Idiopathic Hypertrophic Pyloric Stenosis : Our Experience

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Abstract

Background : A typical infant with idiopathic hypertrophic pyloric stenosis is described as a male child, first in the order of birth with a positive family history. However our experience suggests otherwise which is presented in this report.

Methods : A retrospective analysis of medical records of 8 infants, who were diagnosed to be suffering from idiopathic hypertrophic pyloric stenosis and subjected to surgical treatment, was undertaken.

Results : There were 5 (62.5%) males and 3 (37.5%) females. There was no family history and only one child (12.5%) was first born in the order of birth. One infant was preterm and one case (12.5%) had associated congenital anomaly (single kidney). Definitive diagnosis was established in 6 (75%) babies at admission whereas, other 2 cases (25%) required further evaluation. All the infants were in a state of moderate dehydration and in a varying state of hypochloremic alkalosis. The pH and serum chloride levels ranged from 7.52 to 7.67 and 86-94 mmol/L respectively. All were subjected to traditional Ramstedt's pyloromyotomy after having undergone vigorous correction of fluids and electrolytes for 24-48 hours. Intraoperatively, there was one iatrogenic mucosal perforation, which was closed with an omental patch. Postoperative feeding was initiated 12 hrs after surgery in 6 (75%) babies.

Conclusion : Our series suggests a clinical profile of hypertrophic pyloric stenosis in our subset of patients which is different from what is described in literature.

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Key Words : Idiopathic hypertrophic pyloric stenosis; Pyloromyotomy

Introduction

Idiopathic hypertrophic pyloric stenosis (IHPS) is one of the most common gastrointestinal abnormalities presenting in the first six months of life, affecting up to 1-3 of 1000 births with a male preponderance varying from 2:1 to 5:1 [1,2]. The condition is more prevalent in the western world especially in infants of Caucasian descent and is less common amongst Asians [1]. The aetiology of pyloric stenosis remains unknown; however it is multifactorial with contributions from environmental and familial factors [3].

This paper presents our experience with a series of infants diagnosed to have IHPS. The aim of this paper is to highlight the differences in the disease pattern as seen in our population. Current literature and management protocols have also been briefly reviewed.

Materials and Method

A retrospective analysis of medical records of eight infants who were managed at tertiary care centre between Jan 2001-Dec 2003 was undertaken. The records were analysed for data pertaining to age at onset of symptoms, sex, presenting features, seasonal incidence, feeding habits, family history, birth rank, presence of associated malformations, diagnostic evaluation, pre-operative management, anaesthetic techniques and surgical management. Management of cases was essentially based on Pyloric Stenosis Protocol being

followed at this centre (Table 1). All infants were subjected to general anaesthesia and underwent the traditional Ramstedt's pyloromyotomy.

Results

A total of eight cases were managed at our centre during the period of study. A male preponderance was noted, though three (37.5%) of the eight infants were females. More number of infants presented during winter months. Except for one (12.5%), all our patients were the 2nd or 3rd siblings. The vast majority six (75%) were on top feeds for varied reasons. There was no family history of the disease in any of our patients. A history of prematurity was present in only one of the infant who was born at 34 weeks of gestation and presented at 3 weeks of age. Associated congenital malformation was present in one baby (12.5%) who had a single kidney.

All the cases were symptomatic for a period ranging from 7 to 23 days prior to referral to our centre. Diagnosis of idiopathic hypertrophic pyloric stenosis (IHPS) was established in six (75%) babies at admission to the paediatric surgery centre. However, two (25%) babies required further evaluation for recurrent episodes of vomiting and were subsequently detected to have IHPS. Only one child presented at our centre within three weeks of birth and other cases reported at least after four weeks but within 12 weeks of age. Ultrasonography was confirmatory in all cases. One child needed barium study as well to corroborate the diagnosis. All our patients at admission, were in a state of moderate

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Table 1

Pyloric stenosis protocol

dehydration and in varying state of alkalosis, requiring vigorous correction of fluids and electrolytes before being taken up for surgery. The pH ranged from 7.52 to 7.67. Serum chloride levels were recorded between 86-94 mmol/L. In one infant, the potassium level was unrecordable by the arterial blood gas machine, though the laboratory result was 2.2 mmol/L. However there were no arrhythmias and the ECG was

normal. Other electrolytes were within normal range. Resuscitation took an average of 24-48 hrs before serum chlorides and blood gases were reported as normal.

