9 Gastrointestinal Complications and Their Management After Adult Cardiac Surgery

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Abstract

Gastrointestinal complications (GICs) following cardiac surgery are often severe and contribute to substantial morbidity and mortality. The diagnosis of GICs remains difficult because symptoms and signs are often subtle, or nonspecific, and this commonly leads to delay in definitive diagnosis and treatment. Preventive strategies, coupled with early recognition and aggressive management, provide the foundation of the general clinical approach to addressing these complications. Overall, a high index of clinical suspicion and a low threshold for investigation and definitive management are recommended in patients with nonroutine clinical progress after cardiac surgery. It is imperative that all clinicians who care for postoperative cardiac surgical patients be familiar with the full spectrum of potential GICs in this patient population, as well as the general therapeutic approaches to these complications.

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9.1 Introduction

Gastrointestinal complications (GIC) after cardiac surgery encompass a heterogeneous group of pathologies, ranging from a simple, temporary paralytic ileus to more serious, life-threatening conditions, such as gastrointestinal hemorrhage, cholecystitis, acute pancreatitis, liver failure, and mesenteric ischemia. Although relatively infrequent, these complications are often severe, resulting in a prolonged hospitalization and increased cost and high mortality. Despite the advances in perioperative care of cardiac surgery patients, the incidence of postoperative GICs and associated mortality has not changed substantially since the earliest reports (Diaz-Gomez et al. 2010). Recent improvements in surgical technique and perioperative care to reduce GICs may have been offset by the increasing complexity of cardiac surgery with older and sicker patients undergoing operations.

The clinical diagnosis of GICs is often difficult as a result of multiple factors, including differences in typical clinical signs and symptoms compared with noncardiac surgery patients, the impact of medications affecting assessment (e.g., sedatives, neuromuscular-blocking agents and analgesics), and underlying patient comorbidities. Diagnosis may further be impaired by the tendency to underestimate potentially lethal consequences of GICs because of their relative infrequency and the fact that they lack a "visible connection" to the primary target organ of cardiac surgery (Karangelis et al. 2011).

The pathogenesis of GICs is complex, not fully understood, and often multifactorial. Risk factors for GICs have been identified and may enable the use of preventive strategies, prompt early investigation, and allow early identification of complications in the perioperative period. Delayed recognition of these complications leads to a high incidence of morbidity and mortality; therefore prompt diagnosis and treatment are essential. This chapter reviews the epidemiology, clinical features, and treatment of patients developing GICs after cardiac surgery.

9.2 Epidemiology

9.2.1 Incidence

The incidence of gastrointestinal complications following cardiothoracic surgery ranges widely from <1% to 5.5% of patients and is associated with mortality rates between 14 and 61% (Vassiliou et al. 2008; Zhang et al. 2009).The most common GICs include ileus, gastrointestinal (GI) bleeding, bowel ischemia, and pancreatitis. Perforation of a duodenal ulcer, hepatic failure, and cholecystitis occur

less frequently. The wide variation in incidence of GICs may be accounted for by differences in patients studied, surgical technique, and reporting. Previous reports on the incidence and risk factors for these complications have largely focused on patients undergoing coronary artery bypass grafting (CABG) rather than the larger population of cardiac surgical procedures. With the broader application of percutaneous transluminal coronary angioplasty, the population of patients referred for cardiac surgery has significantly changed with a majority of patients in tertiary centers undergoing more complex procedures including combined valve/CABG, multiple valve, and aortic procedures. In addition the current population of cardiac surgical patients are often older and present with significant preoperative comorbidities including atherosclerotic diseases. These factors potentially increase the risk of abdominal organ hypoperfusion and thromboembolic events, which represent two major pathophysiologic mechanisms of ischemic complications. The high mortality in cardiac surgical patients that develop GICs has been attributed to delayed diagnosis and treatment, which often precipitates multisystem organ failure. However, these patients frequently have serious associated medical problems and limited physiologic reserved, which diminishes their ability to survive a major ischemic, septic, or hemorrhagic insult. During the last decade, significant advances have also been made in the perioperative management of patients undergoing cardiac surgery, which might favorably impact the incidence of GICs. These changes in the cardiac surgical population and advances in management have raised the question of the validity of previously reported incidence and risk factors for the occurrence of GICs.

