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ORIGINAL ARTICLE

Predictors of mortality in children under 5 years of age hospitalized with severe acute malnutrition in a Tertiary Care Hospital.

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ABSTRACT… Objective: To determine the predictors and time of mortality in hospitalized children under 5 years with severe acute malnutrition (SAM). **Study Design:** Prospective, observational cohort study. **Setting:** Nutritional Rehabilitation Unit, Pediatric Medicine Department of National Institute of Child Health, Karachi, Pakistan. **Period:** August 2024 to April 2025. **Methods:** Enrollment of 273 SAM children, aged between 6 and 60 months, hospitalized and getting treatment, was made. All those children who had a weight-for-height Z score of <- 3, < 70% of the expected weight for height, or bilateral edema were considered as SAM children. The length of hospital stay and outcomes were recorded. Data were analyzed in SPSS 26 using chi-square and Mann-Whitney U tests, followed by multivariable logistic regression for variables with p<0.05. **Results:** In 273 children, 160 (58.6%) were male, with a median age of 18.0 months. Sepsis 102 (37.4%) and pneumonia 50 (18.3%) were leading diagnoses. Mortality occurred in 58 (21.2%) children. Multivariable analysis showed low weight adjusted odds ratio (aOR) 0.8 (95% CI 0.5 to 0.9, p=0.005), cough aOR 3.6 (1.2 to 10.1, p=0.018), reluctance to feed aOR 6.3 (1.9 to 20.3, p=0.002), SpO2 below 94% at admission aOR 28.7 (9.3 to 88.4, p<0.001), and skin changes aOR 6.4 (2.4 to 17.2, p<0.001) were independent predictors or mortality. **Conclusion:** Mortality in children with SAM is influenced by the presence of hypoxia, feeding difficulty, respiratory involvement, skin changes, deranged biochemical parameters and low body weight at admission.

Key words: Children, Edema, Hypoxia, Mortality, Severe Acute Malnutrition.

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INTRODUCTION

Malnutrition is a pathological state caused by deficiency and excess or imbalance of various nutrients to the point where body functions are affected.[1] In developing countries deficiency state is prevalent, specifically with a high burden in the Indian subcontinent and Africa.[2] Currently, 33 countries have at least 30% of children who are stunted worldwide.[3] In Pakistan, around 40% of children below 5 years are stunted, 17.7% suffer from wasting, and over 50% suffer from micronutrient deficiencies, while overweight/obesity and diet-related non-communicable diseases also are on the rise.[4] The Sustainable Development Goals (SDGs) proposed to reduce under-5 mortality to at least as low as 25 deaths per 1,000 live births.[5,6]

Identification of factors contributing to mortality is crucial to decrease the mortality in children who are severely malnourished.[7] According to WHO, children with uncomplicated severe acute malnutrition (SAM) should be treated as outpatients.[8,9] Reasons are not clear, but many factors, like the presence of comorbidities, such as diarrhea, sepsis, dehydration, pneumonia, anemia, altered mental status, tuberculosis, HIV, and intravenous fluid use, may be responsible for high mortality rates, with hypoglycemia and severe anemia as the strongest predictors of mortality among the hospitalized children with SAM.[10,11] A study revealed that 15.4% of the SAM children who died in hospital had malaria.[12] According to another study, factors associated with mortality included herbal medication use (p=0.001), poor appetite (p=0.003), mid-upper circumference (MUAC)<11.5 cm (p<0.001), lower respiratory tract infections (LRTIs) (p<0.001), anemia (p=0.021), hypoglycemia (p<0.001), and HIV infection (p<0.001).[13]

Most of the studies in Pakistan have been carried out to determine the frequencies of comorbidities and their outcomes in SAM.

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Therefore, this study was planned with the objective to determine the predictors and time of mortality in hospitalized children under 5 years with SAM.

METHODS

This prospective observational cohort study was conducted at the Nutritional Rehabilitation Unit, Medicine Department of the National Institute of Child Health, Karachi, Pakistan, from August 2024 to April 2025. The study commenced after obtaining approval from the Ethical Review Board of the institution (IERB-29/2023, dated: 19-07-2024). A sample size of 273 was calculated by using the OpenEpi sample size calculator, considering the anticipated proportion of malaria as a common predictor of mortality in SAM children as 15.4%[12], with a confidence limit of 5% and a confidence level of 95%. The inclusion criteria were children of any gender, aged 6-60 months, and who had been hospitalized with a diagnosis of SAM. The exclusion criteria were children with congenital heart disease, congenital anomalies, neuromuscular disorders, inborn errors of metabolism, chronic kidney disease, cerebral palsy, diabetes mellitus, and chronic liver disease. All those children who had a weight-for-height Z score of <- 3, < 70% of the expected weight for height, or bilateral edema were considered as SAM children. The sample selection was carried out using the non-probability consecutive sampling technique. Parents/caregivers were briefed about the objectives and the safety of the study before obtaining informed and written consent from them.

