ISSN: 2474 - 9206

Review Article

Journal of Anesthesia & Pain Medicine

Gastrointestinal Complications Post Cardiac Surgery: A Narrative Review

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Submitted: 23 Mar 2022; **Accepted:** 28 Mar 2022; **Published:** 11 April 2022

Citation: *Naveen Yadav (2022) Gastrointestinal Complications Post Cardiac Surgery: A Narrative Review. J Anesth Pain Med 7(2): 67-71.*

Abstract

Gastrointestinal (GI) complications are one of the most important complications after cardiac surgery which are associated with high mortality and morbidity. The complications can range form benign paralytic ileus to the deadly mesenteric ischemia which has mortality up to 40%. There are several factors which have been associated with these complications. These factors can range from preoperative, intraoperative, and postoperative. The most significant of them are chronic renal failure, high baseline serum creatinine, smoking, cardiopulmonary bypass time greater than 120 min. This narrative review will help understand the incidence, characteristics, outcome, and treatment of the GI complications after cardiac surgery. This review will also help understand the risk stratification models and ways to prevent these complications.

Keywords: Gastrointestinal Complications, Risk Stratification, Cardiopulmonary Bypass

Introduction

Gastrointestinal complications (GIC) following cardiac surgery have been reported since the early days. GIC after cardiac surgery encompass a broad range of pathologies which can range from minor GI bleeding to mesenteric ischemia. Although they occur infrequently, GI events are serious complications which can carry high mortality and morbidity rates. Gastrointestinal complications are still a major problem in clinical practice [1]. post-operatively, most of the cardiac surgery patient are sedated, mechanically ventilated and mostly partially unresponsive which masks the signs and symptoms of GI complications. By the time GIC are identified, they have already reached a significant severity, and this can lead to the significant morbidity and mortality in this patient population. To improve the clinical outcome in these patients, a significant attention needs to be paid on the prevention, early diagnosis, and treatment of GI complications after cardiac surgery. Gastrointestinal complications associated with cardiac surgery are a serious problem for the treating team. These complications can be very severe and lead to prolonged hospitalization and increased costs. In last decade, there has been significant advances in the perioperative and postoperative management, which may have impacted the incidence of GICs. These changes have definitely raised the concerns about the validity of the previously reported incidences of GIC. In recent times, there has been an increase in complex cardiac surgery, this is on the pretext of recent operative techniques and perioperative care which has resulted in a sicker and older patient getting operated. This has also led to many reoperations being done [2, 3]. This has also increased the incidence of GI complications in these subsets of patients.

Incidence

The incidence of the reported GI complications in the last few decades is between 0.41 to 3.7% [4]. In Australia, Saxena et al. reported a 3% incidence of GI complications in patients between age group between 80- and 89-year-old as compared to 1.3% in patients <80 years old after aortic valve replacement [5]. Viana et al published their study in 2013 looking for patients operated for cardiac surgery between 2001 to the year 2011 looking specifically at the incidence, patient profile, and outcomes of GI complications after cardiac surgery in Australia. However, the complexity of cases operated in cardiac surgery and the operative skills have increased significantly in a decade. Although, there were no further studies that looked for GI complications in cardiac surgery patients in the Australian population. The incidence of GI complications in our study was 2.16% as compared to 1.1% in Viana et al.

Pathophysiology

There has been a growing consensus suggesting that visceral hypoperfusion during and after cardiovascular operations with cardiopulmonary bypass seems to be the most common cause for abdominal complications [3]. It can cause injury to the mucosa, leads to the damage the organs, initiates the vicious circle, and this serves as a trigger for the development of multiple organ failure. This all can contribute to the increased mortality from these complications [6, 7].

The gastrointestinal tract, which is supposed to be the main site for systemic inflammation, makes it prone to the damage from both hypoperfusion and inflammatory insults [8]. Since most of these patients have a long history of congestive heart failure with

poor cardiac performance, their ability to tolerate or compensate for ischemia and anoxemia is less.

The episodes of transient hypotension and with indispensable hemodilution during CPB results in shunting blood away from the splanchnic area towards areas of higher priority like the brain. These factors also lead to the hepatic arterial, portal vein, and gut mucosal microcirculation flow get reduced [9]. CPB also leads to the activation of the inflammatory cells, and this stimulates these cells to release cytokines, which can promote systemic fluid sequestration and splanchnic oedema formation. These all factors contribute to bowel microvascular barrier injury which induces the bacterial invasion and following endotoxin transfusion [10, 11].

