Acute Mesenteric Ischemia after Heart Surgery

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Abstract

Introduction: Acute mesenteric ischemia (AMI) is a rare but very severe complication of heart surgery, due especially to the delay in setting the correct diagnosis and choosing the appropriate treatment. There are 4 types, but the most frequent is non-occlusive mesenteric ischemia (NOMI). The main mechanism is represented by great decrease or maldistribution of the splenic blood flow, with negative impact on the integrity of the intestinal mucosa, bacterial translocation and multiorgan failure.

Material and Method: We present a retrospective study conducted on patients who underwent open heart surgery with cardiopulmonary bypass with non-pulsatile flow. 4 cases of angiographically confirmed NOMI (non-occlusive mesenteric ischemia) were identified. When, based on clinical examination and laboratory findings, acute mesenteric ischemia was suspected, superior mesenteric artery angiography was performed via the femoral artery.

Results: The main risk factors were represented by: age over 70 years, left ventricular ejection fraction less than 35%, time of aortic clamp greater than 100 min, chronic renal failure, balloon counterpulsation, use of inotropic drugs, levosimendan, and use of more than 1 unit of red blood cells.

Conclusions: Acute mesenteric ischemia is a rare but very severe complication of heart surgery. It is particularly important to know the main risk factors, and in case of suspicion of the diagnosis, it should be treated as early as possible and with the appropriate treatment.

Cuvinte cheie: ischemie acută mezenterică (IAM), ischemie acută mezenterică non ocluzivă (NOMI), by pass cardio pulmonar (BCP), acidoză metabolică, arteriografie mezenterică superioară, resecție intestinală
70 years old, left ventricle ejection fraction (LVEF) < 35%, aortic clamping time > 100 min., chronic kidney failure, counter-pulsation balloon implant, inotropic medication use, like levosimendan, use of blood components > 1 unit of erythrocyte mass. Clinical signs were nonspecific. All patients presented hypoventilation, arterial hypotension, oliguria and, from a biological standpoint, metabolic acidosis and leucocytosis. Superior mesenteric artery angiography was the investigation method of choice. Treatment approach was initially medical, followed by resection of the intestine. Mortality was 100%.

Conclusions: Acute mesenteric ischemia is a rare but very severe complication in cardiac surgery. It is primordial that the main risk factors be known, and in case of diagnosis suspicion, that it be set as early as possible, along with immediate initiation of an appropriate course of treatment.

Key words: acute mesenteric ischemia (AMI), non-occlusive acute mesenteric ischemia (NOMI), cardiopulmonary bypass (CPB), metabolic acidosis, superior mesenteric angiography, intestinal resection

Introduction

Complications at an abdominal level after cardiac surgery with cardiopulmonary bypass (CPB) are associated with an incidence rate of approximately 0.4 – 2.9% (1,3,5,6,7), acute mesenteric ischemia (AMI) representing 10-67% of cases (1,2,3,5), with a mortality rate of 40-94% determined especially by the delay in setting the correct diagnosis and initiating appropriate treatment measures, given the fact that usually these patients are intubated and under sedation, and AMI is clinically manifested after hours or days (2,8,9).

There are 4 possible causes of AMI:
1. Acute embolism of the superior mesenteric artery;
2. Acute thrombosis on an atheroma at the level of the superior mesenteric artery;
3. Superior mesenteric vein thrombosis (4);
4. Splenic vasoconstriction with decrease in mesenteric flow, known as non-occlusive mesenteric ischemia (NOMI).

NOMI has not been completely understood from a physiopathological point of view, but its main mechanism is represented by an important decrease in or dysfunctional distribution of the splenic blood flow (2,3,10,11). Mesenteric ischemia affects the integrity of the intestinal lining, with bacterial translocation followed by multigangic failure. The small bowel is the splenic territory most inclined to ischemic injury, as the anatomical arrangement of intestinal microcirculation is comprised of 3 parallel systems, which distribute blood equally between the mucosa, submucosa, and muscle layer of the artery, and an intestinal villus is vascularized by an unbranched arteriole which divides directly into capillaries, and by a centrally located venule. (14)

