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Brief Report

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Gastric Pneumatosis and Its Gastrofibroscopic Findings in Life-Threatening Superior Mesenteric Artery Syndrome Complicated by Anorexia Nervosa in a Child

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ABSTRACT

A 14-year-old girl was admitted to the emergency department for excessive bile-containing vomiting and severe abdominal pain. She had been healthy until she intentionally lost 25 kg over a 6-month period. Thick, bloody bile-mixed food particles were drained from the stomach through a nasogastric tube. Abdominal computed tomography revealed huge stomach dilatation with extensive gastric pneumatosis, possible near rupture, acute pancreatitis, and a very narrow third of the duodenum, indicating superior mesenteric syndrome. Gastrofibroscopy revealed multiple hemorrhagic ulcers and numerous bead-like cystic lesions in the stomach. Laboratory examination results were notable for severe deficiencies in critical nutrients, including iron, zinc, proteins, and prealbumin, as well as undernutrition-associated endocrine complications such as hypothyroidism and hypogonadotropic hypogonadism. Excessive vomiting ceased after the endoscopic removal of stagnant gastric contents. Gastric pneumatosis improved after 3 days of supportive care.

Keywords: Gastric dilatation; Superior mesenteric artery syndrome; Anorexia nervosa; Malnutrition; Child

INTRODUCTION

Gastric pneumatosis is a rare condition that occurs in the presence of air in the gastric wall. There are few reports on the endoscopic findings of gastric pneumatosis, particularly in children. To the best of our knowledge, this is the first report describing the endoscopic findings of pediatric gastric pneumatosis in Korea.

In patients with anorexia nervosa, weight loss and consequent malnutrition can cause medial migration of the superior mesenteric artery (SMA) and subsequent narrowing of the normal angle between the SMA and aorta due to the loss of the fatty tissue pad surrounding the SMA. This narrowing results in external mechanical compression of the duodenum as it traverses the space between the SMA and the aorta, leading to intestinal obstruction and dilatation [1].

Conflict of Interest
The authors have no financial conflicts of interest.

Furthermore, the resulting stomach distension causes hypoperfusion and ischemia, with gas penetrating the gastric wall [2,3].

Here, we report the case of a patient who presented with gastric pneumatosis due to SMA syndrome caused by intentional weight loss, which led to undernutrition-associated complications. The patient's condition improved with conservative management.

CASE REPORT

A 14-year-old girl was admitted to our emergency department with excessive bile-containing vomiting and severe abdominal pain. She intentionally lost 25 kg of body weight over a 6-month period. She was 158.3 cm tall (44th percentile), weighed 27.8 kg (<1st percentile), and had a body mass index of 11.09 kg/m^2 (<1st percentile). She had no medical or family history of gastrointestinal disease. Physical examination revealed a distended abdomen, diffuse abdominal tenderness without rebound tenderness, and decreased bowel sounds. Abdominal radiography revealed a markedly distended stomach and duodenum (**Fig. 1**). Air in the stomach wall outlined the fundus and the greater curvature. Abdominal computed tomography (CT) revealed decreased space between the SMA and aorta, leading to compression and near obstruction of the third portion of the duodenum. The aortomesenteric angle was 9.09°, below the normal 28–65° range. An aortomesenteric angle of <22° is suggestive of SMA syndrome [4]. Moreover, significant distension of the stomach and duodenum was observed. Gastric intramural gas outlined almost the entire stomach wall with a risk of rupture. Acute necrotic collection around the tail of the pancreas was also observed, with lipase and amylase levels of 320 and 163 U/L, respectively (**Fig. 1**).

A nasogastric tube was immediately inserted to decompress the stomach. Thick, bloody bile-mixed food particles were drained from the stomach through a nasogastric tube. Gastrofibroscopy was performed for gastric decompression via the removal of a significant volume of gastric contents and to determine the bleeding point and mucosal lesions. Examination revealed multiple mucosal tears in the distal esophagus that developed after excessive vomiting due to SMA syndrome. Thick bile juice mixed with food and blood was observed in the stomach. Blood originated from esophageal tears and necrotic gastric mucosal lesions. As the stagnant intragastric material was removed, severe necrotic mucosal lesions and multiple bleeding ulcers with saccular cystic swelling were observed (**Fig. 2**).

