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PREVALENCE AND PATTERN OF SERUM ELECTROLYTE DERANGEMENTS IN CHILDREN AGED 6–59 MONTHS WITH SEVERE ACUTE MALNUTRITION

Dr Kapil Sharma[1], Dr Mahima Joshi[2], Dr Akanksha Sharma[3], Dr Barkha Lodiwal[4]

[1] Senior Resident, Department of paediatrics, Government Medical College, Kota(raj).
[2] Senior Resident, Department of paediatrics, RNT Medical College Udaipur (raj).
[3] Senior Resident, Department of paediatrics, RVRS Government Medical College Bhilwara (raj).
[4] Resident Doctor, Department of paediatrics, Government Medical College, Kota(raj).

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Corresponding Author:

Dr Kapil Sharma
Senior Resident, Department of paediatrics, Government Medical College, Kota(raj).

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ABSTRACT

Background: Severe acute malnutrition (SAM) is associated with profound alterations in fluid distribution, total-body potassium, renal solute handling and physiological reserve. Serum electrolyte abnormalities may remain clinically occult yet contribute to neurological dysfunction, cardiac instability and circulatory compromise. Institution-specific data on the burden and pattern of these abnormalities are limited in the Hadoti region of Rajasthan.

Objective: To determine the prevalence and pattern of serum sodium, potassium and chloride derangements among children aged 6–59 months with severe acute malnutrition and to evaluate their association with nutritional and clinical characteristics.

Methods: This hospital-based cross-sectional observational study was conducted in the Department of Paediatrics, J.K. Loan Hospital, Kota, from June 2024 to February 2026. Ninety children aged 6–59 months fulfilling World Health Organization criteria for SAM were enrolled after written informed consent. Demographic, clinical, anthropometric and nutritional data were recorded using a structured proforma. A 3 mL venous blood sample was collected within two hours of admission. Serum sodium, potassium and chloride were measured using an EM640 biochemistry autoanalyser. Dyselectrolytemia was defined as at least one abnormal sodium, potassium or chloride value. Descriptive statistics, group comparisons, correlation analysis and binary logistic regression were used; $p<0.05$ was considered statistically significant.

Results: Dyselectrolytemia was present in 65 of 90 children (72.2%). Hyponatremia was the most common electrolyte abnormality, affecting 50 children (55.6%), followed by hypokalemia in 42 (46.7%) and hypochloremia in 30 (33.3%). Hypernatremia and hyperkalemia were uncommon, occurring in 2 (2.2%) and 1 (1.1%) children, respectively. Children with dyselectrolytemia had a significantly lower mean MUAC (10.5 ± 0.6 vs. 11.6 ± 0.5 cm; $p < 0.001$), lower weight-for-height Z-score (−3.9 ± 0.5 vs. −3.3 ± 0.4; $p = 0.002$), higher frequency of diarrhoea at admission (53.8% vs. 28.0%; $p = 0.01$), and lower mean serum albumin levels (1.9 ± 0.6 vs. 2.7 ± 0.5 g/dL; $p < 0.001$). On multivariable analysis, serum albumin <2.0 g/dL (adjusted odds ratio [AOR] 4.4; 95% CI: 1.6–11.8; $p = 0.003$), diarrhea at admission (AOR 3.0; 95% CI: 1.1–8.2; $p = 0.029$), and MUAC <11.5 cm (AOR 2.5; 95% CI: 1.0–6.3; $p = 0.047$) were identified as independent factors associated with dyselectrolytemia. Furthermore, children with oedematous SAM phenotypes had lower mean serum sodium and potassium levels compared with those with marasmus.

Conclusion: Serum electrolyte derangements were highly prevalent among hospitalized children with SAM. Hyponatremia and hypokalemia predominated, and abnormalities were closely associated with severe anthropometric depletion, diarrhea and hypoalbuminemia. Routine admission electrolyte assessment should complement clinical and anthropometric evaluation in hospitalized children with SAM.

***Keywords**: Severe acute malnutrition; dyselectrolytemia; hyponatremia; hypokalemia; hypochloremia; children.*

INTRODUCTION

Severe acute malnutrition (SAM) is the most life-threatening form of childhood undernutrition. In children aged 6–59 months, it is identified by a weight-for-height or weight-for-length Z-score below −3 standard deviations, a mid-upper arm circumference (MUAC) below 11.5 cm, or the presence of bilateral pitting oedema.[1,2] Although SAM is diagnosed using anthropometric and clinical criteria, it represents a multisystem disorder associated with loss of lean tissue, impaired immune function, reduced cardiac and renal reserve, altered intestinal integrity and major disturbances in normal metabolic homeostasis.[3]

Sodium, potassium and chloride are essential for maintaining extracellular osmolality, intravascular volume, acid–base balance, neuromuscular excitability and myocardial electrical stability. Children with SAM commonly have total-body potassium depletion and abnormal sodium and water distribution, even when serum concentrations do not fully reflect intracellular deficits.[4,5] Diarrhea, vomiting, poor feeding, pneumonia, sepsis, altered renal handling and inappropriate fluid administration may further aggravate these abnormalities.[6] Severe electrolyte disturbances may result in altered sensorium, seizures, muscle weakness, cardiac arrhythmias and haemodynamic instability.

