Pyloric Stenosis

14

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14.1 Introduction

Infantile hypertrophic pyloric stenosis (IHPS) is a mechanical obstruction of the gastric outlet, due to a simple benign hypertrophy and hyperplasia of the smooth muscle fibers of the pylorus. The result is a narrowing and elongation of the pyloric channel. The origin remains unknown more than a century after the first treatments.

Gastric outlet obstructions in infants have been described several times (Fabricius Hildanus, 1627 [1]; Patrick Blair, 1717 [2–4]; Christopher Weber, 1758 [5]; George Armstrong, 1777 [6, 7]; H. Beardsley, 1788 [7, 8], Williamson, 1841 [9], Siemon-Dawoski, 1842 [10]) before the first unequivocal modern description of IHPS in 1887 by the Danish Harald Hirschsprung who gave complete clinical details and accurate pathological findings [9, 11, 12] and then by Sir William Osler, from Ontario, Canada, in 1903 [13]. Probably the first successful surgical attempt to solve the problem was by Pietro Loreta from Bologna in 1887 [14]. He described an antral opening to dilate the pylorus from the stomach. Then several procedures were performed, such as a gastroenterostomy by Lobker in 1898 on a 10 weeks old infant. The surgical treatment still in use today is an extramucosal pyloromyotomy (EMP) which bears the names of Fredet-Weber-Ramstedt, referring to those who were supposed to have done it first. However, the procedure performed in 1907 by Pierre Fredet was an extramucosal pyloroplasty [15, 16]. In 1910 Weber did an extramucosal splitting of the muscle followed by a transverse suturing [17], and on August 23, 1911, Conrad Ramstedt (also written Rammstedt, due to a misspelling of his name) performed an EMP leaving the two muscular margins free, but covered the myotomy with an omentoplasty [18]. So the first true EMP was done in Edinborough on February 7, 1910 by Sir Harold Stiles as attested by his original operating report (thanks to Gordon McKinley). However, he did not report it at that time, and the date and records of his operation were published by Mason Brown only in 1956 [19].

14.2 Epidemiology

Infantile hypertrophic pyloric stenosis (IHPS) is the most common cause of vomiting in the postnatal period, occurring with a prevalence rate of ≈2 per 1000 live births in Europe and North America, predominantly in boys compared to girls (4:1 up to 5:1). IHPS is less frequent in children of African or Asian origin [20–23]. In 1927, Still already noticed that it is more frequent in the firstborn [4, 24], but this point is debated by epidemiologists [25]. A decline in the incidence of IHPS has been reported over the past two decades

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M. Lima (ed.), *Pediatric Digestive Surgery*, DOI 10.1007/978-3-319-40525-4_14

in Northern European countries and the United States [20, 25–28]. Decreases in rates of IHPS were observed among foreign-born Hispanics and foreign-born Asians, but not among their US-born counterparts, suggesting an environmental origin [22].

In a case-control study, Svenningsson reports cesarean section, prematurity, primiparity, young maternal age as significant risk factors for IHPS [28].

IHPS affects infants between 3 and 8 weeks. The mean age at diagnosis is about 40 days; 95 % of the cases being diagnosed between age 2 and 11 weeks [25]. However, delayed cases up to 4 years of age have been reported [29].

On the other hand, prematurity is associated with a higher rate of IHPS than term babies [25]. Premature infants develop IHPS at a later chronological age, than term infants [30] and have a higher female preponderance [31], the sex-ratio in preterm being nearly 1:1 [25]. Small weight for gestational age babies have also a significantly higher rate of pyloric stenosis compared with heavier infants [25].

14.3 Etiology

More than a century after its first description, IHPS remains a disease of unclear origin. IHPS can occur as an isolated disease, but it is also well established that it can be associated with chromosomal abnormalities, congenital malformations, and clinical syndromes, which indicate a genetic involvement associated with environmental factors. However, no causal gene or sequence variant has been identified to date and the pathophysiology at a molecular level remains unclear [32].

14.3.1 Genetic Factors

The cases of IHPS reported by Armstrong in 1777 were three siblings [6]. Recurrence risk in families and twin studies [33, 34] provide a high suspicion of a genetic origin, even if debated [32]. Yang recommends that the asymptomatic co-twin should be investigated when one of the twins presents with IHPS [33]. Carter first demonstrated non-syndromic pyloric stenosis as a complex, multifactorial, sex-modified threshold trait [35, 36]. A reanalysis by Mitchell of data from several studies concluded that IHPS is determined by two or three loci of moderate effect conferring individual genotype relative risks of up to 5 [37]. To date five genetic loci (IHPS1 to IHPS5) have been identified. IHPS1, which encodes the enzyme neuronal nitric oxide synthase (NOS1), was considered as a possible evidence that a defect in nitric oxide production may play a role in the etiology of IHPS. However, the evidence for linkage and association is weak and has not been confirmed. Two other loci, IHPS2 (16p13-p12) and IHPS5 (16q24.3), have been identified, suggesting autosomal dominant inheritance [38, 39]. A genome-wide single nucleotide polymorphism (SNP) identified IHPS3 on chromosome 11q14–q22 and IHPS4 on Xq23 [40]. Further analysis provided suggestive evidence for a third locus on chromosome 3q12–q25 [41].

