PRESENTATION AND OUTCOME OF INFANTILE HYPERTROPHIC PYLORIC STENOSIS: A FIVE YEAR RETROSPECTIVE STUDY IN CHITTAGONG MEDICAL COLLEGE

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Summary

Infantile hypertrophic pyloric stenosis (IHPS) is the most common cause of gastric outlet obstruction in infants with a prevalence of 1.5 to 4.0 per 1000 live births. It has been suggested that the incidence is increasing. USG is the most useful method for diagnosis supplanting physical examination and pyloromyotomy is the time honored treatment option. A retrospective review of the patients of IHPS admitted in the department of Pediatric Surgery, Chittagong Medical College Hospital (CMCH) Chittagong, Bangladesh from January 2008 to December 2012 was done. Help was also taken from yearly departmental audits of 2008 to 2012. Data were analyzed with regard to occurrence, seasonal variation, gender variation, presentation, diagnosis, treatment and outcome. Statistical analysis was performed by the chi-square test, and mean and percentage values were calculated. P<0.05 was considered to be statistically significant. There were 42 patients of IHPS. Age range was 5 days to 1 year 8 months with mean age of 79 days. Male to Female ratio was 2.5:1. There was no significant difference between the rate of occurrence of IHPS and gender (P=0.5). There were 2 peaks on seasonal variations of IHPS during summer and winter but quarterly distribution was not statistically significant. Nonbilious vomiting was the most common clinical presentation and USG was the most useful investigation. Pyloromyotomy was done in all patients and persistent vomiting (24%) was the most common complication. The occurrence of IHPS was 0.29% of all admissions. There was no significant gender or seasonal variations. USG has replaced clinical examination as the most effective tool for diagnosis. Ramstedt pyloromytomy is still the choice of treatment on the basis of outcome.

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Key words

Infantile hypertrophic pyloric stenosis; gender; seasonal variation; ramstedt pyloromyotomy.

Introduction

Infantile hypertrophic pyloric stenosis (IHPS) is the most common cause of gastric outlet obstruction in infants [1]. Though the prevalence of IHPS ranges from 1.5 to 4.0 per 1000 live births in Caucasian infants, it is less prevalent in African-American and Asian children [2]. But reports have suggested that the incidence is increasing [3,4]. It occurs more in boys than in girls, with a ratio of approximately 2:1 to 5:1[2]. Though the exact cause is not known, it has been associated with several variables including both environmental and familial factors. It is now thought to be caused by a mechanism other than a developmental defect. Thus it is generally agreed that IHPS is not a congenital abnormality [1,2]. Risk factors for HPS include family history, gender, younger maternal age, being a first-born infant, maternal feeding patterns, the method of feeding (breast-feeding versus formula feeding), seasonal variability, erythromycin exposure, and transpyloric feeding of premature infants [5-9]. The pylorus in IHPS is an enlarged, pale muscle mass usually measuring 2 to 2.5cm in length and 1 to 1.5cm in diameter [2]. Histologically, there is both muscle hypertrophy and hyperplasia primarily involving the circular layer and hypertrophy of the underlying mucosa [1]. Nonbilious projectile vomiting, started at at 2 to 8 weeks of age with a peak occurrence at 3 to 5 weeks; visible peristaltic waves in the left upper part of the abdomen; and hypochloremic, hypokalemic metabolic alkalosis are cardinal features of IHPS [1]. A definitive diagnosis can be made in 75% of infants with IHPS by careful physical examination of the upper part of the abdomen. Unfortunately, this is becoming a lost skill and USG has become not only the most common initial imaging technique for the diagnosis of IHPS but also the standard for diagnosing IHPS. USG has essentially supplanted physical examination as the diagnostic test of choice for IHPS [1]. The generally accepted criteria for a positive US study are a pyloric muscle thickness of 4mm or more and a pyloric

channel length of 16mm or greater [1]. The mainstay of therapy is typically resuscitation followed by pyloromyotomy [1]. Here we report our experiences of IHPS with regard to occurrence, seasonal variation, gender variation, presentation, diagnosis, treatment and outcome.

