Is There a Relation Between Pyloric Muscle Thickness and Clinical and Laboratory Data in Infants with Hypertrophic Pyloric Stenosis?

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Abstract Literature about the association between clinical and laboratory presentation and pyloric muscle thickness in infants with pyloric stenosis is lacking. The objective of this study is to examine the effect of pyloric muscle thickness on clinical and laboratory presentation in infants with pyloric stenosis. Thirty infants with hypertrophic pyloric stenosis presented at the Maternity and Children Hospital, Al-Madinah, Saudi Arabia, were included in this study analysis. Clinical and laboratory data of these infants were recorded. Statistical tests and univariate linear regression analyses were used as appropriate. *P* value was considered significant at a level ≤ 0.05 . The mean age of the studied infant was 5.1 ± 2.6 weeks and mean weight was 3.4 ± 0.7 kg. Most of these infants were full-term (93.3 %), some presented to the hospital dehydrated (80 %), and some were lethargic (56.7 %). The mean pyloric muscle thickness among these infants showed statistically significant difference with regard to the studied clinical and laboratory data. A linear regression analyses also showed positive association between increasing pyloric muscle thickness and almost all studied clinical and laboratory data, with statistically significant difference observed for hyponatremia, hypochloremia, and high bicarbonates. The findings suggest that pyloric muscle thickness is a significant factor that determines the severity of clinical and laboratory presentation in infants with pyloric stenosis.

Introduction

Hypertrophic pyloric stenosis (HPS) is one of the common gastrointestinal emergencies in early infancy with a recognized incidence of 2 to 4 per 1,000 live births in Western countries and 8.2 to 12.3 per 1,000 live births in the US [1, 2]. It is commonly presented during the first 2 to 12 weeks of life [3]. The affected infant usually presents with repeated, non-bilious vomiting after feeding which is initially non-projectile that with time progresses to a projectile one. This is why in the early stages HPS can be misdiagnosed as a gastro-oesophageal reflux disease. This delay in the diagnosis (usually more than 2 weeks) causes continuous loss of hydrochloric acid from persistent vomiting that consequently causes changes in the serum: pH, sodium, potassium and chloride levels. The classical change that has been described is hypochloremic metabolic alkalosis. Persistent vomiting can also lead to dehydration that results in either hyper- or hyponatremia. Some authors [4] reported in their works that the severity of clinical and biochemical changes has a relation to the duration of symptoms. However, others failed to confirm this relation [5]. Based on this pathophysiology, the study was designed to find if there is a relation between pyloric muscle thicknesses and the clinical and laboratory presentation in infants with hypertrophic pyloric stenosis.

Keywords Pyloric stenosis · Dehydration · Infants · Vomiting · Electrolytes

Patients and Method

A retrospective review was performed on all cases of infants with pyloric stenosis admitted at the Maternity and Children Hospital, Al-Madinah, Saudi Arabia, between Jan. 2008 and Dec. 2012. The collected data were the following: patients' demography, clinical presentations, duration of symptoms, laboratory data (pH, HCO_3^- , Na^+ , K^+ , Cl^-), and ultrasound findings on admission. The institutional review board

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approved the study. The socio-demographic variables were categorized during analysis as follows: weight (<4 kg vs. >4 kg, according to its median distribution in the studied sample) and sex (male vs. female), while the clinical data were categorized as follows: vomiting duration (<1 week vs. >1 week, according to the association between the duration of vomiting and severity of clinical presentation) and presence or absence of dehydration (classified based on the clinical examination and laboratory data at admission). The normal ranges for electrolytes based on which we categorized them into normal, high or low were as follows: Na^+ =135–145 mmol/L, Cl^- =98–108 mmol/L, K^+ =3.5–4.5 mmol/L, and HCO_3^- =22–30 mmol/L. The variables were categorized according to their median distribution among the whole studied infants.

Statistical Analysis

The data was analyzed using statistical analysis software package (SAS). The studied data were presented by their frequency number and percent for the categorical variables and mean±SD for continuous variables. Unpaired *t* test and ANOVA were used as appropriate to compare the mean muscle thickness of the pyloric canal by the different studied variables. Fisher's exact tests were used to compare vomiting duration with the studied factors. A univariate linear regression analyses were also used to examine the association of pyloric muscle thickness with the studied clinical and laboratory data. *P* value was considered significant at a level ≤0.05.

