

Gastro-intestinal complications after open heart surgery.

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Abstract

Objectives: Gastrointestinal complications (GIC) are serious consequences after open heart cardiopulmonary bypass (CPB), and associated with high mortality rate. The aim of the present study was to identify the risk factor for this problem in order to optimize medical management before its occurrence.

Patients and methods: We retrospectively analyzed data of 2056 adult patients who underwent open heart surgery between 1994 and 2012. Among them 35 (1.7%) developed GIC in postoperative period. Preoperative, intra-operative and postoperative data were compared into two groups: with GIC (n=35) and those without GIC (n=2021).

Results: The overall mortality rate was higher in the GIC group (48.5% vs. 5.6%, p=0.0001). The most common GIC was upper gastrointestinal bleeding (57.1%), but the lethal GIC was mesenteric ischemia (100%). Univariate analysis identified many predictor factors of GIC but multivariate analysis revealed previous gastric ulcer (OR=7.4, CI=95%, p=0.001), postoperative renal insufficiency (OR=7.5, CI=95%, p=0.006) as significant risk factors for development of GIC.

Conclusion: Our multivariate logistic regression analysis found that risk factors of GIC were: previous gastric ulcer and postoperative renal insufficiency.

Keywords: Cardiac surgery, Risk factors, Gastrointestinal complications

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Introduction

Gastro-intestinal complications (GIC) are one of the serious extra-cardiac events following open heart surgery. These complications are frequently reported in high risk patients. The reported incidence varies from 0.2-14% to 5.5% [1-4]. Although rarely seen, they cause significant mortality and morbidity. The mortality rate varies widely. It ranges from 13.9% to 100% in various series [5-7]. Visceral hypoperfusion during the perioperative period is the main recognized factor that leads to systemic inflammatory response syndrome (SIRS) [8,9]. Some methods that have been suggested to improve splanchnic perfusion and oxygenation are used in various surgical centers in order to reduce abdominal complications [10].

Cardiac surgery itself is a risk factor for intra-abdominal organ injury [8]. It has been established that various factors are combined in this setting, when the patient is fully sedated, the symptoms may be masked by postoperative analgesia and the diagnosis is not well easy. GICs increase hospital costs resulting from specific treatment and prolonged ICU stay. Consequently, the clinician must be vigilant in such patients in order to make early diagnosis. Numerous previous studies have investigated many factors closely associated with these complications. In this study, we used retrospectively collected data to determine the incidence of GICs and assess risk factors for them after open heart surgery. We sought also to study outcomes in patients who developed GICs compared with controls.

Patients and Methods

We retrospectively analyzed 2056 consecutive adult patients underwent open heart surgery with cardiopulmonary bypass at author's surgical center between January 1994 and December 2012. Operative procedures included coronary artery bypass grafting (CABG), isolated valve surgery, combined CABG and valve surgery, aortic root replacement, surgical excision of intracardiac tumors and surgical correction of adult congenital heart defect. Off-pump CABG and thoracic or thoracoabdominal aortic surgery procedures were excluded. The study was approved by our local Ethic Committee. Data were obtained from existing medical records, operative reports and surgical pathology reports.

Informations collected on all patients included demographic characteristics, and cardiovascular history. Surgical procedures included valve repair or valve replacement, coronary artery bypass graft (CABG), CABG combined with valve surgery, or other concomitant procedures. Preoperative comorbidities collected included: smoking, diabetes mellitus (DM), peripheral vascular disease (PVD), renal insufficiency, chronic obstructive pulmonary disease (COPD), history of gastro-duodenal ulcer (GDU). Routine protocol of the anesthesia and the cardiovascular surgery was applied in all patients.

In order to reduce the anxiety, all patients received Hydroxyzine 1 mg/kg as a premedicant. The choice of the anesthetic

method depends on left ventricular function and whether early extubation was suitable. Anesthetic induction used Fentanyl 5 µg/kg and Propofol 3 mg/kg and Cisatracurium 0.15 ng/kg. The maintenance of anesthesia was performed with Propofol 6-12 mg/kg/h combined with Isoflurane 1-2%. Preoperatively, all patients were premedicated with Midazolam 10 mg associated with Morphine.

Anesthesia was induced with Fentanyl 130 µg/kg and Vancuronium for muscle relaxation. Anesthesia was maintained with standard dose of Morphine 10 to 20 µg/kg/h and Vancuronium infusion for basal sedation and analgesia. For stress gastro-duodenal ulcer prophylaxis, patients were medicated with H2-receptor antagonists from the first postoperative day for up one month between 1994 and 2000. After 2000, all patients received proton pump inhibitor (PPI). Cephalosporin was used as antibiotic prophylaxis.

