

The Predictive Utility of Arterial Blood Gas Analysis for ICU Transfer and In-Hospital Mortality Among General Internal Medicine Inpatients

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Purpose: Arterial blood gas (ABG) analysis is a widespread, low-cost diagnostic tool routinely used to assess the metabolic status of patients in internal medicine wards, a population characterized by a high burden of chronic comorbidities. However, its prognostic value for collectively predicting adverse outcomes such as ICU transfer and mortality remains insufficiently investigated. This study aimed to evaluate the ability of ABG parameters to predict the composite outcome of ICU transfer and in-hospital mortality in this patient population.

Patients and Methods: This retrospective cohort study was conducted at Haseki Training and Research Hospital and included 15,698 patients hospitalized in the Department of Internal Medicine between January 2020 and January 2025. Demographic data, medical history, laboratory parameters (including hemoglobin, white blood cell count, creatinine, albumin, procalcitonin, and blood gas analysis), and outcomes (ICU transfer and in-hospital mortality) were retrieved from the electronic hospital information system. Patients were stratified based on ICU transfer status and in-hospital mortality for comparative analysis.

Results: The study included 9057 patients (mean age 63.5±18.1 years; 51% female). The overall ICU admission and mortality rates were 6.2% (n=564) and 1.9% (n=168), respectively. The ICU and non-survivor groups were significantly older and exhibited a more pronounced inflammatory response (elevated CRP, neutrophils; decreased lymphocytes, albumin) along with more severe metabolic disturbances (elevated lactate; decreased bicarbonate) compared to their counterparts. Multivariate analysis identified age, neutrophil count, CRP, albumin, and lactate levels as independent predictors for both ICU admission and mortality.

Conclusion: This study established that current blood gas parameters, particularly lactate and pCO₂, were useful in stratifying the risk for both intensive care unit transfer and in-hospital mortality.

Keywords: internal medicine inpatients, arterial blood gas, prognosis, predictive utility

Introduction

Internal medicine clinicians are responsible for a patient population characterized by a high burden of chronic comorbidities, including diabetes, heart failure, and chronic kidney disease, as well as an advanced age demographic.¹ Given their increased vulnerability to infection and tendency for rapid clinical decline, these patients face substantially higher rates of ICU transfer and in-hospital mortality compared to those in other wards.^{2,3} Despite close monitoring, clinical deterioration in internal medicine wards is often not recognized early, resulting in delayed ICU admission and poorer outcomes. The early recognition of patient deterioration is critical, as it permits the expedited transfer of high-risk individuals to the intensive care unit and allows for the earlier administration of critical interventions.

Arterial blood gas (ABG) analysis is a widespread, routine, and low-cost diagnostic tool used globally to assess pulmonary gas exchange, acid-base balance, and metabolic status.^{4,5} Parameters derived from ABG (pH, bicarbonate, pCO₂, lactate, etc.) provide immediate and critical information about a patient's physiological balance. However, despite

the routine use of these parameters, their comprehensive prognostic value when considered collectively in relation to ICU transfer and mortality has not been sufficiently investigated.

Previous studies have demonstrated that individual ABG parameters, particularly lactate levels, acid–base disturbances, and carbon dioxide abnormalities, are associated with adverse clinical outcomes, including increased mortality and need for intensive care support in hospitalized patients. Among the available evidence, a retrospective study by Hue et al demonstrated the prognostic value of blood gas analysis in a large inpatient cohort. The study revealed significant associations between several blood gas parameters and adverse outcomes. Specifically, general acidosis, metabolic acidosis, lower actual bicarbonate levels and hypocapnia at admission were associated with the development of acute kidney injury during hospitalization. Moreover, both hypocapnia and general acidosis emerged as independent predictors of in-hospital mortality.⁶ In previous studies with COVID-19, abnormalities in arterial blood gas parameters—particularly elevated pH and lactate levels—were shown to be significantly associated with increased mortality, highlighting the potential prognostic value of ABG analysis in predicting adverse clinical outcomes.^{7,8} However, the majority of existing studies focus on selected populations such as critically ill or septic patients, and data on the prognostic value of routinely obtained ABG parameters in a general internal medicine inpatient setting remain limited.

Given these observations, this study seeks to identify arterial blood gas-derived parameters as early indicators associated with clinical deterioration, defined by ICU transfer and in-hospital mortality, in a general internal medicine inpatient population. By focusing on routinely obtained ABG parameters at admission, we aim to contribute to the identification of readily available markers that may assist clinicians in the early risk stratification of internal medicine inpatients.

