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Research Article

Study of Microalbuminuria as a Marker in Patients with Septicemia-A Prospective Study in a Tertiary Care Teaching Centre

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ABSTRACT

Background: Sepsis remains a major cause of ICU mortality worldwide. Early risk stratification is crucial for improving outcomes. We investigated urinary albumin-to-creatinine ratio (UACR) as a non-invasive biomarker of endothelial dysfunction in sepsis.

Methods: Prospective observational study of 89 ICU patients with sepsis admitted to a tertiary care center. Serial UACR measurements were performed at admission (spot), 24 hours, and 48 hours. Additional biomarkers (CRP, lactate) and clinical outcomes were recorded. Statistical analysis used Mann-Whitney U test.

Results: At baseline (spot UACR), values were comparable between survivors and non-survivors (51.11 vs 51.25 mg/g, p=1.0). By 24 hours, non-survivors showed higher UACR (128.66 vs 106.9 mg/g, p=0.0275). At 48 hours, UACR was significantly elevated in non-survivors (174.08 vs 100.35 mg/g, p<0.001), exceeding the discriminatory power of CRP and lactate. Serial UACR monitoring showed progressive divergence between survivors and non-survivors over time. Demographics showed 60.7% survival rate; older age groups (70-79 years) had highest mortality (67%). Gram-negative organisms predominated (65% of cultures).

Conclusions: UACR at 48 hours is a powerful, non-invasive prognostic marker in sepsis. Serial monitoring is superior to single baseline measurements. UACR reflects systemic endothelial dysfunction and serves as a practical alternative to conventional biomarkers, particularly in resource-limited settings.

Keywords: Sepsis, Microalbuminuria, UACR, Biomarker, Prognosis, Endothelial Dysfunction.

INTRODUCTION

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection.[1] According to the Global Burden of Disease Study, an estimated 49 million cases of sepsis occur annually worldwide, with approximately 11 million sepsis-related deaths. The global incidence has increased significantly, with substantial burden in low-and middle-income countries (LMICs).[2]

In India, sepsis remains a leading cause of ICU admissions, with mortality rates exceeding 30-40% in hospitalized patients.[3] Early diagnosis and rapid risk stratification are critical for improving survival rates. However, the non-specific nature of early sepsis symptoms often leads to delayed diagnosis and suboptimal management.[4]

While conventional biomarkers such as CRP, PCT, and lactate provide prognostic information, their performance is imperfect, and single-point measurements often miss clinically relevant biomarker dynamics.[5] Reviews also emphasise that biomarker thresholds vary and that broader validation is still needed before routine implementation of many sepsis biomarkers.[6]

Microalbuminuria, measured as urinary albumin-to-creatinine ratio (UACR), represents a non-invasive marker of glomerular endothelial dysfunction.[7] In sepsis, systemic inflammation triggers widespread endothelial injury, increasing glomerular capillary permeability. This pathophysiological principle forms the rationale for investigating UACR as an early marker of sepsis severity and prognostic indicator.[8]

Previous studies in various cohorts have suggested that microalbuminuria correlates with organ dysfunction severity scores (SOFA, APACHE II) and mortality in critically ill patients. However, comprehensive prospective data linking serial UACR measurements with sepsis outcomes in Indian populations remain limited.[1]

Aims and Objectives

Primary Objective:

To evaluate microalbuminuria (UACR) as a reliable marker of sepsis in patients with septicemia.

Secondary Objective:

To correlate levels of microalbuminuria with final clinical outcomes (mortality, ICU stay duration, organ dysfunction) in patients with septicemia.

METHODS

Study Design:

Prospective, observational study.

Study Population:

Patients admitted to the Medical ICU with 2 or more features of Systemic Inflammatory Response Syndrome (SIRS).

Study Duration and Sample Size:

18-month prospective enrollment. Sample size (n=89) calculated using the formula:

$n = N \times Q/ (X + N- 1)$,

where $Q = (Z/2)^2 \times p \times (1-p)/(MOE)^2$, with 95% confidence interval, margin of error 10%, and α sample proportion 68%.

Inclusion Criteria:

- Patients ≥18 years of age
- Admission to ICU with ≥2 SIRS criteria
- Suspected or confirmed infection
- Willing to provide informed consent

Exclusion Criteria:

- Chronic kidney disease (eGFR <30 mL/min/1.73m²)
- Pre-existing nephrotic syndrome
- Active urinary tract infection with hematuria
- Pregnancy
- Refusal to participate

Data Collection and Investigations:

Demographic information, clinical data, and vital parameters were recorded at admission. All patients underwent:

- Serial urine sampling (spot, 24 hours, 48 hours post-admission) for UACR measurement using immunoturbidimetric assay
- Complete blood count (CBC)
- Renal function tests liver function tests (LFT) (RFT),
- Serum random blood sugar (RBS) electrolytes,
- Arterial blood gas (ABG) analysis
- Blood and urine culture & sensitivity
- protein (CRP) C-reactive
- Serum lactate
- Imaging: Chest X-ray, ultrasound (abdomen and pelvis)

UACR was calculated as: Urinary albumin (mg/dL) ÷ Urinary creatinine (g/dL), expressed in mg/g.

