Laparoscopic Pyloromyotomy

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26.1 Introduction

Little controversy still exists to establish the most appropriate treatment of hypertrophic pyloric stenosis (HPS) in neonates and infants. The nonoperative treatment with oral or intravenous atropine has low acceptance due to the overall success rate of 75–79%, the long-term therapy, and the collateral effects [1, 2]. Surgical treatment described in 1912 by Dr. Conrad Ramstedt remains the suitable standard management option due to the higher success rate (~100%), minimal complications, and shorter hospital stay [1, 3]. In recent years, pediatric laparoscopy and other minimal invasive techniques have found a place in the surgical therapy for HPS offering excellent results but creating some controversy about the benefits when comparing to the open approach [4–6].

HPS is the main cause of gastric outlet obstruction and one of the common pathologies requiring abdominal surgery within the first 2 months of life. It is characterized by hypertrophy of the circular muscle layer of the pylorus with a consequent narrowing and elongation of the pyloric channel. The exact etiology is unknown, but several studies have proposed a relation with genetic factors [8], maternal smoking during pregnancy, being first born, preterm delivery, small weight for gestational age, cesarean section [9], young maternal age [10], and exposure of erythromycin in the neonatal period [11, 12] . Positive family history has been reported in 17% with one family member and 3% in two or more family members [13]. The overall incidence in European countries is 2.0 per 1000 live births [10] and occurs with a fivefold male predominance [9]. The common onset of clinical features such as non-biliary progressive projectile vomiting, observing the gastric peristaltic waves, and palpation of the thickened (olive-shaped) pylorus occur between the third and the sixth week of age. Late-onset presentation has been described [14, 15]. Delay in the diagnosis and treatment causes dehydration, important electrolyte abnormalities such as hypokalemic/hypochloremic metabolic alkalosis, and in some cases jaundice and esophagitis [13].

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C. Esposito et al. (eds.), *ESPES Manual of Pediatric Minimally Invasive Surgery*,
https://doi.org/10.1007/978-3-030-00964-9_26

26.2 Diagnosis

A correct anamnesis and clinical examination allow to establish the diagnosis of HPS in the 75–80% of the cases, but in recent years, this is becoming a lost skill [13, 15].

Haller and Cohen in 1986 reported a pyloric diameter ≥15 mm, a pyloric wall muscle thickness ≥4 mm, and a pyloric channel length ≥18 mm as reliable ultrasound measurements to establish the diagnosis of HPS. Since then, ultrasound has gained popularity as a value tool in the diagnosis of HPS due to the excellent specificity and sensitivity and the ease of obtaining the noninvasive study. Forster et al. reported a sensitivity and specificity of pyloric muscle wall thickness of 91% and 85%, respectively, and sensitivity and specificity of pyloric muscle length of 76% and 85%, respectively [11]. Other authors have reported similar results modifying the US measurements of muscle wall thickness >3 mm, a pyloric diameter >10 mm, and a pyloric channel length >15 mm [12].

Upper GI series may be also helpful in the diagnosis of HPS and could exclude other pathologies. The characteristic features in HPS are delayed gastric emptying, the "string" sign (a single long central streak of contrast filling the pyloric channel), an up-turned pyloric curve, the "beak" sign (as contrast enters the proximal pyloric channel it forms a beak), and indentation of the base of the duodenal bulb by the pyloric muscle mass [14].

26.3 Preoperative Care

Before surgery, a complete stabilization and correction of the dehydration and electrolyte imbalance is mandatory to avoid complications with anesthesia and during the immediate postoperative period [2]. There is wide variance of protocols of fluid and electrolyte replacement in HPS, but typical regime includes correction with a solution containing 0.45% NaCl and 5 or 10% dextrose with KCl added at 10 mmol/500 ml. Controversy still exists regarding the benefits of nasogastric tubes for continuous decompression of the stomach preoperatively, the use of prophylactic antibiotics, and the use of antacids [9].

