



Acute mesenteric ischemia after cardio-pulmonary bypass surgery

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Abstract

Acute mesenteric ischemia (AMI) is a highly-lethal surgical emergency. Several pathophysiologic events (arterial obstruction, venous thrombosis and diffuse vasospasm) lead to a sudden decrease in mesenteric blood flow. Ischemia/reperfusion syndrome of the intestine is responsible for systemic abnormalities, leading to multi-organ failure and death. Early diagnosis is difficult because the clinical presentation is subtle, and the biological and radiological diagnostic tools lack sensitivity and specificity. Therapeutic options vary from conservative resuscitation, medical treatment, endovascular techniques and surgical resection and revascularization. A high index of suspicion is required for diagnosis, and prompt treatment is the only hope of reducing the mortality rate. Studies are in progress to provide more accurate diagnostic tools for early diagnosis. AMI can complicate the post-operative course of patients following cardio-pulmonary bypass (CPB). Several factors contribute to the systemic hypo-perfusion state, which is the most frequent pathophysiologic event. In this particular setting, the clinical presentation of AMI can be misleading, while the laboratory and radiological diagnostic tests often produce inconclusive results. The management strategies are controversial, but early treatment is critical for saving lives. Based on the experience of our team, we consider prompt exploratory laparotomy, irrespective of the results of the diagnostic tests, is

the only way to provide objective assessment and adequate treatment, leading to dramatic reduction in the mortality rate.

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Key words: Acute mesenteric ischemia; Non-occlusive; Cardio-pulmonary bypass; Laparotomy; Prognosis; Mortality

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INTRODUCTION

Acute mesenteric ischemia (AMI) is a life-threatening surgical emergency in which the outcome is closely dependent on the elapsed time to diagnosis and treatment. The diagnosis is typically difficult and delayed due to non-specific results of biological and radiological tests. Since prompt treatment is the key to a better outcome, AMI remains a challenging condition because of controversial algorithms and numerous therapeutic options.

When AMI occurs after a cardio-pulmonary bypass (CPB) procedure, the condition has a more subtle clinical presentation, is more difficult to diagnose and treat, leading to a higher mortality rate.

This report is an updated review of AMI with respect to the pathophysiologic events, diagnostic tests, therapeutic options, mortality rate and promising new areas of research. A separate section is dedicated to AMI in CPB patients and focuses on the main differences in the diagnosis, management and outcome.

AMI IN STANDARD CONTEXT

Definitions

AMI is caused by a sudden decrease in the blood flow to the bowel and abdominal viscera. Important features of AMI include: bacterial translocation, systemic

inflammatory response syndrome and reperfusion injury, which exacerbate the ischemic damage of the intestinal microcirculation and negatively impact the outcome. Although rare, the incidence of AMI is increasing, in parallel with the aging population^[1].

Pathophysiology

AMI is the result of four distinct pathophysiologic mechanisms: arterial embolus, arterial thrombosis, splanchnic vasoconstriction, known as non-occlusive mesenteric ischemia (NOMI) and venous thrombosis.

Arterial embolus is the most common cause, responsible for almost half of all cases^[2,3]. The source of the embolus is usually the heart, and the affected vessel is the superior mesenteric artery in 50% of cases. In general, the obstruction occurs at the mid to distal bifurcation points of the blood vessel^[1,4].

Arterial thrombus is the underlying cause in approximately 30% of patients^[2,3], with rupture of an atherosclerotic plaque in the mesenteric arteries. The site of the occlusion tends to occur at the origin of the blood vessels. The patients can tolerate major visceral artery obstruction because of the slow progressive nature of atherosclerosis, with the development of collaterals. Nearly 75% of patients have pre-existing chronic mesenteric ischemia^[5], and acute bowel ischemia or infarction only ensues if the last remaining visceral artery or an important collateral artery occludes. The extent of bowel ischemia or infarction is typically greater than that seen with embolism.

In NOMI, diffuse vasospasm of the mesenteric and other visceral arteries occurs as a result of a sustained hypoperfusion state^[6,7]. No vascular occlusion is usually demonstrated because pulsatile blood flow is present in larger arteries^[8]. There are several predisposing factors, which are often interrelated, such as heart failure, arterial hypotension, elevated sympathetic activity, hypovolemia, sepsis, use of vasopressors and pre-existing atherosclerotic lesions^[7]. Catecholamines and medications such as digitalis^[9], by interfering with the auto-regulation of mesenteric circulation, can also cause vasospasm^[10].

