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Gastrointestinal Complications in Cardiothoracic Surgery: A Synopsis

Jennifer Schwartz, David E. Lindsey,
Hooman Khabiri and Stanislaw P. A. Stawicki

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1. Introduction

Gastrointestinal complications (GIC) in cardio-thoracic surgery (GIC-CTS) constitute a heterogeneous group of non-cardiac/thoracic complications. Although relatively infrequent, these complications are associated with significant mortality and severe clinical sequelae. It is also well recognized that GIC-CTS are often difficult to identify clinically [1], and the presentation of each specific complication may differ from the presentation of said complication in non-CTS patient populations. The incidence of gastrointestinal complications following CTS ranges from <1% to 4.1% patients [2-4], and is associated with mortality rates between 13.9% and 63% [5-7]. Commonly reported GIC-CTS include gastrointestinal hemorrhage, esophagitis/gastritis, perforated ulcer, acute cholecystitis, acute pancreatitis, and mesenteric ischemia [5]. Predominant factors associated with increased mortality following a gastrointestinal complication after cardiac surgery include patient age, COPD, smoking, NYHA class III and IV heart failure, and hepatic insufficiency [8].

2. Risk factors for GIC-CTS

Numerous studies report on specific risk factors for GIC-CTS. Although some of the factors seem to be universally present across different studies, some others are likely unique to specific study populations. A comprehensive list of commonly cited risk factors compiled from the literature includes: (a) decreased left ventricular ejection fraction (<40%) including post-operative low cardiac output; (b) advanced patient age; (c) pre-existing conditions such as diabetes, renal failure, peripheral vascular disease; (d) valvular surgery or combined coro-

nary artery bypass/valve operation; (e) prolonged mechanical ventilation; (f) emergency surgery; (g) prolonged pump time; (h) need for intra-aortic balloon pump (IABP) or vasopressors during or after surgery; (i) need for re-exploration following surgery (re-sternotomy or re-thoracotomy); (j) pre-existing gastric ulcer disease; (k) stroke; and (l) postoperative sepsis/infectious complications including sternal wound infection [3-5, 9-12].

3. Physiologic and bowel motility changes following cardiac surgery

Despite significant hemodynamic implications of cardiac surgery, the effects on gastrointestinal system function are only modest at best. It is important to note that cardiopulmonary bypass impairs small intestinal transport and increases gut permeability, especially when pump times exceed 100 minutes [13]. Intestinal absorption also appears to be affected in cardio-thoracic surgical patients [14].



Figure 1. Postoperative ileus following thoracoscopic right upper lobe resection. The patient improved markedly following 5 days of therapy consisting of nasogastric suction, electrolyte correction and bowel rest.

The incidence of ileus (Figure 1) in cardio-thoracic surgical patients is between 1-2% [15]. Ileus is among the more common complications following cardio-thoracic procedures [16]. Various forms of ileus following CTS constitute approximately 10% of GIC [4]. Gastrointestinal motility dysfunction following cardio-thoracic procedures can take a number of clinical manifestations, from isolated gastric distention to prolonged bowel dysfunction [9]. It is important to note 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 [15]. Mandatory perioperative fasting, the effect of anesthetic agents, and decreased patient mobility during immediate postoperative recovery, all contribute to temporary intestinal dysfunction, which in the vast majority of cases regresses automatically after the initiation of enteral intake. In a small proportion of patients the ileus persists past the fourth postoperative day, requiring the use of suppositories, enemas, and pro-motility agents (i.e., metaclopramide, erythromycin) to facilitate clinical resolution [17, 18]. In addition, the use of opioids has to be minimized due to the inhibitory effect of these analgesic agents on bowel motility [19]. The abovementioned measures, in conjunction with close clinical monitoring and normalization of serum electrolyte concentrations, are usually successful in restoring or improving intestinal function [20]. Cases that remain unresponsive are treated with a course of nasogastric suction, which should be continued until the return of bowel function.

4. Colonic pseudo-obstruction

Colonic pseudo-obstruction is a rare, poorly understood surgical complication with multifactorial origins [21]. Characterized by marked colonic distention in the absence of distal obstruction (Figure 2), this condition seems to be associated with the disturbance of the autonomic innervation of the colon [22]. Untreated, colonic pseudo-obstruction leads to cecal over-distention and subsequent perforation, with reported mortality as high as 15-50% [21, 22]. The critical cecal diameter range at which perforation is more likely to occur is between 9-12 centimeters [23]. The two main management modalities for colonic pseudo-obstruction, used alone or in combination, are neostigmine administration and colonoscopic decompression [22, 24]. Depending on whether indicated by the finding of bowel perforation or repeated episodes of pseudo-obstruction, surgical options vary from cecal decompression (i.e., cecostomy) to colonic resection with entero-enterostomy or ostomy creation [25]. In the presence of sepsis with hemodynamic instability, damage control surgery may be justified [26-28].

5. Dysphagia

Dysphagia is a common complaint following cardio-thoracic operations [29]. Undoubtedly, there is an association between history of endotracheal intubation, median sternotomy or thoracotomy incisions, postoperative inflammatory changes in the chest/mediastinum and dysphagia in the CTS patient population. The etiology of postoperative dysphagia is multifactorial, including contributions from gastroesophageal reflux, local tissue trauma from surgery and endotracheal intubation, intraoperative trans-esophageal echocardiography (TEE), and other potential factors such as recurrent/superior laryngeal nerve dysfunction or injury [30]. One of the more interesting contributors to post-CTS dysphagia is the performance of intraoperative TEE, with nearly 8 times greater odds of developing dysphagia among patients who underwent TEE versus those who did not [31].