Variation in the incidence of GICs might also be explained by an inconsistency in definitions in different studies. For example, a retrospective study by Mangi et al. analyzed 8709 patients undergoing cardiac surgery and reported an incidence of GICs of 0.5% (Mangi et al. 2005). These authors only reported patients with GICs which required a general surgical consult. Using this definition, only the "sickest" patients were included, particularly those with ischemic bowel disease. Patients with GICs managed with medical or endoscopic treatment without surgical consult were not reported in their series. In contrast in a national multicenter investigation that included more than 2.5 million CABG procedures, the reported GIC rate was 4.1% (Rodriguez et al. 2007). In this analysis rare GICs such as intraabdominal abscess, *Clostridium difficile* infection, esophageal ulceration, and diverticulitis were included which likely explains the higher incidence of GICs in their analysis. Some literature suggests a reduction in the incidence of both ischemic and, more markedly, of hemorrhagic GICs (Filsoufi et al. 2007; Ashfaq et al. 2015).While the precise explanation for this finding has not been elucidated, it has been suggested that the lower incidence of GICs might lie in systematic application of preventive measures and new advances in the intraoperative management of patients undergoing cardiac surgery.

9.2.2 Risk Factors

Numerous risk factors have been reported for GICs after cardiac surgery (Vassiliou et al. 2008; Gulkarov et al. 2014). In general, patients with comorbidities and those with a prolonged or complicated postoperative course are most likely to develop GICs. Although for each complication there may be individual risk factors, as a

Table 9.1 Risk factors for GI complications after cardiac surgery

Preoperative	Intraoperative	Postoperative
Advanced age Decreased left ventricular ejection fraction (<40%) Peripheral vascular disease Peptic ulcer disease Chronic kidney disease Diabetes mellitus COPD Use of preoperative inotropic support or IABP	Valvular or combined CABG/valve surgery Emergency surgery Prolonged CPB time Increased blood transfusion Presence of arrhythmias	Prolonged mechanical ventilation Need for reoperation (re-sternotomy or re-thoracotomy) Stroke Postoperative infection or sepsis (including sternal wound infection) Acute kidney injury Need for vasopressors or IABP after surgery

general principle, those factors that lead to reduced peripheral blood delivery and tissue oxygenation increase the incidence significantly. Some of the identified factors seem to be universally present across different studies; some others are likely unique to specific study populations. Many patients with GICs present with more than one complication (Filsoufi et al. 2007). Risk factors for GICs may be classified as pre-, intra-, or postoperative (Table 9.1). Commonly cited preoperative risk factors include advanced age, chronic renal failure, hepatic insufficiency, peripheral vascular disease, diabetes mellitus, chronic obstructive respiratory disease, preexisting gastrointestinal disease, congestive heart failure, prior myocardial infarction, low cardiac output state, and use of inotropic support or an intra-aortic balloon pump (IABP). Intraoperative risk factors include prolonged cardiopulmonary bypass (CPB) duration, valvular surgery, emergency surgery, increased blood transfusion, use of IABP, and the presence of arrhythmias. Postoperative risk factors for GICs include prolonged mechanical ventilation, an acute kidney injury, a deep sternal wound infection, and a low cardiac output state.

Based on identified risk factors, risk stratification scores have been developed to estimate the probability for the patient to develop a GIC after cardiac surgery (Díaz-Gómez et al. 2010; Karangelis et al. 2011; Vassiliou et al. 2008; Zhang et al. 2009; Mangi et al. 2005; Rodriguez et al. 2007; Filsoufi et al. 2007; Ashfaq et al. 2015; Gulkarov et al. 2014). Risk stratification based on identification of such scores may influence operative strategy or heighten the index of suspicion of the treating clinician when confronted with a postoperative course which deviates from the expected norm. Finally, risk stratification scores for GICs may provide clinically relevant information indicating subsets of patients most likely to benefit from invasive procedures such as laparotomy and may provide a framework for providing patients and their families with realistic expectations should such complications occur.