Patients fulfilling the eligibility criteria went through complete documentation of their demographic features, like gender (male/female), age (months), weight (kg), and height (cm). Weight-for-height z-score was calculated, and MUAC (cm) was measured for each patient. A detailed clinical examination and history taking were performed. Presenting complaints of all children were documented. Using aseptic technique, 5 mL of venous blood was drawn from each child and sent to the institutional laboratory for the assessment of complete blood count, urinary creatinine excretion, serum albumin, C-reactive protein, random blood sugar, ALT, and blood culture and sensitivity. A chest X-ray was taken for each child from the radiological department for the assessment of comorbidities like tuberculosis. All patients were given routine hospital care as per hospital protocol. The length of hospital stay and outcomes in the form of discharge, LAMA, and death were recorded. All the required information was taken on a structured proforma, especially designed for this study.

The statistical analysis was performed using "IBM-SPSS Statistics" version 26.0. The qualitative data were shown in the form of frequency and percentage. For the representation of quantitative data, means and standard deviations (SD), or medians and interquartile ranges (IQR) were calculated. The association of mortality with demographic, clinical and demographical variables were evaluated applying chi-square test and Mann-Whiteny U test (as numeric data were non-normally distributed. Variables with $p<0.05$ in univariatle analysis were further subjected to mulitivariate binary logistic regression analysis for independent association, with adjusted odds ratio (aOR) and 95% confidence interval (CI), taking a p-value<0.05 as significant.

RESULTS

In a total of 273 children, 160 (58.6%) were male, and 113 (41.4%) female. The median age was 18.0 (12.0-29.0) months, ranging betweeen 6 to 56 months. The median weight, height, MUAC (cm), OFC (cm), and weight for height z-score were 6.5 (5.0-6.5) kg, 73.0 (66.0-77.0) cm, 40.0 (9.5-10.5) cm, 44.0 (42.0-45.0) cm, and -4.6 (-5.1 to -3.9), respectively. Residential status of 198 (72.5%) children was urban, and 75 (27.5%) rural. Fever was present among 155 (56.8%) children at the time of admission, and the median duration of fever was 14.0 (7.0-20.0) days. Diarrhea, vomiting, reluctance to feed, cough, and respiratory distress were documented among 162 (59.3%), 81 (29.7%), 77 (28.2%), 63 (23.1%), and 29 (10.6) children, respectively. History of exclusive breastfeeding was reported in 90 (33.0%) children. SpO2 < 94% at the time of admission was noted in 91 (33.3%) children. Marasmus, kwashiorkor,edema, and skin changes were observed in 166 (60.8%), 147 (53.8%), 149 (54.6%), and 128 (46.9%) children, respectively. Anemia (Hb<11 g/dl) was present in 239 (87.5%) children. The median Hb, TLC, Platelets, serum creatinine, serum Na, serum postassium, serum chloride, serum magnesium, serum calcium, serum

albumin, serum ALT, random blood sugar, and C-reactive protein were 7.5 (6.6-8.8) g/dl, 13.2 (8.0-20.5) $10^3/mm^3$, 115.0 (74.0-28.5) $10^3/mm^3$, 0.2 (0.2-0.3) mg/dl, 135.0 (132.0-138.0) mEq/dl, 3.3 (2.7-4.1) mEq/dl, 100.0 (96.0-106.0) mEq/dl, 1.9 (1.8-2.1) mEq/dl, 8.0 (7.8-8.3) mg/dl, 2.5 (1.9-3.3) g/dl, 30.0 (21.0-44.0) U/L, 80.0 (50.0-90.0) mg/dl, and 15.0 (6.8-74.5) mg/dl, respectively. Sepsis, pneumonia, acute gastroenteritis, UTI, malria, acute kidney injury, and enteric fever were diagnosed in 102 (37.4%), 50 (18.3%), 41 (15.0%), 18 (6.6%), 8 (2.9%), 5 (1.8%), and 3 (1.1%) children, respectively.