Material Methods

Patients

Between January 2020 and January 2025, a total of 15,698 patients were hospitalized in the Department of Internal Medicine. Exclusion criteria were defined as follows: patients younger than 18 years, pregnant women, those who decided to refuse treatment patients admitted more than 24 hours after symptom onset, readmissions, and cases with insufficient clinical data (Figure 1). For patients with multiple hospitalizations, only the first admission during the study period was analyzed to avoid duplication. Patients who reached primary endpoints or experienced all-cause mortality due to unnatural causes (eg, accident, suicide, homicide) within the follow-up period were also excluded. Mortality data were recorded from electronic hospital information system. Demographic characteristics, medical history, mortality information, and laboratory results were retrieved from the electronic hospital information system and databases of Haseki Training and Research Hospital.

Ethical Aspects

This study was approved by the Ethics Committee of Haseki Training and Research Hospital, University of Health Sciences, Istanbul, Turkey (Approval No: 66–2023; Approval Date: March 23, 2023). All procedures were carried out in accordance with the principles of the Declaration of Helsinki and good clinical practice guidelines. Written informed consent was obtained from all patients at the time of hospitalization. Data regarding demographic characteristics, medical history, laboratory findings, and follow-up outcomes were obtained from the hospital's electronic management system and the Turkish National Mortality Registry.

Study Design

This retrospective cohort study included patients hospitalized between January 1, 2020, and January 1, 2025. Laboratory parameters recorded at admission included hemoglobin, white blood cell count, neutrophils, lymphocytes, creatinine, uric acid, glucose, albumin, procalcitonin, and blood gas analysis, in addition to demographic variables such as age and sex. Blood samples were obtained after a 12-hour fasting period on the first day of hospitalization. Length of hospital stay, intensive care unit (ICU) transfer, and in-hospital mortality were evaluated using the electronic hospital information system. We hypothesized that arterial blood gas–derived parameters measured at admission are associated with an increased risk of intensive care unit transfer and in-hospital mortality among general internal medicine inpatients. Patients were stratified into two groups

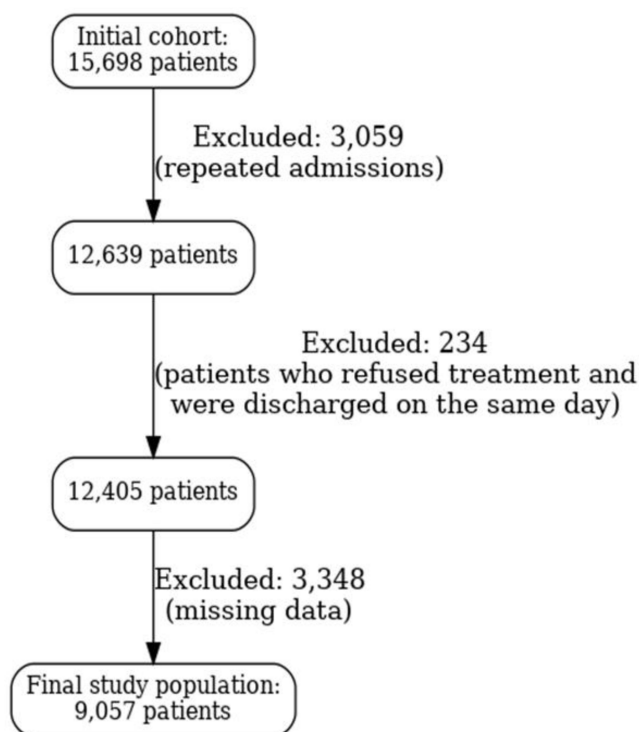


Figure 1 Flow diagram of the selection of patients.

based on ICU transfer status (transferred vs non-transferred), and blood gas and biochemical parameters were compared between these groups. Similarly, patients were categorized according to in-hospital mortality (survivors vs non-survivors), and their blood gas and biochemical parameters were analyzed accordingly.

Statistical Analysis

Descriptive statistics of the data were presented using mean, standard deviation, median, minimum, maximum, frequency, and percentage values. The distribution of the variables was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests. The Mann–Whitney *U*-test was used for the analysis of non-normally distributed quantitative independent variables. The chi-square test was used to analyze qualitative independent variables. Effect sizes were further analyzed using univariate and multivariate logistic regression analyses. All statistical analyses were performed using SPSS version 28.0 (IBM Corp., Armonk, NY, USA).

