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Original Article

Frequency and Risk Factors Associated with Hypophosphatemia in Critically Ill Children

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ABSTRACT

Background: Hypophosphatemia is a clinically important electrolyte disturbance in critically ill children and may affect respiratory, cardiac, neuromuscular, hematological, and metabolic function. Objective: To determine the frequency of hypophosphatemia and assess selected clinical and treatment-related factors associated with hypophosphatemia among critically ill children admitted to a pediatric intensive care unit. Methods: This descriptive observational study with comparative association analysis was conducted in the Pediatric Medicine Department, Intensive Care Unit, Children Hospital, Multan, from December 2024 to May 2025. A total of 160 children aged 2 months to 12 years were enrolled by non-probability consecutive sampling. Hypophosphatemia was defined as serum phosphorus <3.8 mg/dL in children younger than 2 years and <3.5 mg/dL in children aged 2 years or older, using the lowest documented phosphate level during intensive care stay. Data were analyzed using SPSS version 23. Results: The mean age was 4.61 ± 3.21 years, and 92 (57.5%) children were male. Hypophosphatemia was observed in 112 (70.0%) children. Malnutrition, acute kidney injury, steroid use, furosemide use, and beta-2 agonist therapy were significantly associated with hypophosphatemia. Children with hypophosphatemia had longer hospital stay than those with normal phosphate levels, 9.00 ± 4.10 versus 6.10 ± 2.70 days, p<0.001, and higher mortality, 31.3% versus 14.6%, p=0.046. Conclusion: Hypophosphatemia was frequent among critically ill children and was associated with malnutrition, acute kidney injury, and selected medication exposures. Routine phosphate monitoring should be considered in high-risk pediatric intensive care patients. Keywords: Hypophosphatemia, serum phosphate, critically ill children, pediatric intensive care unit, malnutrition, acute kidney injury.

INTRODUCTION

Phosphate is an essential intracellular anion that plays a central role in cellular energy metabolism, skeletal mineralization, nucleic acid synthesis, intracellular signaling, membrane integrity, and oxygen delivery through its contribution to 2,3-diphosphoglycerate production. Although only a small proportion of total body phosphate is present in the circulating compartment, serum phosphate remains clinically important because reduced circulating levels may indicate depleted body stores, altered intracellular redistribution, impaired intake or absorption, or increased renal losses during acute illness. In critically ill children, these disturbances may compromise respiratory muscle function, myocardial performance, neuromuscular stability, hematological function, immune response, and overall metabolic recovery, making phosphate monitoring clinically relevant in pediatric intensive care settings (1-3).

Hypophosphatemia in critically ill patients is usually multifactorial. Reduced nutritional intake, impaired intestinal absorption, intracellular phosphate shift, increased urinary phosphate loss, respiratory alkalosis, insulin therapy, catecholamine exposure, sepsis, malnutrition, refeeding physiology, diuretic therapy, and prolonged critical illness may all contribute to reduced serum phosphate levels. Mild hypophosphatemia may remain clinically silent, but more severe deficiency can present with respiratory muscle weakness, difficulty in weaning from mechanical ventilation,

myocardial dysfunction, arrhythmias, hemolysis, impaired leukocyte function, rhabdomyolysis, seizures, and altered sensorium. These complications are particularly important in pediatric intensive care units because critically ill children often have limited nutritional reserve, high metabolic demand, respiratory compromise, fluid and electrolyte instability, and frequent exposure to medications that may alter phosphate homeostasis (4-7).

