Perioperative complications of surgery for hypertrophic pyloric stenosis

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Abstract

Pyloromyotomy is the tried and tested surgical procedure for successful operative treatment of pyloric stenosis. Over time the operative approach has evolved to take advantage of cosmetically superior incisions and more recently minimally invasive surgery. During and following surgery, complications are uncommon. The specific complications of an inadequate pyloromyotomy requiring repeated procedure and mucosal perforation during an over-zealous pyloromyotomy represent the ends of a spectrum within which sits the perfect procedure. Here we discuss these specific complications together with the other potential complications following surgery for HPS, including anaesthetic considerations.

Introduction

The surgical technique of pyloromyotomy in which the hypertrophied pyloric muscle is divided thereby permitting gastric emptying was established soon after the beginning of the 20th century [1]. Conrad Rammstedt modified the 'submucosal pyloroplasty' described by Dufour and Fredet in 1907 [2] (although attributed by Ramstedt to Weber in 1911) with the omission of transverse muscle closure. Ramstedt serendipitously realised this step was unnecessary after his first pyloromyotomy, where he was forced to simply apply omentum to the exposed mucosa as the stitches kept cutting out of the muscle.

For seven decades surgeons performed pyloromyotomy via midline or right upper quadrant transverse incision until the open approach was refined by the introduction of the Bianchi approach via a circumumbilical skin crease incision to give the subsequent appearance of a scarless abdomen [3]. Adoption of the laparoscopic technique, first described by Alain et al. in 1991 [4], has added another dimension to the well-documented potential sequelae of an open pyloromyotomy.

In recent years several prospective randomised controlled trials (RCTs) comparing the laparoscopic approach against the standard open operation have been completed, allowing contemporary estimation of complication rates from prospectively collected data [5-7].

Overall rates of perioperative complications are obtainable from large, population-based reports. A study analysing readmission post-pyloromyotomy in 1900 infants utilising a patient discharge database in California between 1995 and 2010 demonstrated an in-hospital complication rate of 5.16%, rising to 6.84% for overall complications [8]. At 30 days postoperatively the all-cause readmission rate was 4.01%, of which surgical complications accounted for 36% (27/76) within the first 30 days. Beyond this, respiratory infections were predominantly responsible for readmission.

Interestingly, the largest retrospective study incorporating 11,003 infants drawn from the US National Inpatient Samples database (including data from hospitals across 28 states) revealed a lower complication rate of just 2.71% [9]. The most frequent of these (48%) were intra-operative events, comprising 'accidental puncture or laceration complicating surgery' and haemorrhage. Though there was the suggestion of increased complications in younger infants (<29 days, 3.2% vs. >39 days, 2.1%) this failed to reach statistical significance.

One death occurred during the study period, but there were 19 cardiac arrests, underscoring the key potential contribution of anaesthesia to patient morbidity.

Wound complications

The incidence of wound-healing complications appears to be higher than that encountered with other abdominal wounds within the pediatric population. This observation may have a multifactorial basis including a relatively immature immune system and poor nutritional condition due to the variable period of pre-operative malnourishment.

Infection

Debate persists regarding the indication for prophylactic antibiotics for patients undergoing pyloromyotomy, an ostensibly clean operation.

A retrospective review of 194 patients who underwent laparoscopic pyloromyotomy over a 7 year period (2002 - 2009) looked at the potential benefit of prophylactic antibiotics for this procedure [10]. As individual surgeons within the centre differed on whether antibiotics were indicated, this resulted in two separate groups that did not significantly differ in age, sex, weight or serum bicarbonate on admission or at operation.

Three wound infections occurred in each group - 2.7% (3 of 111) of those that received antibiotic prophylaxis in comparison with 3.5% (3 of 84) that did not - a non-significant difference ($p = 0.73$). They therefore concluded that prophylactic antibiotics do not significantly reduce the rate of wound infection.