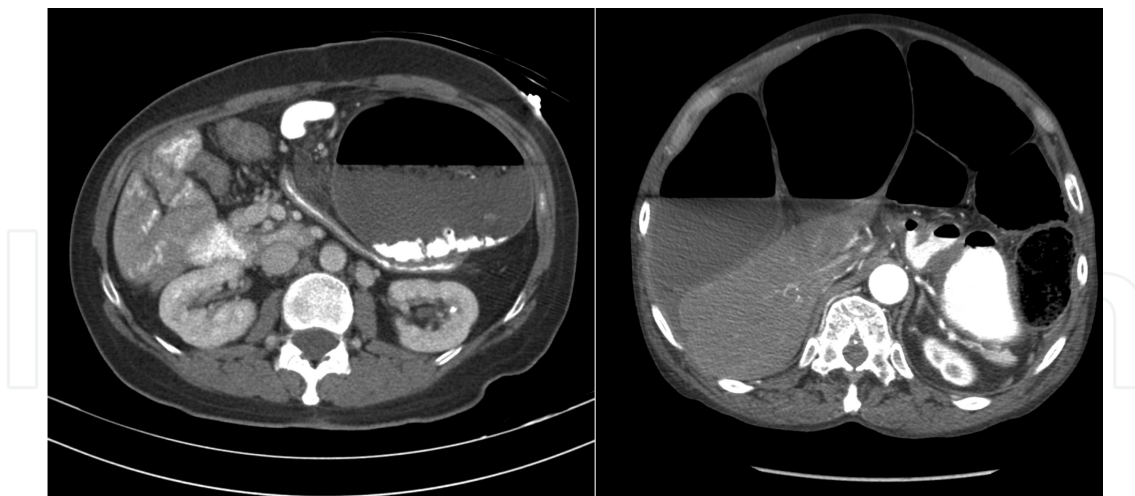


Figure 2. Colonic pseudo-obstruction following cardiac surgery. The first patient (left) presented with increasing abdominal pain/distention, nausea and vomiting following mitral valve replacement. Abdominal CT showed massively dilated left colon with compressive effect on the surrounding small bowel. The pseudo-obstruction resolved with neostigmine therapy. The second patient (right) developed diffuse colonic dilatation following coronary artery bypass grafting. His pseudo-obstruction resolved promptly following emergent colonoscopic decompression.

6. Gastritis and esophagitis

Gastritis and esophagitis are among the more commonly seen gastrointestinal complications in the CTS patient population [32]. In addition to clinical symptoms and history, endoscopy is the most commonly utilized diagnostic modality [33, 34]. Although esophagitis is often associated with gastro-esophageal reflux (GER), the most pressing concern for post-CTS patients with GER is the potential for pulmonary aspiration and associated complications [35]. The etiology of gastritis is multi-factorial, with major contributing elements including mucosal hypoperfusion, previous history of gastric mucosal disorder, and the use of non-steroidal anti-inflammatory drugs [36, 37]. Management includes avoidance of hypotension and hypoperfusion, and aggressive management with H₂-receptor blockers or proton pump inhibitors. For postoperative patients with GER and high pulmonary aspiration risk, the maintenance of 45 degree head-of-bed elevation is an important preventive measure [38].

7. Gastrointestinal hemorrhage

Gastrointestinal bleeding is among the most common GIC following cardio-thoracic procedures. In one study, gastrointestinal bleeding constituted nearly 29% of all GIC-CTS [32]. In general, upper gastrointestinal bleeding occurs more frequently than lower gastrointestinal bleeding, with most hemorrhages (>90%) occurring proximal to the ligament of Treitz [5]. Patients with previous history of peptic ulcer disease may be at higher risk for developing an upper gastrointestinal perforation or hemorrhage following cardiac surgery, although

other traditional risk factors such as *H. pylori* infection alone do not seem contributory [39]. Prolonged mechanical ventilation significantly elevates the risk of upper gastrointestinal bleeding [39]. The two most common etiologies of upper gastrointestinal bleeding are duodenal ulceration and gastric erosion. The appearance of gastric erosions following CTS is likely secondary to systemic hypoperfusion with subsequent development of mucosal ischemia and erosion [40].

The initial step in diagnosis of gastrointestinal bleeding is the placement of a nasogastric (NG) tube and lavage of gastric contents. This aids in determining if the gastrointestinal hemorrhage is proximal to the ligament of Treitz. Medical therapy is attempted first, and includes the administration of H₂-receptor blockers or proton pump inhibitors, red blood cell transfusion, correction of coagulopathy, and temporarily withholding anticoagulation when applicable/possible [41, 42]. If medical management fails, upper endoscopy is the next step in evaluation and treatment of potential bleeding source(s) [43]. Endoscopic attempts aimed at stopping the bleeding by cauterization, vasoconstrictive agent injection, or both are usually effective [42, 43]. In one report, approximately half of the patient with upper gastrointestinal bleeding required upper endoscopy with cauterization to stop the hemorrhage while the other half required surgical intervention to control the bleed [32]. Early surgical intervention if patient fails medical and endoscopic treatment or if significant rebleeding occurs, is recommended. In general, the presence of continued hemodynamic instability, or a pre-determined transfusion threshold (i.e., >4-6 units of packed red blood cells) are utilized as "surgical triggers". Mortality related to gastrointestinal bleeding, even when requiring an operation has decreased over the past two decades.

Lower gastrointestinal bleeding following cardio-thoracic procedures is usually approached according to established clinical algorithms [44]. The first step in management is hemodynamic resuscitation and normalization of coagulation parameters. The bleeding usually stops following these initial maneuvers. If the bleeding does not stop, the next step is the identification of the source of hemorrhage, either endoscopically [45] or by imaging (nuclear scan versus angiography) [46, 47]. In many cases, the bleeding can be controlled endoscopically [48, 49]. Select cases can be treated with endovascular embolization [47]. Surgery should be reserved for refractory cases, with the major determinants for surgery being the failure of non-operative therapies, hemodynamic instability and/or the requirement for transfusion (usually 4-6 units of packed red blood cells) [48, 49].