Previous pediatric studies have reported a substantial burden of hypophosphatemia among critically ill children, although reported frequencies vary according to age distribution, illness severity, nutritional status, timing of phosphate measurement, and diagnostic thresholds. De Menezes et al. reported hypophosphatemia among children admitted to intensive care and observed an association with malnutrition, while Santana e Meneses et al. identified hypophosphatemia during the early phase of pediatric intensive care admission and reported associations with respiratory disease and medication exposure (8,9). Kilic et al. found that hypophosphatemia was associated with prolonged mechanical ventilation and longer pediatric intensive care stay, whereas Shah et al. reported hypophosphatemia in 71.6% of critically ill children and observed an association with prolonged intensive care stay but not with mortality (10,11). El Shazly et al. reported that hypophosphatemia increased from 42% at admission to 62% by the seventh day of pediatric intensive care admission and was more frequent among malnourished children and those receiving furosemide, steroids, and beta-2 agonists (12). Local pediatric intensive care data from Pakistan have also identified hypophosphatemia as a common electrolyte disturbance among critically ill children, supporting the relevance of this issue in regional clinical practice (13).

The clinical significance of hypophosphatemia extends beyond its biochemical definition. Children with severe acute malnutrition, burns, sepsis, or nutritional rehabilitation are especially vulnerable to phosphate depletion because of reduced stores, increased metabolic demand, and intracellular shifts during recovery or refeeding. Recent work in critically ill children has further emphasized the relationship between early hypophosphatemia, nutritional therapy, and clinical recovery, while systematic reviews in adult and pediatric critical care populations have shown associations between hypophosphatemia and adverse outcomes such as longer intensive care stay and increased need for respiratory support. However, whether hypophosphatemia is an independent contributor to poor outcomes or primarily a marker of illness severity remains uncertain, particularly in studies without adjustment for severity of illness, nutritional intake, ventilation status, and medication exposure (14-19).

Despite available international evidence, there remains limited regional evidence from South Punjab regarding the frequency of hypophosphatemia among critically ill children when age-specific phosphate thresholds are used and when clinically relevant factors such as malnutrition, acute kidney injury, steroid therapy, furosemide use, and beta-2 agonist exposure are assessed. Understanding these associations may help clinicians identify children who require closer phosphate monitoring during pediatric intensive care admission. Therefore, this study was conducted to determine the frequency of hypophosphatemia and to assess selected clinical and treatment-related factors associated with hypophosphatemia among critically ill children admitted to the pediatric intensive care unit.

MATERIALS AND METHODS

This descriptive observational study with comparative association analysis was conducted in the Pediatric Medicine Department, Intensive Care Unit, Children Hospital, Multan, Pakistan, from December 2024 to May 2025. The study was designed to determine the frequency of hypophosphatemia among critically ill children and to evaluate selected clinical and treatment-related factors associated with hypophosphatemia during pediatric intensive care admission. The study population comprised children admitted to the pediatric intensive care unit who fulfilled the predefined eligibility criteria during the study period.

The sample size was calculated using the WHO sample size calculator for estimation of a single population proportion. The expected frequency of hypophosphatemia was taken as 71.6% on the basis of previously reported pediatric intensive care data, with a 95% confidence level and 7% absolute precision (11). The calculated sample size was 160 children. Non-probability consecutive sampling was used, and eligible participants were enrolled until the required sample size was achieved.

Children aged 2 months to 12 years, of either gender, admitted to the pediatric intensive care unit were included. Children with chronic kidney disease, chronic liver disease, or postoperative admission were excluded. Chronic kidney disease and chronic liver disease were assessed through clinical history and available medical records. Postoperative children were excluded because perioperative fasting, operative stress, fluid shifts, tissue injury, and postoperative metabolic changes could independently influence serum phosphate levels and confound interpretation of phosphate disturbance during critical illness.

After approval from the institutional ethical review committee, all children admitted to the pediatric intensive care unit during the study period were screened according to the eligibility criteria. Written informed consent was obtained from parents or legal guardians before enrollment. Each participant was assigned a study case number to maintain confidentiality. Baseline demographic and clinical information, including age, gender, area of residence, nutritional status, acute kidney injury status, medication exposure, serum phosphate level, duration of hospital stay, and final hospital outcome, was recorded on a predesigned study proforma by the researcher.