The surgical stress response itself leads to the systemic inflammation and the SIRS response. In addition, the CPB circuit, mechanical ventilation and ischemia may activate and help sustain the SIRS response. The inflammatory and complement cascade release mediators such as thromboxane A2 and B2, leukotrienes, and C5a, which all have vasoconstrictor actions. The activation of the cytokine is implicated in the vascular endothelial dysfunction and damage. The non-pulsatile blood flow also leads to the release of renin and activation of the renin-angiotensin-aldosterone axis with secretion of angiotensin II, which is a potent vasoconstrictor. Hypothermia can lead to the vasoconstriction, and lead to altered region blood flow. In addition, the use of vasoconstricting agents is also known to worsen peripheral tissue perfusion. Moreover, the use of drugs such as muscle relaxants, analgesics, and sedatives in anaesthesia can cause decreased bowel mobility.

However, there are mechanical factors which may contribute to ischemia which includes micro- and macroemboli which results from atheroma, air, thrombus, or debris. There is also GI and hepatic congestion related to venous cannulae placement. There is another proposed mechanism of hypoperfusion which is sympathetic nervous system activation (as occurs in the stress response but may be prolonged or sustained by factors such as prolonged mechanical ventilation) [12]. Nonischemic mechanisms of GI complications which include bacterial translocation (resulting from altered mucosal barriers and blood flow), adverse drug reactions (e.g., overanticoagulation, amiodarone-induced hepatotoxicity), iatrogenic organ injury (e.g., malpositioned surgical drains), and pre-existing pathology [13].

Specific Complications

The above pathophysiology can lead to significant change in the abdominal viscera and leads to the complications which can be fatal. The specific complications related to gastrointestinal systems are described below:

GI Bleeding

Previous data suggests that the bleeding form the gastro-intestinal tract is the most common GIC observed during post operative cardiac surgery [14-17]. The two most common aetiologies of upper GI bleeding are gastro-duodenal ulcer and erosive gastritis. Shin and Abah identified in their study that 21 out of 61 patients with GI complications post-cardiac surgery had significant bleeds [18]. Most of the cases in their study had upper GI bleed as compared to lower GI bleed. (13 vs 5) Three of the patients had both upper and lower GI bleed. These patients are usually managed by the endoscopy and CT angiogram guided embolization. However, in spite of the above treatment modalities, the mortality can be as high as 38%. Most of the patients undergoing cardiac surgery take warfarin and aspirin, both of these drugs increase the chance of bleeding. Ischemia can impair the gastric and duodenal mucosal defence mechanisms causing stress ulceration. The extent of ischemia is dependent upon the duration of CPB and cross clamp time.

Mesenteric Ischemia

Mesenteric ischemia is one of the most severe forms of GIC. It occurs in a total of 18% of all visceral complications observed in open cardiac surgery. The incidence of acute mesenteric ischemia ranges from 0.06% to 0.2%, although it is associated with significant high mortality 46-100% [19]. Schoots et al has reported in their study that the overall mortality in non-surgically treated patients is almost 95% [20]. It has been postulated that the presence of the coronary artery disease is associated with the vasculopathy in the mesenteric bed. This predisposes to the patient to have more ischemia in a peri-operative period. However, the exact pathophysiological mechanism has not been understood. Thus, confirms that the acute mesenteric ischemia after the cardiac surgery is often due to non-occlusive mesenteric ischemia rather than embolic disease [20, 21]. The signs and symptoms for mesenteric ischemia are usually non-specific or can be obscured by sedation or analgesia. The mesenteric ischemia can present as severe shock, abdominal pain, distension, and intolerance to the enteral nutrition. The investigation which is usually done to identify mesenteric ischemia is abdominal radiograph, CT mesenteric angiography and colonoscopy. The treatment for mesenteric ischemia will require intravenous fluid resuscitation, circulatory support, and antibiotic therapy. Most of the time patients with mesenteric ischemia will require laparotomy and bowel resection. Pang et al found that the survivors of the complications had surgical intervention earlier compared to the non-survivors [22].

