ORIGINAL RESEARCH

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Comparative Validation of Blood Gas Analysis, Perfusion Index, and AKIN and RIFLE Scoring Systems in the Diagnosis of Acute Kidney Injury

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Abstract

Objective: This study aimed to evaluate the potential role of the Peripheral Perfusion Index (PI) and arterial blood gas parameters as early indicators of acute kidney injury (AKI) and to compare their diagnostic utility with the Acute Kidney Injury Network (AKIN) and RIFLE scoring systems. We hypothesized that the PI is proportional to the severity of AKI and may aid in early diagnosis and prognosis of the disease.

Materials and Methods: This prospective study included patients who received a new diagnosis of AKI in the emergency department (ED) of two tertiary hospitals based on AKIN and RIFLE criteria. Focusing on ED-diagnosed AKI ensures that the findings reflect early diagnostic decision-making in a time-sensitive setting, where rapid identification of AKI can impact immediate patient management and disposition. The PI of patients with confirmed AKI was measured using the necessary device, and a case report form was completed. This form recorded demographic characteristics, blood gas values, renal function tests, and AKIN and RIFLE scores.

Results: A total of 264 patients were included in this study. The study was divided into two groups: 132 cases (50%) and 132 controls (50%). A statistically significant difference was found between the case and control groups in terms of hypertension ($p=0.001$), coronary artery disease ($p=0.009$), pH ($p=0.015$), HCO_3 ($p=0.001$), sodium ($p=0.001$), PI ($p=0.001$), urea ($p=0.001$), and creatinine ($p=0.001$). A statistically significant difference was found between AKIN score stages and RIFLE index stages regarding pH, CO_2, HCO_3, perfusion index, urea, creatinine, and potassium levels ($p<0.05$).

Conclusion: Our findings suggest that PI and blood gas analysis may serve as valuable adjuncts to conventional AKI classification, providing earlier insights into renal dysfunction.

Keywords: Acute kidney injury (AKI), AKIN Index, Perfusion Index, RIFLE score

Introduction

Acute kidney injury (AKI) is a serious condition characterized by a rapid decline in kidney function, leading to metabolic imbalances and increased morbidity and mortality [1]. While serum creatinine and urine output are the primary diagnostic markers, they are delayed indicators of renal dysfunction [2]. To improve early recognition and risk stratification, various classification systems, including the RIFLE (Risk, Injury, Failure, Loss of kidney function, and End-stage kidney disease) and Acute Kidney Injury Network (AKIN), have been developed [3,4]. However, there is still a need for additional, non-invasive biomarkers that can provide real-time insights into renal perfusion and function.

Patients with AKI commonly present to emergency departments (EDs) with symptoms such as abdominal pain, decreased urine output, diarrhea, and nausea-vomiting [2]. To standardize diagnosis, the Acute Dialysis Quality Initiative (ADQI) developed the RIFLE criteria in 2004 [5]. The RIFLE classification defines AKI severity in three stages (risk, injury, failure) and describes two clinical outcomes (loss and end-stage renal disease) [6].

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Another scoring system, the AKIN, was introduced in 2005 [7]. This system, which serves as a modification of the RIFLE criteria, classifies AKI severity into three stages: stage 1, stage 2, and stage 3 [7]. Both diagnostic systems rely on changes in serum creatinine levels and urine output, regardless of etiology [5]. In both staging systems, mortality increases as the stage progresses.

Despite the widespread use of AKIN and RIFLE scoring systems, early non-invasive markers for AKI remain limited. Perfusion Index (PI), a microcirculatory parameter measured via pulse oximetry, reflects peripheral tissue perfusion and may serve as a dynamic, early marker of AKI severity [8]. Blood gas analysis (BGA), which provides insights into acid-base balance and metabolic disturbances, may further aid in assessing kidney function beyond conventional creatinine-based methods [9].

This study aimed to evaluate PI and BGA as potential adjuncts concomitant with the AKIN and RIFLE scoring systems in patients with AKI. We hypothesize that lower PI values and specific ABG abnormalities are associated with AKI severity and can assist in early diagnosis and risk stratification.

Materials and Methods

Ethical Approval and Patient Consent

This study was conducted in accordance with the principles of the Declaration of Helsinki and approved by the Gaziantep İslam Science and Technology University Non-Interventional Clinical Research Ethics Committee (protocol number: 2022/389, decision number: 389.36.03, date: 26.02.2022). As the study involved a retrospective review of medical records without patient identifiers or potentially identifying data, the institutional review board (IRB) waived the requirement for patient consent.

