

Gastrointestinal Complications and Cardiac Surgery

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Abstract: Gastrointestinal (GI) complications are an uncommon but potentially devastating complication of cardiac surgery. The reported incidence varies between .3% and 5.5% with an associated mortality of .3–87%. A wide range of GI complications are reported with bleeding, mesenteric ischemia, pancreatitis, cholecystitis, and ileus the most common. Ischemia is thought to be the main cause of GI complications with hypoperfusion during cardiac surgery as well as systemic inflammation, hypothermia, drug therapy, and mechanical factors contributing. Several nonischemic mechanisms may contribute to GI complications, including bacterial translocation, adverse drug reactions, and iatrogenic organ injury. Risk factors for GI complications are advanced age (>70 years), reoperation or emergency surgery, comorbidities

(renal disease, respiratory disease, peripheral vascular disease, diabetes mellitus, cardiac failure), perioperative use of an intra-aortic balloon pump or inotrope therapy, prolonged surgery or cardiopulmonary bypass, and postoperative complications. Multiple strategies to reduce the incidence of GI complications exist, including risk stratification scores, targeted inotrope and fluid therapy, drug therapies, and modification of cardiopulmonary bypass. Currently, no single therapy has consistently proven efficacy in reducing GI complications. Timely diagnosis and treatment, while tailored to the specific complication and patient, is essential for optimal management and outcomes in this challenging patient population. **Keywords:** abdominal organs, CPB, complications, outcomes, pathophysiology, perioperative care. *JECT. 2014;46:142–149*

Gastrointestinal (GI) complications after cardiac surgery encompass a broad range of pathologies, ranging from minor GI bleeding to fulminant hepatic failure. Although the overall incidence of these complications is low, significant morbidity and mortality often occur in association with GI complications. The diagnosis of GI complications is often difficult as a result of multiple factors, including altered clinical symptoms and signs in patients, drugs affecting assessment (such as sedatives, neuromuscular-blocking agents, analgesics, and immunosuppressants), and underlying patient comorbidities. The pathogenesis of GI complications is complex and not fully understood and likely multifactorial. Multiple risk factors for complications have been identified, however, and may enable the use of pre-

ventive strategies, prompt early investigation, and allow early identification of complications in the perioperative period. Early identification is important, because early appropriate intervention is critical to optimize outcomes.

This comprehensive, nonsystematic review examines and summarizes the literature to date regarding GI complications after cardiac surgery. A literature search through Medline and PubMed, using the search terms cardiac surgery, coronary artery bypass grafting (CABG), cardiopulmonary bypass, gastrointestinal complications, intra-abdominal complications, and hepatic complications, was performed to collate significant and recent publications, both prospective and retrospective. The findings are summarized and presented as an up-to-date, practical review and guide to the incidence, pathogenesis, risk factors, prevention, and management of GI complications after cardiac surgery.

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INCIDENCE

The incidence of GI complications is variably reported in studies, ranging from .3% to 5.5% (1–8) with an average incidence of approximately 1.2% (8). Reported associated mortality varies even more widely, from .3–87%

(8,9), although average mortality approximates 32% in contemporary studies (8). GI complications are heterogeneous; however, the most common complication reported overall is GI bleeding, accounting for approximately 35% of GI complications (8). Other commonly reported complications include mesenteric ischemia (approximately 14% of GI complications), pancreatitis, cholecystitis, and ileus (3,8). Intestinal obstruction and perforation and hepatic dysfunction are less common (8,9). Rare complications (<2.5% of GI complications) include fulminant hepatic failure, pseudomembranous colitis, peritonitis, and iatrogenic injury to intra-abdominal organs (4,5,8).

PATHOGENESIS

Ischemia is thought to be the primary cause of most GI complications with both splanchnic hypoperfusion and impaired oxygenation implicated in its pathogenesis (10,11).

