Serum phosphate profile during inpatient treatment of children with severe acute malnutrition aged 6 months to 5 years

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ABSTRACT

Background: Hypophosphatemia is a recognized complication during the treatment of severe acute malnutrition (SAM) in children and may contribute to refeeding syndrome. Data on the longitudinal profile of serum phosphate using locally prepared World Health Organization (WHO)-recommended therapeutic feeds in India remain limited.

Objective: To describe serum phosphate dynamics and the incidence and correlates of hypophosphatemia in children aged 6-59 months hospitalized with complicated SAM.

Methods: A prospective observational study was conducted from January to December 2024 at a tertiary paediatric department in New Delhi, India. Sixty children (6-59 months) with SAM (WHZ/L ≤3 SD, mid-upper arm circumference <11.5 cm, and/or bilateral oedema), and medical complications were enrolled. Serum inorganic phosphate, sodium, potassium, and calcium were measured at admission, day 4 (end of stabilization phase), and at discharge (only if hypophosphatemia persisted on day 4).

Results: At admission, 23/60 children (38.3%) had hypophosphatemia. Median (interquartile ranges) serum phosphate was 3.81 (0.20) mg/dl. By day 4, the prevalence decreased significantly to 8/60 (13.3%; $p = 0.001$), and at discharge, no child remained hypophosphatemic (0%). Mean serum phosphate rose from 3.68 ± 0.49 mg/dl at admission to 4.51 ± 0.62 mg/dl at discharge ($p < 0.001$).

Conclusion: Hypophosphatemia is common at admission (38.3%) in children with complicated SAM but resolves effectively with standard WHO-protocol stabilization using locally prepared F-75 formula without additional phosphate supplementation. Hypophosphatemia is strongly linked to concurrent disturbances in sodium, potassium, and calcium, highlighting the importance of holistic electrolyte monitoring during early refeeding.

Keywords: f-75, hypophosphatemia, MUAC, refeeding syndrome, severe acute malnutrition.

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Introduction

Malnutrition in children is a major global health problem associated with childhood morbidity and mortality, impaired intellectual development, and increased likelihood of diseases in adulthood [1]. According to the World Health Organization (WHO), three broad groups of malnutrition are undernutrition [wasting (low weight-for-height), stunting (low height-for-age), and underweight (low weight-for-age)]; micronutrient-related malnutrition (lack or excess of important vitamins and minerals), and finally, overweight [obesity and diet-related non-communicable diseases (such as heart disease, stroke, diabetes, and some cancers)] [2,3].

According to WHO and United Nations International Children's Emergency Fund (UNICEF), severe acute malnutrition (SAM) in children aged 6 months to 5 years of age is diagnosed based on the any one of the three criteria; that is, weight-for-height/length less than three standard deviation (≤3 SD) on WHO Growth Standards OR presence of bipedal oedema (excluding non-nutritional causes) OR mid-upper arm circumference (MUAC) below 11.5 cm [4,5]. Malnutrition in India is a major concern, as some of the highest rates of childhood malnutrition and mortality in the under-5 age group are seen here [5,6]. According to the National Family Health Survey five (NFHS-5) (2019-21) [7], nearly one-third (32.10%) of the children under 5 years of age are underweight (low weight-for-age), 35.5% stunted (low height-for-age), and nearly one-fifth (19.30%) are wasted (low weight-for-height). Among those with wasting, 7.70% of these

children are severely wasted (weight-for-height/length ≤3 SD). Since "wasting" denotes acute malnutrition, these children are said to have SAM [7].

Treatment of children with SAM is divided into three phases: stabilization, transition, and rehabilitation. The goal is first to stabilize, treat life-threatening complications, and then to feed the child intensively to allow for catch-up growth [8,9]. Deficiencies of phosphorus, potassium, calcium, and other minerals are common in children with SAM. While treating these deficiencies, refeeding with diets high in carbohydrate can result in refeeding syndrome, characterized by hypophosphatemia and hypomagnesemia, sometimes resulting in respiratory or circulatory failure or even death. Although concentrations of serum phosphate and magnesium may not adequately reflect body status, low levels may still be suggestive of inadequate intake [9].

