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VISCERAL COMPLICATIONS FOLLOWING CARDIAC SURGERY WITH CARDIOPULMONARY BYPASS: RELEVANCE, MECHANISMS, PROGNOSIS AND MANAGEMENT

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ВИСЦЕРАЛЬНЫЕ ОСЛОЖНЕНИЯ ПОСЛЕ КАРДИОХИРУРГИЧЕСКИХ ВМЕШАТЕЛЬСТВ С ИСКУССТВЕННЫМ КРОВООБРАЩЕНИЕМ: АКТУАЛЬНОСТЬ, МЕХАНИЗМЫ, ПРОГНОЗЫ И ЛЕЧЕНИЕ

Введение. Висцеральные осложнения, возникающие после операции на сердце с искусственным кровообращением, занимают лидирующее место в структуре возможных осложнений.

Цель — проанализировать частоту и структуру осложнений после операции на сердце с искусственным кровообращением**.**

Материалы и методы. Были проанализированны результаты 37 исследований (1976–2004 гг.), охватившие около 172 тыс. случаев и результатов лечения 8410 пациентов, которым выполнялись кардиохирургические вмешательства с искусственным кровообращением на базе «НИИ комплексных проблем сердечно-сосудистых заболеваний» СО РАМН в период 2004–2013 гг.

Результаты. Авторами представлен обзор эпидемиологии, риска и прогностических факторов, лечения и профилактики подобных осложнений, методы диагностики, модификации типов искусственного кровообращения, а также фармакологической защиты желудочно-кишечного тракта для данной категории пациентов.

Выводы. Пациенты, которым выполняются кардиохирургические вмешательства с искусственным кровообращением, требуют тщательного контроля из-за возможных абдоминальных осложнений.

Ключевые слова: искусственное кровообращение, абдоминальные осложнения, прогноз, коррекция.

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Introduction. Abdominal catastrophes, occurring after cardiac surgery with cardiopulmonary bypass (CPB), take the leading place in the structure of possible complications.

Aim. To analyze the frequency and structure of complications after cardiac surgery with cardiopulmonary bypass.

Methods. Review of 37 studies between 1976 and 2004, covering over 172,000 cases and total of 8410 consecutive patients who underwent cardiac surgery with CPB at Research Institute for Complex Issues of cardiovascular disease from 2004–2013 were analyzed.

Results. The authors present a review of the epidemiology, risk and prognostic factors, specific management and preventive strategies for such complications. Ultrasonic imaging (contrast-enhanced ultrasound scanning) and radiological (selective mesenteric angiography) diagnostic tools, surface modifications and several types of cardiopulmonary bypass as well as pharmacological protection of the gastrointestinal are considered to be optimal for such category of patients.

Conclusions. Advanced prevention of abdominal complications includes: choice of adequate techniques to conduct cardiopulmonary bypass (pulsatile flow, minimizing surface area of the extracorporeal circuit) and pharmacological methods to protect the gastrointestinal tract; however, the current work does not allow determining the proper time and place to use these preventive strategies.

Key words: extracorporeal circulation, abdominal complications, prognosis, correction.

Abdominal complications have been described since early days of cardiac bypass surgery. They are relatively uncommon, but are always associated with a high mortality and a high risk of multiple organ dysfunction syndrome (MODS) [5]. Recently, the rates of complications and mortality have not significantly decreased, despite the advanced surgical techniques and perfusion technologies. In addition, splanchnic ischemia may play a major role in the initiation of the systemic inflammatory response syndrome (SIRS) after cardiac surgeries with cardiopulmonary bypass (CPB). SIRS, following cardiac bypass surgery, contributes to the development of MODS, causing injuries in distant organs, including brain, lungs and heart and leading to "remote multiorgan failure" or so-called secondary MODS. Early diagnosis and prognosis, as well as intensive treatment of the risk factors appear to be a basis, able to reduce adverse effects. Unfortunately, the clinical risk assessment is ineffective in avoiding abdominal complications due to the low specificity, displayed by the current methods, common use of sedatives in the postoperative period and, moreover, the symptoms of abdominal complications are often similar to severe cardiac and respiratory failure [1].