8. Mesenteric ischemia

Mesenteric ischemia (Figure 3) is a well known complication of CTS that usually occurs within hours to several days after surgery. The gastrointestinal tract is vulnerable to ischemia because it is often unable to acutely compensate for systemic hypotension. Further, due to the potential for persistent vasoconstriction following the initial "low flow" state, gastrointestinal ischemia may continue despite return of hemodynamic stability (i.e., non-occlusive mesenteric ischemia or NOMI). Intestinal ischemia may lead to complications such as

mucosal sloughing, gangrenous changes of the bowel wall, and perforation. Mortality may exceed 65% for patients with acute mesenteric ischemia [8]. Early recognition of signs and symptoms of bowel ischemia and early intervention are integral to successful outcomes and lower mortality rates [50]. One of the earliest signs of mesenteric ischemia is abdominal pain out of proportion to physical examination findings [51]. However, this can be quite difficult to elicit in postoperative CTS patients as many are mechanically ventilated and sedated following surgery. In the setting of high clinical suspicion, sigmoidoscopy or colonoscopy can aid in diagnosis of colonic ischemia [52]. The subsequent sections will discuss post-CTS mesenteric ischemia as divided into two major pathophysiologic types: (a) "low flow state" secondary to systemic hypoperfusion; or (b) thrombo-embolic events.

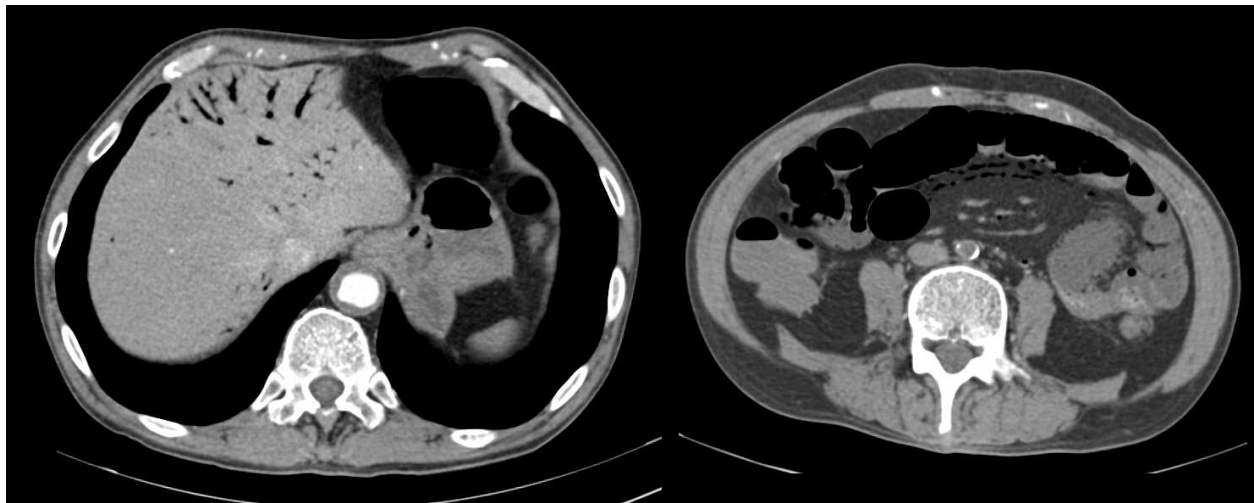


Figure 3. Abdominal CT scan of a patient who developed peritonitis several days after undergoing coronary artery bypass grafting. The study shows diffuse portal venous gas (left) and pneumatosis of the bowel and the mesentery (right). The patient underwent laparotomy with segmental resection of necrotic small bowel. A planned "second-look" laparotomy showed no further bowel necrosis and primary small bowel anastomosis was performed.

9. Ischemia secondary to low flow state

Patients with poor cardiac functional status are at risk for splanchnic hypoperfusion secondary to a number of pre-operative (i.e., pre-existing mesenteric arterial disease), intra-operative (i.e., hypotension/tissue hypoperfusion), and post-operative (i.e., low cardiac output) risk factors. Preoperative presence of conditions such as low left ventricular ejection fraction and peripheral vascular disease have been shown to be significant risks for developing post-operative gastrointestinal ischemia [32].

Intraoperatively, hypovolemia and use of vasoconstrictors can contribute to splanchnic hypoperfusion [53]. Additionally, patients requiring longer cardiopulmonary bypass times may be at greater risk for developing intestinal hypoperfusion [53]. This may be due to the non-pulsatile cardiopulmonary bypass flow characteristics, in conjunction with other factors such as the

associated hemolysis, inflammatory cascade activation, the use of anticoagulation, the presence of hypothermia, and the reduced end-organ perfusion. Further, cardiopulmonary bypass may be associated with increased gastrointestinal permeability and enhanced cytokine release, contributing to microcirculatory dysfunction and mucosal injury [32].

In the postoperative setting, inadequate blood flow to the intestines and subsequent intestinal ischemia/infarction can be associated with hypotension and/or cardiogenic shock [10]. In one study, patients with renal failure (Creatinine >1.4), prior myocardial infarction, and those requiring intra-aortic balloon pump support were at higher risk of developing mesenteric ischemia secondary to "low flow" state [8]. Prolonged mechanical ventilation requiring high positive end-expiratory pressure (PEEP) can also result in hypotension and impaired cardiac output, leading to splanchnic vasoconstriction and hypoperfusion. Furthermore, high PEEP is associated with activation of the renin-angiotensin-aldosterone system and increases in catecholamine levels [54]. This, in turn, results in shunting of blood away from the gastrointestinal system, leading to mismatch between oxygen delivery and demand. Persistent deficit in oxygen delivery then leads to mucosal ischemia and damage. Moreover, during the process of tissue re-perfusion after restoration/normalization of adequate oxygen delivery, the persistent vasoconstrictive state of non-occlusive mesenteric ischemia (NOMI) may be seen [32]. Management of NOMI consists of restoration of adequate circulating intravascular volume, maintenance of adequate cardiac output, and selective angiographic approaches utilizing intra-arterial vasodilating agent infusion therapy [55]. Surgery is reserved for cases requiring resection of necrotic bowel, exploration for suspected perforation, and/or revascularization procedure.

