

Admission Electrolyte Derangement and Preoperative Delay in Infantile Hypertrophic Pyloric Stenosis: A Case–Control Study from Dhi Qar, Iraq

Akram Jooda Al Zaidy
College of Medicine, Al-Ayen Iraqi University, AUIQ, Thi-Qar, Iraq

* Corresponding Author: **Akram Jooda Al Zaidy**

Article Info

ISSN (online): 2582-8940
Volume: 07
Issue: 04
Received: 10-09-2026
Accepted: 08-10-2026
Published: 06-11-2026
Page No: 62-75

Abstract

Background: Pyloromyotomy for infantile hypertrophic pyloric stenosis (IHPS) is deferred until dehydration and hypochloraemic metabolic alkalosis have been corrected. Which admission findings predict a long correction phase is still uncertain, and data from Iraq are scarce.

Objectives: To compare the admission electrolyte and acid–base profile of infants with IHPS with that of healthy infants from the same hospitals, and to identify admission factors associated with the time needed to reach surgical readiness.

Methods: This prospective case–control study was carried out at Al-Nasiriyah Teaching Hospital, Al-Wilada (Maternity) Hospital and Al-Shatrah General Hospital, Dhi Qar Governorate, between 1 October 2025 and 1 March 2026. Twenty-five infants with sonographically confirmed IHPS and 25 healthy infants recruited at well-baby clinics were studied. Venous sodium, potassium, chloride, bicarbonate, pH, base excess, glucose and urea were measured at admission. In cases, the time to electrolyte correction, the admission-to-surgery interval and the length of stay were recorded. Spearman correlation, linear regression and receiver operating characteristic (ROC) analysis were used.

Results: Cases and controls were of similar age (47.2 ± 16.7 vs 51.1 ± 15.6 days) and weight. Infants with IHPS had lower sodium (132.9 ± 3.1 vs 139.7 ± 2.8 mmol/L), potassium (3.45 ± 0.34 vs 4.51 ± 0.48 mmol/L) and chloride (90.2 ± 3.7 vs 103.7 ± 2.4 mmol/L), and higher bicarbonate (32.6 ± 2.6 vs 24.3 ± 1.2 mmol/L), pH, base excess and urea (all $p < 0.001$). Every case was below the chloride target and above the bicarbonate target at admission. Correction took 33.0 ± 7.5 h, surgery followed 44.8 ± 8.7 h after admission, and 8 infants (32%) waited more than 48 h. The admission-to-surgery interval correlated with vomiting duration ($\rho = 0.71$), sodium ($\rho = -0.65$), bicarbonate ($\rho = 0.50$) and chloride ($\rho = -0.44$). Vomiting duration (area under the curve [AUC] 0.83) and sodium (AUC 0.81) discriminated a delay beyond 48 h; chloride did not (AUC 0.67). Once vomiting duration was taken into account, no electrolyte kept an independent association with delay.

Conclusions: Hypochloraemic alkalosis was universal in these infants, so chloride separated cases from controls but did not separate slow from fast correction. A low admission sodium and a long vomiting history identified infants likely to need more than two days of preparation. Earlier referral and a protocol-driven correction pathway are the most promising ways to shorten the preoperative stay in this setting.

DOI: https://doi.org/10.54660/IJMBHR.2026.7.4.62-75

Keywords: hypertrophic pyloric stenosis, hypochloraemia, metabolic alkalosis, hyponatraemia, pyloromyotomy, time-to-treatment, Iraq

1. Introduction

Infantile hypertrophic pyloric stenosis (IHPS) is the most frequent reason for abdominal surgery in the first months of life [1]. Boys are affected several times more often than girls, and population studies have linked the condition to bottle feeding, first-born status and caesarean delivery [2–4]. The operation itself, an extramucosal pyloromyotomy, is short and curative. What makes

the condition dangerous is the metabolic state in which many infants arrive, which is why IHPS is managed as a medical rather than a surgical emergency [1, 5, 6].

Repeated vomiting of gastric contents removes hydrogen, chloride, sodium, potassium and water. The result is the familiar picture of hypochloraemic, hypokalaemic metabolic alkalosis with a variable degree of dehydration [5, 7]. As volume falls, the kidney holds on to sodium at the cost of potassium and hydrogen ions, so the alkalosis is sustained and urine can become paradoxically acidic [5] (Figure 1). Alkalaemia also blunts the ventilatory drive. Perioperative apnoea and other respiratory events have been reported in infants operated on before the alkalosis was corrected [8, 9].

For these reasons, surgery is postponed until biochemical targets have been reached. Older physiological work suggested that a plasma chloride of at least 106 mmol/L reliably signalled correction of alkalaemia [10]. A Delphi panel later agreed on a more practical set of targets: pH ≤ 7.45, base excess ≤ 3.5, bicarbonate < 26 mmol/L, sodium ≥ 132 mmol/L, potassium ≥ 3.5 mmol/L and chloride ≥ 100 mmol/L [11]. Bicarbonate appears to matter in its own right; in one cohort, the risk of preoperative respiratory problems rose with every increment in serum bicarbonate [9]. The chloride load given during resuscitation determines how quickly bicarbonate falls [12].

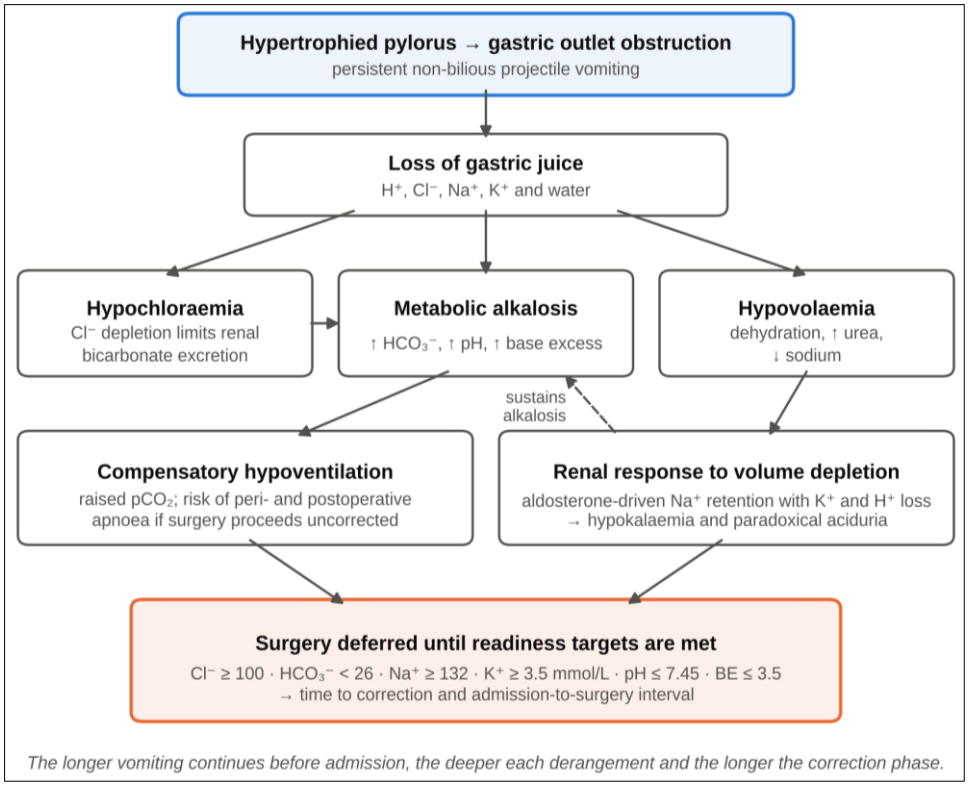


Fig 1: Mechanisms linking gastric outlet obstruction to the electrolyte and acid–base disturbances of IHPS and to the deferral of pyloromyotomy. The readiness targets shown are those of the Delphi consensus [11].

One study found that an infant's serum chloride level on admission predicted the number of saline boluses needed for stabilization; infants with significantly abnormal electrolyte levels stayed longer in the hospital [13]. A structured approach to resuscitation using algorithmic decision-making has been shown to decrease both the time to electrolyte correction and the length of stay [14] and protocol-driven care has been associated with a median time from presentation to definite surgery of less than one day [15]. In a large two-centre series, 75% of infants with malrotation were clinically stable enough for surgery at the time of diagnosis, but less than 50% went to surgery the same day, often because of the time of diagnosis [16]. Performing surgery the same day or at night has been shown to be safe in infants with normal electrolytes [17] and the placement of a gastrostomy tube at presentation has been shown to delay correction of the electrolytes [18]. Delays before definitive surgery can be broken into a physiologic component, dictated by the infant's deranged laboratory values, and an organisational component, dictated by the hospital's ability to arrange surgery in a timely manner.

Infants with long-standing vomiting have higher rates and more severe acid-base abnormalities with higher bicarbonate levels [19, 20]. Delayed presentation to care has also been reported [21, 22], although infants in many wealthier health systems are seen promptly and most require surgery before developing electrolyte abnormalities [13, 16, 25]. New data have identified pyloric measurements, as well as multivariable models as predictors of metabolic derangement and appropriate timing of surgery [23, 24]. In Iraq, the literature has focused almost exclusively on surgical techniques and postoperative care [26]. To our knowledge, no published data from the southern Iraq provide a comparison of the metabolic picture in IHPS patients compared to local healthy controls, or examine how metabolic factors affect wait time before surgery.

The present study had three aims: (1) to compare admission electrolyte and acid–base values between infants with IHPS and healthy infants from the same hospitals in Dhi Qar Governorate; (2) to measure how far infants with IHPS were from consensus readiness targets at admission; and (3) to

identify the admission factors associated with the time to electrolyte correction and the admission-to-surgery interval.