Background

Current medical literature contains several reports, describing a large cohort of patients, suffering from visceral complications. They were included in the appropriate databases; the derived data were used in the epidemiological analysis and allowed assessing incidence rates of such complications. Thus, in a review of 37 studies between 1976 and 2004, covering over 172,000 cases, the incidence of the gastrointestinal tract has ranged 0.2–5.5 %. The Society of Thoracic Surgeons (STS) reported that the incidence of abdominal complications was 2.8 % in a cohort of 206,143 cases, where 2.5 % occurred after first-time coronary artery bypass graft (CABG), 3.2 % — after reoperative CABG, 2.7 % — after heart valve replacement surgeries and 6.8 % — after combined procedures (heart valve replacement surgery + CABG). According to the STS findings, the gastrointestinal tract complication rate was lower compared to renal failure and significantly lower compared to surgical wound infections (Table 1) [3].

Many studies have attempted to identify risk factors for abdominal complications, but these factors were significantly different. Isolated GI complications were not among the most common risk factors, but in combination with several significant complications

Table 1

Types and incidence of visceral complications, n (%)

Type	Incidence	Mortality
GI bleeding	515 (30.7)	435 (26.9)
Ischemic bowel	297 (17.7)	167 (71.3)
Pancreatitis	188 (11.2)	138 (27.5)
Cholecystitis	182 (10.9)	145 (26.9)
Paralytic ileus	77 (4.5)	37 (10.8)
Perforated peptic ulcer	71 (4.2)	64 (43.8)
Hepatic failure	59 (3.5)	39 (74.4)
Diverticulitis	43 (2.6)	35 (17.1)
Small bowel obstruction	34 (2.0)	27 (18.5)
Pseudo-obstruction of colon	32 (1.9)	14 (21.4)
Other	11 (6.7)	—

such as low cardiac output syndrome, low left ventricular ejection fraction, the need for massive transfusion, acute massive blood loss, etc. Less frequently, the authors describe a truly isolated gastrointestinal tract complications. An attempt to identify pathogenetic factors of GI complications has led to the detection of its main components: depressed systemic hemodynamics (decreased oxygen delivery and low cardiac output), systemic inflammation by violating ratio oxygen delivery and consumption, atheroembolism, splanchnic hypoperfusion [7]. Atheroembolism was detected in 22 % of 221 patients who died after cardiac surgery. It is associated with atherosclerosis of the ascending aorta, the incidence of this phenomenon increases with advanced age and the development of polyvascular disease. The authors also associated high rate of atheroembolism with intra-aortic balloon pumping (IABP). Other authors have described the following structure of abdominal complications: paralytic ileus — 33.3 % of cases, GI bleeding — 27.3 %, gastrointestinal perforation — 6.1 %, calculous cholecystitis — 6.1 %, acalculous cholecystitis — 9.1 %, hepatic dysfunction — 12.1 %, bowel ischemia — 6.1 %. They concluded that the risk factors for abdominal complications in the studied group of patients were the following: age over 75 years, high grade heart failure (III and more), a history of peptic ulcers (for complications such as acute gastrointestinal ulcers), low cardiac output after surgery, prolonged mechanical ventilation over 48 hrs. The review of 18,879 cases allowed to identify reliable risk factors for the group of patients suffering from intestinal ischemia, which may be regarded as independent predictors and included the following: elevated levels of creatinine, hemodynamic instability in the perioperative period, a history of stroke, unstable angina and acute coronary syndrome, cardiogenic shock, anticoagulation treatment, clinically significant heart failure class by NYHA (III–IV), peripheral atherosclerosis. The authors noted the presence of non-specific clinical symptoms and risk factors for abdominal complications. Moreover, they suggested to use the GICS scale (gastro-intestinal complication score) to make a potential prognosis for abdominal complications [1].