All operations were performed through a median sternotomy. Cardiopulmonary bypass (CPB) was achieved with a fiber membrane oxygenation. Systemic mild hypothermia (32-34°C) was used and nonpulsatile blood flow was maintained at 2.5 l/min/m² and the perfusion pressure was kept between 60 and 70 mmHg. Before 2000, myocardial preservation was achieved by using cold (4°C) crystalloid cardioplegic solution (St Thomas solution) combined with topical myocardial cooling with iced saline solution. Since 2000, we routinely used cold blood cardioplegia. Cardioplegia was reinfused every 30 minutes during the cross clamping period.

Low cardiac output syndrome (LOS) was defined when the cardiac index was under 2 l/min/m² when there was hemodynamic instability requiring excessive doses of inotropic drugs or intra-aortic balloon pump (IABP). Prolonged ventilator time was defined as the need of mechanical respiratory support more than 48 hours for any reason.

Diagnosis of gastro-intestinal complications (GICs)

GICs were retrospectively reviewed and classified according to Anderson et al. [2]. Only patients with GICs that arose within 30 days of their cardiac surgery were enrolled in this study. GICs were documented by clinical, appropriate biochemical, hematologic, radiologic or endoscopic investigations. Complications encountered included upper gastro-duodenal bleeding, mesenteric ischemia, pancreatitis, cholecystitis and liver dysfunction. Hematemesis or melena which caused a 2 g or more decrease in hemoglobin was defined as GIB. Pancreatitis evidenced by abdominal pain, tenderness, nausea, vomiting and elevated urinary or serum amylase level. The diagnosis was supported by computed tomography CT scan.

Acute cholecystitis was diagnosed on temperature above 38.5°C or below 35°C associated with nausea, vomiting, right upper quadrant abdominal pain echography or CT scan supported the diagnosis. Mesenteric ischemia was considered in case with abdominal pain, distension, nausea, vomiting, diffuse peritoneal irritation, unexplained diarrhea, metabolic acidosis. The diagnosis was made by colonoscopy or confirmed at laparotomy. Liver failure manifested by progressive cholestatic jaundice, rising liver function test values. The authors defined

the groups as patients who developed GICs and controls as patients who did not.

Statistical analysis

Statistical analysis was performed with SPSS version 19.0 (SPSS Chicago Illinois, IL). Normally distributed – continuous variables are presented as mean ± SD and otherwise as median ± interquartile range (IQR). Categorical variables are shown as the percentage of the sample. Univariate analysis for categorical variables was conducted using either the χ^2 test or the Fisher's exact test. We analyzed continuous variables with either the unpaired Student's t test or the non-parametric Mann-Whitney test depending on the normality. Multivariate analysis was used to identify factors that significantly influenced the risk of GICs. These factors were entered in a stepwise logistic regression analysis to identify independent variables associated with postoperative GICs. A probability level of less than 0.05 was considered significant Table 1.

Results

A total of 2056 patients underwent open cardiac surgery under CPB in the author's surgical unit. 1312 valve surgery (63.8%), 585 coronary artery bypass graft (28.5%) and the remaining cases (8.4%) included combined procedures 75 (3.6%), aortic surgery 24 (1.1%), congenital defect 60 (2.9%). Among these patients, gastro-intestinal complications (GICs) were seen in 35 patients, an incidence of 1.7%. The overall mortality rate was 48.5% vs. 5.6% in patients without GICs ($p < 0.0001$). Five patients required surgical treatment and medical management was applied in the remaining patients. Clinical characteristics, comorbidities, operative and postoperative data were summarized in Tables 1 and 2.

Table 1: Patients clinical characteristics and comorbidities.

Variable	Without GICs (n = 2021)	With GICs (n = 35)	p
Age (years)	47.8 ± 14	55.9 ± 13.4	0.001
Sex (F/M)	773 / 1248 (38.2%)	5 / 30 (14.2%)	0.004
BMI (kg/m ²)	24.5 ± 4.4	25 ± 3.6	0.22
Smoking	702 (34.7%)	18 (51.4%)	0.041
Diabetes Mellitus	388 (16.7%)	14 (40%)	0.002
Hypertension	375 (16.5%)	15 (42.8%)	0.0001
Hypercholesterolemia	287 (14.1%)	11 (31.4%)	0.004
COPD	133 (6.5%)	4 (11.4%)	0.29
CRF	114 (5.6%)	8 (22.8%)	0.001
CVA	84 (4.1%)	0 (0%)	0.4
Obesity	322 (15.9%)	10 (28.5%)	0.044
Previous ulcer / or gastritis	80 (3.9%)	7 (20%)	0.001
NYHA III-IV	910 (45%)	13 (37.1%)	0.34
CHF	173 (8.5%)	6 (17.1%)	0.11
Atrial fibrillation	573 (2.8%)	5 (14.2%)	0.057
CTI	0.55 ± 0.07	0.56 ± 0.06	0.91
Anemia	177 (8.7%)	7 (20%)	0.032
Peripheral arterial disease	141 (6.9%)	7 (20%)	0.01
LVEF	56.7 ± 12.3	55.9 ± 12	0.72
Euroscore	2.85 ± 2.86	5.03 ± 4.5	0.009
Valve surgery	1295 (64%)	17 (48.5%)	0.051
Coronary surgery	574 (28.4%)	13 (37.1%)	0.42
Aortic surgery	24 (1.18%)	0 (0%)	0.34