Intriguingly, the institution of prophylactic antibiotics after 1982 in a single-centre 35-year series of 791 infants with pyloric stenosis, coincided with a reduction in wound infection rates from 9 to 3.9% [11]. In this series, the skin commensal *Staphylococcus epidermidis* accounted for 77% of wound infections.

Several authors have attempted to determine whether the operative approach influences wound infection rates. A prospective RCT of the laparoscopic versus open approaches including 200 infants found 4 wound infections in the open group in comparison with 2 in the laparoscopic group ($p =0.68$) [5]. By contrast, a systematic review and meta-analysis in 2009 comparing the two approaches found a significantly lower wound complication rate in the laparoscopic group with an odds ratio of 0.42 (0.2, 0.91), $p = 0.03$ [12].

There is conflicting evidence regarding the risk of wound infection relating to the nature of the open incision selected. Some authors have found a two to threefold increased wound infection rate with the original right upper quadrant incision [11, 13], whilst others noted no difference between the various incisions [14].

Dehiscence

One wound dehiscence occurred in the open group with none in the laparoscopic group during a prospective RCT including 98 patents [6]. A similar rate of developing a 'wound problem requiring re-operation' was observed in 7 (1.6%) from a series of 430 infants treated with either the open or laparoscopic approach at the Royal Hospital for Sick Children Edinburgh between 1999 and 2012 [15]. This was also reflected in a 35-year series from the Hospital for Sick Children, Toronto, where just one of 791 infants developed a wound dehiscence [12].

Incisional hernia

Over a six year period at a single centre, incisional hernia repair was performed in 6 of a total of 255 children who had initially undergone open (4) or laparoscopic (2) pyloromyotomy [16], reflecting an incisional hernia rate of 2.52%.

A slightly lower rate of 1% was observed when combining outcomes from both a pediatric and regional hospital, representing 4 incisional hernias in total - two after right upper quadrant incisions and two following umbilical incisions [14]. A preceding wound infection was only noted in one of these cases.

The occurrence of wound complications - whether dehiscence or incisional hernia - as delineated in the preceding large series, does appear to be higher than might be othewise anticipated.

Incomplete pyloromyotomy

Inadequate division of the hypertrophied pyloric muscle is a recognised specific complication of the procedure. Sufficient pyloromyotomy is commonly assessed at open operation using the 'shoe-shine' manouevre, to confirm that each side of the divided muscle moves freely and independently.

The advent of laparoscopic pyloromyotomy has naturally drawn comparison with the standard open technique, particularly with regard to the risk of incomplete pyloromyotomy, putatively asserted to relate to the absence of direct intraoperative tactile feedback.

A recent meta-analysis revealed a 4% higher rate (risk difference 0.04, 95% confidence interval 0 - 0.08, $p = 0.03$) of incomplete pyloromyotomy with the laparoscopic technique as opposed to the open operation [17].

A large retrospective multicentre study in nine high-volume institutions across the UK and North America analysed a total of 2830 procedures – 1802 of which were performed laparoscopically [18]. Incomplete pyloromyotomy occurred in 3 (0.29%) of the open group and 21 (1.19%) of the laparoscopic group. Binomial logistic regression analysis determined that these figures reflected a significantly higher incidence of incomplete pyloromyotomy following laparoscopic compared with open surgery of 0.87% ($p = 0.046$) [18]. The number needed to treat with laparoscopic pyloromyotomy to encounter a further episode of incomplete pyloromyotomy is 115, highlighting the potentially limited clinical relevance of this small, albeit statistically significant difference between the two approaches.

The group at Mercy Children's Hospital, Kansas, have demonstrated that by creating a pyloromyotomy of approximately 2 cm in length, incomplete laparoscopic pyloromyotomy can be avoided entirely [19]. Alternative strategies to ensure adequacy of muscle division at open operation include passage of a 14 Fr catheter through the pylorus into the duodenum [20], or removal of a section of pyloric muscle from the myotomy edge [21].