Seven percent of children with IHPS had a major malformation compared with 3.7 % of the general population [20]. IHPS is associated with many clinical syndromes that have known causative mutations, such as Cornelia de Lange and Smith-Lemli-Opitz syndromes, chromosomal abnormalities, including translocation of chromosome 8 and 17, and partial trisomy of chromosome 9. An extensive detailed up-to-date review of what we know in the genetics, the molecular studies, and the metabolic studies in IHPS has been published by Peeters et al. [42].

14.3.2 Environmental Factors

A variety of environmental and mechanical factors have been implicated in the occurrence of IHPS. Sleeping position, maternal smoking, and postnatal erythromycin administration are the most commonly evocated factors [21, 32].

In several studies, the rate of IHPS is higher in infants of smoking mothers than among infants of nonsmoking mothers [25, 28].

Sharp decline in the incidence of IHPS in Denmark and Sweden, during the 1990s, coincides with successful campaigns to discourage the prone sleeping position as a prevention of sudden infant death syndrome. This led to the hypothesis that sleeping prone may be a risk factor for IHPS [21, 32]. This could be related to the place where the milk accumulates in the stomach according to the position. However, a similar German study concluded that a common cause was unlikely [27].

Several studies have suggested an increased risk of IHPS following child exposure to erythromycin in the postnatal period, but not through the mother pre- or post birth. Erythromycin is known for its prokinetic effects mediated by its action as a motilin receptor agonist, which could affect gastric motility and/or pyloric contraction [43–45].

The diet itself could play a role as it seems that formula feeding is associated with significantly increased risk of IHPS compared to breastfed children [46]. The development of a delayed pyloric stenosis during transpyloric feedings has also been reported [47, 48].

14.4 Physiopathology

IHPS results in an important thickening of the muscular lays of the pylorus. The enlarged pylorus becomes longer and thicker. This enlargement impairs the normal release of the pyloric sphincter thus occluding the lumen and realizing a gastric outlet obstruction with subsequent vomiting failure to thrive and dehydration.

The pyloric sphincter function involves intrinsic myogenic activity of the smooth muscle, the interstitial cells of Cajal (ICC) which play a role of intestinal pacemaker, gut hormones, and the autonomic and enteric nervous systems. Associated to IHPS, abnormalities have been observed in gastrin levels, enteric nerve terminals, nerve supporting cells, ICC, smooth muscle cells, growth factor synthesis and receptors, and extracellular matrix [49]. But the major hypothesis is that a primary defect in production of nitric oxide (NO) by nitrergic nerves of the enteric nervous system leads to failure of relaxation of the pyloric smooth muscle [50, 51]. Abel brought evidence that nitric oxide synthase (NOS) has been implicated in the pathogenesis of IHPS, since NOS expression is diminished in both circular and longitudinal muscles, as well as in the myenteric plexus [52]. Looking for the ontogeny of the peptide innervation of the pylorus, Abel reports that NOS and vasoactive intestinal polypeptide (VIP) are colocalized to the same nerves in the circular muscle and in the myenteric plexus; they are diminished by the same proportion in IHPS; so he concluded that the initial lesion occurs by 12 weeks of gestation and could be the increment in vasoactive intestinal polypeptide (VIP) in pyloric myenteric ganglia [50, 52].

14.5 Clinical Presentation and Diagnosis

Non-syndromic IHPS affects normally fed healthy children. The story begins with gradually increasing non-bilious vomiting becoming projectile. The vomiting and the inability to be fed lead to dehydration with associated physical signs: loss of weight, skin fold, depressed fontanel, dry mucosa, oliguria, and constipation. Children are hungry and are eager for more to eat without nausea. Given a test meal, visible gastric peristalsis may be seen when the child lays supine.