Hypophosphatemia was defined using age-specific serum phosphate thresholds. In children younger than 2 years, hypophosphatemia was recorded when serum phosphorus was <3.8 mg/dL, while in children aged 2 years or older, hypophosphatemia was recorded when serum phosphorus was <3.5 mg/dL. The lowest serum phosphate value documented during pediatric intensive care stay was used to classify phosphate status. This approach was selected to capture clinically relevant phosphate depletion occurring at any point during intensive care admission rather than limiting assessment to admission values only. Children were then categorized into two groups: those with hypophosphatemia and those with normal serum phosphate levels.

Nutritional status was assessed through anthropometric measurement after enrollment. Weight was measured using a standard weighing scale, while length or height was measured according to age, using an infantometer in children aged ≤2 years and a stadiometer in children aged >2 years. Malnutrition was recorded when the weight-for-height z-score was ≤−2.0. Acute kidney injury was defined according to KDIGO criteria as serum creatinine >1.5 times the baseline value after 24 hours of admission or urine output <0.5 mL/kg/hour for six hours (20). Medication exposure was recorded when the child received steroids, furosemide, or beta-2 agonists during pediatric intensive care stay. These medications were assessed as treatment-related exposures because of their potential relationship with phosphate redistribution, renal electrolyte loss, or illness severity.

Serum phosphate measurement was performed through the same hospital laboratory to minimize inter-laboratory variation. Acute kidney injury was assessed using serum creatinine and urine output according to the operational definition. Duration of hospital stay was calculated in days from admission to discharge or death. Final hospital outcome was categorized as discharged or mortality. To reduce information bias, all variables were recorded using uniform operational definitions on a structured proforma. To improve internal consistency, laboratory-based measurements were taken from hospital records, anthropometric measurements were performed using standard equipment, and all data were reviewed for completeness before entry into the statistical software.

Data were entered and analyzed using SPSS version 23. Numerical variables, including age, serum phosphate level, and duration of hospital stay, were assessed for normality using the Shapiro-Wilk test and expressed as mean and standard deviation. Categorical variables, including gender, area of residence, malnutrition, acute kidney injury, medication exposure, phosphate status, and final outcome, were

expressed as frequency and percentage. Children with hypophosphatemia were compared with children having normal serum phosphate levels. Categorical variables were compared using the chi-square test. Continuous variables were compared using an appropriate independent-group test according to the distribution of data. Stratified analysis was performed for age group, gender, and area of residence, followed by post-stratification chi-square testing. A p-value of ≤0.05 was considered statistically significant. Because the study was observational and based on unadjusted association analysis, findings were interpreted as associations rather than independent predictors or causal risk factors.

Ethical approval was obtained from the institutional ethical review committee before commencement of the study. Written informed consent was obtained from parents or legal guardians of all enrolled children. Participant confidentiality was maintained throughout the study by using coded study numbers instead of personal identifiers. Data were used only for research purposes, and de-identified aggregate findings were reported. No external funding was received, and the authors declared no conflict of interest.

RESULTS

A total of 160 critically ill children admitted to the pediatric intensive care unit were included in the analysis. The mean age was 4.61 ± 3.21 years. Children aged 2 months to <2 years constituted 54 (33.8%) participants, 58 (36.2%) were aged 2 to 5 years, and 48 (30.0%) were aged >5 to 12 years. There were 92 (57.5%) male and 68 (42.5%) female children. Rural residence was recorded in 98 (61.2%) participants, while 62 (38.8%) belonged to urban areas. The overall mean serum phosphate level was 3.12 ± 0.71 mg/dL, and the mean duration of hospital stay was 8.08 ± 3.86 days.