Clinical recognition of fluid and electrolyte abnormalities in SAM is particularly difficult because conventional signs of dehydration and circulatory compromise may be unreliable in severely malnourished children.[4] Oedema, loss of subcutaneous tissue and altered physiological responses may obscure the true degree of intravascular depletion or electrolyte imbalance. Consequently, clinically important abnormalities may remain undetected when assessment is based only on anthropometry and physical examination. Early biochemical evaluation is therefore essential for identifying children who require cautious fluid management, electrolyte correction and close monitoring.

India continues to carry a substantial burden of childhood undernutrition. The National Family Health Survey-5 documented persistent levels of stunting, wasting and underweight among children younger than five years, with important regional and district-level variations, including in Rajasthan.[7] Global estimates also demonstrate that childhood wasting remains a major public-health problem, particularly in South Asia and sub-Saharan Africa.[8] Socioeconomic deprivation, inadequate breastfeeding and complementary feeding, recurrent infections, incomplete immunisation, low birth weight and delayed access to healthcare are recognised contributors to SAM in Indian settings.[9] This burden results in a considerable number of children requiring hospital-based stabilisation for nutritional, infectious and metabolic complications. Previous hospital-based studies have reported a high but variable prevalence of electrolyte abnormalities among children with SAM. Hyponatremia and hypokalemia are consistently described as the predominant abnormalities, while chloride disturbances are less frequently documented.[10,11] Greater biochemical derangement has been reported among children with diarrhea, oedema, severe wasting and hypoalbuminemia.[12-14]

Differences in reported prevalence may reflect variation in illness severity, referral patterns, diarrhoeal burden, laboratory cut-offs and the electrolyte panels evaluated. Despite the recognised clinical importance of dyselectrolytaemia in SAM, institution-specific evidence from the Hadoti region of Rajasthan remains limited. The present study was therefore conducted to determine the prevalence and pattern of serum sodium, potassium and chloride derangements among children aged 6–59 months with severe acute malnutrition admitted to a tertiary-care pediatrics hospital in Kota.

AIMS AND OBJECTIVES

- To determine the prevalence and pattern of serum sodium, potassium and chloride derangements among children aged 6–59 months with severe acute malnutrition.
- To assess the relationship of dyselectrolytemia with anthropometric severity, diarrhea, pedal oedema, serum albumin and SAM phenotype.
- To identify independent factors associated with dyselectrolytemia.

MATERIALS AND METHODS

Study design and setting

This was a hospital-based cross-sectional observational study conducted in the Department of Paediatrics and Nutritional Rehabilitation Centre, J.K. Loan Hospital, Kota, Rajasthan, India. The study period extended from June 2024 to February 2026 after approval from the Institutional Ethics Committee. The present manuscript is restricted to the serum electrolyte component of the parent study.

Study population

Children aged 6–59 months admitted with SAM were screened for eligibility. SAM was defined according to World Health Organization criteria by the presence of any one of the following: weight-for-height/length Z-score below -3 SD, MUAC below 11.5 cm, or bilateral pitting oedema. [1,2]

Eligibility criteria
Children aged 6–59 months fulfilling WHO criteria for SAM were included. Children with chronic liver or kidney disease; chronic pulmonary disease such as cystic fibrosis; organic or metabolic disorders including glycogen storage diseases, channelopathies, muscular dystrophies, diabetes mellitus and lactose intolerance; neurodevelopmental disorders such as cerebral palsy; or absence of parental/caregiver consent were excluded.

Sample size
The final sample size was 90 children. The source protocol calculated the sample size using Z=1.96 for a 95% confidence level, p=7.9% based on the prevalence of severe wasting in Kota district, q=92.1 and an absolute precision of 6%, followed by a 10% allowance for non-response or incomplete observations.[7]

Data collection and clinical assessment
Eligible children were enrolled after written informed consent from a parent or caregiver. Demographic, socioeconomic, clinical, anthropometric, birth, feeding and nutritional information was recorded using a predesigned structured proforma. Socioeconomic status was assessed using the Modified Kuppuswamy scale. Weight, length/height and MUAC were measured using standard calibrated equipment. Bilateral pitting oedema, presenting complaints and systemic examination findings were recorded at admission. SAM phenotype was classified as marasmus, kwashiorkor or marasmic-kwashiorkor according to the clinical and anthropometric profile documented in the parent dataset.