Mesenteric venous thrombosis accounts for approximately 10% of all AMI cases^[2] and involves the superior mesenteric vein in over 90% of patients. Mesenteric venous thrombosis is usually secondary to an underlying coagulopathy, while in 10% the cause is idiopathic^[11-15]. Patients should be screened for genetic thrombophilias. Compromised venous return leads to interstitial swelling in the bowel wall, with subsequent arterial flow disturbances and eventual necrosis. The etiologic factors responsible for venous thrombosis include portal hypertension, intra-abdominal sepsis, cirrhosis, pancreatitis, malignancy and trauma.

Other rare causes of mesenteric ischemia are aortic dissection, lupus, vasculitis, median ligament syndrome, ergot administration and post laparoscopic cholecystectomy^[16]. In young patients, arterial occlusion due to inherited coagulopathy is exceedingly rare and only isolated cases have been reported^[17].

The clinical features of AMI originate from factors such as the site of involvement, systemic inflammatory response triggered by damage to the microcirculation, and reperfusion injury.

At the cellular level, ischemia causes mitochondrial dysfunction, loss of ion transfer regulation, and intracellular acidosis. Alterations in membrane permeability, and the release of free radicals and degradative enzymes leads to cell death and tissue necrosis^[18]. In the ischemic tissue, numerous cells including neutrophils, endothelium, monocytes and platelets are activated. Proinflammatory substances are produced such as tumor necrosis factor, interleukins, platelet-activating factor and leukotrienes. Subsequently, the injury is due to leukocyte adhesion, platelet aggregation^[19] and nitric oxide production impairment^[20]. The activated neutrophils release superoxide substances such as superoxide O_2^- , peroxide H_2O_2 and hydroxyl radicals OH^{\cdot} ^[21], along with neutrophil enzymes, which result in further damage to the surrounding tissues.

Ischemic/reperfusion double-hit injury consists of an initial hypoxic episode followed by the subsequent reperfusion injury due to reestablishment of forward flow^[10]. Superoxide molecules, neutrophil enzymes and pro-inflammatory substances are carried in the bloodstream, causing distant organ damage. Moreover, reperfusion causes swelling of the corresponding organs since capillary permeability is considerably increased during ischemia^[22]. Finally, damage to the intestinal micro-vessels and the disruption of the intestinal mucosal barrier results in leakage of water and bacteria, with resulting endotoxemia^[23] and bacteremia^[24,25]. Ultimately, multi-organ failure ensues and involves the liver^[18], heart^[26], kidneys^[27] and lungs^[28]. Acute pulmonary edema resulting from mesenteric ischemia/reperfusion is caused by an increase in pulmonary microvascular permeability to fluids and proteins, as well as smooth muscle dysfunction^[29,30].

Clinical presentation

Abdominal pain is the primary symptom. The pain is characteristically out of proportion to the clinical findings. It is described as colicky and is most severe in the periumbilical region. Other symptoms are present inconsistently and include nausea (93%), vomiting (80%) and diarrhea (48%)^[31]. Physical examination is unremarkable unless peritonitis has developed. During the late stages, abdominal distension and guarding, as well as systemic complications may be encountered.

Laboratory tests

Soon after onset but prior to the development of mesenteric infarction, the sensitivity of laboratory tests in detecting mesenteric ischemia is poor^[32]. Even at the time when ischemia is confirmed at laparotomy, elevation of serum lactate, amylase, creatine kinase and C-reactive protein (CRP), as well as leucocytes may be absent^[31]. At present, no laboratory test is available for accurately establishing or eliminating the diagnosis^[33,34]. One study

reported that hemoconcentration and hyperamylasemia were independent predictive factors of massive ischemic infarction^[35].

Imaging studies

It is important to remember that when intestinal ischemia is clinically suspected, diagnostic imaging studies should be performed if peritoneal signs are absent.

Plain abdominal radiographs are of little help in the diagnosis of mesenteric ischemia. The presence of dilated loops is non-specific^[36,37], and thickened bowel loops, “ground-glass” appearance suggesting ascites, or “thumbprinting” caused by submucosal edema or hemorrhage are seen in less than 40% patients. Twenty-five percent patients with bowel infarction have negative plain radiographs of the abdomen^[38].