Materials and methods

Study design: Retrospective study

Period of study : Study period was between Jan 2008- Dec 2012 (total 05 years)

Place of study: This study was carried out in the department of Pediatric Surgery, Chittagong Medical College Hospital (CMCH) Chittagong, Bangladesh. Study Subjects: Patients admitted and diagnosed as IHPS in the department of Pediatric Surgery, CMCH were evaluated. Within the study period in a total of 66 patients, provisional diagnosis of IHPS was made. Among them 42 patients were finally diagnosed as IHPS based on operative findings. Other patients were excluded from the study. Incomplete and inadequate records were excluded from the study. Sample size: The sample size was 42.

Data Collection: Hospital records of patients diagnosed as IHPS were evaluated retrospectively. Help was also taken from yearly departmental audits of 2008 to 2012. Record file of each patient was scrutinized to check clinical features (eg. Non bilious vomiting, dehydration, abdominal distention, convulsion, malnutrition, visible peristalsis, palpation of olive like mass) and investigations (electrolytes, abdominal USG) to ascertain whether the diagnosis fit well or not. Outcome including morbidity, mortality and complications were evaluated. Total number of patients in a year and during the study period was calculated.

Data analysis: Year wise data were compiled and compared with these. Data were evaluated to see the yearly percentage of patient bulk, sex, age, seasonal variations, presentation, treatment, surgical intervention and outcome. Data were analyzed by SPSS 17. Statistical analysis was performed by the chi-square test, and mean and percentage values were calculated. P<0.05 was considered to be statistically significant.

Results

Of the 66 patients with provisional diagnosis of IHPS admitted in the department of Pediatric Surgery, CMCH during the 5 year period (Jan 2008-Dec 2012), 42 patients were finally recorded as cases of IHPS based on per-operative findings. Among the other 26 patients, 13 were diagnosed as malrotaion of gut, 2 midgut volvulus, 2 duodenal diaphragms,

1 antral web; 5 patients left the hospital and 3 patients died before operation. Age range was 5 days to 1 year 8 months with mean age of 79 days. 12 patients were less than 1 month, 23 between 1 and 2 months and 7 patients were more than 2 months old. Male (30) suffered more than the female (12) with Male to Female ratio was 2.5:1. There was no significant difference between the rate of occurrence of IHPS and gender (P=0.5). Table I shows the sex distribution of IHPS cases.

There were 2 peaks on seasonal variations of IHPS. One is during the winter in the months of October to January and the other is during the summer in the months of May and June. The highest number was in the month of May. Figure-1 shows the seasonal variations in the occurrence of IHPS. But When quarterly distribution of cases were compared with total number of admitted patients, there was no significant difference found among the quarters (P=0.4). Table-II shows the quarterly distribution of cases.

38(91%) patients had history of non-bilious vomiting. Table-III shows the clinical features of the patients.

On abdominal examination, olive like mass could be palpated in 19(45.24%) patients and was not found in 23 (54.76%) patients. Serum electrolyte was abnormal in 31(73.81%) patients and normal in 11(26.19) patients. USG of pylorus was done in all patients and showed hypertrophied pylorus.

Ramstedtpyloromyotomy was done in all patients with 31(73.81%) patients needing correction of electrolyte imbalance prior to surgery. Figure-2 shows per operative pictures of IHPS.

Most common complication was persistent vomiting after surgery all of which resolved after giving oral gastroprokinetic (Domperidon). Table-IV shows the complications. There were 4 (9.52%) deaths, 2 from severe dyselectrolytemia and 2 from sepsis.

Table I: Sex distribution of IHPS cases.

Sex	Total admissions	IHPS	Percentage
Female	4846	12	0.25
Male	9691	30	0.31
Total	14537	42	0.29

P value=0.5

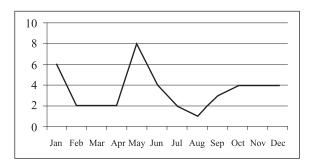


Fig 1: Monthly variations in the occurrence of IHPS.

Table II: Quarterly distribution of cases.

Quarter	Total Admissions	IHPS	Percentage
1 st	3426	10	0.29
$2^{\rm nd}$	3820	14	0.37
$3^{\rm rd}$	3640	6	0.16
4 th	3651	12	0.33

P value= 0.4

Table III: Presentations of the patients.

