



Original Article

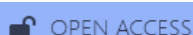
Serum Electrolyte and Blood Glucose Derangements in Children Aged 6-59 Months with Severe Acute Malnutrition: A Cross-Sectional Study

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ABSTRACT

Introduction: Severe acute malnutrition (SAM) is the most life-threatening form of undernutrition in children under five years and is frequently accompanied by clinically important electrolyte and glycaemic disturbances. These abnormalities may worsen neurological status, cardiac rhythm, tissue perfusion and short-term outcomes if not identified early.

Objectives: To determine the frequency of dyselectrolytemia and blood sugar derangements in children aged 6-59 months with severe acute malnutrition.

Methods: This hospital-based cross-sectional observational study was conducted in the Department of Pediatrics at a tertiary care centre in Kota, Rajasthan, from June 2024 to February 2026 after Institutional Ethics Committee approval. Ninety children aged 6–59 months fulfilling World Health Organization criteria for SAM were enrolled. Demographic, clinical, anthropometric and nutritional data were recorded. Serum sodium, potassium and chloride and capillary blood glucose were assessed at admission. Descriptive statistics, independent-samples t tests, chi-square tests or Fisher exact tests, as appropriate, and correlation analysis were used.

Results: Hyponatraemia was observed in 50 (55.6%) children, hypokalaemia in 42 (46.7%) and hypochloraemia in 30 (33.3%). Hypoglycaemia was present in 14 (15.6%), including severe hypoglycaemia in 3 (3.3%); hyperglycaemia was present in 4 (4.4%). Overall, 65 (72.2%) children had dyselectrolytaemia, 18 (20.0%) had a glycaemic derangement and 72 (80.0%) had any metabolic derangement. Dyselectrolytaemia was associated with lower MUAC, lower weight-for-height Z-score, diarrhoea, lower serum albumin and higher SGOT. Hypoglycaemia was associated with lower MUAC, diarrhoea and lower serum albumin.

Conclusion: Electrolyte and blood glucose derangements were common among hospitalised children with SAM. Hyponatraemia, hypokalaemia and hypoglycaemia were clinically important and were associated with greater nutritional and biochemical vulnerability. Early admission screening may support safer fluid, feeding and corrective strategies.

Keywords: Severe acute malnutrition; serum electrolytes; blood glucose; hyponatraemia; hypokalaemia; hypoglycaemia; children.

INTRODUCTION

Severe acute malnutrition (SAM) represents the most life-threatening form of childhood undernutrition and commonly affects children aged 6–59 months. It is defined by a weight-for-height/length Z-score below -3 standard deviations, a mid-upper arm circumference (MUAC) below 11.5 cm, or bilateral pitting oedema.¹ SAM is a systemic condition associated with profound metabolic adaptation, reduced lean body mass, impaired handling of solutes, depleted glycogen stores and reduced physiological reserve.

Serum electrolytes and blood glucose are central to osmotic balance, neuromuscular excitability, membrane potential, cardiac function and tissue perfusion. In children with SAM, intercurrent illnesses such as diarrhea, pneumonia, sepsis, vomiting and poor feeding can rapidly precipitate hyponatremia, hypernatremia, hypokalemia, chloride disturbances and hypoglycemia. These abnormalities are often clinically silent or masked by the altered physiological responses of malnutrition, yet they may contribute to seizures, arrhythmias, altered sensorium, circulatory compromise and avoidable mortality.

Electrolyte abnormalities may also carry prognostic importance across other acute metabolic disorders. In a recent prospective study of adults with diabetic ketoacidosis, Satish et al. documented frequent electrolyte disturbances and reported associations of hyperkalaemia and hypomagnesaemia with adverse clinical outcomes. Although that population is not directly comparable with children with SAM, the study reinforces the broader clinical importance of early electrolyte assessment in acute metabolic illness.²

The burden of childhood undernutrition remains substantial in India. National Family Health Survey-5 data document persistent stunting, wasting and underweight among children under five years, including a considerable burden in Rajasthan.³ This regional burden translates into a clinically important load of SAM admissions in tertiary paediatric hospitals.

Despite established protocols for inpatient SAM management, important gaps remain in the systematic identification and interpretation of electrolyte and glucose abnormalities at admission. Standard signs of dehydration and shock may be unreliable in malnourished children, and inappropriate fluid or feeding strategies can worsen electrolyte imbalance or precipitate metabolic complications. Therefore, biochemical assessment must complement anthropometry and clinical evaluation during early stabilization.