Study Design and Setting

This was a retrospective observational study conducted in the EDs of Gaziantep City Hospital and Gaziantep Training and Research Hospital between February 2022 and February 2025.

All AKI diagnoses were independently verified by an Emergency Medicine Specialist (Assistant Professor with >10 years of clinical experience) using KDIGO criteria. Data were collected by ten emergency medicine residents (PGY-1/PGY-2), who were blinded to the study hypothesis and not involved in the clinical decision-making process.

PI measurements were obtained using Masimo Radical-7 Pulse CO-Oximeter (Masimo Corp., Irvine, CA, USA) with the probe placed on the patient's finger. PI was recorded as a non-invasive, objective ratio of pulsatile to non-pulsatile infrared light absorption, serving as an indicator of peripheral perfusion.

Study Population and Sampling

The study included adult patients (≥18 years) presenting to the participating EDs with a diagnosis of AKI confirmed by KDIGO criteria [10].

Inclusion criteria:

• Complete data on PI, arterial blood gas (ABG), and renal function tests

• Diagnosis of AKI according to KDIGO serum creatinine criteria.

Exclusion criteria:

• Chronic kidney disease stage 4 or 5 or on maintenance dialysis

• Chronic acid-base disorders

• Acute conditions likely to independently alter blood gas or PI (e.g., sepsis, diabetic ketoacidosis, respiratory failure, shock)

• Pregnancy

• Incomplete medical records for key variables.

A sample size calculation was performed using G*Power (version 3.1.9.8). Assuming a medium effect size (Cohen's d=0.5), α=0.05, and 80% power, 64 participants per group were required. A total of 264 participants were included (132 AKI patients and 132 controls), exceeding the minimum requirement.

All patient data were recorded in a dedicated, de-identified Microsoft Excel database created for this study.

Outcomes

• Primary outcome:

Association of PI and ABG parameters with the presence and severity of AKI (staged by AKIN and RIFLE).

• Secondary outcomes:

• Diagnostic and prognostic performance of PI and ABG parameters using receiver operating characteristic (ROC) analysis area under the curve (AUC)

• Independent predictive value of PI and ABG for AKI in multivariate logistic regression.

Definitions and Scoring (AKIN & RIFLE)

AKI diagnosis and staging were based on KDIGO criteria, while AKIN and RIFLE classifications were calculated for all AKI patients:

• AKIN criteria:

• Stage 1: ≥0.3 mg/dL or 1.5–2.0× baseline serum creatinine

• Stage 2: 2.0–3.0× baseline serum creatinine

• Stage 3: >3.0× baseline or initiation of renal replacement therapy

• Urine output <0.5 mL/kg/h for ≥6–12 h was noted where available.

• RIFLE criteria:

• Risk: 1.5× baseline creatinine or urine output <0.5 mL/kg/h for 6 h

• Injury: 2× baseline creatinine or urine output <0.5 mL/kg/h for 12 h

• Failure: 3× baseline creatinine or urine output <0.3 mL/kg/h for 24 h or anuria for 12 h

• Loss and ESRD stages were noted if applicable but were not primary outcomes in this ED cohort.

All scoring and stage assignments were reviewed and validated by the supervising emergency medicine specialist.

Statistical Analysis

All statistical analyses were performed using SPSS version 26 (MacOS).

Data handling and descriptive statistics:

• Continuous variables: mean ± standard deviation (SD) if normally distributed; median (IQR) if non-normally distributed

• Normality: Kolmogorov-Smirnov and Shapiro-Wilk tests, and visual histogram/probability plot assessment.

• Categorical variables: frequency (%).

Comparative analyses:

• Two-group comparisons: Student's t-test (normal) or Mann-Whitney U test (non-normal)

• Multi-group comparisons: ANOVA with Bonferroni post-hoc (normal) or Kruskal-Wallis with post-hoc Mann-Whitney U test (non-normal)

• Categorical variables: Chi-square test or Fisher's exact test as appropriate

• Correlation and predictive modeling: Multivariate logistic regression with odds ratios [OR, 95% confidence interval (CI)]

• Diagnostic performance: ROC curves and AUC with 95% CIs.

Significance: Two-sided $p<0.05$ was considered statistically significant. No significant missing data were observed for key variables.