Results

The study included 30 infants with hypertrophic pyloric stenosis. All the studied infants were male except three. Table 1 presents general, clinical, laboratory, and ultrasonographic findings of the studied infant. The mean age was 5.1 ± 2.6 weeks and mean weight was 3.4 ± 0.7 kg. Twenty-eight of the studied infants (93.3 %) were full-term. Ten of them (33.3 %) presented with history of non-bilious vomiting of less than 1 week. Twenty-four of the studied infants presented with some degree of dehydration (80 %) and 17 with lethargy (56.7 %). High bicarbonate level was found in 56.7 % and alkaline pH in 60 %. Low sodium, potassium, and chloride levels were detected in 36.6 %, 20 %, and 43.3 % of them, respectively. The mean muscle thickness of pyloric stenosis was 5.3 ± 1.1 mm, and the mean pyloric length was 19.5 ± 3.2 mm.

The results showed nearly similar mean thickness of the pyloric muscle among the studied infants regarding their gestational age and weight at diagnosis. The mean of muscle thickness was higher among dehydrated and lethargic infants compared to other infants with statistical significant difference. In addition, there has been statistically significant

Table 1 Characteristics of the studied infants at their presentation

Characteristics	No. (%), N=30
Infant age in weeks, mean±SD (range)	5.1±2.6 (1.7–12.8)
Weight of infant in kg, mean±SD (range)	3.4±0.7 (1.6–4.6)
Gestational age	
Full-term	28 (93.3)
Preterm	2 (6.7)
Duration of vomiting in weeks	
>1 week	23 (77.0)
<1 week	7 (23.0)
Dehydration	
Yes	24 (80.0)
No	6 (20.0)
Lethargy	
Yes	17 (56.7)
No	13 (43.3)
Palpable olive	
Yes	16 (53.3)
No	14 (46.7)
Serum sodium level (Na^+)	
Normal	17 (56.7)
High	2 (6.7)
Low	11 (36.6)
Serum potassium level (K^+)	
Normal	20 (66.7)
High	4 (13.3)
Low	6 (20.0)
Serum chloride level (Cl^-)	
Normal	17 (56.7)
High	0
Low	13 (43.3)
Serum bicarbonate (HCO_3^-)	
Normal	11 (36.7)
High	17 (56.7)
Low	2 (6.6)
Blood pH	
Normal	12 (40.0)
Alkaline	18 (60.0)
Pyloric muscle thickness in mm, mean±SD (range)	5.3±1.1 (4.0–10.0)
Pyloric length in mm, mean±SD (range)	19.5±3.2 (12.0–27.0)

difference of mean muscle thickness by vomiting duration, where the higher thickness was among infants with more than 1 week duration (*P* value<0.01). Also the mean muscle thickness was higher among those infants with clinically palpable pyloric mass, compared to those with no clinically palpable pyloric mass with statistically significant difference (*p*=0.002) (Table 2).

Table 2 Comparison of the studied infants by their pyloric muscle thickness and the studied clinical data ($N=30$ infants)

Clinical data	Mean $\pm$ SD	<i>P</i> value
Gestational age		
Full-term	5.3 $\pm$ 1.1	0.85
Preterm	5.4 $\pm$ 0.1	
Weight of infant		
≤ 4 kg	5.3 $\pm$ 1.2	0.97
>4 kg	5.2 $\pm$ 0.5	
Duration of vomiting in weeks		
≤ 1 week	4.9 $\pm$ 1.2	0.01 ^a
>1 week	5.4 $\pm$ 1.1	
Dehydration		
Yes	5.4 $\pm$ 0.6	0.02 ^a
No	4.3 $\pm$ 0.7	
Lethargy		
Yes	5.5 $\pm$ 1.4	0.03 ^a
No	4.1 $\pm$ 0.7	
Palpable olive		
Yes	5.6 $\pm$ 1.4	0.002 ^a
No	4.9 $\pm$ 0.5	

^a Significant

The mean pyloric muscle thickness among those infants with low sodium and chloride levels and those with alkaline blood pH and high bicarbonate was higher with statistically significant differences (P value 0.04, 0.002, 0.04, and 0.04, respectively). The mean pyloric muscle thickness was nearly the same among infants with different potassium levels (Table 3).

The results of the linear regression analysis for pyloric muscle thickness and clinical and laboratory data revealed that there have been positive associations between pyloric muscle thickness and dehydration and lethargy with regression coefficients which were +0.22 and +0.50, respectively. Also, there were positive associations between increasing muscle thickness of the pylorus and vomiting duration for more than one week (+0.45), clinically palpable mass (+0.60), and all the studied laboratory factors: hyponatremia (+0.94), hypochloremia (+0.75), alkaline pH (+0.50), and high HCO_3 (+0.70).