Our Own Experience

A total of 8410 consecutive patients who underwent cardiac surgery with CPB at our Institute from 2004–2013 were analyzed. The incidence of abdominal complications was 182 (2.16 %) patients. The operations associated with abdominal complications includ-

ed primary CABG (2.35 %), surgical treatment of acquired heart defects (2.5 %), reoperative CABG (3.9 %), and combined CABG and valve replacement surgery procedures (5.8 %), which is consistent with foreign medical literature [3]. The most common abdominal complications were the following: paralytic ileus — 45.1 % (including half of the cases with pancreatitis) and ischemic bowel — 30.5 %. Firstly, the significant differences with foreign data may be explained by the extensive use of early diagnosis of abdominal compartment syndrome based on the negative dynamics of intra-abdominal pressure (and, therefore, the initiation of early conservative treatment); secondly, the impossibility of performing a complete preoperative evaluation of visceral blood flow (by visualization during laparotomy). Thus, sufficiently low rate of bleedings (10.2 %) and perforated (1.6 %) complications have been found. Mortality rate among patients with abdominal complications was 32 (17.6 %), most of them experienced MODS with the major cause of death was late postoperative sepsis.

Common complications

Upper GI bleeding. GI bleeding is the most common event among all possible abdominal complications. The review of 34 studies covering 140,000 cases was performed. The authors found that a history of peptic ulcer was a risk factor, but concluded that routine prophylaxis with secretion inhibitors and other anti-ulcer drugs can not be recommended as preventive therapy. Over the past decade the incidence of acute gastric ulcers and erosion have greatly reduced among other GI complications due to advanced prevention therapy and effective pharmaceuticals, primarily from the group of proton pump inhibitors.

Pancreatitis. Pancreatitis occurs in 11 % of cases with abdominal complications following cardiac surgery. Hyperamylasemia, one of the classic signs accompanying acute postperfusion pancreatitis, occurs in one third of patients. Thus, there was no correlation between elevated levels of amylase and other pancreatic enzymes with autopsy findings, which makes it impossible to consider elevated levels of amylase as a marker of pancreatogenic abdominal complications. However, such complications rarely develop into pancreatic necrosis, but pancreatitis can maintain postoperative ileus.

Bowel ischemia or infarction. Bowel ischemia is considered to be the most life-threatening abdominal complication. It accounts for 18 % of all visceral complications and is associated with the highest mortality rate (about 70 % of all patients with decompensated mesenteric ischemia). Most of these complications are associated with the development of nonocclusive mesenteric ischemia (NOMI). NOMI is a disorder of splanchnic blood flow, provoked by low cardiac output and worsened by vasopressors and pre-existing atherosclerosis. Visceral hypoperfusion during and after CPB is the leading cause of abdominal complications. Ischemia provokes the damage of the gastric mucosal barrier and triggers MODS. CPB activates systemic inflammation, which is considered to be an additional factor, initiating gastrointestinal ischemia injury. Gastrointestinal hypomotility is provoked by the use of neuromuscular blocking agents, sedatives and analgesics, relatively excess volume infusions as well as the administration of vasoconstrictors to stabilize mean arterial pressure.

Hepatic failure accounts for 4 % of all abdominal complications after cardiac surgery. Risk factors includes female gender, history of congestive heart failure, heart valve surgery, and combined surgeries (heart valve replacement + CABG). Besides, massive transfusion and hematoma may be regarded as nonspecific factors as well as low cardiac output and shock. The authors introduced a new term “severe ischemic early liver inju-

Table 2

Pathogenetic components of visceral ischemia

Mechanism	Disorder	Significance
Decreased mesenteric artery blood flow	Occlusion/stenosis	Important for chronic occlusion ischemia
	Decreased blood flow	Preshock reduces the intensity in blood flow to non-vital organs
	Inotropic and vasopressor agents (e. g. epinephrine, digitalis)	Most patients in the ICU
	Opiates	Premedication and postoperative pain relief
Oxygen capacity and hemoglobin	Anemia and hemic hypoxia	Insignificant
Possibility of cells to utilize oxygen	Sepsis	Bacterial translocation
Bowel blood supply	Depressed hemodynamics	Mucosal perfusion < perfusion of serous membranes, assessment of ischemia only by intraluminal techniques
Exchange of oxygen through the tip of the intestinal villi	Depressed hemodynamics	Hypoperfusion zones are found in the region with normal circulation, ischemic villous with normal crypts
Inadequate metabolism and perfusion	—	Hypoperfusion zones are found in the region with normal circulation, ischemic villous with normal crypts

ry" (SIELI) determined by the elevation of liver enzymes within 48 hrs following surgery, which is associated with right-ventricular failure, leading to increased venous pressure. The pulse-dye indocyanine green clearance test of hepatic dysfunction was proved to have the highest sensitivity and specificity compared to the Child-Pugh classification.

