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Recurrent pyloric stenosis: a form of the incomplete pyloromyotomy

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ABSTRACT

Post-pyloromyotomy emesis is common and may be secondary to non-surgical conditions such as pyloric edema, gastroparesis, pylorospasm, or gastroesophageal reflux. Early persistent postoperative emesis is typically attributed to an incomplete pyloromyotomy; whereas delayed postoperative emesis after an asymptomatic period with weight gain has been attributed to recurrent pyloric stenosis. We report a case of an incomplete pyloromyotomy, fulfilling all the criteria of recurrent pyloric stenosis, that suggests recurrent pyloric stenosis is not a separate entity, but a form of the incomplete pyloromyotomy with a failure of the hypertrophied pyloric muscle to regress after an unsuccessful pyloromyotomy.

1. Introduction

Described by Harald Hirschsprung in 1888, infantile hypertrophic pyloric stenosis occurs in 1–3 per 1000 infants in the United States and presents as nonbilious projectile vomiting between 3 and 6 weeks of age [1]. Post-pyloromyotomy emesis is common and may be secondary to non-surgical conditions such as pyloric edema, gastroparesis, pylorospasm, or gastroesophageal reflux [2]. Early persistent postoperative emesis is typically attributed to an incomplete pyloromyotomy; whereas delayed postoperative emesis after an asymptomatic period with weight gain has been attributed to recurrent pyloric stenosis. Cases of recurrent pyloric stenosis have only been reported seven times in the English-language literature [2–9]. To distinguish the incomplete pyloromyotomy from recurrent pyloric stenosis, investigators have defined recurrent cases by the following criteria: “(i) complete resolution of symptoms before recurrence of vomiting; (ii) subsequent weight gain; and (iii) re-stenosis on sonographic and/or operative confirmation” [4]. Fulfilling the criteria for recurrent pyloric stenosis, we describe an operatively proven case of an incomplete pyloromyotomy presenting 5 weeks after a laparoscopic pyloromyotomy with an asymptomatic period of ad libitum feeds and weight gain prior to the recurrence of symptoms. We discuss the natural history of regression of the hypertrophied pylorus after successful pyloromyotomy and conclude that there is no true entity of recurrent pyloric stenosis, only variations of the incomplete pyloromyotomy with a failure of the hypertrophied pyloric muscle to regress.

2. Case report

Our patient was a term infant with a birth weight of 3.29 kg, who at three weeks of age began having intermittent emesis after feedings. The emesis became more frequent, and finally, projectile. At 30 days of age, an ultrasound (Figs. 1 and 2) confirmed the diagnosis of pyloric stenosis (21 mm pyloric length and 3.7 mm thickness) and he was then referred for surgical intervention. He weighed 3.86 kg upon admission and underwent a laparoscopic pyloromyotomy after fluid resuscitation.

At operation, a 5 mm laparoscopic camera was inserted via a supraumbilical skin fold incision after insufflation. Two stab wounds in the left upper and right upper anterior abdominal wall quadrants were used for the pyloric grasper and pylorotome (a myringotomy knife), respectively. The pyloroduodenal and pyloroantral junctions were identified and a pyloromyotomy was performed and completed with a laparoscopic pyloric spreader. The pyloric length was not correlated with the ultrasound findings. A sufficient pyloromyotomy was assumed as both sides of the incised muscle moved independently.

The patient tolerated postoperative feeds and was discharged on postoperative day one. His discharge weight was 4.03 kg. He was seen in follow up two weeks postoperatively with evidence of weight gain and tolerating feeds. By 5 weeks his symptoms recurred with progressive vomiting. A repeat ultrasound demonstrated pyloric dimensions of 22 mm length (Fig. 3) and 3.4 mm thickness (Fig. 4) without air or fluid traversing the channel. A water-soluble contrast study further confirmed gastric outlet obstruction. His weight was 4.49 kg. Following fluid resuscitation, he was taken to the operating room for a redo-pyloromyotomy.

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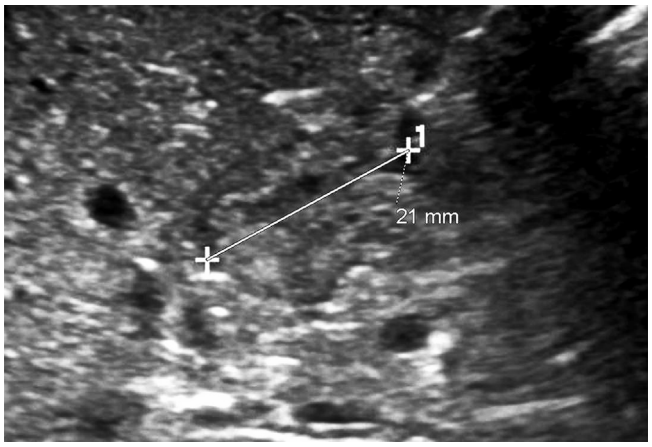

Fig. 1. Pylorus channel on initial presentation.