The median duration of hospital stay was 12.0 (5.0-15.0) days. There were 181 (66.3%) children who recovered and were discharged successfully, while mortality was noted in 58 (21.2%), and 34 (12.5%) LAMA. Among 58 children who died, the most common causes behind mortality were septic shock noted in 41 (70.7%), hypoglycemia 9 (15.5%), pneumonia 3 (5.2%), and severe anemia in 2 (3.4%) children.

Children who LAMA were excluded from the final analysis, and 239 children were analyzed when comparisons were made for evaluating the association of mortality with various study variables. Children who died had significantly less body weight when compared to those who survived (p=0.032). Cough was present in 22 (37.9%) children who died (OR: 3.1, 95% CI: 1.6 to 5.9, p=0.001). Respiratory distress was reported in 13 (22.4%) children who died and in 13 (7.2%) who survivors yielding an odds ratio of 3.7 and a 95% CI 1.6 to 8.6, p=0.001. Reluctance to feed was reported in 23 (39.7%) children who died versus 46 (25.4%) survivors with an OR 1.9 and 95% CI 1.0 to 3.6, p=0.037. SpO2 < 94% at admission was observed in 47 (81.0%) children who died compared to 32 (17.7%) survivors (OR: 19.9, 95% CI: 9.3 to 42.5, p<0.001). Skin changes were reported in 36 (62.1%) children who died versus 77 (42.5%) survivors with OR of 2.2 and 95% CI 1.2 to 4.1, p=0.010 (Table-I).

Serum creatinine was significantly higher among children who died (p<0.001). Serum Na was significantly lower among children who died (p=0.001). Serum ALT was significantly higher among non-survivors (p=0.002). Median random blood sugar was 75.0 (IQR 30.0-86.0) mg/dL among children who died versus 80.0 mg/dL (50.0-91.0) among survivors (p=0.043). Details about the laboratory parameters associated with mortality and survival are presented in TABLE-II.

TABLE-III is showing details about the association between diagnosis at admission and mortality, and no significant associations were observed among children who survived or died.

Each kg in body weight decreased the aOR of mortality by 0.8 with 95% CI 0.5 to 0.9 (p=0.005). Cough at presentation was independently associated with mortality with aOR 3.6 and 95% CI of 1.2 to 10.1 (p=0.018). Reluctance to feed was found to independently predict mortality with aOR 6.3 and 95% CI of 1.9 to 20.3 (p=0.002). SpO_2 < 94% at admission had an aOR 28.7 and 95% CI of 9.3 to 88.4 (p<0.001). Skin changes were independently associated with mortality as aOR 6.4 and 95% CI of 2.4 to 17.2 (p<0.001) (Table-IV).

DISCUSSION

This study revealed that lower body weight was an predictor of mortality and these finding aligns with the meta-analysis by Karunaratne et al.[9], which concluded that lower weight was associated with increased inpatient mortality in children with SAM. This study found that each kg increase in body weight reduced the adjusted odds of death, an observation that supports the concept that diminished physiological reserves increase vulnerability to septic and metabolic deterioration. The similar direction of association between studies indicates that body weight continues to serve as a fundamental prognostic indicator across diverse clinical settings.[14,15] Variations in effect size may reflect differences in baseline nutritional severity, referral delays or local disease burden across populations.

Cough, and respiratory distress were markedly linked with mortality and remained independently associated after adjustment. Podder et al.[11], identified pneumonia in 24.1% of children who died in their study and showed that septicemia and respiratory involvement were key contributors to adverse outcomes.

TABLE-I

Association of demographic and clinical characteristics with mortality (N=239)