9.3 Pathophysiology

Under normal conditions, the splanchnic circulation receives 20% of cardiac output and accounts for 20% of total body oxygen consumption (Allen 2014). Blood is supplied to the liver, stomach, pancreas, and duodenum by the celiac artery, the

superior mesenteric artery supplies the pancreas, duodenum, jejunum and ileum, ascending and transverse colon, and the descending and sigmoid colon are supplied by the inferior mesenteric artery, with each major artery branching from the abdominal aorta. The splanchnic circulation acts not only as perfusion to the abdominal organs but also as a blood reservoir, enabling compensatory autotransfusion into the central circulation (of approximately 800 mL blood) in response to hypovolemia, catecholamines, or low cardiac output (Hessel 2004). The splanchnic supply is usually autoregulated by resistance arterioles, which dilate in response to a decrease in mean arterial pressure (MAP) or accumulation of metabolites. However, the splanchnic circulation is unable to autoregulate perfusion at extremes of pressure or flow and therefore is vulnerable to alterations in these parameters during cardiopulmonary bypass (CPB), hemorrhage, hypovolemia, or arrhythmias.

Splanchnic hypoperfusion and impaired oxygen-induced ischemia are thought to be the primary cause of most GICs after cardiac surgery (Moneta et al. 1985; Ohri and Velissaris 2006). Hypoperfusion may be caused by reduced or suboptimal cardiac output, impaired regional blood flow, or inadequate systemic MAP. Additional injury likely occurs through systemic inflammation and systemic inflammatory response syndrome (SIRS), release of inflammatory mediators, nonpulsatile blood flow, hypothermia, drug therapy, and mechanical factors.

The SIRS response occurs as a result of surgical stress, contact with the CPB circuit, mechanical ventilation, and ischemia itself which may exacerbate SIRS together with reperfusion injury. The inflammatory and complement cascades release mediators which have vasoconstrictor actions and cytokine activation have been implicated in vascular endothelial dysfunction and damage (Ohri and Velissaris 2006). All these factors contribute to a maldistribution of blood flow and impaired mucosal oxygen delivery.

CPB causes renin release and activation of the renin-angiotensin-aldosterone axis with secretion of angiotensin II, a potent vasoconstrictor. Vasoactive drugs commonly used in the perioperative period such as norepinephrine and vasopressin are also associated with splanchnic hypoperfusion. In addition hypothermia used with CPB is associated with vasoconstriction and altered regional blood flow and distribution (Slater et al. 2001).

Mechanical factors that may contribute to GI ischemia include micro- and macro-emboli resulting from air, atheroma, thrombus, or debris and hepatic and GI congestion related to venous cannula placement. Mechanical ventilation especially requiring high positive end-expiratory pressure (PEEP) can result in hypotension and impaired cardiac output, leading to splanchnic vasoconstriction and hypoperfusion. In combination these factors result in the shunting of blood away from the GI system, leading to organ ischemia and damage.

Nonischemic mechanisms may also contribute to the development of GICs after cardiac surgery including bacterial translocation (resulting from altered mucosal barriers and blood flow), adverse drug reactions (e.g., over-anticoagulation, amiodarone-induced hepatotoxicity), and iatrogenic organ injury (e.g., malpositioned surgical drains).

9.4 Diagnostic Considerations

Early diagnosis of GICs in cardiac surgery patients is often challenging. Clinical presentation varies with pathology, no single diagnostic test will reliably diagnose or exclude all intra-abdominal pathology, and investigation should be directed by patient history and presentation. A patient's complaint of abdominal pain in the appropriate clinical setting is often the most sensitive indicator of significant GI pathology. Other clinical indices that should prompt efforts for early recognition of GICs include abdominal bloating, persistent ileus, sepsis, or GI bleeding. Multiorgan failure, metabolic derangement, and cardiovascular instability are nonspecific and often late signs of complications. Overall, a low threshold for investigation in those patients with nonroutine postoperative courses is recommended. In addition to a review of risk factors and physical exam, initial laboratory testing should include serum lactate, glucose, a hepatic panel (transaminases, bilirubin, alkaline phosphatase, and gamma-glutamyl transpeptidase), coagulation parameters, and complete blood count including white blood cell count and differential. These may be followed by abdominal radiography, ultrasound or computed tomography scanning, upper and lower endoscopy, and diagnostic laparoscopy or laparotomy as indicated.

9.5 Treatment Considerations

Since early diagnosis of GICs after cardiac surgery is difficult and patients are often critically ill at the time complications are recognized, management of these serious, often life-threatening, problems is challenging. Initial treatment is often conservative, but when it fails, prompt surgical intervention is necessary. Timely operation or intervention may be lifesaving in patients who are unable to compensate from the severe hemodynamic disturbances of a refractory abdominal complication, such as major bleeding or sepsis. Hesitation to intervene because the patient "had recent cardiovascular surgery" should not be a barrier. Special considerations are necessary for postcardiac surgical patients undergoing abdominal surgery (Dong et al. 2012). The lower end of the sternal wound should be protected from contact with the abdominal incision to prevent contamination and reduce the risk of sternal infection and mediastinitis. Prophylaxis against bacterial endocarditis should be instituted, especially in patients with a prosthetic vessel or valve(s). Finally patients who are anticoagulated require judicious reversal and re-institution of anticoagulation after abdominal surgery.