10. Embolic phenomena

Mesenteric ischemia following cardiac surgery results from embolic disease secondary to macrovascular embolism or thrombosis, such as SMA embolus, or microvascular emboli, such as embolic cholesterol "showering" secondary to aortic manipulation. Septic embolization with occlusive phenomena has also been reported in cases of endocarditis following open heart surgery [56]. The size of the embolus may be an important prognostic factor. For example, patients with large vessel emboli may have better outcomes when compared to patients with microvascular or "distal" emboli [8]. High index of suspicion is critical to optimal patient outcomes. If recognized promptly, occlusive emboli to the mesenteric circulation can be treated via either endovascular and/or open surgical approaches, with acceptable success rates [51]. Patients with hypotension, cardiogenic shock, and/or pump failure requiring intra-aortic balloon pump not only are at risk of significant intestinal hypoperfusion, but are also at risk secondary to embolization and thrombus formation which may further exacerbate the original insult to the intestinal tract. Surgical therapy is indicated if the patient develops peritonitis, perforation, sepsis, and/or end-organ failure in the setting of elevated clinical suspicion [57]. Planned or "second look" surgery is warranted if ischemic (but non-necrotic) bowel segments are noted at the conclusion of the initial procedure [58, 59]. Open

abdominal approaches using temporary abdominal coverage with negative pressure wound therapy have been described in such situations [27].

11. Pancreatitis

Acute pancreatitis is relatively uncommon (incidence 1-3%) following cardiopulmonary bypass [15]. Clinically apparent pancreatitis usually occurs slightly later following cardiac surgery than other gastrointestinal complications, 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 high incidence of hyperamylasemia in cardiac surgery patients (>33%) [15], clinical correlation is required before definitive diagnosis of pancreatitis is made.

The severity of pancreatitis ranges from subclinical (i.e., noted only on laboratory values) to severe hemorrhagic, necrotic pancreatitis (seen in <0.5% of patients) (Figure 4) [60]. In one study, nearly 20% of patients who underwent cardiac surgery were found to have evidence of pancreatitis on autopsy [61]. Although the mechanism explaining the development of pancreatitis after cardiac surgery has not been discovered, it has been hypothesized that low flow state, tissue ischemia, gallstone disease, micro-embolization, and history of pre-existing pancreatic disease all contribute to post-CTS acute pancreatitis.

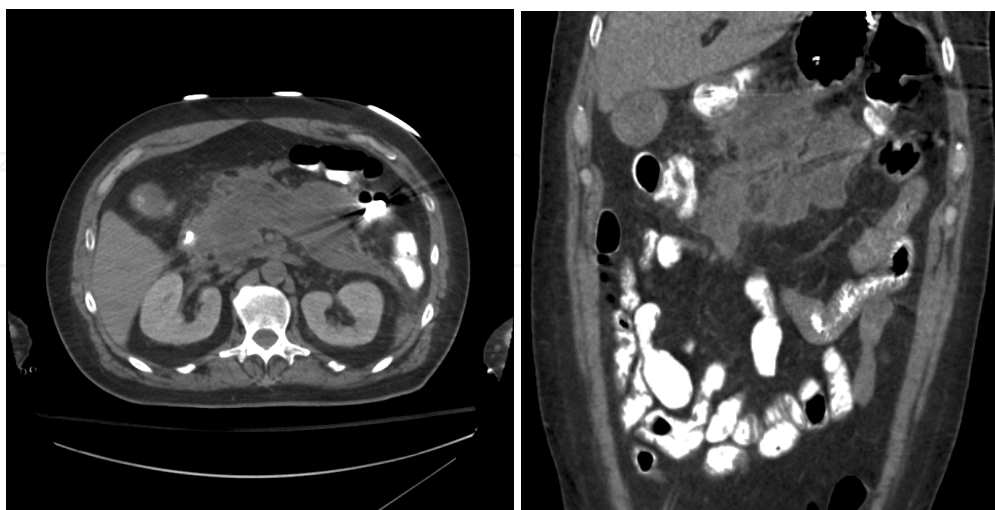


Figure 4. Abdominal CT of a patient who developed acute upper abdominal pain following aortic valve replacement surgery. Representative images of severe necrotizing pancreatitis are shown. Non-operative management resulted in resolution of pancreatitis approximately 2 weeks after the diagnosis was made.

12. Acute cholecystitis

Acute cholecystitis is another commonly seen gastrointestinal complication following CTS (Figure 5). In one study, incidence of acute cholecystitis was approximately 8% among all postoperative gastrointestinal complications [5]. Many cases of acute cholecystitis associated with CTS are termed “acalculous cholecystitis” and are secondary to biliary stasis as a result of multiple factors such as lack of enteral feeding and gallbladder wall ischemia secondary to a “low flow” state. Mortality rates associated with acalculous cholecystitis are significant (>50%) which may reflect the overall poor general health status of patients at risk for this complication [62, 63]. Typical symptoms include right upper quadrant pain and tenderness on examination. However, diagnosis is often delayed secondary to the presence of mechanical ventilation and sedation in significant proportion of patients with acalculous cholecystitis. Patients with acute cholecystitis, diagnosed most often on right upper quadrant ultrasound or cholescintigraphy scan, require surgical intervention or percutaneous cholecystostomy tube placement for treatment of cholecystitis. For poor surgical candidates, percutaneous cholecystostomy can serve as “bridging” therapy that facilitates the patient’s recovery until he or she is ready to undergo cholecystectomy [64].