The detection of incomplete pyloromyotomy – suspected clinically on the basis of persistent postoperative vomiting - is invariably followed by a return to theatre. An upper gastrointestinal contrast study can be useful in confirming this clinical suspicion prior to re-operation [21]. An alternative non-operative approach using antimuscarinic agents has also been described [22]. This follows the same logic applied for the exclusive medical management of HPS as widely implemented in Japan [23]. Endoscopy-guided balloon dilatation (EGBD) has also been successfully performed for incomplete pyloromyotomy, thereby avoiding re-operation altogether [21, 24].

Mucosal perforation

Conversely, an overzealous pyloromyotomy - perhaps with the intention of avoiding the above outcome - can also lead to full-thickness division of the pylorus with perforation.

An unrecognised perforation at the gastric end of a pyloromyotomy has been responsible for the subsequent development of a tension pneumoperitoneum [25]. This initially developed on postoperative day 4, and although the situation was temporised with needle decompression, the pneumoperitoneum reaccumulated within 24 hours prompting surgical exploration and identification of the gastric perforation. More commonly, if unnoticed at the time of operation this can present with symptoms and signs of sepsis and peritonitis, drawing attention to the recent operative procedure.

A retrospective single-centre review over a 21-year period demonstrated a 1.67% incidence (15 of 896) of mucosal perforation [26]. The only difference identified between those who did and did not develop this complication was the age at surgery – 48 days compared with 34 days – which was revealed to be statistically significant ($p = 0.0021$). This figure is similar to the 1.1% rate of mucosal perforation seen within the retrospective study of 11,003 infants across 28 states in the US [9].

A meta-analysis including three RCTs found no significant difference in mucosal perforations between laparoscopic and open groups (odds ratio 0.96, 95% confidence interval: 0.22 - 4.26) [27]. With reference to the large retrospective multicentre study across nine institutions previously described, mucosal perforation was noted in 3 (0.29%) of the open group compared with 15 (0.83%) that underwent the laparoscopic procedure, a difference that did not reach statistical significance.

Surgical strategies for repairing mucosal perforation either involve primary mucosal repair or full-thickness closure, pyloric rotation and repyloromyotomy, as performed by 70% and 27% respectively, of International Pediatric Endosurgery Group (IPEG) survey respondents [28]. Interestingly, 93% of these injuries were identified intraoperatively, and 84% of this patient group were subject to a delay in feeding with or without prior contrast study.

Anaesthetic considerations

Apnoea

It is well known that HPS is not a surgical emergency, rather it is the resulting electrolyte imbalance and metabolic derangement that requires correction before surgery is contemplated. Inadequate attention to this essential component of care can result in both pre- and postoperative apnoea.

The potentially profound metabolic alkalosis is considered responsible for this complication. Within the neonatal and infant population, $PaCO_2$ represents the primary stimulus for ventilation, determining the pH of the cerebrospinal fluid to which the central chemoreceptors are exposed [29].

This appears to largely be of historical significance, in part because the widespread use of ultrasound (US) enables diagnosis earlier in the clinical course prior to the development of profound dehydration and electrolyte abnormalities. Apnoea occurred in a total of three infants from a single-centre series of 791 - preoperatively in one, postoperatively in two - with resulting cardiac arrest and subsequent death on postoperative day 2 in one patient in 1974 [11].

A range of values have been suggested for acceptable chloride levels prior to operating, with one centre's anaesthetic team suggesting a minimum of 106 meq/L [30], whilst another team set the threshold at a

serum chloride of > 100 mEq/l in association with adequate urine output to indicate that sufficient intravascular resuscitation has been achieved [29].

Aspiration risk

Although preoperatively infants are typically managed with a nasogastric tube which is regularly suctioned, the stomach may surprisingly not be satisfactorily empty prior to induction of anaesthesia. Following passage of a wider-bore orogastric catheter after anaesthetic induction, it was shown that the residual volume of gastric fluid was 4.8 +/- 4.3 ml/kg [31], highlighting the need to perform such a manouevre before induction to minimise the risk of aspiration of gastric content.

