Insidious Hepatic Portal Venous Gas after Cardiac Surgery: A Rare Case Report and Review of Literatures

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Abstract

Hepatic portal venous gas (HPVG) is a rare finding as it known as an "ominous sign". This paper presented a rare case of HPVG which occurred on the fourteenth days after cardiac surgery, and conduced a systemic review of literatures. It raised a recognition of the warning sign of a delated HPVG in post-cardiac surgery. To our knowledge, this is one kind of insidious HPVG with unconspicuous symptoms but rapid and fatal deterioration. The report is as follows.

Keywords

Hepatic Portal Venous Gas; Cardiac Surgery; Cardiopulmonary Bypass; Non-occlusive Mesenteric Ischemia; Mesenteric Necrosis.

1. Introduction

HPVG, manifests as gas in the portal venous system, is a devastating omen that mostly occur in bowel ischemia with an extremely high mortality rate up to 75% [1]. Along with the popularization and development of radiographic technology, an increasing number of benign HPVG had been reported with conservative managements, such as cause by inflammatory bowel disease, intraabdominal sepsis, liver transplantation, colectomy, esophageal variceal band ligation and some endoscopic intervention [2-4]. HPVG in post-cardiac surgery is rare, as it mainly due to the onset of non-occlusive mesenteric ischemia (NOMI) [5, 6].

2. Presentation of case

A 64-year-old female was admitted into the cardio-surgery department on November 29, 2019. The report of echocardiography showed the stable multi-thrombus formation in the left atrium with degenerated heart valvular disease. She had the precondition of hypertension and coronary arteriosclerosis. After a comprehensive pre-operative evaluation, including chest-abdomen computed tomography (CT), she was diagnosis as, 1. degenerated heart valvular disease with 66% left ventricular ejection fraction (LVEF), NYHA class II; 2. multi-thrombus formation in left antrum; 3. coronary atherosclerotic; 4. atherosclerosis of the superior mesenteric artery (SMA). On November 7, 2019, she went through a successful 7.5 hours cardiac surgery (Aortic valve replacement + Mitral valve replacement + Tricuspid valvuloplasty + Left atrial thrombectomy + One coronary artery bypass grafting) under cardiopulmonary bypass (CPB).

On the 1st day post-operation, the patient recovered autonomous respiration with an elevate body temperature up to 38.4 ℃ and slight jaundice. Blood investigations showed the elevation in white cell count (WBC) 12.84 × 10^ 9 /L, C-reactive protein (CRP) 74.38 mg/L, procalcitonin (PCT) 190.8 ng/ml, serum lactate 13.2 mmol/L, total bilirubin (TBIL) 79.3umol/L, aspartate aminotransferase (AST) 138 U/L, alanine aminotransferase (ALT) 42 U/L, creatine (CREA) 122umol/L and base excess (BE) 5.7 mmol/L. Considerate it longstanding CPB, her treatment was adopted in antibiotic, relieving jaundice and other symptomatic therapy. She restored a total liquid diet on the 2nd day with a soft and flat abdomen in a slight decrease of bowel sound. All the exam indexes went well and she was recovering gradually, however, her liver function drop down with an obvious jaundice

appearance on the 5th day. Blood test showed an elevation of AST (221 U/L) and ALT (338 U/L) with TBIL (201.0 umol/L). She began hemodialysis treatment daily and her jaundice indexes relieve gradually. On the 7th day, physical examination revealed a flat and soft abdomen accompanied with 6-8/min normal bowel-sound, the patient recovered breaking wind. And she began defecation with yellow-soft stool on the 11th day. The blood test also showed an improvement of internal function as a slowing down WBC (15.26 × 10^ 9 /L), serum lactate (2.79 mmol/L), TBIL (154.5 umol/L), AST (41 U/L), ALT (62 U/L) and BE -1.8 mmol/L. Reexamination of echocardiography revealed an improvement of cardiac function with an 71% LVEF.

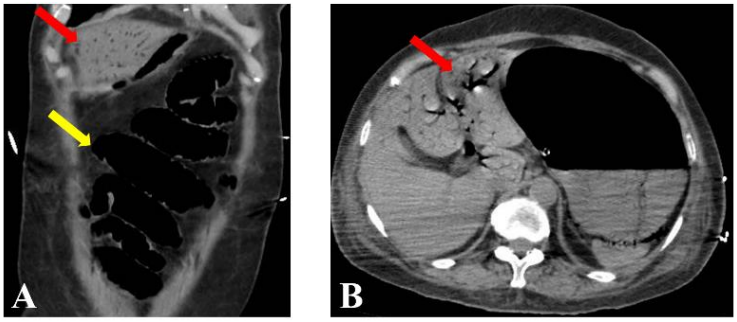

Fig. 1 Abdominal Plain-CT Scan. (A: coronal view, B: cross-section) Showed a large amount of HPVG mainly in the left lobe of liver (red arrowed), accompany with an extensive bowel distension (yellow arrowed).