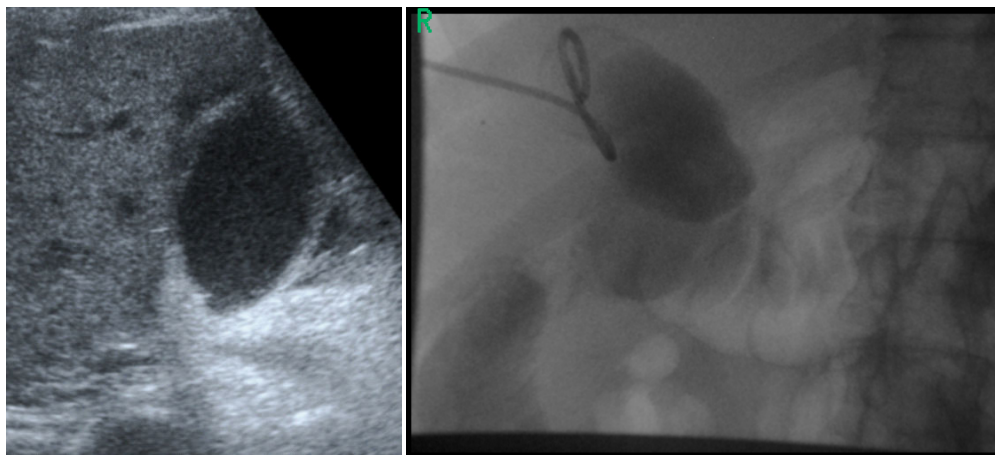


Figure 5. Elderly male patient developed cerebral infarction 2 days after undergoing aortic valve replacement. His recovery was further complicated by acute cholecystitis, as demonstrated by right upper quadrant ultrasound showing distended gallbladder with wall thickening, sludge and pericholecystic fluid (left). His operative risk for cholecystectomy was prohibitive at that time, prompting the placement of percutaneous cholecystostomy (right). Following good functional recovery and hospital discharge, the percutaneous drain was removed and the patient underwent elective laparoscopic cholecystectomy.

13. Gastrointestinal complications unique to transplant recipients and immunosuppression

Immunosuppressive regimens administered to transplant recipients predispose this patient population to elevated risk for bacterial, fungal, parasitic, and viral infections [65]. Within

this broad pathophysiologic spectrum, gastrointestinal infection and associated manifestations feature prominently. While a complete discussion of this topic is beyond the scope of this chapter, we thought it would be important to mention some of the more prominent among these post-transplant sequelae. The list of potential gastrointestinal complications seen after solid organ transplantation is diverse, including cytomegalovirus enteritis [65], herpes simplex virus mucocutaneous manifestations [66], candidal esophagitis [67], *Clostridium difficile* and *Yersinia enterocolitica* infections [4], parasitic (protozoan/metazoan) enteritis [67], and *Helicobacter pylori* infection [68]. Among other post-transplant gastrointestinal complications, organ recipients may be more likely to exhibit diarrhea, luminal ulcerations, perforations, biliary tract complaints, pancreatitis, and gastrointestinal malignancy (i.e., post-transplant lymphoproliferative disorder) [65, 69]. For more detail regarding post-transplant and immunosuppression-related gastrointestinal complications among heart and lung recipients, the reader is referred to more specialized literature on this expansive topic [65, 67, 68].

14. Miscellaneous gastrointestinal and abdominal complications related to cardiac surgery

Among less commonly encountered (and reported) complications of cardiac surgery are those associated with trans-esophageal echocardiography (TEE). Likely under-reported, TEE-related complications in cardiac surgical patients occur in as many as 1.2% of patients [70]. In one series, esophageal and gastric tears were seen within 24 hours of the TEE in 2 patients, with additional gastric ulceration and gastric tear seen within 5 days of the procedure. Moreover, gastric perforations were described presenting between 4-11 days post-TEE. Among the 6 reported cases, 3 required a laparotomy, 2 were treated endoscopically, and 1 patient required transfusion [70].

Epigastric (sub-xiphoid) and chest tube site hernias [71] following cardiac surgery occur in as many as 3-4% of patients following median sternotomy [72]. Another, much less common complications related to the mediastinal tube thoracostomy is superior epigastric artery pseudoaneurysm [73]. Management of these rare conditions is mostly surgical, although minimally symptomatic high-risk surgical patients may be followed with clinical observation.

Due to the growing volume of mechanical cardiac and pulmonary assistive technologies (i.e., ventricular assist devices, intra-aortic balloon pumps, extra-corporeal membrane oxygenation devices), it is important to mention potential gastrointestinal and abdominal complications associated with these devices. Not unexpectedly, the use of cardio-respiratory mechanical assistive devices has been found to be associated with clinically significant abdominal and gastrointestinal complications [32, 74, 75]. For example, extracorporeal membrane oxygenation has been associated with embolic phenomena of the systemic circulation, end-organ ischemia, gastrointestinal hemorrhage, and abdominal compartment syndrome [74, 76-78]. Patients who undergo ventricular assist device placement are also exposed to a number of potential gastrointestinal and abdominal complications, including abdominal infection, bowel injury, acalculous cholecystitis, pancreatitis, various hernias (i.e., incisional,

inguinal, diaphragmatic), peritoneal fluid leaks, and mesenteric ischemia [75, 79-83]. Of note, gastrointestinal hemorrhage has also been reported in patients with ventricular assist devices [84, 85], with higher bleeding rates seen among recipients of non-pulsatile devices as compared to pulsatile devices [86]. There is a trend toward higher mortality among patients receiving ventricular assist devices who experience abdominal complications [75]. Intra-aortic balloon pumps are among known risk factors for gastrointestinal complications following CTS [8, 32]. Some of the reported GIC associated with intra-aortic balloon pump use include gastrointestinal bleeding, bowel ischemia, and pancreatitis [78, 87, 88].

