Serum Electrolytes in Hypertrophic Pyloric Stenosis

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ABSTRACT

Hypochlorimic metabolic alkalosis is regarded as classical electrolyte abnormality occuring with hypertrophic pyloric stenosis (HPS) but atypical electrolyte findings frequently occur and delay establishing the proper diagnosis. A prospective study was performed to study the initial electrolyte changes in patients treated with pyloromyotomy for hypertrophic pyloric stenosis during seven years from 1980 to 1987. Fifty nine patients were divided into four study groups. Group A included one (2%) patient with serum bicarbonate (HCO₃) 17 mmol/L; in group B 20 (35%) with bicarbonate between 18 25 mmol/L(mean 23 ± 0.44); in group C 23 (39%) with bicarbonate between 25 and 30 mmol/L(mean 28 ± 0.36) and in group D 15 (26%) patients with bicarbonate over 30 mmol/L (36±1.36). Established value for normal HCO₃ in neonate is 20.1 ± 2.5 The mean values in group D $(mean \pm SD)$. $HCO_3(36\pm 1.36)$, for potassium (3.2 ± 0.27) , and chloride (79±3.38) mmol/L, were each significantly different (P>0.001) from determinations of similar electrolytes in other groups. The duration of vomiting in group D of 17.7 \pm 4.7 days is almost double the time (P>0.001) in group A and was associated with more dehydration. No significant differences in other demographic characteristics including the age at presentation, gestational age, sex distribution, or type of feeding used was observed. The results of the study emphasize that serum electrolytes in HPS may be normal that HCO3 is significantly lower than established normals for older children and the effects of hydrogen-ion loss elevating the serum HCO3 precedes alterations of other serum electrolytes.

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METHODS

Fifty-nine patients between 11 and 90 days of age who had Hypertrophic Pyloric Stenosis (HPS), were treated by pyloromyotomy at Salmaniya Medical Centre between April 1980 and June 1987 and were assigned to one of four groups determined by admission serum electrolytes value. The groups included patients with: bicarbonate below 18 mmol/L (group A), bicarbonate between 18 and 25 mmol/L (group B); bicarbonate between 25 and 30 mmol/L (group C) and those with bicarbonate above 30 mmol/L (group D). The demographic characteristics and clinical history were examined to determine whether or not any correlations could be made with the acid-base determinations in the four study groups.

The diagnosis of hypertrophic pyloric stenosis is made by the triad of projectile vomiting, visible peristalsis and palpable pyloric tumor or radiographic evidence of "string sign". Metabolic alkalosis occurs in patients with prolonged vomiting and is looked upon by many clinicians as a supportive evidence for HPS in case the physical signs are equivocal. If the baby has a normal or atypical acid-base balance the clinician might miss the diagnosis of HPS.

A prospective study of 59 patients with HPS was undertaken to determine the incidence of "classical" metabolic alkalosis with elevated serum bicarbonate, and depressed potassium and chloride in comparison to other serum electrolyte patterns.

RESULTS

Serum bicarbonate value ranged from 17 to 50 mmol/L. This includes one (2%) patient in group A. 20 (35%) patients in group B, 23 (39%) in group C and 15(26%) patients in group D. The mean serum bicarbonate in group D of 36±1.36(mean+SEM) was significantly higher than mean for other groups; likewise the serum chloride of 79+3.3 in group D was significantly lower than values in other groups

(P>0.001). Mean serum potassium values in group A, B and C ranged from 4.1 to 4.5 mmol/L and significantly higher(P>0.001) for the mean of 3.2 mmol/L for group D. Serum sodium determination showed no significant differences between groups (Table 1).

The demographic characteristics among the groups including the age at presentation, the sex distribution, gestational age and the type of feeding did not show any statistically significant differences (Table 2).

The duration of vomiting was 12 days in group C and 17.7 days in group D, almost double the time in group A and B (9.5 days). The age at presentation was 11 to 90 days with an average of 33 days and showed no statistical difference between groups. The patients in group A and B had mild dehydration while patients in group C and group D had moderate to severe dehydration (Table 3).