Results

A total of 264 patients were included in this study. Methodologically, the study was divided into two equal groups: 132 cases (50%) and 132 controls (50%). The average (AVG) age of the patient group (72.35±10.87) was statistically significantly higher than that of the control group (56.13±19.90) ($p=0.001$). Hypertension was significantly more prevalent in the AKI group (65.2%) compared to the control group (30.0%) ($p<0.001$). Similarly, coronary artery disease was present in 40.9% of AKI patients versus 13.3% of controls ($p=0.002$). These differences suggest a higher baseline cardiovascular risk profile in the AKI group.

The difference in gender distribution between the groups was not found to be statistically significant ($p=0.679$) (Table 1).

Hypertension and coronary artery disease were found to be statistically significantly higher in the patient group compared to the control group ($p=0.001$ and $p=0.009$, respectively). No statistically significant differences were observed between the groups for other variables ($p>0.05$) (Table 1).

In the study, statistically significant differences were observed between the "Patient" and "Control" groups in terms of pH ($p=0.015$), HCO_3 ($p=0.001$), sodium ($p=0.001$), PI ($p=0.001$), urea ($p=0.001$), and creatinine ($p=0.001$) values (Table 2).

A total of 132 patients were categorized using the AKIN and RIFLE scoring systems, (Table 3). According to the AKIN Score, most patients were classified as Stage 1 (63.6%), followed by Stage 2 (28.8%) and Stage 3 (7.6%). In the RIFLE classification, the Risk category was the most common (66.7%), followed by Injury (21.2%), with Failure and Loss each comprising 6.1% of the patient group.

Table 4 summarizes the evaluation of AKIN and RIFLE scores in the patient group, providing the mean AVG, SD, median values, statistical significance (p), and post-hoc comparisons for each parameter.

• AKIN Score Findings:

• PI significantly decreased as AKI severity increased (1>2>3, $p=0.001$).

• Urea and creatinine levels were highest in Stage 3 ($p=0.001$, ranking 3>2>1).

• HCO_3 levels progressively decreased, with Stage 3 having the lowest values ($p=0.001$).

• No significant differences were found for potassium, sodium, lactate, or O_2 levels.

• RIFLE Score Findings:

• PI declined progressively, with the lowest values in the failure stage ($p=0.001$: ranking 1>2>4>3).

• Urea and creatinine levels were significantly higher in the Loss stage compared to all other stages ($p=0.001$, ranking 4>3>2>1).

• O_2 levels were highest in the Failure stage ($p=0.015$, ranking 3>1-2-3.1>2-4).

• No significant differences were found for pH, potassium, sodium, or lactate levels.

Table 1. Demographic and clinical characteristics of patient and control groups

		Control		Case		p
Sex	Male	64	(48.5%)	72	(70.60%)	0.679*
	Female	68	(51.5%)	60	(66.70%)	
Age (AVG ± SD)		56.13	±19.90	72.35	±10.87	0.001**
HT	None	62	(77.50)	18	(22.50)	0.001
	Present	70	(38.04)	114	(61.96)	
CAD	None	74	(66.07)	38	(33.93)	0.009
	Present	58	(38.16)	94	(61.84)	
DM	None	82	(56.16)	64	(43.84)	0.097
	Present	50	(42.37)	68	(57.63)	
CHF	None	88	(49.44)	90	(50.56)	0.233
	Present	44	(51.16)	42	(48.84)	
CKD	None	132	(51.16)	126	(48.84)	0.550
	Present	0	(0.00)	6	(100.00)	
COPD	None	100	(45.87)	118	(54.13)	0.697
	Present	32	(69.57)	14	(30.43)	

*Chi-square, **Mann-Whitney U test.

HT: Hypertension, CAD: Coronary artery disease, DM: Diabetes mellitus, CHF: Chronic heart failure, CKD: Chronic kidney disease, COPD: Chronic obstructive pulmonary disease, AVG: Average, SD: Standard deviation

Table 2. Comparison of Perfusion Index, arterial blood gases and laboratory parameters between patient and control groups