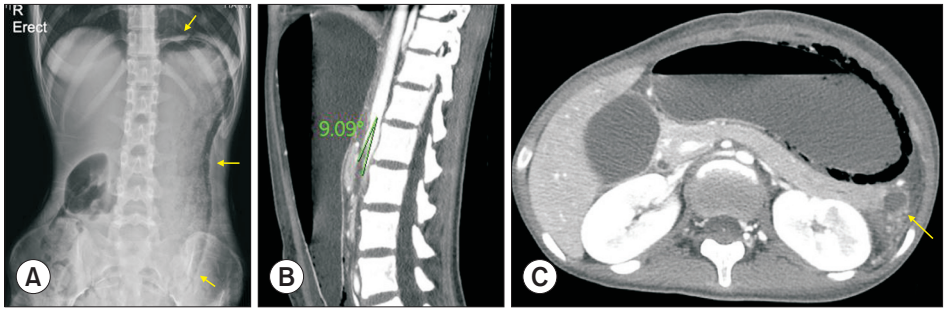

Fig. 1. The stomach and duodenum are markedly distended. Air in the stomach wall outlines (arrow) the fundus and the greater curvature (A). A significant narrowing of the aortomesenteric angle down to 9.09°, suggesting superior mesenteric artery syndrome (B). The abdominal axial scan shows massive distension of the stomach and the duodenum. Gastric intramural gas outlines almost the entire stomach wall. An acute necrotic collection around the tail of the pancreas (arrow) is also observed (C).

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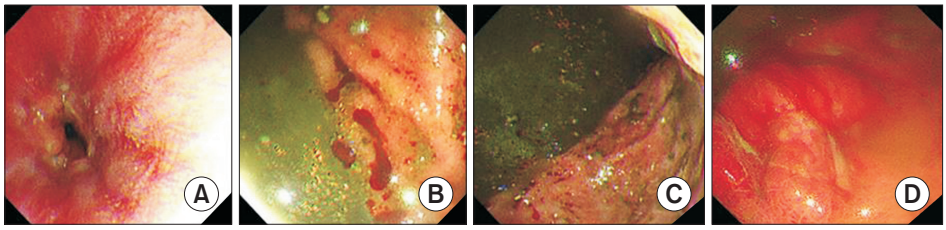

Fig. 2. Multiple mucosal tears in the distal esophagus develop after excessive vomiting due to superior mesenteric artery syndrome (A). Thick bile juice mixed with food and blood in the stomach. The blood originates from the esophageal tears and the necrotic gastric mucosal lesions (B). As the intragastric stagnant material is removed, severely necrotic mucosal lesions (C) and multiple bleeding ulcers with saccular cystic swelling are revealed (D).

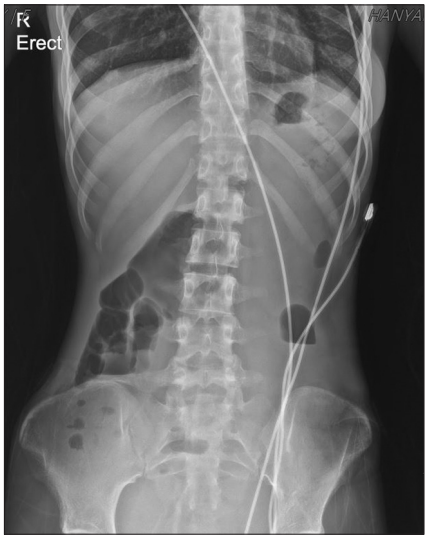

Fig. 3. The follow-up simple abdomen shows that severe gastric dilatation with pneumatosis improved.

Vomiting resolved immediately after the endoscopic removal of 1.6 L of stagnant gastric content. Intravenous proton pump inhibitors, piperacillin/tazobactam, metronidazole, and total parenteral nutrition were administered. Supportive care improved gastric pneumatosis within 5 days (**Fig. 3**), and a soft diet was initiated after 11 days of nil per os (NPO) following an upper gastrointestinal series, which showed delayed contrast passage through the third portion of the duodenum. In addition to NPO, a somatostatin analog was administered to treat acute pancreatitis. Follow-up abdominal CT revealed a decrease in necrotic collection around the tail of the pancreas, and serum lipase and amylase levels improved within 10 days.

