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A Prospective Study of Eighty Cases of Infantile Hypertrophic Pyloric Stenosis

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Abstract
This is a prospective study of (80) cases of infantile hypertrophic pyloric stenosis (I.H.P.S.) who were admitted to the children welfare Teaching Hospital in the period from January 2010 to October 2011. The aim of this study is to analyze the patients according to their age at presentation, sex, family history, body weight at operation, feeding history and general condition of the baby with the result of surgical intervention.
Keywords: IHPS, gastric outlet in infants, CHPS, congenital hypertrophic pyloric stenosis.

1. Introduction

IHPS is most common gastric outlet obstruction in infants. Congenital hypertrophic pyloric stenosis is the commonest cause of gastric outlet obstruction and vomiting in infancy resulting from gradual hypertrophy of smooth muscle of pylorus. Reports have suggested that the incidence of pyloric stenosis is increasing. (1)

The appearance of the pylorus in IHPS is that of an elongated pale mass usually measuring (2 – 2.5 cm) in length and (1- 1.5 cm) in diameter which is due to both hypertrophy and hyperplasia of circular muscle. (3) (4)

Within the pyloric tumor there are hypertrophied autonomic nerve fibers and fewer than normal ganglionic cells which may lead to failure of muscle relaxation. (3)

Grossly the pylorus is hard, whitish mass with different sizes.in autopsy specimens, the pylorus may barely admit 1 mm probe, stomach proximal to the obstruction is dilated and its wall is thick and edematous. There is no evidence of inflammation at first, but submucosal edema and round cell inflammation appear to occur after prolonged vomiting. Histology demonstrated hypertrophy and hyperplasia of circular muscle fibers. (5)

The incidence was more frequent in white populations, males four times than females, hereditary predisposition, for affected male parent, the incidence in offspring as follow:1 :20 male offspring and 1:50 female offspring. While the incidence in male offspring of affected mother is 15% and has incidence in babies with malrotation, obstructive uropathy and esophageal atresia. (3)

The vomitus of the babies with IHPS contain varying amount of Cl, Na, K. The gastric fluid loss through the vomitus or nasogastric tube may be as high as 100 ml/day. This excessive Cl ion loss results in extracellular Cl depletion and metabolic alkalosis. (17)

Secondary hyperaldosteronism developed due to hypovolemia, the high aldosteron cause the kidney to avidly retain Na and increase excretion of K leading to hypokalemia. The body compensate to this alkalosis by hypoventilation leading to increase arterial PaCO2. (17)

- The diagnosis of IHPS is essentially clinical, but when the diagnosis is in doubt ultra sound (U/S) is nearly 100% accurate in diagnosis, sometimes supplemented by Barium meal. (3)
- Complete blood picture, renal function test, serum electrolytes.
- The u/s observation that suggest pyloric stenosis includes bull-eye appearance of central or stellate echo surrounded by thickened pyloric muscles. (12)
- Criteria for positive u/s study include pyloric canal more than 16mm (normal length 5-14mm), diameter larger than 11mm, circular muscle thickness more than 2.5mm, shouldering may be seen at each end of canal.
- U/S must be done without echogenic milk curds and barium, because it impedes transmission of sound waves therefore the infant stomach must be emptied with N/G tube and wash with normal saline, the infant then allowed to take GW to make the stomach as a window for viewing the pylorus. (12)

2. Patient and method

A prospective study of 80 patients with infantile hypertrophic pyloric stenosis that were admitted and managed in Children Welfare teaching hospital over a period from January 2010 to October 2011.

Data collected from those patients focused on age, sex, birth weight, body weight, onset of symptoms, duration of symptoms, family history, feeding history, sequence of the patients in the family, we received the patients either directly from Baghdad city or as a referral from different areas of our country, those patients were admitted to the hospital kepton fluid (maintenance and deficit) and U/S exam were done to all patients on the same day or the next day of admission.

All patients were diagnosed by history and physical examination supported by ultrasonographical examination or by upper GI contrast study which was done infew patients to confirm the diagnosis.

