05 Feb 2016). In12 hours after admission to ICU, after becoming alert again, gaining the adequate breathing, stable hemodynamics and satisfactory muscle tone, the patient was extubated. However, 30 minutes after the extubation, the patient complained of shortness of breath. Noisy wheezing was noted, as well as dyspnea of a mixed character with a RR at 24 per minute. The skin and visible mucous were pale pink, SpO_2 93% due to administration of moistened oxygen (5 l/min) through a face mask. The gas composition of the blood: PaO_2 110 mmHg, PCO_2 35 mmHg, SvO_2 97%, Shunt 7%. After another 30 minutes, the patient was excited, dyspnea reached 34-36 breaths per minute, cyanosis of the visible mucous membranes appeared, SpO_2 – 88%, AP – 150/87 mmHg, CVP – 13-14 mmHg. Gases of the blood: PaO_2 85 mmHg, $PaCO_2$ 56 mmHg, SvO_2 47%, Shunt15%. Taking into account the respiratory failure and hypoxemia, the patient was intubated and transferred to ALV in the CMV mode with moderate hyperventilation f 16-18 per minute, FIO_2– 60%. The analysis of the blood gases after 10 minutes showed: PaO_2 104 mmHg, SvO_2 85%, Shunt 17%. The hemodynamics was stable, diuresis was adequate 1 ml/kg/hour. Propofol sedation was started at a dose of 2-3 mg/kg/hr.

In the following days, the mechanical ventilation continued; with an attempt to transfer the patient to spontaneous breathing, he quickly got tired, and signs of hypoxia appeared. Gases of blood: PaO_2 102 mmHg, PCO_2 34 mmHg, SvO_2 88.1%, Shunt 24.6%.

The worsening of the patient's condition was noted on 05 Feb 2016: hyperthermia up to 38.8^{0}C, increase in leukocytosis up to 21.2*10^9/l, hyperosmolarity (298 mosmol/l) due to hypernatremia (Na+ - 150 mmol/l) and hyperglycemia (blood glucose up to 19 mmol/l). The signs of acute kidney injury (AKI) manifested: blood urea samples 17.5 mmol/l, creatinine 240 μmol/l; decrease in diuresis 0.8 ml/kg/hour; GFR 24.7 ml/min 1.73 m^2; edema on the face and feet due to stimulation of diuresis by loop diuretics. AVL was continued, antibacterial therapy was changed.

Days 4-8 (06-09 Feb 2016). In connection with the progressing renal failure and oliguria at 0.25 ml/kg/hour, there was started daily renal replacement therapy (RRT) in the regime CVVHDF, ultrafiltration (UF) 100 ml/ h with Heparin 5-10 thousand units. GFR (CKD-EPI) – 41.5 ml/min/1.73m^2.

On day 8 after the surgery, after becoming alert again, gaining the adequate breathing, stable hemodynamics, satisfactory muscle tone, normalized acid-base and blood gases balance, the patient was extubated.

Days 9-12 (10-13 Feb 2016). On day 9, the nasogastric tube showed discharge looking like coffee grounds. EGD revealed an acute ulcer in the body of the stomach in the area of greater curvature, with signs of bleeding (Forrest 1a). The endoscopic injection method of hemostasis was administered. The antiplatelets were canceled. Conservative hemostatic therapy was prescribed: two-component antiulcer therapy – a blocker of the third generation of H_2-histamine receptors (Famotidine 40 mg per day) and Nexium proton pump inhibitor (starting dose of 80 mg IV and then a supporting dose of 8 mg/h IV for 72 hours).

The blood tests showed anemia up to 67 g/l (Table 2), a transfusion of fresh frozen plasma and erythrocyte mass was started.

On day 11, the patient had a relapse of gastric bleeding; endoscopic hemostasis was repeated, conservative hemostatic therapy was continued. Despite the treatment, unstable hemodynamics was observed as a manifestation of the hemorrhagic shock. The ongoing infusion-transfusion therapy (ITT) was supplemented by vasopressor (Norepinephrine 0.5-1.0 mcg/kg/min, Adrenaline 50-100 ng/kg/min) and inotropic supports (Dopamine 10-15 mcg/kg/min). Hemodynamic parameters of the mean arterial pressure (MAP) were ≤ 86 mmHg.

In the setting of complex rhythm disturbances in the multiple ventricular extrasystoles type, ventricular fibrillation was observed. Cardiopulmonary resuscitation (CPR) was started: external cardiac massage, artificial lung ventilation, defibrillation 1 discharge (200 J), followed by restoration of the cardiac activity (2 minutes after clinical death).