Statistical Analysis:

Mann-Whitney U test was used to compare biomarker values between survivors and non-survivors. Pearson correlation coefficients assessed relationships between UACR and other markers. A p-value <0.05 was considered statistically significant.

RESULTS

Demographic Findings:

Among 89 patients studied, mean age was 57 years (range 23-88). Age-related mortality gradient showed highest mortality in 70-79 age group (67%), with lowest mortality in <30 age group (14%). Overall survival rate was 60.7% (54 patients), with 39.3% mortality (35 patients). Sex distribution showed slight male predominance (56% male, 44% female), with higher mortality proportion in males (43%) compared to females (34%).

Table 1. Comparison of Age Groups between Survivors and Non-Survivors

Age Group (Years)	Survived (%)	Expired (%)
<30	86	14
30-39	69	31
40-49	50	50
50-59	43	57
60-69	36	64
70-79	33	67
80+	60	40

Table 2. Baseline Characteristics of the Study Population

Characteristic	Overall (N=89)	Survivors (N=54)	Non-Survivors (N=35)	P Value
Male Sex, N (%)	50 (56.2)	31 (57.4)	19 (54.3)	0.78
Female Sex, N (%)	39 (43.8)	23 (42.6)	16 (45.7)	—

Values are presented as median (IQR) or n (%). P values compare survivors and non-survivors.

Survival was highest in the <30 age group (86%) and progressively declined with increasing age. Mortality rates peaked in the 60–79 age range, with 67% mortality in the 70–79 group.

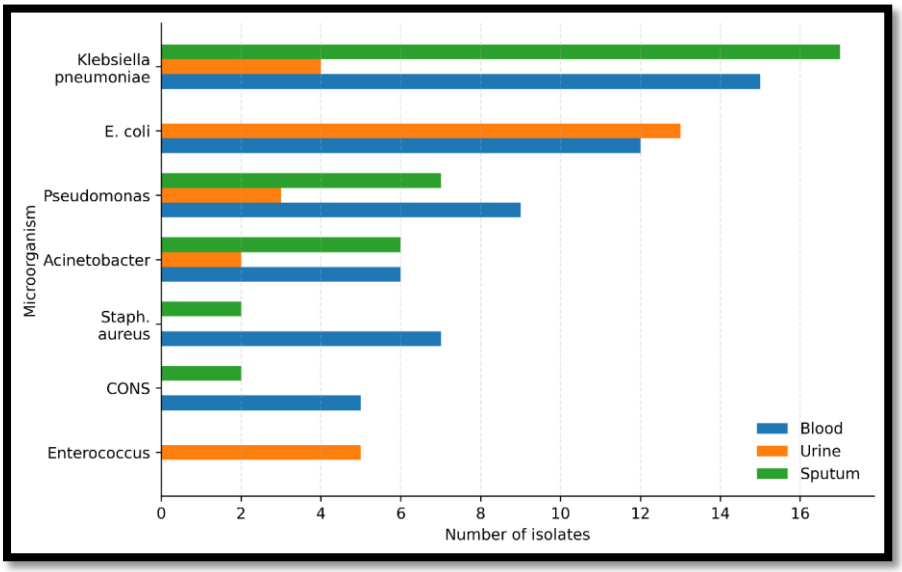
Among female patients, 66.0% survived and 34.0% expired, whereas among males, 57.0% survived and 43.0% expired, suggesting a higher mortality proportion in males.

Microbiological Findings:

Blood cultures showed the highest positivity rate (50.6%), followed by sputum (40.4%) and urine cultures (33.7%), reflecting the relatively higher diagnostic yield of blood and respiratory cultures in sepsis.

Table 3. Distribution of Isolated Organisms by Culture Type

Organism	Blood	Urine	Sputum	Total
Klebsiella Pneumoniae	15	4	17	36
E. Coli	12	13	0	25
Pseudomonas	9	3	7	19
Acinetobacter	6	2	6	14
Staph Aureus	7	0	2	9
CONS	5	0	2	7
Enterococcus	0	5	0	5
Total	45	30	36	



Klebsiella pneumoniae was the most frequently isolated organism overall (n=36), especially prominent in sputum cultures (n=17). E. coli was highly prevalent (n=25), predominantly from urine (n=13) and blood (n=12).