Recurrence

Although vanishingly rare, recurrence of HPS following a satisfactory pyloromyotomy has been described in the literature [32, 33]. This complication was detected in 2 infants over a 30 year period at a single centre (1973 - 2003) [32]. Both had developed non-bilious projectile vomiting at 1 month postoperatively, and following an unsuccessful trial of anti-reflux and prokinetic medications were revealed 2 weeks later to have significant gastric outlet obstruction on contrast studies. Balloon dilatation was initially performed in both cases, however was successful in only one, necessitating re-pyloromyotomy.

Postoperative vomiting

The persistence of non-bilious vomiting following the definitive procedure is a frequent postoperative hurdle. This manifested in 46% and 57% of infants managed with a relaxed or incremental feeding regime, respectively, in a recent RCT investigating which approach is more conducive to achieving feeding goals postoperatively [34]. Several mechanisms have been described for this phenomenon, including gastroparesis, pylorospasm and pyloric oedema.

Within the pyloric stenosis population, it has been revealed that two factors are highly predictive of more severe postoperative vomiting - the degree of preoperative metabolic and electrolyte derangement and a lower weight on admission [35]. These are the infants with a more prolonged duration of gastric outlet obstruction. A variety of feeding regimens have been studied in order to ameliorate postoperative vomiting, with the aim of thereby reaching feeding goals more quickly and reducing hospital length of stay.

Some advocate unstructured ad libitum feeding, as vomiting invariably occurs regardless of the feeding plan and this represents a potentially more straightforward and effective approach. A retrospective single-centre review found no difference in frequency of postoperative vomiting between infants fed ad libitum or using a standardised feeding regime, although full feeds were achieved in a significantly shorter time (19.0 vs. 23.1 hours, $p < 0.01$) with ad libitum feeding [36]. In an RCT comparing 'relaxed' i.e. ad libitum feeding, with incremental feeding, infants randomised to the former approach reached goal feeds significantly more quickly (18.9 +/- 11.4 hours versus 27.0 +/- 10.3 hours, $p < 0.001$) and also had a significantly shorter length of stay (44.3 +/- 19.2 hours versus 53.6 +/- 27.8 hours, $p = 0.0023$) [34].

Similar results in terms of length of stay were found in a recent meta-analysis of the above feeding regimens, although gradual feeding (structured approach) did bring a significant reduction in episodes of vomiting (mean difference -1.7; 95% confidence interval -2.17 to -1.23, $p < 0.00001$) [37].

Although there were no differences in time to full feeding between the laparoscopic and open groups when analysed in a 2006 single-centre RCT, the laparoscopic group did develop significantly fewer episodes of postoperative vomiting - expressed as mean +/- standard error: 1.85 episodes +/- 0.15 versus 2.61 episodes +/- 0.32 [5]. A more recent double-blind multicentre RCT found that postoperative vomiting was similar between these two groups [38].

Misdiagnosis

Seldom described, incorrect diagnosis remains a rare 'intraoperative' complication in the care of infants with suspected HPS. Three such patients were encountered in a 35 year single-centre series, where the diagnosis was thought to be confirmed through a combination of clinical examination and imaging (US, upper gastrointestinal contrast study). Two had no other identifiable abnormality at operation, one had a duodenal duplication causing obstruction just distal to the pylorus [11].

A 1.1% (4 of 343) negative exploration rate has also been reported, 'positive' ultrasound scans were obtained in three [39]. The remaining infant had a negative ultrasound but a positive upper GI contrast study prompting surgery. The authors observed that a 'confident diagnosis' rested on a positive clinical examination and local US in an alkalotic infant, with the addition of on-table examination if 'awake' examination had been negative.

