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Pneumatic dilation in treatment of late-onset primary gastric outlet obstruction in childhood

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Abstract Late-onset primary gastric outlet obstruction in childhood is a rare condition. Only 8 cases of such were reported. Diagnosis should be considered while ruling out mechanical and structural lesions. Up to now, Heineke-Mikulicz pyloroplasty has been the standard treatment. However, we succeeded in treating this condition by using pneumatic dilation. There is no sign of recurrence for 1 year. We propose the etiology, diagnosis, and the relationship between late-onset primary gastric outlet obstruction and esophageal achalasia in childhood.

In 1997, Sharma [1] first reported a new disease that was termed "acquired gastric outlet obstruction." Up to now, only 8 cases are found in the English literature. All of them were cured through the Heineke-Mikulicz pyloroplasty. We add a new case and use a different therapeutic modality. The possible etiology of this disease may be similar to esophageal achalasia in childhood.

1. Case report

A 4-year-old boy had a history of nonbilious projectile vomiting and lost body weight for 1 month. His parents observed abdominal distention after meals, which improved after vomiting. Visible peristalsis in the upper abdomen during vomiting was also observed. All laboratory data were normal. A plain film showed distention of stomach with retained food residue. Upper gastrointestinal study and computed tomography revealed a typical picture of gastric outlet obstruction with a narrow pyloric channel (Figs. 1 and 2A, B). B-mode sonogram demonstrated gastric mucosa protruding into the lumen of the antrum without pyloric muscular hypertrophy in the longitudinal view. Gastroscopy was performed and showed a narrow pylorus without abnormality of gastric mucosa. Histopathological examination of endoscopic biopsy at the pylorus showed a normal cellular pattern without any inflammatory or neoplastic cells. *Helicobacter pylori* study was negative. Those findings were compatible with late-onset primary gastric outlet obstruction in childhood. We decided to use pneumatic dilation for the stenosed pylorus. Upper endoscope was performed under intravenous sedation with thiamylal sodium (5 mg/kg per dose) by a video pediatric endoscope (CV260; Olympus, Tokyo, Japan). The stenosed pylorus was identified and probed under endoscopic visualization with a balloon inflating device (outer diameter, 6-8 mm) (C.R.E. Esopgageal Balloon Dilator Catheter, Microvascular; Boston Scientific Corporation), then inflated at serial

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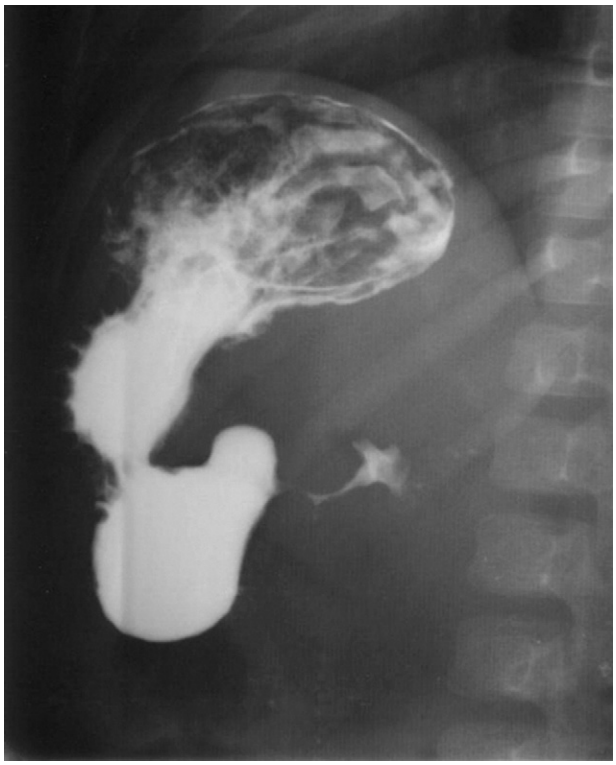

Fig. 1 Upper gastrointestinal series showing a narrow pyloric channel.

pressure of 1.5 and 2 atm for 1 and 2 minutes, respectively. The patient then received partial parenteral nutrition and liquid diet because of inadequate patency of the pylorus after first dilation. We did another pneumatic dilation after 1 week. The inflating device was changed to a balloon with an outer diameter of 8 to 10 mm and pressure of 1.5 and 2 atm for 1 minute each. No fever or abdominal pain occurred after pneumatic dilation. Since then, he has not vomited and is under regular follow-up for 1 year.