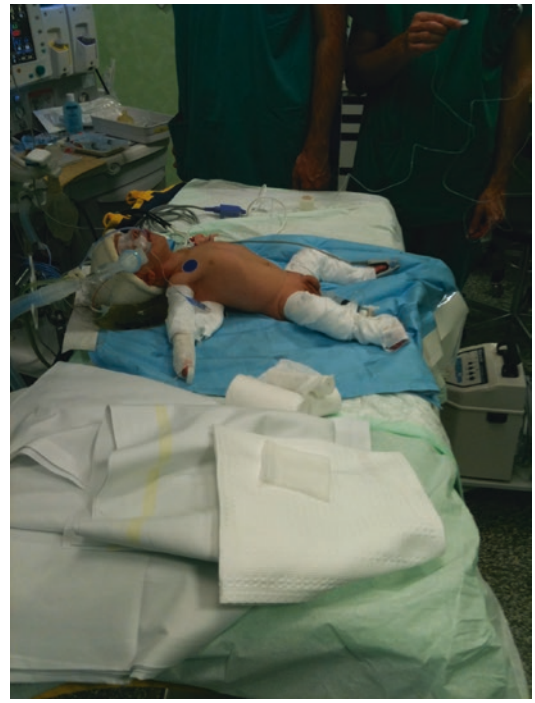


Fig. 26.1 Position

Small babies must be kept warm either with increase of the temperature in the operating room as well as with controlled temperature devices. For the laparoscopic approach, the small patient could be placed transversally in the operating table to facilitate the laparoscopic setting (Fig. 26.1). It is advised to place a nasogastric tube right before surgery to empty and to deflate the stomach and also could be used to exclude perforation of the mucosa during the pyloromyotomy [4].

26.4 Surgical Technique

Extramucosal pyloromyotomy described by Dr. Conrad Ramstedt in 1921 is the gold standard for the surgical treatment of HPS [4, 5]. Due to the higher success rate (~100%), shorter hospital stay, minimal complications, and nearly 100% survival, today, the surgical treatment for HPS

is preferred in comparison with the conservative medical treatment. Moreover, in this new era with the concept of minimal invasive surgery, the classic open approach (right transverse supraumbilical minilaparotomy) to perform a Ramstedt's extramucosal pyloromyotomy (REP) has been losing popularity.

Patient positioning and laparoscopic setup are shown in Figs. 26.1 and 26.2. In our experience, the transverse position of the patient in the operating table allows better instrumentation settings and better ergonomic position to operate.

After disinfection and pose of sterile drapes, a small (~5 mm) curved upper or infraumbilical rim incision is performed. Incision of the aponeurosis and the peritoneum is performed. A U-shaped stitch of a 3-0 reabsorbed suture is placed in the aponeurosis. This suture will be use to fix the umbilical trocar to avoid displacement during surgery and to close the aponeurosis at the end of the procedure. A 5 mm trocar is placed in the umbilicus, and a 6–8 mmHg CO_2 pneumoperitoneum is applied. Special attention is recommended to purge very well the tube of the insufflator with CO_2 before to attach it to the trocar; this is performed to eliminate the room air in the system. Through this trocar, a 0º or 30º 5 mm telescope is inserted. The most common position of the other two trocars of 3 mm for instrumentation is one at the epigastrium and the other in the lateral part of the right hypochondrium (Fig. 26.3). With a 3-mm laparoscopic Babcock clamp, inserted in the right hypochondrium trocar, the hypertrophied pylorus is fixed and exposed. Through the epigastric trocar, a 3-mm laparoscopic retractable scalpel is inserted to perform the pyloromyotomy. The use of a laparoscopic electrocautery monopolar hook to perform the seromuscular pyloric incision has been also reported [6]. An incision is made over the anterosuperior part of the pylorus, beginning at the demarcated pyloroduodenal junction about 2 mm proximal to the pyloric vein and extending the incision onto the gastric antrum. Either with a special 3-mm laparoscopic pyloric spreader (Fig. 26.4) or with an atraumatic grasper, the pyloric muscle fibers are

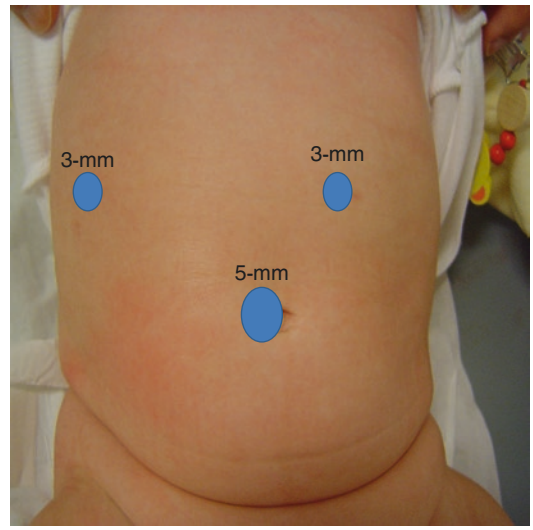
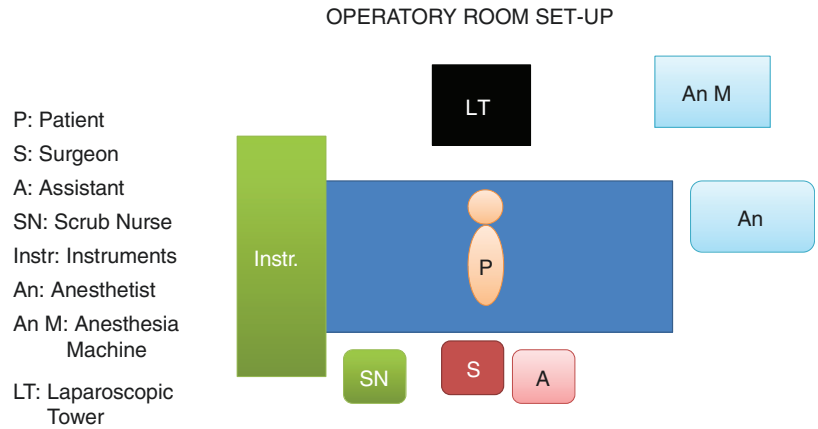