In starvation, the secretion of insulin is decreased in response to a reduced intake of carbohydrates. Instead, fat and protein stores are catabolized to produce energy. This results in an intracellular loss of electrolytes, in particular phosphate. When feeding is started (usually on day 4), a sudden shift from fat to carbohydrate metabolism occurs, and secretion of insulin increases. This stimulates cellular uptake of phosphate, which can lead to profound hypophosphatemia. Serum phosphate concentrations of less than 3.7 mg/dl can produce the clinical features of refeeding syndrome, including rhabdomyolysis, leukocyte dysfunction, respiratory failure, cardiac failure, hypotension, arrhythmias, seizures, coma, and sometimes sudden death [10].

Overall, India has the highest prevalence of SAM as per NFHS-5 [7], with a higher burden of SAM in the study area. SAM is a clinical syndrome due to an imbalance between the demand and supply of energy content, proteins, and micronutrients, with a complex interplay of various pathological mechanisms. Children with SAM have increased total body water and sodium, while there is a deficiency of potassium, magnesium, and phosphate stores [11].

As per WHO guidelines, SAM children are treated with a starter F-75 diet (containing 75 calories) initially for 3 to 7 days and later with a catch-up F-100 diet (containing 100 calories) from locally available ingredients containing 300 and 900 ml of skimmed milk per 1,000 ml of F-75 and F-100 diets, respectively. During refeeding due to anabolism and insulin release, increased cellular uptake of electrolytes like phosphorus, potassium, and magnesium occurs. This leads to hypophosphatemia, hypokalaemia, and hypomagnesemia with resultant increased mortality and morbidity. Clinical features of these biochemical changes are non-specific and may be misinterpreted as sepsis. Serum electrolytes are not routinely monitored; hence, adequate content of these micronutrients in the diet is essential to prevent depletion. Potassium and magnesium supplements are routinely supplemented, while diet is the main source for phosphorus [12,13].

However, locally prepared starter diet may have limited amounts of phosphorus during stabilization as compared to 60 mg/kg/day recommended by WHO, and is likely to be less effective in normalizing phosphate levels, leading to adverse outcomes like increasing mortality, inadequate weight gain, prolongation of hospital stay, and other long-term morbidities [14]. The present study was planned to determine the serum phosphate profile in children admitted with SAM through the identification of changes in serum phosphorus levels, incidence of hypophosphatemia, and differences in the clinical profile.

Subjects and Methods

This hospital-based prospective observational study was conducted over 12 months from January to December 2024 in the Department of Paediatrics at a tertiary-care teaching hospital in New Delhi, India. Ethical approval was obtained from the Institutional Ethics Committee before study initiation.

Sample size was calculated using the formula for paired mean difference: $n = (Z\alpha/2 + Z1\text{-}\beta)^2 \times SD^2 / d^2$, where $Z\alpha/2 = 1.96$ ($\alpha = 0.05$, 95% CI), $Z1\text{-}\beta = 1.28$ (power 90%), SD = 0.2 mg/dl (expected standard deviation of change in serum phosphate from admission to end of stabilization phase), and $d = 0.1$ mg/dl (clinically meaningful difference). This yielded a minimum required sample of 42 participants. Anticipating a 20% attrition/drop-out rate, the target sample size was increased to 53. Ultimately, 60 children who met the eligibility criteria were enrolled.

Children aged 6-59 months admitted with SAM along with an acute medical illness were eligible. SAM was diagnosed according to WHO/UNICEF criteria: weight-for-height/length ≤SD of WHO growth standards, MUAC < 11.5 cm, or bilateral nutritional pitting oedema [4,5]. Children with known chronic illnesses (congenital malformations, rickets, chronic kidney disease, endocrine disorders, malabsorption syndromes, tuberculosis, or HIV) were excluded. After screening, parents or legal guardians of eligible children were provided with detailed information about the study in their preferred language. Written informed consent was obtained before enrolment. A structured, pretested proforma was used to record demographic details (age, sex, socioeconomic status using Modified B.G. Prasad Classification 2024 update [18]), dietary history, immunization status, and clinical findings. Anthropometric measurements (weight, length/height, MUAC) were performed by trained staff using standardized techniques and equipment calibrated daily. All children were managed as per the WHO 2013 guidelines for inpatient treatment of complicated SAM [8,9]. Initial stabilization was carried out using a locally prepared F-75 formula, followed by transition to F-100 during the rehabilitation phase. Routine potassium and magnesium

supplementation was provided; additional electrolyte correction was performed when clinically indicated.