Table 1. Baseline Characteristics of Study Participants

Variable	Category / Measure	n / Mean	% / SD
Total patients		160	100.0
Age, years	Mean ± SD	4.61	±3.21
Age group	2 months to <2 years	54	33.8
	2 to 5 years	58	36.2
	>5 to 12 years	48	30.0
Gender	Male	92	57.5
	Female	68	42.5
Residence	Rural	98	61.2
	Urban	62	38.8
Serum phosphate, mg/dL	Mean ± SD	3.12	±0.71
Hospital stay, days	Mean ± SD	8.08	±3.86

Hypophosphatemia was observed in 112 of 160 critically ill children, giving an overall frequency of 70.0%. Normal serum phosphate levels were recorded in 48 (30.0%) children. This indicates that approximately seven out of every ten children admitted to the pediatric intensive care unit developed hypophosphatemia according to the study's age-specific phosphate thresholds.

Table 2. Frequency of Hypophosphatemia Among Critically Ill Children

Phosphate Status	Frequency	Percentage
Hypophosphatemia	112	70.0
Normal phosphate level	48	30.0
Total	160	100.0

The mean serum phosphate level was substantially lower among children with hypophosphatemia than among children with normal phosphate levels. Children with hypophosphatemia had a mean serum phosphate level of 2.78 ± 0.45 mg/dL, compared with 4.05 ± 0.32 mg/dL in children with normal phosphate levels. The mean difference was −1.27 mg/dL, with a 95% confidence interval from −1.39 to −1.15 mg/dL, and this difference was statistically significant, $p<0.001$.

Table 3. Serum Phosphate Level According to Phosphate Status

Variable	Hypophosphatemia n=112	Normal Phosphate n=48	Mean Difference	95% CI	p-value
Serum phosphate, mg/dL	2.78 ± 0.45	4.05 ± 0.32	−1.27	−1.39 to −1.15	<0.001

Values are presented as mean ± standard deviation. Mean difference represents hypophosphatemia group minus normal phosphate group.

Malnutrition, acute kidney injury, and medication exposures were significantly associated with hypophosphatemia. Malnutrition was present in 62 (55.4%) children with hypophosphatemia compared with 10 (20.8%) children with normal phosphate levels. Children with malnutrition had 4.71 times higher odds of hypophosphatemia than children without malnutrition, OR 4.71, 95% CI 2.14–10.38, p<0.001. Acute kidney injury was present in 38 (33.9%) children with hypophosphatemia and 6 (12.5%) children with normal phosphate levels, corresponding to OR 3.59, 95% CI 1.40–9.21, p=0.010.

Steroid use was recorded in 68 (60.7%) children with hypophosphatemia and 12 (25.0%) children with normal phosphate levels. The odds of hypophosphatemia were 4.64 times higher among children receiving steroids than those not receiving steroids, OR 4.64, 95% CI 2.18–9.87, p<0.001. Furosemide use was also significantly associated with hypophosphatemia, being present in 43 (38.4%) children with hypophosphatemia and 8 (16.7%) children with normal phosphate levels, OR 3.12, 95% CI 1.33–7.28, p=0.012. Beta-2 agonist therapy was recorded in 45 (40.2%) children with hypophosphatemia compared with 9 (18.8%) children with normal phosphate levels, OR 2.91, 95% CI 1.29–6.59, p=0.015.

Table 4. Clinical and Treatment-Related Factors Associated with Hypophosphatemia

Associated Factor	Hypophosphatemia n=112	Normal Phosphate n=48	Odds Ratio	95% CI	p-value
Malnutrition	62 (55.4%)	10 (20.8%)	4.71	2.14–10.38	<0.001
Acute kidney injury	38 (33.9%)	6 (12.5%)	3.59	1.40–9.21	0.010
Steroid use	68 (60.7%)	12 (25.0%)	4.64	2.18–9.87	<0.001
Furosemide use	43 (38.4%)	8 (16.7%)	3.12	1.33–7.28	0.012
Beta-2 agonist use	45 (40.2%)	9 (18.8%)	2.91	1.29–6.59	0.015

Odds ratios and 95% confidence intervals were derived from the reported aggregate 2×2 counts. The reference category for each comparison was absence of the listed factor.

