

Abdominal Complications after Cardiac Surgery

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Abstract

Objective: Abdominal complications after cardiac surgery are associated with a high mortality rate. Due to the absence of early specific clinical signs, diagnosis is often delayed. The present study seeks to determine predictive risk factors for subsequent gastrointestinal complications after cardiosurgical procedures. **Methods:** Within 12 months, all patients (n = 1,116) who had undergone open heart surgery with cardiopulmonary bypass at our institution were studied for abdominal complications. To determine predictive factors, all case histories of the patients were analysed. **Results:** Abdominal complications occurred in 23 (2.1%) patients during the postoperative intensive care unit (ICU) stay, ten of whom had to undergo subsequent abdominal surgery. Of these 23 patients, 20 died. Early complications occurred most likely on postoperative days 6 and 7, consisting of bowel ischaemia or hepatic failure. Late complications consisted of gastrointestinal bleeding, pseudomembranous colitis, cholecystitis and septic rupture of a spleen. The relative risk for abdominal complications after cardiopulmonary bypass was highly increased in association with a cardiac index less than 2.0 l/min⁻¹/(m²)⁻¹ (22.1-fold), postoperative onset of atrial fibrillation (16.6-fold), emergency surgery (10.7-fold), need for vasopressors (10.1-fold), need for intra-aortic balloon counterpulsation (8.6-fold), and the need for re-exploration within the first 24 hours (8.4-fold). All patients with necrotic bowel disease had elevated serum lactate levels. Furthermore, both cardiopulmonary bypass and aortic clamping times were significantly prolonged in patients who developed gastrointestinal complications. **Conclusions:** A number of predictive factors has been described to contribute to the development of abdominal complications subsequently after cardiac surgery on cardiopulmonary bypass. Knowledge of these factors may lead to earlier identification of patients at increased risk and may allow for more efficient and earlier interventions.

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Key words: Cardiopulmonary bypass, Intensive care unit, Risk factors, Mortality

Introduction

Despite intensive therapeutic efforts, both the incidence and mortality of gastrointestinal complications after cardiac surgery employing cardiopulmonary bypass (CPB) have not decreased during the last two decades. Gastric bleeding, ischaemic bowel disease, gallbladder disease, and pancreatitis are reportedly the most common complications.¹⁻⁵ Despite major technological and medical advances, CPB still imposes a considerable physiological stress on the patient.^{6,7} This holds especially true in the case of severely ill or otherwise compromised patients. However, it has been difficult to clearly define risk factors which are associated with a significantly increased risk for intraabdominal complications after CPB. The goal of our present study was to determine risk factors which are

predictive for a significantly increased risk for postoperative gastrointestinal complications to earlier identify patients at risk.

Materials and Methods

A total of 1,116 patients who had undergone cardiosurgical procedures with extracorporeal circulation at our institution during a one-year period were prospectively studied. We evaluated all patients for possible risk factors, which we believed might contribute to or cause the development of postoperative gastrointestinal complications. A routine prophylaxis against ulcers was done with ranitidine (4 x 50 mg/day) in the intensive care unit (ICU). A proton channel blocker (omeprazol 2 x 10 mg/day) was only used in

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TABLE I: PRE-EXISTING RISK FACTORS AND RELATIVE RISK FOR DEVELOPMENT OF POSTOPERATIVE ABDOMINAL COMPLICATIONS

Risk factor	No abdominal complications (n = 1,093)	Abdominal complications (n = 23)	P value	Relative risk
Hypertension	636 (58.2%)	15 (65.2%)	ns	
History of myocardial infarction	527 (48.2%)	11 (47.8%)	ns	
Diabetes mellitus	348 (31.8%)	6 (26.1%)	ns	
Left ventricular ejection fraction <50%	199 (18.2%)	6 (26.1%)	ns	
Chronic obstructive pulmonary disease	129 (11.8%)	6 (26.1%)	0.0495	2.565
Atrial fibrillation	103 (9.4%)	5 (21.7%)	ns	
History of abdominal disease	100 (9.1%)	2 (8.7%)	ns	
Emergency surgery	91 (8.3%)	12 (52.2%)	<0.0001	10.729
Redo cardiac surgery	88 (8.1%)	2 (8.7%)	ns	
Renal insufficiency	60 (5.5%)	5 (21.7%)	0.0085	4.491

Data presented in number and percentage

patients with a history of ulcers. 2 g of ceftriaxon were given perioperatively to all patients.