Ramstedt`s operation (pyloromyotomy) was done to all patients under general anesthesia as semi-emergency rather than an emergency cases.

- All the patients were full term babies with male to female ratio of (5.6\ 1).
- The age at presentation was between three to twelve weeks of age.
- Seventy-six cases presented with projectile vomiting shortly after feeding.
- Forty-six cases were diagnosed by history and physical examination by palpating the mass (the olive) then confirmed the diagnosis by ultrasonographical examination, the remaining cases diagnosed by ultrasonographical examination with or without Barium meal to confirm the diagnosis.

3. Results

All the patients had Ramstedt`spyloromyotomy with a mortality rate of (1.25%)which was due to septicemia resulting from late diagnosis and there was no recurrence of the condition.

All patients were full term babies with birth weight ranging from (2.5 to 4) Kg. The age at presentation was most common between 20 days and 3 months with a peak incidence at the fourth week of age, as illustrated in table (1).

This table showed the peak incidence in forth weeks' age infant. Projectile vomiting was the main symptom at presentation and was gradual in onset in 46 patients while in 34 patients it was abrupt. in 8 patients, the vomitus was bloody.

Jaundice was present in (11) patients (13.75%) and was moderate in severity and of indirect type (less than11mg/dl), yet it didn't preclude operation. In (72) patients (90%) constipation was a significant symptom.

Most of our patients were dehydrated at time of presentation which ranged from mild in (67) patients (83.75%) to severe dehydration in (13) patients (16.25%). There were no associated congenital anomalies, except in one patient who had ventricular septal defect and was in heart failure at time of presentation.

Table (1): The age at presentation.

Age in weeks	No. of patients	Percentage
Third week	12	15%
Forth week	34	42.5%
Fifth week	11	13.75%
Sixth week	9	11.25%
Seventh week	4	5%
Eight week	2	2.5%
Ninth week	1	1.25%
Tenth week	4	5%
Eleventh week	2	2.5%
Twelfth week	1	1.25%
Total	80	100%

The body weight at the time of operation ranged from 2-4Kg. Most of the patients were breast fed51 patients (63.75%,), 18 patients (22.5%) were bottle fed and 11 patients (13.75%) were of mixed feeding (breast &bottle).

A palpable abdominal mass was felt in (46) patients in the upper abdomen. Upper abdominal distension was noticed in (54) patients (67.5%); visible peristaltic movements were noticed in (34) patients (42.5%).

In the (34) remaining patients (42.5%) in which a mass could not be felt, ultrasonography was done for them. In (33) patients we were satisfied by the result, but in one patient the result was not conclusive so Barium study done to confirm the diagnosis.

Although (46) patients were diagnosed by feeling the palpable mass, all case had been sent for ultrasonographical exam to determine the measurement of the mass and to confirm our diagnosis.

The measurements of length of pyloric canal by the ultrasound examination with the number of patients are shown in next table.

Table (2): Length of pyloric canal.

Length in mm	No.	%
15	2	2.5%
16	7	8.75%
17	12	15%
18	19	23.75%
19	10	12.5
20	12	15%
21	8	10%
22	6	7.5%
23	4	5%
Total	80	100%

Table (3): Thickness of pyloric muscle.

Thickness in mm	No.	%
3	16	20%
3.5	32	40%
4	16	20%
4.5	7	8.75%
5	4	5%
5.5	2	2.5%
6	2	2.5%
6.5	1	1.25%
Total	80	100%

Ramstedt`s operation was done to all the patients with no gastric mucosal perforation happened. Nine patients developed vomiting postoperatively who were managed successfully by conservative medical means in a form of i.v fluids and delayed feeding.

One patient died (mortality rate of 1.25%) due to septicemia. The duration of hospital stay ranged from (2 to 4) days. In none of the cases recurrence of the symptoms developed.