Laboratory assessment
A 3 mL venous blood sample was collected under aseptic precautions using the spirit-betadine-spirit technique and transported immediately to the central laboratory. Baseline samples were obtained within two hours of admission to minimize the effect of in-hospital fluids, feeding and corrective treatment. Serum sodium, potassium and chloride were measured using an EM640 biochemistry autoanalyser. Serum albumin, liver enzymes and other clinically indicated investigations were recorded as supportive variables.

Operational definitions
- Hyponatremia: serum sodium <135 mEq/L.
- Hypernatremia: serum sodium >145 mEq/L.
- Hypokalemia: serum potassium <3.5 mEq/L.
- Hyperkalemia: serum potassium >5.0 mEq/L.
- Hypochloremia: serum chloride <98 mEq/L.
- Dyselectrolytemia: presence of at least one abnormal sodium, potassium or chloride value.

Statistical analysis
Continuous variables were summarized as mean±standard deviation and categorical variables as frequency and percentage. Independent-samples t-tests were used for continuous two-group comparisons and Chi-square tests for categorical associations. Spearman correlation assessed selected anthropometric-biochemical relationships. Binary logistic regression generated odds ratios (OR), adjusted odds ratios (AOR) and 95% confidence intervals (CI) for factors associated with dyselectrolytemia. One-way analysis of variance was used to compare biochemical parameters across SAM phenotypes. A p-value <0.05 was considered statistically significant. The statistical software and version were not specified in the source documents and should be inserted before journal submission.
Ethical considerations

Institutional Ethics Committee approval was obtained before study initiation, and written informed consent was obtained from parents or caregivers. The ethics approval number was not provided in the uploaded source and should be inserted in the final submission.

RESULTS
A total of 90 children with SAM were analyzed. The mean age was 24.5±12.8 months; 52 (57.8%) were male and 38 (42.2%) were female. Most participants were from rural areas and lower socioeconomic strata. The mean weight was 7.8±1.9 kg, mean weight-for-height Z-score was -3.8±0.6 and mean MUAC was 10.9±0.7 cm. Marasmus was the predominant phenotype.

Table 1. Baseline characteristics of the study participants (N=90)

Characteristic	Category/statistic	n (%) or mean±SD
Age group (months)	6–12	20 (22.2)
	13–24	26 (28.9)
	25–36	18 (20.0)
	37–48	12 (13.3)
	49–59	14 (15.6)
Age (months)	Mean±SD	24.5±12.8

Characteristic	Category/statistic	n (%) or mean±SD
Sex	Male	52 (57.8)
	Female	38 (42.2)
Residence	Rural	62 (68.9)
	Urban	28 (31.1)
Socioeconomic status	Upper middle	12 (13.3)
	Lower middle	14 (15.6)
	Upper lower	28 (31.1)
	Lower	36 (40.0)
Weight (kg)	Mean±SD	7.8±1.9
WFH Z-score	Mean±SD	-3.8±0.6
MUAC (cm)	Mean±SD	10.9±0.7
SAM phenotype	Marasmus	55 (61.1)
	Kwashiorkor	20 (22.2)
	Marasmic-kwashiorkor	15 (16.7)

The mean serum sodium was 132.5±6.2 mEq/L, mean potassium was 3.2±0.7 mEq/L and mean chloride was 98.8±7.1 mEq/L. Mean serum albumin was 2.1±0.7 g/dL, indicating substantial visceral protein depletion in the study population.

Table 2. Admission electrolyte and protein profile

Parameter	Mean±SD
Serum sodium (mEq/L)	132.5±6.2
Serum potassium (mEq/L)	3.2±0.7
Serum chloride (mEq/L)	98.8±7.1
Serum albumin (g/dL)	2.1±0.7
Total protein (g/dL)	5.4±1.2
Albumin/globulin ratio	0.9±0.3

Overall, 65 children (72.2%) had at least one electrolyte derangement. Hyponatremia was the most frequent abnormality, followed by hypokalemia and hypochloremia. Hypernatremia and hyperkalemia were uncommon. Participant-level overlap counts for isolated and combined electrolyte abnormalities were not available in the supplied manuscript; therefore, the pattern is presented by individual electrolyte abnormality without inventing combination frequencies.

Table 3. Prevalence and pattern of serum electrolyte derangements (N=90)

Electrolyte	Abnormality	n (%)
Sodium	Hyponatremia (<135 mEq/L)	50 (55.6)
	Hypernatremia (>145 mEq/L)	2 (2.2)
Potassium	Hypokalaemia (<3.5 mEq/L)	42 (46.7)
	Hyperkalaemia (>5.0 mEq/L)	1 (1.1)
Chloride	Hypochloraemia (<98 mEq/L)	30 (33.3)
Composite	Any dyselectrolytemia	65 (72.2)

Dyselectrolytemia was defined as at least one abnormal serum sodium, potassium or chloride value. Hypokalemia increased progressively with worsening MUAC category: 60.0% among children with MUAC <10.5 cm, 42.3% among those with MUAC 10.5–11.5 cm and 25.0% among children with MUAC >11.5 cm who qualified through weight-for-height criteria ($p<0.001$).