The palpation of the pylorus is often possible for an experienced examiner. The pylorus must be searched for on the midline just below the edge of the liver. It can be felt as an olive, hence the world "pyloric olive." It was described in 1923 by Sir G. Frederic Still (1868–1941), who is considered to be the "Father of British pediatrics" [24], as "a small barrel-shaped hard tumor (…) varying in size from the thickness of an ordinary lead pencil up to that of a hazelnut" or "as hard as a calcareous gland" [3, 24]. The term "olive" was given by Ladd in 1946 [53]. The palpation of an olive has a 99 % positive predictive value [54].

The patient history, the clinical conditions, and the abdominal palpation of a pyloric olive allowed for a diagnosis of IHPS. Historically, the

diagnosis of IHPS was only made by clinical history and physical examination. By the years 1930s, as radiology improved, the upper gastrointestinal (UGI) came to support diagnosis of IHPS. Today ultrasounds (USs) have replaced UGI. However, UGI can still be used in some unusual circumstances or places where US is not available. Then an isotonic hydrosoluble contrast media should be preferred to the old barium meal, in case of aspiration in a vomiting child [55]. The radiological signs of IHPS are gastric retention, parenthesis shape of the antrum ("shoulder sign") ending with a "beak sign" (the narrowed gastric antrum entering the pylorus), and lengthening of the pylorus with the typical "double-track" sign, or even triple (small trickle of contrast in the thickened and elongated pylorus).

The first diagnostic use of ultrasounds (USs) for IHPS was done by Teele and Smith in 1977 [12, 56]. Today in almost all pediatric medical centers, the high-resolution, real-time US is the modality of first choice to confirm the diagnosis of IHPS [12]. It is a noninvasive technique not using ionizing radiations. It is commonly available with relatively low cost. Ultrasounds have accuracy and sensitivity approaching 100 % [57]. False-positives are rare. However, the distended stomach filled with gas can rotate the pylorus dorsally, thus resulting in its difficult localization and measurements. Thus it requires appropriate equipment, expertise, and clinical experience to produce best results [12].

The positive US diagnosis is based on precise measurements of canal length and muscle thickness. A pylorus is considered hypertrophic when the single hypoechoic muscle layer measured transversely exceeds 3 mm [57–63]. There is some variability for pyloric channel length criteria ranging from 14 to 17 mm in literature, as the pyloric canal lengthens with age [58–63].

US diagnosis can be difficult in infants below 3 weeks or preterms because of the thin pyloric muscle thickness [57]. However, it seems that the normal values are not affected by weight, corrected gestational age, or duration of symptoms [31]. When in doubt, repeated US within 1 or 2 days can be an issue.

14.6 Preoperative Management

IHPS is a medical emergency. The vomiting associated with IHPS leads to depletion of sodium, and hydrochloric acid, thus resulting in hypochloremic metabolic alkalosis that can be partially compensated by a respiratory acidosis. Anesthesia and surgery on an infant in poor metabolic condition can be harmful. Because IHPS is not a surgical emergency, the hypochloremic metabolic alkalosis should be corrected before surgical intervention with adequate fluid and electrolyte IV replacement. This can require a few days. As the potassium is mostly intracellular, its loss may not appear immediately in the kalemia. However, it must be anticipated [64].

14.7 Surgical Treatment

The surgical procedure used to relieve the pyloric obstruction remains the extramucosal pyloromyotomy (EMP) as described more than a century ago. The pyloric serosa is open longitudinally with a blade on its avascular part. Then the thick muscle is split using a smooth grasper or a pyloric spreader, until the mucosa is exposed and bulges out between the muscular edges. It is essential to ensure total opening of the pylorus. Most of the "recurrences" are incomplete myotomies. The splitting of the muscle has to run from the gastric antrum to the pyloroduodenal junction. This is the most dangerous point. At this very place, the mucosa comes up as the muscular wall becomes suddenly thinner, bearing a risk of mucosal perforation (Fig. 14.1). For this reason the surgical procedure must end with a search for potential perforation, using gas insufflation in the stomach via a gastric tube. Bubbles appearing on the pyloric mucosa evidence a leak. A perforation per se is not a major problem as long as it is immediately recognized and sutured. It will only differ the first postoperative meals.