Intestinal villi are susceptible to ischemia, blood flow reaching a minimum at their summits. On the other hand, splenic circulation plays a defensive role against hypovolemia and low cardiac output periods, and as a response to catecholamines, the sympathetic system and the renin-angiotensin system, splenic vasoconstriction occurs, with an increase of approximately 25% in peripheral vascular resistance and of an "auto-transfusion" phenomenon which leads to an average 15% increase in blood volume. When low cardiac output syndrome occurs, the splenic blood flow decreases suddenly, and as the systemic blood flow is restored, the splenic flow recovers its pace slowly and gradually. Splenic hypoperfusion is a normal defence mechanism, and is usually well-tolerated; however, when hypoperfusion is severe and long-lasting, mesenteric ischemia occurs, with loss of the intestinal barrier, absorption of endotoxins and bacteriaemia, triggering of a systemic inflammatory response and multigangic dysfunction (cardiac, pulmonary, renal, cerebral) (3,12-17).

During cardiopulmonary bypass (CPB) similar events can take place, with increased severity in elderly patients.

Splenic blood flow during CPB can be improved by re-establishing volemia, use of phosphodiesterase inhibitors, dopamine, dobutamine. (13,18,19)

Material and Method

We present a retrospective study conducted in the Cardiovascular Surgery Clinic of the “Prof. Dr. Agrippa Ionescu” Clinical Emergency Hospital between 01.01.2010 – 20.12.2013, on 1253 patients with acute mesenteric ischemia (AMI) occurring after having undergone heart surgery with CPB. Patients submitted to off-pump myocardial revascularization were excluded from the study. Among the cardiac surgeries with CPB with non-pulsatile flow, 4 cases of angiographically confirmed NOMI were identified. The study was conducted with the approval of the hospital’s Ethics Committee.

Clinical signs in AMI are non-specific: oliguria, arterial hypotension, respiratory failure. During clinical examination signs of peritoneal irritation may or may not be present: pain (usually diminished by analgesics and sedatives), nausea, vomiting, diarrhoea, abdominal distension (3,15). In case of a vigilant patient, capable of sustaining spontaneous breathing, there are some signs of predictive value: fever, arterial hypotension after previous hemodynamic stability, respiratory or renal failure. (3,19,20)

From a paraclinical standpoint, there is no specific laboratory test, but the most commonly encountered findings are metabolic acidosis and lactate increase, increasing anionic gap, homoconcentration, leucocytosis, amylase increase, creatine kinase (CK) increase.

Paraclinical investigations have various degrees of importance; thusly: abdominal X-ray provides little information, being able to highlight dilated, thickened bowel loops, ground glass appearance (suggesting ascites), thumb printing (indicating submucosal and haemorrhagic oedema). Echo-Doppler, abdominal CT scan, early colonoscopy and peritoneal lavage have limited implications, but can support a
diagnosis. Computer angiotomography (angioCT) is a valuable investigation in sustaining the diagnosis, being able to visualize mesenteric vascularization. (3,16,20)

Superior mesenteric angiography is the most valuable investigation method, providing anatomical data and representing at the same time a therapeutic option, offering the possibility of injecting vasodilating or fibrinolytic medication through the catheter. (2,3,21,22).

Results

Between 01.01.2010 – 20.12.2013 1253 open heart surgeries with non-pulsatile CPB were performed. All four patients presenting NOMI were of male gender, 3 of them being over 70 years old, and all having a preoperative left ventricle ejection fraction (LVEF) under 35%, being under sinus rhythm. In terms of type of surgery, 3 were submitted to coronary artery bypass grafting (CABG), and one to aortic valve prosthesis implant (AVP), mitral valve prosthesis implant (MiVP) and CABG. All 4 patients had a preoperative EUROSCORE over 6. No patient had the extracorporeal circulation time (ECC) longer than 240, and only one patient had an aortic clamping time over 100 minutes, 1 patient presented preoperative chronic renal failure (CRF), 2 patients required postoperative counter-pulsation balloon implant (IABP) which were correctly positioned (verified radiographically). Pre- and intraoperative use of levosimendan (positive inotropic agent) was required in 3 patients. Although cell saver was used intraoperatively in all 4 patients, more than 1 unit of erythrocyte mass needed to be administered. During cardiopulmonary bypass (CPB) all 4 patients presented periods with haematocrir (Ht) bellow 24% or periods of arterial hypotension requiring administration of noradrenaline.