9.6 Specific Conditions

The clinical presentation, suggested diagnostic tests, and management considerations for specific conditions are described in the following sections and summarized in Table 9.2.

Table 9.2 Summary of gastrointestinal complications, common clinical presentations, and suggested investigations (adapted from Allen 2014)

Complication	Clinical presentation	Diagnosis	Management
Ileus	Abdominal distension and pain Nausea/vomiting	Abdominal radiograph	Suppositories, enemas, prokinetic agents Mobilization Minimizing opioids Correction of electrolyte abnormalities Nasogastric tube decompression
Colonic pseudo-obstruction	Abdominal distension and pain	Colonic dilation and fluid levels on abdominal radiograph	Neostigmine and colonoscopic decompression
Dysphagia	Odynophagia, hoarseness Coughing or gagging when swallowing	Videofluoroscopic swallow study or fiber-optic endoscopic evaluation of swallowing	Modification of eating and swallowing Nasoenteral tube or parenteral feeding
Gastritis and esophagitis	Gnawing or burning ache or abdominal pain Nausea or vomiting	Tests for ***H. pylori*** Endoscopy	Initiate enteral feeding H2 receptor blockers or proton pump inhibitors
GI bleeding Upper: duodenal or gastric ulceration Lower GIT: diverticulitis, AV malformations	Blood/melena per rectum Hematochezia Hemodynamic instability	Hemoglobin Lactate dehydrogenase Endoscopy (evidence or site of bleeding identified and potentially treated)	Intravenous fluid resuscitation Correction of coagulopathy Proton pump inhibitors Endoscopy for clipping or sclerotherapy to bleeding vessels Angiography, surgery (if lower gastrointestinal tract bleeding with arteriovenous malformation or diverticulitis)
Mesenteric ischemia Occlusive: emboli or thrombus Nonocclusive mesenteric ischemia (NOMI): hypoperfusion	Shock Abdominal pain and distension Intolerant of enteral nutrition GI bleeding	Complete blood count (leukocytosis) Lactate Abdominal radiograph (distended bowel, thickened bowel, evidence of ileus) Computed tomography Mesenteric angiography (global impairment of perfusion) Colonoscopy (ischemic bowel) Laparotomy (ischemic bowel)	Intravenous fluid resuscitation Circulatory support (inotropes or vasopressors) Antibiotic therapy Embolectomy, thrombectomy, and endarterectomy, as well as endovascular techniques such as balloon angioplasty, percutaneous stenting, and thrombolysis Laparotomy and bowel resection if perforation

(continued)

Table 9.2 (continued)

Complication	Clinical presentation	Diagnosis	Management
Peptic ulcer perforation	Abdominal pain, distension peritonitis	Abdominal radiograph (peritoneal air) Computed tomography scan abdomen (peritoneal air, collection)	Intravenous fluid resuscitation Proton pump inhibitor high dose Laparotomy-vagotomy and oversew of ulcer or resection Enteral rest and nasogastric drainage
Pancreatitis	Shock Epigastric and back pain Nausea and vomiting Abdominal distension	Amylase Lipase Computed tomography scan abdomen (pancreatic inflammation, free fluid, necrosis)	Intravenous fluid resuscitation Postpyloric feeding or intravenous nutrition Supportive therapy Analgesia Percutaneous drainage or surgical treatment
Cholecystitis Calculous or acalculous	Often 10–15 days postsurgery Right upper quadrant pain Fever Leukocytosis SIRS/shock	Liver function tests (elevated) Ultrasound (thickened gallbladder and common bile duct, ±gallstones) CT scan of abdomen Laparoscopy	Surgery (calculous) with cholecystectomy Antibiotics ± percutaneous drainage (acalculous)
Hepatic dysfunction	May be asymptomatic Jaundice	Elevated liver function tests (most commonly hyperbilirubinemia, transaminitis) Abdominal ultrasound (exclude obstruction, thrombosis, collections) Hepatitis serology screen (exclude as cause)	Stop potential hepatotoxins Supportive