On stratified analysis, hypophosphatemia was numerically more frequent among younger children, males, and children from rural areas, although these differences were not statistically significant. Among children aged 2 months to <2 years, 43 of 54 (79.6%) had hypophosphatemia, compared with 40 of 58 (69.0%) children aged 2 to 5 years and 29 of 48 (60.4%) children aged >5 to 12 years, p=0.105. Using children aged >5 to 12 years as the reference group, children aged 2 months to <2 years had higher odds of hypophosphatemia, OR 2.56, 95% CI 1.06–6.17, while children aged 2 to 5 years had OR 1.46, 95% CI 0.65–3.25. However, the overall age-group comparison did not reach statistical significance.

Hypophosphatemia was present in 67 of 92 (72.8%) male children and 45 of 68 (66.2%) female children, OR 1.37, 95% CI 0.69–2.71, p=0.464. Similarly, 74 of 98 (75.5%) rural children had hypophosphatemia compared with 38 of 62 (61.3%) urban children, OR 1.95, 95% CI 0.98–3.87, p=0.083. Although rural residence showed a higher numerical frequency of hypophosphatemia, the confidence interval crossed unity and the association was not statistically significant.

Table 5. Stratified Distribution of Hypophosphatemia by Age, Gender, and Residence

Variable	Total	Hypophosphatemia	Normal Phosphate	Odds Ratio	95% CI	p-value
Age group						0.105
2 months to <2 years	54	43 (79.6%)	11 (20.4%)	2.56	1.06–6.17	
2 to 5 years	58	40 (69.0%)	18 (31.0%)	1.46	0.65–3.25	
>5 to 12 years	48	29 (60.4%)	19 (39.6%)			
Gender						0.464
Male	92	67 (72.8%)	25 (27.2%)	1.37	0.69–2.71	
Female	68	45 (66.2%)	23 (33.8%)			
Residence						0.083

Variable	Total	Hypophosphatemia	Normal Phosphate	Odds Ratio	95% CI	p-value
Rural	98	74 (75.5%)	24 (24.5%)	1.95	0.98–3.87	
Urban	62	38 (61.3%)	24 (38.7%)			

Odds ratios for age were calculated using >5 to 12 years as the reference group. Odds ratios for gender and residence were calculated using female and urban categories as reference groups, respectively. The p-values represent overall group comparisons from the manuscript data.

Children with hypophosphatemia had a longer hospital stay than children with normal phosphate levels. The mean duration of hospital stay was 9.00 ± 4.10 days in the hypophosphatemia group compared with 6.10 ± 2.70 days in the normal phosphate group. The mean difference was 2.90 days, with a 95% confidence interval from 1.81 to 3.99 days, p<0.001. Mortality was also higher among children with hypophosphatemia. Among children with hypophosphatemia, 35 (31.3%) died, compared with 7 (14.6%) children with normal phosphate levels. The odds of mortality were 2.66 times higher in children with hypophosphatemia than in those with normal phosphate levels, OR 2.66, 95% CI 1.09–6.52, p=0.046. However, because this was an unadjusted analysis, this association should be interpreted as a relationship between phosphate status and outcome rather than evidence that hypophosphatemia independently caused mortality.

Table 6. Hospital Stay and Final Outcome According to Phosphate Status

Outcome	Hypophosphatemia n=112	Normal Phosphate n=48	Effect Estimate	95% CI	p-value
Hospital stay, days	9.00 ± 4.10	6.10 ± 2.70	Mean difference 2.90 days	1.81–3.99	<0.001
Discharged	77 (68.8%)	41 (85.4%)	Reference outcome		0.046
Mortality	35 (31.3%)	7 (14.6%)	OR 2.66	1.09–6.52	0.046

Values are presented as mean ± standard deviation or n (%). The mean difference represents hypophosphatemia group minus normal phosphate group. The odds ratio for mortality compares children with hypophosphatemia against children with normal phosphate levels.