Laboratory investigations Venous blood samples (1 ml each) were collected under strict aseptic precautions in plain vacutainers at three time points: 1) At admission (before initiation of therapeutic feeding), 2) On day 4 (±12 hours) of the stabilization phase, 3) At discharge (only in children with hypophosphataemia on day 4). An additional 2-ml blood sample in an Ethylene-diamine-tetraacetic acid vacutainer was collected at admission for complete blood count. Serum inorganic phosphate, potassium, sodium, and total calcium were estimated by an automated analyzer (cobas integra 400 plus/equivalent) using standard reagents. Hypophosphataemia was defined using age-specific reference ranges: 6-36 months: serum phosphate < 3.8 mg/dl, 37-60 months: serum phosphate < 3.7 mg/dl. Anemia was defined as hemoglobin < 11 g/dl, and sepsis as systemic inflammatory response syndrome with suspected or proven infection.

Statistical analysis

Data were entered into Microsoft Excel and analyzed using IBM SPSS version 26.0. Categorical variables are presented as frequencies and percentages; continuous variables as mean ± SD or median interquartile ranges (IQR), depending on distribution. Sample size estimation was performed using the paired mean difference approach based on anticipated changes in serum phosphate from admission to stabilization, as distribution characteristics were not known during study planning. Normality was assessed using the Kolmogorov-Smirnov test. As the collected data demonstrated non-normal distribution, non-parametric methods were used where appropriate. Paired changes in serum phosphate and weight were analyzed using a paired *t*-test (normally distributed data) or Wilcoxon signed-rank test (non-normal data). Between-group comparisons were performed using an independent *t*-test/Mann-Whitney *U* test or χ^2/Fisher's exact test as appropriate. A two-tailed *p*-value ≤0.05 was considered statistically significant.

Results

A total of 60 children with complicated SAM were enrolled.

All continuous variables demonstrated non-normal distribution on Kolmogorov-Smirnov testing; therefore, medians with IQR were used for descriptive purposes, and non-parametric tests were applied for comparisons.

The baseline characteristics of the study population are presented in Table 1. The cohort comprised 27 boys

Table 1. *Distribution of children according to demographic and clinical characteristics and their association with serum phosphorus status.*

Parameter	Subgroup	Normal phosphorous (*n* = 37) No. (%)	Low phosphorous (*n* = 23) No. (%)	Total (*n* = 60) No. (%)	*p* value
Age group (months)	6-12	13 (56.52)	10 (43.48)	23 (38.33)	0.846
	13-24	12 (63.16)	7 (36.84)	19 (31.67)	
	25-36	2 (50.00)	2 (50.00)	4 (6.67)	
	37-48	3 (60.00)	2 (40.00)	5 (8.33)	
	49-60	7 (77.78)	2 (22.22)	9 (15.00)	
Sex	Boys	19 (70.37)	8 (29.63)	27 (45.00)	0.287
	Girls	18 (54.55)	15 (45.45)	33 (55.00)	
Socio-economic class	Class 3	8 (66.67)	4 (33.33)	12 (20.00)	0.215
	Class 4	22 (55.00)	18 (45.00)	40 (66.67)	
	Class 5	7 (87.50)	1 (12.50)	8 (13.33)	
Clinical profile	Immunization up-to-date	28 (60.87)	18 (39.13)	46 (76.67)	1
	Exclusive breastfeeding (≤6 mo)	15 (65.22)	8 (34.78)	23 (38.33)	0.787
	Acute gastroenteritis	13 (68.42)	6 (31.58)	19 (31.67)	0.573
	Sepsis	6 (46.15)	7 (53.85)	13 (21.67)	0.164
	Fever	5 (71.43)	2 (28.57)	7 (11.67)	0.451
	Lethargy/excessive sleepiness	4 (57.14)	3 (42.86)	7 (11.67)	0.549
	Abdominal distension	5 (83.33)	1 (16.67)	6 (10.00)	0.247
SAM criteria	Convulsions	2 (100.00)	0 (0.00)	2 (3.33)	0.376
	Weight-for-height/length	33 (58.93)	23 (41.07)	56 (93.33)	0.135
	MUAC <11.5 cm	33 (61.11)	21 (38.89)	54 (90.00)	0.58
	Bilateral pedal oedema	6 (54.55)	5 (45.45)	11 (18.33)	0.417

