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Research Article

Admission Metabolic Derangements and Mortality Risk in Pediatric Sepsis: Perspectives from a Low-Resource PICU

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ABSTRACT

Background: Sepsis is a leading cause of mortality in pediatric intensive care units (PICUs), particularly in resource-limited settings. Metabolic derangements such as dysglycemia and hypoalbuminemia are recognized as individual risk factors, but their combined prognostic value in pediatric sepsis remains poorly understood.

Aim: To determine if concurrent dysglycemia (hyperglycemia or hypoglycemia) and hypoalbuminemia at PICU admission are superior to either abnormality alone in predicting mortality among children with sepsis.

Methods: This study analyzed 175 children with sepsis upon admission to PICU. Admission blood glucose and serum albumin levels were categorized, and patients were stratified based on their metabolic profiles. The primary outcome was mortality at hospital discharge and at 30 days.

Results: At admission, 17.7% of children were hyperglycemic (blood glucose ≥150 mg/dL) and 28.6% had hypoalbuminemia (serum albumin <3.5 g/dL). Outcomes varied dramatically based on the combined metabolic profile. Children with concurrent hyperglycemia and hypoalbuminemia experienced the highest mortality rates, with 40% mortality at discharge and 50% at 30 days. In contrast, the euglycemic-normoalbuminuric group had a 96.9% survival rate at discharge. Admission hypoalbuminemia was a strong independent predictor of mortality (Area Under the Curve of 0.769), associated with a 3.5-fold increase in PICU mortality and a 2.5-fold increase in post-discharge mortality. Glucose abnormalities alone were not significant independent predictors. Gram-negative organisms were the predominant pathogens in positive blood cultures (62.6%).

Conclusion: The combination of hyperglycemia and hypoalbuminemia at PICU admission is a powerful and easily identifiable predictor of mortality in pediatric sepsis. These findings strongly support the implementation of routine, dual screening with blood glucose and serum albumin, two low-cost, universally available tests to enable early risk stratification and guide interventions for high-risk children, especially in resource-limited settings.

Keywords: *Albumin; Blood Culture; Hyperglycemia; Hypoalbuminemia; Length of Stay; Glucose; Sepsis; Pediatric Intensive Care Unit.*

INTRODUCTION

Clinical hypoglycaemia during hospitalization is a well-established predictor of poorer outcomes in septic patients [1]. Sudden drop in blood glucose can precipitate acute neurological deficits and activate compensatory stress pathways, both of which hinder recovery and increase in-hospital morbidity and mortality. Infection can induce hypoglycaemia by impairing gluconeogenesis and increasing glucose uptake by activated immune cells [2]. Concurrently, hypoalbuminemia resulting from capillary leakage, reduced hepatic synthesis, and accelerated catabolism is recognized as a strong prognostic marker in sepsis[3]. Albumin, the most abundant plasma protein, maintains oncotic pressure, transports a wide array of endogenous and exogenous compounds, and modulates inflammatory responses. Septic patients with low serum albumin

levels face a higher likelihood of organ dysfunction, extended mechanical ventilation, and increased risk of death [4]. Although extensive research has established hypoglycaemia and hypoalbuminemia as independent predictors of poor outcome in sepsis, their combined impact in paediatric patients remains unexplored. As a result, clinicians currently lack an evidence-based framework for interpreting the prognostic significance when hypoglycaemia and hypoalbuminemia co-occur at admission.

Existing paediatric mortality risk scores require multiple laboratory and clinical inputs that may not be feasible in all resource-limited hospital settings[5]. Simpler admission biomarkers namely, point-of-care glucose and albumin measurement could offer a pragmatic solution. Demonstrating their joint predictive power would support a low-cost, scalable triage strategy, enabling prompt treatment in paediatric intensive care units for those most in need. We hypothesized that children presenting with both abnormal glucose and albumin values at PICU admission have a significantly higher in-hospital mortality than those with either abnormality alone or neither. The objective of this study was to assess whether admission glucose–albumin profiles can serve as reliable predictors of death in paediatric sepsis patients managed in a low-resource hospital setting.

Methodology:

Study design and setting: This study was approved by the institute Ethics Committee as a single-centre, prospective observational study from PICU of our hospital between June 2024 and May 2025. This was time bound academic thesis study in which patients were enrolled through purposive sampling, based on predefined eligibility criteria.

Study population: Each morning, the PICU team (principal investigator, attending physicians, and nursing staff) reviewed every patient's chart and enrolled those meeting the inclusion criteria after obtaining informed consent from their caregivers. Paediatric patients over 1 month up to 18 years who were treated for sepsis were included in the study if they had a PICU stay exceeding 24 hours to ensure complete observation and record collection. Sepsis was diagnosed when a confirmed or suspected infection was accompanied by systemic manifestations, with at least two of the following: a core temperature > 38.5°C or < 36°C; a heart rate sustained above 180 bpm or below 100 bpm for more than 30 minutes; an increased respiratory rate or the need for mechanical ventilation due to acute illness; and a white blood cell count that is elevated or depressed for age or shows more than 10 percent immature neutrophils [6].