Results

The age of the patients ranged from 17 to 112 years, with a mean of 63.5 ± 18.1 years. Regarding gender distribution, 51.0% of the patients were female ($n = 4618$) and 49.0% were male ($n = 4439$). A total of 93.8% of the patients ($n = 8493$) were discharged. A total of 6.2% of the patients ($n = 564$) were transferred to the intensive care unit (ICU), and 1.9% ($n = 168$) died.

The ICU group had a significantly higher proportion of male patients and a greater mean age compared to the non-ICU group. White blood cell (WBC), neutrophil count, CRP were significantly higher in the ICU group compared to the non-ICU group. Conversely, hemoglobine, platelet, lymphocyte count and albumin levels were significantly lower in the ICU group ($p < 0.05$). The ICU group had significantly higher pO_2 , lactate, and oxygen saturation (SO_2) levels, while pCO_2 , base excess, and bicarbonate levels were significantly lower than non-ICU group. (Table 1). In the multivariate model, age, platelet, neutrophil count, CRP, uric acid, albumin, pCO_2 , lactate and sO_2 levels were identified as independent and statistically significant predictors for differentiating between ICU and non-ICU patients (Table 2).

In the group of patients who died, the mean age was significantly higher than in the group who survived. There was no statistically significant difference in gender distribution between nonsurvivors and survivor groups ($p > 0.05$).

Table 1 Baseline Characteristics of Patients According to ICU Transfer Status

	Non-ICU (n:8493)		Transfer to ICU (n:564)		P value
	Mean ± SD (n) %	Median	Mean ± SD (n) %	Median	
Age (year)	63.0±18.1	67.0	71.6±16.1	75.0	0.000
Gender: n(%)	Female	4358(51.3%)	260(46.1%)		0.016
	Male	4135(48.7%)	304(53.9%)		
White blood cell count.×10 ⁹ /L	9.0±7.5	7.8	11.3±9.9	8.9	0.000
Hemoglobin (g/dL)	10.4±2.6	10.6	10.2±2.5	10.0	0.002
Platelet.×10 ⁹ /L	225.2±106.3	214.0	200.4±117.0	188.5	0.000
Neutrophil,×10 ⁹ /L	6.82±5.55	5.58	9.07±7.50	7.30	0.000
Lymphocyte,×10 ⁹ /L	1.45±2.15	1.22	1.15±2.50	0.76	0.000
Hs-CRP (mg/L)	65.4±80.4	30.4	113.6±95.6	94.1	0.000
Albumin (g/dL)	34.8±5.5	35.0	30.4±6.4	31.0	0.000
pH (Arterial)	7.39±0.07	7.39	7.38±0.09	7.39	0.397
pCO ₂ (mmHg)	42.7±9.2	42.0	41.5±13.2	39.5	0.000
pO ₂ (mmHg)	49.5±26.0	43.1	55.3±33.3	46.4	0.000
Lactate (mmol/L)	1.79±0.78	1.65	2.26±1.35	1.94	0.000
Base Deficit (mmol/L)	0.19±6.56	0.40	-1.16±7.81	-1.10	0.000
Bicarbonate (mmol/L)	25.2±5.9	25.2	24.0±7.0	23.8	0.000
sO ₂ (%)	71.2±18.8	74.0	73.9±18.8	77.3	0.001
Length of stay (day)	8.6±7.3	7.0	9.4±10.7	6.0	0.002

Note: Statistically significant variables ($p < 0.05$).

Abbreviations: ALT, Alanine aminotransferase;CRP, C-reactive protein; Ph, potential of hydrogen; Pco2, The partial pressure of carbon dioxide; po2, The partial pressure of oxygen; So2, Oxygen saturation.