Table 4. Hypokalaemia according to MUAC category

MUAC category	Hypokalaemia, n/N (%)	p-value
<10.5 cm	18/30 (60.0)	<0.001
10.5–11.5 cm	22/52 (42.3)	
>11.5 cm with WFH Z-score <-3 SD	2/8 (25.0)	

Compared with children without dyselectrolytemia, affected children had significantly lower MUAC and WFH Z-scores, more frequent pedal oedema and diarrhea, lower serum albumin and higher SGOT. Age did not differ significantly between the groups.

Table 5. Comparison of children with and without dyselectrolytemia

Variable	Dyselectrolytemia present (n=65)	Dyselectrolytemia absent (n=25)	p-value
Age (months)	23.5±12.1	27.8±14.2	0.08

Variable	Dyselectrolytemia present (n=65)	Dyselectrolytemia absent (n=25)	p-value
MUAC (cm)	10.5±0.6	11.6±0.5	<0.001
WFH Z-score	-3.9±0.5	-3.3±0.4	0.002
Pedal oedema present	28 (43.1%)	7 (28.0%)	0.02
Diarrhoea at admission	35 (53.8%)	7 (28.0%)	0.01
Serum albumin (g/dL)	1.9±0.6	2.7±0.5	<0.001
SGOT (IU/L)	52±24	40±18	0.03

Serum sodium showed a moderate positive correlation with serum albumin (r=0.48, p<0.001), while serum potassium correlated positively with MUAC (r=0.36, p=0.001). These relationships indicate that worsening nutritional and protein status accompanied lower electrolyte values.

Table 6. Selected correlations relevant to serum electrolyte status

Variables	Correlation coefficient (r)	p-value
Serum sodium and serum albumin	0.48	<0.001
Serum potassium and MUAC	0.36	0.001
Serum albumin and MUAC	0.52	<0.001

On univariate logistic regression, low MUAC, pedal oedema, diarrhea and serum albumin <2.0 g/dL were associated with dyselectrolytemia. In the multivariate model, serum albumin <2.0 g/dL, diarrhea and MUAC <11.5 cm remained independently associated, whereas pedal oedema did not.

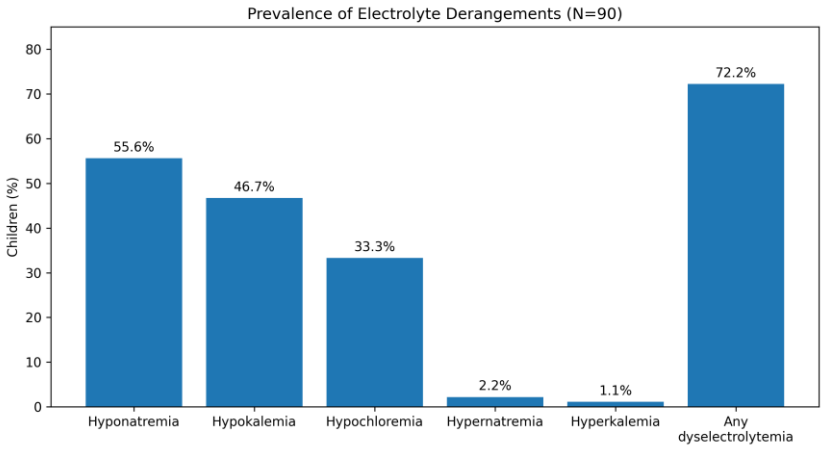
Table 7. Logistic regression analysis of factors associated with dyselectrolytemia