The splanchnic circulation normally receives 20% of cardiac output and consumes 20% of total body oxygen consumption. Blood is supplied to the abdominal organs by the celiac artery (the liver, stomach, pancreas, duodenum), the superior mesenteric artery (the pancreas, duodenum, jejunum and ileum, ascending and transverse colon), and the inferior mesenteric artery (the descending and sigmoid colon), each of which branches 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 (12). This compensatory mechanism may, however, contribute to splanchnic ischemia if prolonged (such as during cardiac surgery, cardiopulmonary bypass, or cardiogenic shock). The mesenteric supply is usually regulated by resistance arterioles, which dilate in response to a decrease in mean arterial pressure (MAP) or accumulation of metabolites. Blood flow is autoregulated and also redistributed so that preferential flow to the intestinal villi usually occurs. 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 arrhythmia.

Hypoperfusion may be caused by reduced or suboptimal cardiac output, impaired regional blood flow, or inadequate systemic MAP. However, the optimal cardiac output and MAP for splanchnic perfusion remain unclear, and appropriate targets may vary between individual patients or vary for an individual patient over time. There are few studies that directly compare MAP or cardiac output targets with splanchnic blood flow. A small ran-

domized study of 16 patients comparing “normal” MAP targets (60–65 mmHg) with “higher” MAP targets (80–85 mmHg) in patients undergoing CPB showed no differences in splanchnic oxygenation, acid-base status, or cytokine production (13). However, prolonged hypotension, use of inotropes, and low cardiac output state in the perioperative period are consistently associated with increased GI complications (5,8,10). Other animal studies have demonstrated preserved total splanchnic flow but impaired distribution of flow within the gut (particularly with prolonged CPB periods), and alterations in oxygen delivery and consumption during and after CPB (14,15).

Multiple other factors may contribute to developing ischemia by alteration of blood pressure, flow, or oxygen delivery, including systemic inflammation and the systemic inflammatory response syndrome (SIRS), release of inflammatory mediators, nonpulsatile blood flow, hypothermia, drug therapy, and mechanical factors (10,16,17).

Systemic inflammation and the SIRS response occur as a result of surgical stress response, contact with the CPB circuit, mechanical ventilation, and ischemia itself (which may activate and sustain SIRS) along with reperfusion injury. The inflammatory and complement cascades release mediators such as thromboxane A₂ and B₂, leukotrienes, and C5a, which all have vasoconstrictor actions. Cytokine activation is implicated in vascular endothelial dysfunction and damage (10). All these factors contribute to maldistribution of blood flow and impaired mucosal oxygen delivery.

Nonpulsatile blood flow causes renin release and activation of the renin–angiotensin–aldosterone axis with secretion of angiotensin II, a potent vasoconstrictor. Hypothermia is also associated with vasoconstriction and altered regional blood flow and distribution. Vasoactive drugs such as noradrenaline and vasopressin are also associated with splanchnic hypoperfusion (1,8).

The most common type of intestinal ischemia after cardiac surgery is nonocclusive mesenteric ischemia, which occurs as a result of a generalized reduction in splanchnic flow, most likely attributable to factors discussed previously rather than occlusive mesenteric ischemia resulting from arterial emboli or thrombosis (18).

However, mechanical factors that may contribute to ischemia include micro- and macroemboli resulting from air, atheroma, thrombus, or debris and hepatic and GI congestion related to venous cannulae placement. Another proposed mechanism of hypoperfusion is sympathetic nervous system activation (as occurs in the stress response but may be prolonged or sustained by factors such as prolonged mechanical ventilation) (19).

Nonischemic mechanisms of GI complications include bacterial translocation (resulting from altered mucosal barriers and blood flow), adverse drug reactions (e.g., overanticoagulation, amiodarone-induced hepatotoxicity)

Table 1. The independent predictors for major gastrointestinal complications after cardiac surgery (risk factors) and calculation of the Gastrointestinal Complication Score.*

| Variable | Odds Ratio (and 95% confidence intervals) for Major Gastrointestinal Complication | Points Score |
|--|---|--------------|
| Age >80 years | 2.4 (1.0–5.8) | 2.5 |
| Active smoker | 2.3 (1.0–5.5) | 2.5 |
| Preoperative inotrope support | 4.0 (1.4–12) | 4.0 |
| NYHA Class III–IV | 2.0 (1.0–3.8) | 2.0 |
| Cardiopulmonary bypass time >150 minutes | 2.5 (1.3–4.8) | 2.5 |
| Postoperative atrial fibrillation | 2.4 (1.3–4.4) | 2.5 |
| Postoperative heart failure | 3.4 (1.6–7.1) | 3.5 |
| Reoperation resulting from bleeding | 3.6 (1.6–7.9) | 3.5 |
| Postoperative vascular complication | 9.3 (1.8–47) | 9.5 |

*Modified from Filsoufi F, Rahmanian PB, Castillo JG, et al. Predictors and outcome of gastrointestinal complications in patients undergoing cardiac surgery. *Ann Surg.* 2007;246:323–9.