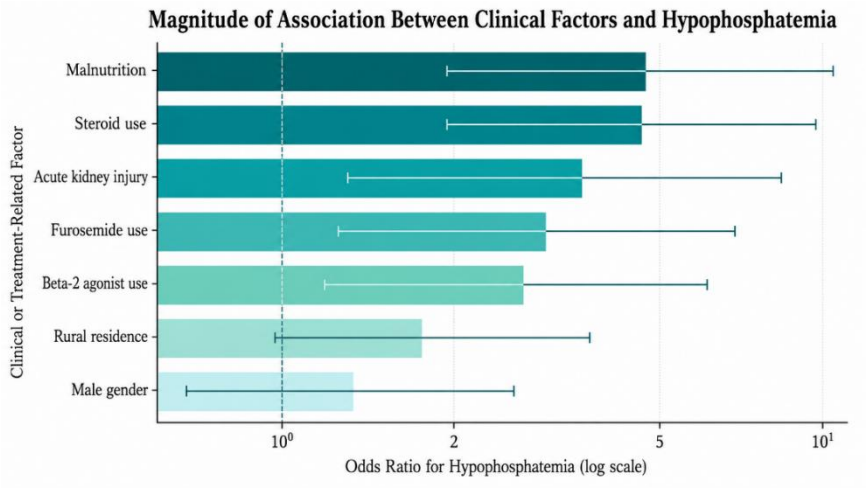

Figure 1 Magnitude of Association Between Clinical Factors and Hypophosphatemia

Figure 1 demonstrates the comparative magnitude of association between selected clinical and treatment-related factors and hypophosphatemia among critically ill children. Malnutrition showed the strongest association with hypophosphatemia, with affected children having 4.71-fold higher odds of hypophosphatemia than non-malnourished children, followed closely by steroid use with an odds ratio of 4.64. Acute kidney injury was also strongly associated with hypophosphatemia, OR 3.59, while furosemide use and beta-2 agonist therapy showed moderate but clinically meaningful associations, OR 3.12 and OR 2.91, respectively. Rural residence demonstrated a higher but statistically less certain association, OR 1.95, whereas male gender showed the weakest association, OR 1.37. The confidence intervals for malnutrition, steroid use, acute kidney injury, furosemide use, and beta-2 agonist therapy remained above the null value, supporting their statistical and clinical relevance as associated factors,

while the wider intervals indicate the need for cautious interpretation in the absence of multivariable adjustment.

DISCUSSION

This study found that hypophosphatemia was highly frequent among critically ill children admitted to the pediatric intensive care unit, affecting 112 of 160 participants, with an overall frequency of 70.0%. This finding indicates that phosphate disturbance was not an occasional biochemical abnormality in this cohort but a common metabolic derangement during critical care admission. The mean serum phosphate level was substantially lower among children with hypophosphatemia than among children with normal phosphate levels, with a mean difference of −1.27 mg/dL, supporting the clinical and biochemical separation between the two groups. The observed frequency is close to the 71.6% reported by Shah et al. among critically ill children, suggesting that a similar burden may be present across pediatric intensive care settings where children have high illness severity, nutritional compromise, and exposure to therapies that may influence phosphate balance (11). However, the frequency in the present study should be interpreted in relation to the methodological approach used, because phosphate status was classified according to the lowest serum phosphate value documented during pediatric intensive care stay rather than admission phosphate alone. This approach captures phosphate depletion occurring during the course of critical illness but may yield a higher frequency than studies limited to admission measurements.