The exclusion criteria included length of stay less than 24 hours, second or third-degree burn patient and those who refuse to take part in the study, had multiple PICU admissions during the study period, diagnosed with Diabetic Ketoacidosis, had known or suspected pre-existing hypoalbuminemia states, including severe malnutrition with weight-for-height below -3 SD, chronic gastrointestinal and renal illnesses affecting nutritional status such as chronic liver disease, cirrhosis, nephrotic syndrome, nephritis, malabsorption and celiac disease, inflammatory bowel disease, had received albumin transfusion or blood products before serum albumin measurement.

Data collection: We collected data on variables including patient demographics, vital signs, anthropometry, reason for admission, sepsis diagnosis, need for ventilatory support, use of inotropes, Pediatric Risk of Mortality Score III (PRISM III), and PICU outcome. All information was retrieved from the patients' medical records and supplemented by direct clinical assessment. As part of routine patient care, laboratory evaluations included admission random blood sugar (RBS) and serum albumin. RBS was measured via finger prick using the Accu-Chek glucometer (Roche Laboratories), and to enhance accuracy, intravenous blood glucose samples were tested in every third patient through the hospital's in-house laboratory. Serum albumin levels were assessed using standard biochemical analyzer. For consistency, only the first value recorded within 24 hours of admission was considered. To support infection diagnosis, additional investigations were carried out as clinically indicated including arterial blood gas analysis, complete blood count, liver and renal function tests, inflammatory markers, and cultures of blood, urine, or other sterile sites to identify the infectious source.

Cases were classified according to admission blood glucose [7] and serum albumin [8]. Hypoglycemia was defined as blood glucose below 80 mg/dL, and hypoalbuminemia as serum albumin below 3.5 g/dL. Patients were then stratified into six subgroups based on the combination of these variables: 1) Hypo-G + Hypo-A: Glucose < 80 mg/dL and albumin < 3.5 g/dL; 2) Normal-G + Hypo-A: Glucose 80–149 mg/dL and albumin < 3.5 g/dL; 3) Hyper-G + Hypo-A: Glucose ≥ 150 mg/dL and albumin < 3.5 g/dL; 4) Hypo-G + Non-Hypo-A: Glucose 80–149 mg/dL and albumin ≥ 3.5 g/dL; 5) Normal-G + Non-Hypo-A: Glucose 80–149 mg/dL and albumin ≥ 3.5 g/dL; and 6) Hyper-G + Non-Hypo-A: Glucose ≥ 150 mg/dL and albumin ≥ 3.5 g/dL .

Patients were prospectively observed from the time of PICU admission until one of three endpoints was reached: discharged alive, death during the PICU stay, or referral to another hospital. Those discharged alive or referred out were then followed up either telephonically or in person at 30-day post-discharge to document final survival status (alive or dead).

Statistical Analysis: SPSS 22 (IBM Statistics Version 22, Chicago, IL, USA) was used for all statistical analyses. Continuous variables were presented as means ± standard deviations and categorical variables expressed in percentage. Variables between groups were compared using the chi square or Mann-Whitney U test. The patients were divided into

various sub groups and data from each group were compared by ANOVA or Kruskal–Wallis analysis. Binary logistic regression analysis was performed to determine the predictive ratio of glucose and albumin levels for mortality. Variables with p values < 0.2 by bivariate analysis were then introduced into the multivariate model for calculating the odds ratios (OR) and 95% confidence interval (CI). ROC analysis was performed and the area under the curve (AUC) value was detected to determine the sensitivity, specificity, and cut-off values of glucose and albumin levels in predicting mortality. A p-value < 0.05 was considered to be statistically significant.

Results:

A total of 175 children (98 male) were enrolled. Mean age was 69.91±66 (range 2-206 months), mean weight was 18.81±48.4 (range 4-72 kilos) and mean length was 102.64±69.4 (range 58-180 cm). Respiratory conditions were the most frequent admission diagnoses, affecting 38.3% of patients (n=67), and included pneumonia (n=28), bronchiolitis (n=15), ARDS (n=11), asthma (n=10), and other respiratory disorders (n=3). Central nervous system disorders accounted for 32.6% of cases (n=57), predominantly meningitis/encephalitis (n=33), with seizures (n=5). Cardiovascular causes were uncommon (1.1%; n=2), both cases being acyanotic congenital heart disease. Hematological disorders comprised 4.6% of admissions (n=8) including leukemia (n=3), anemia (n=2), hemophilia (n=3), while gastrointestinal etiologies represented 12.0% (n=21), with diarrhoea in 16 patients and surgical causes in 5. Endocrine disorders (adrenal insufficiency, n=3; SIADH, n=1) and rheumatological diseases (Kawasaki disease, n=2; systemic lupus erythematosus, n=2) each accounted for 2.3% of admissions. Acute febrile illness was seen in 4 patients (2.3%), and one patient was admitted with acute kidney failure.