2. Materials and Methods

2.1. Study Design and Setting

This was a prospective analytical case–control study conducted between 1 October 2025 and 1 March 2026 in three public hospitals of Dhi Qar Governorate, southern Iraq: Al-Nasiriyah Teaching Hospital and Al-Wilada (Maternity) Hospital in Al-Nasiriyah city, and Al-Shatrah General Hospital in the north of the governorate. These hospitals receive most infants with surgical conditions from the city and the surrounding districts. The report follows the STROBE recommendations for observational studies [27]. The overall design is shown in Figure 2.

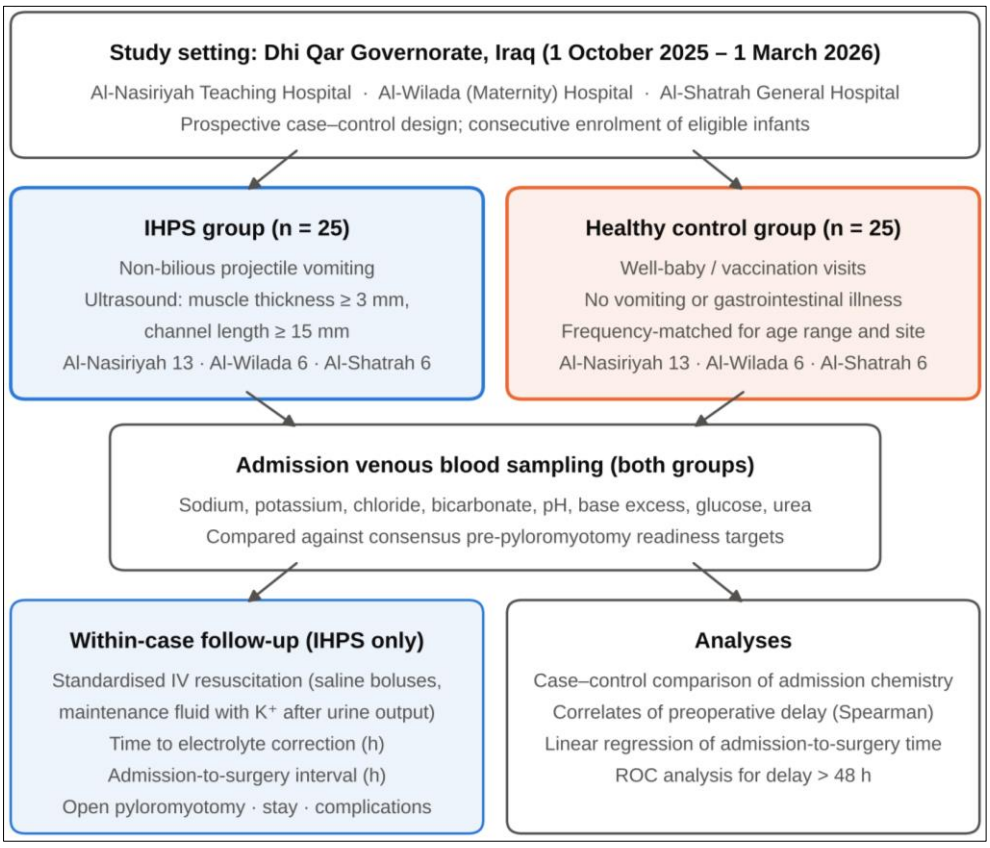


Fig 2: Study design. Cases and controls were recruited in equal numbers from each hospital; laboratory values of both groups were compared, and the preoperative course was followed in cases only.

2.2. Participants

Cases. All infants presenting during the study period with non-bilious projectile vomiting in whom ultrasonography confirmed IHPS were screened. The diagnosis required a pyloric muscle thickness of at least 3 mm and a pyloric channel length of at least 15 mm [28]. Infants were excluded if they had received intravenous fluids at another facility before admission, had a major congenital anomaly or known renal, cardiac or metabolic disease, or if a parent declined participation. Twenty-five infants were enrolled.

Controls. Twenty-five apparently healthy infants were recruited from the well-baby and vaccination clinics of the same hospitals during the same period. Eligible controls had no vomiting, diarrhoea, feeding difficulty or febrile illness in the preceding two weeks, no chronic illness and no previous hospital admission. Controls were frequency-matched to cases by hospital (the same number from each site) and by age range. A single venous sample was taken from each control after written parental consent.

2.3. Clinical Data

A structured form was completed at admission for every infant. It recorded age, sex, weight, feeding pattern (exclusive breastfeeding, formula feeding or mixed feeding), duration of vomiting before admission and the number of vomiting episodes per day. Dehydration was graded clinically by the admitting doctor as mild, moderate or severe. Pyloric muscle thickness and channel length were taken from the diagnostic ultrasound report.

2.4. Laboratory Measurements

Venous blood was drawn at admission, before any intravenous fluid was started. Blood gas analysis provided pH, bicarbonate and base excess. Serum sodium, potassium and chloride were measured by ion-selective electrode, and glucose and urea by routine enzymatic methods, in the central laboratory of each hospital. Glucose and urea are reported in mg/dL, the units used locally. Controls were sampled and analysed in the same way.

2.5. Preoperative Management and Outcome Definitions

Infants with IHPS were resuscitated according to departmental practice. Boluses of 0.9% saline (20 mL/kg) were given for moderate or severe dehydration or marked derangement, followed by maintenance fluid containing 5% dextrose and saline, with potassium chloride added once urine output was established. Electrolytes and blood gases

were repeated during resuscitation at the discretion of the treating team.

Readiness for surgery was judged against the consensus targets of van den Bunder *et al.* [11]: pH ≤ 7.45, base excess ≤ 3.5 mmol/L, bicarbonate < 26 mmol/L, sodium ≥ 132 mmol/L, potassium ≥ 3.5 mmol/L and chloride ≥ 100 mmol/L. The following outcomes were recorded:

- **Time to electrolyte correction:** hours from admission to the first laboratory set meeting all targets.
- **Admission-to-surgery interval:** hours from admission to the start of the operation. A prolonged preoperative delay was defined as an interval longer than 48 h.
- **Number of saline boluses** given before surgery.
- **Length of hospital stay:** days from admission to discharge.
- **Postoperative complications** until discharge, namely wound infection and persistent postoperative vomiting.

All operations were open Ramstedt pyloromyotomies performed under general endotracheal anaesthesia.

The number of the six laboratory targets not met at admission (0–6) was used as a simple index of derangement burden. Glucose (target ≥ 4.0 mmol/L, approximately 72 mg/dL) was reported separately.

2.6. Sample Size

All eligible infants with IHPS presenting during the five-month study window were enrolled. With 25 infants per group, the study had about 79% power to detect a standardised difference of 0.8 between groups (two-sided $\alpha = 0.05$). Within the case group, 25 infants gave 80% power to detect a correlation coefficient of 0.54 or greater.

2.7. Statistical Analysis

Continuous variables were checked for normality with the Shapiro–Wilk test. They are presented as mean ± standard deviation (SD) or as median and interquartile range (IQR). Groups were compared with Welch's t-test, which does not assume equal variances, or with the Mann–Whitney U test when data were skewed. Effect sizes are given as the mean difference with its 95% confidence interval (CI) and as Cohen's d. Categorical variables were compared with Fisher's exact test, and comparisons across three or more groups used the Kruskal–Wallis test.

Within the case group, associations between admission variables and the four outcomes were assessed with Spearman's rank correlation coefficient (ρ). To examine whether electrolyte values carried information beyond the duration of vomiting, partial Spearman correlations were calculated. Ordinary least-squares regression was then used to model the admission-to-surgery interval. Given the sample size, no model contained more than two predictors. Collinearity was checked with the variance inflation factor, and residual normality with the Shapiro–Wilk test.

The ability of admission variables to identify a delay longer than 48 h was assessed with ROC curves. The 95% CI of each area under the curve (AUC) was estimated from 2,000 bootstrap resamples, and the cut-off maximising Youden's index was reported with its sensitivity, specificity and predictive values. All tests were two-sided, with $p < 0.05$ taken as significant. No correction was made for multiple comparisons, and the within-case analyses are presented as exploratory. Analyses were run in Python 3 using SciPy (version 1.18) and scikit-learn (version 1.9).

2.8. Ethical Considerations

The study was approved by the [name of research ethics committee] (approval no. [number], dated [date]) and conducted in line with the Declaration of Helsinki. Written informed consent was obtained from a parent or legal guardian of every participant. Data were anonymised before analysis.

3. Results

3.1. Characteristics of the Participants

Fifty infants were studied, 25 with IHPS and 25 healthy controls. Each group contained 13 infants from Al-Nasiriyah Teaching Hospital and 6 each from Al-Wilada (Maternity) Hospital and Al-Shatrah General Hospital. The two groups were comparable in age and weight (Table 1). Boys made up 84% of cases, a male-to-female ratio of 5.25:1, compared with 52% of controls ($p = 0.032$). Two thirds of the infants with IHPS (17/25) had received formula, either alone or alongside breast milk, whereas every control was exclusively breastfed ($p < 0.001$).