Barium enema has no place in the diagnosis of AMI since it may increase intra-luminal pressure and reduce perfusion to the bowel wall, causing translocation of bacteria and potentially, perforation. In addition, the presence of barium may compromise subsequent diagnostic tests, such as computed tomography (CT) and angiography^[32].

Magnetic resonance imaging has shown promising results in detecting mesenteric ischemia but remains a slow-processing technique that seems to be inadequate in an emergent situation such as AMI^[39,40].

Mesenteric duplex sonography is a highly user-dependent modality that can only confirm diminished blood flow in the trunks of the mesenteric blood vessels. Mesenteric duplex scanning identifies stenosis of the superior mesenteric and celiac arteries by the mean of elevated peak systolic velocities. A velocity > 275 cm/s is indicative of >70% stenosis with a sensitivity of 92% and a negative predictive value of 99%^[41]. Doppler sonography is useful in diagnosing chronic mesenteric arterial occlusive disease but has limited role in AMI^[42-45]. Other applications for duplex sonography are detection of reversible celiac flux alteration such as in median ligament syndrome, and follow-up of mesenteric bypass grafts and stents^[41]. The new technique of contrast-enhanced ultrasonography is a promisingly non-invasive tool for the diagnosis of bowel ischemia^[46].

Angiography is the gold standard diagnostic test in acute mesenteric artery occlusion^[47], providing both anatomical visualization of the vessels and therapeutic options^[48]. The sensitivity and specificity are 74% to 100% and 100%, respectively^[49]. When used in the absence of peritonitis signs, angiography has been shown to improve the survival rate^[50,51]. Mesenteric angiography can usually identify the underlying pathophysiologic event, by differentiating between embolic and thrombotic occlusion^[52]. NOMI characteristically shows narrowing and multiple irregularities of the major SMA tributaries recognized as the “string of sausages” sign. Mesenteric venous thrombosis is characterized by a generalized slowing of arterial flow (up to 20 s) in conjunction with a lack of opacification of the corresponding mesenteric or portal venous outflow tracts. However, angiography is

an invasive, time consuming and potentially nephrotoxic procedure. Its routine use is controversial in emergency situations^[53] and therefore, it is employed only in selected patients.

Since CT is a fast, widely available non-invasive modality, it is considered as the initial imaging test^[54]. It is useful in detecting intestinal signs suggestive of ischemia, as well as vascular abnormalities such as occlusion and stenosis. It is also useful in assessing other causes of acute abdominal pain. Still, the CT findings of mesenteric ischemia and infarction are not pathognomonic, and a direct correlation between CT findings and the final diagnosis is not accurate^[55]. Overall, the sensitivity and specificity of contrast-enhanced CT for mesenteric ischemia are 64% and 92%, respectively^[31,56,57]. Because of these drawbacks, the American Gastrointestinal Association^[49] concluded that CT is of limited use in the diagnosis of AMI and that unremarkable CT findings in the context of a high suspicion of mesenteric ischemia should prompt an angiography without delay. An exception to this rule is when superior mesenteric vein thrombosis is suspected; a situation where CT scan remains the test of choice with sensitivity rate in the range of 90%^[14,50].

Recently, the multi-detector row CT has emerged as a widely established non-invasive technique that not only delineates the blood vessels, but also shows an anatomical three dimensional relationship with the surrounding tissues, and allows evaluation of tissue perfusion^[58]. The sensitivity and specificity rates are 92%-96% and 94%-100%, respectively^[31,59,60], with positive and negative predictive values of 90% and 98%, respectively^[61]. Moreover, when a cardiac source of mesenteric emboli is suspected, scanning of the heart provides a method for concomitant detection of the source of the embolus^[62].

Therapeutic approaches

Therapeutic decisions are taken based on four main considerations: the presence or absence of peritonitis, the presence or absence of irreversible ischemia or infarcted segments of the intestine, the general condition of the patient, and the pathophysiologic phenomenon responsible for the event.

Once a diagnosis of AMI is made, treatment should be initiated without delay. This should include active resuscitation and treatment of the underlying condition, along with efforts directed toward reducing the associated vasospasm. Broad-spectrum antibiotics and intravenous heparin at therapeutic doses should be initiated as early as possible. If the diagnosis was established through angiography, intra-arterial infusion of papaverine, a phosphodiesterase inhibitor, is recommended for NOMI and for occlusive arterial AMI, since arterial vasospasm persists even after successful treatment of the precipitating event^[1,3]. When angiography is not performed, intravenous glucagon may help reduce the vasospasm^[52].