On admission, 15 patients (8.6%) were hypoglycemic, 31 (17.7%) were hyperglycemic, and 129 (73.7%) were euglycemic. Hypoalbuminemia (serum albumin < 3.5 g/dL) was present in 50 patients (28.6%). The largest subgroup was Normal-G + Normal-A (n=96, 54.9%) followed by Normal-G + Hypo-A (n=33, 18.9%), Hyper-G + Normal A (n=21, 12.0%), Hyper-G + Hypo-A ((n=10, 5.7%), Hypo-G + normal-A (n=8, 4.6%), and Hypo-G + Hypo-A at 4.0% (n=7) as mentioned in Table 1.

Outcome

Thirteen patients were referred to other health facilities, eight for plasmapheresis not available at our centre, three for specialized surgical procedures, and two for advanced genetic diagnostic services. Of those referred, four died; additional twelve deaths occurred in our PICU, bringing the total to sixteen. Among the 150 children discharged alive, eight more died during the 30-day follow-up as depicted in Fig 1.

In our cohort of children 25.7% had positive blood cultures. Among these, 62.6% of isolates (n = 28) had gram-negative sepsis, while gram-positive organisms comprised 26.7% (n = 12). The most frequently recovered pathogens were Klebsiella pneumoniae (8 cases, 17.7%), Acinetobacter baumannii (7 cases, 15.5%), Staphylococcus aureus and coagulase-negative staphylococci (6 cases each, 13.3%), Escherichia coli and Candida species (5 cases each, 11.1%), Pseudomonas aeruginosa (4 cases, 8.8%), and Enterobacter and Enterococcus species (2 cases each, 4.4%).

Table 2 summarizes the group-wise duration of PICU stay, inotropic support requirements, and invasive ventilation rates. Patients were stratified into survivors and non-survivors at discharge and at 30-day follow-up to facilitate a comparative analysis of outcome predictors as shown in Table 3. Multiple logistic regression analysis of the admission laboratory data showed that the non-survivors were more likely to be older than 5years, had higher PRISM III score, and presented with lactic acidosis, elevated total leukocyte counts, thrombocytopenia, lower neutrophil-to-lymphocyte ratio, and deranged liver enzymes. At one-month follow-up, non-survivors were more often females, and had admission hypoxia, hyperglycaemia, elevated serum urea, and hypoalbuminemia. Other independent mortality predictors showed no significant association.

As shown in Table 1, before discharge, group 3 exhibited the highest mortality (40%), with group 2 at 27.2%, group 4 at 12.5%, and group 5 at 3.1%. Groups 1 and 6 experienced no deaths prior to discharge. Overall group 3 had highest mortality (50%), followed by group 1 (28.7%), group 2 (27.2%), group 6 (14.2%), group 4 (12.5%), and group 5 (5.2%). Receiver operating characteristic (ROC) analysis of admission variables identified an albumin level ≥3.5 g/dL with 76.7% sensitivity, 81.2% specificity, and an AUC of 0.769 for predicting PICU mortality. In contrast, a random blood sugar (RBS) cut-off ≥169 mg/dL yielded 12.5% sensitivity, 93.7% specificity, and an AUC of 0.378 (Fig. 2).

