Case Report

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Gastric antral web presenting with esophageal stricture and severe malnutrition

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ABSTRACT

Gastric antral web is a rare cause of gastric outlet obstruction in children. Common complaints include intermittent non-bilious vomiting, bloating, pain, loss of appetite, and weight loss. Herein, we report a case of gastric antral web presenting with esophageal stricture and malnutrition, which was followed for many years with a misdiagnosis. An 8year-old African boy was referred to our pediatric gastroenterology clinic due to recurrent vomiting and severe malnutrition. According to his medical history, the patient began experiencing non-bilious vomiting at 40 days of age and a diagnosis of gastroesophageal reflux was made. He was hospitalized multiple times and received nasogastric tube feedings in the following 4 years. He was re-evaluated for growth retardation and malnutrition at 4 years of age. He had been diagnosed with gluten enteropathy and was fed a gluten-free diet. Recently, he developed difficulty swallowing solid food and was only able to consume liquid meals. Esophagography revealed segmental stenosis in the distal portion of the esophagus. After stricture excision with thoracotomy, upper gastrointestinal series with a radiopaque substance indicated normal esophageal passage; however, prolonged gastric emptying time was also noted. Upon examination during laparotomy, a congenital web was detected in the antral region and was excised. Esophageal and gastric pathologies were consistent with stricture caused by reflux esophagitis and gastric antral web. Today, gastric antral web is mostly diagnosed and treated in infancy. Near total obstruction is relatively easy to diagnose; however, many patients with partial obstruction are followed-up for years with an incorrect diagnosis. Therefore, it should be considered in such cases, persistent vomiting and gastroesophageal reflux may be associated with esophageal stricture.

Keywords: Gastric antral web, Esophageal stricture, Gastroesophageal reflux, Malnutrition, Pediatrics

INTRODUCTION

Gastric antral web (GAW) is a rare cause of gastric outlet obstruction in children, with an incidence of 1-3 in 100,000 live births.^{1,2} GAW is characterized by a thin, circumferential mucous membrane with a central opening located 1-2 cm proximal to the pylorus that often does not

contain muscle tissue.^{3,4} The exact etiology is not fully understood. Although the localized endodermal proliferation theory is the most accepted, some authors consider GAW an incomplete form of membranous atresia, and others believe that it results from incomplete recanalization during embryonic development.^{5,6} Clinical presentations vary depending on the degree of anatomical

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obstruction. The most common complaint is non-bilious vomiting, which often begins in infancy. Other symptoms include early satiety, bloating in the epigastrium, and abdominal pain, especially when eating. In almost complete occlusion, the complaints begin early, the course of the disease is severe, and the diagnosis is often made in the infantile period. We were unable to find a case of GAW presenting with esophageal stricture due to gastroesophageal reflux (GER) in the English literature. Herein, we report a case of GAW presenting with esophageal stricture and malnutrition, which was followed for many years with a misdiagnosis.

CASE REPORT

An 8-year-old African boy was referred to our pediatric gastroenterology clinic due to recurrent vomiting and severe malnutrition. According to his medical history, the patient began experiencing non-bilious vomiting at 40 days old. Congenital hypertrophic pyloric stenosis was initially considered; however, no pathology was detected abdominal ultrasound (USG) with gastrointestinal (UGI) series. Therefore, the case was evaluated as GER, and treatment was arranged. The patient was hospitalized multiple times and received nasogastric tube feedings in the following 4 years. He was re-evaluated because of growth retardation and malnutrition at 4 years of age. He had been diagnosed with gluten enteropathy and was fed a gluten-free diet; however, no test results confirmed gluten enteropathy. Recently, he developed difficulty swallowing solid food and was only able to consume liquid meals. Upon arrival, the patient's weight was 6.5 kg (<3rd percentile), and his height was 86 cm (<3rd percentile) (Figure 1a). Hematological examinations revealed no pathology except for slightly elevated liver function test results (white blood cell count: 12×10⁹/l, hemoglobulin: 12.5 g/dl, hematocrit: 38.2%, aspartate aminotransferase: 69 IU/l, alanine aminotransferase: 99 IU/l). Electrolytes were normal. Abdominal USG revealed a mildly dilated stomach and no other pathology. Esophagography revealed segmental stenosis in the distal portion of the esophagus and enlargement of the proximal esophagus (Figure 1b). The stomach and duodenum could not be evaluated. Fiberoptic endoscopy revealed minimal passage through the esophageal stenosis (Figure 1c). We suspected that the stenosis might have been caused by a congenital tracheobronchial remnant, and we believed that the stenosis had increased over time.