Table 1: Demographic characteristics of infants with IHPS and healthy controls

Characteristic	IHPS (n = 25)	Controls (n = 25)	p value
Age (days), mean ± SD	47.2 ± 16.7	51.1 ± 15.6	0.396
Age (days), range	21–73	25–74	
Weight (kg), mean ± SD	4.09 ± 0.51	4.22 ± 0.56	0.385
Male sex, n (%)	21 (84)	13 (52)	0.032
Female sex, n (%)	4 (16)	12 (48)	
Exclusive breastfeeding, n (%)	8 (32)	25 (100)	<0.001[a]
Mixed feeding, n (%)	8 (32)	0 (0)	
Formula feeding, n (%)	9 (36)	0 (0)	
Al-Nasiriyah Teaching Hospital, n (%)	13 (52)	13 (52)	Matched
Al-Wilada (Maternity) Hospital, n (%)	6 (24)	6 (24)	Matched
Al-Shatrah General Hospital, n (%)	6 (24)	6 (24)	Matched

Continuous variables compared with Welch's t-test; categorical variables with Fisher's exact test. [a] Exclusive breastfeeding versus any formula exposure. Hospital distribution was fixed by the matching design.

3.2. Admission Electrolyte and Acid–base Status

Infants with IHPS differed sharply from controls in every electrolyte and acid–base variable except glucose (Table 2, Figure 3). Chloride was on average 13.5 mmol/L lower (95%

CI 11.7–15.3) and bicarbonate 8.3 mmol/L higher (95% CI 7.1–9.4); both effect sizes exceeded four standard deviations. The chloride values of the two groups did not overlap at all: the highest value among cases was 97.5 mmol/L and the lowest among controls 99.6 mmol/L. Urea was also higher in cases, by 13.3 mg/dL, in keeping with dehydration.

Table 2: Admission electrolyte, acid–base and metabolic values in infants with IHPS and healthy controls

Variable	IHPS (n = 25)	Controls (n = 25)	Mean difference (95% CI)	Cohen's d	p value
Sodium (mmol/L)	132.9 ± 3.1	139.7 ± 2.8	−6.84 (−8.51 to −5.18)	−2.34	<0.001
Potassium (mmol/L)	3.45 ± 0.34	4.51 ± 0.48	−1.06 (−1.30 to −0.83)	−2.56	<0.001
Chloride (mmol/L)	90.2 ± 3.7	103.7 ± 2.4	−13.48 (−15.28 to −11.69)	−4.28	<0.001
Bicarbonate (mmol/L)	32.6 ± 2.6	24.3 ± 1.2	8.26 (7.09 to 9.43)	4.04	<0.001
pH	7.493 ± 0.035	7.405 ± 0.021	0.088 (0.071 to 0.104)	3.05	<0.001
Base excess (mmol/L)	7.3 ± 2.2	0.1 ± 1.3	7.20 (6.18 to 8.23)	4.02	<0.001
Glucose (mg/dL)[a]	80.0 (72.6–91.3)	79.4 (75.4–93.3)	−0.55 (−7.67 to 6.56)	−0.04	0.698
Urea (mg/dL)	35.3 ± 9.2	21.9 ± 6.1	13.32 (8.88 to 17.77)	1.71	<0.001

Values are mean ± SD, compared with Welch's t-test. [a] Median (IQR), compared with the Mann–Whitney U test because of skewed distribution.

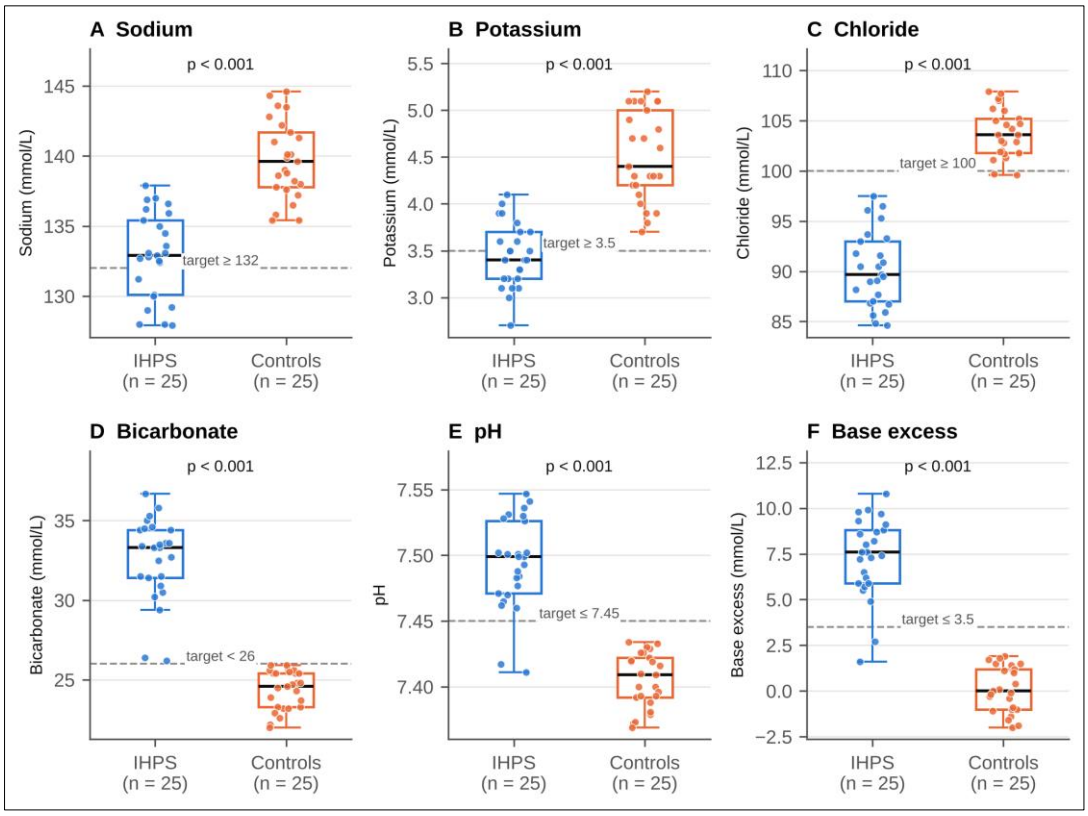


Fig 3: Admission sodium (A), potassium (B), chloride (C), bicarbonate (D), pH (E) and base excess (F) in infants with IHPS and healthy controls. Boxes show the median and interquartile range; whiskers extend to the most extreme values within 1.5 × IQR; each dot is one infant. Dashed lines mark the consensus pre-pyloromyotomy targets [11]. p values from Welch's t-test.

Measured against the consensus targets, every infant with IHPS had a chloride below 100 mmol/L and a bicarbonate of 26 mmol/L or more at admission. Twenty-three (92%) were above the pH and base-excess targets, 13 (52%) had potassium below 3.5 mmol/L and 8 (32%) had sodium below 132 mmol/L (Figure 4A). Thirteen infants (52%) showed the complete picture of hypochloraemia, hypokalaemia and alkalosis. Chloride fell below 90 mmol/L in 13 infants (52%) and below 85 mmol/L in 2 (8%). Most infants missed five or six of the six targets (16/25, 64%), and none met more than four. Among controls, only two infants had a chloride marginally below 100 mmol/L (99.6 and 99.7 mmol/L), and none fell outside any other target. A glucose below 72 mg/dL (4.0 mmol/L) was seen in 6 cases (24%) and 1 control (4%; p = 0.098).

3.3. Clinical Features and Preoperative Course of Infants with IHPS

Infants with IHPS had been vomiting for 11.5 ± 4.1 days before admission (median 13 days, range 3–17), with 7.5 ± 2.1 episodes per day (Table 3). Dehydration was judged severe in 11 infants (44%), moderate in 10 (40%) and mild in 4 (16%). The mean pyloric muscle thickness was 4.60 mm and the mean channel length 19.6 mm.

Correction of the laboratory values took 33.0 ± 7.5 h, and the operation began 44.8 ± 8.7 h after admission. No infant reached theatre within 24 h, 21 (84%) waited longer than 36 h and 8 (32%) longer than 48 h. Once targets had been met, a further 11.8 ± 3.7 h (range 6.4–17.7) passed before surgery, so about a quarter of the preoperative stay was spent waiting after biochemical readiness (Figure 5). Total stay was 4.03 ±

0.69 days and was closely tied to the preoperative interval (ρ = 0.74, p < 0.001). In a linear model, each additional hour before surgery added 0.062 days (about 1.5 h) to the total stay (95% CI 0.040–0.083; R^2 = 0.60).

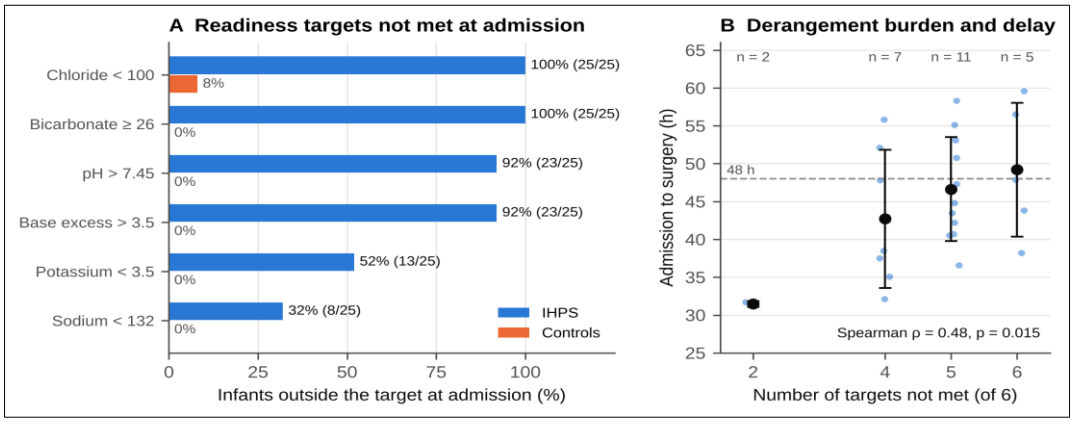


Fig 4: (A) Proportion of infants outside each consensus readiness target at admission. (B) Admission-to-surgery interval according to the number of targets not met at admission in infants with IHPS; black dots and bars show the group mean ± SD and pale dots individual infants. The dashed line marks 48 h.