Characteristics		Mortality		OR (95% CI)	P-Value
		Yes (n=58)	No =181)		
Gender	Male	36 (62.1%)	107 (59.1%)	1.1 (0.6-2.1)	0.690*
	Female	22 (37.9%)	74 (40.9%)	Ref.	
Age in years		18.0 (13.0-36.0)	20.0 (13.0-29.0)	-	0.504^
Weight (kg)		5.5 (5.3-7.5)	6.4 (5.0-7.5)	-	0.032^
Height (cm)		73.0 (66.0-77.0)	74.0 (66.0-78.0)	-	0.277^
MUAC (cm)		10.5 (9.5-10.5)	10.0 (9.5-10.5)	-	0.414^
OFC (cm)		45.0 (42.5-46.0)	44.0 (43.0-45.0)	-	0.323^
Weight for heigh Z-score		-4.5 (-7.8-3.5)	-4.0 (-4.6-4.0)	-	0.287^
Residence	Rural	21 (36.2%)	46 (25.4%)	1.7 (0.9-3.1)	0.111*
	Urban	37 (63.8%)	135 (74.6%)	Ref.	
Fever		35 (60.3%)	97 (53.6%)	1.3 (0.7-2.4)	0.368*
Cough		22 (37.9%)	30 (16.6%)	3.1 (1.6-5.9)	0.001*
Respiratory distress		13 (22.4%)	13 (7.2%)	3.7 (1.6-8.6)	0.001*
Diarrhea		34 (58.4%)	117 (64.6%)	0.8 (0.4-1.4)	0.408*
Vomiting		16 (27.6%)	63 (34.8%)	0.7 (0.4-1.4)	0.309*
Reluctance to feed		23 (39.7%)	46 (25.4%)	1.9 (1.0-3.6)	0.037*
Fits		2 (3.4%)	5 (2.8%)	1.3 (0.2-6.7)	0.787*
History of exclusive breastfeeding		25 (43.1%)	56 (30.9%)	1.7 (0.9-3.1)	0.089*
SpO^2<94%		47 (81.0%)	32 (17.7%)	19.9 (9.3-42.5)	<0.001*
Marasmus		39 (67.2%)	105 (58.0%)	1.5 (0.8-2.8)	0.211*
Kwashiorkor		33 (56.9%)	102 (56.4%)	1.0 (0.6-1.9)	0.942*
Edema		33 (56.9%)	104 (57.5%)	1.0 (0.5-1.8)	0.940*
Skin changes		36 (62.1%)	77 (42.5%)	2.2 (1.2-4.1)	0.010*
Eye changes		15 (25.9%)	35 (19.3%)	1.5 (0.7-2.9)	0.288*
Abdominal distension		23 (39.7%)	90 (49.7%)	0.7 (0.4-1.2)	0.181*

*Chi-square test applied; ^Mann-U Whitney test applied

Baskaran et al.[7], also reported that systemic illness, and respiratory disease were significant predictors of mortality in children with SAM. The present findings reinforce the premise that the combination of malnutrition and respiratory infections exerts synergistic physiological stress.[16] One explanation for this is the established link between malnutrition and compromised cellular immunity which predisposes children to severe pulmonary infections and impaired respiratory compensation.[17] Differences in respiratory pathogen profiles across settings may influence the magnitude of association. This study found that SpO2 < 94% at admission was the strongest independent predictor of mortality. This magnitude of association underscores the severe impact of hypoxia in malnourished children whose physiological compensatory capacity is already diminished.[18] Kassaw et al.[19], found that oxygen saturation below 90% increased the hazard of death and Nduhukire et al similarly reported that impaired respiratory function and the need for oxygen therapy predicted in-hospital mortality.

Reluctance to feed emerged as a strong predictor of mortality. Karunaratne et al.[9], demonstrated that lack of appetite was independently associated with inpatient mortality. In the present study, reluctance to feed had an aOR of 6.3 which demonstrates the

TABLE-II

Association of laboratory parameters with mortality (N=239)

Laboratory Parameters	Mortality		P-Value
	Yes (n=58)	No =181)	
Hemoglobin (g/dl)	8.0 (7.1-11.6)	7.5 (6.6-8.5)	0.085
Total leukocytes count (10^3/mm^3)	16.0 (11.3-20.5)	13.2 (8.0-20.9)	0.513
Platelets (10^3/mm^3)	84.0 (69.0-170.0)	109.0 (59.0-276.5)	0.065
Serum creatinine (mg/dl)	0.4 (0.2-0.7)	0.2 (0.2-0.3)	<0.001
Serum sodium (mEq/dl)	133.0 (130.0-135.0)	135.0 (132.5-138.0)	0.001
Serum potassium (mEq/dl)	3.2 (2.7-3.9)	3.2 (2.4-4.1)	0.895
Serum chloride (mEq/dl)	101.0 (98.0-105.0)	102.0 (96.0-106.0)	0.468
Serum magnesium (mg/dl)	1.8 (1.7-2.0)	1.9 (1.8-2.0)	0.705
Serum calcium (mg/dl)	8.0 (7.6-8.5)	8.0 (7.8-8.2)	0.537
Serum albumin (g/dl)	3.0 (1.8-3.5)	2.4 (1.9-3.2)	0.632
Serum alanine transaminase (U/L)	44.0 (23.0-55.0)	29.0 (20.0-41.5)	0.002
Random blood sugar (mg/dl)	75.0 (30.0-86.0)	80.0 (50.0-91.0)	0.043
C-reactive protein (mg/dl)	17.0 (5.0-72.0)	15.0 (7.0-75.8)	0.608