Initially, we inserted a central venous catheter and began administering total parenteral nutrition. Because balloon dilatation was unlikely to be effective, the patient underwent thoracotomy for excision of the stricture and end-to-end esophageal anastomosis (Figure 1d). On the fifth postoperative day, the patient began eating without any signs of anastomotic leakage; however, on the following day, the patient experienced non-bilious vomiting. Gastroscopy was not performed as the patient's esophageal anastomosis was relatively new. UGI series

indicated normal esophageal passage; however, prolonged gastric emptying time was noted, with the radiopaque substance still present in the stomach after 6 hours (Figure 1e). Upon examination during laparotomy, the gastric contents barely passed through the pylorus despite manual pressure. An incision was made starting from the pylorus, parallel to the long axis of the stomach, and a congenital web with a 4-5 mm aperture was detected in the antral region (Figure 1f).

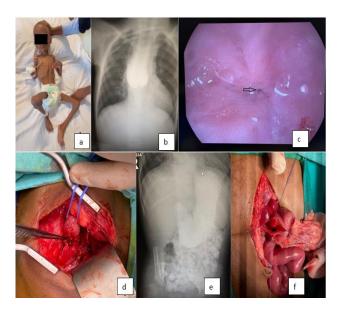


Figure 1: a) Severe malnutrition, b) Stricture in distal esophagus, c) View of the esophageal stricture with fiberoptic endoscopy (arrow), d) External view of esophageal stricture during thoracotomy, e) Upper gastrointestinal series showing slow gastric emptying and knife-like filling defect, f) View of gastric antral web during open surgery.

The antral web was excised, and Heineke-Mikulicz pyloroplasty was performed. In addition, Nissen fundoplication was performed in the same session due to GER. The patient's postoperative period was uneventful, and liquid food was started on the fifth day. The patient resumed normal eating within a few days, and he did not experience any more vomiting. Esophageal pathology was consistent with stricture caused by reflux esophagitis, and no evidence of gluten enteropathy was detected (IgA anti-endomysial antibodies: negative; IgG antiantibodies: negative). endomysial Pathological examination of the sections revealed that the surface was lined with columnar epithelium, and there were hypertrophic muscle bundles in the muscularis propria. The findings were consistent with GAW. The patient was well 4 months after the surgery, without any complaints.

DISCUSSION

Symptoms and signs associated with a GAW are directly related to the degree of obstruction; when the aperture is less than 1 cm, symptoms tend to be severe.^{4,8} Common complaints include intermittent non-bilious vomiting,

bloating, pain, loss of appetite, and weight loss. In patients with early onset of symptoms, failure to thrive and more rarely upper gastrointestinal system bleeding may also occur.^{7,8} Patients with a wide web aperture are often followed for a long time due to misdiagnosis. Growth retardation and malnutrition are the expected results in these patients, as in the presented case. However, this patient had an aperture of less than 1 cm, and late diagnosis may have been suggestive. Perhaps this can be explained by the gradual reduction of web aperture over time. UGI series and USG can diagnose up to 90% of cases of GAW.⁸

An echogenic diaphragm-like structure, gastric dilatation, delay in gastric emptying, and a normal pylorus are the four USG diagnostic criteria for GAW.⁴ Late gastric emptying, double-bulb appearance, and "knife-like" filling defect in the prepyloric region direct the diagnosis with UGI series.^{3,9} We did not detect any findings other than mild gastric dilatation with USG. Unfortunately, although we did not detect any pathology in the preoperative UGI series, we noticed a knife-like septa image in the prepyloric region during postoperative reevaluation. Although it is difficult to visualize this knife-like septum image when the stomach is not filled with contrast medium, it is important to consider a rare pathology in such cases, such as GAW.

Gastroscopy can confirm the diagnosis and rule out other causes, and endoscopic treatment can be performed in appropriate cases.^{3,7} Today, needle-knife incision, balloon dilatations, and triamcinolone injections are common endoscopic treatment modalities for GAW in adults and children.^{3,7,9-11} However, endoscopic treatment methods often require more than one procedure and extensive experience on the part of the technician.^{10,11} When endoscopy is not feasible or in cases of recurrence, web excision and pyloroplasty can be performed via open or laparoscopic surgery.² In this case, we preferred open surgery because of the recent esophageal anastomosis and lack of experience with endoscopic surgery.

One of the undesirable consequences of GER is esophageal stricture. 12 The duration and severity of GER are directly related to the development of esophageal stricture. The development of GER in patients with prolonged vomiting due to GAW is expected and should not be surprising finding. However, we did not find any other published case of esophageal stricture due to GAW in the English literature. We believe that the esophageal stricture developed in the presented case due to bouts of vomiting and GER that persisted for 8 years. Furthermore, the delayed diagnosis has a significant effect on the development of esophageal stricture.

CONCLUSION

Today, GAW is mostly diagnosed and treated in infancy. Near total obstruction is relatively easy to diagnose; however, patients with partial obstruction may be followed-up for years without a proper diagnosis. Therefore, it should be considered in such cases, persistent vomiting and gastroesophageal reflux may be associated with esophageal stricture.

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