	Group	n	AVG	SD	Median	p
pH	Case	132	7.36	0.10	7.36	0.015
	Control	132	7.40	0.03	7.41	
CO_2	Case	132	39.12	8.19	37.35	0.059
	Control	132	42.77	8.25	40.85	
HCO_3	Case	132	22.08	5.40	21.8	0.001
	Control	132	27.01	8.7	25	
Potassium	Case	132	4.11	0.68	4.095	0.383
	Control	132	3.99	0.32	4.02	
Sodium	Case	132	134.18	5.17	135.3	0.001
	Control	132	138.69	2.18	138.8	
Lactate	Case	132	2.13	1.92	1.65	0.559
	Control	132	1.71	0.77	1.415	
O_2	Case	132	73.05	21.18	77.15	0.649
	Control	132	74.93	18.3	84.05	
Perfusion Index	Case	132	1.19	0.57	1.1	0.001
	Control	132	4.07	1.65	3.95	
Urea	Case	132	71.69	46.02	56.5	0.001
	Control	132	33.73	10.4	29.95	
Creatinine	Case	132	2.02	1.77	1.5	0.001
	Control	132	0.92	0.17	0.935	

*Mann-Whitney U test, **Independent Groups t-test.

AVG: Average, SD: Standard deviation

Table 3. Distribution of the AKIN and RIFLE scores in patient group

		n	%
AKIN score	Stage-1	84	63.6
	Stage-2	38	28.8
	Stage-3	10	7.6
	Total	132	100
		n	%
RIFLE score	Risk	88	66.7
	Injury	28	21.2
	Failure	8	6.1
	Loss	8	6.1
	Total	132	100

AKIN: Acute Kidney Injury Network

A multivariate logistic regression analysis was performed to determine whether PI independently predicted AKI. The model included PI, age, and sex as covariates. PI was found to be a statistically significant independent predictor of AKI (odds ratio (OR): 0.05, 95% CI: 0.01-0.21, $p<0.001$), indicating that lower PI values were strongly associated with the presence of AKI. These results suggest a much stronger association between PI and AKI than previously reported, supporting its role as a potential early diagnostic marker.

A comparison of PI values between AKI and control groups revealed a significant difference. As shown in Figure 1, patients diagnosed with AKI had markedly lower PI values compared to the control group ($p<0.001$), highlighting the potential of PI as an early and non-invasive indicator of renal dysfunction.

To further assess the diagnostic value of PI in predicting AKI, a ROC curve analysis was performed. As illustrated in Figure 2, the PI demonstrated excellent discriminative ability, yielding an area AUC of 0.97, indicating that PI can effectively differentiate between AKI and non-AKI patients in the emergency setting.

The model demonstrated excellent discriminative power, with an AUC of 0.97, suggesting that PI can reliably differentiate between AKI and non-AKI patients.

Discussion

This study demonstrates that lower PI values and ABG abnormalities are significantly associated with AKI presence and severity, with PI showing excellent discriminative ability (AUC =0.97).

AKI is commonly diagnosed using serum creatinine and urine output, both of which are delayed indicators of renal dysfunction [11]. This study highlights the

Table 4. comparative analysis of perfusion index, arterial blood gases and laboratory parameters based on the AKIN and RIFLE scores in patient group

Parameter	AKIN stage 1 (n=84)	AKIN stage 2 (n=38)	AKIN stage 3 (n=10)	p value (AKIN)	Post- Hoc (AKIN)	RIFLE risk (n=88)	RIFLE injury (n=28)	RIFLE failure (n=8)	RIFLE loss (n=8)	p value (RIFLE)	Post--hoc (RIFLE)
pH	7.38±0.06	7.37±0.10	7.18±0.19	0.023	1>3	7.38±0.06	7.36±0.10	7.40±0.13	7.1±0.21	0.091	-
CO_2 (mmHg)	41.04±7.53	37.29±8.40	29.88±5.53	0.006	1>3	40.42±7.92	40.26±7.65	30.03±1.87	29.85±6.39	0.007	1-2>3-4
HCO_3 (mEq/L)	23.55±4.69	21.39±4.08	12.32±5.50	0.001	1>3.2>3	23.31±4.74	21.94±4.40	18.93±4.38	12.15±6.33	0.007	1-2>3-4.3>4
Potassium (mEq/L)	4.01±0.51	4.17±0.87	4.74±0.95	0.067	-	3.97±0.52	4.27±0.95	4.47±0.40	4.67±1.08	0.091	-
Sodium (mEq/L)	134.70±5.55	133.81±4.16	131.32±5.17	0.283	-	134.69±5.51	134.89±3.34	130.48±4.96	129.90±4.71	0.116	-
Lactate (mmol/L)	1.93±1.78	2.23±1.41	3.41±4.00	0.436	-	2.03±1.85	2.00±1.10	1.76±1.27	4.07±4.28	0.900	-
O_2 (mmHg)	75.02±20.13	69.5±24.07	69.98±20.44	0.619	-	74.81±19.70	63.21±23.77	95.75±5.22	65.48±20.54	0.015	3>1-2-3.1>2-4
Perfusion Index	1.36±0.56	0.97±0.45	0.59±0.15	0.001	1>2>3	1.37±0.56	0.96±0.40	0.51±0.20	0.65±0.06	0.001	1>2>4>3
Urea (mg/dL)	55.69±30.27	76.58±22.95	187.54±55.49	0.001	3>2>1	56.24±29.67	74.61±22.35	135.45±91.60	167.68±38.39	0.001	4>3> 2>1
Creatinine (mg/dL)	1.39±0.36	2.04±0.43	7.28±3.21	0.001	3>2>1	1.40±0.36	2.05±0.46	3.13±1.69	7.68±3.55	0.001	4>3>2>1