The present findings are broadly consistent with previous pediatric intensive care literature reporting hypophosphatemia as a common electrolyte disturbance among critically ill children. El Shazly et al. observed that hypophosphatemia increased from 42% at admission to 62% by the seventh day of pediatric intensive care admission, supporting the concept that phosphate depletion may develop or worsen during intensive care rather than being limited to baseline status (12). Kilic et al. reported hypophosphatemia in 60.2% of critically ill pediatric patients and found associations with prolonged ventilation and longer pediatric intensive care stay, while Santana e Meneses et al. described associations with respiratory illness and medication exposure (9,10). A Pakistani pediatric intensive care study also identified hypophosphatemia as a frequent abnormality among critically ill children, although reported frequencies may vary according to population characteristics, age-specific thresholds, nutritional status, timing of measurement, and case-mix differences (13). The high frequency observed in the present study therefore appears clinically plausible, especially in a pediatric intensive care population in which malnutrition, acute kidney injury, and medication exposure were common.

Malnutrition showed the strongest association with hypophosphatemia in the present analysis. Malnutrition was present in 55.4% of children with hypophosphatemia compared with 20.8% of children with normal phosphate levels, and malnourished children had 4.71-fold higher odds of hypophosphatemia. This association is biologically plausible because malnourished children often have reduced intracellular phosphate stores, lower dietary intake, impaired metabolic reserve, and increased susceptibility to phosphate shifts during acute illness or nutritional rehabilitation. Phosphate depletion in malnutrition has been reported in children recovering from severe acute undernutrition and in children with sepsis, where intracellular depletion and refeeding-related shifts may contribute to clinically relevant hypophosphatemia (18,19). In critically ill children, the combination of poor baseline reserve, inflammation, catabolism, and nutritional therapy may further increase vulnerability to phosphate disturbance. This finding supports the need for careful nutritional assessment and phosphate monitoring in malnourished children admitted to pediatric intensive care.

Acute kidney injury was also significantly associated with hypophosphatemia, with affected children having 3.59-fold higher odds of hypophosphatemia than children without acute kidney injury. Although renal impairment is classically associated with phosphate retention and hyperphosphatemia, phosphate balance in critical illness is complex and may be influenced by fluid therapy, diuretic exposure, altered

tubular handling, intracellular redistribution, poor intake, and concurrent metabolic stress. In this context, acute kidney injury may function not only as a renal variable but also as a marker of greater systemic illness severity. The use of KDIGO criteria provided a standardized operational definition for acute kidney injury, but interpretation remains limited by the absence of illness severity scores and the lack of detailed data on baseline creatinine estimation, urine output trends, fluid balance, and phosphate replacement (20). Therefore, the association between acute kidney injury and hypophosphatemia should be interpreted as an unadjusted clinical relationship rather than evidence of an independent renal mechanism.

Medication exposure was an important treatment-related finding. Steroid use, furosemide use, and beta-2 agonist therapy were all significantly more frequent among children with hypophosphatemia. Steroid exposure showed a strong association, with an odds ratio of 4.64, followed by furosemide use with an odds ratio of 3.12 and beta-2 agonist therapy with an odds ratio of 2.91. These findings are consistent with known mechanisms of medication-related phosphate disturbance. Steroids may contribute to altered renal phosphate handling and metabolic shifts, furosemide may increase urinary electrolyte losses, and beta-2 agonists may promote intracellular phosphate redistribution through adrenergic stimulation (5). Similar associations have been reported by El Shazly et al. and Kilic et al., where furosemide, steroids, and beta-2 agonists were linked with hypophosphatemia among critically ill children (10,12). Nevertheless, these medication associations should be interpreted cautiously because drug exposure may also reflect underlying disease severity, particularly respiratory compromise, fluid overload, inflammation, or hemodynamic instability.

The stratified analysis showed that hypophosphatemia was numerically more frequent among younger children, males, and rural residents, but these associations did not reach statistical significance. Children aged 2 months to <2 years had the highest frequency of hypophosphatemia at 79.6%, compared with 69.0% among children aged 2 to 5 years and 60.4% among those aged >5 to 12 years. This gradient may reflect greater nutritional vulnerability, higher metabolic demand, or age-related differences in phosphate requirements among younger children, although the overall age-group comparison was not statistically significant. Rural residence also showed a higher numerical frequency of hypophosphatemia, 75.5% versus 61.3% among urban children, but the confidence interval crossed unity. These findings suggest possible demographic patterns that warrant further investigation in larger multicenter studies, but they should not be interpreted as confirmed independent associations.