Types of GIC were summarized in Table 3. The most common complication was upper gastro-intestinal bleeding (UGIB), representing 57.1% (n=20) despite the routine use of stress ulcer prophylaxis. Of the 20 patients with bleeding, 2 required surgery because of massive hemorrhage. Therapeutic endoscopy was applied in six patients. Hemostasis was achieved by medical management alone (blood red transfusion + intravenous histamine 2 receptor blockers or proton pump inhibitor (PPI) in 12 patients. 18 patients underwent endoscopic investigation. Two patients were not investigated by endoscopy because their hemodynamic conditions were worse. Of the 18 patients who underwent endoscopic exploration, the following diagnosis were established: duodenal ulcer n=6, gastric ulcer n=4, gastric + duodenal ulcer n=3, ulcero-hemorrhagic esophagitis n=2, erosive gastritis n=3. The mean interval between cardiac surgery and onset of gastro-intestinal bleeding was 13 ± 5.5 days. Seven patients had a history of previous gastro-duodenal ulcer. The mortality rate in those patients was 20% (n=4 deaths).

Six patients developed intestinal ischemia (17.1%). In 3 patients the diagnosis was established by colonoscopy, in 3 others cases mesenteric ischemia was documented at autopsy, because death preceded diagnosis. All those patients underwent coronary artery bypass graft. Mesenteric ischemia occurred on average mediane 3 days after the operation (range 2 to 17.25 days). Two patients required colectomy for necrotic bowel. Six patients had liver failure, the diagnosis was made 8.6 ± 3.2 days after the cardiac surgery (range 5 day to 11 days). Acute pancreatitis was developed in 2 patients (1 day and 4 days).

Table 2: Operative and postoperative patient data.

Variables	Without GICs	With GICs	P value
Non elective surgery	93 (4.6%)	6 (17.1%)	0.006
CPB time	99.9 ± 41.3	125 ± 74	0.001
Cross clamping time	66.4 ± 30.9	79.48 ± 56.5	0.018
Time of surgery (min)	205 ± 61	239.5 ± 81.8	0.007
MV (hours)	8 (6 – 18)	18 (9 – 96)	< 0.0001
MV > 48 hours	128 (6.3%)	15 (42.8%)	< 0.0001
ICU stay (days)	13 ± 12.7	18.4 ± 10.2	0.031
Need for inotropic drugs	263 (13%)	17 (48.5%)	< 0.0001
IABP	100 (4.9%)	6 (17.1%)	0.008
LOS	190 (9.4%)	15 (42.8%)	< 0.0001
Reexploration for bleeding	81 (4%)	3 (8.5%)	0.17
ARF	125 (6.2%)	19 (54.2%)	< 0.0001
Acute cerebrovascular accident	23 (1.1%)	2 (5.7%)	0.065
Infection	137 (6.7%)	11 (31.4%)	< 0.0001
Hemodialysis	28 (1.3%)	6 (17.1%)	< 0.0001
Transfusion	704 (34.8%)	25 (71.4%)	< 0.0001
MOF	71 (3.5%)	14 (40%)	< 0.0001
30-days mortality	115 (5.6%)	17 (48.5%)	< 0.0001
Lactates	1.23 ± 0.04	4.24 ± 4.4	< 0.001

Table 3: Type and characteristics of GICs after open heart surgery.

Variables	No of GIC	Time to diagnosis	No of deaths
UGI bleeding	20 (57.1%)	13 ± 5.5 day	4
Intestinal ischemia	6 (17.1%)	3 (2-17.2)	6
Acute pancreatitis	2 (5.7%)	—	1
Acute cholecystitis	1 (2.8%)	25 days	1
Liver failure	6 (17.1%)	8.6 ± 3.2 day	5

Acute cholecystitis was rare, it occurred too late in one patient (25th day after surgery). The patient required emergency cholecystectomy but he died within 2 days of operation because of severe sepsis and multi-organ failure. The most lethal gastro-intestinal complication was intestinal ischemia (100%).