14.7.1 Open Surgery

If EMP has not evolved over time, the surgical access has changed substantially. The initial

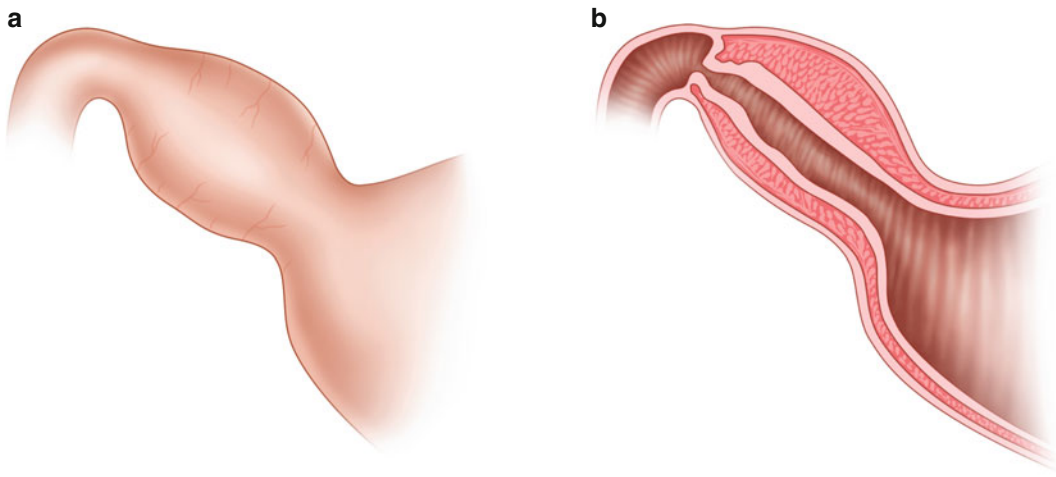

Fig. 14.1 Drawing of the hypertrophic pylorus showing the dangerous place where the mucosa comes up as the muscular wall becomes suddenly thinner

open approach was a midline laparotomy, which has moved toward a transverse laparotomy and then a smaller transrectal (from the rectus abdominis) approach in the right upper quadrant. A first major change toward minimal invasive surgery was suggested by Bianchi, with a circumumbilical approach, which rapidly spreads among pediatric surgeons [65]. Tan and Bianchi described in 1986 a semicircular supraumbilical skinfold incision leaving an almost invisible scar. Through this minimal incision, the pylorus is palpated, seized with a Babcock clamp, and delivered through the umbilicus to perform the EMP out of the abdomen. Somehow it can be difficult to bring out a big firm pylorus. Then the aponeurotic fascia must be open longitudinally on the midline as far as needed, to allow easy extraction. Once the EMP is done, the fascia is sutured. The transumbilical incision for EMP allows excellent access to the pylorus, while leaving an almost undetectable scar.

Modifications of the Bianchi's umbilical approach were suggested by some authors. As there are some obvious technical difficulties in delivering a large pyloric tumor through the umbilicus even after opening the midline fascia, instead of bringing the pylorus out through the umbilicus with subsequent traction, the pylorus is kept in situ and the EMP is performed intracorporeally [66–69]. To stabilize the pylorus and draw it up just under the umbilical wound, suspension threads are placed in the hypertrophic muscle [67, 69].

14.7.2 Laparoscopic Pyloromyotomy

One of the first laparoscopic procedures even done in children were pyloromyotomies performed by Dominique Grousseau and Jean-Luc Alain from Limoges, France, in 1989, and published first in French in 1990 [70] then in English with ten cases in 1991 [71]. The technique was long to gain popularity but by some pediatric surgeons involved in pediatric minimal invasive surgery.

Initially three ports were used: a 5 mm in the umbilicus for the telescope, using the Hasson's open technique, and two 3 mm for instruments, one on the midline, the second on the right midclavicular line just below the liver. The pylorus was caught in a Babcock grasper. The pyloric serosa was opened longitudinally on its anterior face using a 3-mm retractable knife. Then the pyloric muscle was split with a laparoscopic pyloric spreader.

The laparoscopic EMP has evolved toward simpler technique. Nowadays, only one 5-mm port is placed in the umbilicus and none for the instruments. As the left hand is used only to seize the pylorus, the instrument is left in place from beginning to end and do not require a port. As it appeared difficult to grab the big firm pylorus

with a small Babcock grasper, today we use a smooth Johann grasper placed transversally on the duodenum just below the pyloroduodenal junction. This allows to lift up or to rotate the pylorus with a good exposure of the pyloroduodenal junction. A small 2–3-mm disposable knife (designed for ophthalmology or for arthrotomies) is inserted through the skin on the midline just in front of the pylorus. The blade is not pushed down to the pylorus, but the pylorus is lifted up toward the blade. Then the wound site is used to insert a smooth 2 or 3-mm grasper (Johann, Maryland) to split the muscle. The use of one of the specially designed pyloric spreaders is helpful but not mandatory.

There are no contraindications to laparoscopic IHPS. However, prematures bear a risk of cerebral bleeding due to the elevation of pressure in the superior vena cava related to insufflation, even done at a low pressure (5–6 mmHg). Children with cardiac defect shunting from left to right could embolize in their brain and therefore should be recused for laparoscopy as those with lung anomalies (Figs. 14.2 and 14.3).

