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Case Report

Delayed stomach necrosis in a patient with injured celiac artery branches after penetrating abdominal trauma

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ABSTRACT

Injuries of the celiac artery and its branches are rare, but potentially lethal. Ligation of these arteries is performed to control significant hemorrhage. However, few reports have described the adverse effects of ligating these arteries.

A 69-year-old woman with a self-inflicted stab wound was brought to our hospital. Her blood pressure could not be measured, therefore aortic cross-clamping was performed, and epinephrine was administered for resuscitation, an emergency laparotomy was performed, and the roots of splenic artery and common hepatic artery were ligated. The left gastric artery which was anomalous and arose directly from the aorta, was also injured and had to be ligated. Norepinephrine was required after the surgery. Enhanced computed tomography performed on hospital day 4 revealed a disrupted celiac artery. The patient developed gastric necrosis on hospital day 23 and, hence, underwent total gastrectomy was performed.

The possibility of delayed stomach necrosis should be considered during the postoperative management of patients who undergo ligation of all of the celiac artery branches and experience global hypoperfusion after the surgery.

Introduction

Celiac artery branch injury (CABI) is rare but life-threatening and has a reported mortality rate of 38–75 % owing to celiac artery injury (CAI) [1]. Penetrating injuries account for 90–95 % of these cases [2]. In most cases of CABI, an emergency laparotomy for hemostasis is required, and ligation of the celiac artery is well tolerated [4]. However, few studies have examined the adverse effects of ligating the celiac artery and its main branches. In addition, global hypoperfusion could cause gastrointestinal ischemia.

This report describes a case of CABI due to a self-inflicted stab wound, for which an emergency laparotomy was performed and the branches of the celiac artery were ligated. The patient was under a state of hypoperfusion after the surgery, and subsequently developed gastric necrosis. We report the case along with the relevant literature.

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Case

A 69-year-old woman stabbed herself in the abdomen with a kitchen knife with suicidal intent and was admitted to our emergency department. On arrival, her blood pressure could not be measured, and the cardiac monitor showed pulseless electrical activity. Her body temperature was 35.0 °C. Oxygen saturation was 99 % with a 10-L oxygen reservoir mask. She had a Glasgow Coma Scale score of 3 (E1V1M1) and developed agonal respiration. A wound measuring 5 cm was present on her upper abdomen. Ultrasonography revealed intra-abdominal fluid collection. An emergency thoracotomy was performed, and, subsequently, aortic cross-clamping and open cardiac massage were performed to maintain blood pressure. The aorta was clamped for 15 min, and 1 mg of epinephrine was administered thrice at 3-min interval for resuscitation. Temporary return of spontaneous circulation was observed, but she was still hemodynamically unstable. A laparotomy was performed, revealing injuries to the common hepatic and splenic arteries, pancreas, spleen, and liver. The injured arteries were ligated to achieve hemostasis. The left gastric artery was not identified; however, it seemed that there was a branch arising directly from the aorta that was also injured; thus, ligation was performed on the branch as well. After that, distal pancreatectomy and splenectomy were performed, and the liver was sutured. Norepinephrine was administered at 0.25 µg/ kg/min intraoperatively. A second-look surgery the following day revealed no signs of active bleeding or any ischemic change macroscopically. On hospital day 3, no vasopressor requirement was noted, and abdominal wall closure was performed. Regular examinations were conducted to detect ischemic changes during her stay in the intensive care unit. An enhanced computed tomography (CT) scan on hospital day 4 demonstrated disruption of the celiac artery. The gastroduodenal artery was seen arising from the superior mesenteric artery as collateral flow. Other arteries, that supply the stomach, such as the right gastric arteries, left and right gastroepiploic arteries, and short gastric arteries, were not observed during CT angiography (Fig. 1). Gastroscopy was performed for follow-up on hospital day 9, and revealed patchy mucosal necrosis on the gastric upper body (Fig. 2). Despite administration of conservative treatment, there was no significant change even after 2 weeks. However, on hospital day 23, the patient developed a fever of 39 °C, and complained of pain in the stomach. Blood examination was conducted revealing a white blood cell count of 34,000/mm³, C reactive protein of 13.4 mg/dL. CT scan was performed and demonstrating the presence of air in the gastric wall, and intraabdominal free air. Gastric necrosis was suspected and, subsequently, gastroscopy was performed revealing extensive mucosal necrosis on the stomach (Fig. 3). An emergency surgery revealed necrosis of the stomach; hence, total gastrectomy with Roux-en-Y reconstruction was performed. Histological findings of the stomach revealed diffuse necrotic changes, (significant degeneration and necrosis of the tissues on the mucosal side of the gastric wall), and various inflammatory cell infiltrations (including numerous neutrophils and eosinophils). No evidence of invasive fungal infection was noted. Due to a leakage on the duodenal stump identified on hospital day 29, continuous tube drainage was performed. Her consciousness became clear and she underwent rehabilitation in bed for a few weeks. However, she developed sepsis due to multidrug-resistant Pseudomonas aeruginosa infection and disseminated intravascular coagulation. Subsequently, the patient's general condition gradually deteriorated, and she died 70 days after admission.