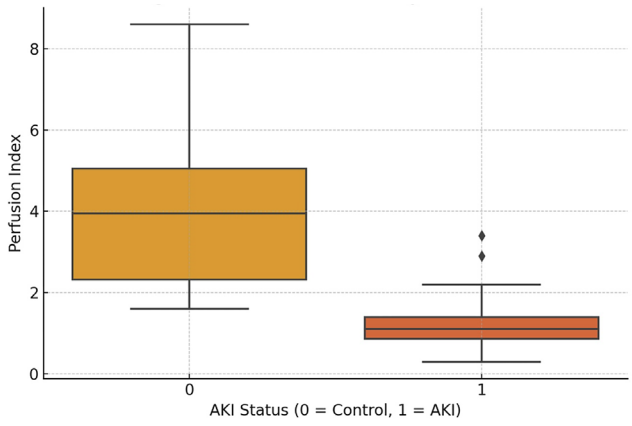

Figure 1. Boxplot of perfusion index in patients with and without acute kidney injury (AKI).

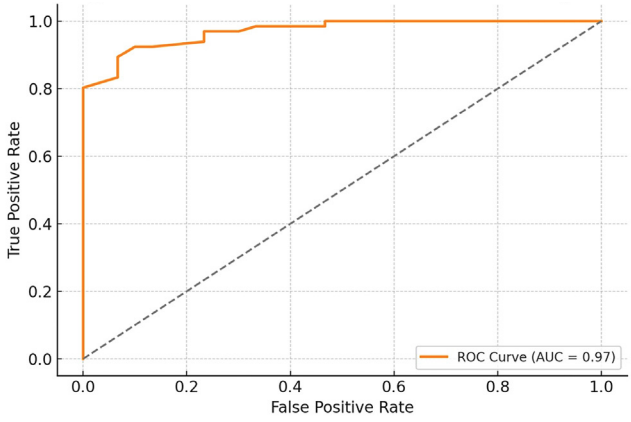

Figure 2. Receiver operating characteristic (ROC) curve of perfusion index for predicting acute kidney injury (AKI)

potential role of PI and ABG The AKI group demonstrated significantly lower perfusion index values compared to controls ($p<0.001$), supporting its role as a strong predictor of renal dysfunction parameters as early, non-invasive markers of AKI severity. Prior to the adoption of the KDIGO classification, the RIFLE (Risk, Injury, Failure, Loss, End-stage) and AKIN criteria were commonly used to define and stage AKI. These systems varied in thresholds and timing for serum creatinine changes, or urine output reduction. The KDIGO classification later harmonized these definitions into a single unified system, improving diagnostic consistency and facilitating comparisons across studies [5,12].

In our cohort, the distribution of AKI severity differed slightly between the AKIN and RIFLE classifications. The RIFLE system identified a greater number of patients in early AKI stages (Risk and Injury), suggesting that its broader creatinine thresholds and the inclusion of more permissive urine output criteria may capture mild renal impairment earlier. In contrast, AKIN classification demonstrated better alignment with patients who progressed to severe AKI as its staging is more sensitive to small but clinically relevant rises in serum creatinine over shorter time intervals. Our findings highlight that using both systems in parallel can provide complementary information, with RIFLE favoring early detection and AKIN better reflecting clinically significant disease progression.