Table 1: Distribution of Outcome and Laboratory Variables Across Study Groups

	Group 1 (n=7) (Hypo-G + Hypo-A)	Group 2 (n=33) (Normal-G + Hypo-A)	Group 3 (n=10) (Hyper-G + Hypo-A)	Group 4 (n=8) (Hypo-G + Normal-A)	Group 5 (n=96) (Normal-G + Normal-A)	Group 6 (n=21) (Hyper-G + Normal-A)	Total	p-value
Male: Female(n=175)	3:4	23:10	5:5	6:2	52:54	9:12	98:77	
Deaths: Male: Female (Discharge) (n=16)	-	5:4	2:2	-	1:2	-	8:8	0.61
Additional deaths: Male: Female (Day 30) (n=6)	0:1	-	0:1	0:1	0:2	0:3	0:8	
Total deaths: Male: Female (Day 30) (n=24)	0:1	5:4	2:3	0:1	1:4	0:3	8:16	0.01
Vital Sign								
Heart Rate (bpm)	108.29 ± 27.75	111.70 ± 30.46	105.30 ± 23.85	123.75 ± 42.06	112.13 ± 34.92	105.48 ± 34.65	111.29 ± 33.90	0.199
Respiratory Rate (breaths/min)	31.43 ± 12.46	32.61 ± 17.37	33.60 ± 11.45	39.50 ± 28.42	33.66 ± 20.09	31.24 ± 14.93	33.29 ± 19.03	0.797
Systolic BP (mmHg)	98.29 ± 21.70	98.55 ± 23.33	104.60 ± 26.91	105.75 ± 25.60	101.38 ± 25.46	94.67 ± 23.65	100.50 ± 24.85	0.546
Diastolic BP (mmHg)	61.43 ± 16.22	62.24 ± 13.82	68.60 ± 16.95	69.50 ± 18.43	63.56 ± 15.22	58.29 ± 14.24	63.09 ± 15.23	0.269
Mean BP (mmHg)	73.71 ± 17.49	74.34 ± 15.83	80.60 ± 19.72	81.58 ± 20.22	76.17 ± 17.96	70.41 ± 16.13	75.56 ± 17.69	0.415
Glasgow Coma Scale	13.14 ± 3.24	13.73 ± 2.92	14.10 ± 2.64	13.38 ± 3.42	13.72 ± 3.26	14.57 ± 1.94	13.78 ± 3.08	0.252
Temperature (°C)	36.79 ± 0.83	36.91 ± 0.81	36.82 ± 0.75	36.95 ± 0.88	36.85 ± 0.79	36.96 ± 0.79	36.87 ± 0.79	0.961
Oxygen Saturation (%)	91.14 ± 14.79	90.85 ± 11.93	88.40 ± 14.17	91.25 ± 15.60	92.27 ± 13.26	95.81 ± 7.75	92.18 ± 12.86	0.049*
Laboratory Parameter								
Random Blood Sugar (mg/dL)	66.43 ± 11.84	106.79 ± 20.36	229.30 ± 75.13	64.13 ± 10.36	107.27 ± 20.81	213.57 ± 65.38	118.81 ± 73.04	<0.001*
Serum Albumin (g/dL)	2.86 ± 0.38	2.91 ± 0.37	2.95 ± 0.42	4.70 ± 0.63	4.34 ± 0.70	4.15 ± 0.57	4.30 ± 0.72	<0.001*
pH	7.26 ± 0.24	7.36 ± 0.13	7.33 ± 0.14	7.26 ± 0.14	7.34 ± 0.11	7.32 ± 0.14	7.34 ± 0.13	0.224
PaCO2 (mmHg)	44.43 ± 9.11	30.45 ± 8.83	30.30 ± 9.36	39.13 ± 13.14	35.42 ± 11.40	29.19 ± 4.95	33.97 ± 10.73	0.001*
PaO2 (mmHg)	59.14 ± 21.10	83.91 ± 47.37	82.90 ± 33.62	86.63 ± 60.67	84.94 ± 38.09	91.67 ± 38.67	84.48 ± 40.39	0.632
Bicarbonate in ABG (mmol/L)	21.00 ± 5.13	19.06 ± 5.56	18.30 ± 5.12	19.00 ± 7.29	20.50 ± 4.59	17.81 ± 4.03	19.73 ± 4.95	0.182
Lactatein ABG (mmol/L)	2.29 ± 1.35	2.82 ± 4.72	2.80 ± 2.27	1.76 ± 0.68	2.11 ± 1.87	1.64 ± 0.98	2.22 ± 2.57	0.04*
Hemoglobin (g/dL)	11.50 ± 1.84	11.12 ± 2.76	10.65 ± 2.32	10.94 ± 3.17	11.44 ± 2.25	11.56 ± 2.53	11.33 ± 2.40	0.893
Total Leukocyte Count (x10³/µL)	12143.57 ± 6356.37	10353.03 ± 6092.92	10797.00 ± 6788.63	10607.50 ± 8189.94	11778.44 ± 6217.43	11046.48 ± 8599.27	11326.81 ± 6578.49	0.918

Neutrophil: Lymphocyte Ratio	4.76 ± 5.55	6.02 ± 8.74	1.98 ± 2.06	2.99 ± 3.42	3.92 ± 5.35	3.44 ± 2.51	4.14 ± 5.77	0.332
Platelet Count ($x10^3/\mu L$)	267714.29 ± 118736.00	245090.91 ± 144326.10	162013.40 ± 160095.81	258875.00 ± 161729.44	264354.17 ± 137511.49	226058.67 ± 167949.90	250162.09 ± 144602.29	0.372
CRP (mg/L)	73.13 ± 113.73	59.22 ± 86.70	63.00 ± 102.67	49.04 ± 107.88	51.50 ± 86.19	60.58 ± 93.35	55.45 ± 89.06	0.983
INR	0.90 ± 0.11	1.14 ± 0.37	1.33 ± 0.44	1.13 ± 0.55	1.12 ± 0.72	1.09 ± 0.11	1.12 ± 0.58	0.790
PT (seconds)	12.71 ± 1.11	14.01 ± 4.71	16.45 ± 4.90	14.88 ± 5.67	13.94 ± 8.36	13.31 ± 1.52	14.02 ± 6.76	0.863
aPTT (seconds)	24.14 ± 3.58	25.24 ± 8.12	26.94 ± 9.72	27.38 ± 8.45	24.18 ± 9.61	23.80 ± 7.43	24.64 ± 8.83	0.843
Urea (mg/dL)	27.71 ± 16.75	32.67 ± 16.07	35.70 ± 21.50	57.63 ± 51.61	31.27 ± 24.46	33.77 ± 42.00	33.15 ± 27.36	0.203
Creatinine (mg/dL)	0.41 ± 0.16	0.57 ± 0.30	0.66 ± 0.25	1.07 ± 1.05	0.59 ± 0.60	0.94 ± 1.23	0.65 ± 0.68	0.111
Sodium (mmol/L)	140.29 ± 7.02	137.76 ± 5.79	140.00 ± 8.56	139.00 ± 7.35	138.27 ± 5.07	138.62 ± 3.28	138.43 ± 5.43	0.803
Potassium (mmol/L)	4.09 ± 1.33	4.15 ± 0.67	4.61 ± 0.67	4.70 ± 0.63	4.34 ± 0.70	4.15 ± 0.57	4.30 ± 0.72	0.165
Total Bilirubin (mg/dL)	0.44 ± 0.48	0.70 ± 0.56	1.31 ± 0.95	0.46 ± 0.43	1.23 ± 2.96	1.07 ± 1.60	1.05 ± 2.30	0.779
SGPT (U/L)	37.43 ± 29.31	52.55 ± 95.67	150.90 ± 272.98	99.13 ± 183.15	158.50 ± 571.79	68.00 ± 88.51	119.67 ± 434.37	0.829
SGOT (U/L)	48.43 ± 27.93	111.03 ± 240.96	131.70 ± 195.64	307.25 ± 696.30	187.21 ± 602.85	46.33 ± 54.36	152.70 ± 484.63	0.721