Impairment of splanchnic perfusion. In order to present a complete description of its pattern, a brief description of the visceral perfusion is presented (Table 2).

Splanchnic blood flow plays an important defensive role in hypovolemia and during low cardiac output. In response to the release of catecholamines, sympathetic and renin-angiotensin stimulation, splanchnic vasoconstriction is 25 % of the increase in total peripheral vascular resistance and leads to a return of 15 % of the blood volume. Similar mechanism is observed during extracorporeal circulation. Intestinal and liver autoregulation is weaker in comparison with other vascular beds, but the liver due to its dual blood supply, is better protected from hypoxia and ischemia. Acute impairment of blood flow results in impaired gastrointestinal blood flow, but the timing of the recovery for systemic and splanchnic blood flow are not comparable. Splanchnic hypoperfusion is a common defense mechanism (to compensate shock). However, this mechanism is prolonged and severe, hypoperfusion causes damages of the gastrointestinal tract (bleeding, pancreatitis, acalculous cholecystitis, liver necrosis).

Moreover, the loss of the intestinal barrier may result in the absorption of endotoxins, bacteria and other substances causing SIRS, remote organ injury and MODS. Moreover, splanchnic ischemia/reperfusion may induce SIRS. Intestinal villi are extremely sensitive to ischemia. Arterial inflow enters at its base, hence arterial oxygen partial pressure and content is lower at the tip. Metabolic demand is highest at the tip, making it more sensitive to hypoxia. The data obtained with the introduction of microspheres and performing ultrasound procedure CEUS (see below) confirmed that microcirculatory dysfunction develops away from the central arteriole [2]. Red blood cells can not normally desaturate at the villus tip leading to malnutrition of mucosa tissue despite normal saturation indices and blood oxygen capacity. The differentiative role in damaging the gastrointestinal tract is given to reperfusion: in acute arterial occlusive ischemia, the ischemic and reperfusion processes are consistent, but in NOMI — parallel. The ischemic phase involves acute impairment of ATP recycling, disorder of epithelial cell membrane channels, damage of "dense" contacts between the cells of enterocytes, bacterial translocation. Reperfusion involves an increase in the synthesis of xanthine oxidase, oxidative DNA damage, destruction of membrane integrity, the imbalance between pro-and anti-oxidant systems.

Prognostic methods

In the early 1970s experimental studies focused on the effects of high flow, normothermic, nonpulsatile CPB in primates on organ blood flow measured with radionuclide-labeled microspheres. They found a 50 % decrease in hepatic arterial flow and a 50 % increase of pancreatic flow and a 100–200 % increase in stomach and gastrointestinal flow with initiation of CPB. During 1 hr of CPB hepatic arterial blood flow increased by 100 %, and other flows remained stable [4].

Bastien et al compared the importance of flow preservation vs pressure on gut mucosal and liver flow assessed by LDF in a rabbit model of normothermic CPB. Mucosal flow was 60 % higher in the presence of high pump flow regardless of pressure. Mucosal blood flow did not change under the administration of vasopressors, used to increase systemic blood pressure. Liver flow was also higher by 40 %, regardless of pressure, but in case of vasoconstrictors administration, liver flow decreased, while systemic blood pressure increased (vasoconstrictors administration with low flow decreased liver flow, whereas administration of vasodilators with high flow decreased liver flow). The authors concluded that hepatic flow as well as intestinal mucosal flow has different response and, therefore, prevention of visceral ischemia should focus on perfusion rates, but not systemic pressure. However, visceral flows during perfusion was 35 % of prebypass flow.

