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Case Report

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Gastric Necrosis – An Untold Catastrophe in Splenectomy: How we Manage in a Surgical District Hospital

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1. Abstract

Gastric necrosis post splenectomy carries a high risk of mortality and should be identified and diagnosed early. It is a rare event, only reported in less than 1% of splenectomies. We would like to report a 57 years old lady involved in an alleged MVA causing a splinic injury grade 5 underwent an exploratory laparotomy with splenectomy we advocate an endoscopic is a safe and feasible method in diagnosing the extend of gastric necrosis and able to manage the bleeding from the slough from gastric mucosa by using endoscopic clips and by injecting adrenaline. However, in case of extensive bleeding from gastric mucosa an exploratory laparotomy with on table endoscopic underruning suture at the bleeding at gastric fundus can help to avoid a partial gastrectomy.

2. Introduction

Emergency splenectomy post trauma has been known to carry a high morbidity with its complications. Such complications include gastric necrosis, pancreatic duct injury causing fistula and overwhelming post-splenectomy infection (OPSI). These complications, although rare, are challenging to be treated. We report a case of a lady who developed gastric necrosis post splenectomy from a motor vehicle accident (MVA), and how we managed her in a district hospital setting. We advocate an endoscopic is a safe and feasible method in diagnosing the extend of gastric necrosis and able to manage the bleeding from the slough from gastric mucosa by using endoscopic clips and by injecting adrenaline. However, in case of extensive bleeding from gastric mucosa an exploratory laparotomy with on table endoscopic underruning suture at the bleeding at gastric fundus can help to avoid a partial gastrectomy.

3. Case Report

A 57 years old lady with underlying Diabetes Mellitus, Hypertension and Dyslipidaemia was involved in an alleged MVA. She had loss of consciousness post trauma, with retrograde amnesia, and abdominal pain. Upon arrival to the emergency department, she was drowsy, tachypneic, and tachycardic. Clinical examination revealed pallor, abdominal bruising and generalised peritonitis. A bedside FAST scan showed free fluid at right upper quadrant, and left upper quadrant. She was hypotensive, and despite fluid resuscitation, she was a non-fluid responder. In view of the patient's response, she was subjected to an explorative laparotomy. Intraoperative findings revealed a shattered spleen grade 5 and liver injury grade 3. A splenectomy was performed and she was admitted to the intensive care unit (ICU) for post-op stabilisation and close monitoring.

On day 8 post-op, she developed bouts of hematochezia. A proctoscope examination was done which noted blood clots, but no oozing, no spurting, and no evidence of SRUS. An urgent OGDS revealed a sloughy area with streakiness of necrotic tissue over gastric fundus region (Figure 1). She underwent a relaparotomy. Intraoperative findings was an infected hematoma at the splenic bed and a peritoneal washout and drain was inserted.

The patient had an episode of hematemesis seven days after the relaparotomy. She was subjected to a re - OGDS which showed a huge Forrest III ulcer at the fundus, obscured by a huge blood clot, and the mucosa looks very thin with no active bleeding noted. A

repeated OGDS on the next day noted a fundal necrosis complicated with ulceration and mucosal sloughing. (Figure 2). Her condition did not improve, and was posted for a 3rd relaparotomy with underruning of stomach ulcer. There was a huge ischaemic gastric ulcer at the posterior wall. The affected segment was underunned and plicated using non absorbable prolene 3/0. However, the intraabdominal collection over the splenic bed, and left pleural effusion was drained percutaneously via ultrasound guidance. Post operative, patient had to be nursed in ICU for antibiotics and oxygenation support via tracheostomy. Reaasesment OGDS was done 2 weeks later showed intact sutures and reduction of the area of necrosis (Figure 3 and 4). Fortunately, patient was discharged home well.