Table 2: Comparison of Length of Stay and Mechanical Ventilation Across Study Groups

Characteristic	Group 1 (n=7) (Hypo-G + Hypo- A)	Group 2 (n=33) (Inter-G + Hypo-A)	Group 3 (n=10) (Hyper-G + Hypo-A)	Group 4 (n=8) (Hypo-G + Normal-A)	Group 5 (n=96) (Inter-G + Normal-A)	Group 6 (n=21) (Hyper-G + Normal-A)	Total (n=175)	p-value
Invasive ventilation, n(%),	4 (57.1%)	14 (42.4%)	2 (20.0%)	6 (75.0%)	52 (54.2%)	7 (33.3%)	85 (48.6%)	0.001*
Duration of invasive ventilation in days	0.00	1.71 ± 1.27	3.50 ± 0.71	0.75 ± 0.72	1.33 ± 1.14	11.00 ± 9.90	1.51 ± 1.44	0.002*
Inotropes Given, n(%)	4 (57.1%)	20 (60.6%)	5 (50.0%)	3 (37.5%)	41 (42.7%)	7 (33.3%)	80 (45.7%)	0.053
PICU stay in days	8.29 ± 3.45	6.00 ±2.99	5.30 ± 2.11	3.88 ± 2.36	4.49 ± 2.62	6.62 ± 3.66	5.13 ± 2.91	<0.001*
Total hospital stay in days	8.86 ± 3.98	8.36 ± 4.49	7.70 ± 3.80	6.00 ± 2.98	7.76 ± 4.66	11.14 ± 6.11	8.14 ±4.78	0.188

Duration of stay in days, Mean ± SD

Table 3: Comparison of Survivors and Non-Survivors Across Study Groups

Parameters	Alive (Discharge)	Dead (Before discharge)	OR (95% CI)	p-value	Dead (Day 30 follow-up)	OR (95% CI)	p-value
Gender, Male, n(%)	90(91.8)	8(8.2)		0.61	8(8.2)	2.4(1.2-5.8)	0.04
Female, n(%)	69(89.6)	8(10.4)			16(20.8)		
Median age in months(range)	42(2-204)	66(2-206)	0.8(0.6-.98)	0.03	54(20206)		0.37
Median PICU stay in days(range)	3(1-20)	4(1-18)	1.3(1.1-1.27)	0.01	3(1-18)		0.052
Median hospital stays in days(range)	6(1-30)	4(1-18)	0.87(0.67-0.94)	0.02	6(1-18)		0.06
PRISM 3 score (Mean±SD)	4.75(±3)	13.3(±6.2)	1.2(1.15-1.3)	0.01	11.2(±3.4)	1.18(1.03-1.24)	0.01