4. Discussion

- Infantile hypertrophic pyloric stenosis is the most common cause of gastric outlet obstruction in infancy and is also one of the most frequent conditions requiring surgery. [24]
- In all of the studies dealing with IHPS there is a male predominance, the ratio differs in different studies. In our study male to female ratio was (5.6: 1) which is different to the result of Ferman A.N. [25] study in 1999 where he found a ratio of (11.5:1) and near the results that done by University of Naples [26] in 1998 which gave a ratio of (4.4:1) and that done by Janet R. Reid who found a ratio of (4.6:1). [10]
- Still the first-born baby is affected more than the others, in our study (52.5%) of cases were first born baby compared with (58%) of cases in the study of University of Naples [26] and (44%) in Ferman A.N. study. [25]
- Family history was positive in only one case in this study 1.25%, but it was positive in (2%) of Ferman A.N. study [25] and (12%) of Frieda Hulka study in 1997. [27]
- The age of presentation in this study ranged from (3-12) weeks with the peak incidence at (4) weeks of age, this is similar to Frieda Hulka study [27], Janet study [10], and Gudrun Aspelund. [33]
- The main symptom of presentation was projectile vomiting which is found in (95%) of our patients, this result is similar to that of Zeidan B in 1988. [28]
- The vomitus was mostly milky, but sometimes blood stained because of gastritis and esophagitis. [4]
- Constipation was found in (90%) of cases compared with (60%) in Ferman AN study [25] and (65%) in Janet study. [10]
- Body weight at time of presentation ranged from (2-4) Kg which is near the result found by Frieda Hulka that was (2-6) Kg. [27]
- The degree of dehydration ranged from mild dehydration in (83.75%) of cases to severe dehydration in (16.25%) of cases, this is comparable with the results found by Ferman A.N [13] in which (36%) of their patients have mild dehydration and (14%) of severely dehydrated patients. This means that the

presentation is almost the same in all infants and the difference in weight depends on the time of diagnosis and the degree of dehydration of the patient.

- Most of our patients were breast fed (63.75%) ,(22.5%) were bottle fed and (13.75%)mixed compared with (68%) breast fed , (12%) bottle fed and (20%) mixed fed in Naples study [26] and (67.8%) ,(12.2%) and (20%) respectively in Alfredo Pisacane study [29] which means that it is more prevalent in breast fed babies. while it is more common in bottle feeding in Gudrun Aspelund study 2007. [33]
- By physical examination, palpable abdominal mass was found in (57.5%) of cases compared with (70%) in Frieda Hulka study [27] and (65%) in Janet study.[10],this is because feeling of the mass need soft abdomen and this need calm patient and most of our patients are irritable during examination.
- Visible gastric peristaltic waves were found in (42.5%) of cases and abdominal distension in (67.5%) compared with (58%) and (62%) in Frieda study [27] respectively.
- These results were near those of Salih W.M. [30] study who found that more than (14) mm in length and more than (3) mm in thickness of pyloric mass could give the picture of IHPS , and near the results of Janet [10] who found the measurement of more than (3)mm and more than (17)mm as diagnostic. In Riccabona study [31] in 2001 they found that the length of the "tumor" was (20) mm as a mean and the thickness of the mass was (4.5) mm as a mean.
- Presently there is general agreement that ultrasonography is the investigation of choice in diagnosing IHPS and is almost (100%) accurate, though it depends on the experience of the radiologist.
- There was no operative complication of perforated duodenal mucosa. This result is similar to Gudrun Aspelund 2007 [33] which has also 0% and Different from Zeidan B. study [28] with (8%) perforated duodenal mucosa with the same management. In Daniel A. Beals study [32], this complication occurred in (12%) of cases.
- Nine of our patients developed vomiting postoperatively who were managed conservatively with success which is similar to Gudrun Aspelund 2007[33] which has 3.5% postoperative vomiting treated conservatively also.
- No wound infection or dehiscence happened in our study as compared to 1% in Gudrun Aspelund 2007. [33]
- Mortality rate was (1.25%) in our study that is not directly related to the operative procedure, but to the general condition of the patient, this is less than the result of Zeidan B. study [28] who gave a mortality rate of (2%) related to the congenital anomalies and sepsis, and more than (0.3%) rate in Jagvis study. [3]
- This means that Ramstedt`s operation is a safe procedure with controlled complication if the patient is prepared preoperatively and had no serious congenital anomalies that interfere with life or is immunologically compromise.
- The duration of hospital stay in our study was (2-4) days postoperatively compared with (1-7) days in Ferman A.N study. [25] This means that the preoperative general condition of the patient with time needed for resuscitation and the need for further investigation may lead to prolonged hospital stay. Furthermore, the operative procedure itself and its complication may delay the starting of oral feeding or need more time for postoperative observation till the vomiting ceases.
- No recurrence of symptoms in our study was encountered