All infants were subjected to general anaesthesia and there were no adverse anaesthetic event. Intraoperatively, there was one case of iatrogenic mucosal perforation, which was closed with an omental patch. Postoperative feeding was

Fig. 1 : Hypertrophied pylorus delivered out of incision

initiated 12 hrs after surgery in six (75%) babies. One baby had prolonged postoperative vomiting for 48 hrs, which settled slowly on 'drip and suck' therapy.

Discussion

An infant having IHPS has been classically described as a full term, male infant who presents with onset of non-bilious projectile vomiting between 3-6 weeks of age. Non bilious vomiting after feeds, a palpable 'pyloric tumour' in the right hypochondrium, visible peristalsis in the upper abdomen and a positive test feed are considered diagnostic of IHPS [1]. The present study involving eight cases, suggests subtle differences in the clinical picture of IHPS in our setting as compared to what is described in the literature. Typically, the patient is described as first-born of a family, presenting with non-bilious projectile vomiting. However, in our series only one infant was first born and majority (87.5%) were of other birth ranks. Our study also shows a substantially increased incidence in female babies which formed 37.5% of all cases though, it confirms to male preponderance. Positive family history, an important diagnostic clue seen in upto 14% of cases in most series, was not seen in our cases [3]. Other factors such as seasonal incidence, effects of feeding, prematurity and associated malformations were same as in other studies [4].

In our series, delayed diagnosis and protracted vomiting in all cases, resulted in dehydration, hypokalemia, hypochloremia, and primary metabolic alkalosis. It is universally agreed that no newborn should be subjected to the additional hazards of anaesthesia and surgery until stabilised medically. Alkalosis is known to have adverse effects, such as enhancing oxygen affinity of haemoglobin and therefore less unloading of oxygen at the tissue level besides decreasing the ionised calcium levels and increasing the potential for seizures [5]. Satisfactory fluid and electrolyte replacement may take 12 to 72 hours, depending on the patient's condition

Fig. 2 : Pylorus after Ramstedt's myotomy

[6]. Our resuscitation approach (Table 1) included treatment of initial hypovolaemia with bolus of Ringer lactate, followed by correction of deficit by using calculated volumes of normal saline. Then 5% dextrose with one-fifth strength normal saline, was used to provide maintenance fluids. Balanced general anaesthesia along with anti aspiration prophylaxis, is the technique of choice [7]. The importance of early diagnosis and referral needs no emphasis. As noted in our series, delayed reporting to a tertiary level centre resulted in dehydration, dyselektrolytaemia and metabolic alkalosis, requiring aggressive preoperative management. Correct diagnosis is based on a good history, careful examination supported by biochemical and radiological investigations. Ultrasound is becoming the most common diagnostic modality for management. The commonly used criteria for a positive USG study are, a pyloric muscle thickness of >4 mm and a pyloric channel length of > 16mm [2]. One infant who presented with bilious vomiting, underwent a barium contrast study to exclude other causes of upper gastrointestinal obstruction. This baby was detected to have a large stomach, gastric stasis and an elongated pyloric channel with indentation of the antrum. Intra-operatively a diagnosis of IHPS was confirmed.

All patients except for one, were subjected to saline gastric lavage in the preoperative period. Lavage helps in cleansing the stomach by removing the sloughed mucosa. It also enhances the tonicity of stomach and reduces gastric stasis in the post operative period. One baby who did not receive preoperative gastric lavage inadvertently, suffered from repeated episodes of vomiting in the postoperative period.