Five infants (20%) had a postoperative complication: three wound infections (12%) and two episodes of persistent vomiting (8%). All 25 infants were discharged alive.

Table 3: Clinical features, ultrasound findings and perioperative course of infants with IHPS (n = 25)

Variable	Mean ± SD or n (%)	Median (IQR)	Range
Duration of vomiting (days)	11.5 ± 4.1	13.0 (9.0–15.0)	3–17
Vomiting episodes per day	7.5 ± 2.1	7.0 (6.0–9.0)	5–12
Non-bilious projectile vomiting	25 (100)		
Dehydration: mild	4 (16)		
Dehydration: moderate	10 (40)		
Dehydration: severe	11 (44)		
Pyloric muscle thickness (mm)	4.60 ± 0.64	4.60 (4.10–5.20)	3.3–5.6
Pyloric channel length (mm)	19.6 ± 2.0	19.9 (18.2–21.6)	15.8–22.5
Saline boluses: one	8 (32)		
Saline boluses: two	6 (24)		
Saline boluses: three	11 (44)		
Time to electrolyte correction (h)	33.0 ± 7.5	32.4 (29.2–40.1)	18.7–47.1
Correction to surgery (h)	11.8 ± 3.7	12.2 (8.6–15.2)	6.4–17.7
Admission to surgery (h)	44.8 ± 8.7	43.8 (38.2–52.1)	31.2–59.6
Admission to surgery > 48 h	8 (32)		
Length of hospital stay (days)	4.03 ± 0.69	4.10 (3.50–4.40)	2.9–5.3
Any postoperative complication	5 (20)		
Wound infection	3 (12)		
Persistent postoperative vomiting	2 (8)		

3.4. Duration of Vomiting and Severity of Derangement

The longer an infant had been vomiting, the more deranged the admission chemistry (Figure 6). Vomiting duration correlated with sodium (ρ = −0.69, p < 0.001), bicarbonate (ρ = 0.59, p = 0.002), potassium (ρ = −0.47, p = 0.018), pH (ρ = 0.46, p = 0.022), base excess (ρ = 0.45, p = 0.025) and chloride (ρ = −0.44, p = 0.030). It was not related to urea (ρ = 0.29, p = 0.163), pyloric muscle thickness (ρ = −0.17, p = 0.413) or channel length (ρ = 0.03, p = 0.889). Grouping infants by vomiting duration showed the same gradient, with the clearest steps in sodium, bicarbonate and the two time intervals (Table 4). Infants who had vomited for 15 days or more needed on average 15 h longer to correct, and 16 h longer to reach theatre, than those who had vomited for a week or less.

The clinical dehydration grade did not follow vomiting duration (p = 0.931), time to correction (p = 0.869) or the admission-to-surgery interval (p = 0.957). Potassium was the only laboratory value that differed by grade, falling from 3.77 ± 0.28 mmol/L in mild to 3.28 ± 0.32 mmol/L in severe dehydration (p = 0.042).

3.5. Admission Factors Associated with Preoperative Delay

Vomiting duration was the variable most strongly associated with both preoperative intervals (Table 5). Among the laboratory values, sodium had the strongest relation: lower sodium went with longer correction (ρ = −0.56, p = 0.004), a longer admission-to-surgery interval (ρ = −0.65, p < 0.001) and a longer stay (ρ = −0.58, p = 0.002). Bicarbonate correlated with both intervals (ρ = 0.49 and 0.50). Chloride correlated with the admission-to-surgery interval (ρ = −0.44, p = 0.030) but not significantly with time to correction (ρ = −0.39, p = 0.057) (Figure 7). Potassium was related only to time to correction. No admission variable, chloride included, correlated with the number of saline boluses. The

derangement-burden index also followed the delay (ρ = 0.48, p = 0.015; Figure 4B): infants missing two targets reached theatre after a mean of 31.5 h, against 49.2 h for those missing all six.

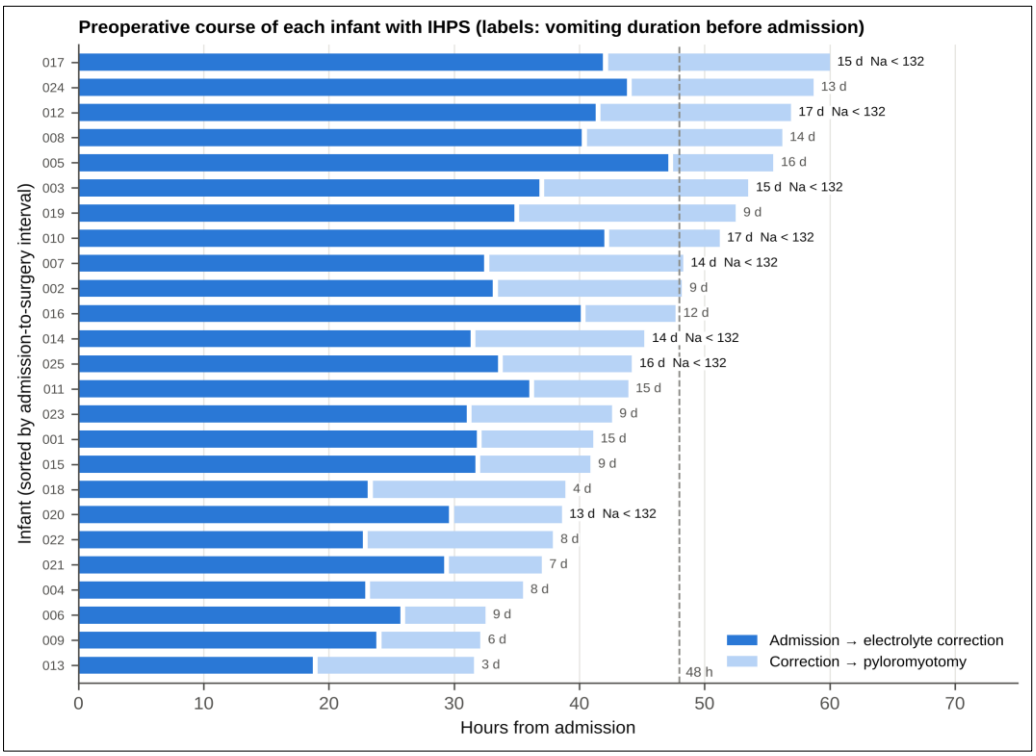


Fig 5: Preoperative course of each infant with IHPS, sorted by admission-to-surgery interval. Dark segments show the time from admission to electrolyte correction and pale segments the time from correction to pyloromyotomy. Labels give the duration of vomiting before admission and flag infants with an admission sodium below 132 mmol/L. The dashed line marks 48 h.

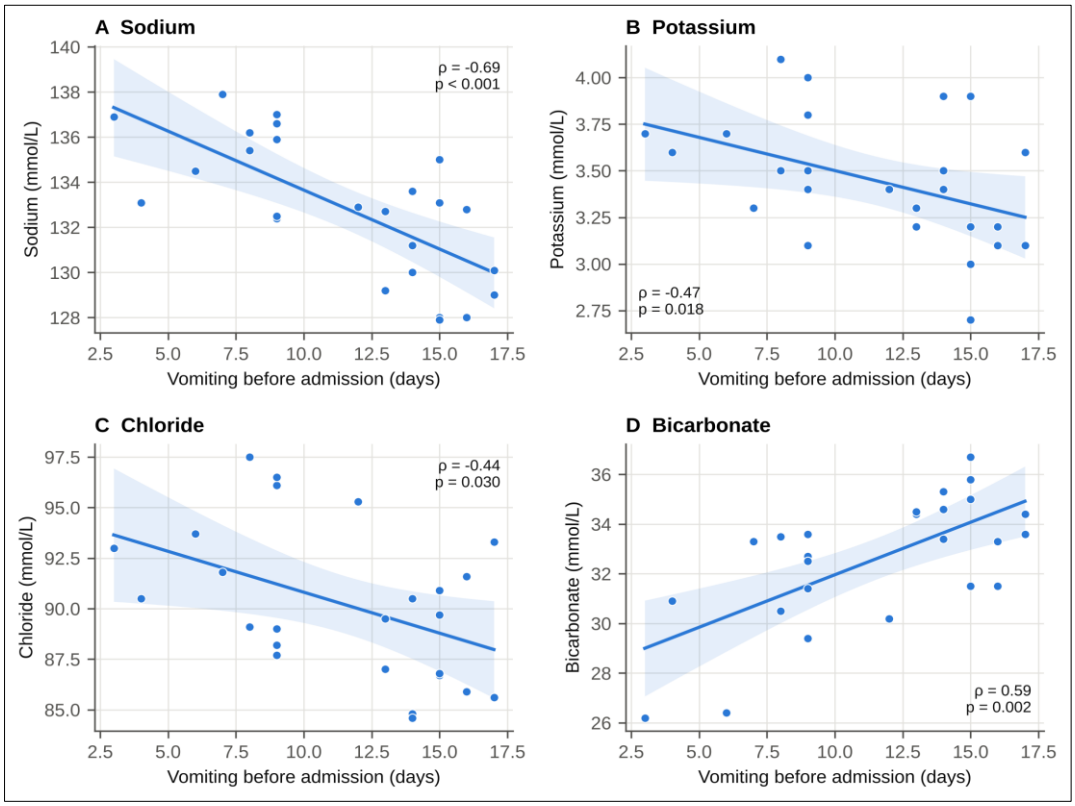


Fig 6: Duration of vomiting before admission plotted against admission sodium (A), potassium (B), chloride (C) and bicarbonate (D) in infants with IHPS (n = 25). Lines show the least-squares fit with its 95% confidence band; ρ is Spearman's rank correlation coefficient.