Parameters that emerged as predictors of GICs on univariate analysis are summarized in Table 4. Patients with GICs were older than patients without GICs. Women were less represented in the group with GICs (14.2 vs. 38.2%, p=0.007).

Smoking, diabetes, obesity, hypertension and hyperlipidemia occurred more frequently in the patients with GICs. Significantly, more patients with GICs had a history of previous gastric ulcer, peripheral vascular disease (PVD), worse preoperative renal function and anemia. A higher number of no elective cardiac surgery was observed in patients with GICs. Mean duration of cardiopulmonary bypass and aortic cross clamp was significantly prolonged in patients who developed GICs. Mechanical ventilation and ICU stay were found to be significantly prolonged in the same group.

The need for IABP, use of inotropic support transfusion were associated with GICs. LOS, pulmonary infection, deep sterna infection, acute renal failure were also more prevalent in patients with GICs. Multivariate analysis revealed previous gastric ulcer (OR=7.4; 95% CI=2.28-24.5) and postoperative acute renal failure (ARF) (OR=7.5; 95% CI=1.79-31.8) as significant risk factors for development of gastro-intestinal events Table 5.

Discussion

Gastro-intestinal complications are relatively rare after cardiac surgery with an incidence ranging from 0.2% to 5.5% [1,4].

Table 4: Univariate risk factors for GICs after cardiac surgery.

Variables	Without GICs N = 2021	With GICs N = 35	P value
Age	47.8 ± 14	55.9 ± 13.4	0.001
Sex female	773 (38.2%)	5 (14.2%)	0.007
Smoking	702 (34.7%)	18 (51.4%) 48 hours	0.045
Diabetes Mellitus	388 (16.7%)	14 (40%)	0.003
Hypertension	375 (16.5%)	15 (42.8%)	0.001
Hyperlipidemia	287 (14.1%)	11 (31.4%)	0.006
Anemia	177 (8.7%)	7 (20%)	0.026
Peripheral vascular disease	141 (6.9%)	7 (20%)	0.011
CRF	114 (5.6%)	8 (22.8%)	0.0001
Previous gastric ulcer	80 (3.9%)	7 (20%)	0.0001
CPB time	99.9 ± 41.3	125 ± 74	0.001
Cross clamp time	66.4 ± 30.9	79.48 ± 56.5	0.018
Time of surgery	205.7 ± 61	239 ± 81.8	0.007
Mechanical ventilation	8 (6 – 18)	18 (9 – 96)	0.0001
MV ≥ 48 hours	128 (6.3%)	15 (42.8%)	0.0001
ICU stay (hours)	46 (24 – 48)	44 (72 – 168)	0.0001
Need for inotropic drug	263 (13%)	17 (42.8%)	0.0001
LOS	190 (9.4%)	15 (42.8%)	0.0001
Infection	137 (6.7%)	11 (31.4%)	0.0001
Obesity	322 (15.9%)	10 (28.5%)	0.049
Euroscore	2 (1 – 4)	3 (1 – 9)	0.0001
No elective surgery	93 (4.6%)	6 (17.1%)	0.002
IABP	101 (5%)	6 (17.1%)	0.008

Table 5: Multivariate predictors of GICs after cardiac surgery.

Variables	Odds Ratio	95% CI	p value
Previous gastric ulcer	7.4	2.28 – 24.5	0.001
Postoperative renal failure	7.5	1.79 – 31.8	0.006

But it carries high mortality when they occur. The average mortality in various series of patients experiencing GIC was 33% (range 13% - 87%) [5]. Deaths from GIC accounted for about 14% (2.5% - 40%) of all deaths after cardiac surgery [10,11]. Our present series had a 1.7% incidence of GIC and a 48.5% mortality rate, both of which are consistent with existing reports.

GIC are largely attributed to splanchnic ischemia which plays a key role in the initiation and perpetuation of the systemic inflammatory response syndrome (SIRS) that often follows cardiac surgery under CPB. There are conflicting opinions about the rate of CPB as a principal cause of adverse GI events. Many, have suggested that beating heart surgery could substantially minimize the risk of digestive complications. According to the Rajah [11] finding, the risk of GIC is seven times higher in on-pump compared to off-pump group.

On recent prospective randomized study, Chrome et al. [12] did not find any differences of total GIC between the off-pump and on-pump group. The same results are reported by Poirier [13]. The effect of CPB and the peri-operative period on splanchnic perfusion have been reviewed by some authors [14-18]. They demonstrated that pHi is lower during and after CPB in patients experiencing GIC. In our series, mean lactate concentration during CPB was higher in GIC group (p<0.001) compared to control group. Many have found this phenomenon to be associated with an adverse outcome [19,20].

