

# Brief Overview and Updates on Infantile Hypertrophic Pyloric Stenosis: Focus on Perioperative Management

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## ABSTRACT

Infantile hypertrophic pyloric stenosis is the most common surgical cause of vomiting in infancy. After adequate resuscitation, surgical pyloromyotomy is the standard of care for infants with pyloric stenosis. This article provides a brief overview and updates on hypertrophic pyloric stenosis in infants, with a focus on the surgical approach as well as perioperative management of this pathology. The most controversial aspect of the management of infants with pyloric stenosis is post-pyloromyotomy feeding, as there is no clear consensus in the literature on the best regimen. More randomized controlled trials are needed to establish the optimal resuscitation protocol in the preoperative phase and the ideal feeding regimen in the postoperative phase for infants with hypertrophic pyloric stenosis. [*Pediatr Ann.* 2021;50(3):e136-e141.]

Infantile hypertrophic pyloric stenosis (IHPS) was first described by Patrick Blaire in 1717, and the first large cohort of patients with IHPS was reported by Harold Hirschsprung in 1888.<sup>1</sup>

## EPIDEMIOLOGY

IHPS is the most common surgical cause of vomiting in infancy, with an incidence rate ranging between 0.5 and 4.2 per 1,000 live births in the

Western world; it is less common in the rest of the world.<sup>2</sup> Studies in the United States generally report higher preponderance of IHPS among non-Hispanic White mothers, with significantly lower prevalence among non-Hispanic Black mothers.<sup>2,3</sup> IHPS is considerably more common among boys, with a 4 to 5 times greater prevalence than in girls in most Western European and North American studies.<sup>2,3</sup> Seasonal variation in the incidence of IHPS has been described by many authors, the etiology of which remains elusive.<sup>2,3</sup>

IHPS is usually an isolated entity; however, it has been described in multiple genetic syndromes including Cornelia de Lange, Apert, trisomy 21, trisomy 18, and Denys-Drash.<sup>2</sup>

## ETIOLOGY

IHPS is a complex condition resulting from the interaction of genetic and environmental factors. The exact etiology remains unknown.

Five genetic loci linked to IHPS have been identified, and two of these (*IHPS2* and *IHPS5*) are associated with autosomal dominant inheritance of the disease. The gene for nitric acid synthetase

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(*NOS1*) and other genes implicated in smooth muscle hypertrophy are among the few candidates under investigation.<sup>2</sup>

The association between several perinatal factors and the development of IHPS has been investigated in many studies with controversial results. A recent meta-analysis proved a significant correlation between IHPS and children who are first-born, those born via caesarean delivery, those born prematurely, and those who are bottle-fed.<sup>4</sup> Another meta-analysis demonstrated a significant correlation between postnatal erythromycin exposure and IHPS development, especially if the exposure occurred in the first 2 weeks of life;<sup>5</sup> however, the association with the maternal intake of macrolides was less significant.<sup>6</sup> Breast milk was found to have a protective effect against IHPS as it contains high levels of vasoactive intestinal peptide, an agent of smooth muscle relaxation in the gastrointestinal tract that might enhance pyloric relaxation.<sup>7</sup>

## **PATHOGENESIS**

In infants with IHPS, the muscle of the pylorus progressively gets abnormally thickened, resulting in elongation and narrowing of the pyloric channel, leading to gastric outlet obstruction with compensatory dilation and hyperperistalsis of the stomach.

Sphincter function is controlled by a complex system involving the enteric nervous system, gastrointestinal hormones, and interstitial cells of Cajal. Abnormalities in hormonal control, extracellular matrix, smooth muscle fibers, growth factors, interstitial cells of Cajal, and pyloric innervation have all been implicated in the pathogenesis of IHPS.<sup>2</sup> The most widely accepted theory is the lack of neuronal nitric oxide in pyloric tissue, resulting in pylorospasm and pyloric muscle hypertrophy.<sup>8</sup>

## **CLINICAL PRESENTATION AND DIAGNOSIS**

The age of presentation of IHPS has been historically described to be between 2 and 12 weeks of life.<sup>9</sup> This was investigated and supported by Aboagye et al.<sup>10</sup> in 2014, with 93% of cases presenting between 3 and 10 weeks of age. The typical presentation is usually in the form of progressive, postprandial, projectile, nonbilious emesis in previously well infants. Vomiting usually starts as small nonbilious spit-ups after feeds and gradually progresses to become projectile (forceful); the emesis can become tinged with blood at later stages because of gastritis. At presentation, patients with IHPS are usually hungry; however, they may be profoundly dehydrated and lethargic in case of late presentation.