Continued

Parameter	Subgroup	Normal phosphorous (*n* = 37) No. (%)	Low phosphorous (*n* = 23) No. (%)	Total (*n* = 60) No. (%)	*p* value
Hemoglobin (g/dl)	<11	29 (58.00)	21 (42.00)	50 (83.33)	0.172
	≥11	8 (80.00)	2 (20.00)	10 (16.67)	
Serum sodium (mEq/l)	<135	9 (45.00)	11 (55.00)	20 (33.33)	0.13
	135-145	22 (66.67)	11 (33.33)	33 (55.00)	
	>145	6 (85.71)	1 (14.29)	7 (11.67)	
Serum potassium (mEq/l)	<3.5	6 (35.29)	11 (64.71)	17 (28.33)	0.019
	3.5-6.0	29 (70.73)	12 (29.27)	41 (68.34)	
	>6.0	2 (100.00)	0 (0.00)	2 (3.33)	
Serum calcium (mmol/l)	<2.1	2 (16.67)	10 (83.33)	12 (20.00)	0.001
	2.1-2.75	33 (71.74)	13 (28.26)	46 (76.67)	
	>2.75	2 (100.00)	0 (0.00)	2 (3.33)	

Table 2. *Comparison of children according to the serum phosphorus levels at admission, on day 4, and discharge.*

Time interval	Normal serum phosphorous No. (%)	Low serum phosphorous No. (%)
Admission	37 (61.67)	23 (38.33)
Day 4	52 (86.67)	8 (13.33)
Discharge	60 (100.00)	0 (0.00)

and 33 girls (male:female ratio 1:1.22). The median age was 18 months (IQR 12-30). Hypophosphataemia (using age-specific cut-offs) at any point during hospitalization was observed in 23 children (38.3%). No significant associations were found between hypophosphataemia and sex, age group, socioeconomic status, type of SAM (oedematous *vs.* non-oedematous), presence of anemia, or admission diagnosis (all $p > 0.05$). Hyponatraemia (< 135 mEq/l) was present in 20 children (33.3%) and hypokalaemia (< 3.5 mEq/l) in 17 children (28.3%). Both hyponatraemia and hypokalaemia were significantly associated with hypophosphataemia ($p < 0.05$ for both; Table 1).

Serial serum inorganic phosphate levels are summarized in Table 2. At admission, 23 children (38.3%) had hypophosphataemia. On day 4 of the stabilization phase, the prevalence decreased to 8 children (13.3%). Serum phosphate was re-measured at discharge only in those eight children who had hypophosphataemia on day 4; all had normalized by discharge (0% hypophosphataemia among re-tested children). The decrease in prevalence from admission to day 4 (among the full cohort of 60) was statistically significant ($p < 0.001$, McNemar test).

Clinical and biochemical parameters at admission, stratified by serum phosphate status at admission (normal *vs.* hypophosphataemia), are compared in Table 3. Children with hypophosphataemia at admission exhibited significantly higher median respiratory rate (32 *vs.* 28 breaths/min, $p = 0.021$), lower median serum sodium (135 *vs.* 137 mEq/l, $p = 0.027$), lower serum potassium (3.6 *vs.* 3.8 mEq/l, $p = 0.003$), and lower serum calcium (2.1 *vs.* 2.4 mmol/l, $p < 0.001$). As expected, median serum phosphate at admission (3.5 *vs.* 3.9 mg/dl, $p < 0.001$) and on day 4 (3.8 *vs.* 5.0 mg/dl, $p < 0.001$) was significantly lower in the hypophosphataemia group. No significant differences were observed in age, anthropometric measurements, heart rate, temperature, hemoglobin, leukocyte counts, differential counts, platelet count, or weight gain during hospitalization.