Presentations	No	Percentage
Non-bilious vomiting	38	90.48
Bilious vomiting	4	9.52
Severe dehydration	10	23.81
Convulsion	3	7.14
Visible peristalsis	2	4.76
Abdominal distension	2	4.76
Malnutrition	21	50.00
Jaundice	2	4.76





Fig 2 : Per-operative view of IHPS (A-Olive like mass of pylorus, B-pyloromyotomy).

Table IV: Complications of pyoloromyotomy.

Complication		
Perforation at myotomy site		4.76
Duodenal perforation by forceps injury		2.38
Wound infection	3	7.14
Burst abdomen	1	2.38
Persistent vomiting needing gastro-prokinetics	10	23.81
Convulsion	3	7.14
Delayed recovery from anesthesia	6	14.29
Mesenteric tear	3	7.14
Needed re-exploration (pyloric perforation,		
duodenal perforation, burst abdomen)		7.14
Death	4	9.52

Discussion

During these five years of study period there were 42 patients of IHPS in whom pyloromyotomy was done which was 0.29% of all admissions. 26 other patients who presented with persistent non-bilious vomiting were initially diagnosed as IHPS, but were later ruled out on the basis of investigation and operative findings. Mean age of presentation (2 months 19 days) was later than the peak occurrence of vomiting in the literature (3 to 5 weeks), which signifies that patients were presented late for surgical care [1]. There were only 12 (29%) patients who presented before 1 month of age. It may be due to this delayed presentation, 10 (24%) of our patients presented with severe dehydration and 31 (74%) had electrolyte imbalance which needed correction. There were also 3 patients who were much delayed presenter (more than 1 year), probably with milder form of IHPS [10].

Though male suffered more than the females with a male to female ratio of 2.5:1, it was not statistically significant as the number of admission for other disease was also more in male with a male to female ratio approaching towards 2:1. There were 2 peaks on seasonal variations of IHPS. One is during the winter in the months of October to January and the other is during the summer in the months of May and June. The highest number was in the month of May. The seasonal variation of the occurrence of IHPS has varied in different studies in the past [11-15]. But causes of disease prevalance are not clear. One previous study suggested that a higher incidence of IHPS was found in infants born in the third quarter (July-September) [12]. Dodge et al. reported that more IHPS infants were born in winter months, and the peak admission season was during the winter [11]. However, seasonal variation was not found by Rasmussen et al. and Lemessa et al[13-14]. Our study did not show significant differences among the

four seasons. Most of the patients (38) presented with non-bilious vomiting and 4 had bilious vomiting. 3 patients presented with convulsion who were 2.5 months, 2 months and 1.5 months old. Two of the patients presenting with convulsion died of their disease. Twenty one patients had malnutrition and 10 had severe dehydration. One patient had jaundice. Literature suggests that 2% to 5% of infants have jaundice from indirect hyperbilirubinemia, which is believed to be secondary to glucuronyltransferase deficiency [5]. Though IHPS is not usually associated with other anomalies, 1 of our patients had associated tongue tie and 1 had right inguinal hernia (Hydrocele). Clinical examination had been debated to be the most effective tool for diagnosis; but in our series in only 45% patients a clear mass could be felt and all the patients underwent USG. This is consistent with other studies where USG is gradually replacing clinical examination as a more reliable tool for diagnosis [1]

Ramstedtpyloromyotomy was done in all patients. Operations were done by different surgeons with different level of expertise. Prophylactic antibiotic (Amoxycillin/Ceftriaxone) was used in all cases, though there are debates regarding its usefulness [16-19]. A recent survey of members of the British Association of Paediatric Surgeons on the Surgical practice for infantile hypertrophic pyloric stenosis in the United Kingdom and Ireland showed that more than 50% of pediatric do not routinely prescribe antibiotics [20]. Prophylactic antibiotic was used by more surgeons using the umbilical incision due to an increased risk of wound infection by umbilical skin flora (Staphylococcusepidermidis)[20]. All the operations in our series were performed through a classical right upper quadrant (RUQ) incision; though several other approaches have been currently advocated for pyloromyotomy including the umbilical skinfold incision popularized by Tan and Bianchi and laparoscopy pioneered by Alain et al[16-17]. Though medical treatment by Atropine was used in early days and still is practiced in some centers, our protocol is to do pyloromyotomy for IHPS patients [21].