Fig. 26.3 Trocar position

Fig. 26.2 Setup



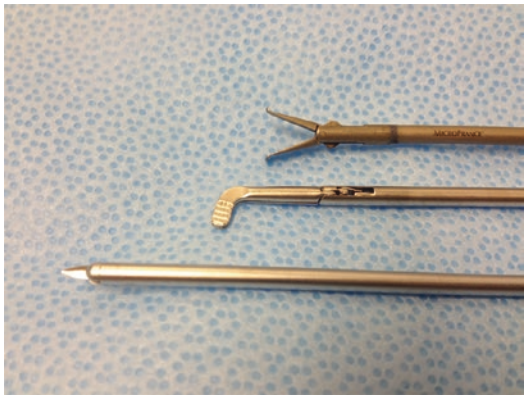


Fig. 26.4 Laparoscopic instruments for pyloromyotomy

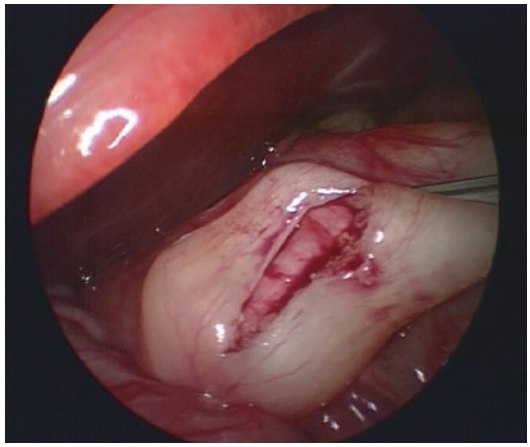


Fig. 26.5 Laparoscopic pyloromyotomy

then widely split until the mucosa is visible and bulging (Fig. 26.5). Hemostasis is assured. A perforation test could be performed dropping saline with a 3-mm laparoscopic irrigation system and insufflation of the stomach with air through a nasogastric tube, searching for bubbles. Although the absence of bubbles suggests a low suspicion of mucosal impairment, this technique does not exclude completely the possibility of perforation [2]. The pneumoperitoneum is evacuated, and the incisions of the trocars are closed with interrupted stitches of 4-0 absorbable sutures. The skin could be closed with rapid absorbable sutures or with glue.

26.5 Postoperative Care

Maintenance of intravenous fluids is continued until the patient is feeding satisfactorily. Most patients start oral feeds 4–6 h after surgery. Although several protocols to increase the volume and concentration of the meals for the reintroduction of oral feeds exist, some institutions advocate feeding *ad libitum* with excellent results [9]. Table 26.1 shows a common protocol of oral feeding. Most patients tolerate full feedings 24–48 h after surgery and could be discharged from the hospital [3]. Postoperative esthetics of the scars is very good (Fig. 26.6).

26.6 Complications

Randomized controlled trials comparing outcomes after open and laparoscopic pyloromyotomy have been published [6, 7]. These studies concluded that there were no statistically significant differences in complication rates.