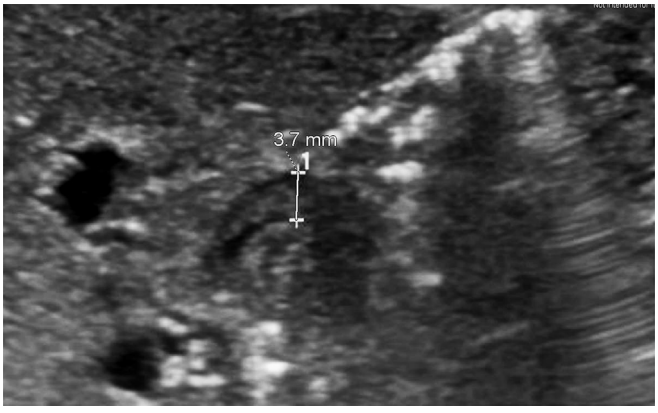

Fig. 2. Pylorus wall thickness on initial presentation.

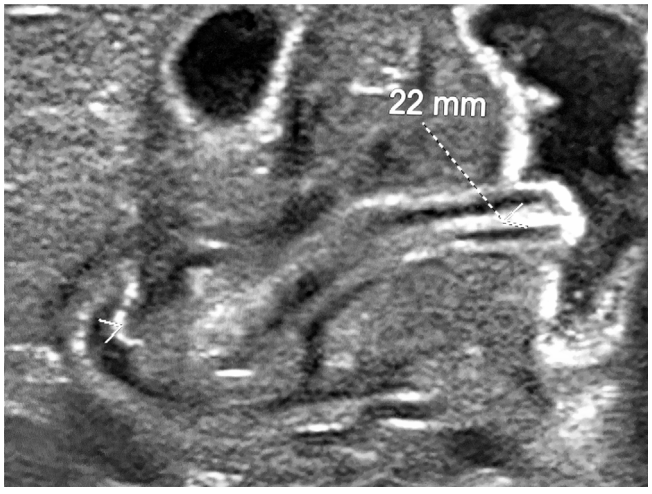

Fig. 3. Pylorus channel on representation.

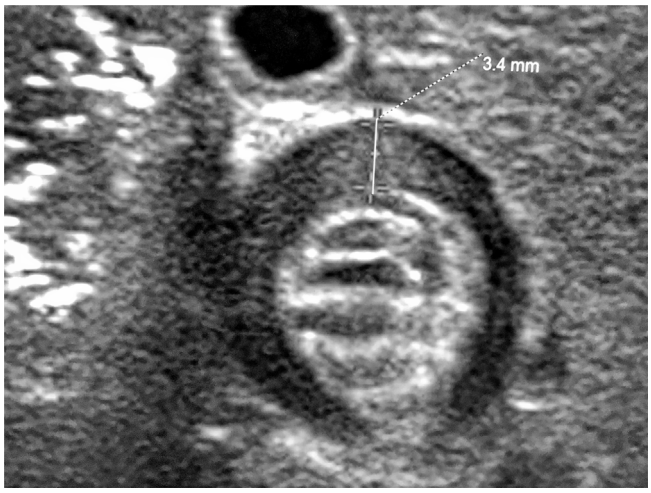

Fig. 4. Pylorus wall thickness on representation.

Fig. 5. Well-healed scar from original pyloromyotomy.

Fig. 6. Newly created pyloromyotomy.

At operation, we observed a well-healed pyloromyotomy that measured 19 mm (Fig. 5) from the pyloric duodenal junction to the antrum of the stomach. On careful evaluation there was residual undivided pyloric muscle appreciated at the pyloroantral junction suggesting that the initial pyloromyotomy was incomplete consistent with the initial ultrasound length of the pylorus of 21 mm. We, therefore, constructed another pyloromyotomy 45° lateral from the initial one, incising from the pyloroduodenal junction to the pyloroantral junction (Fig. 6). Upon concluding the antral portion, we measured this pyloromyotomy at 22 mm in length, which corresponded to the ultrasound length. Both sides of the pyloric muscle moved independently and there was no evidence of perforation. He was discharged home on postoperative day one. No further vomiting was reported on follow up.

3. Discussion

Hypertrophic pyloric stenosis can be treated with an open or a laparoscopic pyloromyotomy. In comparing these approaches, an

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