In a porcine model of normothermic CPB, Tao et al. found an increase in global visceral blood flow (measured in the superior mesenteric artery and by microspheres), but a reduction of mucosal blood flow. Mesenteric oxygen consumption increased, but oxygen delivery decreased (by reducing hematocrit) and mucosal pH decreased, indicating ischemia. The authors concluded that mucosal ischemia was formed due to the redistribution of blood flow away from the mucosa, reducing oxygen delivery due to anemia and increasing oxygen consumption, but not owing to reduced global splanchnic blood flow.

In a similar model, Tofukuji et al. reported a decrease of ileal mucosal blood flow, a reduction of interstitial pH, an increase of permeability, edema and endothelial dysfunction as manifestations of vasoconstriction and depression of endothelium-dependent re-

laxation. They emphasized that CPB resulted in increased sensitivity of the mesenteric vessels to vasoconstriction and decreased sensitivity of muscle microcirculation; thus, postoperative vasoconstrictor therapy may induce mucosal ischemia.

Attempts to use biochemical markers to predict the development of abdominal complications and possible biochemical monitoring for efficient prevention have been made repeatedly. They focused on the main stage of the complications development, including: impaired visceral vessel patency, microcirculatory disturbances of the intestinal mucosa, intramucosal (interstitial) acidosis, impaired permeability, translocation of endotoxin. Unfortunately, except of relatively promising approach to evaluate intracellular pH of gastric mucosa and duodenum, the other biochemical methods applied in assessing the prognosis reported their incompetency. The study performed by Holmes et al appeared to be promising in this area. The authors measured levels of intestinal FABP in the blood serum of patients after CABG. IFABP are released into the blood during intestinal ischemia. 15 patients with no risk factors for developing abdominal complications did not have elevated levels of IFABP, whereas one-third of patients with one or more risk factors had an increase of the protein concentration.

The recent report dedicated to NOMI mechanisms was provided by Groesdonk et al. who studied 865 patients, included in the registery after on-pump cardiac surgery (patients after CABG prevailed). Patients underwent elective surgery. All patients with suspected NOMI and at least two indications (oliguria over the next 6 hrs after surgery with urine output of 0.5 ml/kg/hour, abdominal hypertension with absent peristaltic sounds, serum lactate levels >5 mmol/l, metabolic acidosis with B. E. levels >5 mmol/l) underwent angiography of the mesenteric arteries. Additionally, angiography was performed in all patients with CI>1.8 l/min/m^2 SI. NOMI assessment scale was proposed (Table 3).

Contrast-Enhanced Ultrasound Scanning (CEUS)

To date, computed tomography and ultrasonography have been regarded as the "gold standard" for diagnosis in patients abdominal catastrophes, including acute visceral ischemia. The sensitivity and specificity of these methods range from 82 to 96 %. Common manifestations seen during the visualization are: presence / absence of arterial / venous obstruction, bowel wall thickening, presence / absence of spastic ileus, presence / absence of hypotension bowel pneumatosis, fluid in the abdominal cavity, parenchymal ischemia (kidney, liver, spleen) [2]. Furthermore, the authors suggested to use the gradation of ischemic phases and microcirculatory disorders, pointing out several stages (1) the early phase (splanchnic ischemia, heterogenous disorder by multi-layer spiral CT, impaired blood supply to the small SMA branches, a mild bowel wall thickening), (2) an intermediate phase: bowel wall thickening, dilated loops of bowel, possible response to the ongoing reperfusion therapy, (3) the late phase: significant improvement in diagnostic capabilities are achieved by the use of CEUS, allowing the visualization of small vessels, which are not defined by the standard ultrasound scanning [6; 8; 9]. It is worth noting that there is no any information about the diagnostic significance of intra-abdominal pressure monitoring (IAPM) and application of abdominal compartment syndrome's criteria in the current medical literature. This occurs despite the fact that intra-abdominal pressure monitoring and scales, containing these criteria, are regarded as the "gold standard" for the diagnosis of abdominal complications in almost all critically ill patients (pancreatitis, trauma, burns). Of course, it is referred to the cardiac surgeries, but