Discussion

The diagnosis of pyloric stenosis (PS) can be suspected based on patient age at presentation and the characteristic of the vomiting which is non-bilious projectile vomiting. The affected infants may be dehydrated and/or lethargic. The presence of the classical hypochloremic metabolic alkalosis aids in the diagnosis, but it is not always present in PS [6–8]. Although

Table 3 Comparison of the studied infants by their pyloric muscle thickness and the studied laboratory data ($N=30$ infants)

Laboratory data	Mean $\pm$ SD	<i>P</i> value
Serum sodium level (Na^+)		
Normal	4.9 $\pm$ 0.5	0.04 ^a
High	5.2 $\pm$ 0.2	
Low	5.9 $\pm$ 1.5	
Serum potassium level (K^+)		
Normal	5.4 $\pm$ 1.2	0.65
High	4.9 $\pm$ 0.5	
Low	5.4 $\pm$ 0.8	
Serum chloride level (Cl^-)		
Normal	5.0 $\pm$ 0.6	0.002 ^a
Low	5.7 $\pm$ 1.4	
Serum bicarbonate (HCO_3)		
Normal	5.0 $\pm$ 0.5	0.04 ^a
High	5.6 $\pm$ 1.3	
Low	4.9 $\pm$ 0.6	
Blood pH		
Normal	5.0 $\pm$ 0.7	0.04 ^a
Alkaline	5.5 $\pm$ 1.3	

^a Significant

palpation of the pyloric mass (olive) is diagnostic, it may be not detected in 11 % to 51 % of cases [9, 10]. In this study, most of the studied infants were presented with some degree of dehydration (80 %) and lethargy (57 %), alkalemia (60 %), and hypochloremia (44 %). This study and most of the previous ones which studied the clinical and biochemical presentation of PS were retrospective. Nevertheless, there was a difference in the reported prevalence of the biochemical changes. Glatstein et al. [11] found hypochloremia in 23 % and alkalosis in 14.4 % of their patients, while Goh et al. [12] reported alkalemia in 63 % and hypochloremia in 46 % of 50 infants. Shaoul and his colleagues [13] noticed alkalosis only in 20 % of their patients. Papadakis and co-workers [14] noted only 12 % of their studied children having electrolyte abnormalities on admission. Researchers based on the understanding of the physiological response believed that the clinical and laboratory findings in PS related to the duration of symptoms.

Quinn et al. [15] found a statistically significant positive correlation between the duration of symptoms and the low level of chloride, and a positive correlation also has been found with the pH and bicarbonate levels, but was statistically insignificant. In the same line, Beasley and his associates [16] noticed patients with severe vomiting for more than 1 week and those with more than 5 % dehydration having metabolic abnormalities. Nmadu [17] found that the duration of presentation affects the laboratory abnormalities. He found that if the duration is 3 weeks or less the metabolic changes

were metabolic alkalosis and normal potassium, and if the duration is more than 3 weeks the changes were metabolic alkalosis and hypokalemia. Kildeberg [18] shares the same concept and reported that the presence of metabolic alkalosis implies the long duration of illness. Malcom et al. [19] found normal bicarbonate levels in eight non-consecutive cases despite all they had projectile vomiting and increased vomiting frequencies although the duration of history was not mentioned. In the same context, Aljazaeri et al. [5] showed that there was no relation between duration of vomiting and biochemical changes. More than that, they believed that the metabolic abnormalities are best related to the severity of pyloric stenosis rather than the duration of symptoms. In this study, however, we found a statistically significant relation between the duration of vomiting and muscle thickness. Moreover, we revealed a significant relation between muscle thickness and presence of dehydration, lethargy, and palpation pyloric mass. Based on the pathophysiology point of view, the severity of the pyloric stenosis is related to the muscle thickness; as the muscle thickness increases, the gastric outlet narrowed and, hence, the vomiting persists and the amount of vomitus will be more as minimal or no gastric content passes distally. Our findings support this theory. We have found that the pyloric muscle thickness was higher among infants with vomiting duration more than 1 week and in those with clinically palpable olive mass (P value 0.01 and 0.002, respectively). The mean muscle thickness was higher among dehydrated and lethargic infants. Regarding laboratory data, it has been found in this study that low levels of chloride, sodium, and alkaline pH were marked among those infants with higher mean of muscle thickness. These associations have been proved by the linear regression analysis which showed that the severity of the studied clinical and laboratory data was increased according to each unit increase in muscle thickness at the time of diagnosis.

This concept may also be supported by the finding of profound metabolic alkalosis in infants with vomiting due to PS in comparison to those with vomiting due to low obstruction or non-obstructive causes [20].