The splanchnic circulation plays an important defensive role in hypovolemia and during low output syndrome. Splanchnic vasoconstriction accounts for about 25% of the increase in total systemic vascular resistance and results in autotransfusion of about 15% of the blood volume. The same events occur during CPB [20-22]. In low-flow states, splanchnic blood flow falls promptly, but when systemic flow recovers, splanchnic hypoperfusion is usually well tolerated. However, when it is severe or prolonged, it may proceed to splanchnic ischemia that can lead to visceral organ damage [20]. Besides contributing to GIC, splanchnic ischemia may play another important role in morbidity during postcardiac surgery by causing wide spread inflammation. This process contributes to multi-organ disorders and functional changes.

To our knowledge, hemorrhage from upper gastro-intestinal tract is the most common GIC observed during postoperative cardiac surgery [23-27]. It accounts for about 31% of visceral complications after cardiac surgery. Interestingly, in numerous published papers, we noted that GIC especially, UGIB did not decline through the years despite constant improvement in medical management. The two most common aetiologies of UGIB are gastro-duodenal ulcer and erosive gastritis [5,28-32]. In our series, it accounts 57.1%. Characteristics of patients with UGIB such as incidence, mortality, onset of occurring, history of previous ulcer, aetiologies of bleed and medical approach correspond well to the previous reports [33].

Intestinal ischemia is one of the most severe GIC. It occurs for about 18% of all visceral complications observed in open cardiac surgery. Incidence of acute mesenteric ischemia (AMI) range from 0.06% to 0.2%, but associated with extremely high mortality 46-100% [33-36]. In our series, 5/6 patients who developed AMI underwent coronary artery bypass graft. All patients died because of delayed diagnosis and the salvage is uncommon. Schoots [34] reported an overall mortality rate of about 95% for non-surgically treated patients.

It is possible that presence of coronary artery disease is associated with vasculopathy in mesenteric bed, thus potentially predisposing a patient to more ischemia in peri-operative period. Exact pathophysiological mechanism is not understood. Thus confirms that AMI after cardiac procedure most often is due to a non-occlusive mesenteric ischemia (NOMI) rather than embolic disease [35-37].

Theoretically, the avoidance of CPB should diminish the risk of GIC. Unfortunately 2 previous studies have failed to demonstrate this protective effect [38,39]. At present, no laboratory test is available for accurately establishing or eliminating the diagnosis [40]. Our data indicates that UGIB remains the main GIC observed. In contrast, in a more recent studies, Mangi et al. [41] and Filsoofi et al. [24] reported AMI as the dominant GIC observed.

The precise incidence of pancreatitis following cardiac surgery is unclear (1 – 3%) [42,43] because the severity of damages ranges from subclinical to hemorrhagic form, or necrotic pancreatitis. Previous studies found an incidence between 11% - 20% in autopsy [44,45]. When it occurs overtly, it is associated with high mortality ranging from 28% to 50% [46]. The pathogenesis of acute pancreatitis after cardiac surgery is still a matter debate.

According to the literature reports, some factors may contribute to pancreatitis such: corticosteroid, chlorothiazid diuretics, hypercalcemia and complement activation [47]. In our serie, one of the two patients who developed acute pancreatitis had a fulminant form and died 3 days after. Rose [47] reported 14 cases of severe pancreatitis, six of them who developed acute fulminant pancreatitis died with 21 days.

Acute cholecystitis (AC) is another commonly seen GIC following cardiac surgery. It accounts approximately 3% to 8% among all GICs and is often acalculous [5,48]. In a study published by Rady [49] AC was diagnosed a median of 26 days (11-41 days) of cardiac surgery corresponding well with our series. The precise pathophysiology of AC is still unclear. Visceral hypoperfusion of gallbladder, increased viscosity and lithogenicity of bile secondary to stasis, endotoxemia, and overproduction of inflammatory mediators have been suggested as mechanisms for AC [50].

Hepatic failure is relatively rare after cardiac surgery (0.1 – 1.1%) [51]. Clinically, hepatic failure usually occurs slightly later after hyperbilirubinemia and is associated with a very high mortality, about 74%. Clues to the pathogenesis of liver injury following cardiopulmonary bypass may be due to ischemia hepatitis [52]. In some previous reports, these patients found to have more frequently elevated central venous pressure [52,53].

Visceral complications rarely occur during uncomplicated recovery. Most of patients with GIC experienced other complications [2,8,28,54]. Recht et al. reported high prevalence of neurologic, pulmonary, renal and infections in patients with GIC [54]. On the other hand, Mc Sweeney et al. [4] in a multicentric study have found that patients developing GIC were 6.5 times more likely to die than those without GIC. The mean duration of ICU stay as the median post-surgical hospital stay increased by more than 1 week and 3 weeks respectively compared with uncomplicated cases. This explain excessive consumption of materials, inotropic drugs and expensive antibiotics and many have a large economic impact. In the current study, GIC occurred at a mean of 12.2 ± 8.6 days at surgery. As delayed diagnosis of GIC is often associated with unfavorable outcome, identification of patients at risk seems desirable.

