Acute Cholecystitis Complicating Cardiac Surgery
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Citation

Abstract
Cardiac operations are the most common elective procedures currently done. Gastrointestinal complications are rare after cardiac surgery, with high morbidity and mortality rate. The incidence of acute cholecystitis after cardiac surgery is 0.2% to 0.5%. Identification of specific predictors for acute cholecystitis in patients undergoing cardiovascular surgery should be attempted early in order to identify at-risk patients and prompt earlier diagnosis and treatment. Early and aggressive treatment in such cases is required if mortality is to be reduced.

INTRODUCTION
Cardiac operations are currently among the most common elective procedures done. With the progressive reduction in postoperative cardiac-related morbidity and mortality, even in elderly, understanding of pathogenesis and improved management of non-cardiac complications is of great importance.

Gastrointestinal complications after cardiac surgery are rare with high morbidity and mortality rate, the reported incidence ranges from 0.5% to 2.1% and morbidity is between 15% and 42%.

Acute cholecystitis comprises 6% to 18% of these complications and the incidence of acute cholecystitis after cardiac surgery is 0.2% to 0.5%.

A study in Cleveland clinic, Ohio, showed that, out of 11330 patients who went for cardiac surgery, 876 patients stayed in the ICU more than 7 days and 30 of them (3%) developed postoperative acute cholecystitis, 17 of them underwent cholecystectomy and eight of 17 patients (47%) were noted to have gangrene or perforation of the gallbladder wall as complication of acute cholecystitis.

CASE PRESENTATION
A 73-year-old male patient was admitted to the cardiac center in KKKUH for Coronary Artery Bypass Grafting (CABG). He was complaining of chest pain and shortness of breath; he had ischemic heart disease, hypertension and diabetes. He was smoker and had stopped smoking since 5 years. Preoperative laboratory investigation showed a fasting blood sugar of 6.6 mmol/L; full blood count, liver function test profile and coagulation profile were all normal.

The patient was admitted to ICU after CABG operation. On the 3rd postoperative day; the patient developed abdominal pain, mainly in the right hypochondrium with nausea. On examination; the patient was conscious and oriented, his general condition was satisfactory with no jaundice, a temperature of 38 °C, a pulse of 100/min, a BP of 110/60 mmHg on renal dose of dopamine and adrenaline and a respiratory rate of 35/min. Abdominal examination revealed tenderness, mild muscle guarding with mild rebound in the epigastric region and right hypochondrium; the rest of the abdomen was soft and lax with mild distension, intestinal sounds were audible but sluggish and rectal examination revealed an empty rectum. Laboratory investigation revealed a random blood sugar of 13.4 mmol/L. Na⁺, K⁺ and Cl⁻ were normal, Hb was 10.8, WBC 18000, PLT 51, urea 17 umol/L, creatinine 340 umol/L, total bilirubin 22umol/L, and serum amylase 137 umol/L. The coagulation profile showed a PT of 18.7 sec, an aPTT of 45.6 sec and an INR of 1.5.

From the above picture, the initial clinical diagnosis was bowel ischemia or acute cholecystitis. Ultrasound revealed a thickened gallbladder wall with no stones or pericholecystic fluid. CT of the chest revealed right pleural effusion with sub-segmental basal atelectasis (figures 1A & B).
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**Figure 1**
Figure 1A: CT of the chest

**Figure 2**
Figure 1B: CT of the chest (lung window)

**Figure 3**
Figure 2A: CT of the abdomen

**Figure 4**
Figure 2B: CT of the abdomen (kidney infarction)

Figure 1A and B show pleural effusion (right side) with sub-segmental basal atelectasis.

Abdominal CT revealed a right sub-hepatic focal area of fat streaking (stranding) denoting infection just caudal to the dilated gallbladder; the gallbladder showed no calculi, surrounding fluid or internal air lucencies (figure 2A).

CT also showed a thickened wall of a loop of small bowel with no intramural air, a right large ischemic renal infarction with a relatively small infarction of the left kidney (figure2B), with delayed bilateral renal excretory power, and a splenic infarction (figure 2C). The picture is suggestive of bowel ischemia.
Laparoscopic exploration was decided. Operative findings revealed a dilated and mildly edematous dusky segment of the ileum, 15cm long, with mildly sluggish peristalsis and no sign of acute ischemia (figure 3).

The gallbladder was covered by omentum with presence of mild turbid brownish fluid in the sub-diaphragmatic and pericholecystic areas (figure 4&5), it was found to be greenish in color with dark greenish patches and it was successfully removed laparoscopically (figure 6).

Figure 6
Figure 3: Demonstrating an ischemic (?) ileal loop with dilated segment

Figure 7
Figure 4: Sub-diaphragmatic collection

Figure 8
Figure 5: Pericholecystic collection
Postoperatively, the patient was transferred to ICU and kept on mechanical ventilation. After 24 hours the patient became feverish (38.5°C), developed tachycardia (120/min.) and WBC increased to 20000. Based on that and on the possibility of developing ischemic bowel, the patient was transferred to the OR for a second laparoscopic look. On second look; the whole bowel was explored and found healthy with normal color and peristalsis, even the previously dusky ileal loop had become normal in color and peristalsis. There was no leak from the gallbladder bed. On the next day, the patient was extubated, inotropic drugs were discontinued and his WBC went down. Two days later, the patient was tolerating normal diet and was discharged from cardiac ICU in a stable condition.