Children with hypophosphatemia had longer hospital stay and higher mortality than children with normal phosphate levels. The mean hospital stay was 9.00 ± 4.10 days in the hypophosphatemia group compared with 6.10 ± 2.70 days in the normal phosphate group, with a mean difference of 2.90 days. Mortality was also higher among children with hypophosphatemia, 31.3% versus 14.6%, corresponding to 2.66-fold higher odds of mortality. These findings are clinically important because phosphate is essential for energy metabolism, respiratory muscle activity, myocardial function, oxygen delivery, and cellular recovery. Previous studies have also reported associations between hypophosphatemia and prolonged intensive care stay, mechanical ventilation, or adverse clinical outcomes (10-12). However, the present study did not include multivariable adjustment for severity of illness, sepsis, ventilation status, nutritional intake, or medication dose and duration. Therefore, hypophosphatemia may represent both a potentially modifiable metabolic abnormality and a marker of greater underlying illness severity. The relationship with mortality should consequently be interpreted as an unadjusted association rather than proof that hypophosphatemia independently caused death.

The findings have practical implications for pediatric critical care. Routine phosphate monitoring may be especially relevant in children with malnutrition, acute kidney injury, steroid exposure, furosemide use, or beta-2 agonist therapy. Early identification of phosphate disturbance may allow timely clinical review of nutrition, renal function, medication exposure, and replacement needs. However, the study does not establish treatment thresholds or prove that phosphate correction improves outcomes.

Systematic reviews have shown that hypophosphatemia is associated with adverse outcomes in critically ill adults and children, but uncertainty remains regarding causality, optimal monitoring intervals, replacement strategies, and outcome benefits of correction (16,17). Future studies should therefore evaluate serial phosphate trends, severity-adjusted predictors, medication dose-response patterns, nutritional intake, phosphate replacement, mechanical ventilation duration, and clinically meaningful recovery outcomes.

This study has several limitations. It was conducted at a single center and used non-probability consecutive sampling, which may limit generalizability. Severity of illness scoring was not included, and therefore residual confounding by illness severity cannot be excluded. Phosphate status was based on the lowest documented serum phosphate value during pediatric intensive care stay, which captured in-stay hypophosphatemia but may not be directly comparable with admission-only studies. Serial phosphate trends, timing of hypophosphatemia onset, nutritional intake, refeeding status, phosphate replacement, mechanical ventilation duration, sepsis status, and medication dose or duration were not assessed. Multivariable logistic regression was not performed, so the reported associations should be considered unadjusted. Despite these limitations, the study provides clinically useful local evidence showing a high burden of hypophosphatemia and identifying key clinical and treatment-related factors associated with phosphate disturbance among critically ill children.

CONCLUSION

Hypophosphatemia was frequent among critically ill children admitted to the pediatric intensive care unit, affecting 70.0% of the study population. Malnutrition, acute kidney injury, steroid use, furosemide use, and beta-2 agonist therapy were significantly associated with hypophosphatemia, while younger age, male gender, and rural residence showed higher numerical frequencies without statistically significant associations. Children with hypophosphatemia also had longer hospital stay and higher mortality than those with normal phosphate levels, although these outcome associations should be interpreted cautiously because the analysis was unadjusted for illness severity and other potential confounders. Routine phosphate monitoring should be considered in critically ill children, particularly those with nutritional compromise, acute kidney injury, and exposure to medications that may disturb phosphate balance, while future multicenter studies with serial phosphate assessment and adjusted analysis are needed to identify independent predictors and clarify the clinical impact of phosphate correction.

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