Non-operative measures such as frequent small feeds, oral or intravenous atropine have been described but are only of historical importance [1]. Extramucosal pyloromyotomy is the only definitive treatment for these infants. Laparoscopic approach has been reported to

be associated with a better postoperative course and avoidance of any skin scar [8]. During surgery, one intraoperative complication of mucosal perforation was encountered, which was managed with primary closure with omental patch. Mucosal perforation has been correlated with age of patients. The incidence is higher in older patients probably due to different tissue characteristics and greater adherence of mucosa to overlying muscularis and less pliable muscle layer [9]. We subscribe to this ideology, as the perforation occurred in the baby, who was 3 months old. An alternative to an omental patch repair is to close the incision and redo the pyloromyotomy at a different site. However, the outcome of both these alternatives has been found to be equally effective [9].

Post operatively, majority of infants can be started on feeds within 6 hrs of surgery [1]. In our study six (75%) babies were started on oral feeds within 12 hrs, one after 48 hrs, owing to vomiting in post operative period and in one case, feeding was withheld till 72 hrs after surgery due to a mucosal perforation. In the post-anaesthesia recovery period, the infant should be carefully observed for signs of respiratory depression and periods of apnoea secondary to effects of metabolic alkalosis, general anaesthesia, and decreased body temperature [10]. Anaesthesia related morbidity rate, once noted to be as high as 3.7%, is on the decline and recent studies have shown no complications after general anaesthesia [11,12]. There were no adverse anaesthetic events noted in our series.

In conclusion, the present study brings out a clinical profile of IHPS in our subset of patients, which is different from what is seen in other parts of the world. These 'atypical features' may delay correct diagnosis and hence should be taken into account while evaluating a child with IHPS to reach an early diagnosis.

Conflicts of Interest

None identified

References

1. Dillon PW, Cilley RE. Idiopathic hypertrophic pyloric stenosis. Ashcraft KW, Murphy JP, Sharp RJ, Sigalet, DL, editors. Paediatric surgery. 3rd ed. Philadelphia: WB Saunders, 2000; 391-405.
2. O'Neill JA, Rowe MI, Grosfeld JL, Fonkalsrud EW, Colan AG, editors. Essentials of Paediatric Surgery. 5th ed. St. Louis: Mosby, 1995.
3. Poon TSC, Zhang AL, Cartmill T, Cass DT. Changing patterns of diagnosis and treatment of infantile hypertrophic pyloric stenosis: A clinical audit of 303 patients. J Pediatr Surg 1996; 31: 1611-5.
4. White JS, Clements WDB, Heggarty P, Sidhu S, Mackie E and Stirling I. Treatment of infantile hypertrophic pyloric stenosis in a district general hospital: a review of 160 cases. J Pediatr Surg 2003; 38: 1333-6.
5. Barash PG, Cullen BF, Stoelting RK. editors. Clinical Anaesthesia, 3rd ed, Philadelphia: Lippincott-Raven, 1997.
6. Berry FA. Physiology and surgery of the infant. In : Berry FA, editor. Anaesthetic management of difficult and routine paediatric patients. New York: Churchill Livingstone, 1990; 162-3.
7. Hall SC. Anaesthesia for neonates. In : Badgwell JM, editor. Clinical Paediatric anaesthesia. Philadelphia: Lippincott-Raven 1997; 189-90.
8. Alain JL, Grousseau D, Longis B, Ugazzi M, Terrier G. Extramucosal pyloromyotomy by laparoscopy. Eur J Pediatr Surg 1996 ; 6: 10-12.
9. Royal RE, Linz DN, Gruppo DL, Ziegler MM. Repair of mucosal perforation during pyloromyotomy: surgeon's choice. J Pediatr Surg 1995; 30: 1430-2.
10. Andropoulos DB, Heard MB, Johnson KL. Postanaesthetic apnoea in full term infants after pyloromyotomy. Anaesthesiology 1994; 80: 216-9.
11. MacDonald NJ, Fitzpatrick GJ, Morre KP et al. Anaesthesia for idiopathic hypertrophic pyloric stenosis: A review of 350 patients. Br J Anaesth 1987; 59: 672-7.
12. Moschini V, Sartori A, Sogni A, Lanata M. Anaesthesia in hypertrophic pyloric stenosis. Minerva Anaesthesiol 1995; 61: 259-64.