NYHA, New York Heart Association.

(20), pre-existing pathology, and iatrogenic organ injury (e.g., malpositioned surgical drains).

RISK FACTORS

Patients with comorbidities and those with a prolonged or complicated postoperative course are most likely to develop GI complications. Studies have variably reported risk factors, but those consistently identified may be classified as pre-, intra-, or postoperative. Preoperative risk factors include advanced age (>70 years), reoperation, chronic renal failure, peripheral vascular disease, diabetes mellitus, chronic obstructive respiratory disease, gastrointestinal disease pre-existing, congestive heart failure (New York Heart Association [NYHA] Class III or IV), low cardiac output state, and use of inotropic support or intra-aortic balloon pump (IABP) (5,7,8,17). Intraoperative factors include prolonged CPB duration, valvular surgery, emergency surgery, increased blood transfusion, use of IABP, and presence of arrhythmias. Postoperative factors associated are prolonged mechanical ventilation, acute kidney injury, deep sternal wound infection, and postoperative low cardiac output state (4,5,8,11,19,21,22). Several studies have found no difference in the incidence or mortality of GI complications in on-pump versus off-pump surgery (23–28). This is an interesting and important finding, because modification of CPB as a preventive strategy to reduce GI complications has been proposed and frequently investigated with few strategies shown to be clearly effective in reducing incidence or severity of GI complications (12). In fact, the role of CPB in the pathogenesis of GI complications may be much less important than previously proposed with the SIRS response to surgery and anesthesia, pharmacotherapy, and hypothermia more important. This is further discussed in the prevention section of this review.

The Gastrointestinal Complication Score (GICS) is a risk score model specific for GI complications after cardiac surgery. Developed using prospectively collected data from 5593 patients in a single center undergoing cardiac surgical procedures, the model was subsequently validated at the same single center on a further 1031 single center cardiac surgery patients. Receiver operating curves (ROCs) were used to assess the score's predictive ability with a ROC area under curve in the validation group of .83. Of note, there was no significant difference between the GICS ROC and the EuroSCORE ROC in the validation data set. The GICS model uses the following risk factors in calculating a score: age >80 years, active smoker, preoperative inotropic support, NYHA Class III–IV symptoms, CPB duration >150 minutes, postoperative atrial fibrillation, postoperative heart failure, reoperation resulting from bleeding, and postoperative vascular complication. In the validation study, the probability of a GI complication at a GICS 15 or above was >20%, while at a GICS of 5 or below, was only <.4% (27). Table 1 provides the GICS variables, odds ratios, and point values for calculating the total GICS.

PREVENTION

Preoperative risk stratification with scoring systems, or risk factor identification, may allow preventive strategies to be used pre- and intraoperatively as well as prompt earlier investigation, diagnosis, and management of complications postoperatively. It is likely that early detection and intervention can improve both morbidity and mortality related to GI complications.

Preoperative optimization of a hemodynamic state with correction of hypovolemia and anemia and optimization of cardiac output (e.g., inotrope therapy or IABP if

required) are recommended. However, there are currently no large randomized controlled trials to validate this approach. Institutional practice is variable in the management of preoperative anemia with recent recommendations to use iron supplementation and erythropoietin in selected elective patients (28), whereas in some institutions, preoperative transfusion is considered in those requiring nonelective surgery. However, there remains considerable debate around the use of preoperative transfusion, and to date, no minimal preoperative hemoglobin or hematocrit target or threshold is established.