Admission Vitals							
Heart Rate (bpm)	132(±34)	147(±32)	0.92(0.84-0.98)	0.01	140(±43)	1.02(1.01-1.04)	0.01
Respiratory Rate(breaths/min)	40(±19)	44(±24)	0.9(0.75-0.98)	0.02	42(±26)		0.3
Systolic BP (mmHg)	94(±23)	89(±38)	0.97(0.95-0.99)	0.02	82(±42)	0.98(0.97-0.99)	0.01
Diastolic BP (mmHg)	56(±16)	52(±22)			48(±24)		
Mean BP (mmHg)	70(±16)	64(±27)			58(±30)		
Glasgow Coma Scale	13(±2)	9(±5)	0.96(0.89-0.98)	0.01	10(±5)	0.75(0.67-0.84)	0.01
Temperature (°C)	37.2(±0.7)	37.7(±0.9)		0.72	37.4(±1.1)		0.94
Oxygen Saturation (%)	87(±12)	83(±16)		0.08	80(±18)		0.01
Laboratory Parameter							
pH	7.33(±1.3)	7.28(±0.1)	2.14(1.3-7.3)	0.03	7.3(±1.5)		0.9
PaCO2 (mmHg)	34.7(±10)	26.6(±8)			32(±11)		
PaO2 (mmHg)	90(±34)	84(±41)			78(±36)		
Bicarbonate in ABG (mmol/L)	20(±4.8)	17(±5.2)	0.9(0.8-0.96)	0.03	18(±5)		0.09
Lactate in ABG (mmol/L)	2.1(±2.5)	3.8(2.2)	1.6(1.1-1.9)	0.02	2.5(±1.9)		0.1
Haemoglobin (g/dL)	11.2(±2.4)	11.6(±2.5)		0.6	11.8(±2.2)		0.5
Total Leukocyte Count ($x10^3/\mu L$)	11335(±6400)	112340(±7920)	1.3(1.1-1.9)	0.01	11171(±7600)	1.12(1.1-2.9)	0.01
Neutrophil: Lymphocyte Ratio	4.2(±1.5)	3(±2)	0.94(0.8-0.97)	0.02	3.2(±1.2)	0.7(0.52-0.92)	0.02
Platelet Count ($x10^3/\mu L$)	251170(±141410)	240080(±17830)	0.91(0.89-0.95)	0.01	228408(±156758)	0.93(0.90-0.99)	0.01
CRP (mg/L)	54(±8)	62(±10)	0.78(0.52-0.91)	0.01	86(±24)	0.85(0.51-0.92)	0.01
INR	1.1(±0.6)	1.0(±0.2)		0.8	1.1(±0.2)		0.9
PT (seconds)	14(±7)	13.5(±3)		0.6	14(±2.8)		0.9
aPTT (seconds)	24.8(±9)	22(±6)	0.94(0.86-0.98)	0.03	24(±8)		0.8
Urea (mg/dL)	33(±23)	34(±18)		0.3	38(±18)	0.42(0.21-0.65)	0.01
Creatinine (mg/dL)	0.65(±0.7)	0.55(±0.3)		0.7	0.8(0.3)		0.3
Sodium (mmol/L)	138(±5)	139(±7)		0.9	138(±6)		0.9
Potassium (mmol/L)	4.3(±0.7)	4.1(±0.7)		0.4	4.2(±0.7)		0.7
Total Bilirubin(mg/dL)	1.08(±0.4)	0.7(±0.2)		0.2	0.9(±0.3)		0.5
SGPT (U/L)	122(±45)	98(±22)	0.8(0.61-0.9)	0.01	88(±14)	0.91(0.5-0.92)	0.01
SGOT (U/L)	152(±48)	100(±58)			82(±13)		0.01
Positive blood culture	13	4		0.35	4		0.25
Glycaemic groups							
Hypoglycaemia (<80 mg/dL)	15	0		0.36	2		0.09
Euglycemia (80–149 mg/dL)	117	12			14		
Hyperglycaemia (≥150 mg/dL)	27	4			8		
Albumin groups							
Hypoalbuminemia (<3.5 g/dL)	37	13	0.28(0.13-0.5)	0.01	15	0.35(0.1-0.5)	0.01
Normal Albumin (≥3.5 g/dL)	122	3			9		