Table 4: Admission chemistry and preoperative intervals according to the duration of vomiting before admission

Variable	≤ 7 days (n = 4)	8–14 days (n = 13)	≥ 15 days (n = 8)	p (Kruskal–Wallis)	p for trend
Sodium (mmol/L)	135.6 ± 2.2	133.5 ± 2.6	130.5 ± 2.8	0.019	0.003
Potassium (mmol/L)	3.58 ± 0.19	3.55 ± 0.31	3.22 ± 0.37	0.069	0.024
Chloride (mmol/L)	92.3 ± 1.4	90.5 ± 4.5	88.8 ± 2.9	0.239	0.112
Bicarbonate (mmol/L)	29.2 ± 3.5	32.8 ± 1.9	34.0 ± 1.9	0.037	0.011
pH	7.46 ± 0.05	7.50 ± 0.03	7.51 ± 0.03	0.224	0.091
Base excess (mmol/L)	4.7 ± 3.2	7.6 ± 1.5	8.1 ± 1.9	0.134	0.080
Time to correction (h)	23.7 ± 4.3	32.3 ± 6.4	38.8 ± 5.1	0.003	<0.001
Admission to surgery (h)	34.5 ± 3.6	44.6 ± 8.0	50.4 ± 6.9	0.009	0.002
Hospital stay (days)	3.78 ± 0.78	3.85 ± 0.64	4.44 ± 0.62	0.194	0.107

Values are mean ± SD. p for trend from Spearman correlation between the ordered duration band and each variable.

Table 5: Spearman correlations between admission variables and preoperative and hospital outcomes in infants with IHPS (n = 25)

Admission variable	Time to correction	Admission to surgery	Saline boluses	Hospital stay
Vomiting duration (days)	**0.80 (<0.001)**	**0.71 (<0.001)**	0.00 (0.991)	**0.49 (0.014)**
Vomiting episodes per day	−0.22 (0.282)	−0.21 (0.312)	0.28 (0.177)	−0.13 (0.545)
Age at admission (days)	0.36 (0.081)	0.35 (0.083)	0.19 (0.364)	0.32 (0.114)
Weight (kg)	0.38 (0.063)	0.39 (0.051)	0.08 (0.720)	0.36 (0.073)
Sodium	**−0.56 (0.004)**	**−0.65 (<0.001)**	−0.14 (0.490)	**−0.58 (0.002)**
Potassium	**−0.43 (0.030)**	−0.32 (0.116)	0.25 (0.237)	−0.26 (0.211)
Chloride	−0.39 (0.057)	**−0.44 (0.030)**	−0.02 (0.914)	**−0.44 (0.029)**
Bicarbonate	**0.49 (0.013)**	**0.50 (0.011)**	0.07 (0.734)	0.36 (0.073)
pH	0.29 (0.153)	0.35 (0.087)	−0.01 (0.967)	**0.41 (0.040)**
Base excess	0.39 (0.051)	0.40 (0.050)	0.08 (0.703)	0.35 (0.091)
Urea	0.26 (0.214)	0.19 (0.355)	0.12 (0.563)	0.13 (0.528)
Glucose	0.31 (0.135)	0.36 (0.077)	−0.08 (0.686)	0.30 (0.138)
Pyloric muscle thickness	0.02 (0.921)	0.01 (0.961)	−0.27 (0.184)	0.02 (0.939)
Pyloric channel length	0.04 (0.844)	−0.02 (0.939)	0.10 (0.622)	−0.01 (0.970)

Values are Spearman's ρ (p value). Bold type marks p < 0.05.

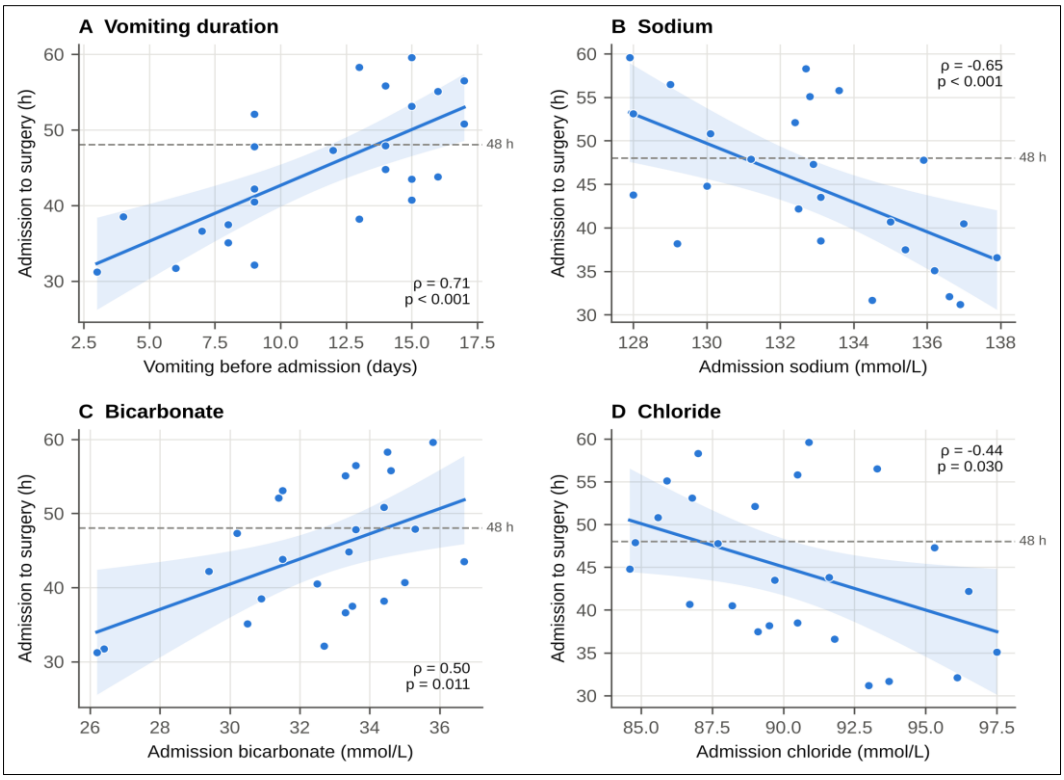


Fig 7: Admission-to-surgery interval plotted against vomiting duration (A), admission sodium (B), bicarbonate (C) and chloride (D) in infants with IHPS. Lines show the least-squares fit with its 95% confidence band; the dashed line marks 48 h.

Splitting the cohort at clinically meaningful values gave a consistent picture. Infants with bicarbonate ≥ 33 mmol/L (n = 14) took longer to correct (35.9 ± 6.9 vs 29.3 ± 6.8 h, p = 0.026) and to reach surgery (48.1 ± 8.1 vs 40.7 ± 7.9 h, p = 0.031) than those below that level. Hypokalaemic infants (n = 13) took longer to correct (36.1 ± 6.0 vs 29.6 ± 7.8 h, p = 0.030), although their admission-to-surgery interval was not significantly longer (46.9 ± 7.9 vs 42.5 ± 9.2 h, p = 0.216). A split at a chloride of 90 mmol/L produced no significant difference in either interval (admission to surgery 47.0 ± 6.7

vs 42.5 ± 10.2 h, p = 0.221). Infants with sodium below 132 mmol/L (n = 8) stayed longer in hospital (4.45 ± 0.52 vs 3.83 ± 0.68 days, p = 0.022), and their admission-to-surgery interval pointed the same way (49.3 ± 7.1 vs 42.7 ± 8.7 h, p = 0.059). Lower sodium was also associated with a longer wait between correction and surgery (ρ = −0.40, p = 0.045).

The eight infants who waited more than 48 h had vomited for longer, had lower sodium and were heavier than the other 17 (Table 6). Their chloride, bicarbonate, potassium and pH did not differ significantly. They stayed about one day longer in hospital, and none of the five complications occurred in this group.

Table 6: Comparison of infants with an admission-to-surgery interval above and below 48 h

Variable	> 48 h (n = 8)	≤ 48 h (n = 17)	p value
Age (days)	54.1 ± 8.7	43.9 ± 18.7	0.075
Weight (kg)	4.37 ± 0.22	3.96 ± 0.56	0.016
Vomiting duration (days)	14.5 ± 2.6	10.1 ± 4.0	0.003
Severe dehydration, n (%)	3 (38)	8 (47)	1.000
Sodium (mmol/L)	130.8 ± 2.3	133.8 ± 2.9	0.013
Sodium < 132 mmol/L, n (%)	4 (50)	4 (24)	0.359
Potassium (mmol/L)	3.35 ± 0.36	3.49 ± 0.33	0.359
Chloride (mmol/L)	88.6 ± 2.8	91.0 ± 4.0	0.105
Bicarbonate (mmol/L)	33.6 ± 1.5	32.1 ± 2.9	0.096
pH	7.50 ± 0.03	7.49 ± 0.04	0.377
Base excess (mmol/L)	8.0 ± 1.8	7.0 ± 2.4	0.226
Urea (mg/dL)	35.6 ± 8.5	35.1 ± 9.7	0.889
Saline boluses (n)	2.4 ± 0.9	2.0 ± 0.9	0.349
Time to correction (h)	41.0 ± 3.8	29.2 ± 5.6	<0.001
Hospital stay (days)	4.75 ± 0.41	3.69 ± 0.51	<0.001
Postoperative complication, n (%)	0 (0)	5 (29)	0.140

Values are mean ± SD (Welch's t-test) or n (%) (Fisher's exact test).

