Gastric bleeding detected by transesophageal echocardiography during cardiopulmonary bypass

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Abstract Gastric bleeding during cardiopulmonary bypass (CPB) is a rare but life-threatening complication for patients undergoing cardiac surgery. We present a case of a patient undergoing coronary bypass grafting in whom we detected gastric bleeding by transesophageal echocardiography (TEE) during CPB. After separation from CPB, bleeding was stopped by endoscopic clipping of the mucosa. If a fluid deficit is noted and TEE reveals a hypoechoic space within the stomach, gastric bleeding might be suggested.

Keywords Gastric bleeding · Transesophageal echocardiography · Complications

Introduction

Low cardiac output or any intervention leading to a low cardiac output state impairs splanchnic blood flow and increases the risk for gastrointestinal bleeding during cardiac surgery [1]. During cardiopulmonary bypass (CPB) in particular, low perfusion pressure, hypothermia, and systemic heparinization may predispose to mucosal damage and bleeding [2].

We present a case of gastric bleeding detected by transesophageal echocardiography (TEE) during CPB.

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TEE is a safe and effective procedure frequently used for intraoperative monitoring in patients undergoing cardiac surgery. Early detection of the gastric bleeding by TEE and active treatment with iced saline gastric lavage, administration of a thrombin product, and endoscopic gastric mucosal clipping successfully terminated the bleeding.

Case report

A 68-year-old man (158 cm, 60 kg) with episodes of chest pain at rest presented for coronary artery bypass graft surgery. He had a history of old myocardial infarction, hypertension, diabetes, brain infarction, and lumbar spinal cord stenosis. He had no evidence of gastric ulcer or gastritis by endoscopic examination 6 months before surgery, but he was empirically prescribed a histamine-2 receptor antagonist because of the use of nonsteroidal antiinflammatory drugs (NSAIDs) for lumbago caused by his spinal cord stenosis. The patient had taken aspirin 100 mg daily as anticoagulant therapy for the 3 years before surgery, but it was stopped 7 days before surgery, and the prothrombin time was not prolonged. Electrocardiography showed normal sinus rhythm with slight ST-T changes at rest. Transthoracic echocardiography showed inferior wall hypokinesis and low global left ventricle (LV) function. Coronary angiography revealed 75–100% stenosis of all three coronary vessels, and left ventriculography showed reduced cardiac wall motion.

General anesthesia was induced with intravenous propofol (25 mg initial dose followed by 5 mg kg^{-1} h^{-1}), ketamine (50 mg), remifentanil (0.1–0.4 μg kg^{-1} min^{-1}), and rocuronium (40 mg), and the patient was intubated with a tracheal tube. The patient was monitored with five-lead electrocardiography, invasive arterial pressure

monitoring, pulmonary arterial and central venous pressure measurement, pulse oximetry, capnometry, and TEE.

A TEE omniplane probe covered by a lubricated protective sheath was inserted uneventfully into the esophagus. Probe controls were in the neutral position to allow the probe to follow the natural course of the esophagus. Heart images were obtained from the standard position, and a midesophageal bicaval view was obtained for pulmonary artery catheter insertion. After catheter insertion, the heart was imaged in the transgastric midpapillary short-axis view for monitoring of regional wall motion abnormality. The angular fixation device to lock the probe was not used. Before CPB, a bolus of heparin 18,000 U was given to raise the activated coagulation time to 521 s. After systemic heparinization, CPB was established by cannulation of the ascending aorta and right atrium. After anesthetic induction, the patient's hemoglobin and hematocrit were 10.4 g/dl and 29.9%, respectively, but decreased to 7.4 g/dl and 22.5%, respectively, at the beginning of CPB because of bleeding in the surgical field. Myocardial revascularization was performed with right and left internal thoracic artery and saphenous vein grafts. We noticed the blood volume in the reservoir to be gradually decreasing and treated the hypovolemia with infusion of crystalloids (13,360 ml) and transfusion of blood products (840 ml) to maintain the reservoir level during CPB. We suspected complications from either venous cannulation or retention of blood in the surgical field including the intrathoracic cavity, but neither could be identified. However, TEE revealed a hypoechoic space in the stomach (Fig. 1). At this point (45 min from the start of CPB), hemoglobin and hematocrit had dropped to 6.5 g/dl and 19.6%, respectively. We thought that serious gastric bleeding might have occurred. In consideration of TEE-associated gastric complication, we stopped TEE monitoring and removed the probe. We inserted a nasogastric tube, which resulted in immediate drainage of 500 ml red blood. The patient was

Fig. 1 Hypoechoic space in the stomach revealed by transesophageal echocardiography (TEE)

treated with intravenous administration of a histamine-2 receptor antagonist, iced saline gastric lavage, and administration of 5,000 U thrombin product through the nasogastric tube. CPB lasted for 240 min, and separation was done with the help of dopamine, dobutamine, and noradrenalin. Just after separation, hemoglobin and hematocrit were 5.8 g/dl and 17.5%, respectively. After heparin reversal with protamine 150 mg, hemodynamic status was unstable, and the hematocrit was 17–20% despite the transfusion of an additional 2,500 ml blood products, suggesting that the gastric bleeding was continuing. We performed immediate endoscopic examination and although no mucosal tear was seen near the gastroesophageal junction and esophagus, we found several erosions and sites of bleeding at the greater curvature of the stomach. These lesions were successfully treated with mucosal clipping, after which hemodynamics immediately stabilized. Thereafter, hemoglobin and hematocrit gradually rose to 12.1 g/dl and 33.5%, respectively. In total, 1,500 ml red blood was drained via the nasogastric tube, and 4,840 ml blood products was given. Total input/output balance was +13,980 ml. This excess fluid balance made it difficult to wean the patient from mechanical ventilation; however, the patient was extubated on the fifth postoperative day and made a full recovery.