Intraoperative monitoring and maintenance of adequate cardiac output and oxygenation is clearly important; however, as previously outlined, the exact parameters for adequate cardiac output and oxygen delivery are unknown and likely vary between patients. Mucosal ischemia and altered barrier function may occur despite adequate global flow and oxygen delivery. Several methods for monitoring GI perfusion, including measurement of gastric pH, ultrasound of blood flow in hepatic or mesenteric vessels, measurement of intestinal transport functions, measurement of transsplanchnic changes in interleukin (IL)-6, IL-10, pH, and lactate have been described (13,29). These methods are impractical for routine use and interpretation in the operating room and are not used clinically. Specific intraoperative strategies are discussed subsequently.

Vasoactive Drug Therapies

Several drug therapies have been associated in small studies with benefit in reducing GI complications, but findings have not been consistent, and evidence from large randomized controlled trials (RCTs) is still lacking.

Aspirin treatment within 48 hours postoperatively has been associated with a reduction in both the incidence and mortality of GI complications in CABG surgery (30).

Milrinone infusion in patients undergoing CABG resulted in reduced gastric mucosal acidosis and lower inflammatory marker and endotoxin levels in a small RCT (31), but clinically significant effects have yet to be demonstrated.

Vasoconstrictor therapy, particularly norepinephrine and epinephrine, has been associated with increases in MAP and systemic blood flow but with decreases in splanchnic flow and redistribution of blood flow from the splanchnic to the systemic circulation (32). It is likely that these changes are attributable at least in part to the mesenteric afferent arteriolar constriction described earlier, which is marked in response to systemic vasoconstrictors, and overrides normal autoregulation. Interestingly, phenylephrine has been demonstrated to increase MAP but not systemic blood flow and to have no redistribution effect on splanchnic blood flow (32). Dopamine and dobutamine can both increase cardiac output; however,

neither have shown clear benefit in terms of GI perfusion, and recent studies suggest potential harm associated with the use of either of these therapies (12,33). Vasopressin has been demonstrated to have adverse effects on gastric mucosal perfusion in patients with septic shock, and in critically ill patients, and on colonic mucosal perfusion during CPB in animal studies (12,34,35). These findings re-emphasize that maintenance of adequate MAP is not the only, nor most important, strategy for ensuring adequate perfusion and that blood flow and oxygen delivery may be globally adequate but regionally inadequate and both of these factors may be worsened by therapies measured only by a global parameter such as MAP or cardiac index.

Modification of Cardiopulmonary Bypass

Multiple strategies have been proposed, focusing on maintaining adequate perfusion, avoiding hemodilution and severe anemia, the use of pulsatile flow, the use of filters and strategies to reduce emboli and finally, and avoidance of CPB with off-pump surgery.

Maintenance of adequate cardiac output and oxygenation seems prudent, but as previously discussed, the thresholds for adequacy may be difficult to measure and are difficult to define for any individual patient. Minimizing the use of pure vasoconstrictors is recommended with augmentation through inotropes suggested if support for MAP targets is required.

Hemodilution resulting in a hematocrit $<.25$ is associated with reduced oxygen delivery and in some studies with increased mortality, although these findings are not consistent (36). The use of smaller bypass circuit components and retrograde autologous priming may help with blood conservation and reduction of anemia and transfusion requirement and is a reasonable approach, supported by limited evidence, but with no evidence demonstrating harm (28).

Debate has been extensive over the role of pulsatile versus nonpulsatile flow in CPB. In some trials, pulsatile flow has resulted in improved mucosal oxygenation and perfusion, but in others, no differences were demonstrated (15,37). Clinical outcomes with pulsatile versus nonpulsatile flow have not differed.

Minimizing the risk of emboli and subsequent hypoperfusion is recommended. Atheroemboli may be reduced or avoided by careful selection of aortic cannulation site, echo assessment of the aorta, avoidance of excess manipulation of the aorta, and avoidance of IABP in those with severe atheroma. Meticulous deairing techniques along with CPB filters are warranted to reduce gaseous emboli, and CPB circuit filters have been demonstrated to reduce micro- and macrogaseous emboli, but none of these strategies has proven successful in reducing GI complications.