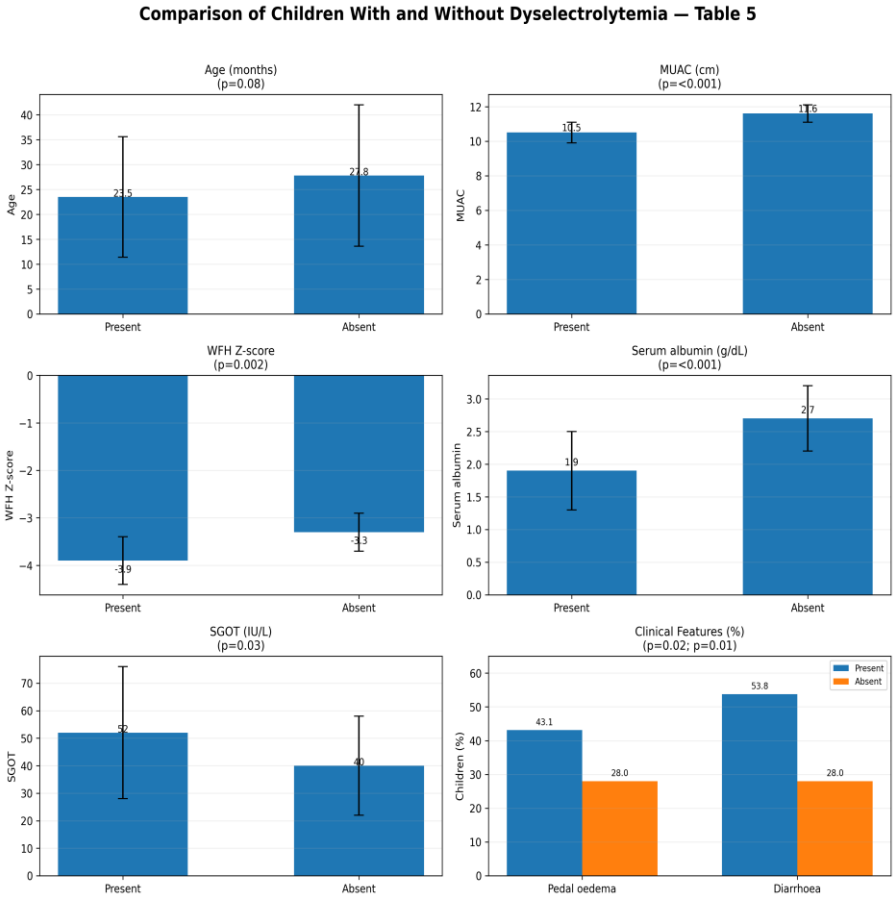
Predictor	Unadjusted OR (95% CI)	p-value	Adjusted OR (95% CI)	p-value
MUAC <11.5 cm	4.0 (1.8–9.1)	0.001	2.5 (1.0–6.3)	0.047
Pedal oedema	3.5 (1.2–10.3)	0.020	1.8 (0.6–5.5)	0.290
Diarrhoea at admission	4.3 (1.8–10.1)	0.001	3.0 (1.1–8.2)	0.029
Serum albumin <2.0 g/dL	6.5 (2.5–16.8)	<0.001	4.4 (1.6–11.8)	0.003

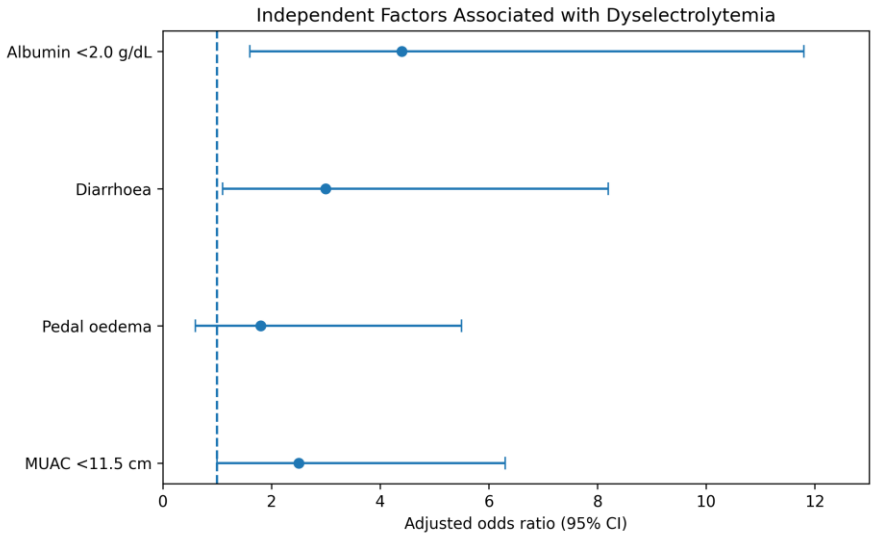
Electrolyte values differed significantly across SAM phenotypes. Children with kwashiorkor had the lowest mean sodium and potassium, followed by those with marasmic-kwashiorkor; children with marasmus had comparatively higher values. Serum albumin was also lowest in kwashiorkor.

Table 8. Biochemical profile according to SAM phenotype

Parameter	Marasmus (n=55)	Kwashiorkor (n=20)	Marasmic-kwashiorkor (n=15)	p-value
Serum albumin (g/dL)	2.3±0.5	1.6±0.4	1.8±0.5	<0.001
Serum sodium (mEq/L)	133.6±5.4	129.6±6.8	131.0±6.1	0.03
Serum potassium (mEq/L)	3.3±0.6	2.9±0.7	3.0±0.7	0.02







DISCUSSION

The present study demonstrates that serum electrolyte abnormalities are a common biochemical feature among hospitalized children with severe acute malnutrition (SAM). Nearly three-fourths of the study population had at least one sodium, potassium, or chloride abnormality at admission. Hyponatraemia was the most frequent abnormality, followed by hypokalaemia and hypochloraemia, whereas hypernatraemia and hyperkalaemia were uncommon. These findings indicate a substantial burden of electrolyte disturbance among hospitalized children with SAM, with a clear predominance of low serum electrolyte concentrations.