14.7.3 Which Is Better: Lap or Open?

We have had to wait for more than a decade until data were available to compare open with laparoscopic EMP (lap).

The French team of Nantes has performed a randomized prospective study of respectively 50 EMPs done by laparoscopy with 52 open. The

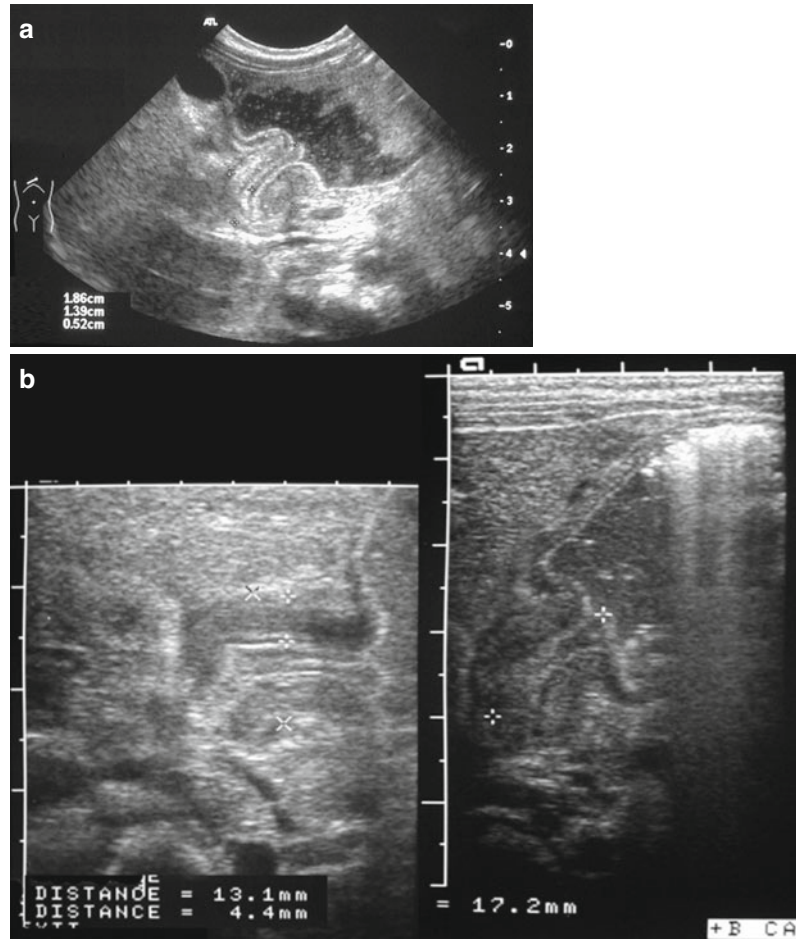

Fig. 14.2 (**a**, **b**) Ultrasounds of IHPS. Measures are taken between calipers + and X. The lamina muscularis mucosae appear as a white stripe in the thick muscle. Note the dilated stomach filled with echoes

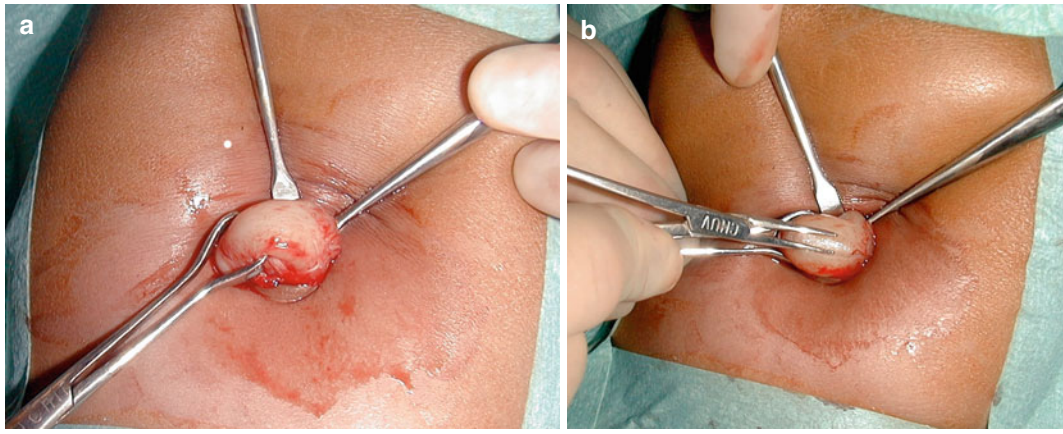

Fig. 14.3 Extramucosal plyloromytotomy through the umbilical approach. (**a**) The pylorus has been delivered through the umbilicus. (**b**) The pyloric muscle is split using a mosquito and the mucosa is exposed between the muscular edges

durations of surgery and anesthesia were longer in the lap group. There was no difference in the incidence of postoperative vomiting, and the complications were similar (1 perforation each ie 1 %; 2 wound complications open versus 1 lap ie 3 %; 3 incomplete myotomies after lap ie 3 %) but significantly with less pain in the lap group ($p < .0001$) [72].