Laboratory results revealed severe deficiencies in critical nutrients, including iron, zinc, proteins, and prealbumin, likely due to chronic malnutrition resulting from anorexia nervosa. The patient's white blood cell count was 20,000/mm^3 with 92.0% neutrophils, and her serum C-reactive protein level was 1.80 mg/dL. Her hemoglobin, iron, ferritin, total iron-binding capacity (TIBC), and transferrin saturation levels were 9.3 g/dL, 10 μg/dL, 275 ng/mL, 157 μg/dL, and 6.4%, respectively. After 11 days of intravenous administration of ferric hydroxide, hemoglobin, iron, ferritin, and transferrin saturation levels increased to 15.8 g/dL, 107 μg/dL, 514 ng/mL, and 64%, respectively, while TIBC decreased to 157 μg/dL. Iron deficiency anemia was successfully corrected. The prealbumin and zinc levels were 9.41 mg/dL and 65.8 μg/dL, respectively. These levels normalized following the administration of intravenous zinc and amino acids.

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The patient exhibited hirsutism and amenorrhea for 2 months, and laboratory testing revealed low levels of follicle-stimulating hormone (FSH, 0.4 mIU/mL), progesterone (<0.1 ng/mL), estradiol (<5.0 pg/mL), and beta-human chorionic gonadotropin (<0.2 ng/mL). Thyroid function also decreased; T3, T4, free T4, and thyroid stimulating hormone (TSH) levels were 26 ng/dL, 4.12 μg/dL, 0.72 ng/dL, and 0.50 μIU/mL, respectively. Further evaluation was conducted using a thyrotropin-releasing hormone stimulation test, demonstrating that the basal and peak TSH levels were 0.16 μIU/mL and 6.25 μIU/mL, respectively. These findings suggested that chronic malnutrition results in hypothalamic dysfunction and hypogonadotropic hypogonadism. Treatment with liothyronine and levothyroxine improved the hypothyroidism, and the patient received adequate nutrient supplementation.

Chronic malnutrition also resulted in hypotension (systolic/diastolic blood pressure of 80 mmHg/40 mmHg) and bradycardia (average heart rate, 37 beats/min), leading to admission to the telemetry unit for 24-hours electrocardiogram monitoring, where the patient was managed with intravenous atropine and hydration.

The patient was advised to remain hospitalized until adequate weight gain and recovery from nutritional imbalances were achieved. However, despite gaining only 2.1 kg body weight, the patient was discharged 16 days after admission due to financial constraints. Subsequently, the patient was readmitted to our hospital three more times with similar symptoms related to intentional weight loss and binge eating.

Ethics statement

The authors obtained approval from the Institutional Review Board of Hanyang University Hospital (File No. 2023-04-013). Informed consent was waived by the board.

DISCUSSION

Gastric pneumatosis has various etiologies, including mechanical force, pulmonary disease, bacterial infection, and ischemic conditions [5,6]. In children, it can be associated with pyloric stenosis [7], gastric malrotations [8], and annular pancreas [9]. In the present patient with anorexia nervosa, intentional weight loss caused the loss of fatty tissues surrounding the SMA and subsequent narrowing of the angle between the SMA and the aorta, resulting in duodenal obstruction. This obstruction led to gastric dilatation, causing hypoperfusion of the stomach and subsequent gastric pneumatosis. Currently, there is no standard treatment for gastric pneumatosis. Most reported cases have been managed by conservative treatment, such as with empirical antimicrobial management, including agents effective against gram-negative and anaerobic bacteria, hemodynamic stabilization with intravenous fluids, and nutrition [2,5].

Endoscopic findings in patients with gastric pneumatosis include diffusely ulcerated, edematous hemorrhagic gastric mucosa, mucosal hemorrhage [10], absence of mucosal folds, mucosal edema, necrotic mucosa [11], gastric mucosal congestion [12], severe circumferential erythema, erosions, exudates, friability of irregularly thickened proximal gastric folds [13], violaceous and pale mucosa in the stomach, gastric infarction [14], strictures [15], ulcer-like lesions covered by pus, multiple small mucosal defects [16], and diffuse erythema, edema, erosion, and sloughing of the gastric mucosa [17]. In this present

case, the endoscopic findings helped diagnose gastric pneumatosis. Furthermore, the endoscopic intervention involved the extraction of 1.6 L of sluggish gastric content and the application of epinephrine to manage the bleeding lesions. These findings emphasize the importance of early endoscopic intervention. The patient's severe essential nutrient deficiencies, anemia, endocrine complications, including euthyroid sick syndrome and hypogonadotropic hypogonadism, and unstable vital signs highlight the critical importance of timely intervention and sustained management of individuals with eating disorders.