Cardiopulmonary Bypass

CPB was performed with membrane oxygenators under mild hypothermia (32°C). A non-pulsatile blood flow rate of not less than 2.4 l/min⁻¹/m² was used whenever possible. Buckberg blood cardioplegia was used for myocardial protection. A mean arterial perfusion pressure of 5.33 kPa was maintained by application of norepinephrine if necessary. Also, if needed, epinephrine (max. 0.3 µg/kg⁻¹/min⁻¹) or enoximone (10 µg/kg⁻¹/min⁻¹) was administered. Our guidelines for haemodynamic management were to use initially dopamine and dobutamine up to a maximum dose of 10 µg/kg⁻¹/min⁻¹. If this proved ineffective, we used epinephrine as needed.

Risk Factors

Our definition of preexisting gastrointestinal disease included all patients with a history of gastric or duodenal ulcers and colitis, as well as hepatobiliary disease such as pancreatitis, cholelithiasis and cholecystitis. Renal insufficiency was considered significant with a serum creatinine level of more than 178 µmol/l. All other preoperative risk factors are given in Table I. Intraoperatively, both cardiopulmonary bypass and aortic cross-clamping times were considered as potential risk factors. During the postoperative period, we looked for postoperative catecholamine support with either epinephrine and/or norepinephrine, loss of sinus rhythm and onset of atrial fibrillation, counterpulsation by intra-aortic balloon pump, low cardiac output with a cardiac index of less than 2.0 l/min⁻¹/m², and the need for redo thoracotomy within the first 24 postoperative hours.

Gastrointestinal Complications

We defined postoperative gastrointestinal complications as follows: gastrointestinal bleeding confirmed by

endoscopy with a decrease of at least 2 g/dl in haemoglobin content, mesenteric ischaemia or infarction, cholecystitis, pancreatitis and hepatic failure confirmed by history and laboratory data.

Statistics

The prevalence of risk factors between the groups with or without gastrointestinal complications was compared in terms of statistical significance with Fisher's Exact test, and the relative risk was calculated, provided statistical significance. Demographic data, as well as CPB and aortic clamping times were compared using the Wilcoxon Mann-Whitney test. All statistical calculations were performed with GraphPad InStat Version 3.00 (GraphPad Software, Inc.). Statistical significance was confirmed with a probability of error less than 5% ($P < 0.05$).

Results

Out of a total of 1,116 patients who underwent cardiac surgery with CPB during a 12-month period, 23 (2.1%) developed gastrointestinal complications during their postoperative ICU stay. Both groups were identical in terms of demographic data and the surgical procedure performed. The mortality rate for 1,093 patients who did not develop gastrointestinal complications was 2.7% ($n = 30$). Of those 23 patients with postoperative gastrointestinal complications, 20 (87.0%) died ($P < 0.0001$) (Table II). In 9 patients with hepatic failure or mesenteric ischaemia, the actual cause of death was most likely prolonged low cardiac output. One patient died in direct association with gastric bleeding. In the remaining 10 patients who died, it remained unclear whether death was directly related to the gastrointestinal complications or rather was a result of prolonged hypoperfusion from circulatory failure or prolonged ICU stay.