Postoperatively the 4 patients were taken off intubation after the first 24 hours, but during the first postoperative day they all presented with non-painful abdominal distention, absence of bowel movement and bowel sounds. Although immediately postoperatively all 4 patients were haemodynamically stable, arterial hypotension occurred, requiring adrenaline administering in all 4 cases. Hypoventilation requiring reintubation with mechanical ventilation occurred, all 4 patients developing a postoperative fever, with oliguria and need of diuretic medication. From a biological point of view, on the second postoperative day, all patients presented metabolic acidosis with serum lactate over 5 mmol/l, excess of bases (BE) over 5 mmol/l, high creatine kinase (CK), leucocytosis. As there was suspicion of AMI from the first postoperative day, abdominal X-ray showing dilated intestinal loops was performed in these patients. The abdominal CT scan indicated thickening of the small bowel’s wall.

Superior mesenteric artery angiography was performed in these patients on the third postoperative day and revealed specific findings: moderate-severe constriction of the trunk, branches and arcs of the superior mesenteric artery, moderate-severe reflux of the contrast agent in the aorta, with an angiographic sum up score of 9-10 (severe mesenteric insufficiency). (24).

Seeing how all the AMI forms were NOMI, immediately after the diagnosis was made, medical treatment was initiated, meaning: volemic rebalancing, correction of the metabolic imbalance, antibiotic therapy, heparin treatment, mechanical ventilation, diuretic treatment, vasodilators (pentoxifylline), antispasmodics. No arterial flow reestablishment through fibrinolytic treatment was required at the level of the superior mesenteric artery. A catheter was placed in the superior mesenteric artery, arteriographically introduced, under papaverine 50 mg/h (2,3,20). Unfortunately the evolution of these patients was towards deterioration, with multiorganic failure (pulmonary, cardiac, renal, cerebral ± hepatic). A patient deceased on the 4th postoperative day, without there being time to perform a laparotomy. In the remaining 3 patients irreversible small bowel gangrene was observed, followed by intestinal resection. The catheter was maintained intra- and postoperatively for intra-mesenteric administration of papaverine. The evolution of these 3 patients was gradually deteriorating, with exits in under 10 days.

Discussions

AMI is a rare but very severe complication of cardiac surgery, with a statistical frequency of approximately 1%. (1,3,5,6,7)

In our records, the frequency of this complication was far lower (0.31%), while mortality was however 100% compared to 67-100% reported by other authors. (2,5-9,12,13)

The physiopathological mechanism is not yet fully understood, but it is believed to be represented by an important decrease or abnormal distribution of the splenic blood flow (2,3,10,11,23). Also, the clinical and biological signs are non-specific. The main risk factors for occurrence of NOMI are considered to be: age over 70 years old, counter-pulsation balloon (IABP) use, haemodialysis, surgical reexploration due to haemorrhage, postoperative need for at least 1 unit of erythrocyte mass (EM), postoperative serum lactate over 5 mmol/l, postoperative use of levosimendan, noradrenaline, postoperative loss of sinus rhythm (2,24). All these factors were revealed by our paper to be favouring factors. (7,9,12,15,18,24)

During cardiopulmonary bypass (CPB) the decrease in blood flow is most poignantly felt at the level of the jejunum and ileum, with the risk of NOMI occurrence. An alteration of the intestinal barrier develops, intensified by the administering of vasopressor medication (noradrenaline) (2,25,29). Levosimendan has a vasodilator effect, but requires dosage supplementation with α stimulant medication (noradrenaline), with worsening of the intestinal ischemia (30). In patients with low cardiac output, the use of an IABP is common, which may lead to mesenteric ischemia even when the balloon is in a radiologically supervised position.

Mesenteric ischemia triggers a systemic inflammatory response with the possibility of modifying the coagulation system in the direction of either hypo- or hypercoagulation. (29)

The methods for improving splenic blood flow and, implicitly, reducing AMI incidence are presented in Table 1.
According to E. Cunneyt (31), a series of elements pertaining to CPB which could diminish AMI incidence – according to E. Cunneyt, are presented in Table 2 (31).