9.7 Ileus

Ileus is a common GIC which in its simplest form affects almost every patient undergoing cardiac surgery (Karangelis et al. 2011). Perioperative fasting, effects of anesthetic agents, opioids, and decreased patient mobility all contribute to the intestinal dysfunction, which in the vast majority of cases resolves spontaneously in the early postoperative period. In a small proportion of patients, the ileus persists after the fourth postoperative day and requires the use of suppositories, enemas, and promotility agents (i.e., metoclopramide, erythromycin) or methylnaltrexone to facilitate clinical resolution. Close clinical monitoring, patient mobilization, minimizing the use of postoperative opioids, and correction of serum electrolyte abnormalities are usually successful in restoring or improving intestinal function. Cases that remain unresponsive are often managed with nasogastric suction, which should be

continued until the return of bowel function. It is important to recognize that the appearance of clinically significant new ileus, especially when accompanied by severe abdominal pain, may indicate a more serious underlying problem such as mesenteric ischemia or pancreatitis.

9.8 Colonic Pseudo-obstruction

Colonic pseudo-obstruction (Ogilvie's syndrome) is an acute dilatation of the colon without any mechanical obstruction which occurs in up to 3.5% of patients undergoing cardiac or thoracic surgery (Guler et al. 2011). It may also develop after noncardiac surgery or systemic illness. It is characterized by massive colonic dilatation and the presence of fluid levels on abdominal radiograph. While the pathophysiology has not been fully elucidated, this condition seems to be associated with a disturbance of the autonomic innervation of the colon. Untreated, colonic pseudo-obstruction can lead to cecal overdistention and subsequent perforation. Perforation is more likely to occur if the cecal diameter exceeds 10–12 cm (De Giorgio and Knowles 2009). Two common management modalities for colonic pseudo-obstruction, used alone or in combination, are neostigmine administration and colonoscopic decompression. Surgical options for more refractory disease include cecal decompression (i.e., cecostomy) and colonic resection with ostomy creation.

9.9 Dysphagia

Dysphagia is a common complaint following cardiac surgery. The etiology of postoperative dysphagia is often multifactorial, including contributions from gastroesophageal reflux, local tissue trauma and inflammation from surgery, endotracheal intubation, intraoperative transesophageal echocardiography, and other potential factors such as recurrent/superior laryngeal nerve injury (Grimm et al. 2015). Affected patients are at increased risk for complications such as aspiration and pneumonia. Risk factors for dysphagia after cardiac surgery include male gender, low body mass index, chronic lung disease, cerebrovascular disease, placement of ventricular assist device or heart transplantation, hypothermic circulatory arrest, and prolonged postoperative mechanical ventilation. Early consultation with a speech language pathologist is vital to accurately diagnose patients with dysphagia in the immediate postoperative period. Therapy consists of modification of eating behavior and swallowing technique and in some more severe cases enteral tube or parenteral feeding.

9.10 Gastritis and Esophagitis

Gastritis and esophagitis are among the more common GICs in the cardiac surgery patient population. The etiology is multifactorial, with contributing factors including mucosal hypoperfusion, preexisting history of gastric or esophageal mucosal

disorders, and the use of nonsteroidal anti-inflammatory drugs. Esophagitis is often associated with gastro-esophageal reflux, a particular concern for postsurgical patients due to the potential for pulmonary aspiration. Management of esophagitis and gastritis during the perioperative period includes avoidance of hypotension, avoiding delay in enteral feeding, and aggressive management with H2 receptor blockers or proton pump inhibitors. Diagnosis of esophagitis and gastritis is typically based on history and clinical symptoms with endoscopy as the most commonly utilized diagnostic modality. Maintaining head-of-bed elevation is an important preventive measure for postoperative patients with gastro-esophageal reflux and high pulmonary aspiration risk.

9.11 GI Hemorrhage

Gastrointestinal bleeding (GIB) is among the most common GICs following cardiac surgical procedures. In general, upper GIB occurs more frequently than lower GIB, with most hemorrhages (>90%) occurring proximal to the ligament of Treitz (Yilmaz et al. 1996). The two most common etiologies of upper GIB in cardiac surgical patients are duodenal ulceration and gastric erosion. Gastric and duodenal ulceration following cardiac surgery are likely secondary to systemic hypoperfusion with subsequent development of mucosal ischemia and erosion. Contributing factors include preoperative fasting, coagulation disorders, history of gastric or duodenal ulcer disease, and prolonged mechanical ventilation.