The overall prevalence of dyselectrolytaemia in the present study was 72.2%, which is consistent with the high burden reported in previous studies, although considerable variation exists between different cohorts. Raza et al. reported electrolyte abnormalities in more than 90% of hospitalized children with SAM, with hypokalaemia, hypocalcaemia, and hyponatraemia occurring frequently.[10] Similarly, Azizullah et al. documented a high prevalence of electrolyte disturbances among children admitted to a nutritional stabilisation centre.[14] Studies from India and neighbouring

countries have also reported frequent sodium and potassium abnormalities, particularly among children with complicated SAM and those presenting with diarrhoea.[11-17] A prospective Ethiopian study published in 2026 further demonstrated an association between admission electrolyte abnormalities and treatment outcomes in hospitalized children with SAM.[18] Differences in prevalence across studies may be explained by variations in the electrolytes assessed, biochemical cut-off values, inclusion criteria, clinical severity, prevalence of diarrhoeal illness, and treatment setting. Some studies have also included calcium and magnesium abnormalities within the definition of dyselectrolytaemia, making direct comparison of prevalence estimates difficult.

Hyponatraemia was observed in 55.6% of participants and was the most common electrolyte abnormality in the present study. Several mechanisms may contribute to low serum sodium concentrations in children with SAM, including gastrointestinal sodium losses, infection-associated non-osmotic release of antidiuretic hormone, altered renal sodium handling, changes in fluid distribution associated with oedema, and previous administration of hypotonic fluids.[4-6,10-14] Importantly, serum sodium concentration does not necessarily reflect total-body sodium stores in malnourished children, particularly in the presence of oedema. Nevertheless, clinically significant hyponatraemia may contribute to lethargy, altered consciousness, seizures, and circulatory instability, particularly when severe or when corrected inappropriately. The high frequency observed in this study therefore supports the importance of biochemical assessment rather than relying solely on clinical signs for identification of electrolyte abnormalities.

Hypokalaemia was present in 46.7% of children. Potassium depletion is well recognised in SAM and may result from reduced dietary intake, loss of lean tissue mass, gastrointestinal losses, and altered cellular potassium distribution.[3,4,12,19,20] Diarrhoea and vomiting may further aggravate potassium losses, while changes during initial treatment and refeeding may also influence serum potassium concentrations. Clinically important hypokalaemia may lead to muscle weakness, gastrointestinal hypomotility, respiratory muscle dysfunction, and cardiac arrhythmias. In the present study, the frequency of hypokalaemia increased from 25.0% in the least severely depleted MUAC category to 60.0% among children with MUAC <10.5 cm. Although the cross-sectional design prevents establishing a temporal or causal relationship, this pattern suggests that more severe anthropometric wasting is associated with a greater likelihood of hypokalaemia and is biologically consistent with the substantial depletion of lean tissue and intracellular potassium stores that accompanies severe malnutrition.

Hypochloraemia was identified in approximately one-third of the study population. Chloride abnormalities have received less attention than sodium and potassium disturbances in the SAM literature, although chloride plays an important role in maintaining electroneutrality and acid–base balance.[6,10,13-18] Low serum chloride may occur in association with sodium depletion, gastrointestinal losses, vomiting, diarrhoea, and alterations in hydration status. The simultaneous predominance of hyponatraemia, hypokalaemia, and hypochloraemia suggests that electrolyte disturbances in hospitalized children with SAM are not confined to a single electrolyte but may represent a broader disruption of fluid, electrolyte, and metabolic homeostasis.

Nutritional severity was significantly associated with electrolyte status. Children with dyselectrolytaemia had a lower mean MUAC and a more negative WFH Z-score than children without electrolyte abnormalities. In addition, serum potassium showed a positive correlation with MUAC, suggesting that greater preservation of muscle and tissue mass was associated with better potassium status. Similar relationships between biochemical abnormalities and nutritional or clinical severity have been reported in previous hospital-based studies of children with SAM.[11,12,17,18] These findings support the concept that severe anthropometric depletion and biochemical instability frequently coexist. However, because the present study was cross-sectional, the direction of this relationship cannot be established.

Diarrhoea was independently associated with the presence of dyselectrolytaemia after adjustment for other relevant factors. This association is clinically plausible because children with SAM have limited physiological reserves, while diarrhoea may result in additional sodium, potassium, and chloride losses, reduced oral intake, impaired absorption, and disturbances in hydration. Previous studies among malnourished children with acute diarrhoea have demonstrated frequent sodium and potassium abnormalities, while studies evaluating modified rehydration approaches have highlighted the importance of carefully tailored fluid and electrolyte replacement in this population.[13,19,21] The present findings therefore emphasise the importance of prompt electrolyte assessment in children with SAM who present with diarrhoea, together with careful assessment of hydration status and appropriate rehydration therapy.