Previous studies have documented frequent electrolyte disturbances among hospitalised children with SAM, particularly hyponatraemia and hypokalaemia, with greater abnormalities in children with diarrhoea, oedema or more severe wasting.⁴

⁸ Context-specific data from the Hadoti region of Rajasthan remain limited. This study therefore assessed the frequency and pattern of serum electrolyte and blood glucose derangements among children aged 6–59 months with SAM admitted at the study centre.

MATERIALS & METHODS

Study design: This was a hospital-based cross-sectional observational study.

Study period: The study was conducted from June 2024 to February 2026 after obtaining approval from the Institutional Ethics Committee.

Place of study: The study was conducted in the Department of Paediatrics, J.K. Loan Hospital, Kota.

Study population: Children aged 6-59 months admitted to J.K. Loan Hospital with severe acute malnutrition were included.

Sample size: The final sample size was 90 children. Sample size was calculated using $Z=1.96$ for 95% confidence interval, $p=7.9$ based on prevalence of severe wasting in Kota district as per NFHS-5 (2019-2021), $q=92.1$ and $d=6$, with addition of 10% for dropouts.

Inclusion criteria: Children aged 6-59 months fulfilling WHO criteria for SAM were included if they had any of the following: weight-for-height/length < -3 SD, MUAC < 11.5 cm, or bilateral pitting oedema.

Exclusion criteria: Children with chronic liver or kidney disease, chronic pulmonary diseases such as cystic fibrosis, organic and metabolic disorders including glycogen storage diseases, channelopathies, muscular dystrophies, diabetes mellitus and lactose intolerance, neurodevelopmental disorders such as cerebral palsy, and children whose parents did not give consent were excluded.

Data collection: All children admitted to the Nutritional Rehabilitation Center were screened according to inclusion and exclusion criteria. Eligible children were enrolled after written informed consent from parents or caregivers. Baseline demographic, clinical, anthropometric, nutritional and feeding-related details were recorded using a predesigned structured proforma. Socioeconomic status was assessed using the Modified Kuppuswamy scale.

Laboratory assessment: Blood samples of 3 ml were collected under aseptic precautions and transported immediately to the central laboratory. Serum sodium, potassium, chloride, capillary blood glucose, renal profile, haematological parameters, liver enzymes and protein profile were tested.

Statistical analysis: Data were summarised as mean $\pm$ standard deviation or frequency and percentage. Independent-samples t tests were used for continuous variables. Categorical variables were compared using chi-square tests or Fisher exact tests, as appropriate. Correlation analysis examined relationships between anthropometric and biochemical variables. All tests were two-sided, and $p < 0.05$ was considered statistically significant.

RESULTS

The mean age was 24.5 $\pm$ 12.8 months, with the highest proportion belonging to the 13-24 months age group [26 (28.9%)], followed by 6-12 months [20 (22.2%)]. Males constituted 52 (57.8%) children and females 38 (42.2%). Most children were from rural areas [62 (68.9%)] and belonged predominantly to lower socioeconomic strata, with 36 (40.0%) in the lower class and 28 (31.1%) in the upper-lower class. The mean weight was 7.8 $\pm$ 1.9 kg, mean weight-for-height Z-score was -3.8 $\pm$ 0.6, and mean MUAC was 10.9 $\pm$ 0.7 cm. Marasmus was the most common SAM phenotype [55 (61.1%)], followed by kwashiorkor [20 (22.2%)] and marasmic-kwashiorkor [15 (16.7%)]. Admission biochemical assessment showed low mean serum sodium, potassium and albumin levels, indicating biochemical vulnerability among children with SAM. Mean capillary blood glucose was 78.6 $\pm$ 28.4 mg/dL, while mean serum albumin was 2.1 $\pm$ 0.7 g/dL, reflecting poor metabolic and visceral protein reserve.

Hyponatremia was the most common electrolyte abnormality (55.6%), followed by hypokalemia (46.7%) and hypochloremia (33.3%). Glycemic derangement was observed in 20.0% of children. Overall, 72.2% had dyselectrolytemia, and 80.0% had at least one metabolic abnormality.

Hypokalaemia showed a graded numerical increase with worsening MUAC category, but the displayed three-group comparison was not statistically significant ($p = 0.132$). Dyselectrolytaemia was associated with lower MUAC, lower WFH Z-score, diarrhoea, lower albumin and higher SGOT; pedal oedema was not significantly associated after recalculation from the displayed counts. Hypoglycaemia was associated with lower MUAC, diarrhoea and lower albumin.