The most common complication was persistent vomiting after surgery (24%) which resolved with gastroprokinetics within a week. Emesis has been reported to be a frequent finding for a few days in the postoperative period but usually resolves spontaneously. This emesis may be from pyloric edema, gastroparesis, pylorospasm, or GER [22]. In 1 patient there was unfortunate complication of duodenal perforation caused as a result of holding

the duodenum with Babcock forceps during delivering the pylorus through the wound. This patient later needed re-exploration and repair of perforation and recovered uneventfully. Two patients developed perforation of pyloric mucosa which was closed and myotomywas done on the opposite site. These patients also recovered uneventfully. This complication was far less than some other reported series [22]. The available literature suggests that earlyrecognition and closure of an incidental perforation is not associated with an increased incidence of morbidity[22-24].

Complications after pyloromyotomy (mucosal perforation, bleeding, wound infection, wound dehiscence, and incisional hernia) have been reported with a rate of up to 8% [25-26]. The 3 largest studies looking at pyloromyotomy have involved a total of 570 infants and have shown rates of wound infection of between 3% and 7%, dehiscence rates of 0% to 2.6%, and perforation rates of 3.3% to 14.8% [27-29]. There were 4 deaths; 2 from severe dyselectrolytemia and 2 from sepsis. Death rates were more than that of reported in most literatures, probably due to the delayed presentation as well as failure of early correction of fluid and electrolyte loss. There were no cases of recurrence which was reported in some reports[30].

Conclusion

The occurrence of IHPS was 0.29% of all admissions. There was no significant gender or seasonal variations. USG has replaced clinical examination as the most effective tool for diagnosis. Ramstedtpyloromytomy is still the choice of treatment on the basis of outcome.

Disclosure

All the authors declared no competing interest.

References

- **1.** Schwartz M Z. Hypertrophic Pyloric Stenosis. In Pediatric Surgery .Ed Coran AG. 2012;7:1021-1028.
- **2.** Mitchell L.E., Risch N.: The genetics of infantile hypertrophic pyloric stenosis. Am J Dis Child 1993; 147:1203.
- **3.** Mason P.F.: Increasing infantile hypertrophic pyloric stenosis? Experience in an overseas military hospital. J R CollSurgEndinb 1991; 36:293.
- **4.** O'Donoghue J.M., Connolly K.D., Gallagher M.M., et al: The increasing incidence of infantile hypertrophic pyloric stenosis. Ir J Med Sci 1993; 162:175.