Serum albumin concentration <2.0 g/dL emerged as the strongest independent factor associated with dyselectrolytaemia. Serum sodium also showed a positive correlation with albumin, and children with electrolyte abnormalities had substantially lower mean albumin concentrations than those without abnormalities. Hypoalbuminaemia in SAM reflects multiple processes rather than protein deficiency alone and may be influenced by inflammation, impaired hepatic protein synthesis, increased capillary permeability, altered fluid distribution, and dilutional effects associated with oedema. Hospital-based studies have similarly reported associations between hypoalbuminaemia, biochemical abnormalities, and greater clinical severity in children with SAM.[11-13,17,18] Thus, the association between low albumin and dyselectrolytaemia should not be interpreted as evidence of a direct causal relationship. Rather, low albumin may serve as

an accessible marker of underlying nutritional and systemic severity and may help identify children who are at greater risk of biochemical instability.

The analysis according to SAM phenotype demonstrated lower serum sodium, potassium, and albumin concentrations among children with kwashiorkor and marasmic-kwashiorkor compared with those with marasmus. Oedematous forms of SAM are associated with substantial alterations in protein metabolism, fluid distribution, and cellular function, which may contribute to differences in biochemical profiles between phenotypes.[3,12,18] Kumar et al. similarly reported a more pronounced biochemical derangement among children with oedematous SAM.[12] These findings suggest that children with kwashiorkor and marasmic-kwashiorkor may require particularly close biochemical monitoring during hospitalisation. However, the present analysis was based on an overall one-way analysis of variance (ANOVA), and because post-hoc pairwise comparisons were not reported, the findings should be interpreted as demonstrating an overall difference between phenotype groups rather than confirming statistically significant differences between every individual pair of phenotypes.

The clinical implications of these findings are important. Anthropometric assessment remains central to the diagnosis and classification of SAM, but anthropometric measurements alone may not fully capture the immediate biochemical risk of a hospitalized child. Measurement of serum sodium, potassium, and chloride at admission can identify clinically occult abnormalities and may assist in the selection of appropriate fluids, electrolyte supplementation, feeding strategies, and monitoring intensity.[2,4,5] Recent paediatric evidence from resource-constrained inpatient settings has also demonstrated that electrolyte abnormalities are common and may coexist with adverse clinical outcomes.[22,23] Nevertheless, the present cross-sectional study does not establish that routine electrolyte testing independently improves morbidity or mortality. Rather, the high prevalence of abnormalities observed provides a strong rationale for incorporating electrolyte assessment into the initial evaluation of hospitalized children with complicated SAM, particularly those with severe wasting, diarrhoea, oedema, or marked hypoalbuminaemia.

Overall, the findings indicate that electrolyte disturbances constitute an important component of the biochemical burden of hospitalized children with SAM. The predominance of hyponatraemia, hypokalaemia, and hypochloraemia, together with their association with anthropometric severity, diarrhoea, and hypoalbuminaemia, highlights the close relationship between nutritional depletion, intercurrent illness, and biochemical instability. Recognition of these abnormalities at admission may therefore facilitate more individualised monitoring and supportive management during inpatient treatment.

CONCLUSION

Serum electrolyte derangements were present in 72.2% of hospitalized children aged 6–59 months with severe acute malnutrition (SAM). Hyponatremia, hypokalemia, and hypochloremia were the predominant abnormalities, whereas hypernatremia and hyperkalemia were uncommon. Greater anthropometric depletion, diarrhoea, and severe hypoalbuminemia were associated with an increased risk of dyselectrolytaemia, while children with oedematous SAM phenotypes demonstrated more pronounced sodium and potassium abnormalities. These findings highlight the importance of incorporating routine assessment of serum sodium, potassium, and chloride into the initial evaluation of hospitalized children with SAM, in addition to anthropometric and clinical assessment. Particular attention should be given to children presenting with diarrhoea, severe wasting, oedema, or serum albumin <2.0 g/dL, as these features may identify children at greater risk of electrolyte disturbances and who may require closer biochemical monitoring and appropriately tailored supportive management.

DECLARATIONS

Conflict of Interest: None.
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Ethical Approval: Obtained.
Consent: Written informed consent was obtained from parents/caregivers.