Acute Cholecystitis

Acute cholecystitis (AC) is another complication which is seen with cardiac surgery. It usually accounts for the 3% to 8% among all GICs and is often calculous [23]. Rady et al published a study in which AC was diagnosed a median of 26 days (11-41 days) of the cardiac surgery [24]. The precise pathophysiology of the AC is not known, and the early diagnosis is always a challenge. Symptoms like fever and right upper quadrant pain are highly nonspecific especially in the setting of post operative cardiac surgery. Several mechanisms have been suggested which can cause acute cholecystitis which include visceral hypoperfusion of the gallbladder, endotoxemia, increased viscosity of bile because of the stasis, and overproduction of inflammatory mediators are some of them [25]. The liver function tests are usually deranged, and patient may require an US or CT scan depending upon the preference by the physician.

Paralytic Ileus

Paralytic ileus is usually defined by its abdominal distension and

typical radiographic changes. Paralytic ileus is sometimes caused by the colonic-pseudo-obstruction and is rare. Paralytic ileus is poorly understood surgical complication with multifactorial origins [25]. This condition is associated with the disturbance of the autonomic innervation of the colon [26]. If untreated, the colonic pseudo-obstruction can lead to caecal over-distension and possible perforation which usually carries a high mortality [26, 27]. The critical caecal diameter range at which perforation is more likely to occur is between 9-12 centimetres [28]. Neostigmine, and colonic decompression are the two main modalities for the management of colonic obstruction [27, 29]. They can be both used as combination. Paralytic ileus can lead to high mortality and should not be taken lightly. It should be investigated and treated early.

Perforation

Perforated duodenal or gastric ulcer post-cardiac surgery accounts for approximately 6-8 % of all GI complications with a reported mortality of around 38% [30]. The clinical presentation of perforation is abdominal pain, peritonism, and distension, The suggested investigation required to diagnose the perforation is to look for peritoneal air on the abdominal Xray or CT scan of the abdomen. The initial treatment required for the perforation is intravenous fluid resuscitation to restore euvolemia. The literature on adult critical care medicine suggests the use of proton pump inhibitors for prophylaxis of gastric or duodenal stress ulcers for high risks patients is usually appropriate [31].

Hepatic Dysfunction

Hepatic dysfunction maybe asymptomatic. Transient jaundice maybe present in the 40% of the patients and hepatic failure can be seen <0.4% of patients. Clues to the pathogenesis of liver injury following cardiopulmonary bypass may be due to ischemia hepatitis [32]. In some previous reports, these patients were found to have more frequently elevated central venous pressure [32, 33]. The incidence of liver failure post cardiac surgery in the international literature is 2.2% of all GI complications and 0.026% of all cardiac operations [30].

Management of deranged liver physiology involves interventions to improve the liver perfusion and, removal of hepatotoxic drugs. An abdominal ultrasound to assess for biliary obstruction, a laboratory evaluation for an underlying hematologic and infectious cause, portal venous thrombosis or fluid collection or abscess should also be looked at and specific treatment is initiated. There is also need to stop the exposure to the potential hepatotoxins.

Preoperative Risk Factors

The preoperative factors which increase the increased likelihood of individuals to gastrointestinal complications following cardiac surgery are advanced age (Age >65 years), diabetes, higher illness severity, renal dysfunction, and arrythmias (mostly atrial fibrillation), emergency surgery, and preoperative elevated creatinine levels. Many studies have identified various risk factors trying to elucidate the correlation with GI complication. A few authors have used multivariate analysis, but with little concordance except for age, renal dysfunction, low EF, prolonged ventilation, and NYHA functional class [34, 35]. Aithoussa et al has shown that smoking, diabetes, obesity, hypertension, and hyperlipidaemia occurred more frequently in patients with the GI complications. They have also shown that the significantly more patients with GI complications had a history of previous gastric ulcer, peripheral vascular disease (PVD), worse preoperative renal function, and anaemia.

Intraoperative Risk Factors

Most of the previous data have shown that the intraoperative factors associated with increased gastrointestinal complications following cardiac surgery include significant vasopressor inotropic support, increased cardiopulmonary bypass time, prolonged duration of surgery, prolonged aortic cross-clamp time, and use of intra-aortic balloon pump [15, 17, 35, 36]. McSweeny et al have shown that the hypotension and duration of the CPB have been implicated as the strongest predictors of an adverse GI outcome. In this study, the seven percent of patient with GI complications had CPB > 100 min as compared to the four percent of patients with no GI complications. The data has shown that where 30 % of patients with GI complication had CPB > 120 min as compared to the 15% in cohort with no GI complication.