In AKI, the ability of the kidneys to maintain acid-base balance may be impaired, leading to acidosis [13]. Our findings demonstrate a significant decline in pH and bicarbonate (HCO_3) levels in the AKI group, consistent with metabolic acidosis. The mean pH in the AKI group was 7.36, compared to 7.40 in controls ($p=0.015$), while bicarbonate levels were also significantly lower ($p=0.001$). These changes align with previous studies describing impaired acid-base regulation in AKI due to reduced bicarbonate reabsorption and increased acid retention [9]. The worsening of metabolic acidosis with advancing AKI stages further supports its role as an indicator of disease progression [14]. The development of metabolic acidosis may result from the kidneys' inability to excrete acid loads. This was further supported by Hoste al. [6], who stated that kidney failure can lead to decreased serum bicarbonate levels, triggering metabolic acidosis. In advanced stages, CO_2 levels also decreased alongside HCO_3, reflecting progressive metabolic acidosis [15]. These findings emphasize the importance of integrating ABG analysis into AKI assessment, as metabolic disturbances can serve as early warning signs of worsening kidney function.

Additionally, PI was significantly lower in AKI patients compared to controls ($p=0.001$), indicating a reduction in renal perfusion. This aligns with findings from Legrand et al. [8], who reported that AKI is associated with microcirculatory disturbances due to both intrarenal and systemic factors. The correlation between PI and AKI severity suggests that renal hypoperfusion contributes to ischemic kidney injury, further exacerbating cellular damage [16]. As AKI progresses, PI declines, supporting its potential use as a dynamic, real-time indicator of renal microvascular health. This was further supported by a study, which stated that microcirculatory disturbances in AKI could lead to reduced PI [17]. Patients in the most severe AKI stages exhibited lower PI values, indicating worsening renal hypoperfusion, which likely contributes to metabolic deterioration and adverse clinical outcomes [18]. Our findings demonstrated a progressive decline in PI with increasing AKI severity. This suggests that PI may not only serve as a diagnostic marker but also as a potential indicator of disease progression. The observed trend aligns with the pathophysiology of AKI, in which renal microcirculatory dysfunction and impaired perfusion worsen with advancing stages. As ischemia intensifies, tissue oxygen delivery diminishes, which may be reflected by declining PI values. These findings underscore the potential utility of PI as a dynamic biomarker for real-time assessment of renal perfusion

status and AKI severity. Monitoring PI trends could therefore aid in stratifying risk and guiding therapeutic interventions in critically ill patients. Our results suggest that both PI and ABG parameters could serve as valuable adjuncts to traditional AKI classification systems, such as AKIN and RIFLE, by providing earlier and more dynamic physiological insights.

Given the delayed nature of creatinine-based AKI diagnosis, incorporating PI and ABG analysis into routine assessment may facilitate earlier intervention. PI, in particular, may serve as an immediate, non-invasive marker of renal perfusion that reflects real-time changes in kidney function. Clinicians may consider PI and ABG monitoring in emergency and critical care settings to improve risk stratification and guide treatment decisions.

Contrary to previous studies, which reported modest associations between PI and renal outcomes, our analysis revealed a significantly stronger relationship. The multivariate logistic regression and ROC curve, (AUC =0.97), both demonstrate that PI is a highly effective, non-invasive predictor of AKI in emergency settings. This discrepancy may be due to differences in population selection, stricter exclusion of confounding conditions, or more stringent use of a validated pulse co-oximeter device. Further studies with larger, more diverse populations are warranted to validate these findings.

Study Limitations

This study has several limitations. The relatively small sample size may reduce generalizability and limit statistical power. Its cross-sectional design prevents the establishment of causality between PI, ABG parameters, and AKI outcomes. As the study was conducted in tertiary care centers, the findings may not be fully applicable to non-tertiary or outpatient settings. Selection bias is also possible, as mild AKI cases that resolved without hospitalization could be underrepresented.

In addition, physiological confounders that influence PI measurements—such as body temperature, peripheral vascular tone, vasoconstriction, and the use of vasoactive medications, -were not controlled for, which may affect measurement accuracy. Only single time-point measurements of PI and ABG were obtained, limiting the assessment of dynamic trends or progression. Urine output data were not available for all patients, which may reduce the accuracy of AKIN and RIFLE staging. Finally, the heterogeneity of the control group may have introduced unmeasured confounding, as their underlying clinical conditions were diverse.