Upon physical examination a small mass may be palpable in the right upper quadrant of the abdomen, referred to as a “palpable olive,” which has a positive predictive value of 99%.<sup>11</sup> Positive test feeds can be used in which an infant is fed a small volume of milk or water while being examined to allow for a potentially easier palpation of the enlarged pylorus; however, this has fallen out of favor because of its inaccuracy.<sup>12</sup> In advanced cases, a visible gastric peristaltic wave can be demonstrated from left to right across the upper abdomen. The differential diagnosis of nonbilious vomiting in infants includes IHPS; gastroesophageal reflux; viral gastroenteritis; over-feeding; duodenal atresia, webs, and duplication; metabolic disorders; central nervous system lesions; and sepsis.

Icterypyloric syndrome occurs in approximately 15% of infants with IHPS, in whom the IHPS is associated with clinical jaundice. This syndrome improves after surgery and initiation of feeding. Jaundice in these patients might be an early manifestation of Gilbert syndrome.<sup>13</sup>

Currently, in most medical centers the diagnosis of IHPS depends largely on a real-time ultrasound scan with high sensitivity and specificity. A pyloric thickness (serosa-to-mucosa) of 3 mm or more and channel length of 12 mm or more by ultrasound imaging is accepted by many clinicians as the diagnostic criteria for IHPS.<sup>14,15</sup>

The ultrasonic evidence of IHPS is not always clear-cut, and the measurements might be borderline. In borderline cases, it is sometimes useful to visualize the relaxation of the pyloric canal after introduction of fluid into the stomach; this finding reliably excludes IHPS.<sup>16</sup>

Current guidelines may not be sufficient for accurate diagnosis of IHPS in infants younger than age 3 weeks because of the thin pyloric muscle thickness at this age.<sup>17</sup> In these situations, lower cut-off measurements may be more appropriate, and repeating the ultrasound in 1 or 2 days is sometimes useful.

A fluoroscopic upper gastrointestinal contrast study can also be used; however, this is time consuming and unnecessary in most instances, as the narrowed elongated pyloric channel is usually demonstrated in a string-like fashion.

The repetitive vomiting that occurs with IHPS will result in dehydration as well as depletion of hydrogen ions, chloride, potassium, and sodium. This classically leads to hypochloremic hypokalemic metabolic alkalosis. Hypokalemia is caused by loss of gastric juices, renal excretion, and intracellular movement. As dehydration becomes more profound, hypovolemia triggers sodium reabsorption in exchange for K<sup>+</sup> and H<sup>+</sup> ions. This leads to urinary excretion of H<sup>+</sup> ions in the distal tubules when K<sup>+</sup> is not available, resulting in paradoxical urinary acidosis.<sup>18,19</sup>

Because of the high index of suspicion, as well as early use of ultrasound

scans, the diagnosis of IHPS is usually made early, before the development of the electrolyte and acid-base balance derangements. This results in shorter duration of required preoperative resuscitation and smoother postoperative course.

### PREOPERATIVE MANAGEMENT

Metabolic alkalosis can reduce the inspiratory drive of the infant, potentially causing postoperative apnea and thus complicating extubation after general anesthesia.<sup>20</sup> IHPS is not a surgical emergency, as the priority is to resuscitate the infant and to correct the acid base and electrolyte derangements. This approach has been proven to improve the outcome in infants with IHPS.<sup>21</sup>

The resuscitation requires admission to a regular ward and administration of intravenous fluid, usually for 24 to 48 hours. Infants generally require one or more bolus of a 20-mL/kg normal saline solution, and they are usually placed on 1.5 to 2 times the volume of maintenance physiologic fluid (5% dextrose in 0.45 normal saline and 20 mEq/L of potassium chloride). Potassium is added to the fluid infusion after the urine output has normalized. Patients are usually kept *nil per os* (ie, nothing by mouth), and a nasogastric tube may or may not be placed. Indicators of adequate resuscitation include skin turgor, moist mucous membranes, and normalization of urine output. Serum bicarbonate levels less than 28 mEq/L and serum chloride levels greater than 100 mEq/L are commonly used as objective markers indicating that the infant is ready for general anesthesia. A recent study suggested a resuscitation algorithm based on preoperative serum chloride level.<sup>22</sup> The authors of this study recommended the administration of two 20-mL/kg normal saline boluses separated by a 1-hour interval before rechecking the laboratory results if the initial serum chloride value