TABLE 1
Serum Electrolytes with HPS

Serum Electrolyte (mmol/L)								
Group		Na	K	Cl	HCO_3			
A	Bicarbonate < 18 (n=1)	138 ± 0.0	4.4 ± 0.0	106 ± 0.0	17 ± 0.0			
В	Bicarbonate 18 - 25 (n=20)	134.5 ± 1.0	4.5 ± 0.19	93.5 ± 2.53	23 ± 0.44			
С	Bicarbonate >25-30 (n=23)	137 ± 0.57	4.1 ± 0.17	91.5 ± 1.91	28.5 ± 0.36			
D	Bicarbonate >30 (n=15)	135 ± 3.12	3.3 ± 0.27	86.5 ± 3.3	36 ± 1.36			

TABLE 2
Demographic Characteristics with HPS

Group	Sex M/F	Gestation FT/Premature	Family History
A (n=1)	1/0	1/0	_
B (n=20)	15/5	20/0	_
C (n=23)	21/2	23/0	-
D (n=15)	7/8	15/0	-

TABLE 3
Clinical History with HPS

Group	Feeding Breast/ Formula	Vomiting Duration (days)	Age (Weeks)	Dehydration (<5;5-10;>10)
A (n=1)	1/0	5	5	1/0/0
B (n=20)	20/0	9.5	4.5	20/0/0
C (n=23)	22/1	12	5	17/6/0
D (n=15)	14/1	17.6	5	6/8/1

DISCUSSION

The wide spectrum of serum bicarbonate determination observed in our patients with HPS has been previously described by Touloukian¹. Its significance requires knowledge of baseline value for normal infants. There is no doubt that the serum bicarbonate and potassium for healthy neonates and infants are significantly different than older children and adults. Touloukian 1 reported a mean bicarbonate for normal infant in their hospital laboratory to be 21 mmol/L with a range of 18 to 25 mmol/L compared to a normal of 25 to 30 mmol/L for older children and adults. Meites 2 gives a mean bicarbonate of 20 mmol/L with a range of 17.2-23.6 mmol/L for infants. The serum bicarbonate in Thomas and Reichelderfer's 3 analysis of serum electrolytes in premature infants during the first 7 weeks of life was 20.6±3.1 (mean+SD). A mean bicarbonate value for normal infants in our hospital laboratory was estimated to be 21 with a normal range of 18-25 mmol/L compared to normal of 25 to 30 mmol/L for older children and adults. The low threshold for the renal tubular reabsorbtion of bicarbonate in neonates and infants might be the possible explanation for lower serum bicarbonate value 4.

The normal serum potassium for neonate is reported to range from 4.3 ± 0.7 to 5.7 ± 0.5 mmol/L (mean±SEM) ^{2. 3. 4}. In our hospital, the value of venous or arterial serum potassium ranges between 3.9 to 5.2 mmol/L. Heel-stick sampling, prolonged application of tourniquet and tube haemolysis of red blood cells can increase the serum potassium value.

The prolonged vomiting of large quantities of hydrochloric acid and smaller amounts of sodium and potassium in HPS produces metabolic alkalosis with an elevated serum bicarbonate and depressed potassium and chloride 5. Initially, the urine is alkaline but as potassium and bicarbonate stores become depleted from unreplaced gastric and renal losses, the kidney excretes an acid urine as the metabolic acidosis perpetuated. Additional potassium is lost secondary to dehydration with volume depletion and stimulation of aldosterone secretory mechanism. Group D in this series had the typical serum electrolytes changes of elevated serum bicarbonate and depressed potassium and chloride with occasional depressed sodium. This group had a significantly longer history of vomiting and more severe dehydration compared to babies in the other groups. The patients in group C had moderate elevation of serum bicarbonate but normal potassium and chloride indicating that hydrogen-ion loss from vomiting predominates in the earlier stages and precedes significant alterations in other electrolytes. One patient in group A with a bicarbonate of 17 mmol/L constitute groups of babies with HPS who have a short history of vomiting and otherwise normal serum electrolytes. The anion gap (serum sodium + potassium – chloride + bicarbonate) in group A is 19.4 which is within the normal anticipated range found in the other groups.

Eight (15%) patients in this series had normal serum electrolytes. Except for a shorter history of vomiting, there are no demographic characteristics to distinguish these patients from those with typical metabolic alkalosis.

CONCLUSION

We conclude our study by emphasizing the importance of a careful clinical examination for the diagnosis of hypertrophic pyloric stenosis. Contrast study is indicated in doubtful cases to provide an accurate diagnosis. Serum electrolytes can show atypical findings which might delay the diagnosis.

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