Postoperative Risk Factors

Post-operative factors such as need for inotropic agents, requirement of ventilation for more than 24 hours, and stroke were more common in patients with GI complications. and it corresponds well with previous reports [1, 14-16]. Filsoufi et al has shown that age, myocardial infarction, hemodynamic stability, renal and hepatic failure as the independent predictors of the GI complications. Aithoussa et al suggested that severe infection is a risk factor for GI complication in univariate analysis but not multivariate analysis [37]. Several studies have shown that the septicaemia, pneumonia and multiorgan failure were also more prevalent in patients with GI complications.

Clinical Outcomes

Studies have shown that in terms of resource utilisation perspective, the patient with GI complication had a longer stay `in ICU and hospital as compared to the comparator group. The McSweeny et al has shown that the adverse GI outcome more than doubled the mean post-surgical stay to 3.5 week and also doubled the median hospital length of stay [36]. It has also been seen that; the median length of the ICU stay is three times in GI complication group as compared to the no GI complication group. Similarly, the median length of hospital stay is doubled in the GI complication group as compared to the non- GI complication group.

Prevention

Because of the significant morbidity and mortality associated with the GI complications after cardiac surgery are rare, it is important to recognize them early and then treat them as appropriate.Risk Stratification Models The preoperative risk stratification is an important part of any surgical procedure, and it aims to reduce the mortality and morbidity by identifying and assessing factors which may contribute to poor prognostic outcomes. The European System for Cardiac Operative Risk Evaluation (EuroSCORE) is used to predict in-hospital mortality, direct costs, postoperative complications, and postoperative length of stay in

hospital following cardiac surgery [38]. It has been seen that the EuroSCORE can successfully predict some specific complications post-cardiac surgery, such as sepsis, renal failure, and respiratory failure. However, it has been seen that it fails to predict other major complications such as stroke, myocardial infarction, and GI complications [38]. A newer scoring model, named the GI complication score (GICS), was proposed by Andersson et al [39]. and aimed to predict complications arising specifically from abdominal viscera following cardiac surgery. The study was able to successfully validate this model and GICS showed to have good predictive ability for GI complications [39]. The risk stratification models are effective, cheap, and easy to use. However, there are certain key limitations of them as the physicians may become over-reliant on their use and they may miss broader risk factors not considered in the original design of the model.

Perioperative Preventive Measures

There has been ongoing debate as to whether routine use of prophylactic proton pump inhibitors (PPIs) can reduce GI complications, particularly stress ulcers and haemorrhagic events. Shin and Abah in their study were marginally in favour of acid suppression drugs due to the high morbidity and mortality associated with GI bleeding following cardiac surgery [18]. However, these uses of these drugs have also been demonstrated to increase the risk of hospital-acquired pneumonia [18]. Bhat et al. have argued against the use of acid suppression drugs as PPIs and found that PPIs does not significantly alter the incidence of postoperative haemorrhagic events. As it has been seen that the formation of stress ulcers is thought to be due to ischemia rather than pH related and splanchnic hypoperfusion [40]. Because of above conflicting views, further studies will be required in order to recognise the efficacy of prophylactic PPI therapy prior to cardiac surgery. Allen et al in their review article has suggested that there are number of potential preventive measures including preoperative correction of a hemodynamic state with careful attention to hypovolemia, anaemia, and cardiac output. In addition, the intraoperative monitoring and maintenance of adequate cardiac output; monitoring of GI perfusion via ultrasound of mesenteric blood flow and measurements of intestinal transport functions can help reduce the GI complications [41]. However, it has been seen that postoperatively there are only limited number of measures which can be taken to reduce GI complications. One study has shown that early aspirin therapy post-coronary bypass surgery to be linked to a 62% reduction in the incidence of bowel infarction. This has also caused a reduction in mortality and other ischemic complications involving the GI tract [42]. The modifications to CPB may help to reduce the likelihoods of GI complications occurring as it appears to play a crucial role in the pathophysiology behind these complications. Zhang et al. in their study analysed 206 patients who had GI complications following CPB surgery from 2000 to 2007 and compared to 206 matched control patients. In their study, they have suggested that GI complications could be reduced by maintaining a higher perfusion pressure and shortening the time on CPB and ventilation [43].

Conclusion

GI complications following cardiac surgery are relatively uncommon but are associated with high mortality. There is pre-operative, intra-operative, and post-operative factors which can lead to high incidence of GI Complication. The early identification of GI complications following cardiac surgery will help to facilitate pre-operative assessment and optimisation in high-risk patients. There has been the development of a risk stratification mode which may require more validation, and timely post-operative intervention, which can lead to the reduced GI complication.

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