Discussion

This case describes a patient with massive gastric bleeding detected by TEE during CPB.

Perioperative gastric bleeding in patients on CPB is rare, with an incidence ranging from 0.33% to 0.87%; however, the mortality rate ranges from 0% to 33% [1–8]. The main causes of gastric bleeding during cardiac surgery are gastric ulcer, acute gastric mucosal lesions, and TEE-associated complications [3, 9].

Gastric ulcer and acute gastric mucosal lesions are the most frequent perioperative gastrointestinal complications [4]. Although the pathogenesis of gastrointestinal lesions is complex and multifactorial, the major factor implicated in cardiac surgery is reduced systemic blood flow, which leads to inappropriate oxygen delivery and energy deficit. Hypovolemia, prolonged CPB, and administration of vasoconstrictors can cause gastrointestinal hypoperfusion [5]. CPB is associated with a broad range of systemic complications, including nonpulsatile flow, hemolysis, activation of the inflammatory cascade, anticoagulation, hypothermia, and, finally, reduced end-organ perfusion [4].

The reported incidence of TEE-associated gastrointestinal bleeding is low at 0.03% [10]. Direct trauma to the gastrointestinal tract may result from blind insertion and advancement of the probe, large movements of the probe
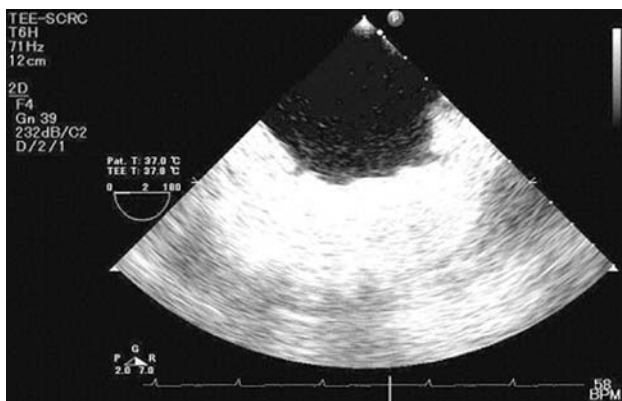
tip during flexion and manipulation required to obtain certain images, and the presence of unknown gastrointestinal pathology. Indirect trauma of the gastrointestinal tract may be related to excessive or prolonged continuous pressure at the TEE probe–mucosal interface that can result in tissue ischemia and necrosis [9, 11]. Alternatively, gastrointestinal injury may occur secondary to thermal injury produced by piezoelectric crystal vibration at the TEE probe tip or from ultrasound energy absorption by adjacent tissue [11, 12]. Risk factors that can precipitate gastric bleeding caused by TEE following cardiac surgery include a previous ulcerative process, vasoactive drug utilization, and absence of H2 antagonist in the perioperative period. Other factors such as a long CPB period, urgent surgery, reoperation, or aspirin use have also been implicated [13].

Our patient did not have clear evidence of a gastric ulcer, but a histamine-2 receptor antagonist had been prescribed until the day before surgery. Although our patient had several risk factors for gastric bleeding such as preoperative anticoagulation, prolonged CPB, and peripheral vascular disease in addition to the insertion of the TEE probe while he was fully heparinized for CPB [3], we omitted the histamine-2 receptor antagonist as a preoperative medication. We do not routinely use histamine-2 receptor antagonist during cardiac surgery because its use may increase postoperative infection rate [14, 15].

Several cases of gastrointestinal bleeding from a Mallory-Weiss tear caused by TEE have been reported [9–13, 16, 17]. In our patient, however, several erosions and bleeding sites were seen at the greater curvature of the stomach. There appeared to be no tear caused by the TEE probe, but there was evidence of acute gastric mucosal lesions and an old gastric ulcer. Endoscopic findings showed that the gastric bleeding might not have been caused by a TEE complication but rather by acute mucosal lesions and the old ulcer. The gastric bleeding might have been caused by the stress of surgery, preoperative administration of NSAIDs and aspirin, and absence of the histamine-2 receptor antagonist [4]. Although we were unable to discover the cause of the gastric bleeding during CPB, TEE allowed us to detect and immediately treat the bleeding.

In previous reports, patients with gastrointestinal bleeding were treated successfully by conservative histamine-2 receptor antagonist therapy [4, 13]. De Vries et al. successfully stopped massive bleeding using iced saline gastric lavage and endoscopic infiltration of 5 ml adrenaline solution (100 µg ml^{-1}) for bleeding [16]. Kihara et al. treated their patient successfully using endoscopic argon plasma coagulation therapy [11]. Our patient may have recovered after conservative therapy without endoscopic treatment. However, we thought it necessary to actively treat our patient during this critical situation. Iced saline gastric lavage, administration of thrombin product, and endoscopic gastric mucosal clipping were successful, resulting in hemodynamic stabilization.

Although gastric bleeding is a rare and unusual complication during cardiac surgery, special attention should be paid to the reservoir level during CPB. If a fluid deficit is noted and complications of venous cannulation and retention of blood in the surgical field and intrathoracic cavity are ruled out, TEE may reveal a hypoechoic space within the stomach. If gastric bleeding is recognized, prompt diagnosis and adequate treatment may be needed to avoid further complications from massive bleeding or excessive fluid balance resulting from blood transfusion.

In conclusion, gastric bleeding during cardiac surgery is rare, but this complication may lead to a critical situation. TEE monitoring appears to be useful not only to evaluate cardiac performance but also to detect unexpected gastric bleeding during cardiac surgery.

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