

Gastrointestinal complications and its predictors after cardiac surgery

Kalp cerrahisi sonrası gastrointestinal komplikasyonlar ve prediktörleri

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INTRODUCTION

Gastrointestinal (GI) problems are infrequent but serious complications of cardiac surgery, with high rates of morbidity and mortality. The occurrence of life-threatening GI complications after cardiopulmonary bypass (CPB) was reported for the first time by Berkowitz in 1963, a few years after the clinical introduction of the heart-lung machine. The purpose of this paper is to review the current literature regarding the predictors and occurrence of this condition.

Predictors

The incidence, pattern and outcome of GI complications after CPB have been investigated in numerous studies. The reported incidence of GI complications varies between 0.12 to 3.0% with an associated mortality of 11 to 63% (1).

Data regarding the predictors of these complications are not well characterized, and the role of fundamental variables remains controversial. It has been well established, however, that the severity of these complications varies widely; consequently, the associated treatments and outcomes are similarly diverse (2). In addition, splanchnic ischemia may play a key role in the initiation and perpetuation of the systemic inflammatory response syndrome (SIRS) that often follows cardiac surgery. It may contribute to further injury of the intraabdominal organs and kidneys as well as remote organs such as the lungs, heart and brain, leading to additional morbidity, sometimes multiorgan failure, and death.

The mortality is highest for hepatic failure (average 74%) and bowel ischemia (average 71%); intermediate with perforated ulcer (average 44%); and lowest, but still substantial, with GI bleeding, pancreatitis, cholecystitis, diverticulitis, paralytic ileus, pseudo-obstruction of the colon, and bowel obstruction (11%–27%). The occurrence of GI complications is also associated with a prolonged length of stay in the intensive care unit (ICU) and hospital (3).

Despite major technological and medical advances, CPB still imposes considerable physiologic stress on the patient. Intraabdominal ischemic injury is a likely contributing factor in most GI complications after cardiac surgery (4).

Factors such as mechanical ventilation, renal failure and sepsis are the strongest predictors of GI complications, causing splanchnic hypoperfusion, hypomotility and hypoxia. Furthermore, excessive anticoagulation after valve replacement may lead to GI hemorrhage. Valve surgery, often requiring anticoagulant treatment, increases bleeding. Monitoring mechanical ventilation and hemodynamic parameters, adopting early extubation and mobilization measures, preventing infections, and strictly monitoring renal function and anticoagulation may prevent catastrophic abdominal complications. Although the pathogenesis of GI lesions is complex and multifactorial, the major factor implicated in cardiac surgery is reduced systemic blood flow, which leads to insufficient oxygen delivery

and energy deficit. The GI organs are at particular risk for ischemic events for several reasons, and oxygen shunting and consequent distal hypoxia can occur at the tip of the intestinal villi even under normal conditions. The GI tract does not have the ability to autoregulate to compensate for reductions in blood pressure. Furthermore, due to persistent vasoconstriction, splanchnic hypoperfusion may continue even after hemodynamic stability has been regained.

Preoperative, intraoperative, and postoperative variables may all influence abdominal perfusion. Comorbid conditions, such as low left ventricular ejection fraction (EF) and peripheral vascular disease, may all cause splanchnic hypoperfusion and have actually been identified as determinants of GI complications in patients undergoing cardiac surgery. In one study, only peripheral vascular disease was more common in the group with GI complications, although statistical significance was achieved solely by univariate analysis. During the intraoperative phases, hypovolemia, prolonged CPB, and administration of vasoconstrictors can cause GI hypoperfusion. CPB is associated with a broad range of systemic complications, including nonpulsatile flow, hemolysis, activation of the inflammatory cascade, anticoagulation, hypothermia, and, finally, reduced end-organ perfusion. Furthermore, CPB can increase GI permeability and, as a consequence, enhance the release of cytokines that will lead to mucosal damage and microcirculation problems. Findings regarding the relationship between CPB duration and GI complications are not uniform (5). D'Ancona and co-authors (5) concluded that prolonged mechanical ventilation, acute renal failure, sepsis, valve surgery, chronic renal failure, and sternal wound infection are major determinants of GI complications after cardiac surgery. However, another group of authors reported that CPB including cardioplegic arrest is the main independent predictor of postoperative GI complications in patients undergoing cardiac surgery (6).

In one study, previous revascularization, combined cardiac procedures (coronary artery bypass graft [CABG] plus an intracardiac or proximal aortic procedure), EF <0.4, preoperative total bilirubin level >1.2 mg/dl, low platelet count, and prolonged partial thromboplastin time (PTT) were independent preoperative predictors of adverse GI outcome in the combined preoperative and intraoperative multivariate model ($p < 0.05$). The trans-

fusion of bank blood and the use of pharmacologic cardiovascular support were identified as independent intraoperative risk factors (7). Improvements in surgical techniques such as off-pump surgery do not seem to have made any significant impact on the incidence or prognosis of adverse GI outcome (8). The incidence rates of GI complications were found to be similar in the on- and off-pump CAB groups; the type of GI complications, however, was different. The mortality rate due to these complications was also similar and remained high, regardless of the type of surgery (9, 10).