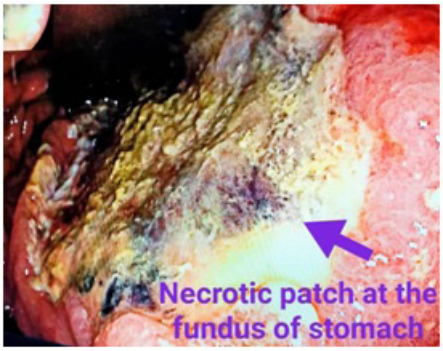

Figure 1: showed an OGDS findings area of necrotic patch over the fundus of the stomach with contact bleeding.

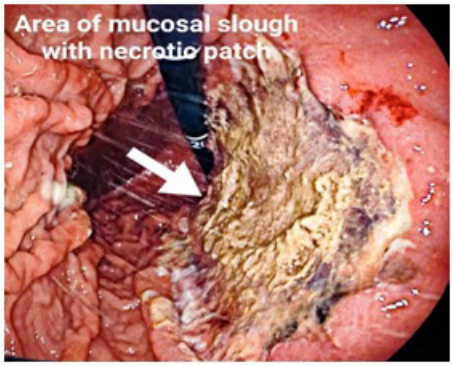

Figure 2: showed an OGDS findings area of mucosal sloughing with patches of necrosis and ulceration at fundus of the stomach.

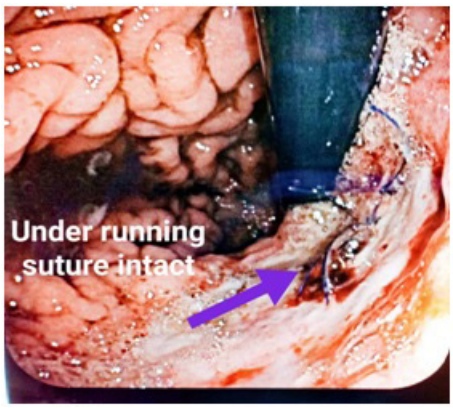

Figure 3: showed an OGDS findings resolution of mucosal sloughing and necrosis. The underrunning sutures were intact. No evidence of bleeding.

4. Discussion

The stomach is a highly vascularized organ located in the intraabdominal cavity. It has a rich intramural and extramural anastomotic network and is mainly supplied by the branches of the coeliac trunk which are the left gastric artery, common hepatic artery and splenic artery. The splenic artery gives rise to the left gastroepiploic artery, short gastric arteries and pancreatic branches. The short gastric arteries supply the fundus of the stomach [1]. With the vast blood supply that the stomach has, it makes the organ resistant to a postoperative ischemic event. However, there are certain surgical procedures that may disrupt the blood supply, inadvertently causing an ischemic event such as gastric necrosis, and more cases are being reported today [2].

Gastric necrosis after splenectomy is a devastating complication, which carries a high mortality rate ranging 53 to 79% [3]. It normally occurs after traumatic close ligation of the short gastric vessels near to the greater curvature of the stomach. The splenic gastric ligament is in close contact with the gastric wall, and during close ligation of the short gastric vessels, there may be involvement and trauma to the gastric wall, which leads to vascular insufficiency to the affected area, thus leading to ischemia and gastric necrosis [4]. Factors that lead to gastric necrosis post splenectomy can be divided into patient and surgical factors. Patient factors such as atherosclerosis, diabetes mellitus, systemic hypotension, vasculitis, steroids and disseminated thromboembolism carry a significant risk [5]. Surgical factors normally depend on the type of surgery. There are surgical procedures that can lead to gastric necrosis, although rare. For instance, in fundoplication, the patient has a risk of developing gastric necrosis. During the mobilization of the fundus of the stomach in an attempt to create non-tension anti-reflux valves, short gastric vessels supplying the fundus may be needed to be ligated and released, further subjecting that region to vascular deficiency. This could lead to localized gastric necrosis. Other procedures, such as proximal gastric vagotomy can lead to necrosis of the lesser curvature of this stomach, in view of the limited blood supply of that region [6]. Colorectal surgeries especially left hemicolectomy, during mobilization of the left colon, can severe the blood supply to the greater curvature of the stomach. The type of surgery, whether it is an emergency or elective splenectomy, carries different risks as well. Emergency splenectomies are associated with high risk of gastric necrosis compared to elective splenectomies.

