Superior Mesenteric Artery Syndrome with Massive Gastric Dilatation

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Superior mesenteric artery (SMA) syndrome is a rare disorder characterized by extrinsic compression of the third portion of the duodenum between the superior mesenteric artery and aorta, resulting in intermittent obstruction, thereby resulting in proximal duodenal and stomach dilatation. Although the most characteristic symptoms are postprandial epigastric pain, fullness, voluminous vomiting, and eructation, severe symptoms including acute massive gastric dilatation to the extent of surgical abdomen was rarely reported. We report a case of SMA syndrome in a 24-year-old patient with an eating disorder. CT and an upper gastrointestinal contrast series revealed massive gastric dilatation which induced vascular compressions. Endoscopy showed deep extensive ulcerations of the whole stomach with duodenal necrosis and ischemia, which prompted immediate surgical laparotomy, but no remarkable intra-abdominal peritonitis evidence was noted. We treated the patient conservatively and the patient recovered from all the symptoms. (Korean J Helicobacter Up Gastrointest Res 2014;14:268-272)

Key Words: Superior mesenteric artery syndrome; Eating disorders; Gastric dilatation

INTRODUCTION

Superior mesenteric artery (SMA) syndrome is a rare condition resulting from compression of the third portion of the duodenum by the SMA. Patients may present with acute or chronic forms of SMA syndrome. The acute presentation usually occurs in young and thin women, and presents often with massive gastric dilatation (MGD). The emergency physician needs to be aware of this condition because it is life threatening, and early recognition will reduce the extent of surgery required.

CASE REPORT

A 24-year-old woman presented to our emergency department with a 5-hour history of severe pain and distension of the abdomen after a reported bout of binge eating. Her blood pressure was 140/90 mmHg; pulse, 110 beats/min; respiratory rate, 24 breaths/min; and temperature, 36.0°C. She was 152.7 cm tall, weighed 50.4 kg, and her BMI was 21.6 kg/m². The abdomen was distended and tympanic, with diffuse pain and tenderness. Bowel sounds were hypoactive. Blood pH was 7.211; leukocyte count, 7,200/μL; platelet count, 241,000/μL; and hemoglobin concentration, 8.0 g/dL. Other laboratory values, including electrolytes, were almost within the normal range. Plain abdominal radiography revealed a massively distended stomach with no free air (Fig. 1). Abdominal CT revealed a marked dilatation of the stomach and proximal duodenum, with abrupt luminal narrowing at the third portion of the duodenum. Diffuse air densities were noted in both portal veins, perigastric vessels, and gastric wall. The MGD occupied the abdominal cavity entirely from the diaphragm to the pelvis, causing collapse of the abdominal aorta and perigastric small vessels (Fig. 2).

The stomach was lavaged using a large-bore (36 Fr) nasogastric tube with a net return of 5,000 mL of undigested food that smelt of chocolate. After lavage, her abdominal girth was decreased. However, she still complained diffuse pain and mild rebound tenderness. The patient underwent an emergency laparotomy to reduce the risk of life-threatening bowel ischemia or infarction caused by the severe distension, portal venous gas, and compressed vessels. During laparotomy, small amounts of serosanguinous fluid collection were noted in the ab-
dominal cavity, but no micro-perforation was evident. No abnormality was seen in the small and large intestines, and no volvulus or adhesions were observed. Normal pulsations were seen in the SMA. No necrosis was found in the stomach area, so excision was not required. Conservative management with fluid and electrolyte replacement via a nasogastric tube relieved abdominal distension and pain. Psychiatric assessment suggested a possible diagnosis of bulimia. Hypotonic duodenography performed 14 days after admission revealed a blockage in the third portion of the duodenum.

SMA syndrome was diagnosed on the basis of CT findings (Fig. 3). Upper endoscopy (EGD) revealed extensive deep ulcerations and dirty exudates in the stomach and duodenum (Fig. 4). After surgery, the patient was well initially, although she could not tolerate
Fig. 3. Abdominal CT shows compression of the third portion of duodenum between the aorta and superior mesenteric artery (A) Aorto-mesenteric distance is 4.21 mm. (B) Aorto-mesenteric angle 16.3°. (C) In hypotonic duodenography, dilatation of the second portion of the duodenum and blocked barium passage with a cut-off sign (arrow).

