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Prostaglandin-induced foveolar hyperplasia simulating pyloric stenosis in an infant with cyanotic heart disease

M. G. Mercado-Deane[1,2], E. M. Burton[1,2], A. V. Brawley[2], R. Hatley[3]

[1] Department of Radiology, Medical College of Georgia, Augusta, GA 30912, USA
[2] Department of Pediatrics, Medical College of Georgia, Augusta, GA 30912, USA
[3] Department of Surgery, Medical College of Georgia, Augusta, GA 30912, USA

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Abstract. Prostaglandin infusion is used to maintain patency of the ductus arteriosus in infants with cyanotic congenital heart disease. Recently, gastric outlet obstruction as a result of prostaglandin infusion has been described. In our case, an upper gastrointestinal contrast study seemed to depict the typical appearance of pyloric stenosis in an infant who had received an infusion of prostaglandin for a prolonged period. Serial ultrasonograms, however, disclosed progressive elongation of the antropyloric channel *without* wall thickening. This report is the second to illustrate prostaglandin-induced gastric outlet obstruction in a vomiting infant with a gastrointestinal series diagnosis of pyloric stenosis.

Hypertrophic pyloric stenosis, a common cause of nonbilious vomiting in infants, can be imaged by an upper gastrointestinal contrast study or by ultrasonography. This is the second report to recognize the gastrointestinal series appearance simulating pyloric stenosis in a patient treated with prostaglandin.

Case report

A 2.5-kg term female infant was the product of a pregnancy complicated by hypertension and polyhydramnios. On the 1st day of life, echocardiography showed transposition of the great arteries. This was treated by continuous prostaglandin infusion for 23 days. The dosage of prostaglandin was 0.05 μg/kg per minute (total dose 4140 μg). On the 3rd day of life, cardiac catheterization confirmed the diagnosis of transposition of the great arteries; balloon atrial septostomy was then performed. At 25 days of age, a Jatene arterial switch procedure was performed.

Persistent non-bilious vomiting and feeding intolerance began at 32 days of age. No pyloric mass was palpable. An upper gastrointestinal series at 35 days of life showed gastric outlet obstruction, elongation of the pylorus, and double-channel sign (Fig. 1).

Ultrasonography demonstrated that the pyloric length and wall thickness were 19 mm and 2.1 mm, respectively. Prominent antral folds were also noted. Ultrasonography 3 days later disclosed that the pyloric length and wall thickness were 22.7 mm and 2.3 mm, respectively. At 49 days of life, ultrasonography (Fig. 2) showed that the pyloric length had increased to 27.5 mm; the wall thickness was 2.5 mm. Transverse images depicted the double channel. Because of persistent gastric outlet obstruction, a pyloroplasty and transpyloric feeding tube placement were performed. The gastric antrum was narrowed by redundant mucosa; there was no evidence of pyloric muscle thickening. After the transpyloric stent was removed, vomiting recurred. The vomiting subsided spontaneously by the 85th day of life, 2 months after the prostaglandin infusion was discontinued.

Discussion

Although a pyloric mass can be felt by pediatric surgeons in 80–89 % of infants with hypertrophic pyloric stenosis [1, 2], imaging studies are frequently obtained to confirm the clinical impression. Between 1980 and 1984, 61–97 % of patients referred to each of four children's hospitals had an imaging study to confirm the clinical suspicion of

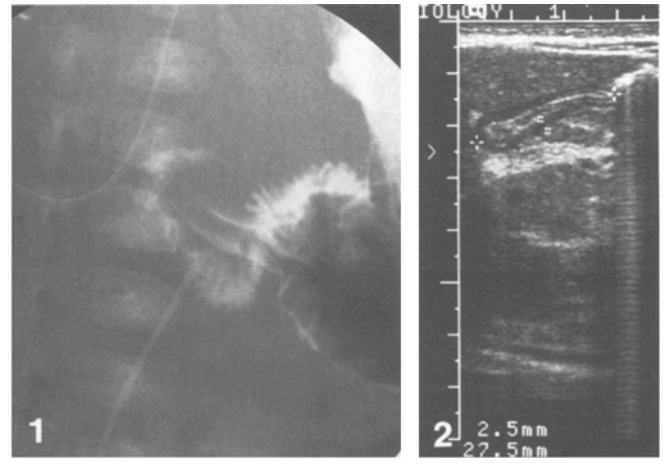

Fig. 1. An upper gastroinestinal series demonstrates elongation of the pylorus and a "double-channel" sign consistent with pyloric stenosis

Fig. 2. Longitudinal ultrasound depicts pyloric length of 27.5 mm. Note the normal wall thickness of 2.5 mm

Correspondence to: M. G. Mercado-Deane

hypertrophic pyloric stenosis [2]. An upper gastrointestinal contrast study is the procedure most commonly performed to depict the pyloric channel, but ultrasonography has been increasingly used [2].

The sonographic criteria for the diagnosis of hypertrophic pyloric stenosis include pyloric channel length of greater than 17–19 mm and pyloric wall thickness of greater than 3–4 mm [3–8]. Tunell and Wilson advocate the pyloric channel length as the more reliable single criterion [6]; others advocate the pyloric wall thickness [4, 5]. Additional recommendations include using multiple abnormal measurements, including the "double track sign", for the sonographic diagnosis of hypertrophic pyloric stenosis [4, 8, 9].

In our patient, the upper gastrointestinal contrast study appeared to show typical findings of pyloric stenosis, including gastric outlet obstruction, elongation of the pyloric channel, and double-channel sign. No pyloric mass was palpable. Ultrasonography showed progressive lengthening of the pyloric channel from 19 to 27 mm but a normal wall thickness of 2.1–2.5 mm. Although the length of the antro-pyloric channel was consistent with that of pyloric stenosis, the pyloric muscle was not hypertrophic. Thus, our patient did not have hypertrophic pyloric stenosis.

Recently, Peled et al. described five neonates with gastric outlet obstruction induced by prostaglandin infusion. Each had radiologic or pathologic evidence of gastric outlet obstruction consistent with antral mucosal hyperplasia. The duration of treatment in patients without gastric outlet obstruction was 1–123 h. Patients with gastric outlet obstruction had received prostaglandin infusion for 272–1109 h. The mean rate of prostaglandin infusion was similar in the two groups [10]. Using adult human volunteers, Tytgat et al. demonstrated antral and foveolar hypertrophy following the oral intake of prostaglandin for 2 months followed by 2 months of placebo. Increased thickness of the antrum was due to expansion of the foveolar layer and enlargement of mucus-secreting cells. The changes reversed completely within 2 months after stopping prostaglandin [11]. Our patient, who had received prostaglandin for 23 days, demonstrated antral mucosal redundancy, elongation of the antropyloric channel, and gastric outlet obstruction, which resolved 2 months after the cessation of therapy. The prostaglandin-induced antral mucosal redundancy in our patient resembles the antral focal foveolar hyperplasia described by McAlister et al., whose two patients had not been on prostaglandin therapy [12, 13].

We agree with Peled et al. that newborns receiving prostaglandin therapy at a dose of 0.05 μg/kg per minute for more than 120 h should be monitored for evidence of antral mucosal thickening and gastric outlet obstruction. The fluoroscopic and sonographic findings in such patients should not be confused with those in cases of hypertrophic pyloric stenosis. The absence of pyloric wall thickening distinguishes between hypertrophic pyloric stenosis and prostaglandin-induced mucosal redundancy.

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