15. Conclusions

Gastrointestinal complications following cardio-thoracic procedures continue to significantly contribute to morbidity and mortality in this patient population. Preventive strategies, coupled with early recognition and aggressive management of GIC-CTS constitute the foundation of the general clinical approach to these complications. Therefore, it is imperative that all practitioners who care for postoperative cardiac and thoracic surgical patients are familiar with the full spectrum of potential gastrointestinal complications in this patient population, as well as with general therapeutic approaches to these complications.

Author details

Jennifer Schwartz¹, David E. Lindsey^{1,2}, Hooman Khabiri³ and Stanislaw P. A. Stawicki^{1,2}

1 Department of Surgery, The Ohio State University College of Medicine, Columbus Ohio, USA

2 Division of Critical Care Trauma and Burn, The Ohio State University College of Medicine, Columbus Ohio, USA

3 Department of Radiology, Section of Interventional Radiology, The Ohio State University College of Medicine, Columbus Ohio, USA

References

- [1] Andersson, B, et al. Gastrointestinal complications after cardiac surgery. *Br J Surg*, (2005). , 326-333.
- [2] Zacharias, A, et al. Predictors of gastrointestinal complications in cardiac surgery. *Tex Heart Inst J*, (2000). , 93-99.

- [3] Negargar, S, et al. Gastrointestinal complications after cardiac surgery. *Journal of Iranian Society of Anesthesiology and Intensive Care*, (2003). , 66-74.
- [4] Geissler, H. J, et al. Incidence and outcome of gastrointestinal complications after cardiopulmonary bypass. *Interact Cardiovasc Thorac Surg*, (2006). , 239-242.
- [5] Yilmaz, A. T, et al. Gastrointestinal complications after cardiac surgery. *Eur J Cardiothorac Surg*, (1996). , 763-767.
- [6] Huddy, S. P, Joyce, W. P, & Pepper, J. R. Gastrointestinal complications in 4473 patients who underwent cardiopulmonary bypass surgery. *Br J Surg*, (1991). , 293-296.
- [7] Lazar, H. L, et al. Gastrointestinal complications following cardiac surgery. *Cardiovasc Surg*, (1995). , 341-344.
- [8] Mangi, A. A, et al. Gastrointestinal complications in patients undergoing heart operation: an analysis of 8709 consecutive cardiac surgical patients. *Ann Surg*, (2005). discussion 901-4., 895-901.
- [9] Halm, M. A. Acute gastrointestinal complications after cardiac surgery. *Am J Crit Care*, (1996). quiz 119-20., 109-118.
- [10] Christenson, J. T, et al. Postoperative visceral hypotension the common cause for gastrointestinal complications after cardiac surgery. *Thorac Cardiovasc Surg*, (1994). , 152-157.
- [11] Franz, W. D. Postoperative abdominal complications after thoracic surgery (author's transl). *Zentralbl Chir*, (1977). , 621-623.
- [12] Mlekodaj, S, & Musialek, W. Acute abdominal complications following thoracic surgery]. *Gruzlica*, (1971). , 541-546.
- [13] Ohri, S. K, et al. Cardiopulmonary bypass impairs small intestinal transport and increases gut permeability. *Ann Thorac Surg*, (1993). , 1080-1086.
- [14] Berger, M. M, et al. Intestinal absorption in patients after cardiac surgery. *Crit Care Med*, (2000). , 2217-2223.
- [15] Akpınar, B, et al. Acute gastrointestinal complications after open heart surgery. *Asian Cardiovasc Thorac Ann*, (2000). , 109-113.
- [16] Simic, O, et al. Abdominal complications after heart surgery interventions]. *Zentralbl Chir*, (1997). , 893-897.
- [17] Miller, F, & Fenzl, T. C. Prolonged ileus with acute spinal cord injury responding to metaclopramide. *Paraplegia*, (1981). , 43-45.
- [18] Korolkiewicz, R. P, & Kuziński, K. Use of erythromycin in prevention or treatment of postoperative ileus. *Urology*, (2008). , 231.

- [19] Kraft, M. D. Methylnaltrexone, a new peripherally acting mu-opioid receptor antagonist being evaluated for the treatment of postoperative ileus. *Expert Opin Investig Drugs*, (2008). , 1365-1377.
- [20] Karangelis, D, et al. Gastrointestinal Complications Following Heart Surgery: An Updated Review. *European Journal of Cardiovascular Medicine*, (2011). , 34-37.
- [21] Apostolakis, E. R, & Thiel, W. Bircks, Acute pseudo-obstruction of the colon (Ogilvie's syndrome) following open heart surgery. *Thorac Cardiovasc Surg*, (1990). , 371-373.
- [22] Vanek, V. W, & Al-salti, M. Acute pseudo-obstruction of the colon (Ogilvie's syndrome). An analysis of 400 cases. *Dis Colon Rectum*, (1986). , 203-210.
- [23] Nanni, G, et al. Ogilvie's syndrome (acute colonic pseudo-obstruction): review of the literature ((October 1948 to March 1980). and report of four additional cases. *Dis Colon Rectum*, 1982. 25(2): , 157-166.
- [24] Connor, F. L. and C. Di Lorenzo, Chronic intestinal pseudo-obstruction: assessment and management. *Gastroenterology*, (2006). Suppl 1): , S29-S36.
- [25] Simic, O, et al. Incidence and prognosis of abdominal complications after cardiopulmonary bypass. *Cardiovasc Surg*, (1999). , 419-424.
- [26] Stawicki, S. P, et al. The concept of damage control: extending the paradigm to emergency general surgery. *Injury*, (2008). , 93-101.
- [27] Smith, B. P, et al. Review of abdominal damage control and open abdomens: focus on gastrointestinal complications. *J Gastrointestin Liver Dis*, (2010). , 425-435.
- [28] Stawicki, S. P, Cipolla, J, & Bria, C. Comparison of open abdomens in non-trauma and trauma patients: a retrospective study. *OPUS 12 Scientist*, (2007). , 1-8.
- [29] Gundry, S. R, et al. Candida esophagitis following cardiac operation and short-term antibiotic prophylaxis. *J Thorac Cardiovasc Surg*, (1980). , 661-668.
- [30] Andersson, B, et al. Gastrointestinal complications after cardiac surgery- improved risk stratification using a new scoring model. *Interact Cardiovasc Thorac Surg*, (2010). , 366-370.
- [31] Ziegelin, M, et al. Does clopidogrel rather than aspirin plus a proton-pump inhibitor reduce the frequency of gastrointestinal complications after cardiac surgery? *Interact Cardiovasc Thorac Surg*, (2007). , 534-537.
- [32] Ancona, D, et al. Determinants of gastrointestinal complications in cardiac surgery. *Tex Heart Inst J*, (2003). , 280-285.
- [33] The role of endoscopy in the management of esophagitis. Guidelines for clinical application. *Gastrointest Endosc*, (1988). Suppl): , 9S.
- [34] Sartori, S, et al. Role of endoscopy in the diagnosis of gastritis. *Medicina (Firenze)*, (1988). , 309-310.