Thirteen patients received conservative treatment (5 for gastric bleeding, 5 for hepatic failure, and 3 for pseudomembranous colitis), and 10 patients had a

TABLE II: DEMOGRAPHIC DATA AND SURGICAL INTERVENTIONS IN 1,116 PATIENTS WHO UNDERWENT CARDIAC SURGERY

		No abdominal complications (n = 1,093)	Abdominal complications (n = 23)
In-hospital mortality		30 (2.7%)*	20 (87.0%)*
Age (years)		66.1 ± 8.12	64.6 ± 9.71
Sex	Male	775 (70.9%)	15 (65.2%)
	Female	318 (29.1%)	8 (34.8%)
Intervention	Coronary artery bypass grafting (CABG)	765 (70.0%)	14 (60.9%)
	Valve repair or replacement (VR)	169 (15.5%)	3 (13.0%)
	CABG and VR	100 (9.1%)	2 (8.7%)
	Other	59 (5.4%)	4 (17.4%)

Data presented as mean ± (standard deviation) or number and (%).

* $P < 0.0001$

laparotomy. Five of these patients had ischaemic colitis, and 2 of them had to undergo repeat laparotomies, twice and three times, respectively. Two patients had gastric bleeding, and 2 more patients had acute cholecystitis. One patient died from rupture of a septic spleen (Table III).

Preoperative Risk Factors

As shown in Table I, the most significant risk factors for the development of postoperative gastrointestinal complications were emergency surgery and renal insufficiency. Furthermore, patients with co-existing chronic obstructive pulmonary disease are also at increased risk for gastrointestinal complications.

Intraoperative Risk Factors

Patients who developed gastrointestinal complications during their ICU stay had significantly prolonged periods of both cardiopulmonary bypass (205.6 ± 83.16 versus 114.9 ± 49.08 minutes, $P < 0.0001$) and aortic cross clamping

TABLE III: PATIENTS WITH ABDOMINAL COMPLICATIONS AFTER CARDIAC SURGERY

	Patients (n)	Survivors (n)	Manifestation of complication (days after cardiac surgery)*
Hepatic failure	5	0	6 (3 – 8)
Ischaemic or necrotic bowel disease	5	1	7 (1 – 11)
Gastric bleeding	7	1	13 (9 – 20)
Pseudomembraneous colitis	3	0	27 (8 – 40)
Acute cholecystitis	2	1	45 (30 – 60)
Septic rupture of the spleen	1	0	43

* Data presented as median and range

(106.3 ± 46.67 versus 74.0 ± 38.66 minutes, $P < 0.0001$). However, there was no clear threshold at which the risk for complications significantly increased.

Postoperative Risk Factors

As listed in Table IV, patients with low cardiac output were at a 22-fold increased risk for gastrointestinal complications. Loss of sinus rhythm and onset of atrial fibrillation during the postoperative period increased the risk by almost 17-fold. Another significant risk factor was the postoperative need for vasopressor support using epinephrine and/or norepinephrine, as was the use of intra-aortic balloon counterpulsation. Also, redo thoracotomy within the first postoperative day resulted in an 8-fold increased likelihood of gastrointestinal complications. All patients with necrotic bowel and hepatic failure ($n = 10$) had reasonably elevated serum lactate levels. In 70% of these patients, the serum lactate levels exceeded 10 mmol/l. In all other patients, the serum lactate levels were normal regardless of which gastrointestinal complication occurred in particular.

Discussion

Despite major advances in cardiopulmonary bypass

TABLE IV: POSTOPERATIVE RISK FACTORS AND RELATIVE RISK FOR DEVELOPMENT OF POSTOPERATIVE ABDOMINAL COMPLICATIONS

Risk factor	No abdominal complications (n = 1,093)	Abdominal complications (n = 23)	P value	Relative risk
Low cardiac output syndrome	49 (4.5%)	13 (56.5%)	<0.0001	22.100
Loss of sinus rhythm and onset of atrial fibrillation	119 (10.9%)	16 (69.6%)	<0.0001	16.610
Vasopressor support with epinephrine and/or norepinephrine	97 (8.9%)	12 (52.2%)	<0.0001	10.078
Intra-aortic balloon counterpulsation	30 (2.7%)	5 (21.7%)	0.0005	8.579
Redo thoracotomy < 24 hours	39 (3.6%)	6 (26.1%)	0.0002	8.400