Table 1. Sociodemographic Profile of Study Participants (N = 90)

Variable	Category	n	%
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
	Mean $\pm$ SD (Months)	24.5 $\pm$ 12.8	
Sex	Male	52	57.8
	Female	38	42.2
Socioeconomic Status (Modified Kuppuswamy Scale)	Upper Middle	12	13.3
	Lower Middle	14	15.6
	Upper Lower	28	31.1
	Lower	36	40

Table 2: Admission biochemical profile among children with SAM

Parameter	Mean $\pm$ SD / n (%)
Serum sodium, mEq/L	132.5 $\pm$ 6.2
Serum potassium, mEq/L	3.2 $\pm$ 0.7
Serum chloride, mEq/L	98.8 $\pm$ 7.1
Capillary blood glucose, mg/dL	78.6 $\pm$ 28.4
Haemoglobin, g/dL	8.6 $\pm$ 1.7
WBC count, /uL	11,200 $\pm$ 4,500
Platelet count, /uL	280,000 $\pm$ 75,000
Haematocrit, %	27.8 $\pm$ 5.2
MCV, fL	72 $\pm$ 8
Serum albumin, g/dL	2.1 $\pm$ 0.7
Total protein, g/dL	5.4 $\pm$ 1.2
A/G ratio	0.9 $\pm$ 0.3

Table 3. Distribution of Metabolic Abnormalities among Children with SAM (N = 90)

Parameter	Abnormality	n (%)
Serum sodium	Hyponatremia (<135 mEq/L)	50 (55.6)
	Hypernatremia (>145 mEq/L)	2 (2.2)
Serum potassium	Hypokalemia (<3.5 mEq/L)	42 (46.7)

	Hyperkalemia (>5.0 mEq/L)	1 (1.1)
Serum chloride	Hypochloremia (<98 mEq/L)	30 (33.3)
Blood glucose	Hypoglycemia (<70 mg/dL)	14 (15.6)
	Severe hypoglycemia (<40 mg/dL)*	3 (3.3)
	Hyperglycemia (>150 mg/dL)	4 (4.4)
Composite outcomes	Any dyselectrolytemia	65 (72.2)
	Any glycemic derangement	18 (20.0)
	Any metabolic derangement	72 (80.0)

*Severe hypoglycemia was included within the hypoglycemia group.

Table 4: Association of nutritional and clinical parameters with dyselectrolytemia and hypoglycemia

Analysis	Variable	Affected group	Comparator group	p-value
Hypokalemia by MUAC	MUAC <10.5 cm	18/30 (60.0%)	-	0.132
Hypokalemia by MUAC	MUAC 10.5-11.5 cm	22/52 (42.3%)	-	
Hypokalemia by MUAC	MUAC >11.5 cm with WFH <- 3 SD	2/8 (25.0%)	-	
Dyselectrolytemia	Age, months	23.5 +/- 12.1	27.8 +/- 14.2	0.189
Dyselectrolytemia	MUAC, cm	10.5 +/- 0.6	11.6 +/- 0.5	<0.001
Dyselectrolytemia	WFH Z-score	-3.9 +/- 0.5	-3.3 +/- 0.4	<0.001
Dyselectrolytemia	Pedal oedema present	28 (43.1%)	7 (28.0%)	0.189
Dyselectrolytemia	Diarrhea on admission	35 (53.8%)	7 (28.0%)	0.028
Dyselectrolytemia	Serum albumin, g/dL	1.9 +/- 0.6	2.7 +/- 0.5	<0.001
Dyselectrolytemia	SGOT, IU/L	52 +/- 24	40 +/- 18	0.013
Hypoglycemia	MUAC, cm	10.3 +/- 0.6	11.0 +/- 0.7	<0.001
Hypoglycemia	Diarrhea present	11 (78.6%)	31 (40.8%)	0.009
Hypoglycemia	Albumin, g/dL	1.7 +/- 0.5	2.2 +/- 0.7	0.004
Hypoglycemia	Capillary glucose, mg/dL	48 +/- 12	85 +/- 26	<0.001

Table 5. Correlation between anthropometric and biochemical parameters

Variables	Correlation coefficient (r)	p-value
Serum sodium and serum albumin	0.48	<0.001
Serum potassium and MUAC	0.36	0.001
Capillary blood glucose and MUAC	0.29	0.005
Serum albumin and MUAC	0.52	<0.001
WFH Z-score and capillary blood glucose	0.33	0.002