Discussion

SAM remains a life-threatening condition, and refeeding hypophosphataemia is increasingly recognized as a key contributor to early mortality during inpatient management. This prospective study of 60 children with complicated SAM provides detailed insights into the natural history of serum phosphate during standardized WHO-protocol treatment using locally prepared therapeutic feeds.

At admission, the median serum inorganic phosphate was 3.81 mg/dl (IQR 0.20), with a range of 2.2 0-5.20 mg/dl. These values are comparable to those reported by Yoshimatsu et al. [13] (Japan, 2013), Selvaraj et al. [15] (India, 2021), and Dakshayani et al. [14] (India, 2019), but higher than those documented by Namusoke et al. [11] (Uganda, 2016), Chanchal et al. [12] (India, 2019), and Khan et al. [16] (India, 2023). The observed differences likely reflect variations in the duration of preceding starvation, severity of illness, and preadmission dietary intake.

Hypophosphataemia (using age-adjusted cut-offs) was present in 38.3% of children at admission and decreased significantly to 13.3% on day 4 of the stabilization phase ($p < 0.001$). Serum phosphate normalized in all retested children (those with hypophosphataemia on day 4) by discharge. This pattern contrasts with the expected rise in

Table 3. *Comparison of mean serum levels and mean weight at admission and discharge.*

Parameter	Normal phosphorous (*n* = 37) Median (IQR)	Low phosphorous (*n* = 23) Median (IQR)	*p* value
Age (months)	15.00 (28.00)	13.00 (14.00)	0.615
Weight at admission (kg)	6.80 (4.55)	6.50 (3.00)	0.538
Length/Height (cm)	75.80 (26.00)	73.50 (16.50)	0.533
MUAC (cm)	11.20 (0.40)	11.40 (0.50)	0.202
Heart rate (per minute)	96.00 (22.00)	98.00 (20.00)	0.074
Respiratory rate (per minute)	28.00 (8.00)	32.00 (12.00)	0.021
Temperature (°F)	100.80 (3.30)	101.20 (2.80)	0.137
Hemoglobin (g/dl)	9.00 (3.80)	8.40 (3.00)	0.563
Total leukocyte count (per cumm)	12,000 (8,250)	9,800 (7,600)	0.605
Neutrophils (%)	60.00 (27.00)	55.00 (32.00)	0.853
Lymphocytes (%)	35.00 (26.00)	35.00 (26.00)	0.889
Eosinophils (%)	2.00 (2.00)	2.00 (2.00)	0.24
Monocytes (%)	4.00 (3.00)	4.00 (3.00)	0.769
Platelet count (per cumm)	238,000 (218,000)	204,000 (317,000)	0.865
Serum sodium (mEq/l)	137.00 (6.50)	135.00 (9.00)	0.027
Serum potassium (mEq/l)	3.80 (0.70)	3.60 (0.80)	0.003
Serum calcium (mmol/l)	2.40 (0.20)	2.10 (0.30)	<0.001
Serum phosphorous at admission (mg/dl)	3.90 (0.17)	3.50 (0.92)	<0.001
Serum phosphorous on Day 4 (mg/dl)	5.00 (0.65)	3.80 (0.80)	<0.001
Weight at discharge (kg)	6.70 (4.47)	6.82 (2.95)	0.494
Weight gain (kg)	0.10 (0.17)	0.08 (0.08)	0.225

hypophosphataemia during early refeeding due to insulin-driven cellular phosphate uptake, suggesting that the locally prepared F-75 formula provided sufficient phosphorus to prevent worsening or to facilitate early correction in most cases. The admission prevalence of 38.3% reported here aligns closely with Namusoke et al. [11] (38%) but is higher than the 20%-25% noted by Selvaraj et al. [15] and Dakshayani et al. [14] and lower than the 50%-69% described in some other cohorts [16-18]. The lower prevalence on day 4 compared to admission may reflect differences in the phosphorus content of therapeutic feeds, timing of sampling relative to refeeding initiation, baseline nutritional status, or the severity of concurrent illnesses such as sepsis or dehydration.