Discussion

Blood flow to the upper abdominal viscera is mainly supplied by branches of the celiac artery, i.e., common hepatic, splenic, and



Fig. 1. Enhanced computed tomography (CT) scan shows disruption of the celiac artery(arrow) and gastroduodenal artery arising from superior mesenteric artery(arrowheads) on hospital day 4.



Fig. 2. Gastroscopy shows patchy mucosal necrosis in the upper body of the stomach on hospital day 9.



Fig. 3. Gastroscopy shows extensive mucosal necrosis of the stomach on hospital day 23.

left gastric artery, and hence various complications due to interruption of blood flow may occur when the celiac artery is injured. Whereas the celiac artery has several anatomical variants, and, in the present case, the left gastric artery arose directly from the aorta, an occurrence which was reported to be 0.40 % in a systematic review [3]. In this case, the main branches of the celiac artery were injured by penetrating trauma, making equivalent to a case of CAI.

Delayed necrosis of the stomach can occur after ligation of the celiac artery, which is reported as being well tolerated [4]. In a review on 11 cases of CAI, all 3 patients who underwent ligation of the celiac artery survived [5]. Thus, this review suggested that ligation could be performed to promote hemostasis in hemodynamically unstable patients, who require damage control surgery. However, a few studies reported delayed necrosis as a complication. A case was reported in which the celiac artery was accidentally ligated during pediatric surgery, and stomach necrosis developed on post-operative day (POD) 59 [6]. In another case, liver failure was noted 6 days after abdominal trauma with CAI [7]. Necrosis probably developed gradually as the blood flow decreased slowly. In a pediatric case, necrosis occurred on POD 59 [6], which is long after the ligation of the celiac artery, indicating that blood flow was maintained to some extent even after the ligation of the celiac artery. In the current case, the root of the celiac artery was not ligated; however, the branch arteries were ligated; therefore, it is comparable with a case of CAI. Patchy necrosis was detected by gastroscopy

on hospital day 9, indicating that the obstruction to blood flow may have developed gradually and was partial. The post-operative course was uneventful until reoperation was performed; hence, the circulatory disturbance can be considered as having developed slowly. In addition to the ligation of celiac artery, the aortic cross-clamping and vasopressor use in the resuscitative phase could have caused low blood flow to the stomach. A state of hypoperfusion in the diuretic phase could have influenced the blood flow to the organs. An observational study suggested that aortic cross-clamping affects splanchnic hypoperfusion, and causes subsequent gastrointestinal dysfunction in patients with abdominal aortic aneurysms [8]. Another observational study revealed administration of vasopressors, e.g. vasopressin and norepinephrine, is associated with acute mesenteric ischemia [9].

In the presented case, further interventions could have been undertaken, given that patchy mucosal necrosis was observed in gastroscopy on hospital day 9. CT angiography should have been performed again to detect the low blood flow to the stomach, therefore, further treatment should have been initiated, such as vasodilator administration, blood transfusion, and fluid infusion. The effectiveness of vasodilators has been reported against visceral ischemia, such as non-occlusive mesenteric ischemia [10]. Duodenal stump leakage occurred 5 days after the gastrectomy. It could be affected by the ligation of the common hepatic artery. Blood flow from the gastroduodenal artery could have been decreased, which would have influenced the healing of the duodenal stump. Tube drainage was performed, which seemed to be effective to some extent. However, finally, the patient died of septic shock 70 days after admission.

Conclusion

We encountered a case of delayed gastric necrosis following the ligation of the celiac artery branches and global hypoperfusion. Thus, the possibility of delayed necrosis in a patient with CABI should be considered during postoperative management.

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Not applicable.

Ethical approval

Not required.

Informed consent

Written informed consent was obtained from the patient's family for publication of this case report and any accompanying images.

Declaration of competing interest

None.

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