DISCUSSION

Most gastrointestinal complications after cardiopulmonary bypass (CPB) seem to be ischemic in origin, and half of all patients having cardiac operations have transient episodes of gut mucosal ischemia. Hepatic arterial flow is reduced during CPB. Ischemia has also been linked to the development of acute acalculous cholecystitis and upper gastrointestinal bleeding. Preoperative hypotension, prolonged CPB, vasoconstricting agents, arrhythmias, hemorrhage and pre-existing vascular disease alone or in combination reduce mucosal perfusion, injure the mucosa and damage the organ.

The pathogenesis of acute acalculous cholecystitis complicating cardiac surgery is not clearly delineated but it seems to be multifactorial. Bile stasis due to increased viscosity of bile and ampullary constriction due to narcotic analgesia can lead to distension of the gallbladder with increased wall pressure. Positive pressure ventilation has been shown in experimental studies to increase intraluminal pressure in the common bile duct and contributes to increased pressure of the gallbladder wall. Visceral hypoperfusion in the postoperative period due to low cardiac output syndrome has been documented as a risk factor for the development of acute acalculous cholecystitis.

The combination of increased wall tension of the gallbladder wall and hypoperfusion potentially leads to ischemia of the gallbladder mucosa and this is supported by a series of angiographic studies of acute acalculous cholecystitis gallbladder specimens, showing multiple arterial occlusions and absent or minimal venous filling. The release of cytokines such as factor XII and platelet-activating factor due to surgical trauma have also been implicated in the development of acute acalculous cholecystitis.

RISK FACTORS

Preoperative risk factors for abdominal complications after cardiac surgery are presented in table (1).
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Table 1

| Intraoperative risk factors: The type of cardiac surgery (coronary bypass or valve procedure) seems to have a role. Emergency operation, preoperative myocardial infarction, intraoperative hemorrhage, arrhythmias and low cardiac output (CO) are appreciably more common in patients who subsequently develop such a complication. Prolonged CPB is a serious risk factor. | Postoperative risk factors: The development of non-abdominal complications such as cerebrovascular or lower extremity ischemia, acute renal failure, postoperative hemorrhage, arrhythmias and low postoperative CO correlates with the development of abdominal complications. On applying these risk factors retrospectively to our patient (age 70, history of heavy smoking, hypertension, diabetes, generalized vascular disease with splenic and kidney infarction, critical illness, on dopamine) we come to the conclusion that he was at risk of developing an abdominal complication and this should bring the attention of the surgeon (either cardiac or general) to apply them routinely for their patients. The incidence of acute cholecystitis (AC) complicating cardiac surgery is 0.2%–0.5%, in other series it was reported as 0.08%, the incidence in KKUH series is 0.07%. The overall low incidence could reflect the rarity of this condition or, alternatively, that only the clinically severe form of AC was identifiable after cardiovascular surgery. Milder forms of AC after cardiovascular surgery could have escaped diagnosis because of lack of clinical manifestation. A prospective surveillance study using strict criteria of imaging of the gallbladder by ultrasonography estimated the incidence of AC at 18% in a group of young trauma patients, many of these cases did not have sufficient clinical manifestations for suspicion of AC because we did not rely on gallbladder imaging alone and did not screen all admissions to ICU for this diagnosis. The incidence of AC after cardiovascular surgery could be underreported. Symptoms usually develop 5–15 days postoperatively, with a median period from heart surgery to onset of abdominal symptoms and signs of 5 days. In our reported case, they started after 3 days while the patient was in ICU. In previous studies, arterial vascular disease, multiple surgical procedures, mechanical ventilation and nosocomial infections were predictors for subsequent development of AC after cardiovascular surgery. Characteristic predictors for AC after cardiovascular surgery were inadequate preoperative oxygen transport, longer times on cardiopulmonary bypass, postoperative bacteremia and cardiac arrhythmia as postoperative cardiac arrhythmia implied periods of global and regional hypoperfusion, continued early during the ICU stay after initial surgery. The diagnosis of acute cholecystitis in the critically ill patient is challenging. Clinical evaluation is delayed by alterations in mental status due to sedation, analgesia and communication problems because of mechanical ventilation. Classical signs and symptoms of acute cholecystitis as right upper quadrant pain with tenderness, nausea and vomiting were not found to be reliably present in previous series as these signs and symptoms could be related to the cardiac procedure and are common among critically ill patient. |
Laboratory findings were non-diagnostic. A leukocytosis was present but appeared to be unrelated to the development of acute cholecystitis in the absence of perforation; mixed abnormalities of liver function tests were found but were nonspecific. These findings are going with previous publications. 

No general agreement exists on the imaging modality of choice. Suggested radiologic investigations include ultrasonography, CT and radionuclide studies. Sensitivity of ultrasonography in the diagnosis of acute cholecystitis has been reported between 30% and 100% with specificity ranging from 80% to 94%. Positive findings include gallbladder distension, wall thickening, pericholecystic fluid and sludge formation. 

We believe that ultrasonography should be the first imaging investigation as it is easily performed at the bedside. CT is also very helpful. However, negative imaging should not be used to exclude acute cholecystitis if clinical findings are suggestive, as false negative studies are possible and delay in treatment may have dire consequences. 

As in many published reports, laparoscopic cholecystectomy is the procedure of choice for acute cholecystitis in the critically ill patient. Mortality is high, ranging from 65% to 100%, probably because of delayed diagnosis, the general condition of most patients and often the surgeon’s reluctance to intervene again and worsen the prognosis. 

CONCLUSION
Early and aggressive treatment of abdominal complications is required if mortality is to be reduced.

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References
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