Conclusion

Overall, perioperative complications of surgery for HPS can be overwhelmingly considered to represent the exception rather than the rule. Regardless of operative approach, the key complications of incomplete pyloromyotomy and mucosal perforation occur rarely, with wound-related complications also uncommon. Pre- or postoperative apnoea with close attention to detail is preventable and this complication appears to have been consigned to history. Excellent outcomes can clearly be achieved with meticulous attention to pre-operative preparation and intra-operative surgical technique.

References

1. Rammstedt C. Zur Operation der Angeborenen Pylorusstenose. Med Klin 1912; undefined: pp. 1702-1706
2. Dufour H, Fredet P. La Stenose Hypertrophique Du Pylore chez le Nourrisson et son Traitement Chirurgical. Bull Mem Soc Med Paris 1907;24: pp. 1221
3. Tan KC, Bianchi A. Circumumbilical incision for pyloromyotomy. Br J Surg 1986;73(5):399
4. Alain JL, Grousseau D, Terrier G. Extramucosal pyloromyotomy by laparoscopy. Surg Endosc 1991; 5(4):174-5

5. St Peter SD, Holcomb GW 3rd, Calkins CM, et al. Open versus laparoscopic pyloromyotomy for pyloric stenosis: a prospective, randomized trial. Ann Surg 2006;244(3):363–70.

6. Siddiqui S, Heidel RE, Angel CA, et al. Pyloromyotomy: randomized control trial of laparoscopic vs open technique. J Pediatr Surg 2012;47(1):93-8

7. Leclair MD, Plattner V, Mirallie E, et al. Laparoscopic pyloromyotomy for hypertrophic pyloric stenosis: a prospective, randomized controlled trial. J Pediatr Surg 2007;42(4):692-8

8. Marks IH, Chang DC, Masiakos PT, et al. Is all-cause readmission an appropriate performance measure for pediatric surgeons? A case study in pyloromyotomy. J Pediatr Surg 2017;52(9):1426-29

9. Safford SD, Pietrobon R, Safford KM, et al. A study of 11,003 patients with hypertrophic pyloric stenosis and the association between surgeon and hospital volume and outcomes. J Pediatr Surg 2005;40(6):967-72

10. Katz MS, Schwartz MZ, Moront ML, et al. Prophylactic antibiotics do not decrease the incidence of wound infections after laparoscopic pyloromyotomy. J Pediatr Surg 2011;46(6):1086-88

11. Ein SH, Masiakos PT, Ein A. The ins and outs of pyloromyotomy: what we have learned in 35 years. Pediatr Surg Int 2014;30(5):467-80

12. Sola JE, Neville HL. Laparoscopic vs open pyloromyotomy: a systematic review and meta-analysis. J Pediatr Surg 2009;44(8):1631-7

13. van den Ende ED, Allema JH, Hazebroek FW, et al. Can pyloromyotomy for infantile hypertrophic pyloric stenosis be performed in any hospital? Results from two teaching hospitals. Eur J Pediatr 2007;166(6):553-7

14. Podevin G, Missirlu A, Branchereau S, et al. Umbilical incision for pyloromyotomy. Eur J Pediatr Surg 1997;7(1):8-10

15. Keys C, Johnson C, Teague W, et al. One hundred years of pyloric stenosis in the Royal Hospital for Sick Children Edinburgh. J Pediatr Surg 2015;50(2):280-4

16. Mullassery D, Pedersen A, Robb A, et al. Incisional hernia in pediatric surgery - experience at a single UK tertiary centre. J Pediatr Surg 2016;51(11):1791-94

17. Sathya C, Wayne C, Gotsch A, et al. Laparoscopic versus open pyloromyotomy in infants: a systematic review and meta-analysis. Pediatr Surg Int 2017;33(3):325-333

18. Hall NJ, Eaton S, Seims A, et al. Risk of incomplete pyloromyotomy and mucosal perforation in open and laparoscopic pyloromotomy. J Pediatr Surg 2014;49(7):1083-6