In the setting of a hemodynamically stable patient, with no signs of peritonitis, conservative medical

management may be attempted. For embolus- and thrombus-induced events, thrombolytic agents such as streptokinase, urokinase or recombinant tissue plasminogen activator are effective treatments^[63-66]. Thrombolytic therapy seems to be most successful in distal clots, when used within 12 h after the onset of symptoms^[66]. Ultimately, primary endovascular techniques and surgical resection may prevent mesenteric infarction when performed promptly in hemodynamically stable patients with arterial mesenteric ischemia^[67]. For NOMI, especially when diagnosed by angiography, selective intra-arterial infusion of papaverine at the usual dose of 30 to 60 mg/h, is an adequate treatment^[1,52,53]. It reduces the mortality rate from 70% to 50%-55%^[68]. Early treatment with continuous intravenous high dose prostaglandin E(1)^[69] or a prostacyclin analogue^[70] have shown promising results in the treatment of NOMI. As for venous mesenteric ischemia, the standard treatment is anticoagulation, while venous thrombectomy has not improved the outcome and is controversial^[71,72]. Heparin should be initiated as soon as the diagnosis is established, and is associated with reduction in the recurrence rate and mortality^[13,14]. Another appropriate therapeutic modality is thrombolysis^[73-75].

At any time during evaluation, should signs of peritonitis develop, the patient should undergo exploratory laparotomy without delay. First, the intestine is assessed for viability. Visual evaluation of the bowel relies on arterial pulsations and intestinal peristalsis and colour, although these findings are not specific^[52]. Another technique is the use of sodium fluorescein, which is injected intravenously; it is detected with a Wood's lamp in the presence of hypoxic damage. Both retrospective analysis and randomized trials have shown that this technique is more reliable than clinical evaluation of mesenteric viability^[76-78]. When compared with histological results, intraoperative laser Doppler flowmetry has been shown to be 100% accurate in assessing bowel viability^[79]. Doppler ultrasound can be used intraoperatively but does not provide a quick assessment of the entire length of the intestine and thus does not carry any advantage over clinical judgment^[80].

In patients with arterial occlusive AMI, when sufficient bowel is potentially viable, revascularization prior to resection of the infarcted bowel may improve the survival^[2]. Although surgical revascularization is the standard procedure^[81], embolectomy, thrombectomy, endarterectomy^[38], as well as endovascular techniques such as antegrade percutaneous stenting^[82], and open retrograde stenting^[83-85] provide attractive alternatives with good short-term outcome. A high stent restenosis rate is the drawback of these techniques, requiring close follow-up of the patients^[85,86]. Contraindications to revascularization include obvious infarction of the bowel supplied by the affected artery, patient's instability precluding further resection, and mesenteric vein thrombosis^[53,87].

Surgical bowel resection must include all of the clearly non viable and infarcted portions of the bowel. Primary anastomosis can be performed if perfusion is

adequate. A second-look laparotomy is scheduled within 12 to 24 h, if large portions or multiple segments of intestine of questionable viability were left behind^[88], provided that complete resection should not result in a short bowel syndrome. Although widely approved^[1,3], some authors question its routine use, and limit second-look laparotomy to individual cases^[89].

Alternatively, second-look laparoscopy has emerged as a minimally-invasive, technically simple procedure that can provide diagnostic and therapeutic advantages^[90] despite the fact that the evaluation is limited to the serosa and that mucosal lesions can be missed^[91]. As a result, the value of second-look laparotomy in preventing morbidity is uncertain^[90,92-94].

Outcome

Despite advances in the identification of mortality risk factors and greater therapeutic options, the overall mortality associated with AMI is as high today as it was several decades ago^[31,95], ranging from 60% to 90%^[49,96-99]. When specific etiologies are considered separately, arterial thrombosis has the highest mortality rate of 70% to 100%^[3,98,100] in part because of the extensive ischemia-infarction of the bowel, and the need for more complex surgical revascularization. The mortality associated with NOMI is also within this range^[98], whereas arterial embolism and venous thrombosis have much better prognosis, with mortality rates of 0% to 50%^[15,98,101] and 20%^[5], respectively. The peri-operative factors predicting mortality after mesenteric ischemia have been extensively studied^[7,99]. Of the various factors examined, age > 70 (where diagnosis is more frequently overlooked), and prolonged duration of symptoms were independent predictors of mortality^[97,102-104]. It cannot be overemphasized that a high index of suspicion, prompt diagnosis and aggressive early treatment are the only surgeon-dependent factors that have a positive influence on the outcome.