Table 2. The modification strategies for conduct of cardiopulmonary bypass that may reduce gastrointestinal complications.*

| |
|--|
| Maintain adequate cardiac index and mean arterial pressure |
| Avoid vasoconstrictors |
| Augment circulation with inodilators, inotropes, vasodilators |
| Minimize gaseous microemboli and atheroemboli (cannula site selection, epiaortic scanning, avoidance of excess aortic manipulation, meticulous deairing) |
| Avoid severe anemia |
| Biocompatible surface modification of cardiopulmonary bypass (CPB) circuit |
| Minimize surface area and volume of CPB circuit |
| Pulsatile flow |
| Antithrombotic therapy (aspirin) |

*Modified from reference Hessel EA. Abdominal organ injury after cardiac surgery. *Semin Cardiothorac Anesth.* 2004;8:243–63.

Off-pump CABG surgery avoids the CPB circuit entirely and is associated with reduced bleeding complications but not a reduction in GI complications (12).

Other unproven strategies include biocompatible surfaces in the CPB circuit, minimization of circuit surface area and volume, and reduction of blood–air interfaces.

Table 2 summarizes the modification of CPB strategies that may reduce GI complications (12).

Other Strategies

Pharmacotherapy with gastric acid suppression using a proton pump inhibitor (PPI) is currently recommended to reduce the risk of peptic and duodenal ulcers and GI bleeding and has been shown to be superior to no prophylaxis and to use of a histamine receptor antagonist in preventing these complications (38). Of note, a recent meta-analysis and systematic review of the literature regarding gastric ulcer prophylaxis and acid suppression, which did not include patients undergoing cardiac surgery, associated the use of PPIs with increased hospital-acquired pneumonia (39). A practical approach to gastric acid suppression perioperatively is for the initial high-risk perioperative period to include treatment with a PPI with discontinuation of prophylaxis once normal oral intake is re-established for patients.

DIAGNOSIS

In the patient undergoing cardiac surgery, sedatives, analgesics, neuromuscular blockade, and vasoactive drugs may alter clinical symptoms and signs of GI complications. Multiorgan failure, metabolic derangement, and cardiovascular instability are nonspecific signs and may make clinical assessment even more difficult. 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 his-

tory and presentation. Overall, a low threshold for investigation in those patients with nonroutine postoperative progress is recommended.

Initial investigations will include biochemistry and hematology blood analysis with serum lactate, glucose, liver function tests (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. Delayed diagnosis and management is associated with worse outcome (5,8).

The incidence, clinical presentation, and suggested diagnostic tests for the most common GI complications are summarized in Table 3.

MANAGEMENT

Determined by the specific condition, management is usually identical to standard management for the specific condition. It is important to emphasize that treatment should not be withheld as a result of recent cardiac surgery, and prompt diagnosis and intervention may be associated with improved outcomes.

The routine management for the most common GI complications is summarized in Table 4.

SUMMARY

This review summarizes the literature to date regarding GI complications after cardiac surgery. GI complications are relatively uncommon after cardiac surgery but occur overall in approximately 1.2% of patients undergoing cardiac surgery, a rate that has not decreased significantly in recent years. These complications remain associated with a very high burden of morbidity and mortality despite advances in diagnostic modalities and perioperative care. The diagnosis remains difficult, because symptoms and signs are often subtle, or nonspecific, and this commonly leads to delay in definitive diagnosis and treatment. Preventive strategies are largely unproven currently and have focused on single areas of the surgical, anesthesia, perfusion, or critical care management. The pathogenesis of GI complications however is complex and multifactorial and it is likely any effective preventive strategies will involve mediation or attenuation of several interrelated factors such as the SIRS response and loss of autoregulation during and after CPB. In the future, further large high-quality trials are needed to establish efficacy of preventive strategies. Currently, strategies for prevention center around optimization of hemodynamic and respiratory parameters,

Table 3. Summary of gastrointestinal complications, common clinical presentations, and suggested investigations.