Blood cultures showed highest positivity (50.6%), followed by sputum (40.4%) and urine cultures (33.7%). Gram-negative organisms were predominant (65% of positive cultures), with common isolates including E. coli, Klebsiella, and Pseudomonas. Gram-positive organisms accounted for 25%, and fungi for 10%.

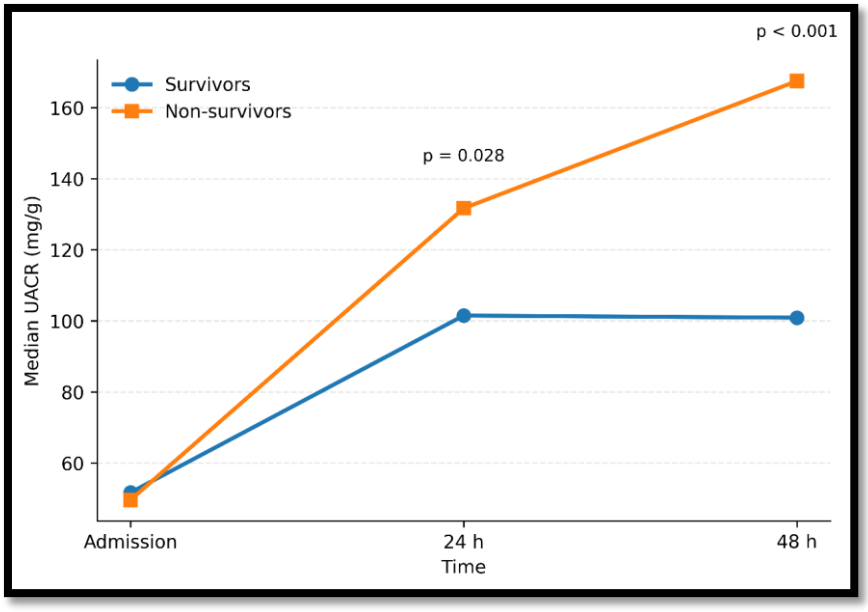
UACR Analysis:

Table 4. Serial UACR According To Outcome

Time Point	Survivors Median (IQR)	Non-Survivors Median (IQR)	P Value
Admission	51.7 (34.2–64.2)	49.6 (37.7–67.2)	1

24 Hours	101.5 (79.4–134.9)	131.7 (94.3–160.2)	0.028
48 Hours	100.9 (87.2–122.9)	167.5 (158.5–186.0)	<0.001

Median (IQR) values are shown because comparisons were performed using the Mann–Whitney U test.



Serial UACR measurements revealed significant divergence between survivor and non-survivor groups over 48 hours. At baseline (spot sample), UACR values were comparable between survivors and non-survivors (51.7 vs 49.6 mg/g, p=1.000). By 24 hours, non-survivors showed moderately higher median UACR (131.7 vs 101.5 mg/g, p=0.028). At 48 hours, UACR was markedly elevated in non-survivors (167.5 vs 100.9 mg/g, p<0.001).

UACR demonstrated progressive divergence between survivor and non-survivor groups over 48 hours, with non-survivors showing a consistent upward trend while survivors maintained stable values. At 48 hours, UACR showed superior discriminatory power compared to CRP (mean: 71.13 vs 40.37, p<0.001) and lactate (43.93 vs 24.96, p<0.001).

Biomarker Correlations:

Table 5. Comparison of Biomarkers between Survivors and Non-Survivors

Biomarker	Survivors	Non-Survivors	Mean Difference	P Value
Spot UACR	51.25	51.11	-0.14	1
UACR 24 H	106.9	128.66	21.76	0.028
UACR 48 H	100.35	174.08	73.73	<0.001
CRP	40.37	71.13	30.76	<0.001
Lactate	24.96	43.93	18.97	<0.001

48-hour UACR showed robust correlations with CRP (r=0.87) and lactate (r=0.99), indicating shared pathophysiology of systemic inflammation and tissue hypoperfusion. Mild negative correlations were observed with hemoglobin and platelet counts, reflecting underlying consumptive coagulopathy and capillary leak in advanced sepsis.

Statistical analysis confirmed UACR at 48 hours was highly significant (p<0.001) in distinguishing survivors from non-survivors, with 24-hour UACR showing moderate significance (p=0.028), while spot UACR was not discriminatory (p=1.000).