Table 26.1 Feeding protocol 4–6 h after pyloromyotomy

Substance	Quantity	Time of interval	Times of administration
Pedialyte or water with 10% glucose	30 ml	Every 3 h	2
Half-strength formula or complete breast milk	30 ml	Every 3 h	1
Full-strength formula or breast milk	30 ml	Every 3 h	1
Full-strength formula or breast milk	60 ml	Every 3 h	2
Full-strength formula or breast milk	90 ml	Every 3 h	2
Full-strength formula or breast milk	*Ad libitum*	Every 3 h	

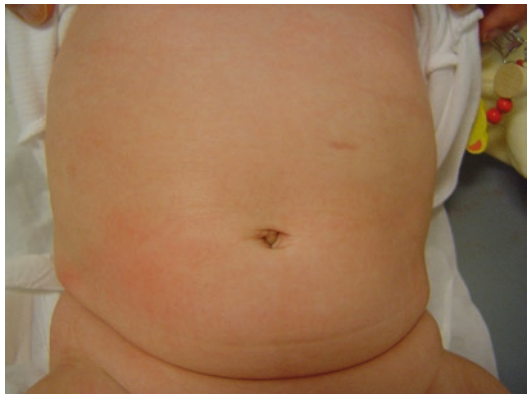


Fig. 26.6 Postoperative results of the scars

The rate of complications during or after pyloromyotomy is very low and generally related to the inexperience of the surgeon during the learning curve. Common major complications include mucosal perforation or incomplete pyloromyotomy. In both cases, the treatment of these complications should be performed with or by an experienced surgeon and could be performed by open or laparoscopic approach. Mucosal perforation occurs mainly secondary to excessive separation of the muscular fibers in the duodenal side of the pylorus; in this case, the perforation is closed with an absorbable suture, and a patch of omentum is applied over the suture. A new pyloromyotomy in the posterosuperior face of the pylorus may be performed. Incomplete pyloromyotomy is mainly secondary to a short incision or incomplete separation of the muscle fibers of the gastric side of the pylorus. In this case, a new intervention is necessary to complete the pyloromyotomy.

Other severe and very rare complications such as air or carbon dioxide embolism during laparoscopic pyloromyotomy have been reported [3].

26.7 Discussion

Diagnosis of HPS has made important changes since the introduction of ultrasound. Prompt diagnosis and stabilization of the dehydration and electrolyte imbalance allow a sooner surgical correction of the gastric outlet obstruction with a consequent better outcome. Regarding the surgical procedure, Ramstedt extramucosal pyloromyotomy is considered still the gold standard.

The randomized controlled trials existing in the literature, comparing the open and the laparoscopic pyloromyotomy, concluded that both approaches are equally safe and reproducible in experienced hands. From the cosmetic point of view, no real benefits exist between the transumbilical open approach and the laparoscopic approach. The surgeon must choose the best approach in base of his surgical experience and skills and in the resources of the institution where he works. Moreover, a close supervision by an experienced pediatric surgeon faculty is mandatory while training residents during an open or a laparoscopic pyloromyotomy to decrease the risk of major complications. Further randomized controlled trials regarding new techniques such as the needlescopic approach [6], single-incision approach [7], and the new endoscopic intraluminal pyloromyotomy [3] are necessary to evaluate their real benefits, complications, and contraindications in this domain.

Publications in the medical literature evaluating the long-term follow-up after pyloromyotomy are scanty. The outcome of the pyloric hypertrophy after a pyloromyotomy was studied in 103 infants by Muramori et al. [9]. They performed serial ultrasonographic measurements regarding channel length, muscle thickness, and diameter of the pylorus for a period of 1 year after surgery. In contrast to the prompt improvement of clinical symptoms, they found that the length of the pyloric channel reached a normal length (~12.7 ± 2.8 mm) around 4 months after surgery, the muscle thickness reached a normal range (~2 mm) until 8 months after surgery, and the pyloric diameter did not reach a normal diameter (~10–12 mm) even by the end of 1 year after surgery.

Walker et al. [3] analyzed the neurological development of infants operated for HPS and

compare them with healthy control infants at 1 year of age. They found that the cognitive, receptive language and motor score were significantly lower in HPS infants than in controls. Other authors have reported chronic abdominal pain probably secondary to irritable bowel syndrome, functional dyspepsia, and functional abdominal pain in children operated of HPS in a mean follow-up period of 7 years when compared to healthy control children [4]. These findings raised concerns over the potential impact of HPS and its surgical treatment. Further studies are necessary to elucidate these results.

In conclusion, laparoscopic pyloromyotomy is safe and feasible and offers excellent postoperative results in neonates and infants with congenital pyloric stenosis. During the learning curve process of surgeons in training, a correct supervision by experienced laparoscopic surgeons is mandatory in order to decrease the risk of complications. Further studies regarding new techniques such as the needlescopic approach, the single-incision approach, and the endoscopic intraluminal pyloromyotomy are necessary to evaluate their real benefits, complications, and contraindications in this domain.

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