- [35] Russell, G. N, et al. Gastroesophageal reflux and tracheobronchial contamination after cardiac surgery: should a nasogastric tube be routine? *Anesth Analg*, (1996). , 228-232.
- [36] Bennett, J. S, et al. The use of nonsteroidal anti-inflammatory drugs (NSAIDs): a science advisory from the American Heart Association. *Circulation*, (2005). , 1713-1716.
- [37] Yapici, N, & Ince, C. Gastrointestinal complications and its predictors after cardiac surgery. *Turk J Gastroenterol*, (2010). , 3-6.
- [38] Keeley, L. Reducing the risk of ventilator-acquired pneumonia through head of bed elevation. *Nurs Crit Care*, (2007). , 287-294.
- [39] Halm, U, et al. Helicobacter pylori infection: a risk factor for upper gastrointestinal bleeding after cardiac surgery? *Crit Care Med*, (2000). , 110-113.
- [40] Egleston, C. V, et al. Gastrointestinal complications after cardiac surgery. *Ann R Coll Surg Engl*, (1993). , 52-56.
- [41] Lau, J. Y, et al. Effect of intravenous omeprazole on recurrent bleeding after endoscopic treatment of bleeding peptic ulcers. *N Engl J Med*, (2000). , 310-316.
- [42] Sung, J. J, et al. Endoscopic clipping versus injection and thermo-coagulation in the treatment of non-variceal upper gastrointestinal bleeding: a meta-analysis. *Gut*, (2007). , 1364-1373.
- [43] Soehendra, N, Grimm, H, & Stenzel, M. Injection of nonvariceal bleeding lesions of the upper gastrointestinal tract. *Endoscopy*, (1985). , 129-132.
- [44] Barnert, J, & Messmann, H. Diagnosis and management of lower gastrointestinal bleeding. *Nat Rev Gastroenterol Hepatol*, (2009). , 637-646.
- [45] Green, B. T, et al. Urgent colonoscopy for evaluation and management of acute lower gastrointestinal hemorrhage: a randomized controlled trial. *Am J Gastroenterol*, (2005). , 2395-2402.
- [46] Winzelberg, G. G, et al. Radionuclide localization of lower gastrointestinal hemorrhage. *Radiology*, (1981). , 465-469.
- [47] Gordon, R. L, et al. Selective arterial embolization for the control of lower gastrointestinal bleeding. *Am J Surg*, (1997). , 24-28.
- [48] Zuckerman, G. R, & Prakash, C. Acute lower intestinal bleeding. Part II: etiology, therapy, and outcomes. *Gastrointest Endosc*, (1999). , 228-238.
- [49] Zuckerman, G. R, & Prakash, C. Acute lower intestinal bleeding: part I: clinical presentation and diagnosis. *Gastrointest Endosc*, (1998). , 606-617.
- [50] Chang, R. W, Chang, J. B, & Longo, W. E. Update in management of mesenteric ischemia. *World J Gastroenterol*, (2006). , 3243-3247.