There are however authors who disagree with the fact that heart surgery without CPB or with pulsatile CPB diminishes the frequency rate of AMI occurrence. (11,18,31)

Superior mesenteric angiography was the most valuable paraclinical investigation, the characteristics of NOMI being: thickening and irregularities of the superior mesenteric artery, contrast agent reflux in the aorta (signs of venous occlusion). Klotz recommends 4 elements for performing mesenteric angiography: absence of bowel movements for more than 3 days from the cardiac surgery, severe abdominal distension, clinical and radiological signs of dynamic ileus and increased level of serum lactate. Mesenteric arteriography via the femoral artery was decided for, with substitution when at least 2 of the four clinical and paraclinical elements were present: oligo-anuria, abdominal distension of lack of bowel sounds, serum lactate over 5 mmol/l, metabolic acidosis with base excess (BE) lower than 5 mmol/l (2,3,20)

When, based on the above mentioned risk factors (although clinical and paraclinical signs are unspecific), a suspicion of AMI is raised, medical and surgical treatment must be initiated as early as possible, individually, taking into consideration the four types of AMI. In patients in which mesenteric angiography reveals arterial thrombosis with no peritoneal signs, thrombolysis with recombined plasminogen activator (ACTILYSE) – 5 mg in bolus, followed by 1 mg/h can be attempted. The arteriography can be reperformed after 24 hours. (24,31).

In case of NOMI, primary treatment is the medical one presented above, which is instated simultaneously with mesenteric angiography. A catheter is fixed in the superior mesenteric artery, with local administration of papaverine 30-60 mg/h, 24-48 hours (27). When peritoneal signs appear, laparoscopy or laparotomy with evaluation of intestinal villi viability is necessary. The surgical treatment standard is represented by: resection of nonviable bowel, systemic reperfusion with warm peritoneal lavage and repeated perfusion with warm lactated Ringer solution. For patients in whom mesenteric angiography reveals arterial ischemia, revascularisation is indicated: mesenteric angiography, via the femoral artery, followed by hemodialysis (24,31,30).

Although the number of detected cases was small, only 4, the mortality rate was total (100%), so it may be considered that preoperative detection of the main risk factors for AMI can help efficiently prevent the development of this very severe complication.

In the Romanian medical literature there are authors such as Văsilie I., Medina C. et al. (2,3) who present their own experiences with AMI and one case of successfully operated NOMI, who survived. Similarity factors with our paper are represented by symptomatology, clinical signs, paraclinical investigations, treatment applied, as well as some etiologic factors: age, arterial hypertension, arterial atheromatosis, but the essential difference resides in the fact that NOMI occurrence after cardiac surgery with CPB presents essential risk factors: significant haemodilution, use of alpha adrenergic medication, of an IABP, of levosimendan, which determined an unfavourable evolution and a high mortality rate.

### Conclusion
AMI is a rare but extremely severe complication of cardiac surgery, especially when the diagnosis and treatment are delayed. Immediately after setting the diagnosis, superior mesenteric artery angiography must be performed, followed closely by specific treatment of each aetiological form. An extremely important element is identification of preoperative risk factors, thus being able to influence greatly the incidence of this complication and reduce it.

### References

### Table 1. Possible methods to improve splanchnic blood flow and reduce the incidence of abdominal complications after cardiac surgery

<table>
<thead>
<tr>
<th>Recommended Method</th>
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<tbody>
<tr>
<td>Phosphodiesterase (PDE) III Inhibitors</td>
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<td>Inotropic agents</td>
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<tr>
<td>Volume loading</td>
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<tr>
<td>Selective digestive decontamination</td>
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<tr>
<td>Prophylactic intra-aortic balloon pumping (IABP)</td>
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<td>Modify conduct of cardiopulmonary bypass (CPB)</td>
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### Table 2. Modification of conduct of cardiopulmonary bypass that may reduce visceral ischemia and abdominal complications

<table>
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<tr>
<th>Recommended Method</th>
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<tbody>
<tr>
<td>Maintain high flow ± pressure</td>
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<tr>
<td>Pulsatile flow</td>
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<tr>
<td>Limit administration of vasoconstrictors</td>
</tr>
<tr>
<td>Administration of inotropes and vasodilators</td>
</tr>
<tr>
<td>Minimizing gaseous micro-emboli (GME) and atheroemboli: Embol-X, Distal aortic cannula</td>
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<tr>
<td>Avoid severe anaemia</td>
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<tr>
<td>Temperature management</td>
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<tr>
<td>Surface modification of the extracorporeal circuit (ECC)</td>
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<tr>
<td>Minimize surface area and prime volume of ECG</td>
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<tr>
<td>Anti-inflammatory strategies: Aprotinin, Leukodepletion, Hemofiltration, Steroids</td>
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<tr>
<td>Anti-thrombotic therapy: Aspirin</td>
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<tr>
<td>Perform procedure off-pump (OPCAB)</td>
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