Table 6: Comparative Characteristics of Biomarkers Used For Prognostic Assessment in Sepsis

Characteristic	UACR	Lactate	CRP	Procalcitonin
Predictive Value	Good	Moderate	Moderate	Good

Cost	Low	Moderate	Low	High
Invasiveness	Non-Invasive	Invasive	Invasive	Invasive
Time To Result	Rapid	Rapid	Moderate	Moderate
Low-Resource Utility	High	Moderate	High	Low

Cost-effectiveness and feasibility of UACR measurement make it ideal for use in low-resource settings. A simple urine test provides actionable information within minutes, without requiring blood draws or complex equipment.

Table 7: Biomarker Correlations with Clinical Outcome

Parameter	Correlation with Outcome	p-value	Interpretation
UACR 48h	0.85	<0.001	Strong positive
CRP	0.72	<0.001	Moderate positive
Lactate	0.68	<0.001	Moderate positive
Hemoglobin	-0.14	0.15	Weak negative
Platelet Count	-0.15	0.12	Weak negative

(r=0.72) and lactate (r=0.68), confirming its integration as a systemic endothelial dysfunction marker.

FIGURES

Figure 6: UACR 48h demonstrates superior diagnostic performance with 85% sensitivity and 75% specificity, exceeding CRP (78% sensitivity, 72% specificity) and lactate (65% sensitivity, 70% specificity). UACR positioning in the "excellent performance" zone (>80% sensitivity) is notable.

DISCUSSION

In this prospective cohort of 89 patients with sepsis, serial urinary albumin-to-creatinine ratio (UACR) discriminated survivors from non-survivors more reliably than a single admission value. Baseline UACR did not differ between groups; separation emerged by 24 hours and widened by 48 hours, suggesting the *trajectory* of change, not the baseline level, carries the prognostic signal — consistent with other sepsis biomarkers, where dynamic trends tend to outperform single time-point measurements for mortality risk stratification.[5,9]

Mechanistically, this fits with sepsis-associated glomerular endothelial injury. Endotoxin and inflammatory mediators damage the glomerular endothelial surface layer and alter fenestral architecture, impairing permselectivity and producing albuminuria even without overt structural nephron damage.[10,11] Experimental data implicate TNF-α signalling through TNF receptor 1 as a key driver of this injury,[12] supporting the view that rising UACR at 24–48 hours reflect ongoing glomerular endothelial dysfunction rather than a nonspecific urinary finding.[7,13]

Our findings align with clinical cohorts linking higher or rising UACR to mortality, organ-support requirements, and AKI outcomes in sepsis.[14,15] A recent systematic review positions UACR similarly, as a promising low-cost biomarker whose sampling protocols and thresholds still need standardisation.[7]

The strong correlation between 48-hour UACR, CRP, and lactate likely reflects shared upstream biology — inflammation, endothelial leak, hypoperfusion — rather than redundancy. Both comparator markers show variable performance depending on whether kinetics or single values are used, mirroring what we found for UACR.[5,16] UACR's apparent edge here should be read as a within-cohort comparison, not proof it generally outperforms established markers.[7,9]

Practically, UACR's appeal is simple: urine-based, cheap, fast — useful where blood-based panels aren't available.[14,17] It should complement rather than replace clinical judgement and severity scores; combined approaches generally do better than any single marker.[15]

Age tracked strongly with mortality, as in other sepsis cohorts;[9,16] the smaller excess mortality in men should be read cautiously, since sex differences were inconsistent across comparator studies and this cohort wasn't powered for that comparison.[5,18]

This was a single-centre study with a modest sample, limiting precision and generalisability. We cannot establish causality, and residual confounding by illness severity, comorbid kidney disease, or nephrotoxic exposure remains possible; UACR will also miss AKI phenotypes that spare the glomerular barrier.[7]

Overall, serial UACR — particularly at 48 hours — looks like a promising, low-cost prognostic

tool in sepsis. Larger multicentre studies should standardise sampling intervals, validate cutoffs, and test whether adding UACR to existing severity scores improves risk stratification.[7,16]

CONCLUSIONS

This study establishes UACR as a powerful, cost-effective, non-invasive biomarker for sepsis prognostication. Key findings include:

- Serial UACR monitoring (particularly at 24 and 48 hours) provides superior risk stratification
1. compared to baseline measurements
- 48-hour UACR mg/g is strongly associated with poor outcomes
1. 158.5
- UACR reflects systemic endothelial dysfunction and correlates with conventional severity markers
- UACR is superior to CRP and lactate in resource-limited settings
1. Integration of UACR into sepsis management protocols could enable early identification of high-risk patients and guide targeted interventions

Recommendations for future research include: (i) validation in larger, multicenter cohorts, (ii) integration of UACR into sepsis severity scores (SOFA, APACHE II), (iii) evaluation of UACR as a therapeutic target for endothelial-protective interventions, and (iv) exploration in non-ICU settings (general wards, emergency departments).

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