GASTROINTESTINAL COMPLICATIONS AFTER CARDIAC SURGERY

Essay

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بسم الله الرحمن الرحيم
قَالُوا سُبْحَانَكَ
لاَ عِلْمَ لَنَا
إِلاَّ مَا عَلَّمْتَنَا
إِنَّكَ أَنتَ
الْعَلِيمُ الْحَكِيمُ
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Karim Sobhy Sayed

مضاعفات الجهاز الهضمي بعد عمليات القلب المفتوح

رسالة

توطئه للحصول علي درجة الماجستير في العناية والتخدير

مقدم من

الطبيب/ كريم صبحي سيد

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List of Abbreviations

GI	Gastrointestinal
AC	Acute cholecystitis
aGST	A-glutathione S-transferase
ALT	Serum alanine amino transferase
AMI	Acute mesenteric ischemia
CABG	Coronary artery bypass graft
CHF	Congestive heart failure
CO	Cardiac output
CPB	Cardiopulmonary bypass
CRP	C-reactive protein
CT	Computed tomography
DO2	Oxygen delivery
GIP	Gastrointestinal perforation
GUCH	Grown-up congenital heart
IABP	Intra-aortic balloon pump
ICAM-1	Intercellular adhesion molecule-1
IL	Interleukins
LDD	Low-dose dopamine
LGIB	Lower gastrointestinal bleeding
MAP	Mean arterial pressure
MEGEX	Monoethylglycinexylidide
NO	Nitric oxide
NOMI	Non-occlusive mesenteric ischemia
OPCAB	Off pump coronary artery bypass
PAOP	Pulmonary artery occlusion pressure
PEEP	Positive end-expiratory pressure

List of Abbreviations

PEEP	**Positive end expiratory pressure**
pHi	Intramucosal ph
SIELI	*Severe ischemic early liver injury*
SIRS	Systemic inflammatory response syndrome
SMA	Superior mesenteric artery
SVRI	Systemic vascular resistance index
TNF	Tumour necrosis factor
UGH	Upper gastrointestinal haemorrhage
UGI	Upper gastrointestinal
VAD	Ventricular assist device
VO2	Oxygen consumption

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INTRODUCTION

Organ and tissue injuries are sometimes lethal sequelae to cardiac surgery, but until the advent of off-pump coronary artery bypass procedures, these are considered a consequence of cardiopulmonary bypass. Despite the deterioration of the risk profile of patients presenting for cardiac surgery, the overall incidence of gastrointestinal complications has changed little over the last two decades, which may be a result of improvements in cardiac surgical techniques, anaesthesia and intensive care. The lethality of gastrointestinal (GI) complications has also altered little over this time frame, which support their clinical importance and the need to identify and intervene at the earliest stage if outcome is to be influenced ***(Sunil et al., 2006)***.

The most common clinical complications are GI haemorrhage, intestinal ischaemia, pancreatitis and cholecystitis, diverticulitis, liver dysfunction. A period of ileus or constipation is probably the most common complication, but is usually clinically insignificant and, therefore, not recorded as a morbidity. Overall, the incidence ranges from 0.3 to 2.0%, with mortality ranging from 10 to 60% ***(Nilson et al., 2005).***

There were identified seven risk factors:

1- Inotropic support

2- Intra-aortic balloon pump support

3- Arrythmias

4- Pre-existing renal dysfunction (creatinine>125 mmol/L)

5- Established renal failure

6- Pre-existing hepatic dysfunction (bilirubin>14 mmol/L)

7- Protracted ventilation (>24 h)

These all pointed to hypoperfusion of the splanchnic bed as the common factor in the perioperative period in the pathogenesis of these complications ***(Sunil et al., 2006).***

The prevention, early diagnosis and treatment of Gastrointestinal complications after cardiac surgery have gained significant clinical attention in order to improve the outcome of these patients ***(Fabiano et al., 2013).***

AIM OF THE WORK

The aim of the work is to shed the light on the most common intestinal complications which occur after cardiac surgery and management of those complications in order to decrease morbidity and mortality after cardiac surgery.