A multicenter international double-blind controlled trial across six tertiary pediatric surgical centers has been published with 180 infants randomly assigned to open ($n = 93$) or laparoscopic EMP ($n = 87$) [73]. Complications were similar (17 vs 15). All perforations (1 vs 2) and the 3 incomplete myotomies by laparoscopy were done by nontrainees. Full oral feeding was achieved faster in the lap group ($p = .002$); there were less pain in the lap group ($p = .011$); and the postoperative hospital stay was shorter in the lap group ($p = .027$). The postoperative vomiting and complications were similar. The parental satisfaction was higher for the lap group ($p = 0.011$). The design of the study was to recruit 200 infants (100 per group). However, the data monitoring and ethics committee recommended halting the trial before full recruitment because of significant treatment benefit in the laparoscopy group at interim analysis. Their conclusions were: "Both open and laparoscopic pyloromyotomy are safe procedures for the management of pyloric stenosis. However, laparoscopy has advantages over open pyloromyotomy, and we recommend its use in centers with suitable laparoscopic experience."

Keith Georgeson and his team from Birmingham, AL, have compared the incidence and type of technical complications seen in a retrospective series of pyloromyotomies done by open (225) and by laparoscopic (232) EMP in similar groups performed by multiple surgeons. The overall incidences of complications were similar in the two groups (open 4.4 %; lap 5.6 %). There was a greater rate of perforation with the open technique (3.6 % vs 0.4 %) and a higher rate of postoperative problems including incomplete myotomy in the laparoscopic group (0 vs 2.2 %). They conclude that: "This lower rate of perforation could be attributed to improved visualization because of the magnification provided by laparoscopy. Alternatively, the lower perforation rate could be owing to a less "aggressive" pyloromyotomy" [74].

Sola published a meta-analysis upon six prospective studies of level 1 (5) or 2 (1) in which it appeared that laparoscopic EMP had a lower total complication rate ($p = .04$) due to a lower wound complication rate ($p = .03$). The laparoscopic EMP had shorter time to full feedings ($p < .00001$) and shorter postoperative hospital stay ($p = .0005$) with no statistically significant differences in mucosal perforation (0.9 % vs 1.3 %), wound infections, and postoperative vomiting. There were six incomplete myotomies (4 lap vs 2 open).

The conclusion was: "This systematic review and meta-analysis favors the laparoscopic approach with significantly reduced rate of total complications, which is mostly due to a lower wound complication rate" [75].

Finally, Carrington and the British team from Great Ormond Street Hospital for children, London, have compared the costs of the laparoscopic EMP with the open approach in a multicenter randomized double-blind controlled trial, for which the primary outcomes were time to full feeds and time to discharge. Operation costs were similar between the two groups. A shorter time to full feeds and shorter hospital stay in lap versus open patients resulted in a highly significant difference in ward costs ($ 2,650 ± 126 lap versus $ 3,398 ± 126 open; $p = .001$) and a small difference in other costs. Overall, laparoscopic patients were $ 1,263 less expensive to treat than open patients [76].

To summarize, the quoted advantages of laparoscopic pyloromyotomy compared to the open approach are reduced postoperative pain, shorter hospital stay, earlier return to normal activity, and cosmetic benefits [77, 78].

14.7.4 Postoperative Period

Postanesthetic apnea in the premature <60 WGA is well known and specific recommendations have been made to prevent them. However, postanesthetic apnea can occur in full-term babies without perinatal problem after some surgical procedures including cures of IHPS [79]. Some recommendations have been made but a strict postoperative monitoring and supervision in a specialized environment is wise [80, 81].

The nasogastric tube is suctioned at the end of the procedure before its removal, except in case of sutured perforation. Wounds are infiltrated with 0.25 % bupivacaine 2 mg/kg for postoperative pain relief. Antibiotics are given only in case of mucosal tear. Oral feeding may be resumed on return to the ward and increased as tolerated.

Isolated vomiting can endure for a few hours/days (≤2 days) after surgery in 10–15 % of cases. They are related to the gastric irritation associated with preoperative vomiting and to the traction on the pylorus during the procedure [82]. Subsequently, they are less frequent after laparoscopic or transumbilical intracorporeal EMP than after exteriorized one. However, they must not be minimized as they can reveal a perforation.