Data presented in number and percentage

technology and improved strategies in intensive care medicine, the 1% to 3% incidence of gastrointestinal complications after CPB remained constant during the last two decades.¹⁻⁵ In our study, the incidence of postoperative gastrointestinal complications was 2.1% which conforms with the low incidence demonstrated by other investigators. Once a gastrointestinal complication occurs, however, overall mortality ranges from 13.5% to 72%.^{1-5,8} In particular, patients with gastric bleeding or cholecystitis are most likely to survive, whereas almost every patient with mesenteric ischaemia dies.^{9,10} Since clinical features are often subtle, it is essential to identify patients at risk for early diagnosis and aggressive treatment.

The most severe gastrointestinal complications, i.e. ischaemic bowel and hepatic failure, occurred in almost half of our patients. None of these patients survived. Diagnosis was most frequently made during the first postoperative week, and no later than 11 days after cardiac surgery. These findings suggest an obvious relationship of ischaemic bowel disease or hepatic failure and previous surgery. Prolonged CPB and aortic clamping result in significant intestinal hypoperfusion and subsequent inflammatory response,⁷ as does postoperative low cardiac output¹⁰ which often requires application of highly potent vasoactive catecholamines that further augment intestinal hypoperfusion.¹¹ Use of intra-aortic balloon counterpulsation (IABC)—which is often also required in these patients—may further aggravate the situation, especially in cases of malposition of the device.¹² We could demonstrate that the use of intra-aortic balloon counterpulsation increases the risk for gastrointestinal disease by more than 8-fold. Intestinal hypoperfusion, which may progress to acute mesenteric infarction, results in an ischaemic lesion of the bowel with necrosis and perforation. This, in turn, may lead to sepsis and peritonitis. The occurrence of hepatic failure also seems to be the consequence of a refractory low cardiac output syndrome with prolonged application of vasoactive catecholamines. We believe that both hepatic failure and mesenteric ischaemia represent complications of prolonged circulatory failure which was the actual cause of death. Indeed, hepatic failure or mesenteric ischaemia due to a state of hypoperfusion from low cardiac output syndrome can rarely be treated sufficiently even if diagnosis is made early. In contrast, if mesenteric ischaemia results from thromboembolic events that most likely occur in patients who present with postoperative onset of atrial fibrillation, more timely diagnosis may possibly improve outcome.

Regardless of the fact that catecholamine support can rarely be avoided in patients with low cardiac output, the risks and benefits must be considered very carefully. Because of a 10-fold increased likelihood of gastrointestinal

complications, our data demand a thoughtful risk-benefit analysis regarding vasoactive support, particularly with norepinephrine and epinephrine. Phosphodiesterase inhibitors (PDEI) may be used to reduce the requirements of epinephrine or norepinephrine, however, the sole use of PDEI without epinephrine or norepinephrine support may result in clinically significant decrease in blood pressure, that further augments peripheral and intestinal hypoperfusion, especially in hypovolaemic patients. Dopexamine, that interferes with intestinal perfusion to a lesser degree than does norepinephrine or epinephrine,¹³ may be used additionally to reduce the requirement of vasopressors. Patients with poor cardiac function who require vasopressors and/or mechanical support devices such as IABC are at highly increased risk for gastrointestinal complications, however, hepatic failure or mesenteric ischaemia only accelerates the death of most patients who most likely would have died from circulatory failure anyway, with or without gastrointestinal complications.