5. Conclusions

- Infantile hypertrophic pyloric stenosis is more common in first born male baby in the first four weeks of his life.
- It should be considered in every infant with projectile vomiting in the first three months of his age.
- Palpation of a mass is sufficient to make the diagnosis.

References

1. Baily and Love's, short practice of surgery, hypertrophic pyloric stenosis of infant, 25th edition, Chapman and Hall, 2008: pp 78-79.
2. Lewis Spitz, Arnold G. Coran. pediatric surgery. Rob and smith operative Surgery, pyloromyotomy ,5th edition.Chapman and Hall . 2005: pp 321-327.
3. Jagvir Singh, eMedicine Specialities, Emergency Medicine, pyloric stenosis, Electronic Article. Jagvir department of emergency medicine, division of pediatric emergency medicine, pyloric stenosis August 1,2007, Lutheran General Hospital of Park Ridge
4. Oue T, Puri P. Abnormalities of elastin and elastic fibers in infantile Hypertrophic Pyloric Stenosis, emedicine specialities, Emergency Medicine, pediatric. Pediatr Surg Int 1999; 15(8):540-2Abstract quote.
5. James A. O. Neill Jr, Mare I. Rowe, Jay L. Grosfeld. Pediatric Surgery, Hypertrophic Pyloric Stenosis. 6th edition, Library of Congress Cataloging –in- publication Data. 2006: ch: 76:1215-1224.