The more electrolyte abnormalities children have at the time of diagnosis, the longer they stay in the hospital [64].

14.7.5 Complications

Complications are between 1 and 3 % in the hands of pediatric surgeons and mostly related to incomplete myotomies or perforations [72, 82]. Infections of the umbilical wounds have been described (1–7 %). However, with the increment of laparoscopy in neonates and infants, pediatric surgeons have learned how to clean the umbilicus, and the rate of umbilical infections is decreasing.

Complications per surgeon drop with experience. This has been evidenced in laparoscopic EMP. Mucosal perforation was experienced by 8.3 % of the patients in the initial series, as compared with 0.7 % in the later series reported by Van der Bilt [78]. Insufficient pyloromyotomy occurred in 8.3 % of the initial series, as compared with 2.7 % of the later series. He suggested that the learning curve could be 15 laparoscopic IHPS [78] (Fig. 14.4).

14.8 Nonsurgical Conservative Treatment for IHPS

Before the era of the EMP and until the years 1960s, IHPS were treated conservatively using atropine or equivalents (belladonna, atropine methylnitrate (eumydrin)). Although pyloromyotomy became the first choice of treatment in Western countries, several authors, mostly from Asian countries (Japan [83–86], Taiwan [87], and India [88]) but also from Germany [89], have revisited the nonsurgical treatment using intravenous or oral atropine for IHPS. Atropine sulfate is given daily for 1–8 days at various regimens [89] with increasing doses until vomiting stopped

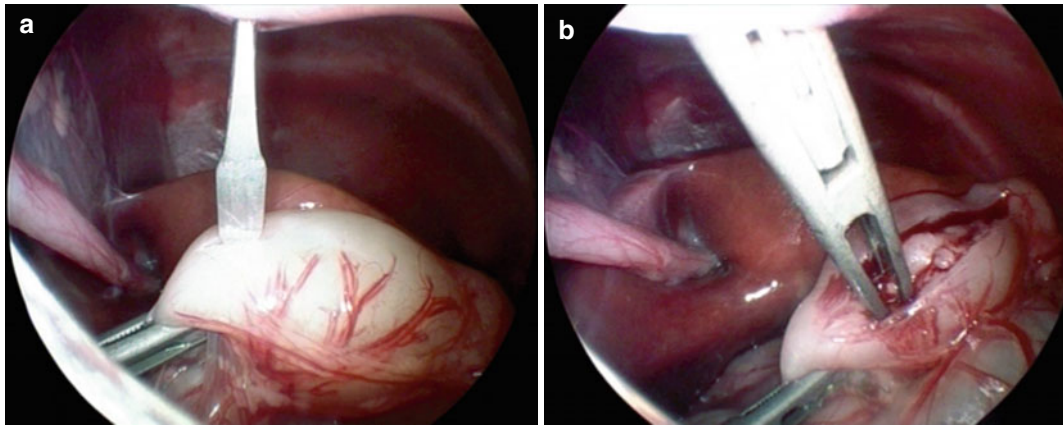

Fig. 14.4 Laparoscopic extramucosal plyloromytotomy. (**a**) Opening the serosa. Note the Johann grasper holding the duodenum just below the enlarged pylorus. The opening of the serosa is done with a disposable ophthalmologic knife in the avascular zone. (**b**) Splitting the muscle with a standard smooth dissector. The mucosa is already bulging proximally to the grasper

then maintained for 2 weeks. The rationale for atropine therapy is that the physiopathology of IHPS may be partially due to impaired function of acetylcholine and muscarinic receptors, thus releasing the pyloric muscle. Medical treatment may require 7 days or more of skilled nursing and careful follow-up. The results are fairly good with no significant complications. About 10–25 % patients require surgery for failure of medical treatment.

Conservative medical treatment with atropine is an option. To date there are no randomized controlled studies answering the question whether therapy with atropine can achieve sufficient resolution of IHPS to avoid surgery but only case series and a retrospective cohort study with low level of evidence [90]. Mercer studied ten relevant articles on the use of atropine for IHPS. The success rate of atropine therapy is about 85 %, whereas surgical EMP is >95 % [90]. Under a humoristic editorial title ("Medical Treatment of Idiopathic Hypertrophic Pyloric Stenosis: Should We Marinate or Slice the "Olive"?"), Rudolph, a pediatric gastroenterologist, advocates for the surgery arguing it solves the problem within 48–72 h with less than 1 % complications for a lower cost [91]. As per Aspelund, we believe conservative medical treatment with atropine should be considered as an alternative in infants with contraindications to anesthesia or surgery [92].