Correlation of Anthropometric and Biochemical Parameters

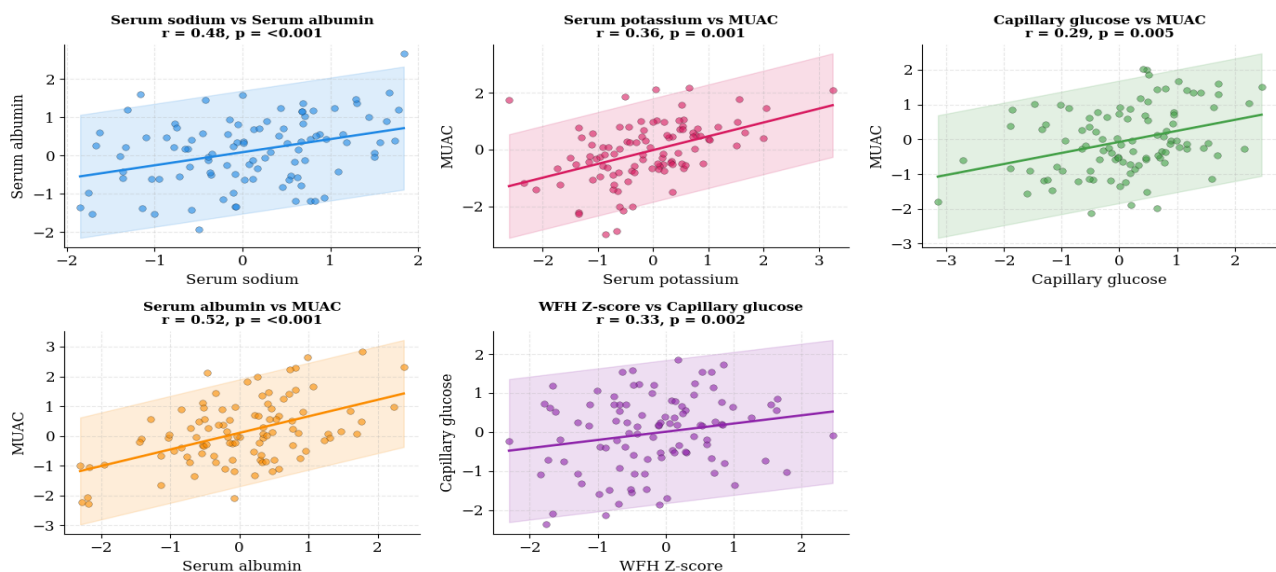


Figure 1: Correlation of anthropometric and biochemical parameters

Table 6. Biochemical profile according to SAM phenotype

Domain	Variable	Marasmus	Kwashiorkor	Marasmic-kwashiorkor	p-value
Biochemical profile	Albumin, g/dL	2.3 +/- 0.5	1.6 +/- 0.4	1.8 +/- 0.5	<0.001
Biochemical profile	Sodium, mEq/L	133.6 +/- 5.4	129.6 +/- 6.8	131.0 +/- 6.1	0.03
Biochemical profile	Potassium, mEq/L	3.3 +/- 0.6	2.9 +/- 0.7	3.0 +/- 0.7	0.02
Biochemical profile	Glucose, mg/dL	84 +/- 26	68 +/- 22	72 +/- 24	0.04

DISCUSSION

The present hospital-based cross-sectional study evaluated serum electrolyte and blood glucose derangements in 90 children aged 6–59 months with SAM. Most participants were young and came from socioeconomically disadvantaged households, consistent with the recognised clustering of SAM where food insecurity, suboptimal feeding, recurrent infection and delayed access to care coexist.^{3,9}

The central finding was the high burden of dyselectrolytaemia (72.2%). Hyponatraemia affected 55.6%, hypokalaemia 46.7% and hypochloraemia 33.3%; hypernatraemia and hyperkalaemia were uncommon. These findings are clinically important because sodium and chloride regulate extracellular osmolality and intravascular volume, while potassium is essential for cardiac and neuromuscular membrane stability. Frequent electrolyte abnormalities among children with SAM have also been reported by Raza et al., Verma et al., Saini et al., Kumar et al. and Owais and Sridhar.^{4–8}

Glycaemic derangement was less frequent than electrolyte disturbance but remained clinically relevant. Hypoglycaemia affected 14 children (15.6%), including 3 (3.3%) with severe hypoglycaemia, while hyperglycaemia occurred in 4 (4.4%). Bandsma et al. described impaired endogenous glucose production in malnourished children, supporting the biological plausibility of hypoglycaemia in the setting of depleted energy reserves.¹⁰ Saini et al. likewise reported reduced blood glucose among children with SAM.⁶