Andersson et al. [2] tried to develop a risk score model specific for predicting GIC after cardiac surgery named gastrointestinal complications score (GICS). The model helps to create Framework for medical management for this patient group, but it need still more studies for validation. Because patients developing GIC after cardiac surgery are at high risk for morbidity and death, the determination of who are a greater risk of GI events is extremely important and clinically relevant because it may direct perioperative management and influence the decision regarding therapeutic measures.

Many studies have identified various risk factors trying to elucidate the correlation with GI, but often not the same. A few authors [4,8,29,39] have used multivariate analysis, but with little concordance except for age, renal dysfunction, low EF, prolonged ventilation and NYHA functional class III – IV. Our study revealed 19 independent determinants of GIC after cardiac surgery but univariate analysis.

Smoking, diabetes mellitus, peripheral vascular disease and chronic renal insufficiency reflect a high prevalence of atherosclerotic disease. We found that CPB time was significantly longer than in uncomplicated cases. This is in accordance with other published data [2-4,25,29,55]. Perioperative factors such as LOS, need for excessive inotropic agents, concomitant infection were correlated with increased incidence of GIC, and it correspond well with previous reports [4,8,24-27,30,32].

The relationship between CRF, ARF and GIC after CPB has been documented in many previous investigations [1,24-26,30,56] in present study; both CRF and postoperative ARF are independent determinants for GIC. Opinions regarding the relationship of sepsis to GIC are not uniform in the literature. To our knowledge, the septic state may lead to systemic hypoperfusion and finally, to multi-organ failure. Our result revealed severe infection as a risk factor for GIC in univariate analysis but not in multivariate analysis. Use of an IABP has also been previously identified as a predisposing risk factor for developing GIC [5,24,42]. In present study we found that need for IABP was more common in patients with GIC, but it's impact after logistic regression was not significant.

This study failed also to demonstrate low EF as an independent predictor of GIC in contrast with previous reports [8,29]. In this

regard, prolonged mechanical ventilation has been implicated as the strongest predictor of an adverse GI outcome. Mutlu et al. have elegantly documented the role of prolonged ventilator support in modifying the splanchnic blood flow [56]. These findings are reported by other investigations [57]. Prolonged mechanical ventilation was found to be a risk factor in our analysis but not significant in multivariate analysis.

Limitations

The major limitation of our study is its retrospective design that could not allow us to determine the precise explanation for some of GIC like intestinal ischemia.

The small size of cohort of patient did not help to make investigation easier and certain risk factor that were reported as determinants previously failed to be significant.

Our cohort also is heterogeneous because we included all cardiac surgery procedures. The large period of the study didn't help to make our results homogenous. Among this period there were different changes in cardiac diagnosis methods, cardiac surgery technics, CPB procedures and anesthetic cares.

Conclusion

GIC after open heart surgery are infrequent (1.7%) with a higher hospital mortality rate (48.5%). Our multivariate logistic regression analysis found that risk factors of GIC were: previous gastric ulcer and postoperative renal insufficiency.

Future Direction

We believe that we should make prospective large cohort to make results strong and more interpretable.

Declaration of Conflicting interest

The authors declared no conflicts of interest.

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References

1. Rodriguez F, Nguyen TC, Galanko JA, et al. Gastrointestinal complications after coronary artery bypass grafting: a national study of morbidity and mortality predictors. *J Am Coll Surg.* 2007;205:741-7.
2. Andersson B, Nilsson J, Brandt J, et al. Gastrointestinal complications after cardiac surgery. *Br J Surg.* 2005;92:326-33.
3. Geissler HJ, Fischer UM, Grunert S, et al. Incidence and outcome of gastrointestinal complications after cardiopulmonary bypass. *Interact Cardiovasc Thorac Surg.* 2006;5:239-42.
4. McSweeney ME, Garwood S, Levin J, et al. Adverse gastrointestinal complications after cardiopulmonary bypass: can outcome be predicted from preoperative risk factors? *Anesth Analg.* 2004;98:1610-7.
5. Yilmaz AT, Arslan M, Demirkilç U, et al. Gastrointestinal complications after cardiac surgery. *Eur J Cardiothorac Surg.* 1996;10:763-7.