This case serves as a crucial illustration of life-threatening complications associated with anorexia nervosa, offering valuable insights to pediatricians regarding the potential manifestations of the various phenomena outlined in this case report. Surgical intervention should be considered if conservative treatment fails to address the patient's condition resulting from the underlying SMA syndrome. However, it is essential to acknowledge that the effective implementation of nonsurgical treatment for multiorgan dysfunction, leading to an overall improvement in the patient, poses a significant challenge for pediatricians.

REFERENCES

1. Watters A, Gibson D, Dee E, Mascolo M, Mehler PS. Superior mesenteric artery syndrome in severe anorexia nervosa: A case series. Clin Case Rep 2019;8:185-9.
PUBMED | CROSSREF
2. López-Medina G, Castillo D, Heredia-Salazar AC, Hernández-Salcedo DR. Gastric emphysema a spectrum of pneumatosis intestinalis: a case report and literature review. Case Rep Gastrointest Med 2014;2014:891360.
PUBMED | CROSSREF
3. Merrett ND, Wilson RB, Cosman P, Biankin AV. Superior mesenteric artery syndrome: diagnosis and treatment strategies. J Gastrointest Surg 2009;13:287-92.
PUBMED | CROSSREF
4. Agrawal GA, Johnson PT, Fishman EK. Multidetector row CT of superior mesenteric artery syndrome. J Clin Gastroenterol 2007;41:62-5.
PUBMED | CROSSREF
5. Van Mook WN, Van der Geest S, Goessens ML, Schoon EJ, Ramsay G. Gas within the wall of the stomach due to emphysematous gastritis: case report and review. Eur J Gastroenterol Hepatol 2002;14:1155-60.
PUBMED | CROSSREF
6. Pineda B, Diehl DL, Babameto GP, Smith RE. Massive hepatic portal venous gas and gastric pneumatosis secondary to gastric ischemia. Gastrointest Endosc 2013;78:540-1.
PUBMED | CROSSREF
7. Lester PD, Budge AF, Barnes JC, Kirks DR. Gastric emphysema in infants with hypertrophic pyloric stenosis. Am J Roentgenol 1978;131:421-3.
PUBMED | CROSSREF
8. Kawano S, Tanaka H, Daimon Y, Niizuma T, Terada K, Kataoka N, et al. Gastric pneumatosis associated with duodenal stenosis and malrotation. Pediatr Radiol 2001;31:656-8.
PUBMED | CROSSREF
9. Franquet T, Gonzalez A. Gastric and duodenal pneumatosis in a child with annular pancreas. Pediatr Radiol 1987;17:262.
PUBMED | CROSSREF
10. Lee YM, Song HJ, Na SY, Boo SJ, Kim HU, Kim SH. Gastric emphysema related with superior mesenteric artery syndrome. Ewha Med J 2014;37:141-5.
CROSSREF
11. Carlson AP, Chan JW, Ketai LH, Demarest GB. Emphysematous gastritis in a severely burned patient: case report and literature review. J Trauma 2007;62:765-7.
PUBMED | CROSSREF
12. Yu HH, Tsang S, Cheung TT, Lo CM. Surviving emphysematous gastritis after hepatectomy. Case Rep Hepatol 2013;2013:106383.
PUBMED | CROSSREF

13. Binmoeller KF, Benner KG. Emphysematous gastritis secondary to gastric infarction. Am J Gastroenterol 1992;87:526-9.
PUBMED

14. Qureshi T, Sparkman J, Husain NS. Emphysematous gastritis in a patient with abdominal pain after dialysis. Clin Gastroenterol Hepatol 2019;17:A18.
PUBMED | CROSSREF

15. Gaspar BL. The significance of sarcina in routine surgical pathology practice. APMIS 2016;124:436-43.
PUBMED | CROSSREF

16. Verhelst X, Cabooter M, De Wilde V, Laukens P, Orlent H. A case of emphysematous gastritis. Endoscopy 2012;44 (Suppl 2):E159.
PUBMED | CROSSREF

17. Soon MS, Yen HH, Soon A, Lin OS. Endoscopic ultrasonographic appearance of gastric emphysema. World J Gastroenterol 2005;11:1719-21.
PUBMED | CROSSREF