was less than or equal to 97 mEq/L, and three boluses of 20 mL/kg of normal saline if the presenting serum chloride level was less than 85 mEq/L.<sup>22</sup> Recent reports have suggested that the use of H<sub>2</sub>-receptor antagonists may reduce the severity of electrolyte disturbances in patients with IHPS. H<sub>2</sub>-receptor antagonists reduce gastric acid secretion, and as a result, pyloromyotomy can be performed earlier, hence reducing both hospital stay and costs.<sup>23,24</sup>

### EXTRAMUCOSAL PYLOROMYOTOMY

Extramucosal longitudinal pyloromyotomy, as first described by the French surgeon Pierre Fredet in 1908<sup>25</sup> and later by the German surgeon Wilhelm Conard Ramstedt in 1912,<sup>26</sup> remains the standard surgical procedure for IHPS. This procedure entails longitudinal splitting of the thickened pyloric muscle, resulting in bulging out of the pyloric submucosa and thus relieving the gastric outlet obstruction.

To perform the procedure, the patient is placed in the supine position. Prophylactic antibiotics are generally not indicated;<sup>27</sup> however, many pediatric surgeons opt to administer one prophylactic dose of antibiotics within 1 hour of starting the procedure. The classic open approach has been traditionally described through a right upper quadrant transverse abdominal incision; however, such an approach has led to cosmetically unappealing results. Several other approaches were then introduced; the most widely used is the supraumbilical skin-fold incision described by Tan and Bianchi<sup>28</sup> in 1986. This approach resulted in better cosmetic outcomes.

The edge of the liver is retracted cranially via a protected malleable retractor. The greater curvature of the stomach is then identified, which is then grasped and pulled to the left to expose and sta-

bilize the hypertrophied pylorus. The distal extent of the enlarged pylorus is marked by the pyloric vein of Mayo. A longitudinal serosal incision is then made over the thickened pylorus. The hypertrophied muscle is then split using a blunt instrument, until the pyloric submucosa bulges into the incision. Air may be injected into the stomach via a nasogastric tube to test for inadvertent duodenal perforation

Laparoscopic pyloromyotomy was first described in 1991<sup>29</sup> and is currently the surgical approach of choice for many surgeons across the globe. Like in open pyloromyotomy, prophylactic antibiotics are not indicated prior to laparoscopic pyloromyotomy;<sup>30</sup> however, many pediatric surgeons all over the world still prefer to administer antibiotics within 1 hour of the procedure to decrease the risk of wound infection. It is usually performed via a 3-mm or a 5-mm umbilical laparoscope; a “stab wound” incision on either side of the upper abdomen is used for the passage of 3-mm instruments. The duodenum is gently grasped to stabilize the thickened pylorus, then, similarly to the open approach, a longitudinal incision is made over the thickened pylorus using an arthrotomy knife or electrocautery, followed by gentle splitting of the hypertrophied pyloric muscle using a pyloric spreader.

### POSTOPERATIVE CARE

There is currently no consensus on a standardized post-pyloromyotomy feeding regimen in the literature. Some authors allow patients to feed immediately after surgery, whereas others, in an attempt to avoid postoperative emesis, have suggested a period of withholding feeds for several hours postoperatively. Some authors prefer to start *ad libitum* feeds, and others tend to follow a scheduled incremental feeding regimen. A retrospective analysis of 227

infants showed that a standardized feeding regimen offers no advantage over *ad libitum* feeds after pyloromyotomy.<sup>31</sup> Based on inconsistent evidence, Graham et al.<sup>32</sup> advocated a delay in the initiation of feeds for at least 4 hours postoperatively, followed by *ad libitum* feedings. This is in agreement with the findings of a recent prospective randomized trial in which infants who were allowed *ad libitum* feeds after a 4-hour period nothing by mouth were able to reach full feeds much faster and had a shorter length of hospital stay than the infants who were put on an incremental feeding regimen; there was no difference in the postoperative vomiting and readmission rate between the two groups. The same study also showed that infants with preoperative serum chloride less than 100 mEq/L needed longer time to reach goal feeds compared to infants with normal laboratory values.<sup>33</sup>

### OPERATIVE COMPLICATIONS

Despite the fact that pyloromyotomy is currently considered a fairly safe operation with reproducible excellent outcomes, multiple potential complications of the procedure have been reported in the literature. The overall complication rates vary between 4.6% and 12% in most series.<sup>34</sup> Incomplete pyloromyotomy (mainly occurring on the stomach side), mucosal perforation (mainly occurring on the duodenum side), wound dehiscence and infection, delayed feeding, and postoperative vomiting are among the most commonly reported complications.