In single-predictor regression models, vomiting duration explained 49% of the variance in the admission-to-surgery interval, sodium 36%, bicarbonate 26% and chloride 19% (Table 7A). Sodium and bicarbonate both remained significant when entered together (adjusted R^2 0.45). Once vomiting duration was added to any electrolyte, however, it was the only significant predictor, with each additional day of vomiting adding roughly 1.2–1.5 h to the interval. Partial correlations adjusted for vomiting duration agreed: sodium (ρ = −0.32, p = 0.126), chloride (ρ = −0.20, p = 0.351), bicarbonate (ρ = 0.14, p = 0.508) and potassium (ρ = 0.02, p = 0.931) were no longer associated with the interval. Variance inflation factors were below 2.0 in every two-predictor model, and no model showed a significant departure of residuals from normality.

In ROC analysis, vomiting duration (AUC 0.83) and admission sodium (AUC 0.81) discriminated a delay beyond 48 h, whereas bicarbonate, chloride, base excess, potassium and pH did not (Table 7B, Figure 8). A vomiting history of 13 days or more, or a sodium of 132.8 mmol/L or less, each identified 7 of the 8 delayed infants. Both cut-offs had a negative predictive value of 92%.

Table 7: (A) Linear regression models of the admission-to-surgery interval (h). (B) ROC analysis of admission variables for an admission-to-surgery interval > 48 h

A. Regression Models

Model	Predictor	B (95% CI), h per unit	p value	R^2 (adjusted R^2)
1	Vomiting duration (days)	1.48 (0.83 to 2.12)	<0.001	0.49 (0.47)
2	Sodium (mmol/L)	−1.69 (−2.67 to −0.72)	0.002	0.36 (0.33)
3	Bicarbonate (mmol/L)	1.70 (0.48 to 2.92)	0.009	0.26 (0.23)
4	Chloride (mmol/L)	−1.01 (−1.91 to −0.11)	0.030	0.19 (0.16)
5	Sodium (mmol/L)	−1.40 (−2.33 to −0.48)	0.005	0.49 (0.45)
	Bicarbonate (mmol/L)	1.25 (0.16 to 2.33)	0.026	
6	Vomiting duration (days)	1.16 (0.25 to 2.07)	0.015	0.52 (0.47)
	Sodium (mmol/L)	−0.60 (−1.82 to 0.62)	0.319	
7	Vomiting duration (days)	1.33 (0.61 to 2.06)	0.001	0.51 (0.47)
	Chloride (mmol/L)	−0.36 (−1.16 to 0.45)	0.367	
8	Vomiting duration (days)	1.36 (0.47 to 2.24)	0.004	0.50 (0.45)
	Bicarbonate (mmol/L)	0.28 (−1.11 to 1.67)	0.678	

B, unstandardised regression coefficient; CI, confidence interval; AUC, area under the curve (95% CI from 2,000 bootstrap resamples); PPV and NPV, positive and negative predictive values. Cut-offs maximise Youden's index.

B. ROC Analysis (8 Delayed vs 17 Non-Delayed Infants)

Predictor	AUC (95% CI)	p value	Cut-off	Sensitivity	Specificity	PPV	NPV
Vomiting duration	0.83 (0.64–0.97)	0.009	≥ 13 days	88%	65%	54%	92%
Sodium (lower)	0.81 (0.62–0.96)	0.017	≤ 132.8 mmol/L	88%	71%	58%	92%
Bicarbonate	0.68 (0.44–0.88)	0.171	≥ 31.4 mmol/L	100%	35%	42%	100%
Chloride (lower)	0.67 (0.44–0.89)	0.180	≤ 90.9 mmol/L	88%	47%	44%	89%
Base excess	0.61 (0.36–0.83)	0.382	≥ 7.4 mmol/L	75%	53%	43%	82%
Potassium (lower)	0.59 (0.33–0.82)	0.483	≤ 3.6 mmol/L	88%	35%	39%	86%
pH	0.59 (0.32–0.83)	0.484	≥ 7.541	25%	100%	100%	74%

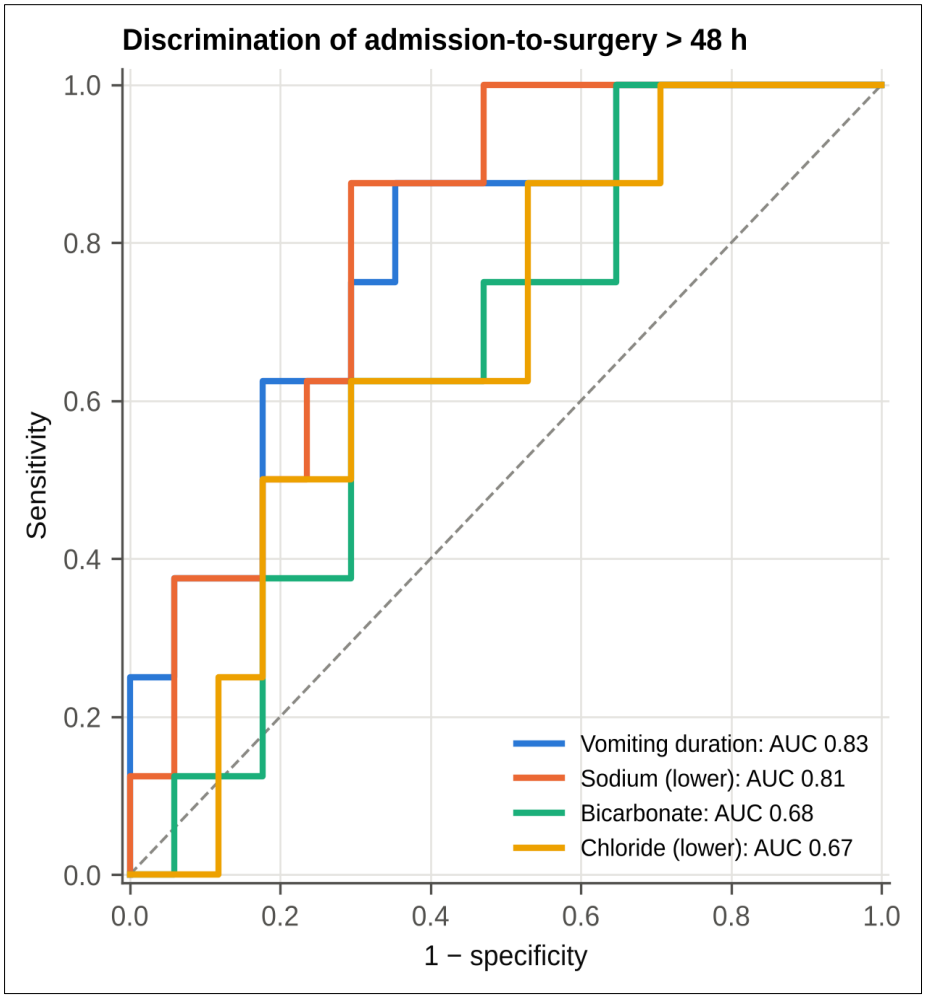


Fig 8: ROC curves of vomiting duration, sodium, bicarbonate and chloride for identifying an admission-to-surgery interval longer than 48 h. For sodium and chloride, lower values were treated as positive. The dashed diagonal marks chance discrimination.

3.6. Hospital, Feeding Pattern and Complications

The three hospitals did not differ in time to correction (p = 0.367), admission-to-surgery interval (44.5 ± 8.8 h at Al-Nasiriyah Teaching Hospital, 48.5 ± 10.0 h at Al-Wilada Hospital and 41.9 ± 7.1 h at Al-Shatrah General Hospital; p = 0.489), length of stay (p = 0.763) or admission chloride (p = 0.281). Within the case group, feeding pattern was unrelated to the admission-to-surgery interval (p = 0.874), chloride (p = 0.459) or vomiting duration (p = 0.996). Infants who developed a complication did not differ from the others in admission chloride (91.9 ± 3.6 vs 89.8 ± 3.8 mmol/L, p = 0.308), bicarbonate, potassium, sodium or admission-to-surgery interval (39.3 ± 5.5 vs 46.2 ± 8.9 h, p = 0.129).

4. Discussion

This multicentre study from southern Iraq shows that infants with IHPS arrived with a marked and almost uniform hypochloraemic metabolic alkalosis, far from the values used to judge fitness for anaesthesia. Correction took a day and a half on average, and surgery followed nearly two days after admission. Within the case group, the time to surgery depended less on how low the chloride was than on how long the infant had been vomiting, and the electrolyte most closely tied to delay was sodium.

4.1. A Late-Presenting Population

The metabolic and electrolyte abnormalities that characterized the infants in this study are striking, particularly when compared to cohorts from high-income nations. At diagnosis, Dalton *et al*. found normal electrolytes in 60% of the 505 infants included in their study of pyloric stenosis [13]. Furthermore, Wilhelm *et al*. reported that when diagnosed, 75% of their patients were already in suitable condition to undergo surgery [16]. In a study of 67 Korean patients, Kim found hyponatremia in just 7.5% and metabolic alkalosis in around half [23]. In our cohort, on the contrary, 92% were

alkalemic, 68% had a sodium level <135 mmol/L and none had target bicarbonate or chloride levels. These significant metabolic and electrolyte abnormalities are presumably due to the delayed presentation to medical care that characterizes our patient population. Indeed, vomiting had lasted for a median of 13 days by the time pyloric stenosis was diagnosed. This notion of delayed presentation is in keeping with a study on Chinese infants, which found similar rates of metabolic and electrolyte abnormalities, and a median duration of symptoms of 11 days before diagnosis of pyloric stenosis [19]. In other contexts, delayed presentation is attributed to poor access to health care [21, 22]. We suspect the reasons for delayed presentation of our patients may be multifactorial and include the distance many from the Dhi Qar region have to travel to tertiary referral centers, misdirection of evaluation of vomiting as being due to gastroesophageal reflux, or feeding intolerance, and delays in obtaining an ultrasound. Future studies should aim to prospectively elucidate the reasons for delayed presentation in our setting.