Perspectives for the future

A number of biochemical and genetic studies are in progress, designed to elucidate the pathophysiologic changes in an ischemic/reperfused intestine. Several molecules have been identified which attenuate intestinal injury and reduce the production of proinflammatory cytokines^[105-110]. Intra-luminal infusion of hyperoxygenated solution during ischemia may improve the functional and structural status of the enterocyte mitochondria associated with ischemia/reperfusion syndrome^[111]. Most therapies have to be initiated prior to the onset of ischemia, making their clinical application difficult to foresee.

Since the prognosis is closely related to delay in the treatment, and since the diagnostic tools currently available are not very accurate, great effort is being focused on identifying more accurate methods of early diagnosis. Serum assays such as D-dimers^[112-114], alcohol dehydrogenase^[115], glutathione S-transferase^[116] and cobalt-albumin binding^[117], and measurement of pH and potassium in peritoneal irrigation fluid^[118], as well

as liver tissue oxygenation index^[119] are good examples of current research. Seidel *et al* showed that mesenteric electrical activity may detect mesenteric ischemia with a high degree of sensitivity and specificity^[120]. However, promising studies in animals remain to be validated under clinical conditions.

Experimental studies in which laparoscopy was combined with ultraviolet light and IV injection of fluorescein showed that this technique may be useful in detecting mesenteric ischemia and viability of the intestine at an early stage^[121,122]. Moreover, trans-serosal pulse oximetry may help determine bowel viability and resection extension prior to laparotomy^[123].

AMI AFTER CPB

Incidence and frequency

Abdominal complications after CPB for cardiac surgery are seen in < 1% of patients^[124-132] but carry a high mortality of 14.1%^[131]. AMI represents 10%-67% of these complications^[129,133,134], and is the most lethal, with a case-fatality rate of 70% to 100%^[129,131,132,135,136].

Pathophysiology

AMI occurring after CPB, is due to NOMI in the vast majority of cases^[137-139]. The various contributory factors are: low cardiac output (frequent in this category of patients), use of vasopressors and underlying atherosclerotic disease. It is well established that CPB is responsible for mesenteric endothelial dysfunction and microcirculation disturbances even under stable hemodynamic conditions. An increase in the contractile response to alpha1-adrenergic agonist and an early release of pro-inflammatory substances has been observed after CPB^[140-142]. Nevertheless, the effect of pulseless extracorporeal circulation on bowel hypoperfusion is still under debate^[143,144], and off-pump coronary artery bypass does not prevent subsequent mesenteric ischemia^[134,145,146]. The physiologic changes in intestinal perfusion during cardiac surgery remain to be elucidated. It has been observed that there is significant mesenteric hypoperfusion followed by hyperemic response^[147] after off-pump cardiac surgery along with an increase in the resistive and pulsatility indexes^[148]. Moreover, studies focusing on the identification of predisposing factors for mesenteric ischemia after CPB^[135,124-126], have produced different, and even opposite results. Rare cases of embolic acute arterial infarction can be prevented when a calcified aorta is detected on pre-operative CT scan^[149].

Clinical presentation

In the context of CPB, patients are usually sedated and mechanically ventilated for a few days. Consequently, symptoms are not reported and the physical examination is equivocal due to masked, late-appearing, or missing clinical signs^[125,139,150]. This accounts for the delay in diagnosis, and the disease may progress to a late, even irreversible stage by the time clinical signs become obvious (i.e. cyanosis). Finally, extracorporeal

circulation induces a systemic inflammatory response with vasodilatation, such that hemodynamic instability can no longer be interpreted as a clue to an underlying mesenteric ischemia. A high index of suspicion in detecting subtle clinical evidence of AMI is the key to reducing the delay in diagnosis.