In the early postresuscitation period, the patient continued with ALV, medication sedation (Propofol 1-2 mg/kg/h). Due to the development of paroxysmal atrial

Table 1

The dynamics of indices of the excretory function of the kidneys

Days after the surgery	Indicator			
	Urea, mmol/l	Creatinine, μmol/l	GFR ml/ min/1.73m^2	Diuresis, ml
1	7	139	47,7	4840
3	17,5	240	24,7	2400
5	12,7	156	41,5	650
7	22	174	36,4	630
9	21	142	46	700
11	28	201	30,6	600
13	27	274	21	540
15	26	300	18,5	515
23	19	369	14,6	420
35	26	642	7	0
45	26	331	16,7	200
50	27	312	18	5000>
81	24	280	19	4000
109 (day of transfer from ICU)	10	168	38	3000

Table 2

The dynamics of the complete blood count indicators

Days after the surgery	Indicator				
	Erythrocytes, *1012/l	Hb, g/l	Ht, %	Thrombocytes, *109/l	Leucocytes, *109/l
1	3,27	104	26,7	168	16
3	3,22	102	30	130	21
5	5,73	109	53,1	68	10
7	3,06	90	28,5	140	22
9	3	90	28	138	21
11	2,6	68	29	142	24
13	2,6	68	25	90	26
16	3,2	82	26	94	22
28	2,84	81	25,6	248	8,7
81	3,4	84	25,6	287	14
109 (day of transfer from ICU)	3,05	79	27,78	183	7,8

flutter with 1:1ratio, antiarrhythmic therapy was initiated (Amiodarone 1200 mg/day). Given the extremely difficult condition, it was decided to refrain from open surgical intervention; the endoscopic clipping of the bleeding stomach vessel was repeated.

On day 12 after the surgery, the nasogastric tube again showed copious discharge of the dark brown color. An emergency laparotomy and transverse gastrotomy were performed. The revision of the stomach showed sound vascular clips, imposed previously, an ulcer 7-8 mm with active bleeding in the lower third of the body of the stomach along its posterior wall. Sewing (Z-shaped nodular suture) of the bleeding site and ligation of the left gastric artery were performed. With further revision of the abdominal cavity, a destructive gallbladder with necrosis areas was revealed, and therefore a cholecystectomy was executed.

Days 13-15 (14-16 Feb 2016). The patient's condition remained extremely serious; there were no signs of gastrointestinal hemorrhage. The staff continued medication sedation, ALV, ITT, vasopressor/ inotropic support (MAP ≤86 mmHg), RRT (using Calcium citrate instead of Heparin), parenteral nutrition. Through the tube, inserted into the duodenum, enteral feeding was started. Due to prolonged ventilation, the need for adequate sanitation of the tracheobronchial tree was performed with medial tracheostomy.

On day 15, EGD revealed a linear rupture in the region of the nasal esophagus, which was an indication for the installation of the Blackmore tube. In setting of replacement transfusion therapy, anemia remained (Table 2).

There were signs of pronounced ARF (Table 1). The patient had peripheral anasarca edema, fluid in the pleural cavities. Pleurofix system pleural drainage was installed; 700 ml of serous hemorrhagic fluid was evacuated from the both sides.

Days 16-27 (17-28 Feb 2016). The condition of the patient was still extremely serious. There were no signs of gastrointestinal bleeding, but the anemia remained. The patient received enteral feeding through a tube inserted into the duodenum. There were signs of hemorrhagic bronchitis, CKD-EPI – 11.1 ml/min/1.73 m^2. Anasarca. The unstable hemodynamics remained against a background of microfine infusion of norepinephrine 0.25-0.5 μg/kg/min, epinephrine ≤50 ng/kg/min, and dopamine at a dose of 5-10 μg/kg/min (MAP ≥ 89 mmHg).

In the following days, in the setting of the ongoing mechanical ventilation, the patient developed a clinic of acute respiratory failure with a decrease in SpO_2 ≤ 92%, hypoxemia $PaO_2$89-93 mmHg, SvO_2 66-72 mmHg against the background of FiO_2 0.7-1.0 fraction of oxygen. A computed tomography of the chest organs revealed signs of acute respiratory distress syndrome (ARDS).

Days 28-80 (29Feb – 20 April 2016). During this period, vasopressor amines were cancelled, and the dose of Dopamine was reduced to 3-5 μg/kg/min. The patient demonstrated positive dynamics, caused by the restoration of diuresis, and then polyuria in the first 10 days (1.0-2.0 ml/kg/h). GFR was 7-19 ml/min /1.73 m^2. The computer tomography of the chest organs showed positive dynamics – there were no signs of ARDS, there were foci of fibrous changes in the lungs and insignificant stagnation in the small circle of blood circulation. RRT continued under the indications, on average, 1 session every 3 days.