14.9 Other Gastric Outlet Obstructions

In infants, gastric outlet obstruction (GOO) is most often due to IHPS. However, several conditions other than IHPS may cause non-bilious vomiting in infants and children that we must be aware of (Table 14.1).

Gastric polyps may be either hyperplastic or adenomatous. Hyperplastic polyps are most common in children and account for 70–90 % of benign gastric polyps. A study at Johns Hopkins University reported that the prevalence of duodenal polyps in children was 0.4 % (22 of 5,766) of upper gastrointestinal endoscopies. Most of the duodenal polyps in that series were syndromic and were commonly associated with familial adenomatous polyposis [63, 94, 96]. However, sporadic cases have been described before or inside the pylorus [93].

An ectopic pancreas is not uncommon in children and the pyloric location has been described. Besides GOO, they can cause epigastric pain [101] and develop gastrointestinal bleedings or late malignant transformation. Thus surgical removal is suggested [100].

Even very unusual in infants, antral or pyloric malignancies have been reported and should always be considered as a possible etiology of a pyloric obstructive mass in older children [97, 105, 107]. The literature concerning such

Table 14.1 Gastric outlet obstructions non-IHPS

Anatomical anomalies	Treatment	Refs.
Prepyloric masses:		
Pyloric polyp	Endoscopic resection	[93–97]
Ectopic pancreas	Lap or open resection	[93, 97–102]
Tumors	Surgical resection, pyloroplasty	[97, 103–107]
Pyloric web	Endoscopic or lap pyloric opening	[63, 93, 108–113]
Pyloric atresia	Surgical resection	[114, 115]
Gastric volvulus	Lap gastropexy	[116]
Acquired gastric outlet obstructions		
Peptic ulcer disease (gastric or pyloroduodenal)	Medical TTT	[93, 117]
Brunner's glands hyperplasia	Lap pyloroplasty + medical	[118]
Eosinophilic gastritis	Surgical resection	[119]
Drug induced (ibuprofen)	Endoscopic pneumatic dilatation	[120]
Foreign bodies	Endoscopy	[121]

gastropyloric tumors in children is mainly limited to case studies. Gastrointestinal stromal tumor (GIST) [106], Burkitt's lymphoma, gastroblastoma [103], adenomyoma [104], and plasma cell granuloma [97] have been reported.

Prepyloric webs are unusual mucosal partial or total diaphragms that may cause GOO. Histologically, the web consists in normal mucosa and submucosa. It appears in the early infancy in most cases, but it has been reported in older children and even in adults. The treatments are either endoscopic or surgical resection [110–113].

Acute gastric volvulus in newborns and infants is known as a rare but life-threatening emergency that requires prompt recognition and treatment. The first description of this condition was made in 1866 by Berti based on the autopsy of a 61-year-old woman. Oltmann described the first pediatric patient in 1899. To date, more than 250 gastric volvulus in children have been described [116]. Gastric volvulus can be defined as torsion of more than 180° of the stomach around itself thus occluding the pylorus and inducing intermittent or persistent vomiting. The diagnosis is done by upper gastrointestinal contrast studies. The radiological signs include horizontalness of the stomach, the greater curvature being above the lesser one and crossing in front of the lower esophagus with the pylorus looking downward. Once recognized the surgical procedure is an anterior gastropexy with reinforcement of the esophagogastric angle performed by laparoscopy, without antireflux-associated procedure [116].

In the acquired conditions, children are older than the former one, i.e., after 1 year of age. Albeit unusual, peptic ulcers can occur in children and according to their sites may occlude the pylorus. Prior to proton pump inhibitors (PPI) and H2 blockers, peptic ulcer disease secondary to *Helicobacter pylori* was a more common cause of GOO than today. *Helicobacter pylori* are evidenced by urease test and medically treated. However, even at the era of PPI, persistent ulcer under adequate treatment can require for surgery [117].

The ingestion of foreign bodies is a common problem in infants, but fortunately the majority of them will pass through the digestive tract without any adverse effects. The peak incidence of foreign body ingestion is between 6 months and 3 years and coins are the most common. It has even been described in neonates (esophageal zipper in a 2 months old baby) [121, see also Chap. 16]. Most ingested foreign bodies remain entrapped in the esophagus at the level of its anatomic narrowing. However, some of them can be trapped in the antrum occluding the pylorus. There are no guidelines available to determine which type of object will pass safely. The size depends on the age of the child. The eventuality of foreign body impaction must always be considered in infants below 5 years of age and searched for.

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