Table 2 Correlation Analysis Between ICU Transfer Status and Laboratory Parameters

	Univariate Model			Multivariate Model		
	OR	%95 GA	P value	OR	%95 GA	P value
Age	1.032	1.026–1.038	0.000	1.021	1.014–1.028	0.000
Gender	1.232	1.039–1.462	0.017			
White blood cell	1.020	1.012–1.029	0.000			
Hemoglobin	0.959	0.928–0.991	0.012			
Platelet	0.997	0.997–0.998	0.000	0.998	0.997–0.999	0.000
Neutrophil	1.052	1.038–1.066	0.000	1.019	1.007–1.031	0.001
Lymphocyte	0.694	0.614–0.785	0.000			
Hs-CRP	1.005	1.005-1.006	0.000	1.003	1.002-1.004	0.000
Albumin	0.886	0.874-0.899	0.000	0.903	0.888-0.918	0.000
pCO ₂	0.986	0.976-0.995	0.003	1.010	1.000-1.020	0.039
pO ₂	1.007	1.004-1.009	0.000			
Lactate	1.553	1.440-1.675	0.000	1.426	1.308-1.555	0.000
Base Deficit	0.969	0.957-0.982	0.000			
Bicarbonate	0.965	0.951-0.980	0.000			
sO ₂ (%)	1.008	1.003-1.013	0.001	1.010	1.005-1.015	0.000
Length of stay	1.013	1.003-1.023	0.009			

Note: Statistically significant variables ($p < 0.05$).

Abbreviations: CRP, C-reactive protein; Pco2, The partial pressure of carbon dioxide; po2, The partial pressure of oxygen; HCO₃, Bicarbonate; So2, Oxygen saturation.

WBC, neutrophil count, CRP, uric acid levels were significantly higher in the nonsurvivor group compared to the survivor group ($p < 0.05$). Conversely, HGB, PLT, lymphocyte count and albumin levels were significantly lower in the nonsurvivor group. However, $p\text{CO}_2$, base excess and bicarbonate levels were significantly lower, and lactate levels were significantly higher in the nonsurvivor group (Table 3).

The multivariate analysis demonstrated that age, neutrophil count, CRP uric acid, albumin, and lactate levels were independently and significantly associated with mortality, highlighting their potential utility as predictors in clinical mortality risk assessment (Table 4).

For Figure 2, in the ROC analysis of the PCO_2 variable for mortality prediction, the following were found: The area under the curve (AUC) was 0.571 (95% CI=0.545–0.598, $p < 0.000$). The optimal cut-off value was determined as 38.3 mmHg. The sensitivity of this value was calculated as 53%, and its specificity was 69.7%.

For Figure 3, in the ROC analysis of the Lactate variable for mortality prediction, the following were found: The area under the curve (AUC) was 0.621 (95% CI=0.595–0.646, $p < 0.000$). The optimal cut-off value was determined as 1.87 mmol/L. The sensitivity of this value was calculated as 55%, and its specificity was 63.1%.

Discussion

In this large cohort of internal medicine patients, our analysis demonstrates that arterial blood gas parameters, in conjunction with routinely measured laboratory values, possess significant prognostic utility for predicting both intensive care unit (ICU) transfer and in-hospital mortality. Specifically, lower partial pressure of carbon dioxide ($p\text{CO}_2$), decreased oxygen saturation, and elevated lactate concentrations were independently associated with adverse clinical outcomes. These findings align with established literature positing that disturbances in acid-base homeostasis and tissue hypoperfusion, indicated by rising lactate levels, act as critical early warning signs of clinical deterioration. Furthermore, the persistence of $p\text{CO}_2$, lactate, and oxygen saturation as significant predictors in multivariate analysis underscores their role as independent prognostic indicators beyond conventional inflammatory and biochemical markers. However, the