Chapter (1):

PATHOPHYSIOLOGY OF GASTROINTESTINAL COMPLICATIONS POST-CARDIAC SURGERY

Numerous studies have identified factors, including low ejection fraction and peripheral vascular disease, which may be important in identifying at risk patients, but more recently, multivariate analysis has been applied to identify independent risk factors. Prolonged mechanical ventilation >24 h, Postoperative renal failure, Sepsis, Valve surgery, Preoperative renal failure, Deep sternal infection ***(D'Ancona et al., 2003).***

It is interesting that, despite the time interval, two factors appear to be consistently prominent and these are pre/postoperative renal impairment and protracted ventilation (> 24 h). The link between renal dysfunction and GI complications may be explained by hypoperfusion, which affects the renal and gut beds in a similar fashion, and injury to these two beds is likely to run in parallel. Renal failure also results in impaired jejunal motility and decreases colonic transit time ***(Lefebvre et al., 2008).***

Protracted ventilation has been identified as a risk factor for GI morbidity by numerous univariate analyses and was first noted in multivariate analysis ***(Sunil et al., 2006).***

During mechanical ventilation, hypoperfusion to the splanchnic bed may be induced by the use of positive end-expiratory pressure (PEEP). PEEP reduces venous return and, thereby, cardiac output and blood pressure ***(Mutlu et al., 2001).***

High PEEP levels can induce angiotensin and catecholamine production, leading to splanchnic vasoconstriction ***(Raja et al., 2003).***

Patients who have had a stormy operative course are, therefore, put at further risk for GI complications by the need to use prolonged mechanical ventilation, often with intra-aortic balloon pump (IABP) support, with or without renal support. All these factors have the potential to reduce the limited flow to the splanchnic bed in patients who already have a compromised cardiac output ***(Filsoufi et al., 2007).***

Atheroembolism may also be the mechanism of injury in patients who have aorto-iliac disease and receive IABP support. although GI haemorrhage is the most frequently reported GI complication after both coronary and valve surgery, the aetiology is probably two-fold; haemorrhage secondary to over anti-coagulation of mechanical valve patients and haemorrhage due to primary mucosal injury from hypoperfusion. The majority of patients sustain GI haemorrhage secondary to mucosal injury, since only 24% of valve patients who sustained GI bleeding had been over anti-coagulated ***(Andersson et al., 2010).***

Liver dysfunction following cardiac surgery has been less well documented, but is worthy of mention because liver failure is extremely difficult to treat and often fatal after cardiac surgery. Liver dysfunction may present as mild hyperbilirubinaemia, or much less commonly, as liver failure as part of multi-system organ failure. The incidence of hyperbilirubinaemia has been reported at 14-20% post-cardiopulmonary bypass (CPB) ***(Rodriguez et al., 2010).***

It is documented in patients undergoing CPB, that preoperative alcohol intake, hepatic enzymes and bilirubin were not associated with subsequent liver dysfunction, but raised right atrial pressure, and multiple valve replacements were significant prognostic factors, which may have importance to the rising population of adult grown-up congenital heart (GUCH) patients presenting for surgery. This contention has been supported by Chu et al. who documented a 21.7% incidence of hyperbilirubinaemia amongst patients undergoing congenital cardiac procedures. Valve patients also had a high incidence; these findings correlated well with high right-sided pressures which would cause liver congestion or valve lesions causing tricuspid incompetence ***(Karangelis, 2011).***

These patients have reduced hepatic reserve and do not sustain the insult of cardiac surgery and CPB as well those without pre-existing hepatic congestion. Using the galactose

clearance technique, hepatic blood flow was found to be reduced by 19% during CPB ***(Smith, 2010).***

The blood supply to the liver is also dependent on the perfusion protocol adopted. It was found that hepatic perfusion was preserved during CPB by pulsatile flow at normothermia, but this advantage is less apparent at hypothermic temperatures (28-30 degree celsuis) ***(Mathie et al., 2006).***

The duration of CPB (>80 min) has also been described as a risk factor for hepatic damage, as measured by reduced monoethylglycinexylidide (MEGEX) hepatic production and a-glutathione S-transferase (aGST) release as a marker of liver injury ***(Kumle et al., 2003).***

This mortality is higher than that reported in other series of ischaemic hepatitis and was associated with low cardiac output and high right-sided filling pressures, postoperatively. Although multivariate analysis identified female gender as the only independent risk factor, the authors concluded that the main risk factors were hepatic hypoperfusion and elevated cardiac filling pressures. With the aim of identifying the potential benefit of eliminating CPB as a predisposing factor for liver injury, a prospective, randomized study in low-risk patients undergoing coronary artery bypass graft (CABG). There was no difference in liver dysfunction between patients undergoing CPB using pulsatile flow at 32 degree celsius and off pump coronary artery bypass (OPCAB) grafting. The