6. Simic O, Strathausen S, Geidel S, et al. Abdominal complications following cardiac surgery. *Acta Med Croatica.* 1997;51:191-6.
7. Hashemzadeh K, Hashemzadeh S. Predictors and outcome of gastrointestinal complications after cardiac surgery. *Minerva Chir.* 2012;67:327-35.
8. Sakorafas GH, Tsiotos GG. Intra-abdominal complications after cardiac surgery. *Eur J Surg.* 1999;165:820-7.
9. Hessel EA. Abdominal organ injury after cardiac surgery. *Semin Cardiothorac Vasc Anesth.* 2004;8:243-63.
10. Díaz-Gómez JL, Nutter B, Xu M, et al. The effect of postoperative gastrointestinal complications in patients undergoing coronary artery bypass surgery. *Ann Thorac Surg.* 2010;90:109-16.
11. Raja SG, Haider Z, Ahmad M. Predictors of gastrointestinal complications after conventional and beating heart coronary surgery. *Sur J R Coll Surg Edin Irel.* 2003;221-8.
12. Croome KP, Kiaii B, Fox S, et al. Comparison of gastrointestinal complications in on-pump versus off-pump coronary bypass grafting. *Can J Surg.* 2009;52:125-8.
13. Poirier B, Baillet R, Bauset R, et al. Abdominal complications associated with cardiac surgery. Review of a contemporary surgical experience and of a series done without extracorporeal circulation. *Can J Surg.* 2003;46:176-82.
14. Mathieu RT. Hepatic blood flow during cardiopulmonary bypass. *Crit Care Med.* 1993;21:S72-6.
15. Chapman MV, Woolf RL, Bennett-Guerrero E, et al. The effect of hypothermia on calculated values using saline and automated air tonometry. *J Cardiothorac Vasc Anesth.* 2002;16:304-7.
16. Chapman MV, Mythen MG, Webb AR, et al. Report from the meeting: Gastrointestinal Tonometry: State of the Art. 22nd-23rd May 1998, London, UK. *Intensive Care Med.* 2000;26:613-22.
17. Bennett-Guerrero E, Panah MH, Bodian CA, et al. Automated detection of gastric luminal partial pressure of carbon dioxide during cardiovascular surgery using the Tonocap. *Anesthesiology.* 2000;92:38-45.
18. Mythen MG, Webb AR. Gastrointestinal tonometry comes of age? *Br J Anaesth.* 1998;81:667-8.
19. Karavana MN, Frumento RJ, Hirsch AL, et al. Gastric hypercarbia and adverse outcomes after cardiac surgery. *Int Care Med.* 2003;29:742-8.
20. Takala J. Determinants of splanchnic blood flow. *Br J Anaesth.* 1996;77:50-8.
21. Ackland G, Grocott MP, Mythen MG. Understanding gastrointestinal perfusion in critical care: So near, and yet so far. *Crit Care.* 2000;4:269-81.
22. Sever K, Ozbek C, Goktas B, et al. Gastrointestinal complications after open heart surgery: Incidence and determinants of risk factors. *Angiology.* 2014;65:425-9
23. Bolcal C, Iyem H, Sargin M, et al. Gastrointestinal complications after cardiopulmonary bypass: Sixteen years of experience. *Can J Gastroenterol.* 2005;19:613-7.
24. 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.
25. Aouifi A, Piriou V, Bastien O, et al. Severe digestive complications after heart surgery using extracorporeal circulation. *Can J Anaesth.* 1999;46:114-21.
26. Khan JH, Lambert AM, Habib JH, et al. Abdominal complications after heart surgery. *Ann Thorac Surg.* 2006;82:1796-801.
27. Egleston CV, Wood AE, Gorey TF, et al. Gastrointestinal complications after cardiac surgery. *Ann R Coll Surg Engl.* 1993;75:52-6.
28. Zacharias A, Schwann TA, Parenteau GL, et al. Predictors of gastrointestinal complications in cardiac surgery. *Tex Heart Inst J.* 2000;27:93-9.
29. Yoshida K, Matsumoto M, Sugita T, et al. Gastrointestinal complications in patients undergoing coronary artery bypass grafting. *Ann Thorac Cardiovasc Surg.* 2005;11:25-8.
30. Bhat M, Larocque M, Amorim M, et al. Prediction and prevention of upper gastrointestinal bleeding after cardiac surgery: a case control study. *Can J Gastroenterol.* 2012;26:340-4.
31. Gulkarov I, Trocciola SM, Yokoyama CC, et al. Gastrointestinal complications after mitral valve surgery. *Ann Thorac Cardiovasc Surg.* 2014;20:292-8.
32. Abboud B, Daher R, Sleilaty G, et al. Is prompt exploratory laparotomy the best attitude for mesenteric ischemia after cardiac surgery? *Interact Cardiovasc Thorac Surg.* 2008;7:1079-83.
33. M Aithoussa, A Abetti, A Abdou, et al. Does pre-existent gastroduodenal ulcer increase gastrointestinal bleeding after cardiac surgery? *J Gastrointest Disord Liver Func.* 2016;2:1-4.
34. Schoots IG, Koffeman GI, Legemate DA, et al. Systematic review of survival after acute mesenteric ischaemia according to disease aetiology. *Br J Surg.* 2004;91:17-27.
35. Kazui T, Yamasaki M, Abe K, et al. Non-obstructive mesenteric ischemia: a potentially lethal complication after cardiovascular surgery: report of two cases. *Ann Thorac Cardiovasc Surg.* 2012;18:56-60.
36. Ghosh S, Roberts N, Firmin RK, et al. Risk factors for intestinal ischaemia in cardiac surgical patients. *Eur J Cardiothorac Surg.* 2002;21:411-6.
37. Venkateswaran RV, Charman SC, Goddard M, et al. Lethal mesenteric ischaemia after cardiopulmonary bypass: A common complication? *Eur J Cardiothorac Surg.* 2002;22:534-8.

