Is the gut safe? Rare postoperative gastrointestinal complications following cardiac surgery

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Received: 14 November 2020 Revised: 28 May 2021 Accepted: 2 June 2021 Published: 16 December 2020

The Egyptian Journal of Cardiothoracic Anesthesia 2021, 15:92–94

The incidence of gastrointestinal (GI) complications following cardiac surgeries is 0.3-5.5%, with 13-87% mortality, and GI bleeding occurs commonly [1]. Splanchnic circulation receives 20% of cardiac output with 20% of total oxygen consumption [2]. It perfuses the gut, is a reservoir of 800 ml blood, and increases total systemic vascular resistance by 25% during compensation [3]. Loss of autoregulation causing transient hypoperfusion is tolerated [2]; if prolonged or severe, ischemia initiating extensive inflammation and multi organ dysfunction syndrome (MODS) occur [3]. Perfusion pressure maintained at 50-70 mmHg serves as an indicator of organ perfusion, although the ideal cardiac output and mean arterial pressure (MAP) for splanchnic circulation remains ill-defined [2,4]. Two patients with adverse outcome reinforced the need for morbidity-specific risk stratification perioperatively.

Case Report

A 67 year male, type II diabetic on oral hypoglycemic agents, hypertensive, smoker with 5 pack years, presented with extensive anterior wall ischemia and non sustained ventricular tachycardia. Coronary angiogram: left main thrombus causing 90% stenosis, patient was thrombolysed. Cardiogenic shock ensued requiring dobutamine infusion 5 µg/kg/min and Intra aortic balloon pump (IABP). Emergency coronary artery bypass grafting (CABG) with three venous grafts was planned. cardio pulmonary bypass (CPB) duration was 120 min, and MAP was maintained above 70 mmHg throughout. Intraoperative esophageal trans assessment echocardiography (TEE) was not performed. He was uneventfully weaned from CPB with adrenaline, dobutamine, and IABP support. Postoperative arterial blood gas (ABG) showed metabolic acidosis with lactate of 6.2, corrected over 24 h with intravenous fluids. Hemodynamic support was continued up to postoperative day (POD) 3 and weaned. Perioperatively, 20U of blood and products were transfused based on the complete blood count and coagulation profile. On post-extubation day 2, abdominal distension developed, decompressed with nasogastric tube (NG), and was monitored. On POD 5, he had melena and collapsed and was resuscitated. GIscopy result was normal, and computed tomography of the abdomen suggestive of toxic megacolon, which was managed conservatively with antibiotics vancomycin and metronidazole. He had two episodes of hemodynamically stable atrial fibrillation on POD 7 and 9 and was treated with infusion of amiodarone and electrolyte correction. He succumbed to MODS on POD 17 despite CPR (Fig. 1).

A 75-year-old female patient had COPD, severe aortic stenosis, ejection fraction (EF) 36%, past history of thalamic infarct without residual deficits, and underwent aortic valve replacement (AVR). CPB duration was 90 min. MAP of 65–70 mmHg was maintained throughout. Aorta was not assessed for plaques during TEE examination. She was weaned with dobutamine, adrenaline, and levosimendan infusions after receiving internal DC shocks, with a blood pressure of 130/84 mmHg. Postoperative ABG was normal with lactate 3.3 and base deficit –2.6. She was extubated on POD 2, with EF 40%. She required

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dobutamine up to POD 3. No postoperative atrial fibrillation was noticed. She was icteric on POD 5, thought as hemolytic; deranged liver function tests (LFT), normal peripheral smear, and reticulocyte hepatic etiology. count favored Computed tomography of the abdomen suggested acalculous cholecystitis. Anticoagulants were withheld owing to increasing trend of International Normalized Ratio (INR) and activated partial thromboplastin time (APTT). LFT was monitored daily and was managed conservatively. On POD 9, the patient desaturated, became unresponsive, and succumbed despite 20 cycles of CPR, probably owing to pulmonary embolism (Fig. 2).