Vomiting in the first few days after surgery is common and usually resolves spontaneously. Persistent emesis after pyloromyotomy may be caused by viral gastroenteritis, gastroesophageal reflux, aggressive feeding protocols, or incomplete pyloromyotomy (least common).<sup>35</sup> If significant vomiting is still present after more than 1 week postoperatively,

an upper gastrointestinal contrast study should be performed to rule out incomplete pyloromyotomy or possible duodenal perforation. An ultrasound in these situations is not useful because the pyloric hypertrophy needs up to 4 to 5 months to resolve.

Several studies have looked at the predisposing factors for postoperative emesis after pyloromyotomy. A recent prospective analysis showed that a lower body weight at the time of the operation and the presence of electrolyte abnormalities on admission (chloride, potassium, and anion gap) are associated with a significant increase in the incidence of postoperative emesis.<sup>36</sup>

Many studies have proven the safety of the laparoscopic approach, which also results in a lower incidence of postoperative emesis, less pain, and faster recovery as compared to the open technique.<sup>37</sup> In a retrospective analysis of data from nine high-volume institutions from the US, UK, and Canada, the incidence of incomplete pyloromyotomy was 0.29% in the open group as opposed to 1.16% in the laparoscopic group. Mucosal duodenal perforation occurred in 0.29% of the patients in the open group and 0.83% in the laparoscopic group. The authors concluded that despite the fact that laparoscopy is associated with a statistically significant increase in the risk of incomplete pyloromyotomy, the effect size is small and of questionable clinical relevance.<sup>38</sup> A recent retrospective analysis of 3,256 infants derived from the 2013 and 2014 American College of Surgeons National Surgical Quality Improvement Project-Pediatric demonstrated that the outcomes after laparoscopic pyloromyotomy are equivalent or superior to the open approach.<sup>39</sup> In this study the authors were able to show a statistically significant reduction in the overall morbidity in the laparoscopic group (2.4% vs 1.2%,  $P = .007$ ) and no

significant difference in the incidence of return to the operating room for a second pyloromyotomy between the two groups (0.42% in the open group versus 0.29% in the laparoscopic group,  $P = .2$ ).<sup>39</sup>

### ALTERNATIVE MANAGEMENT APPROACH

Many practitioners, particularly from Asian countries, have reported good results with using intravenous and oral atropine in the management of some patients with IHPS, thus avoiding surgery and anesthesia. Atropine is an anticholinergic agent with significant antimuscarinic activity. It works in patients with IHPS by relaxing the smooth muscles of the pyloric channel, thus relieving the obstruction.

This approach requires a prolonged hospital stay, possible need for parenteral nutrition, as well as high failure rate; however, it might be helpful if anesthesia or surgery are not available or are medically contraindicated.

A recent study from Japan retrospectively analyzed data of 585 patients; the overall success rate of intravenous atropine was 78.9%, compared to a 100% success rate of pyloromyotomy, with the rate of postoperative complications as low as 2.8% of the patients. Medical management required longer hospital stay than surgery. The authors of this study concluded that surgery remains the suitable standard management option for IHPS, with its high success rate, minimal complications, and shorter hospital stay compared with intravenous atropine therapy.<sup>40</sup>

### CONCLUSION

IHPS is a relatively common surgical condition affecting infants from age 2 to 12 weeks, and its etiology is unknown. Pyloromyotomy, either open or laparoscopic, is the most suitable management option and has a low rate of

postoperative complications. The most controversial aspects of the management of infants with pyloric stenosis is the optimal resuscitation protocol in the preoperative phase, as well as the ideal feeding regimen in the postoperative phase, as there is no clear consensus in the literature. More randomized controlled trials are needed to optimize these management protocols in infants with hypertrophic pyloric stenosis.

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