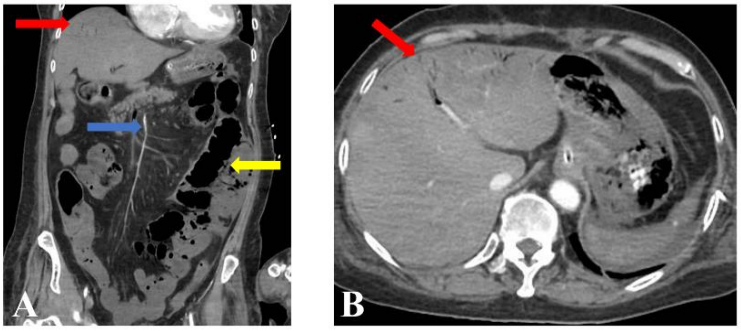

Fig. 2 Abdominal Contract-CT Scan. (A: coronal view, B: cross-section) Showed a reducing HPVG (red arrowed), an extensive bowel distension with low enhancement (yellow arrowed), a poor perfusion in superior mesenteric artery (blue arrowed).

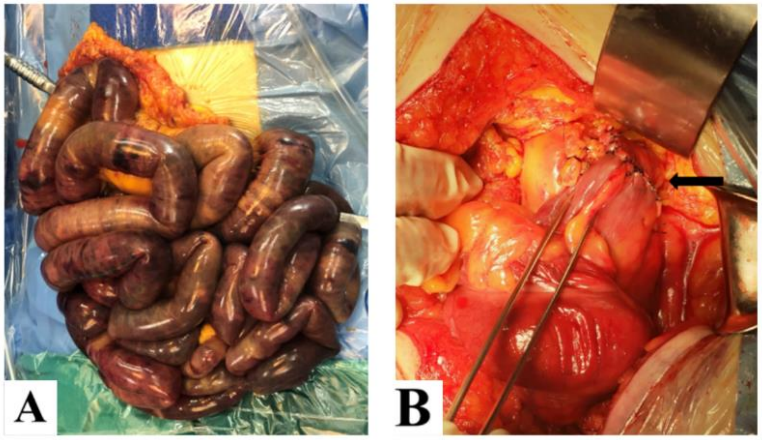

Fig. 3 Laparotomy findings. Showed a large proportion of purple-black bowel ischemia without ulceration or perforation, and a total Jejunum and subtotal ileum resection with terminal ileum-duodenal anastomosis (remain 8cm distal ileum).

In the evening of 13rd day, patient appeared an episode of diarrhea with yellow-green watery stool. However, her situation suddenly aggravated on the next morning. The patient became poorly spirit complained about abdominal pain. Physical examination revealed an abdominal distention with a weak bowel-sound, extensive tenderness without rebound tenderness. Arterial blood gases indicated a hypoxia condition with a compensated metabolic acidosis. The serum lactate was going up to 14.03 mmol/L with a deficiency of BE -8.4mmol/L. Emergency plain CT revealed an extensive bowel distention with extensive HPVG sign, without obvious sign of bowel ischemia (Fig. 1). Considering a diagnosis of paralytic ileus, conservative therapy was adapted with continuously gastrointestinal decompression, emergency hemodialysis and other supportive treatment.

At noon, her condition became worsens as she became tachypnea and finally septic shock. A reexamination of contract-CT scan revealed an extensive bowel ischemia with reducing HPVG (Fig. 2). She was sent to an emergency laparotomy. Laparotomy revealed a subtotal small bowel necrosis without perforation or gangrenous (Fig. 3A). A successful total Jejunum and subtotal ileum resection with terminal ileum-duodenal anastomosis were performed (Fig. 3B). The next afternoon, her condition got worsen with multiple organ failure. Her family decided to cease therapy and the patient died shortly. The primary diagnosis of death is NOMI, and it was confirmed by the post-operational pathology as no thrombosis formation in the SMA.

3. Discussion

Hepatic portal venous gas, a rare radiological finding, firstly introduced in infants with necrotizing enterocolitis in 1955 [4]. Its fatal consequence raised a great concern due to its 75% mortality rate in which bowel necrosis occupy 72% of those cases [7]. As the popularization and development of radiographic technology, an increasing HPVG had been reported with various cause, broadly categorized as bowel disease (such as mesenteric infarction, intestinal obstruction, inflammatory bowel disease, perforation, diarrhea), systematic disorder (suppurative cholangitis, intra-abdominal abscess, abdominal trauma, seizure, chemotherapy, chronic obstructive pulmonary disease), others iatrogenic or idiopathic causes and so on, mesenteric ischemia still occupies the majority [8-12]. More and more benign HPVG as well as its timely treatment, the overall mortality rate of HPVG reduces to 29–39% [13, 14]. However, there is scant literature of HPVG relate to cardiac surgery which is vulnerable to ignore.