REFERENCES

1. World Health Organization. Identification of severe acute malnutrition in children 6–59 months of age [Internet]. 2025
2. World Health Organization. WHO guideline on the prevention and management of wasting and nutritional oedema (acute malnutrition) in infants and children under 5 years. Web Annex C. GRADE evidence profiles. World Health Organization; 2024 Mar 1.
3. Dipasquale V, Cucinotta U, Romano C. Acute malnutrition in children: pathophysiology, clinical effects and treatment. Nutrients. 2020 Aug 12;12(8):2413.
4. World Health Organization. Pocket book of hospital care for children: guidelines for the management of common childhood illnesses. World Health Organization; 2013.
5. Bhan MK, Bhandari N, Bahl R. Management of the severely malnourished child: perspective from developing countries. Bmj. 2003 Jan 18;326(7381):146-51.
6. Zieg J, Ghose S, Raina R. Electrolyte disorders related emergencies in children. BMC nephrology. 2024 Aug 30;25(1):282.

7. Kabdwal NC, Yadav NK, Purwar N, Gautam V. Changing Patterns of Child Malnutrition in India: A Comparative Analysis Across NFHS-3, NFHS-4 and NFHS-5.
8. World Health Organization. Levels and trends in child malnutrition child malnutrition: UNICEF/WHO/World Bank Group Joint Child Malnutrition Estimates: Key findings of the 2023 edition. World Health Organization; 2023 May 24.
9. Mishra K, Kumar P, Basu S, Rai K, Aneja S. Risk factors for severe acute malnutrition in children below 5 y of age in India: a case-control study. The Indian Journal of Pediatrics. 2014 Aug;81(8):762-5.
10. Raza M, Kumar S, Ejaz M, Azim D, Azizullah S, Hussain A. Electrolyte imbalance in children with severe acute malnutrition at a tertiary care hospital in Pakistan: a cross-sectional study. Cureus. 2020 Sep 19;12(9).
11. Verma GK, Yadv RK, Chand R, Khan IA, Katiyar SB, Singh M, Verma G, Yadav RK, Khan IA, Katiyar SB. Prognostic significance of serum biochemistry profile in children with severe acute malnutrition. Cureus. 2022 Nov 8;14(11).
12. Kumar D, Rao SK, Singh TB. Clinico-biochemical profile of sick children with severe acute malnutrition. Journal of Family Medicine and Primary Care. 2020 May 31;9(5):2269.
13. Ahmad F, Hossain MA, Islam MN, Chowdhury B, Aktar A, Amin SE, Kabir MA, Tusnim I, Alam MT, Pandit P. Serum Electrolyte, Serum Calcium and Blood Sugar Status in Severe Acute Malnutrition and Their Relationship with Edema, Vomiting and Diarrhea. Mymensingh Medical Journal: MMJ. 2022 Apr 1;31(2):281-8.
14. Chand S, Shah MA, Radhan AH, Baloch HN, Chohan MN. Prevalence and Patterns of Electrolyte Imbalance in Children Diagnosed with Acute Severe Malnutrition at a Nutritional Stabilization Center: Electrolyte Imbalance in Severe Acute Malnutrition. Pakistan Journal of Health Sciences. 2024 Sep 30:96-100.
15. Owais MJ, Sridhar NL. A study of serum electrolytes in malnourished children. Asian Journal of Clinical Pediatrics and Neonatology. 2020 Jan;8:41-3.
16. Dakshayani B, Ravi M, Premalatha R. A study of serum electrolytes in severe acute malnourished children with and without complications. Indian J Child Health. 2018 Feb;5(2):120-3.
17. Barman M, Rabha J, Rohman SM. Changes in biochemical profiles of children with severe acute malnutrition on admission and after nutritional rehabilitation. Indian J Child Health. 2023;10(4):40-6.
18. Hailu M, Kassahun W, Yesuf HA, Lingerew A. Serum electrolyte disturbances and treatment outcomes in hospitalized children with Severe Acute Malnutrition in Ethiopia. Scientific Reports. 2026 May 10.
19. Bilal A, Sadiq MA, Haider N. Frequency of hyponatremia and hypokalemia in malnourished children with acute diarrhea. J Pak Med Assoc. 2016 Sep 1;66(9):1077-80.
20. Alasad SM, Salih OA, Hassan M. Insight into potassium's role in childhood mortality due to severe acute malnutrition. Sudanese journal of paediatrics. 2019;19(1):44.
21. Alam NH, Hamadani JD, Dewan N, Fuchs GJ. Efficacy and safety of a modified oral rehydration solution (ReSoMaL) in the treatment of severely malnourished children with watery diarrhea. The Journal of pediatrics. 2003 Nov 1;143(5):614-9.
22. Gupta A, Kasundriya S, Shrivastava S, Purohit M, Pathak A. Prevalence of Electrolyte Abnormality and its Correlation with Clinical Features and Patient Outcomes in Children Admitted to Pediatric Intensive Care Unit of a Resource-Constrained Setting in India. SAGE Open Medicine. 2025 Nov;13:20503121251391990.
23. Das K, Das S, Mohapatra S, Swain A, Mohakud NK. Risk and adverse outcome factors of severe acute malnutrition in children: a Hospital-Based Study in Odisha. Cureus. 2021 Sep 28;13(9).