As it is a retrospective study, it has its limitations. Furthermore, the small number enrolled in the study makes it difficult to draw a rigid conclusion. However, the use of linear regression analysis in this small size study is an advantage to investigate the association of severity of clinical and laboratory with each unit increase in pyloric muscle. This study may encourage researchers to do further prospective study to evaluate these findings and may open the door to test the relation of the response rate to medical treatment with atropine sulfate and the muscle thickness in pyloric stenosis.

References

- O'Neill JA, Grosfeld JL, Fonkalsrud EW et al (eds) (2004) Principles of pediatric surgery (ed 2, chapter 45). Mosby, St. Louis, MO, p 467
- Safford SD, Pietrobon R, Safford KM et al (2005) A study of 11,003 patients with hypertrophic pyloric stenosis and the association between surgeon and hospital volume and outcomes. *J Pediatr Surg* 40: 967
- Schechter R, Torfs CP, Bateson TF (1997) The epidemiology of infantile hypertrophic pyloric stenosis. *Paediatr Perinat Epidemiol* 11:407
- Tigges CR, Bigham MT (2012) Hypertrophic pyloric stenosis: it can take your breath away. *Air Med J* 31(1):45–48. doi:10.1016/j.amj.2011.06.009
- Al-Jazaeri A, Al-Shehri A, Zamakhshary M, Al-Zahem A (2011) Can the duration of vomiting predict postoperative outcomes in hypertrophic pyloric stenosis? *Ann Saudi Med* 31(6):609
- Breaux CW Jr, Hood JS, Georgeson KE (1989) The significance of alkalosis and hypochloremia in hypertrophic pyloric stenosis. *J Pediatr Surg* 24:1250
- Hulka F, Campbell TJ, Campbell JR, Harrison MW (1997) Evolution in the recognition of infantile hypertrophic pyloric stenosis. *Pediatrics* 100:E9
- Chen EA, Luks FI, Gilchrist BF, Wesselhoeft CWI, DeLuca FG (1996) Pyloric stenosis in the age of ultrasonography: fading skills, better patients? *J Pediatr Surg* 31:829
- Freund H, Berlatzky Y, Katzenelson R, Schiller M (1976) Diagnosis of pyloric stenosis. *Lancet* 1(7982):473
- Macdessi J, Oates R (1993) Clinical diagnosis of pyloric stenosis: a declining art. *BMJ* 306:553
- Glatstein M, Carbell G, Boddu SK, Bernardini A, Scolnik D (2011) The changing clinical presentation of hypertrophic pyloric stenosis: the experience of a large, tertiary care pediatric hospital. *Clin Pediatr (Phila)* 50(3):192
- Goh DW, Hall SK, Gornall P, Buick RG, Green A, Corkery JJ (1990) Plasma chloride and alkalaemia in pyloric stenosis. *Br J Surg* 77(8): 922
- Shaoul R, Enav B, Steiner Z, Mogilner J, Jaffe M (2004) Clinical presentation of pyloric stenosis: the change is in our hands. *Isr Med Assoc J* 6(3):134
- Papadakis K, Chen EA, Luks FI, Lessin MS, Wesselhoeft CW Jr, DeLuca FG (1999) The changing presentation of pyloric stenosis. *Am J Emerg Med* 17(1):67
- Quinn N, Walls A, Milliken I, McCullagh M (2011) Pyloric stenosis — do males and females present differently? *Ulster Med J* 80(3):145
- Beasley SW, Hudson I, Yuen HP, Jones PG (1986) Influence of age, sex, duration of symptoms and dehydration of serum electrolytes in hypertrophic pyloric stenosis. *Aust Paediatr J* 22(3):193
- Nmadu PT (1992) Alterations in serum electrolytes in congenital hypertrophic pyloric stenosis: a study in Nigerian children. *Ann Trop Paediatr* 12(2):169
- Kildeberg P (1964) Metabolic alkalosis in hypertrophic pyloric stenosis: clinical significance and treatment. *Acta Paediatr* 53:132
- Malcom GE, Raio CC, Del Rios M, Blaivas M, Tsung JW (2009) Feasibility of emergency physician diagnosis of hypertrophic pyloric stenosis using point-of-care ultrasound: a multi-center case series. *J Emerg Med* 37(3):283
- Smith GA, Mihalove L, Shields BJ (1999) Diagnostic aids in the differentiation of pyloric stenosis from severe gastroesophageal reflux during early infancy: the utility of serum bicarbonate and serum chloride. *Am J Emerg Med* 17:28