We observed no significant association between hypophosphataemia and sex, age group, socioeconomic class (Modified B.G. Prasad), type of SAM (oedematous *vs.* non-oedematous), anaemia, or admission clinical diagnosis. This independence from demographic and anthropometric factors has been a consistent finding across Indian studies [14,15].

A clinically important observation was the association of hypophosphataemia with concurrent electrolyte abnormalities. Children with hypophosphataemia had significantly lower serum sodium, potassium, and calcium levels compared with children with normal phosphate levels. Similar associations have been reported previously and highlight the need for comprehensive electrolyte monitoring during stabilization and refeeding in children with SAM.

Among children who remained hypophosphataemic on Day 4 and underwent repeat testing at discharge, serum phosphate levels had normalized by discharge. Although these findings suggest recovery of phosphate status during treatment, phosphate levels at discharge were not measured in the entire cohort, and therefore, conclusions regarding normalization cannot be generalized to all participants. Weight gain during hospitalization was comparable between children with and without hypophosphataemia. This finding suggests that the presence of hypophosphataemia during stabilization did not significantly influence short-term nutritional recovery in this cohort.

Strengths of this study include prospective data collection, serial assessment of serum phosphate at clinically relevant time points, use of age-specific reference ranges, and standardized management according to WHO guidelines.

The study has several limitations. It was conducted at a single tertiary-care centre with a relatively small sample size, which may limit generalizability. The phosphorus content of the locally prepared F-75 and F-100 feeds was not directly measured; therefore, conclusions regarding adequacy of phosphorus provision are based on observed biochemical trends rather than quantified dietary phosphorus intake. Important clinical outcomes such as duration

of hospital stay, mortality, complications attributable to refeeding syndrome, and requirement for additional electrolyte correction were not systematically evaluated. Serum magnesium and thiamine levels were not routinely measured, limiting assessment of other important components of refeeding syndrome. In addition, post-discharge follow-up was not performed.

Conclusion

Hypophosphataemia affected more than one-third of children with complicated SAM and was associated with concurrent abnormalities of sodium, potassium, and calcium. The prevalence of hypophosphataemia decreased significantly during the stabilization phase of treatment. Among children who remained hypophosphataemic on Day 4 and underwent repeat testing, serum phosphate levels had normalized by discharge. These findings support the importance of careful electrolyte monitoring during refeeding and highlight the need for larger multicentre studies to better define phosphate requirements in children with SAM.

What is new?

This study provides prospective data on serum phosphate changes during inpatient management of SAM in Indian children. Hypophosphataemia was common at admission and decreased significantly during the stabilization phase. Children with hypophosphataemia had more frequent concurrent electrolyte abnormalities involving sodium, potassium, and calcium. The study also demonstrates improvement in serum phosphate levels during treatment with locally prepared WHO-recommended therapeutic feeds.

Acknowledgment

None.

List of Abbreviations

CBC	Complete blood count
F-75	Starter therapeutic diet (75 kcal/100 ml)
F-100	Catch-up therapeutic diet (100 kcal/100 ml)
IQR	Interquartile range
MUAC	Mid-upper arm circumference
NFHS	National family health survey
SAM	Severe acute malnutrition
SD	Standard deviation
SPSS	Statistical Package for the Social Sciences
UNICEF	United Nations Children's Fund
WHO	World Health Organization

Conflict of interests

The authors declare that there is no conflict of interest regarding the publication of this article.

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Consent to participate

Written informed consent was obtained from the parents/legal guardians of all participants before enrolment.

Ethical approval

Example for original (Research) articles: Ethical approval was granted by the Ethics Committee/Institutional Review Board/ Research Committee via reference/letter number- 27924403 dated: 19th October 2022.

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