Table 3

NOMI Scale

Criterion	Scores	Definition
Vascular morphology	0 — no	SMA — trunk, branches and arcade within norm
	1 — mild	SMA — trunk within norm, several branches and/or arcades are moderately spastic
	2 — moderate	SMA — trunk is mildly spastic, branches within norm
	3 — severe	SMA — trunk and branches are spastic
Reflux of contrast into the aorta	0 — no 1 — mild 2 — moderate	No reflux of contrast Insignificant reflux Significant reflex on serial angiograms
Parenchymal opacification	0 — total	Homogeneous bowel opacification
	1 — moderate deficit	Inhomogeneous bowel opacification
	3 — severe deficit	Bowel is not contrasted
Bowel distension capacity	0 — no 1 — mild 2 — severe	Less than 3 bowel loops contain air More than 3 bowel loops contain air All bowel loops contain air
Time to contrast portal vein	0 — normal 1 — rather slow 2 — slow	<8 sec 9–12 sec >13 sec
Total scoring	No Mild Moderate Severe	0 1–5 6–7 8–11

Note. SMA — superior mesenteric artery.

not to the surgical procedures, performed on the SMA branches and abdominal aorta, where the prognostic and diagnostic significance of IAPM is beyond doubt.

Splanchnic ischemia and the Systemic Inflammatory Response Syndrome. In addition to standard abdominal complications, splanchnic ischemia may play a significant role in morbidity postcardiac surgery, by activating systemic inflammation and causing remote organ dysfunction. One suggested pathological mechanism involves endotoxin. The development of endotoxemia and bowel ischemia usually occurs early after initiation of CPB. One of the proposed theories suggested ischemia to lead to translocation of LPS into the blood flow. LPS binds to LPS-binding protein (LBP), which stimulates the release of tumor necrosis factor (TNF) from macrophages. Then, neutrophils, lymphocytes, and proinflammatory cytokines are activated. These mediators injure the endothelium, aggravating splanchnic ischemia. A prospective study of 100 elective CABG, Rothenburger et al. suggested high levels of antiendotoxin antibody (AEA) to correlate with prolonged mechanical ventilation over 24 hrs. There are several studies, limiting this hypothesis. Riddington et al. found an increase of AEA levels during CPB, whereas mucosal ischemia occurred a few hours after CPB (with a peak at 12 postoperative). Moreover,

it was suggested that SIRS mediators have been already activated during CPB and mucosal injury and ischemia are the secondary events to active SIRS mediators [10].

Methods to Improve Perfusion and Reduce the Incidence of Complications may be divided into pharmacological (inotropic agents, volume loading, selective intestinal decontamination, glutamine) and modification techniques to conduct CPB (maintaining high flow ± pressure, pulsatile flow, limit administration of vasoconstrictors, minimizing gaseous and atheroemboli, temperature control, surface modification of the extracorporeal circuit, off-pump procedures). Their diversity and disparity need to be discussed in a separate review dedicated to this issue.

Conclusions

1. Despite the fact that the incidence of visceral complications following on-pump cardiac surgery does not prevail among other common complications, it is associated with high mortality rates and, therefore, requires significant expenditures for the treatment of multiple organ dysfunction syndrome.

2. Nonocclusive mesenteric ischemia is difficult to predict, to make prognosis and treat due to significant dysfunction of visceral microcirculation and inability to use reliable and precise diagnostic methods.

3. Early mesenteric angiography (CT angiography) and contrast-enhanced ultrasound scanning may be considered as promising diagnostic tools.

4. Further clinical investigation should be conducted to define the role of intra-abdominal hypertension in the diagnostic and prognostic algorithms for abdominal complications.

5. Biochemical markers of intestinal mucosal injury require careful study and analysis to assess their validity.

6. Advanced prevention of abdominal complications includes: choice of adequate techniques to conduct cardiopulmonary bypass (pulsatile flow, minimizing surface area of the extracorporeal circuit) and pharmacological methods to protect the gastrointestinal tract; however, the current work does not allow determining the proper time and place to use these preventive strategies.

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