Days 81-108 (**21April – 21May 2016).** The condition of the patient showed positive dynamics. There was a decrease in peripheral edema, GFR 31.9 ml/min/1.73 m^2. Against this background, the RRT was cancelled. A total of 33 sessions of the filtration-dialysis method of detoxification (hemodialysis, ultrafiltration, hemodiafiltration) were conducted.

In the period of polyuria, which lasted for 1 day, the daily diuresis averaged to 3,127 ± 245.6 ml. During this period, the focus of abscess in the retrostenal hematoma was sanitized.

The patient activation started, the medication sedation was cancelled. The patient is without the neurological deficit. Alert, adequate, he understood the phrases addressed to him and performed simple commands. The discontinuation of the patient from mechanical ventilation went for 2 days. The respiratory gymnastics and exercise therapy were done. The patient can take food unassisted. There were no signs of gastrointestinal hemorrhage. There was an improvement in the radiographic pattern in the lungs, normalization of the clinical and biochemical parameters of the blood.

On 22 May 2016, on day 109 after the surgery, the patient was transferred from ICU to the Cardiac Surgery Ward in a state of moderate severity; in 14 days, in satisfactory condition, he was discharged home.

In total, the patient spent 268 days in the hospital, including 109 days in the ICU.

When analyzing this clinical case retrospectively, one can see that the complications were observed from the first hours of the postoperative period. These were signs of laryngospasm with acute respiratory failure, which developed after extubation of the patient and led to a repeated intubation of the trachea and transfer to mechanical ventilation. Another complication was ARF with uremia and hypervolemia, marked in the patient from day 3 after the surgery, in respond to which 33 sessions of RRT were performed. According to the published data [4], the risk of developing AKI in patients after surgical treatment of CAD is highest with concomitant diabetes and metabolic syndrome, which were present in this patient. On days 9-12, the patient had bleeding from an acute stomach ulcer with severe anemia and a hemorrhagic shock, resulting in a two-minute clinical death. The cardiopulmonary resuscitation, started immediately and conducted with taking into account the type of the cardiac arrest, as well as the adequate management of the early postresuscitation period, allowed restoring blood circulation and avoiding a subsequent neurological deficit.

Thus, a comprehensive intensive therapy, including vasopressor/inotropic hemodynamic support, aggressive respiratory therapy, efferent methods of detox-

ification, resulted in the successful management of all the severe complications, stabilization of the patient's condition, restoration of the functions of organs and systems, and the recovery of the patient.

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CLINICAL CASE OF BRONCHIECTASIS IN THE TEENAGER OF 16 YEARS

ABSTRACT

This article is devoted to the observation during 3 years (2014-2017) of the clinical case of bronchiectasis in a teenager of 16 years. His complaints were shortness of breath during physical exertion, a wet cough with purulent sputum. From an early age he often (about 5 times a year) had bronchitis with an obstructive syndrome. He was hospitalized in the pulmonology department with the diagnosis: community-acquired pneumonia, middle-lobe, moderate severity. According to the results of computed tomography of chest organs, a conclusion was made: bronchiectasis of the middle lobe of the right lung.

Keywords: bronchiectasis, fibrosis, teenager, pneumonia, bronchial asthma.

Bronchiectasis (BE) occurs in about 0.5-1.5% of the population, developing predominantly in childhood and young age (from 5 to 25 yrs). The disease occurs in the form of recurrent bronchopulmonary infections and is accompanied by a constant cough. The lesion of the bronchi with bronchiectasis may be limited to one segment or a lobe of the lung or be widespread (1-2).

Acquired bronchiectasis occurs as a result of frequent respiratory infections, the migrated in childhood - pneumonia, chronic deforming bronchitis, tuberculosis or lung abscess. Sometimes bronchiectasis develops as a result of ingress of foreign bodies in the bronchial lumen.

Chronic inflammation of the bronchial tree causes changes in mucosal and muscular layers of bronchi and in the peribronchial tissue. Becoming malleable, affected walls of the bronchial tubes to dilate. Pneumosclerosis processes in the lung tissue after suffering bronchitis, pneumonia, tuberculosis or lung abscess leads to scarring of the pulmonary parenchyma and dilation, distortion of the bronchial walls. Destructive processes also affect the nerve endings, arterioles and capillaries that feed the bronchi.

Fusiform and cylindrical bronchiectasis affect large and medium-sized bronchi,