Fig. 4. Esophagogastroduodenoscopy findings. Extensive deep ulcerations and ischemic changes with dirty exudates were noted.
oral feeding because of recurrent nausea and vomiting. EGD performed on day 43 of admission revealed diffuse ulcerative friable mucosal changes and loss of gastric rugae with deformity of the antrum and bulb. This was deemed to be a healing stage from previously active ischemic and ulcerative mucosa (Fig. 5). Her general medical condition improved with time, and she was discharged symptom free on day 64.

She was re-admitted to hospital 3 months later with vomiting and weight loss; at 36.5 kg, her weight was 14 kg less than that at discharge. Subsequent management of the patient focused on the delivery of adequate nutrition and psychiatric assessment.

**DISCUSSION**

Acute gastric dilatation in patients with eating disorders occurs in patients with binge eating and drinking habits associated with anorexia nervosa and bulimia, or during re-feeding when BMI is low.2

In the present case, body weight was normal and the patient was not diagnosed with an eating disorder. However, when she was readmitted, she had lost 14 kg of body weight, which suggests that bulimia made her body weight fluctuate sharply; therefore, she was at risk of SMA syndrome even though her BMI was normal.

First-line treatment for SMA syndrome with MGD consists of prompt decompression of the stomach and vigorous fluid and electrolyte replacement therapy. If conservative treatment fails or gastric infarction or perforation is suspected, immediate surgical intervention is mandatory.3,4 In fact, for most cases of MGD reported in the literature, the treatment consisted of exploratory laparotomy, and then, surgical decompression or partial or total resection of the stomach in case.5-9 Surgical management includes duodenojejunostomy, gastrojejunostomy, and Strong’s operation, which consists of lysis of the ligament of Treitz, and derotation of the small bowel and right colon. The most frequent used procedure is duodenojejunostomy, which is successful in approximately 90% of cases.7 During conservative therapy, close monitoring is essential to detect signs of surgical abdomen as soon as possible. In this case, we decided to emergency laparotomy to reduce the risk of life-threatening bowel ische-

**Fig. 5.** Esophagagogastroduodenoscopy findings. Diffuse healed ulcerations and deformed ulcer scars with loss of gastric rugae were seen.
mia or infarction reflected by the acute severe distension, portal venous gas, and compressed vessels. And also, it was necessary to rule out volvulus or adhesion. No necrosis was found in the stomach area, so excision was not required. However, if we had the decompression surgery during emergency laparotomy, it might be helpful in improving of clinical symptom and preventing the recurrence during the treatment period of eating disorder.

Severe ischemia with extensive mucosal necrosis in MGD is not always mandatory indication for surgery. Prompt adequate conservative therapy may avoid an unnecessary gastrectomy. In the present case, we performed prompt decompression for the distended stomach and an urgent laparotomy. Fortunately, the patient recovered well using conservative management, and total gastrectomy was not required.

The stomach is very resistant to ischemia because of the rich blood supply and sufficient intramural anastomoses. Several experimental studies have revealed that gastric ischemia and necrosis occur only after both arterial and venous circulations are interrupted. Intra-gastric pressure exceeding 20 cmH2O, which exceeds venous pressure, results in mucosal necrosis. In cases of acute MGD, intra-gastric pressure usually exceeds 30 cmH2O and produces a rapid decrease in intramural blood flow, followed by necrosis and perforation.

In this case, chronic bile regurgitation and bile stasis by compression of distal duodenum may induce gastric mucosal injury. Binge eating-induced MGD can aggravate mechanical obstruction and increase the intra-gastric pressure, leading to gastric ischemia, pneumatosis of the gastric wall, and portal venous gas.

In summary, we experienced a case of SMA syndrome with acute complications such as severe acute MGD, deep extensive ulcerations of whole stomach and proximal duodenum, caused by bulimia nervosa in a 23-year-old young woman. For its great rarity, we report this case with a review of literature.

REFERENCES