6. Richard E. Behrman,MD.Robert M. Kliegman,MD.Hal B. Jenson,MD. Nelson Text book of Pediatric. Pyloric Stenosis and congenital anomalies of the stomach.17th edition. USA Library of congress cataloging-in-Publication data, 2004, ch: 310:1229-1231
7. Richard Snell, Clinical anatomy for medical students, Embryology of stomach.2nd edition USA: Little, Brown and company, 2005, ch: 5 pp 143 – 150.
8. Ohshiro K, Puri P. Increased insulin-like growth factor-I mRNA expression in pyloric muscle in infantile hypertrophic pyloric stenosis.Ped Surg Int 1998;13:253-5
9. Schwartz Shires Spencer: Principle of surgery. pyloric stenosis. 9th edition USA@.2010:1425-1426.
10. Janet Reid, HPS, Medicine Warld Medical Library, June 21, 2002. Cleveland clinic children`s Hospital.
11. Ceccarelli: M., Villirillo-A Balsano-L Hypertrophic Pyloric Stenosis Electronic Article. Medical Journal ,1992.
12. Waldoe Nelson: congenital hypertrophic pyloric stenosis in Nelsons textbook of paediatrics(4th edition)1998,pp.984-950
13. Carol A.Redel and R.Jeff Zwiener: infantile hypertrophic pyloric stenosis in Sleisenger and Fordtrans gastrointestinal and liver disease,6th edition,1998,volume1,pp.566-567.
14. Kobayashi H, O, Briain DS, Puripp: Immunochemical characterization of neural cell adhesion molecule, nitric oxide synthase and neurofilament protein expression in pyloric muscle of patient with pyloric stenosis in journal of pediatric gastroenterology (April 1995) 20 (3); pp.319-325.
15. Michel T. Nitric oxide synthesis in hypertrophic pyloric stenosis.N.Engl.Jornal of medicine. (Dec.1999)327(23); pp.1690-1691.
16. Thom E. Lobe: Pyloromyotomy in Robe & Smiths (Operative surgery for paediatric surgery) 4th edition, 1996, pp.267-272.
17. Bayle Bastos F, Mayol Belda MJ, Mira Navarro J, et al:Physiopathology of hypertrophic pyloric stenosis. Cir. Paediatric. (Oct. 1998) 2(4); pp. 172-174.
18. Schmuel Katz, Danial Basel, & David Branski: Prenatal gastric dilatation & infantile hypertrophic pyloric stenosis: Journal of paediatric surgery (Nov. 2001) vol.23, No.11, pp. 1021-1022.
19. Raffensperger JG.: pyloric stenosis Ch.26, in swensons Paediatric Surgery (5th edition)1990 pp.211-219.
20. Levine-D and Edwards-DK: The Olive on end: A useful variant of the shoulder sign in barium x-ray diagnosis of idiopathic hypertrophic pyloric stenosis. Paediatric Radiology 1992,22(4), pp.275-276.
21. Emmink-B,Hadley-GP and Wiersma-R: infantile hypertrophic pyloric stenosis in third world environment,South Africa medical jornal,(Sept.2002),82(3),pp.168-170.
22. Maher-M:Infantile hypertrophic pyloric stenosis: long term audit from a general surgical unit.Ir.Journal of medical science, (Apr.1996), 165(2), pp.115-117.
23. Ng WT, Lee SY. Hypertrophic pyloric stenosis, congenital or notcongenital: a critical overview. Pediatr Surg Int 2007; 18:563-4.
24. Keith W. Aschraft.Pediatric surgery, Lesion of the stomach, Hypertrophic pyloric stenosis. 5th edition W.B Saunders company .2009. pp. 391-395.
25. Ammar N. Forman, M. B. Ch. B. Analysis of Fifty Cases of Infantile Hypertrophic Pyloric Stenosis in Iraq. Athesis submitted to the Scientific Council of the Commission for the Medical Specialization.1999.
26. Pisocane MD. Infantile Hypertrophic Pyloric Stenosis. Journal of University of Naples, Electronic Article,Santobono pediatric Hospital,Italy,1998.
27. Frieda Hullka, Timothy J. Campell, John R. Campell, and Marvin W. Harrison. Evolution in the recognition of Infantile Hypertrophic Pyloric. Stenosis. Electronic Article Copyright 1997 by American Academy Pediatrics. Pediatrics vol. 100 No. 2. August 1999. P. 9.
28. Zeidan B. Jwyatt J; Mackersie A.; Brereton R. J. Recent results in the treatment of Infantile Hypertrophic Pyloric Stenosis. Journal: ARCH. DIS. CHILD. 1998, 63/9 (1060-1064). Copyright Elsevier Science B.V.2006.
29. Alfredo Pisacanse . Breast feeding and HPS,Astudy of breast feeding and hypertrophic pyloric stenosis dose not conflict with others,Electronic Article eMedicine Specialities,Emergency Medicine, Pediatric 1996;312,1674(29 June).
30. Waad M. Salh. diagnostic value of ultrasound in IHPS. A Thesis submitted to the Scientific Council of the Commission for the Medical Specialization. P.5 1997.
31. M. Riccabona, C. Weitzer, F. Lindbicher and J. Mayr Sonography and Doppler sonography for monitoring conservatively treated Infantile Hypertrophic Pyloric Stenosis. Journal of ultrasound in medicine, Journal Article, Sep 2008;20 (9): 997 -1002 abstract quote.
32. Daniel A. Beals, MD, Medicine instant. access to the minds of medicine, section of pediatric surgery, pyloric stenosis, university of Kentuky 11, 2002.
33. Gudrun Aspeland, Jacob C. Current management of pyloric stenosis. Seminar in paediatric surgery (2007) 16, p27_33.