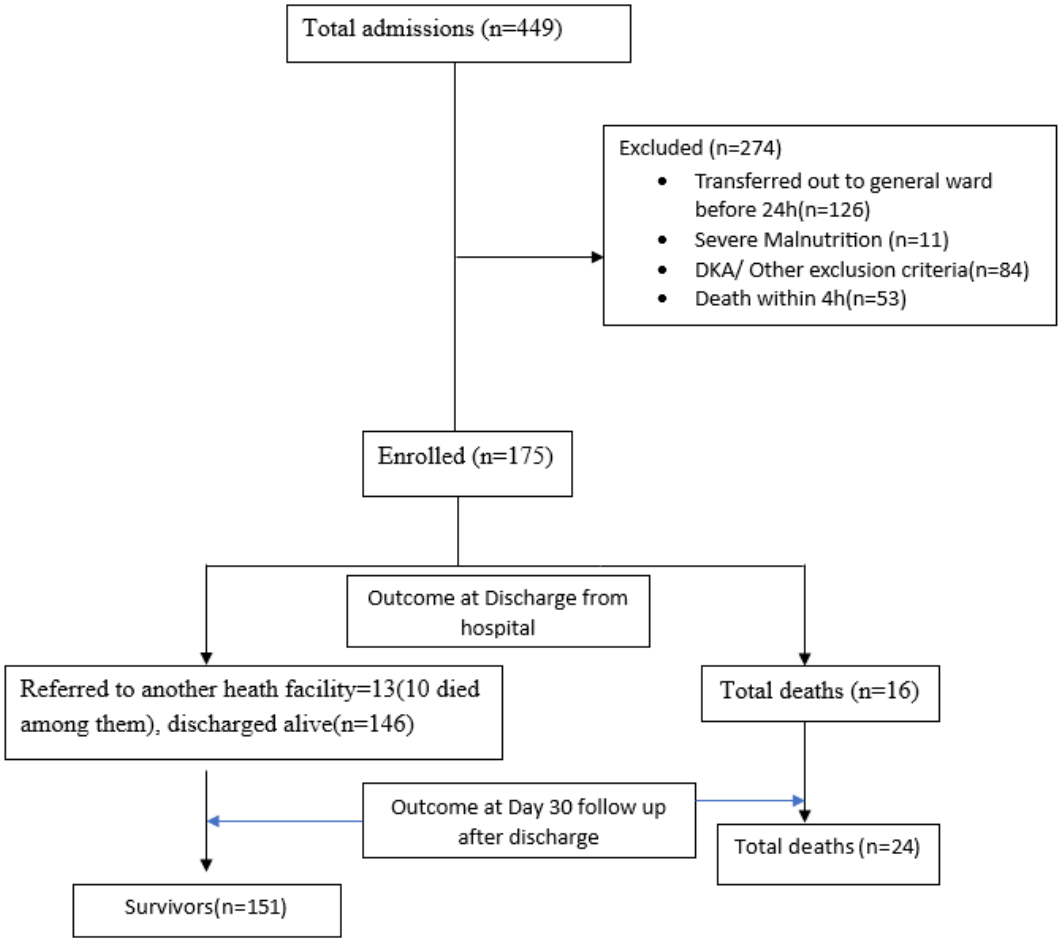

Fig 1: Study Participant Flow Diagram

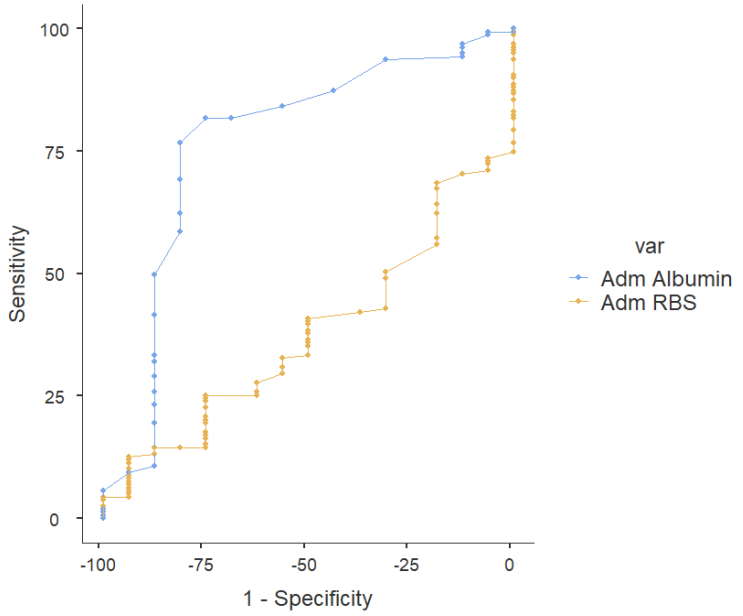

Fig 2: ROC Curve; Albumin level ≥3.5 g/dL with AUC 0.769 (76.7% sensitivity, 81.2% specificity), and RBS value ≥169 mg/dL yielded AUC of 0.378 (12.5% sensitivity, 93.7% specificity)

DISCUSSION:

Sepsis remains a formidable cause of morbidity and mortality in children globally, with the burden especially heavy in resource-limited settings despite advances in supportive care. Early identification of sick children at greatest risk of adverse outcomes upon PICU admission is crucial for prompt, targeted interventions that may improve survival. In this context, we evaluated whether admission hypoglycemia and hypoalbuminemia independently and together predict mortality in children with sepsis. Our cohort had a mean age of 70 months and spanned infancy through adolescence, with wide variations in weight and height reflecting diverse nutritional and developmental profiles[9], [10].

Before PICU discharge, mortality rates did not differ by sex. However, on follow-up girls experienced higher mortality after hospital discharge. This finding contrasts with the general pediatric population, where female infants and children typically have lower mortality rates than boys. Paradoxically, in the PICU setting, boys not only constitute a larger proportion of admissions but also demonstrate superior survival than females, consistent with findings of Almossawi et al[11].

In line with our findings, prior studies from both low- and high-income countries have identified respiratory and central nervous system infections as the predominant causes of paediatric sepsis requiring intensive care. Across aggregate cohorts, the central nervous system was implicated in 33.2% of cases and the respiratory system in 23.1%, mirroring the organ-involvement distribution observed in our dataset[12], [13].

El-Nawawy et al[14] performed a prospective cohort on children aged 1 month to 12 years admitted to the PICU with severe pneumonia and reported that hospital-acquired and ventilator-associated pneumonia were overwhelmingly due to Gram-negative organisms (91.67% and 87.8%, respectively), with Klebsiella, Acinetobacter, and Pseudomonas species predominating, Similarly, our cohort demonstrated a predominance of Gram-negative sepsis. These findings highlight the dual challenge in PICU patients, the metabolic derangements on admission and a high burden of Gram-negative pathogens during their ICU course. Invasive ventilation was most frequently required by children in Group 4, whereas those in Group 6 endured the longest duration of invasive ventilation and the most prolonged PICU stay. Abnormal blood glucose level was encountered among critically ill children.

In our cohort, 54.9% of children were classified as Group 5 on admission, exhibiting normoglycemia and normal serum albumin. This finding highlight that, despite the overall high prevalence of metabolic derangements, a substantial proportion of critically ill children present with entirely normal metabolic profiles to PICU. Children in Group 3 presenting with hyperglycaemia and hypoalbuminemia experienced the worst outcomes, with mortality reaching 40 percent at hospital discharge and climbing to 50 percent by 30 days follow up. The simultaneous derangement of glucose and albumin likely reflects severe systemic inflammation, endothelial dysfunction, and markedly reduced physiological reserve. Normoglycemia was associated with favourable outcomes both in terms of hospital stay and mortality in this study.