| Complication | Clinical Presentation | Suggested Investigations and Expected Results |
|---|--|--|
| Hemorrhage 30–35% of GI complications Upper GIT: duodenal or gastric ulceration | Altered blood/melena per rectum Hematochezia Hemodynamic instability | Hemoglobin (↓) Lactate dehydrogenase (↑) Endoscopy (evidence or site of bleeding identified and potentially treated) |
| Lower GIT: diverticulitis, AV malformations Mesenteric ischemia 14–20% complications Occlusive: emboli or thrombus | Shock Abdominal pain and distension Intolerance enteral nutrition GI bleeding | Complete blood count (leucocytosis) Lactate (↑) Abdominal radiograph (distended bowel, thickened bowel, evidence of ileus) Computed tomography mesenteric angiography (global impairment of perfusion) Colonoscopy (ischemic bowel) Laparotomy (ischemic bowel) |
| Nonocclusive (NOMI): hypoperfusion | | Abdominal radiograph (peritoneal air) Computed tomography scan abdomen (peritoneal air, collection) |
| Peptic ulcer perforation 6–8% complications | Abdominal pain, distension Peritonism | Amylase (↑) Lipase (↑) Computed tomography scan abdomen (pancreatic inflammation, free fluid, necrosis) |
| Pancreatitis .5–1.0% of all patients NB: hyperamylasemia common (25–35% of patients postcardiac surgery) | Shock Epigastric and back pain Nausea and vomiting Abdominal distension | |
| Cholecystitis 6–11% of complications | SIRS Shock Often 10–15 days postsurgery Right upper quadrant pain | Liver function tests deranged (elevated) Ultrasound biliary tract (thickened gallbladder and common bile duct, ±gallstones) Computed tomography scan abdomen Laparoscopy |
| Calculous or acalculous | Fever Leucocytosis Systemic inflammatory response syndrome Shock | |
| Hepatic dysfunction | May be asymptomatic | Elevated liver function tests (most commonly hyperbilirubinemia, transaminitis) |
| Transient up to 40% patients | Jaundice | Abdominal ultrasound (exclude obstruction, thrombosis, collections) |
| Hepatic failure <.4% of patients | | Hepatitis serology screen (exclude as cause) |

GI, gastrointestinal; GIT, gastrointestinal tract; AV, atrioventricular; SIRS, systemic inflammatory response syndrome.

Table 4. Suggested management for common gastrointestinal complications after cardiac surgery.

| | |
|--------------------------|--|
| Hemorrhage | Intravenous fluid resuscitation (to restore euolemia) Correction of coagulopathy Proton pump inhibitors (consider infusion) Endoscopy (enables diagnosis and treatment) Clipping or sclerotherapy to bleeding vessels Angiography and intervention/surgery (if lower gastrointestinal tract bleeding with arteriovenous malformation or diverticulitis) |
| Mesenteric ischemia | Intravenous fluid resuscitation (to restore euolemia) Circulatory support (inotropes or vasopressors) Antibiotic therapy Laparotomy and bowel resection |
| Peptic ulcer perforation | Intravenous fluid resuscitation (to restore euolemia) Proton pump inhibitor high dose Laparotomy—vagotomy and oversew of ulcer or resection |
| Pancreatitis | Enteral rest and nasogastric drainage Intravenous fluid resuscitation (to restore euolemia) Postpyloric feeding or intravenous nutrition Supportive therapy Analgesia Occasionally percutaneous drainage or surgical treatment (rare) |
| Cholecystitis | Surgery (calculous) with cholecystectomy Antibiotics ± percutaneous drainage (acalculous) |
| Hepatic dysfunction | Stop potential hepatotoxins Supportive |

metabolic state, and supportive care along with minimization of vasoconstrictor drug therapy and potential sources of perfusion perturbation (such as emboli). However, important questions remain regarding the appropriate hemodynamic targets and how best to measure these. Further randomized trials are needed to elucidate whether a “pressure” or “flow” target are important in preventing complications and to establish appropriate targets for hemoglobin and hematocrit as well as the best method to achieve these targets. Critical to improving outcomes is the prompt diagnosis and definitive treatment when GI complications do occur, with full recovery possible after even the most serious of GI complications (such as mesenteric ischemia) but high mortality rates associated with conservative management or failure to diagnose complications. Ideally, the development of more sensitive and specific diagnostic tests (such as GI ischemia biomarkers) will allow earlier definitive treatment and therefore improved outcomes, and this is another area warranting further research and investigation.

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.

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