4.2. Why Chloride Did Not Predict Delay

Traditionally, chloride is used in IHPS as a marker of severity, as a lower chloride is associated with a greater need for saline [13] and resolution of the alkalemia after re-hydration is marked by a normalization of the chloride value [10]. Our study does not negate these principles, but demonstrates their limitations in a cohort of infants where hypochloratemia is ubiquitous. Indeed, every infant in our cohort was hypochloremic, and fell within a relatively narrow range of values (84.6 to 97.5 mmol/L) as compared to values in our control group. While chloride was an effective way of discriminating sick infants from healthy ones in this study, its ability to predict more rapid correction was negated by the compressed range of values in which it fell. It should be noted that Dalton's [13] study consisted largely of infants with normal baseline chemistries, which allowed a greater analytical range and thus the ability for the chloride to be a predictor of need for fluids. In our cohort, the volume of normal saline received was not associated with either the initial chloride value or the grade of clinical dehydration. This speaks to the fact that the decision the administration of fluid bolus inIHPS is made on a case by case basis and heavily depends on the astuteness and experience of the surgeon, and standardized protocols based on chloride measures were generated to address this surgeon dependent variability in management [13, 14].

4.3. Sodium and Vomiting Duration

Infants with low bicarbonate and those whose vomits have persisted longer are more likely to have hyponatraemia, reflecting a large deficit of total body sodium and water after prolonged losses, during which the kidney retains sodium at the expense of potassium and hydrogen ions [5]. Thus hyponatraemia should be corrected gradually to avoid rapid shifts in serum sodium and also because potassium can only be added once urine flow is established. Small changes in serum sodium can go a long way towards indicating delays.

Consistent with Kim's finding of lower sodium among infants with high bicarbonate [23], we found that sodium correlates inversely with vomiting duration ($\rho = -0.69$). In this sample, the AUC for sodium as a predictor of vomiting for longer than 48 hours was 0.81. Although this lagged behind chloride and bicarbonate, this electrolyte is one of the consensus targets [11] and no doubt has been attracted less attention as a predictor. Once vomiting duration was taken into account, sodium's link with delay vanished, in other words, low sodium best reflects the length of the illness, not that it contributes towards delay. Nonetheless it can still prove a valuable predictor. Hyponatraemia is obvious from the first blood sample and must be avoided no matter how long the parent reports his baby has vomited. In this sample, a sodium of 132.8 mEq/L or less identified seven of the eight infants who vomitted for longer than 48 hours, and a sodium above this threshold made such a protracted history unlikely (NPV 92%). Of course these cut-offs are derived from a small sample and need validation elsewhere before they can safely be used in clinical practice.

4.4. Bicarbonate and Potassium

Infants with an admission bicarbonate of above 33 mmol/L needed about 6.6 h longer to correct, and half of them were hypokalaemic, taking about 6.5 h longer to correct. Potassium is usually withheld until the infant passes urine and, once started, is limited by safe infusion concentrations, so correction can take some time.

Correction of metabolic alkalosis can be difficult, especially with a very high admission bicarbonate level. Miozzari *et al.* estimated that 10 mmol/kg of chloride will lower bicarbonate by about 3 mmol/L [12]. Our mean admission bicarbonate level was 32.6 mmol/L, and so to bring this down to around 26 mmol/L would require about 21 mmol/kg of chloride. With normal saline (0.9% saline) this would equate to about 140 mL/kg. This simple calculation may help to explain why it can take quite a long time to correct metabolic alkalosis, especially when bicarbonate levels are very high.

There is still much debate about the optimal target bicarbonate level. One study from the group that has reported the relationship between pre-operative bicarbonate, chloride and respiratory problems suggested a pre-operative bicarbonate cut-off of around 25.7 mmol/L might predict post-operative respiratory problems [9]. However, a later study from this group suggested that pre-operative pH, bicarbonate and chloride levels were not good predictors of peri-operative respiratory events [29].

4.5. The Organisational Part of Delay

Not all of the delay was biological. After the targets had been met, infants waited almost 12 more hours for surgery, about a quarter of the whole preoperative stay. This interval probably reflects theatre and anaesthetic availability and a reluctance to operate on infants at night. Elsewhere, postponing surgery to the next day after an afternoon diagnosis has been identified as a common and modifiable cause of longer stay [16], and operating on the same day, or after one night of fluids, was safe in infants with normal electrolytes [17]. Infants with lower sodium also waited longer after correction, perhaps because clinicians preferred a further check before anaesthesia.

The wider comparison is sobering. Our mean admission-to-surgery interval of 44.8 h is more than twice the median arrival-to-surgery time of 19.5 h reported under a resuscitation protocol in the United States, although that cohort also included infants without derangement [15]. Our mean correction time of 33 h is nearly three times the median of 11.9 h achieved among deranged infants after a fluid algorithm was introduced [14]. Because each extra hour before surgery added about 1.5 h to the total stay, a shorter

preoperative phase would translate almost directly into shorter admissions. Clinical pathways have reduced stay and charges by removing variation in practice [30], and chloride-guided bolus regimens reduce repeated blood sampling [13, 14]. We did not record whether gastric tubes were placed on admission. Since that practice has been associated with slower correction [18], it is worth examining locally.

4.6. Pyloric Dimensions, Complications, Sex and Feeding

Pyloric dimensions were unrelated to chemistry or delay. Kim found a strong correlation between channel length and bicarbonate, with a length above 23.5 mm identifying severe alkalosis [23], and a machine-learning study listed muscle thickness among the predictors of early surgery [24]. The narrow range of channel lengths in our infants (15.8–22.5 mm, none above 23.5 mm), measurement by different sonographers at three sites and the small sample may explain the difference. Chuang *et al*. reported that muscle thickness increased with symptom duration [20], which we did not observe.

Wound infection occurred in 12% of infants. This is well above the 1.2% rate of superficial infection reported in a large North American registry in which most operations were laparoscopic [31]. Differences in definitions, the open technique used in all our infants and the small denominator limit the comparison, but the figure justifies a local audit of antibiotic prophylaxis and wound care. Complications of pyloromyotomy depend on technique and setting as well as on the infant [32], and surgeon and hospital volume influence outcomes [33]. In our series, complications were not linked to admission chemistry, and none occurred in the delayed group. We therefore found no sign that waiting for correction caused harm, nor that it protected against it.

The male-to-female ratio of 5.25:1 fits the known excess risk in boys [2]. Formula exposure was common among cases and absent among controls, which is consistent with cohort and meta-analytic evidence linking bottle feeding to IHPS [2–4, 34]. The study was not designed to test that association, however. Controls were recruited at vaccination clinics, where exclusively breastfed infants may be over-represented, so the contrast should not be read as an estimate of effect.

4.7. Strengths and Limitations

The study was prospective and drew on three hospitals in a region that is rarely represented in the literature. Healthy local controls were sampled and analysed in the same way as cases, data were complete for every infant, and the consensus targets provided a common yardstick. The analysis also separated the biological and organisational components of delay.

Limitations include the following: The sample was small which limits the power of subgroup and multivariable analyses and it is likely that the ROC cut-offs derived from the same sample are optimistic. Many comparisons were made without correction, so the within-case findings should be viewed as exploratory. No screening log was kept and so it is hard to say with certainty if the most severely ill referrals were included or excluded. Indeed infants who had received IV fluids prior to referral were excluded. It is possible that the most severely ill referrals were under-represented in this study. Controls were frequency matched and so differed from cases in sex and pattern of feeding. The controls served as a biochemical reference only and were not individually matched. Dehydration was graded clinically, and is a subjective assessment. This may explain its weak correlation with electrolyte derangements. Venous rather than arterial blood gases were used. Only admission values were analysed. The volume of IV fluids and chloride received, urine output, repeat electrolytes, timing of diagnosis, use of a gastric tube, theatre availability, readmission rates and long-term complications were not documented. This limits the ability to comment on the role of biological and organisational factors in the observed derangements.

5. Conclusions

In Dhi Qar Governorate, infants with IHPS presented late, universally had metabolic alkaloses with low chloride, frequently having also low potassium and sodium levels, and spent on average almost two days admitted before pyloromyotomy. While the low admission chloride distinguished infants from healthy children, it was not associated with those who were quickest to become operative. A prolonged history of vomiting and low admission sodium level were the best early indicators of those children who would be delayed more than 48 hours. Earlier diagnosis and referral, a written clear chloride and bicarb driven resuscitation protocol and operating room scheduling so that surgery could be carried out as soon as the targets were met would probably reduce the inpatient stay. However, this needs to be tested by a bigger prospective study including more detailed information on management including the fluid and chloride given and the sequence of events.

Declarations

Ethics Approval and Consent to Participate

Approved by the ethics committee of AUIQ, approval no. (AUIQ 20261112). Written informed consent was obtained from a parent or legal guardian of every infant.

Consent for Publication

Not applicable; the manuscript contains no identifiable patient information.