Previous studies have also established that glycaemic variability including hyperglycaemia or hypoglycaemia is frequently observed in severe sepsis, pneumonia, traumatic brain injury, and acute malnutrition, and independently predicts higher mortality, prolonged hospital stay, and an increased risk of nosocomial infections and adverse neuro logical outcomes[15], [16]. Hyperglycaemia was linked to higher mortality in our cohort, although this association did not reach statistical significance. The stress induced hyperglycaemia results from increased gluconeogenesis, glycogenolysis, and insulin resistance, and a longer duration of hyperglycaemia is known to worsen prognosis. In one PICU cohort, 56% of children with hyperglycaemia during the first 24 hours experienced higher mortality and longer PICU stays compared to 14% of their normoglycemic peers, yielding an overall mortality of 36% in that period[17].

In this study, patients in group 2 with hypoalbuminemia comprised the highest proportion requiring inotrope support. James et al. also demonstrated that in children with oncologic and hematologic diseases, hypoalbuminemia, particularly serum albumin below 3 g/dL was associated with a greater need for invasive mechanical ventilation and longer ICU stays[18]. Similarly, Singh et al. found that among multiple risk factors, only hypoalbuminemia and severe acute malnutrition remained independently linked to increased PICU mortality[19]. Low albumin undermines immune defence, prolongs recovery, and heightens susceptibility to severe infections. Together, these findings underscore the necessity of early nutritional screening and individualized support for critically ill children.

Sickest patients in the PICU often exhibit low admission RBS and/or low serum albumin levels, which are indicators of poor outcomes, increased mortality risk, prolonged PICU stays, and higher likelihood of needing mechanical ventilation. In our cohort, hypoalbuminemia (serum albumin < 3.5 g/dL) carried a 3.5-fold increase in odds of PICU death and a 2.5-fold greater risk of post-discharge mortality compared with normo-albuminemic children. In contrast, hypoglycaemia and hyperglycaemia did not independently predict mortality. Similarly, Saafvi et al found that albumin < 3.03 g/dL conferred a 1.2-fold higher risk of requiring ventilatory support, while admission hyperglycaemia (> 200 mg/dL) did not[20]. Both studies observed that non-survivors had significantly lower albumin and higher glucose levels on admission than survivors, indicating that concurrent metabolic derangements at presentation mark greater disease severity. These findings underscore

the importance of rapid metabolic screening at admission and early intervention; tailored glycaemic control protocols alongside albumin-focused nutritional support may improve PICU outcomes.

An admission albumin threshold of ≥3.5 g/dL in our cohort yielded an AUC of 0.769 (sensitivity 76.7%, specificity 81.2%) for predicting PICU mortality, slightly more discriminative than findings from Ari et al study [21], which identified a 3.785 g/dL cut-off with an AUC of 0.731 (sensitivity 68.3%, specificity 67%). The higher sensitivity and specificity in our dataset may stem from differences in patient demographics, baseline nutritional reserves, or the timing of albumin measurement. By contrast, evidence for admission random blood sugar as a prognostic tool in Pediatrics remains sparse. In adult critical care, hyperglycaemia thresholds often range from 140–180 mg/dL with ROC AUCs of 0.6–0.7,[2] whereas our RBS ≥169 mg/dL achieved a poor AUC of 0.378 (sensitivity 12.5%, specificity 93.7%). This discrepancy highlights that admission glycaemic status alone is a weak marker of paediatric critical illness severity. However, glycaemic trends may still hold value when interpreted alongside other inflammation and nutritional markers including CRP and albumin to better capture the metabolic complexity of paediatric critical illness.

This study demonstrates that serum albumin measured at PICU admission is a simple, inexpensive, and widely available test with strong predictive value for both in-hospital and post-discharge mortality in paediatric sepsis. Its favourable performance compared with multicentre data supports reliability across varied contexts, including low- and middle-income settings. However, the single-centre, time-bound design, small sample size, reliance on a one-time admission measurement, and focus on short-term outcomes limit generalizability and overlook potential long-term impacts. Future research should prioritise prospective multicentre validation, serial biomarker profiling, extended follow-up, and economic evaluation to optimise adoption and impact.

CONCLUSION:

Serum albumin at PICU admission is a practical, low-cost, high-yield biomarker whereas glucose alone lacks sufficient prognostic sensitivity. When both are paired this enables rapid, scalable risk stratification in pediatric sepsis and can facilitate timely escalation of care and improve clinical outcomes especially in resource-limited settings.

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Informed consent statement: Prior informed written consent was taken from participants legal guardian before enrolment into the study.

Conflict-of-interest statement: No conflict of interest

Data sharing statement: Dataset available from the corresponding author at [drmanglasood@gmail.com].

STROBE checklist: The authors have read the STROBE Statement checklist of items, and the manuscript was prepared and revised according to the STROBE Statement checklist of items.

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