The initial step in diagnosis of GIB is the insertion of a nasogastric tube and lavage of gastric contents which aids in determining if the GI hemorrhage is proximal to the ligament of Treitz. Medical therapy is attempted first and includes the administration of proton pump inhibitors, red blood cell transfusion, and correction of coagulopathy. If medical management fails, upper endoscopy is the next step in evaluation and treatment of potential bleeding source(s). Endoscopic interventions are aimed at stopping the bleeding by cauterization, vasoconstrictive agent injection, or both. Endovascular embolization is now considered the first-line therapy for massive UGI bleeding that is refractory to endoscopic management (Loffroy et al. 2015). Surgical intervention is indicated if the patient fails medical endoscopic and endovascular treatment. In general, the presence of continued hemodynamic instability and a predetermined transfusion threshold (e.g., >4–6 units of packed red blood cells) are utilized as "triggers" for surgery.

As with upper GI bleeding, management of lower GI hemorrhage following cardiac surgical procedures begins with hemodynamic resuscitation and normalization of coagulation parameters. If the bleeding does not stop, the next step is the identification of the source of hemorrhage, either endoscopically or by imaging (e.g., angiography). In many cases, the bleeding can be controlled endoscopically or with endovascular embolization. Surgery is reserved for the failure of non-operative therapies.

Of note, gastrointestinal hemorrhage has been reported with increased frequency in cardiac patients with ventricular assist devices, with higher bleeding rates seen among recipients of nonpulsatile devices as compared to pulsatile devices (Cheng

et al. 2014). Nonpulsatile ventricular assist devices far outnumber pulsatile devices, and thus GI hemorrhage will continue to be an important issue in this patient population for the foreseeable future.

9.12 Acute Cholecystitis

Acute cholecystitis after cardiac surgery is rare but carries a high mortality (Passage et al. 2007). Many cases of acute cholecystitis are "acalculous" and result from a variety of factors including systemic hypoperfusion, the SIRS, prolonged fasting, and the use of opioid medications. Typical symptoms of acute cholecystitis include right upper quadrant pain and tenderness on examination. However, diagnosis is often delayed in postcardiac surgical patients due to the presence of mechanical ventilation and sedation. For patients with acute cholecystitis, diagnosis is most often confirmed with right upper quadrant ultrasound or CT scan. Initial conservative treatment with transition to surgery in cases with lack of clinical improvement after 48 h is often recommended. The definitive treatment of acute cholecystitis is cholecystectomy for patients who are able to tolerate surgery. For poor surgical candidates, percutaneous cholecystostomy can serve as "bridging" therapy until the patient is ready to undergo cholecystectomy. Mortality rates associated with acalculous cholecystitis are significant which may reflect the overall poor general health status of patients at risk for this complication.

9.13 Acute Mesenteric Ischemia

Acute mesenteric ischemia (AMI) is a potentially life-threatening complication of cardiac surgery that can occur within hours to several days after surgery. AMI can affect any part of the small or large intestine and lead to devastating complications including mucosal sloughing, gangrenous changes of the bowel wall, and perforation. Mortality rates for patients with AMI exceed 40% in recent series (Eris et al. 2013; Viana et al. 2013). Common causes of AMI include embolism to the superior mesenteric artery, acute thrombosis of an atherosclerotic plaque with previous partial occlusion, splanchnic vasoconstriction leading to low flow and regional ischemia (referred to as nonocclusive mesenteric ischemia, NOMI), and mesenteric venous thrombosis. AMI after cardiac surgery most often is due to a NOMI and is related to a reduction in the splanchnic blood flow, which can be due to low cardiac output, and aggravated by cardiovascular support, such as vasopressors, and by preexisting atherosclerotic disease. The classic sign of AMI is abdominal pain out of proportion to physical examination findings; however many patients with intestinal ischemia after cardiac surgery have vague and nonspecific symptoms. Other symptoms are also present inconsistently and may include nausea, vomiting, and diarrhea. Physical examination is often unremarkable unless peritonitis has developed. During the late stages of AMI, abdominal distension and guarding, as well as systemic complications, may develop.