Typical symptoms of GI tract disease may be masked by routine analgesic treatment in the conscious patient, or GI symptoms may be practically absent in the sedated and ventilated ICU patient. Furthermore, brief episodes of unspecific GI complaints such as poor appetite, lack of taste, or nausea, occur in about 20–40% of patients after cardiac surgery. It is important to note that the clinical onset of GI complications is not necessarily immediately after surgery, but may develop after a delay of several days (1).

Due to the absence of early specific clinical signs, GI complications after cardiac surgery are often diagnosed late with resultant high mortality. Common in-hospital late complications (beyond 7 to 10 days postoperatively) include bowel ischemia from embolization; low flow, upper or lower GI bleeding from gastritis; peptic ulceration and diverticular disease; and diarrhea from pseudomembranous colitis, pancreatitis, cholecystitis, or septic rupture of the spleen.

CLINICAL FEATURES

Intestinal Complications

Mesenteric ischemia is a rare but catastrophic complication, with mortality rates as high as 91%. Nonocclusive ischemia (45% to 60%) and embolic events are the common causes of mesenteric ischemia. Physical examination findings of ileus or acute abdomen with elevated lactate and amylase levels should usually arouse suspicion. Radiologic and angiographic studies are generally used to confirm diagnosis. Interventions include intra-arterial papaverine injection, embolectomy (9%), bowel resection (36%), and exploratory laparotomy (55%) (11). There is a need to adopt an aggressive approach to diagnosis in suspected cases of non-occlusive mesenteric ischemia, and the role of mesenteric angiography needs to be evaluated (12).

Hepatic Complications

Synthetic and secretory dysfunction of the liver, icterus, and hepatic encephalopathy are rare complications of cardiac surgery utilizing CPB. Risk factors predicting hepatic dysfunction and adverse outcomes include preoperative hepatic dysfunction evidenced by elevated SGOT, SGPT, prothrombin time, conjugated and unconjugated bilirubin, thrombocytopenia, right heart failure with severe hepatic congestion, portal hypertension, cardiac cirrhosis, hypotension, hypoxemia, and amount of transfusions. Age, sex, underlying cardiac lesions, and the presence or absence of hepatitis B are not predictive of postoperative jaundice. Good pre-, intra-, and postoperative management of systemic perfusion (with high flow rates), optimization of heart and liver function, and pharmacologic/mechanical circulatory support facilitate better outcomes in patients with liver dysfunction.

Pancreatic Complications

The incidence of pancreatic complications in cardiac surgery patients is reported to be 0.44%. Hyperamylasemia is common after cardiac procedures (32%). About 20% of patients with hyperamylasemia (less than 1000 IU/L) exhibit no serum lipase elevations and the elevated amylase is predominantly the salivary isoenzyme. About 3% of patients with elevated amylase and lipase have overt clinical and diagnostic signs of severe pancreatitis. Patients with hyperamylasemia exhibit higher mortality compared to controls. The risk factors include pre/postoperative hypotension, excessive use of inotropic support, renal failure, prolonged ventilatory support, intra-aortic balloon usage, and administration of calcium at doses of 800 mg or more in the perioperative period.

Splenic Complications

Splenic injury and rupture following cardiac sur-

gery are very rare complications, sometimes noted in patients with myeloid dyscrasias and enlarged spleen or in those with erosion from fulminant pancreatitis or operative trauma exacerbated by full heparinization.

Biliary Complications

Acalculous and calculous cholecystitis have both been described following cardiac surgery and cardiothoracic organ transplantation. In a study of 645 cardiothoracic organ transplantation patients, 5.7% had symptomatic cholecystitis with all of them containing gallstones. All patients were female with a higher body mass index, and a significant proportion also had common bile duct stones (13).

Gastrointestinal bleeding, followed by acute pancreatitis, acalculous cholecystitis, peptic disease without bleeding or perforation, perforated ulcer, bowel infarction, diverticulitis, and liver dysfunction are all manifestations of hypoperfusion and response to the stress of surgery. Bowel infarction is the most lethal of all intraabdominal complications. Relative visceral ischemia may result from prolonged CPB, vasoconstriction, low cardiac output, maldistribution of perfusion, obstructive splanchnic disease, and embolism (14).

CONCLUSION

Early diagnosis and timely therapeutic interventions appear to be the most effective means for improving the poor outcome of GI complications after CPB. Although GI complications were associated with prolonged CPB time, the role of CPB in splanchnic injury remains controversial. Further research into the pathophysiology of GI hypoperfusion during cardiac surgery, either on-pump or off-pump, seems to be required.

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