Metabolic abnormalities increased with nutritional severity. Children with dyselectrolytaemia had lower mean MUAC and WFH Z-scores. Hypokalaemia increased numerically across worsening MUAC categories, although the recalculated three-group comparison was not significant. Prior studies similarly describe substantial electrolyte disturbance in SAM, with heterogeneity according to case mix and diarrhoeal illness.^{4,5,7,8}

Serum albumin was lower among children with dyselectrolytaemia and among those with hypoglycaemia. This finding supports the use of hypoalbuminaemia as a marker of greater nutritional and clinical vulnerability, but the cross-sectional design does not establish causality.^{5–7}

The biochemical profile differed across SAM phenotypes. Oedematous phenotypes showed lower mean albumin, sodium, potassium and glucose values than marasmus. Similar biochemical vulnerability in clinically severe SAM has been described previously.⁷

The clinical implication is that children admitted with SAM require early biochemical assessment in addition to anthropometry. Admission measurement of serum sodium, potassium and chloride and capillary blood glucose may identify children requiring careful fluid selection, timely correction, close monitoring and feeding adjustment. Rehydration and electrolyte correction must remain cautious and protocol guided in SAM.¹¹

LIMITATIONS

This single-centre study had a modest sample size and included only hospitalised children, which limits generalisability. The cross-sectional design does not establish temporal or causal relationships. Biochemical measurements were obtained at admission without serial assessment.

CONCLUSION

This study found a high frequency of electrolyte and blood glucose derangements among children aged 6–59 months with SAM. Hyponatraemia and hypokalaemia were the leading electrolyte abnormalities, while hypoglycaemia affected a clinically important minority. Greater abnormalities were observed among children with lower anthropometric indices, diarrhoea and hypoalbuminaemia. These findings support routine admission biochemical screening and targeted management of serum electrolytes and blood glucose in hospitalised children with SAM.

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REFERENCES

1. World Health Organization, United Nations Children's Fund. WHO child growth standards and the identification of severe acute malnutrition in infants and children. Geneva: World Health Organization; 2009.
2. Satish BV, Amrut BR, Gowda SS. Electrolyte abnormalities in diabetic ketoacidosis and their clinical implications: a prospective observational study. *Int J Med Pharm Res.* 2026;7(3):1064-7.
3. International Institute for Population Sciences (IIPS), ICF. National Family Health Survey (NFHS-5), 2019–21: Rajasthan. Mumbai: IIPS; 2021.
4. 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;12(9):e10541. doi:10.7759/cureus.10541.
5. Verma GK, Yadav RK, Chand R, Khan IA, Katiyar SB, Singh M, et al. Prognostic significance of serum biochemistry profile in children with severe acute malnutrition. *Cureus.* 2022;14(11):e31266. doi:10.7759/cureus.31266.
6. Saini A, Agarwal P, Rakholia R. Study of clinico-demographic profile, serum electrolytes, blood sugar, and albumin levels in children with malnutrition. *Int J Med Pharm Res.* 2025;6(5):836–42. doi:10.5281/zenodo.17347167.
7. Kumar D, Rao SK, Singh TB. Clinico-biochemical profile of sick children with severe acute malnutrition. *J Family Med Prim Care.* 2020;9(5):2269–72.
8. Owais MJ, Sridhar NL. A study of serum electrolytes in malnourished children. *Asian J Clin Pediatr Neonatol.* 2020;8(2):41–3.
9. Mishra K, Kumar P, Basu S, Rai K, Aneja S. Risk factors for severe acute malnutrition in children below 5 years of age in India: a case-control study. *Indian J Pediatr.* 2014;81(8):762–5. doi:10.1007/s12098-013-1127-3.
10. Bandsma RHJ, Mendel M, Spoelstra MN, Reijngoud DJ, Boer T, Stellaard F, et al. Mechanisms behind decreased endogenous glucose production in malnourished children. *Pediatr Res.* 2010;68(5):423–8. doi:10.1203/PDR.0b013e3181f2b959.
11. Kumar R, Kumar P, Aneja S, Kumar V, Rehan HS. Safety and efficacy of low-osmolality ORS versus modified rehydration solution for malnourished children for treatment of children with severe acute malnutrition and diarrhea: a randomized controlled trial. *J Trop Pediatr.* 2015;61(6):435–41. doi:10.1093/tropej/fmv054.