Surprisingly, patients with pre-existing atrial fibrillation were not at significantly increased risk for gastrointestinal complications, but those who lost sinus rhythm during the postoperative period were. Sixteen of the 23 patients with gastrointestinal complications who had been in sinus rhythm preoperatively, developed atrial fibrillation after surgery. If administration of antiarrhythmic drugs proved ineffective, electric cardioversion was performed. However, return to and maintenance of sinus rhythm could be achieved in only 4 patients. Atrial fibrillation represents one of the most common rhythm disturbances after cardiac procedures.¹⁴ The loss of atrial contraction is associated with haemodynamic impairment, reduced stroke volume and thromboembolic events.¹⁵ The combination of postoperative onset of atrial fibrillation and gastrointestinal symptoms is highly suggestive of a thromboembolic event that must be confirmed or disproved without delay.

Furthermore, we strongly recommend frequent determinations of serum lactate levels in patients at increased risk for gastrointestinal complications. The serum lactate levels were highly elevated in all our patients with ischaemic bowel disease. In contrast, the lactate levels in patients with hepatic failure or low cardiac output syndrome virtually always were only moderately elevated. Regardless of the fact that serum lactate levels are not quite specific for ischaemic bowel disease, they represent a sensitive diagnostic marker and valuable screening tool, and highly elevated serum lactate levels must be a reason for concern, particularly in patients at risk. If bowel ischaemia is suspected in patients at risk, invasive diagnostic measures such as explorative laparotomy and angiography should follow without delay.

Late gastrointestinal complications consisted of gastric

bleeding, pseudomembranous colitis, acute cholecystitis, and septic rupture of the spleen. These complications occurred most frequently during the second postoperative week, but were also noted as late as 60 days after cardiac surgery. Despite the routine perioperative use of histamine H_2 -blockers and administration of proton channel blockers in patients with a history of ulcers, episodes of gastric bleeding are still the most common gastrointestinal complication after cardiac surgery.⁴ In 7 patients in our study who had gastric bleeding, bleeding control by endoscopic clipping was successful in 5 patients, and only 2 patients needed to undergo abdominal surgery. Therefore, gastroscopy should be done early if gastric bleeding is suspected in a patient at risk. Regardless of the fact that 6 of 7 patients died, only 1 patient died in direct association with gastric bleeding. It can only be speculated to what extent compromised cardiac function and resulting hypoperfusion contributed to the development of gastric bleeding in these patients.

In contrast to other investigators who reported the development of severe, acute pancreatitis after cardiac surgery,^{4,5} there was no case of acute pancreatitis in our study. Acute cholecystitis occurred in 2 patients. It is speculated that both acute pancreatitis and cholecystitis after cardiac surgery are caused by hypotension and hypoperfusion which result in inflammation and reperfusion injury. Furthermore, calcium which is often administered for haemodynamic reasons during weaning from CPB, has been described as a significant risk factor for pancreatic injury.¹⁶ Since acute cholecystitis in our patients did not occur earlier than 30 days after cardiac surgery, it can most likely be attributed to the prolonged stay in the ICU rather than to the previous surgical procedure. Similarly, the late complications of septic rupture of the spleen and pseudomembranous colitis are probably also sequelae of prolonged intensive care treatment with steroids and antibiotics or the use of external organ support devices, such as veno-venous haemofiltration and aggressive ventilation strategies to support the lungs.

Conclusion

The results of our study show that a number of postoperative complications are associated with a highly increased risk for the development of gastrointestinal complications after cardiac surgery. Intestinal hypoperfusion as a result of poor cardiac output is likely to result in hepatic failure and ischaemic bowel disease early after surgery. Gastric bleeding, cholecystitis and pseudomembranous colitis are late complications and are probably sequelae of prolonged ICU treatment. The risk factors described allow the prediction of who is at increased risk for gastrointestinal complications. Knowledge of these factors and increased awareness of patients at risk may lead

to more timely diagnosis, earlier therapeutic intervention and therefore a reduction of the alarmingly high mortality rate of gastrointestinal complications, at least in patients whose gastrointestinal complications are not sequelae of prolonged poor cardiac function and subsequent intestinal hypoperfusion.

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