The most common laboratory abnormalities in patients with AMI are an unexplained lactic acidosis, hemoconcentration, and leukocytosis. However, even at the time when mesenteric ischemia is confirmed at laparotomy, an elevation of serum lactate may not be present (Acosta et al. 2009). Abdominal radiographs are of little help in the diagnosis of mesenteric ischemia. The presence of dilated loops of bowel is nonspecific, and thickened bowel loops or "thumbprinting" caused by submucosal edema or hemorrhage is inconsistently seen. Doppler sonography is useful in diagnosing chronic mesenteric arterial occlusive disease but has limited role in AMI. In the setting of high clinical suspicion, sigmoidoscopy or colonoscopy can aid in diagnosis of colonic ischemia. Computed tomography angiography provides direct visualization of the mesenteric vasculature, intestines, and mesentery allowing fast and accurate diagnosis of AMI. Angiography is the gold standard diagnostic test in acute mesenteric artery occlusion, providing both anatomical visualization of the vessels and therapeutic options (e.g., intravascular administration of vasodilators and thrombolytics).

Treatment of AMI consists of volume resuscitation, broad-spectrum antibiotics, vasodilators, and intravenous heparin at therapeutic doses which should be initiated without delay. Although surgical revascularization has been the standard management for restoring visceral blood flow, embolectomy, thrombectomy, and endarterectomy as well as endovascular techniques such as balloon angioplasty, percutaneous stenting, thrombolysis, and thrombus extraction have all been used with favorable outcomes. Angiographically proven NOMI can be treated with intra-arterial infusion of tolazoline, papaverine, or prostaglandin E2, after the selective intra-arterial catheterization of the SMA. If the patient develops signs of bowel infarction such as peritonitis, worsening sepsis, or metabolic acidosis during treatment, laparotomy is indicated.

9.14 Liver Dysfunction

Liver dysfunction can affect up to 10% of patients after cardiac surgery and can range in severity from mild liver enzyme elevation to fulminant failure (Sabzi and Faraji 2015). Consequences of severe liver failure include impaired clearance of hepatically metabolized drugs, coagulopathy, and encephalopathy. Risk factors for postoperative liver dysfunction include preexisting liver disease, prolonged CPB time, low cardiac output states necessitating administration of inotropic agents and/or IABP, volume of blood transfusion, and combined CABG and valve operations. Side effects of anesthetic drugs, as well as mechanical pressure from a low-placed inferior vena cava cannula, can also contribute to postoperative hepatic dysfunction. Treatment is supportive with control of fluids and electrolytes and replenishment of nutrient and coagulation factors. Worsening encephalopathy, jaundice, and ascites are important clinical markers of decompensation of liver function. Monitoring of coagulation and also maintaining vigilance for signs of postoperative bleeding should be continued beyond the usual postoperative monitoring period. Intravascular catheters should be removed as soon as they are no longer needed because of the increased risk of catheter-related sepsis in patients with liver impairment.

9.15 Pancreatitis

Acute pancreatitis is a relatively uncommon complication following cardiac surgery ranging in severity from subclinical amylase and lipase elevations to severe hemorrhagic, necrotic pancreatitis. Hypoperfusion from CPB, perioperative bleeding, SIRS, micro-embolization, and a history of preexisting pancreatic or gallstone disease are factors that increase the risk of acute pancreatitis. Clinically significant pancreatitis usually occurs slightly later following cardiac surgery than other GICs, such as bleeding or mesenteric ischemia. Patients typically complain of upper abdominal and left upper quadrant pain, nausea, vomiting, and/or abdominal distension. Laboratory values including elevated amylase and lipase are usually present. However, due to the high incidence of hyperamylasemia in cardiac surgery patients (exceeding 33%), clinical correlation is required before definitive diagnosis of pancreatitis is made (Fernandez-del Castillo et al. 1991). Management of acute pancreatitis postcardiac surgery follows that for noncardiac surgery patients.

9.16 GICs Related to Mechanical Assist Devices

The expanding use of mechanical cardiac assist technologies, i.e., ventricular assist devices (VADs), intra-aortic balloon pumps (IABPs), and extracorporeal membrane oxygenation devices (ECMO), is associated with clinically significant GICs.

- Patients who undergo VAD placement are at risk for a number of potential GICs, including abdominal infection, bowel injury, acalculous cholecystitis, pancreatitis, various hernias, gastric outlet obstruction, peritoneal fluid leaks, and mesenteric ischemia.
- IABPs have long been used for perioperative circulatory support in patients with low cardiac output. Despite improving coronary perfusion and reducing left ventricular afterload, IABP use is a known risk factor for gastrointestinal complications including gastrointestinal bleeding, bowel ischemia, and pancreatitis. In addition, malposition of the IABP balloon has been established as a primary factor leading to compromised visceral blood flow (Rastan et al. 2010).
- ECMO has been associated with embolic phenomena of the systemic circulation, end-organ ischemia, gastrointestinal hemorrhage, and abdominal compartment syndrome.