19. Ostlie DJ, Woodall CE, Wade KR, et al. An effective pyloromyotomy length in infants undergoing laparoscopic pyloromyotomy. Surgery 2004;136(4):827-32

20. Shaw RB. Simple technique for assuring completeness of a pyloromyotomy. J Am Coll Surg 1994;178(3):303-4

21. Khoshoo V, Noel RA, LaGarde D, et al. Endoscopic balloon dilatation of failed pyloromyotomy in young infants. J Pediatr Gastroenterol Nutr 1996;23(4):447-51

22. Owen RP, Almond SL, Humphrey GM. Atrophine sulphate: rescue therapy for pyloric stenosis. BMJ Case Rep 2012;Aug 2

23. Kawahara H, Takama Y, Yoshida H, et al. Medical treatment of infantile hypertrophic pyloric stenosis: should we always slice the “olive”? J Pediatr Surg 2005;40(12):1848-51

24. Tam PKH, Carty H. Endoscopy-guided balloon dilatation for infantile hypertrophic pyloric stenosis. Pediatr Surg Int 1991;6(4-5):306-308

25. Eke N. Postoperative tension pneumoperitoneum in an infant. Pediatr Surg Int 2001;17(2-3):204-5

26. Royal RE, Linz DN, Gruppo DL, et al. Repair of mucosal perforation during pyloromyotomy: surgeon’s choice. J Pediatr Surg 1995;30(10):1430-2

27. Jia WQ, Tian JH, Yang KH, et al. Open versus laparoscopic pyloromyotomy for pyloric stenosis: a meta-analysis of randomized controlled trials. Eur J Pediatr Surg 2011;21(2):77-81

28. Waldron LS, St Peter SD, Muensterer OJ. Management and Outcome of Mucosal Injury During Pyloromyotomy – An Analytical Survey Study. J Laparoendosc Adv Surg Tech A 2015;25(12):1044-6

29. Kamata M, Cartabuke RS, Tobias JD. Perioperative care of infants with pyloric stenosis. Paediatr Anaesth 2015;25(12):1193-206

30. Bissonnette B, Sullivan PJ. Pyloric stenosis. Can J Anaesth 1991;38(5):668-76

31. Cook-Sather SD, Tulloch HV, Liacouras CA, et al. Gastric fluid volume in infants for pyloromyotomy. Can J Anaesth 1997;44(3):278-83

32. Ankermann T, Engler S, Partsch CJ. Repyloromyotomy for recurrent infantile hypertrophic pyloric stenosis after successful first pyloromyotomy. J Pediatr Surg 2002;37(11):E40

33. Nasr A, Ein SH, Connolly B. Recurrent pyloric stenosis: to dilate or operate? A preliminary report. J Pediatr Surg 2008;43(2):e17-20

34. Markel TA, Scott MR, Stokes SM, et al. A randomized trial to assess advancement of enteral feedings following surgery for hypertrophic pyloric stenosis. J Pediatr Surg 2017;52(4):534-539

35. St Peter SD, Tsao K, Sharp SW, et al. Predictors of emesis and time to goal intake after pyloromyotomy: analysis from a prospective trial. J Pediatr Surg 2008;43(11):2038-41

36. Adibe OO, Nichol PF, Lim FY, et al. Ad libitum feeds after laparoscopic pyloromyotomy: a retrospective comparison with a standardized feeding regimen in 227 infants. J Laparoendosc Adv Surg Tech A 2007;17(2):235-7

37. Sullivan KJ, Chan E, Vincent J, et al. Feeding Post-Pyloromyotomy: A Meta-analysis. Pediatrics 2016;137(1)

38. Hall NJ, Pacilli M, Eaton S, et al. Recovery after open versus laparoscopic pyloromyotomy for pyloric stenosis: a double-blind multicentre randomised controlled trial. Lancet. 2009;373(9661):390-8

39. Mullassery D, Mallappa S, Shariff R, et al. Negative exploration for pyloric stenosis - is it preventable? BMC Pediatr. 2008;8:37