- [51] Park, W. M, et al. Contemporary management of acute mesenteric ischemia: Factors associated with survival. *J Vasc Surg*, (2002). , 445-452.
- [52] Brandt, C. P, Piotrowski, J. J, & Alexander, J. J. Flexible sigmoidoscopy. A reliable determinant of colonic ischemia following ruptured abdominal aortic aneurysm. *Surg Endosc*, (1997). , 113-115.
- [53] Moneta, G. L, Misbach, G. A, & Ivey, T. D. Hypoperfusion as a possible factor in the development of gastrointestinal complications after cardiac surgery. *Am J Surg*, (1985). , 648-650.
- [54] Aneman, A, et al. Hemodynamic, sympathetic and angiotensin II responses to PEEP ventilation before and during administration of isoflurane. *Acta Anaesthesiol Scand*, (1997). Pt 1): , 41-48.
- [55] Trompeter, M, et al. Non-occlusive mesenteric ischemia: etiology, diagnosis, and interventional therapy. *Eur Radiol*, (2002). , 1179-1187.
- [56] Misawa, S, et al. Septic embolic occlusion of the superior mesenteric artery induced by mitral valve endocarditis. *Ann Thorac Cardiovasc Surg*, (2011). , 415-417.
- [57] Caleb, M. G. Acute bowel ischemia after coronary bypass surgery--a catastrophic event. *Singapore Med J*, (2001). , 33-37.
- [58] Wang, Y. Z. Staged second-look laparoscopy to evaluate ischemic bowel. *JSLs*, (2009). , 560-563.
- [59] Slutzki, S, et al. The laparoscopic second look for ischemic bowel disease. *Surg Endosc*, (1996). , 729-731.
- [60] Haas, G. S, et al. Acute pancreatitis after cardiopulmonary bypass. *Am J Surg*, (1985). , 508-515.
- [61] Feiner, H. Pancreatitis after cardiac surgery; a morphologic study. *Am J Surg*, (1976). , 684-688.
- [62] Leitman, I. M, et al. Intra-abdominal complications of cardiopulmonary bypass operations. *Surg Gynecol Obstet*, (1987). , 251-254.
- [63] Krasna, M. J, et al. Gastrointestinal complications after cardiac surgery. *Surgery*, (1988). , 773-780.
- [64] Elmunzer, B. J, et al. Percutaneous cholecystostomy as a bridge to definitive endoscopic gallbladder stent placement. *Clin Gastroenterol Hepatol*, (2011). , 18-20.
- [65] Bennett, S. M, et al. The scope and impact of perinatal loss: Current status and future directions. *Professional Psychology-Research and Practice*, (2005). , 180-187.
- [66] Brajtman, S, et al. Providing direction for change: assessing Canadian nursing students learning needs. *Int J Palliat Nurs*, (2007). , 213-221.

- [67] Prendergast, T. J, & Puntillo, K. A. Withdrawal of life support: intensive caring at the end of life. *JAMA*, (2002). , 2732-2740.
- [68] Dimitrakakis, G, Keefe, P. A. O, & Von Oppell, U. O. eComment. Gastrointestinal complications in cardiac surgery. *Interact Cardiovasc Thorac Surg*, (2012). , 627-628.
- [69] Rodriguez, R, et al. Gastrointestinal complications following cardiac surgery: a comprehensive review. *J Card Surg*, (2010). , 188-197.
- [70] Lennon, M. J, et al. Transesophageal echocardiography-related gastrointestinal complications in cardiac surgical patients. *J Cardiothorac Vasc Anesth*, (2005). , 141-145.
- [71] Kesime, E. B, et al. Tube thoracostomy: complications and its management. *Pulm Med*, (2012). , 256878.
- [72] Davidson, B. R, & Bailey, J. S. Repair of incisional hernia after median sternotomy. *Thorax*, (1987). , 549-550.
- [73] Sadat, U, et al. Superior epigastric artery pseudoaneurysm--a rare complication of chest drain insertion in coronary artery bypass grafting. *J Cardiothorac Surg*, (2007). , 21.
- [74] Augustin, P, et al. Abdominal compartment syndrome due to extracorporeal membrane oxygenation in adults. *Ann Thorac Surg*, (2010). , e40-e41.
- [75] Costantini, T. W, Taylor, J. H, & Beilman, G. J. Abdominal complications of ventricular assist device placement. *Surg Infect (Larchmt)*, (2005). , 409-418.
- [76] Park, P. K, et al. Extracorporeal membrane oxygenation (ECMO) in patients with ARDS. (2012 [cited 2012 September 9]. Available from: <http://www.thoracic.org/clinical/critical-care/salvage-therapies-h1n1/pages/ecmo.php>.
- [77] Sarosiek, K, et al. Adult extracorporeal membrane oxygenation and gastrointestinal bleeding from small bowel arteriovenous malformations: a novel treatment using spiral enteroscopy. *J Thorac Cardiovasc Surg*, (2012). , 1221-1222.
- [78] Muthiah, K, et al. Gastrointestinal (GI) hemorrhage in patients undergoing extracorporeal membrane oxygenation (ECMO). (2012 [cited 2012 September 9]. Available from: <http://www.pulsus.com/cddw2012/abs/082.htm>.
- [79] Chatterjee, S, et al. Diaphragmatic hernias associated with ventricular assist devices and heart transplantation. *Ann Thorac Surg*, (2004). , 2111-2114.
- [80] Herman, S. C, et al. Colonic perforation from left ventricular assist device: a rare complication. *Interact Cardiovasc Thorac Surg*, (2010). , 369-370.
- [81] Cipolla, J, et al. Negative pressure wound therapy: Unusual and innovative applications. *OPUS 12 Scientist*, (2008). , 15-29.
- [82] Baradarian, S, et al. Case series: clinical management of persistent mechanical assist device driveline drainage using vacuum-assisted closure therapy. *ASAIO J*, (2006). , 354-356.

- [83] Scherer, M, et al. Extracorporeal membrane oxygenation as perioperative right ventricular support in patients with biventricular failure undergoing left ventricular assist device implantation. *Eur J Cardiothorac Surg*, (2011). discussion 944., 939-944.
- [84] Meyer, M. M, et al. Endoscopic evaluation and management of gastrointestinal bleeding in patients with ventricular assist devices. *Gastroenterol Res Pract*, (2012). , 630483.
- [85] Smith, V, et al. Surgical management of unusual gastrointestinal bleeding and a left ventricular assist device. *Interact Cardiovasc Thorac Surg*, (2010). , 612-613.
- [86] Crow, S, et al. Gastrointestinal bleeding rates in recipients of nonpulsatile and pulsatile left ventricular assist devices. *J Thorac Cardiovasc Surg*, (2009). , 208-215.
- [87] Rivard, J, Vergis, A, & Kassum, D. Case report of visceral ischemia: the "tail" of an intra-aortic balloon pump. *J Thorac Cardiovasc Surg*, (2008). , 1167-1168.
- [88] Rizk, A. B, & Rashkow, A. M. Acute pancreatitis associated with intra-aortic balloon pump placement. *Cathet Cardiovasc Diagn*, (1996). , 363-364.