9.17 Prevention

A number of strategies have been suggested for prevention of GICs after cardiac surgery:

Preoperative preparation

- Use of preoperative risk stratification models may allow preventive strategies to be used pre- and intraoperatively as well as prompt earlier investigation, diagnosis, and management of complications postoperatively.
- Prophylactic gastric acid suppression using H2 blockers or proton pump inhibitors has been recommended to reduce the risk of peptic and duodenal ulcers and GI bleeding (Patel and Som 2013). Prophylactic acid-suppressive therapy has been associated with an increased incidence of pneumonia and other complications in hospitalized patients; therefore a practical approach is to initiate treatment during perioperative period and discontinue the therapy once normal oral intake is re-established.
- Preoperative hemodynamic optimization with correction of hypovolemia and anemia and support of cardiac output (e.g., inotrope therapy or IABP if required) may be beneficial for maintaining perfusion of the organs of the GI tract, thereby reducing GICs. However there are no large clinical trials to validate this approach. At present there remains considerable debate around the use of preoperative transfusion, and to date, no minimal preoperative hemoglobin target or threshold has been established.

Intraoperative prevention

- Intraoperative maintenance of adequate cardiac output and oxygenation is important; however, the exact hemodynamic parameters for adequate cardiac output and oxygen delivery are unknown and likely vary between patients.
- Several methods for monitoring GI perfusion, including measurement of gastric pH, ultrasound of blood flow in hepatic or mesenteric vessels, and measurement of intestinal transport functions, have been described but are not presently used clinically.
- Aspirin treatment within 48 h postoperatively has been associated with a reduction in both the incidence and mortality of GICs in CABG surgery (Mangano 2002).
- Milrinone infusion in patients undergoing CABG resulted in reduced gastric mucosal acidosis and lower inflammatory marker and endotoxin levels in a small RCT (Mollhoff et al. 1999). However other inotropic and vasoactive therapies such as epinephrine, dopamine, dobutamine, and vasopressin have been associated with reduced blood flow to the splanchnic circulation despite increases in MAP and systemic blood flow. It is likely that these detrimental effects are attributable at least in part to the mesenteric arteriolar constriction which is marked in response to systemic vasoconstrictors and overrides normal autoregulation. Minimizing the use of pure vasoconstrictors has been suggested through the use of inotropes if support for MAP targets is required.
- Modification of CPB as a preventive strategy to reduce GICs has been proposed and investigated with few strategies shown to be clearly effective in reducing the incidence or severity of GICs (Table 9.3).

Table 9.3 Modifications of CPB have been proposed to reduce GI complications investigations (adapted from Allen 2014)

• Minimize gaseous microemboli and atheroemboli (through the use of epiaortic scanning for cannula site selection, avoidance of excess aortic manipulation, meticulous de-airing)
• Transfusion to avoid severe anemia
• Use of CPB circuits with biocompatible surfaces
• Reduction of blood-air interfaces
• Minimize surface area and volume of the CPB circuit
• Use of pulsatile CPB flow
• Off-pump CABG surgery
• Use of internal mammary-based pedicled grafts
• Use of proximal anastomotic devices to avoid clamping the aorta in such patients
• Screening for heparin-induced thrombocytopenia (HIT) and active prophylaxis against HIT
• Fast-track extubation pathways and/or minimizing sedation to enable earlier recognition of a GICs

Conclusion

GICs following cardiac surgical procedures continue to significantly contribute to morbidity and mortality. The diagnosis of GICs remains difficult because symptoms and signs are often subtle, or nonspecific, and this commonly leads to delay in definitive diagnosis and treatment. Preventive strategies, coupled with early recognition and aggressive management, provide the foundation of the general clinical approach to addressing these complications. Overall, a high index of clinical suspicion and a low threshold for investigation and definitive management are recommended in patients with nonroutine clinical progress after cardiac surgery. Therefore, it is imperative that all clinicians who care for postoperative cardiac surgical patients be familiar with the full spectrum of potential GICs in this patient population, as well as the general therapeutic approaches to these complications.

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