38. Musleh GS, Patel NC, Grayson AD, et al. Off-pump coronary artery bypass surgery does not reduce gastrointestinal complications. *Eur J Cardiothorac Surg.* 2003;23:170-4.
39. Sanisoglu I, Guden M, Bayramoglu Z, et al. Does off-pump CABG reduce gastrointestinal complications? *Ann Thorac Surg.* 2004;77:619-25.
40. Woo K, Major K, Kohanzadeh S, et al. Laparotomy for visceral ischemia and gangrene. *Am Surg.* 2007;73:1006-8.
41. Mangi AA, Christison-Lagay ER, Torchiana DF, et al. Gastrointestinal complications in patients undergoing heart operation: an analysis of 8709 consecutive cardiac surgical patients. *Ann Surg.* 2005;241:895-901.
42. Akpınar B, Sagbas E, Guden M, et al. Acute gastrointestinal complications after open heart surgery. *Asian Cardiovasc Thorac Ann.* 2000;8:109-13.
43. Feiner H. Pancreatitis after cardiac surgery: A morphologic study. *Am J Surg.* 1976;131:684-8.
44. Warshaw AL, O'Hara PJ. Susceptibility of the pancreas to ischemic injury in shock. *Ann Surg.* 1978;188:197-201.
45. Lefor AT, Vuocolo P, Parker FB, et al. Pancreatic complications following cardiopulmonary bypass. Factors influencing mortality. *Arch Surg.* 1992;127:1225-30.
46. Horn JK, Ranson JH, Goldstein IM, et al. Evidence of complement catabolism in experimental acute pancreatitis. *Am J Pathol.* 1980;101(1):205-16.
47. Rose DM, Ranson JH, Cunningham JN, et al. Patterns of severe pancreatic injury following cardiopulmonary bypass. *Ann Surg.* 1984;199:168-72.
48. Shapiro MJ, Luchtefeld WB, Kurzweil S, et al. Acute acalculous cholecystitis in the critically ill. *Am Surg.* 1994;60:335-9.
49. Rady MY, Kodavatiganti R, Ryan T. Perioperative predictors of acute cholecystitis after cardiovascular surgery. *Chest.* 1998;114:76-84.
50. Sakurai T, Ichimiya H, Miyazaki H, et al. Profiling of eicosanoids in inflamed gall bladder wall by gas chromatography with selected-ion monitoring. *J Chromatogr.* 1991;15:571.
51. Raman JS, Kochi K, Morimatsu H, et al. Severe ischemic early liver injury after cardiac surgery. *Ann Thorac Surg.* 2002;74:1601-6.
52. Seeto RK, Fenn B, Rokey DC. Ischemic hepatitis: Clinical presentation and pathogenesis. *Am J Med.* 2000;1:109-13.
53. Recht MH, Smith JM, Woods SE, et al. Predictors and outcomes of gastrointestinal complications in patients undergoing coronary artery bypass graft surgery: A prospective, nested case-control study. *J Am Coll Surg.* 2004;198:742-7.
54. Movahedi N, Karimi A, Ahmadi H, et al. Laparotomy due to gastrointestinal complications after open heart surgery. *J Cardiovasc Surg (Torino).* 2011;52:111-6.
55. Guler M, Yamak B, Erdogan M, et al. Risk factors for gastrointestinal complications in patients undergoing coronary artery bypass graft surgery. *J Cardiothorac Vasc Anesth.* 2011;25:637-41.
56. Mutlu GM, Mutlu EA, Factor P. GI complications in patients receiving mechanical ventilation. *Chest.* 2001;119:1222-41.
57. Cullen JJ, Ephgrave KS, Caropreso DK. Gastrointestinal myoelectric activity during endotoxemia. *Am J Surg.* 1996;171:596-9.

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