- **5.** Roth B., Statz A., Heinisch H.M., Gladtke E.: Elimination of indocyanine green by the liver of infants with hypertrophic pyloric stenosis and the icteropyloric syndrome. J Pediatr 1981; 99:240.
- **6.** Honein M.A., Paulozzi L.J., Himelright I.M., et al: Infantile hypertrophic pyloric stenosis after pertussis prophylaxis with erythromycin: A case review and cohort study. Lancet 1999; 354:2101.
- 7. Rasmussen L., Green A., Hansen L.P.: The epidemiology of infantile hypertrophic pyloric stenosis in a Danish population. Int J Epidemiol 1989; 18:413.
- **8.** Tack E.D., Perlman J.M., Bower R.J., McAlister W.H.: Pyloric stenosis in the sick premature infant: Clinical and radiologic findings. Am J Dis Child 1988; 142:68.
- **9.** Curt S, Wulkan M. Lesions of the stomach. In Holcomb GW, Murphy JPeds: Ashcraft's pediatric surgery. 2010; 5:391-395.
- **10.** Mac Cann and Dean, 1950, Mac Connell, 1996. Hypertrophic pyloric stenosis. F. Med. genet (1996) 6,126-download from site; jmg.bmj.com on Dec. 25,2013,-published by group.bmj.com
- **11.** Dodge JA Infantile hypertrophic pyloric stenosis in Belfast, 1957-1969. Arch Dis Child 1975; 50: 171–178.
- **12.** Adelstein P, Fedrick J Pyloric stenosis in the Oxford Record Linkage Study area. J Med Genet 1976; 13: 439–448.
- **13.** Rasmussen L, Green A, Hansen LP The epidemiology of infantile hypertrophic pyloric stenosis in a Danish population, 1950-84. Int J Epidemiol 1989; 18: 413–417.
- **14.** Lemessa D Infantilehypertropic pyloric stenosis in a children's hospital a retrospective study. Ethiop Med J 1990; 28: 169–173.
- **15.** Leong M-M, Chen SC-C, Hsieh C-S, Chin Y-Y, Tok T-S, et al. (2011) Epidemiological Features of Infantile Hypertrophic Pyloric Stenosis in Taiwanese Children: A Nation-Wide Analysis of Cases during 1997–2007. PLoS ONE 6(5): e19404. doi:10.1371/journal.pone.0019404
- **16.** Tan KC, Bianchi A. Circumumbilical incision for pyloromyotomy. Br J Surg 1986;73:399.
- **17.** Alain JL, Grousseau D, Terrier G. Extramucosalpylorotomy by laparoscopy. J PediatrSurg 1991;26:1191-1192.
- **18.** Nour S, MacKinnon AE, Dickson JA, Walker J. Antibiotic prophylaxis for infantile pyloromyotomy. J R CollSurgEdinb 1996;41:178-180.

- **19.** Ladd AP, Nemeth SA, Kirincich AN, et al. Supraumbilicalpyloromyotomy: a unique indication for antimicrobial prophylaxis. J PediatrSurg 2005;40:974-977.
- **20.** DhanyaMullassery, Daniel Perry, AnjuGoyal, Edwin C. Jesudason, Paul D. Losty. Surgical practice for infantile hypertrophic pyloric stenosis in the United Kingdom and Ireland: A survey of members of the British Association of Paediatric Surgeons.. Journal of Pediatric Surgery 2008; 43: 1227–1229.
- **21.** Hisayoshi Kawahara, Yuichi Takama, Hideki Yoshida, et al.Medical treatment of infantile hypertrophic pyloric stenosis: should we always slice the olive? Journal of Pediatric Surgery 2005; 40: 1848–1851.
- **22.** J.S. White, W.D.B. Clements, P. Heggarty, et al.Treatment of Infantile Hypertrophic Pyloric Stenosis in a District General Hospital: A Review of 160 Cases. Journal of Pediatric Surgery, 2003; 38(9): 1333-1336.
- **23.** Graham DA, Mogridge N, Abbott GD, et al: Pyloric stenosis: The Christchurch experience. N Z Med J 1993; 106:57-59.
- **24.** Royal RE, Linz DN, Gruppo DL, et al: Repair of mucosal perforation during pyloromyotomy: Surgeon's choice. J PediatrSurg 1995; 30:1430-1432.
- **25.** Hulka F, Harrison MW, Campbell TJ, et al. Complications of pyloromyotomy for infantile hypertrophic pyloric stenosis. Am J Surg 1997;173:450-452.
- **26.** Zeidan B, Wyatt J, Mackersic P, et al. Recent results of treatment of infantile hypertrophic pyloric stenosis. Arch Dis Child 1988;63:1060-1064.
- **27.** Zhang AL, Cass DT, Dubois RS, et al: Infantile hypertrophic pyloric stenosis: A clinical review from a general hospital. J Paediatr Child Health 1993; 29:372-378.
- **28.** Saunders MP, Williams CR: Infantile hypertrophic pyloric stenosis: Experience in a district general hospital. J R CollSurgEdinb1990; 35:36-38.
- **29.** Maher M, Hehir DJ, Horgan A, et al: Infantile hypertrophic pyloric stenosis: Long-term audit from a general surgical unit. Irish J Med Sci 1996; 165:115-117.
- **30.** Ahmed Nasr, Sigmund H. Ein, Bairbre Connolly. Recurrent pyloric stenosis: to dilate or operate? A preliminary report. Journal of Pediatric Surgery 2008; 43: E17–E20.