Laboratory tests

Pulseless perfusion during extracorporeal circulation causes systemic hypoperfusion, as illustrated by major derangements in the biochemical tests. These abnormalities are difficult to distinguish from those related to an underlying AMI. Moreover, laboratory test abnormalities are observed inconstantly in AMI. Even if unexplained metabolic acidosis with elevation of lactate level is considered as an early sign^[151], several studies have shown that serum lactate may remain normal in the presence of extensive mesenteric infarction^[139]. As indicated by Edwards *et al*^[130], neither routine clinical investigations nor biological tests (such as leucocytosis, and elevation in serum creatinine, creatine kinase, hepatic or pancreatic enzymes) are discriminatory for mesenteric ischemia when the diagnosis is clinically suspected.

Imaging

When AMI is suspected after CPB, imaging studies should be such that they provide a rapid and accurate diagnosis, while being safe and avoid further morbidity in an already fragile patient. Traditional radiologic studies are not accurate for the diagnosis of AMI (as stated above) and are not recommended. Abdominal ultrasound is a non-invasive technique but remains highly operator-dependent. Its accuracy decreases significantly in emergent situations, especially in the presence of ileus and dilated bowel loops, which have a negative impact on image quality. Multi-detector abdominal CT scan theoretically provides good diagnostic results in AMI, but its specificity and sensitivity after CPB are reduced dramatically; accurate diagnosis correlates with laparotomy findings in < 50% of patients^[139]. This is in part due to the limitation in the use of intravenous contrast because of borderline renal function and frequent presence of diabetes in these patients. This consideration applies also to angiography that remains the gold standard diagnostic tool in peripheral splanchnic disease^[152], despite its invasive nature and time-consumption.

Therapeutic options

An early diagnosis and prompt treatment based on a high index of suspicion are the only hope for reducing the mortality and improving the outcome^[153,154]. The initial treatment consists of hemodynamic support, but if these measures fail, prompt intervention is mandatory. Some experts believe that surgery within the first 6 h has a positive impact on the prognosis^[151]. Since most mesenteric ischemic episodes after CPB are due to NOMI, some authors argue in favour of selective angiography as the initial test, as it provides the potential for both diagnosis and therapy. Intra-arterial infusion

of papaverine^[155] or tolazoline with heparin^[125] are both effective treatments.

Perspectives for the future

AMI occurring in the post CPB period remains a challenging surgical emergency, characterized by extremely high mortality and a controversial management approach. As stated above, clinical assessment as well as laboratory and radiological tests are typically unreliable in establishing the diagnosis.

In view of these considerations, we have adopted a uniform treatment strategy. When a diagnosis of AMI after CPB was suspected, exploratory laparotomy was carried out, irrespective of the results of the diagnostic tests. We performed a retrospective analysis on 1634 consecutive patients undergoing CPB for coronary artery bypass alone or combined with valvular surgery, between January 1st, 2000 and July 31st, 2007. A total of thirteen patients were suspected to have mesenteric ischemia, based on clinical and/or laboratory and/or radiological findings. All patients underwent exploratory laparotomy and were divided into two groups (Group 1 and Group 2) depending upon whether or not ischemic bowel was present. There was no difference in the clinical findings, laboratory tests and radiological results between the two groups. The mean delay in laparotomy was 13.7 h and 51.4 h in Group 1 and Group 2, respectively; the difference was statistically significant. Mortality rates in Group 1 and Group 2 were 42.8% and 50%, respectively. Based on these findings, we concluded that in the context of post-CPB AMI, diagnostic tests do not provide any information of practical value, but instead consume valuable time. By performing early exploratory laparotomy, we were able to reduce the mortality rate considerably. Since all of our patients had NOMI, no revascularization was required and resection of the irreversibly ischemic and infarcted segments of the bowel helped in preventing the vicious circle leading to multi-organ failure and death.

CONCLUSION

Although AMI has been known for several decades, it remains a highly lethal emergency, characterized by numerous controversies. The pathophysiologic process has not been completely resolved, the clinical presentation is often subtle and misleading, and despite the introduction of new technologies the diagnostic tools are often inaccurate. A high index of suspicion and prompt treatment are the only means to reduce mortality.

AMI in cardiac patients undergoing CPB is an extremely challenging surgical emergency. The role of clinical evaluation becomes even more relevant since the laboratory and radiological tests are no longer effective. In this context, prompt laparotomy is the only method of providing objective assessment and targeted treatment. Using this approach we achieved considerable improvement in the mortality rate. Although promising, this practice needs to be confirmed in a larger series of patients.

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