Table 3 Baseline Characteristics of Patients According to the Mortal Status

	Survivals (n:8889)		Non-Survivals (n:168)		P value
	Mean \pm SD (n)%	Median	Mean \pm SD (n)%	Median	
Age (year)	63.3 \pm 18.1	67.0	75.1 \pm 14.3	77.0	0.000
Gender: n (%)	Female	4544(51.4%)	74(44.0%)		0.069
	Male	4345(49.2%)	94(56.0%)		
White Blood Cell Count. $\times 10^9$ /L	9.1 \pm 7.6	7.9	12.2 \pm 11.0	9.3	0.000
Hemoglobin (g/dL)	10.4 \pm 2.6	10.6	10.0 \pm 2.2	9.8	0.003
Platelet. $\times 10^9$ /L	224.1 \pm 107.0	213.0	203.0 \pm 114.1	195.0	0.001
Neutrophil, $\times 10^9$ /L	6.9 \pm 5.6	5.6	10.1 \pm 9.3	7.8	0.000
Lymphocyte, $\times 10^9$ /L	1.44 \pm 2.14	1.20	1.24 \pm 3.48	0.71	0.000
Hs-CRP (mg/L)	67.4 \pm 81.8	32.3	119.1 \pm 89.2	99.1	0.000
Albumin (g/dL)	34.6 \pm 5.6	35.0	28.3 \pm 6.0	27.0	0.000
pH (arterial)	7.39 \pm 0.07	7.39	7.37 \pm 0.10	7.38	0.171 ^m
$p\text{CO}_2$ (mmHg)	42.7 \pm 9.5	41.9	39.8 \pm 11.2	38.4	0.000 ^m
$p\text{O}_2$ (mmHg)	49.8 \pm 26.4	43.2	56.4 \pm 34.4	43.8	0.078 ^m
Lactate (mmol/L)	1.81 \pm 0.81	1.65	2.47 \pm 1.56	2.15	0.000 ^m
Base deficit (mmol/L)	0.16 \pm 6.64	0.30	-2.78 \pm 6.93	-2.50	0.000 ^m
Bicarbonate (mmol/L)	25.2 \pm 6.0	25.2	22.5 \pm 6.1	22.5	0.000 ^m
sO_2 (%)	71.4 \pm 18.8	74.2	72.2 \pm 19.8	75.2	0.460 ^m

Note: Statistically significant variables ($p < 0.05$).

Abbreviations: ALT, Alanine aminotransferase; CRP, C-reactive protein ; Ph, potential of hydrogen; Pco2, The partial pressure of carbon dioxide; po2, The partial pressure of oxygen; So2, Oxygen saturation.

Table 4 Correlation Analysis Between Survival Status and Laboratory Parameters

	Univariate Model			Multivariate Model		
	OR	%95 GA	P value	OR	%95 GA	P value
Age	1.050	1.038–1.062	0.000	1.043	1.029–1.057	0.000
White blood cell	1.016	1.008–1.025	0.000			
Hemoglobin	0.932	0.879–0.988	0.018			
Platelet	0.998	0.996–1.000	0.010			
Neutrophil	1.044	1.023–1.066	0.000	1.021	1.006–1.037	0.007
Lymphocyte	0.842	0.690–1.027	0.090			
Hs-CRP	1.005	1.004-1.007	0.000	1.002	1.000-1.004	0.033
Uric acid	1.213	1.165-1.263	0.000	1.138	1.088-1.191	0.000
Albumin	0.848	0.828-0.869	0.000	0.854	0.830-0.878	0.000
pCO ₂	0.963	0.946-0.981	0.000			
Lactate	1.587	1.434-1.757	0.000	1.428	1.262-1.616	0.000
Base Deficit	0.935	0.914-0.956	0.000			
Bicarbonate	0.924	0.901-0.949	0.000			

Note: Statistically significant variables ($p < 0.05$).

Abbreviations: CRP, C-reactive protein; Pco2, The partial pressure of carbon dioxide.

interpretation of arterial blood gas parameters requires careful consideration of the underlying clinical context, as similar abnormalities may reflect distinct pathophysiological processes with different therapeutic and prognostic implications.

Oxygen saturation, pCO₂ and lactate demonstrated discriminative ability for ICU admission. These parameters may assist in identifying patients at lower risk for ICU transfer when interpreted alongside clinical judgment, rather than serving as standalone decision tools. Paradoxical finding was that patients subsequently admitted to the ICU exhibited comparable or even higher mean oxygenation parameters. The observation of elevated oxygen saturation in patients subsequently transferred to the ICU may reflect the administration of high-concentration oxygen therapy prior to transfer and the associated pathophysiological consequences of high concentration oxygen therapy. It is known that high-concentration oxygen therapy can induce oxidative stress, leading to cellular damage, heightened inflammatory