Conclusion

This study highlights the potential role of PI and ABG parameters as complementary tools for the diagnosis and severity stratification of AKI in the ED. PI independently predicted AKI severity (OR =0.05) and demonstrated excellent discriminative ability (AUC =0.97), underscoring its diagnostic and prognostic utility alongside conventional scoring systems such as AKIN and RIFLE. Integrating PI into AKI risk algorithms may facilitate earlier identification and more informed management decisions, although external validation in larger, multi-center cohorts is warranted to define standardized cutoff values and confirm its clinical impact.

Ethics

Ethics Committee Approval: This study was conducted in accordance with the principles of the Declaration of Helsinki and approved by the Gaziantep Islam Science and Technology University Non-Interventional Clinical Research Ethics Committee (protocol number: 2022/389, decision number: 389.36.03, date: 26.02.2022).

Informed Consent: As the study involved a retrospective review of medical records without patient identifiers or potentially identifying data, the institutional review board (IRB) waived the requirement for patient consent.

Footnotes

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References

1. Almazmomi MA, Esmat A, Naeem A. Acute kidney injury: definition, management, and promising therapeutic target. Cureus. 2023;15:51228.
2. Mercado MG, Smith DK, Guard EL. Acute kidney injury: diagnosis and management. Am Fam Physician. 2019;100:687 694.
3. Saingam P, Jain T, Woicik A, Li B, Candry P, Redcorn R, et al. Integrating socio economic vulnerability factors improves neighborhood scale waste water based epidemiology for public health applications. Water Res. 2024;254:121415.
4. Andrikos E, Tseke P, Balafa O, Cruz DN, Tsinta A, Androulaki M, et al. Epidemiology of acute renal failure in ICUs: a multi center prospective study. Blood Purif. 2009;28:239 44.
5. Bellomo R, Ronco C, Kellum JA, Mehta RL, Palevsky P; Acute Dialysis Quality Initiative Workgroup. Acute renal failure-definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) group. Crit Care. 2004;8:204 12.
6. Hoste EA, Bagshaw SM, Bellomo R, et al. Epidemiology of acute kidney injury in critically ill patients: the multinational AKI EPI study. Intensive Care Med. 2015;41:1411 23.
7. Kidney disease: improving global outcomes (KDIGO). KDIGO Clinical Practice Guideline for acute kidney injury. 2012. Available from: https://kdigo.org/wp-content/uploads/2016/10/KDIGO-2012-AKI-Guideline-English.pdf.
8. Legrand M, Dupuis C, Simon C, Gayat E, Mateo J, Lukaszewicz AC, et al. Association between systemic hemodynamics and septic acute kidney injury in critically ill patients: a retrospective observational study. Crit Care. 2013;17:278.

9. Kraut JA, Madias NE. Metabolic acidosis of CKD: an update. Am J Kidney Dis. 2016;67:307-17.

10. Kidney Disease: Improving global outcomes (KDIGO) acute kidney injury work group. KDIGO clinical practice guideline for acute kidney injury. Kidney Int Suppl. 2012;2(1):1-138.

11. Devarajan P. Biomarkers for the early detection of acute kidney injury. Curr Opin Pediatr. 2011;23:194-200.

12. Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, Warnock DG, et al. Acute kidney injury network: report of an initiative to improve outcomes in acute kidney injury. Crit Care. 2007;11:31.

13 Kamel KS, Halperin ML. Use of urine electrolytes and urine osmolality in the clinical diagnosis of fluid, electrolytes, and acid base disorders. Kidney Int Rep. 2021;6:1211-24.

14. Bellomo R, Kellum JA, Ronco C. Acute kidney injury. Lancet. 2012;380:756 66.

15. Kraut JA, Madias NE. Metabolic acidosis: pathophysiology, diagnosis and management. Nat Rev Nephrol. 2010;6:274-85.

16. Ince C, Mik EG. Microcirculatory and mitochondrial hypoxia in sepsis, shock, and resuscitation. J Appl Physiol (1985). 2016;120:226-35.

17. Watchorn J, Huang D, Bramham K, Hutchings S. Decreased renal cortical perfusion, independent of changes in renal blood flow and sublingual microcirculatory impairment, is associated with the severity of acute kidney injury in patients with septic shock. Crit Care. 2022;26:261.

18. Wan L, Bagshaw SM, Langenberg C, Saotome T, May C, Bellomo R. Pathophysiology of septic acute kidney injury: what do we really know? Crit Care Med. 2008;36:198-203.