Figure 1



Diffuse dilatation of colon, ileum, filled, with air. Transverse colon \sim 9 cm. Stomach collapsed.

Figure 2



No calcified gallstones. Gall bladder over distended (3.9 cm anteroposterior), abnormally enhancing wall, suggestive of acalculous cholecystitis.

Mc Sweeny *et al.* [5] studied preoperative risk for GI complications after CPB, concluding that adverse GI outcomes possibly portend myocardial infarction, stroke, renal failure, and increased postoperative ICU stay, with mortality of 6.5 times greater. GI risk stratification was not done in the above cases; hence, complications were unanticipated, delaying diagnosis and management.

Nonmodifiable risk factors, delayed diagnosis due to sedation altering clinical signs, difficulty in shifting for imaging, no single effective therapy, and reluctance in resubmitting patient for surgery pose challenges [2,3].

Increased age, acuteness at presentation, and advanced disease with multiple comorbidities in present population require morbidity-specific risk stratification rather than mortality scores used earlier [5].

Often, perioperative risk is ascertained for organs forgetting the gut; hence, protective strategies overlook the gut. A bedside postoperative diagnostic tool [the gastro intestinal complication score (GICS)], including risk factors such as age more than 80, smoker, preoperative ionotropic support, NYHA III and IV, CPB more than 150 min, postoperative heart failure, reoperation, fibrillation, atrial and vascular complication, has been formulated by Andersson et al. [6]. Each risk factor is assigned points compiling the score. A GICS more than 15 indicates probability of more than 20% of GI complications, and a GICS less than 5 indicates probability of less than 4%. Retrospectively, GICS of the first patient was 8.5 and for the second patient was 5.5. The EuroSCORE, a widely used cardiac surgical mortality score, was compared with GICS for predicting GI complications, and the GICS proved to be better [6]. GICS augments surgical decision making [5].

Risk of clostridium difficile infection after cardiac surgery increases with temporary circulatory support and use of more than 8 packed red blood cell transfusion (PRBC) or 5 fresh frozen plasma (FFP), highlighting gut ischemia owing to low-output states [7]. These risk factors were superadded to the GICS of 8.5 in our patient. Toxic megacolon was a clinical and radiological diagnosis, and further evaluation of stool was not done.

Ampullary constriction by narcotics, increased viscosity of bile, and greater wall tension cause hypoperfusion of gall bladder mucosa, leading to acalculous cholecystitis [8]. Mucosal injury leads to bacterial invasion. Our

Variables	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 min	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

Table 1 The independent predictors for major gastrointestinal complications after cardiac surgery (risk factors) and calculation of the gastrointestinal complication score [9]

patient had a stroke previously, suggesting visceral atherosclerosis predisposing to hypoperfusion. Low perioperative cardiac output aggravated hypoperfusion.

Both patients were managed conservatively owing to the reluctance in resubmitting for surgery, which probably contributed to adverse outcome apart from delayed diagnosis due to lack of GI-specific risk stratification.

Multiple perioperative strategies exist for prevention of GICs and can be incorporated with awareness of risk factors. Detailed history and examination reveal pointers for further evaluation. Morbidity-specific risk stratification scores enable close surveillance and prompt early, correct diagnosis and treatment. The gut is not safe but can be perioperatively protected by vigilance and early intervention in high-risk patients (Table 1).

Acknowledgements

Dr. Laji Samuel, Head of Department, Department of Anesthesia, Bangalore Baptist Hospital, Hebbal, Bangalore, and Dr. Varghese Zachariah, Professor, Department of Anesthesia, Bangalore Baptist Hospital, Hebbal, Bangalore.

Earlier published/ presented: presented as e-poster in IACTACON2020 on 09/02/2020.

Criteria for inclusion of authors: Dr. Sarkar M. Eunice, the anesthesiologist for the case, and Dr. Kini M. Satish, the surgeon for the case.

Declaration: The manuscript has been read and approved by all the authors, the requirements for authorship as stated have been met, and each author believes that the manuscript represents honest work. The authors alone are responsible for the contents and writing of the manuscript.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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