2. Discussion

In 1997, Sharma et al [1] first described 5 unusual cases with acquired gastric outlet obstruction during infancy and childhood. In 2004 and 2005, 3 new cases were reported by Abuhandan et al [2] and Nazir and Arshad [3]. Patients of late-onset primary gastric outlet obstruction have a good well-being, then develop recurrent projectile, nonbilious vomiting after meals with varied duration from 1 month to 8 years (mean, 2.3 years). The patient age ranged from 3 months to 17 years (mean, 9.9 years). There were 3 who were younger than 1 year, 4 who were between 2 and 6 years, and 1 who was 16 years old. They were all male. The stomach was dilated secondary to the stenotic pyloric ring. Other than stenosis, the pylorus was grossly normal without muscular hypertrophy or ganglion cell deficiency. The obstruction was partial and long-standing. All reported cases were cured through the Heineke-Mikulicz pyloroplasty.

Diagnosis of primary gastric outlet obstruction is considered only after mechanical and structural lesions have been ruled out. Upper gastrointestinal series showed a dilated stomach with delayed gastric emptying time and a narrow pylorus. Sudden narrowing of the pylorus is a typical gastroscopic finding. The sonogram reveals that gastric mucosa protrudes into the antrum (antral nipple sign) without pyloric muscular hypertrophy (Fig. 3A, B). This is the first sonographic description, and we are unsure if it is a specific finding for late-onset primary gastric outlet obstruction in childhood.

The etiology of late-onset primary gastric outlet obstruction is still unknown. Sharma et al [1] suggested that the function of the pylorus changed permanently because of localized pyloric neuromuscular incoordination; hence, a drainage operation was necessary. We speculated that esophageal achalasia in childhood and late-onset primary gastric outlet obstruction shared common pathophysiological mechanisms but involved different sites. This speculation is based on 4 reasons. First, they are both motility

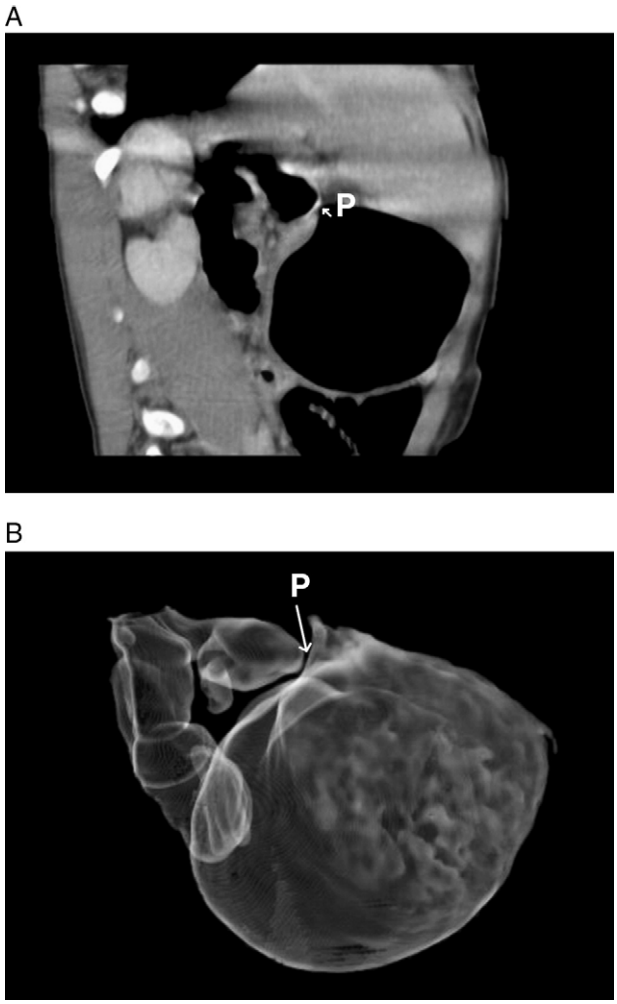

Fig. 2 A, Computed tomography revealed a short and narrow pyloric channel (P). B, Multiplane reformation of computed tomography imagine.