The exact pathogenesis of HPVG is still unclear, currently recognized as: (1) Mechanical: the increasing pressure of intra-bowel push the hyper-pressure air flow into the portal venous system under microcirculation; (2) Bacterial translocation: some cause of bacteremia, the disturbance of intestinal flora or even the normal bowel flora could migrate from the submucosa into the bloodstream, producing gas in the portal system [10]; (3) Impairment of mucosal barrier: the damaged mucosa is more vulnerable to gas and bacteria [7, 15, 16].

HPVG demonstrates a peripheral gas sign of the liver capsule, called “centrifugal lucencies”, predominantly in the anterior segments of the left liver lobe, while pneumobilia demonstrates a “centrally lucencies” within the liver center due to its centripetal flow of bile [17-19]. Those difference can be easily distinguished in CT-scan as it becomes the golden standard for diagnosis. Besides that, ultrasound could be used as a quick detection method especially in infant, manifested a hyper-echoic or dot-like foci within the portal veins or liver parenchyma [20, 21]. But its limitation of inter-operator variability and low sensitivity restrict its application [22].

Non-occlusive mesenteric ischemia, a hazardous types of acute mesenteric ischemia (AMI), was firstly reported in 1958 in cardiac surgery [23]. The total incidence rate of AMI in cardiac surgery is 0.07-0.5% with high mortality of 37-77% [24-26], in which NOMI occupies its 20–30% proportion as its major part [5, 6]. The etiology of NOMI is wildly recognized as low cardiac output or vasospasm, mainly causes by shock, septicemia dehydration or vasoactive inotrope [24, 27, 28]. Advanced age, peripheral arterial disease, sepsis, arrhythmia, prolonged ventilation and cardiopulmonary bypass are regarded as its dangerous predisposition [24, 26, 29, 30]. Angiography is the gold standard diagnosis

method as well as therapeutic tool [31]. Besides that, contrast-CT scan is regarded as a non-invasive "gold standard" with quick diagnosis and easier access.

When it came back to this case, a high-age woman developed a NOMI with the sign of HPVG after cardiac surgery. The damaged intestinal mucosa is the crucial etiology. Both intro-operative CPB state or post-operative sepsis state contribute to its mesenteric hypoperfusion[32]. The damaged mucosal barrier leads to the escape of gas as well as gas-produce bacteria from bowel to the portal system, result in HPVG. Besides that, the compromised of intestinal mucosa could further aggravates the systemic inflammation even bacteremia, conjugating the consequence of dead. However, the asymptomatic median window from cardiac surgery to the diagnosis of NOMI is 14 (10.3-20.3) days, that "delayed" complication could relax clinician's vigilance easily[30]. Furthermore, the rapid aggravate nature of NOMI also contribute to its high mortality.

Early intervention is particularly crucial. The bowel ischemia after cardiac surgery is rare. HPVG alone is not an emergency surgery indicator, but when it appeals following cardiac surgery, possibly represent an ominous sign. Once serious abdominal pain occur, emergency laparotomy should be more aggressive if necessary.

The present report describes a rare case of delay HPVG after a successful cardiac surgery. An episode of diarrhea accompanied with abdominal pain interrupted the process of recovery with rapid deterioration, the patient finally died although emergency laparotomy was performed.

About HPVG after cardiac-surgery, several recognition should be raised: first, difficulty in early diagnosis, the unconspicuous symptoms as well as the large variance span of onset time are easy to lose the alert of bowel ischemia; second, the importance of comprehensive evaluation before cardiac surgery, routinely abdominal CT-scan could evoke the alerts of probable post-surgery complications; third, revascularization of the severe narrow mesenteric vessel before cardiac surgery, a large proportion of cardiac patients developed various degree of stenosis within SMA, which can be treated in endovascular approach; fourth, pay close attention to the post-operative recovery as the intestinal ischemia could occur and deteriorate suddenly; fifth, the sign of HPVG after cardiac surgery indicates a highly potential of bowel ischemia; sixth, difficulty in decisive therapy, emergency laparotomy should bases on the hematological and examinational profiles accompanied with the overall dispositions of patient; seventh, high mortality rate, the sign of HPVG after cardiac surgery is rare with poor prognosis in spite of timely intervention.

4. Conclusion

Since benign HPVG has been increasing with conservative therapy, contract with the rarity of HPVG after cardiac surgery, ischemia alert should keep in mind. For those who has high risk of mesenteric ischemia, surgeons are supposed to keep a high suspicion of NOMI after cardiac surgery, HPVG sign probably indicates the onset of bowel necrosis and timely laparotomy are mandated.

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