Availability of Data

The anonymised dataset is available from the author on reasonable request.

Competing Interests

The author declares no competing interests.

Funding

The author received no specific funding for this work.

Author Contribution

The sole author conceived and designed the study, collected the data, performed the statistical analysis, interpreted the results, wrote the manuscript and approved the final version.

Acknowledgements

The author thanks the paediatric surgical, anaesthetic, nursing and laboratory staff of Al-Nasiriyah Teaching Hospital, Al-Wilada (Maternity) Hospital and Al-Shatrah General Hospital, and the parents who agreed to take part.

Use of AI-Assisted Tools

Artificial intelligence (AI)-assisted tools were used solely for language editing and proofreading to improve the clarity, grammar, and readability of the manuscript. AI was not used

for generating scientific content, data analysis, interpretation of results, or drawing conclusions. The author reviewed and approved all revisions and takes full responsibility for the final content of the manuscript.

References

1. Jobson M, Hall NJ. Contemporary management of pyloric stenosis. Semin Pediatr Surg. 2016;25(4):219–24.
2. Obaid YY, Toubasi AA, Albustanji FH, Al-Qawasmeh AR. Perinatal risk factors for infantile hypertrophic pyloric stenosis: a systematic review and meta-analysis. J Pediatr Surg. 2023;58(3):458–66. doi:10.1016/j.jpedsurg.2022.08.016
3. Krogh C, Biggar RJ, Fischer TK, Lindholm M, Wohlfahrt J, Melbye M. Bottle-feeding and the risk of pyloric stenosis. Pediatrics. 2012;130(4):e943–9. doi:10.1542/peds.2011-2785
4. Zhu J, Zhu T, Lin Z, Qu Y, Mu D. Perinatal risk factors for infantile hypertrophic pyloric stenosis: a meta-analysis. J Pediatr Surg. 2017;52(9):1389–97. doi:10.1016/j.jpedsurg.2017.02.017
5. Kamata M, Cartabuke RS, Tobias JD. Perioperative care of infants with pyloric stenosis. Paediatr Anaesth. 2015;25(12):1193–206. doi:10.1111/pan.12792
6. Aspelund G, Langer JC. Current management of hypertrophic pyloric stenosis. Semin Pediatr Surg. 2007;16(1):27–33.
7. Touloukian RJ, Higgins E. The spectrum of serum electrolytes in hypertrophic pyloric stenosis. J Pediatr Surg. 1983;18(4):394–7.
8. van den Bunder FAIM, van Wijk L, van Woensel JBM, Stevens MF, van Heurn LWE, Derikx JPM. Perioperative apnea in infants with hypertrophic pyloric stenosis: a systematic review. Paediatr Anaesth. 2020;30(7):749–58. doi:10.1111/pan.13879
9. van den Bunder FAIM, van Woensel JBM, Stevens MF, van de Brug T, van Heurn LWE, Derikx JPM. Respiratory problems owing to severe metabolic alkalosis in infants presenting with hypertrophic pyloric stenosis. J Pediatr Surg. 2020;55:2772–6. doi:10.1016/j.jpedsurg.2020.05.041
10. Goh DW, Hall SK, Gornall P, Buick RG, Green A, Corkery JJ. Plasma chloride and alkalaemia in pyloric stenosis. Br J Surg. 1990;77(8):922–3. doi:10.1002/bjs.1800770827
11. van den Bunder FAIM, Hall NJ, van Heurn LWE, Derikx JPM. A Delphi analysis to reach consensus on preoperative care in infants with hypertrophic pyloric stenosis. Eur J Pediatr Surg. 2020;30:497–504. doi:10.1055/s-0039-3401987
12. Miozzari HH, Tönz M, von Vigier RO, Bianchetti MG. Fluid resuscitation in infantile hypertrophic pyloric stenosis. Acta Paediatr. 2001;90(5):511–4. doi:10.1111/j.1651-2227.2001.tb00790.x
13. Dalton BG, Gonzalez KW, Boda SR, Thomas PG, Sherman AK, St Peter SD. Optimizing fluid resuscitation in hypertrophic pyloric stenosis. J Pediatr Surg. 2016;51(8):1279–82. doi:10.1016/j.jpedsurg.2016.01.013
14. Fraser JA, Osuchukwu O, Briggs KB, *et al*. Evaluation of a fluid resuscitation protocol for patients with hypertrophic pyloric stenosis. J Pediatr Surg. 2022;57(10):386–9. doi:10.1016/j.jpedsurg.2021.10.052
15. Cruz-Centeno N, Fraser JA, Stewart S, *et al*. Hypertrophic pyloric stenosis protocol: a single center study. Am Surg. 2023;89(12):5697–701. doi:10.1177/00031348231175126
16. Wilhelm S, Studzinski D, Alslaim H, *et al*. Optimizing throughput of babies with infantile hypertrophic pyloric stenosis. Am J Surg. 2024;230:68–72. doi:10.1016/j.amjsurg.2024.01.013
17. Longi FN, Reiter AJ, Patel S, *et al*. Timing of pediatric pyloromyotomy on hospital length of stay. Surg Pract Sci. 2023;13:100177. doi:10.1016/j.sipas.2023.100177
18. Lee LK, Burns RA, Dhamrait RS, *et al*. Retrospective cohort study on the optimal timing of orogastric tube/nasogastric tube insertion in infants with pyloric stenosis. Anesth Analg. 2019;129(4):1079–86. doi:10.1213/ANE.0000000000003805
19. Feng Z, Nie Y, Zhang Y, *et al*. The clinical features of infantile hypertrophic pyloric stenosis in Chinese Han population: analysis from 1998 to 2010. PLoS One. 2014;9(2):e88925. doi:10.1371/journal.pone.0088925
20. Chuang YH, Chao HC, Yeh HY, Lai MW, Chen CC. Factors associated with pyloric hypertrophy severity and post-operative feeding and nutritional recovery in infantile hypertrophic pyloric stenosis. Biomed J. 2022;45(6):948–56. doi:10.1016/j.bj.2021.12.011
21. Reich DA, Giraldo G, Canty W, *et al*. Disparities in insurance status negatively affect patients with infantile hypertrophic pyloric stenosis. Pediatr Surg Int. 2024;40(1):127.
22. Feliz A, Holub JL, Azarakhsh N, Bachier-Rodriguez M, Savoie KB. Health disparities in infants with hypertrophic pyloric stenosis. Am J Surg. 2017;214(2):329–35. doi:10.1016/j.amjsurg.2016.07.009
23. Kim HJ. Utility of pyloric length measurement for detecting severe metabolic alkalosis in infants with hypertrophic pyloric stenosis. Pediatr Gastroenterol Hepatol Nutr. 2024;27(2):88–94. doi:10.5223/pghn.2024.27.2.88
24. Genisol I, Boztas AE, Koska IO, *et al*. Machine learning-based prediction of surgical timing and discharge in infantile hypertrophic pyloric stenosis: a multimodal predictive approach. Pediatr Surg Int. 2026;42:161.
25. Tutay GJ, Capraro G, Spirko B, Garb J, Smithline H. Electrolyte profile of pediatric patients with hypertrophic pyloric stenosis. Pediatr Emerg Care. 2013;29(4):465–8.
26. Alhassani AA. Infantile hypertrophic pyloric stenosis: postoperative management without prolonged fasting or nasogastric tube. Bas J Surg. 2015;21:50–4.
27. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Lancet. 2007;370(9596):1453–7.
28. Hernanz-Schulman M. Infantile hypertrophic pyloric stenosis. Radiology. 2003;227(2):319–31.
29. van den Bunder FAIM, Stevens MF, van Woensel JBM, van de Brug T, Derikx JPM, van Heurn LWE. Perioperative hypoxemia and postoperative respiratory events in infants with hypertrophic pyloric stenosis. Eur J Pediatr Surg. 2023;33(6):485–92. doi:10.1055/a-1984-9803
30. Michalsky MP, Pratt D, Caniano DA, Teich S. Streamlining the care of patients with hypertrophic

pyloric stenosis: application of a clinical pathway. J Pediatr Surg. 2002;37(7):1072–5. doi:10.1053/jpsu.2002.33847
31. Parikh RM, Ata A, Edwards MJ. A contemporary review of surgical approach and outcomes in pediatric hypertrophic pyloric stenosis. J Surg Res. 2023;285:142–9. doi:10.1016/j.jss.2022.12.034
32. Kelay A, Hall NJ. Perioperative complications of surgery for hypertrophic pyloric stenosis. Eur J Pediatr Surg. 2018;28(2):171–5.
33. Safford SD, Pietrobon R, Safford KM, Martins H, Skinner MA, Rice HE. A study of 11,003 patients with hypertrophic pyloric stenosis and the association between surgeon and hospital volume and outcomes. J Pediatr Surg. 2005;40(6):967–72. doi:10.1016/j.jpedsurg.2005.03.011
34. McAteer JP, Ledbetter DJ, Goldin AB. Role of bottle feeding in the etiology of hypertrophic pyloric stenosis. JAMA Pediatr. 2013;167(12):1143–9.

How to Cite This Article
Al Zaidy AJ. Admission electrolyte derangement and preoperative delay in Infantile hypertrophic pyloric stenosis: a Case–Control study from Dhi Qar, Iraq. International Journal of Medical and All Body Health Research. 2026;7(4):62-75. doi:10.54660/IJMBHR.2026.7.4.62-75.

Creative Commons (CC) License
This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution - NonCommercial -ShareAlike 4.0 International (CC BY -NC - SA 4.0) License, which allows others to remix, tweak, and build upon the work non -commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.