Mann-U Whitney test applied

TABLE-III

Association of diagnosis at the time of admission with mortality (N=139)

Diagnosis at Admission	Mortality		OR (95% CI)	P-Value
	Yes (n=58)	No =181)		
Sepsis	22 (37.9%)	77 (42.5%)	0.8 (0.5-1.5)	0.535
Pneumonia	14 (24.1%)	27 (14.9%)	1.8 (0.9-3.8)	0.105
Acute gastroenteritis	2 (3.4%)	30 (16.6%)	0.2 (0.1-0.8)	0.011
Urinary tract infection	2 (3.4%)	16 (8.8%)	0.4 (0.1-1.7)	0.176
Malaria	1 (1.7%)	7 (3.9%)	0.4 (0.1-3.6)	0.430
Enteric fever		3 (1.7%)	-	0.324
Acute kidney injury	-	5 (2.8%)	-	0.201

Chi-square / fisher's exact test applied

TABLE-IV

Multivariate binary logistic regression for the predictors of mortality

Predictors	aOR (95% CI)	P-Value
Weight	0.7 (0.5-0.9)	0.005
Cough	3.6 (1.2-10.1)	0.018
Respiratory distress	1.2 (0.4-4.1)	0.719
Reluctance to feed	6.3 (1.9-20.3)	0.002
History of exclusive breastfeeding	1.9 (0.7-5.5)	0.207
SpO_2 < 94% at admission	28.7 (9.3-88.4)	<0.001
Skin changes	6.4 (2.4-17.2)	<0.001
Serum creatinine (mg/dl)	1.6 (0.6-4.8)	0.360
Serum sodium (mEq/dl)	1.0 (0.9-1.1)	0.481
Serum alanine transaminase (U/L)	1.0 (0.9-1.0)	0.808
Random blood sugar (mg/dl)	0.9 (0.9-1.0)	0.226

aOR: adjusted odds ratio; CI: confidence interval

clinical significance of impaired intake in destabilizing fluid, electrolyte, and metabolic balance. Sturgeon et al.[10], also reported that electrolyte disturbances, persistent diarrhoea and feeding difficulties contributed to early mortality in hospitalized children with SAM.

Skin changes were another variable independently associated with mortality. Podder et al.[11], identified dermatosis in 46.2% of children who died and described it as a major contributor to mortality in their cohort. Gavhi et al.[13], also documented that children with skin breakdown and secondary infection had higher odds of mortality. The shared direction of association between studies can be attributed to the fact that dermatoses in SAM frequently reflect hypoalbuminemia, micronutrient deficiency, and compromised immunity, all of which increase the risk of systemic infection.[20,21]

The overall mortality rate in this study was 21.2% which is higher than the mortality rates reported in Alemu et al.[22], at 9.5%, Wagnew et al.[23], at 12.52%, and Abdu et al.[24], at 10.4%. Several possible explanations can account for the higher mortality in this population. A substantial proportion of children presented with prolonged illness, advanced malnutrition, hypoxia, and sepsis at admission. The presence of multiple overlapping comorbidities including severe anaemia and prolonged fever may have contributed to more rapid clinical decline.[25]

This study has few limitations. The study was conducted in a single tertiary centre which may limit external generalizability. The use of a cross-sectional design restricted the ability to establish temporal causality beyond associations. Prospective cohort studies with daily monitoring of physiological parameters would improve understanding of the progression toward mortality.

CONCLUSION

Mortality in children with SAM is influenced by the presence of hypoxia, feeding difficulty, respiratory involvement, skin changes, deranged biochemical parameters and low body weight at admission. Strengthening early detection systems, ensuring timely correction of metabolic abnormalities and enhancing the quality of inpatient stabilization care are essential steps toward reducing mortality in this vulnerable population.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHORSHIP AND CONTRIBUTION DECLARATION	
1	**Amrita:** Data collection, drafting.
2	**Mohsina Noor Ibrahim:** Concept, study design.
3	**Misbah Anjum:** Critical revisions.