Patients normally present with vomiting, upper gastrointestinal bleeding, abdominal pain, and ongoing sepsis most likely due to localized intraabdominal collection from the affected area. Signs and symptoms usually arise between the 2nd and 10th day post-operatively, and a high index of suspicion is needed to accurately

diagnose this condition. It is vital for us clinicians to understand the histological changes that occurs in gastric necrosis. Early changes would include capillary dilatation, mucosal edema, vascular congestion, followed by superficial necrosis. This will then progress to mucosal coagulative necrosis, and as ischemia continues, it results in full-thickness hemorrhagic necrosis leading to deep ulceration of the gastric wall.

Once there is suspicion of gastric necrosis, the next step for diagnosis workup would be endoscopy, with addition of imaging studies such as a CT scan. Endoscopy offers a quick and early diagnosis, and it also allows us to assess the extent of gastric necrosis. In our patient, she showed signs of upper gastrointestinal bleeding post-operatively, which raised our suspicion and we proceeded with an OGDS which revealed patches of necrosis over the gastric fundus, further confirming our diagnosis, saving time and hasten our next step of management, which was an emergency laparotomy.

The management of gastric necrosis can be divided into conservative, endoscopy and surgery. Conservative treatment involves close observation fluid resuscitation, nasogastric tube placement for gastric decompression, aggressive acid reduction therapy with intravenous PPI, and selective use of broad-spectrum antibiotics. Patients would require a repeat OGDS to reassess to affected necrotic site. Patients might also be complicated with intraabdominal collection, and these collections should be drained percutaneously. Endoscopically, if the area of necrosis is small, clips can be applied to approximate the affected area. Cyanoacrylate glue, fibrin sealant glue and thrombin can also be applied to necrotic segment, further preventing the sequelae of gastric necrosis. The type of surgery strongly depends on the general condition of the patient, along with the conditions of the peritoneal cavity and surgical field, in terms of the degree of contamination, and the extent of gastric necrosis. Areas of gastric necrosis that are distal can be managed with distal gastrectomy. Patients that are complicated with a gross extent of gastric necrosis, with large perforation will benefit from partial or total gastrectomy [7]. However, in cases where there are patches of necrosis that are localized to the fundus, a simple surgical procedure such as underrunning of the affected mucosa by plicating with surgical sutures is enough to address the issue, as evident from our patient. A gastrostomy was also performed, as it serves as a method to deviate and drain excess gastric juices, which further aids to the healing of the affected mucosa. There are also a few steps that a surgeon can make, during a splenectomy, in an attempt to prevent such a complication. Although it is difficult in a patient post trauma with a clouded surgical field from hematoma and blood clots in the peritoneal cavity, is important for a surgeon to carefully identify the gastrosplenic ligament and its nearby structures. Care should be taken not to involve the gastric wall during ligation of the short gastric vessels. It has also been advocated for surgeons to plicate the upper greater curvature of the stomach or fundus, to invert them, in order to prevent complications such as perforation from gastric necrosis after a splenectomy [8].

5. Conclusion

A surgeon must have a differential of gastric necrosis in mind, especially when a patient is deviating from the normal post-operative course after a splenectomy. As evident from our patient, OGDS is the mainstay in diagnosing gastric necrosis by assessing the affected mucosa. If an intraabdominal collection is suspected, then a CT scan should be done to assess the size and the collection should be drained. Based on our case, we advocate an endoscopic is a safe and feasible method in diagnosing the extend of gastric necrosis and able to manage the bleeding from the slough from gastric mucosa by using endoscopic clips and by injecting adrenaline. However, in case of extensive bleeding from gastric mucosa an exploratory laparotomy with on table endoscopic underruning with simple plication of the necrotic gastric mucosa with non absorbable sutures, can serve as a less invasive at the bleeding at gastric fundus can help to avoid a partial gastrectomy.

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