Pyloric Stenosis in Pediatric Surgery

An Evidence-Based Review

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KEYWORDS

• Pyloric stenosis • Pyloromyotomy • Projectile vomiting • Hypochloremic
• Hypokalemic metabolic alkalosis

KEY POINTS

- Hypertrophic pyloric stenosis (PS) is a benign condition presenting with projectile, non bilious emesis in the newborn infant. The etiology of this condition is still unclear.
- Ultrasound is the preferred diagnostic study if the history, physical and labs aren't sufficient to make the diagnosis.
- Fluid replacement and electrolyte correction will correct the hypochloremic, hypokalemic metabolic alkalosis, and are necessary to avoid post operative apnea.
- Pyloromyotomy is curative after resuscitation. The laparoscopic approach is becoming increasingly common, and seems to have some advantages over the open method.
- Early post operative feeding is safe and effective, frequently allowing discharge on the first post operative day.

In 1960, Willis J. Potts[1] wrote the following in his classic text on pediatric surgery *The Surgeon and the Child:*

> *"The operation for pyloric stenosis is the most satisfactory procedure in the entire field of pediatric surgery. The sick baby vomits all its feedings, the mother is distraught, a simple operation is performed, the baby thrives, and the mother is happy."*

Pyloric stenosis (PS) is a well-known surgical problem within the pediatric population. The treatment is quickly successful and is very rewarding to both parents and surgeon. The patient swiftly recovers and is returned to normal diet and activity within days.

The authors have nothing to disclose.

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This article outlines the classic elements necessary to care for the patient in a safe and effective manner. Because variation in care has been associated with increased levels of error, evidenced-based clinical practice guidelines are set forth, which can be modified easily to work well in a general surgery environment. This article has been crafted specifically with the adult general surgeon in mind.

HISTORICAL PERSPECTIVE

Although first described by Hildanus in 1627, it was not until Hirschsprung's unequivocal clinical and autopsy description in 1887 the pathologic basis of this disease is understood.[2] After this description, a variety of surgical approaches including the creation of a gastroenterostomy or forceful dilation via gastrostomy were practiced. The results, however, were poor with mortality rates approaching as high as 50%. Fredet in 1908 was the first to suggest a full-thickness incision of the pylorus followed by a transverse closure. Although this was successful, Ramstedt modified the technique and later described the sutureless, extramucosal longitudinal splitting of the pyloric muscle, which left an intact mucosa.[2] This technique continues to be the guiding principle of current surgical approaches for PS to this day.

EPIDEMIOLOGY AND ETIOLOGY

The incidence of PS varies with geographic and ethnic populations for reasons unclear. For example, PS occurs in approximately 2 to 4 per 1000 live births in the Western population, whereas the incidence has been reported to be approximately 4 times lower in the Southeast Asian and Chinese populations.[3–5] There have been numerous reports in the literature citing a wide variety of etiologic factors, some of which are discussed below.

Although no specific gene has been identified as the cause of PS, genetic syndromes, such as Smith-Lemli-Opitz, Cornelia de Lange, and other chromosomal abnormalities, have been associated with PS.[6] In addition, PS is 4 times more likely to present in boys than girls. The exact reason for the gender bias is still unknown. However, children of affected men are only affected between 3% and 5% of the time, whereas children of affected women are affected between 7% and 20% of the time.[7]

Pharmaceutical agents, hormones, and growth factors have all been linked to PS through small case reports.[6] Erythromycin, through its action as a motilin agonist, induces strong gastric and pyloric contractions that may eventually lead to hypertrophy of the pylorus.[6] SanFilippo[8] deduced that postnatal use of erythromycin estolate in infants might have resulted in a transient increase in the incidence of PS. In those infants, the incidence seemed to normalize after terminating the administration of erythromycin estolate. Since this early description, a few subsequent studies have described a 10-fold increase in the incidence of PS in infants treated with erythromycin, whereas other studies have failed to corroborate the findings.[9] Conversely, in terms of antenatal exposure, erythromycin estolate does not have a positive correlation for developing PS. Exposure of the lactating mother to erythromycin estolate does show increased rates of PS in whom the drug is clearly contraindicated.[6]

Several studies have also implicated higher acid exposure to have a causal effect on the development of PS.[10] A study in canines conducted by Dodge demonstrated that gastrin exposure to either puppies or pregnant female dogs resulted in an increased incidence of PS or duodenal ulcers.[11] This study was not replicated in other species and a definitive link has not been established.[12]

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