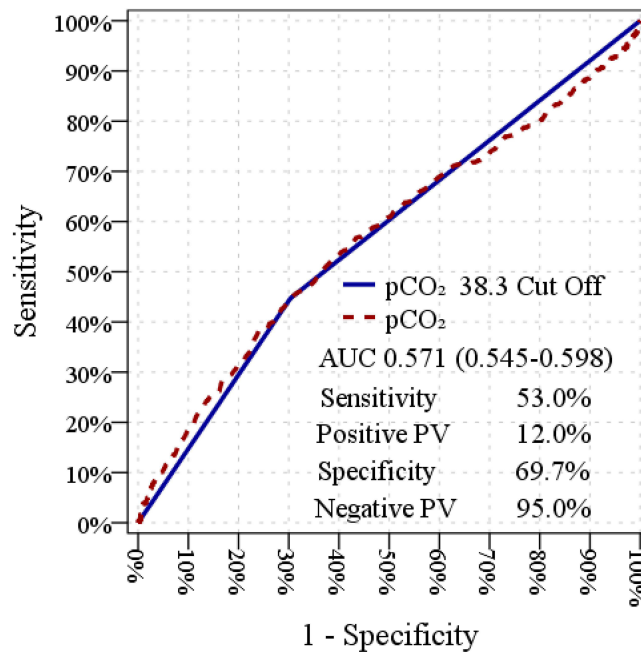


Figure 2 Curve for the prediction of mortality by PCO₂. The ROC curve for PCO₂ in estimating mortality was constructed, and the area under the curve of PCO₂ 0.571 (95% CI=0.545 to 0.598, P<0.000) was found. The cut-off values of PCO₂ was 38,3 (mmHg) with sensitivity of 53% and specificity of 69.7%.

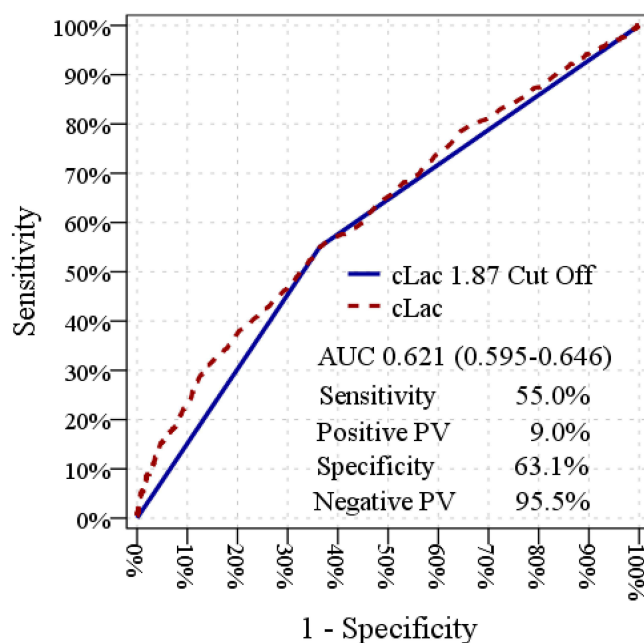


Figure 3 Curve for the prediction of mortality by Lactate. The ROC curve for lactate in estimating mortality was constructed, and the area under the curve of lactate 0.621 (95% CI=0.595 to 0.5646, $P<0.000$) was found. The cut-off values of lactate was 1.87 (mmol/L) with sensitivity of 55% and specificity of 63.1%.

responses, and mitochondrial dysfunction.^{9,10} These processes can initiate a vicious cycle of tissue injury through the excessive production of reactive oxygen species and activation of immune pathways.^{11,12} Prolonged oxygen exposure may also compromise pulmonary gas exchange by promoting absorptive atelectasis and ventilation-perfusion mismatch.^{13,14} Collectively, these mechanisms substantiate the hypothesis that elevated oxygen saturation may be a marker of, or contributor to, hyperoxia-induced lung injury. Accordingly, elevated oxygen saturation in ICU transferring patients should not necessarily be interpreted as clinical stability but may instead reflect aggressive oxygen therapy and potential hyperoxia-related pathophysiological effects. This distinction is clinically relevant, as hyperoxia has been associated with oxidative stress, inflammatory activation, and impaired pulmonary gas exchange.

Patients in internal medicine wards typically present with numerous comorbidities, which complicates the interpretation of hypocapnia. The development of low $p\text{CO}_2$ may be attributed to a multitude of factors. The prognostic significance of hypocapnia may vary substantially depending on underlying comorbid conditions. In patients with chronic obstructive pulmonary disease, alterations in $p\text{CO}_2$ may reflect baseline respiratory physiology, whereas in patients with renal failure or sepsis, hypocapnia often represents a compensatory response to metabolic acidosis. These distinctions underscore that blood gas abnormalities should be interpreted in conjunction with clinical diagnoses rather than as isolated markers. Acute conditions such as pulmonary embolism, pneumothorax, pneumonia, and asthma can induce tachypnea, leading to reduced arterial carbon dioxide levels. Additionally, systemic conditions including infections, vascular diseases, and chronic kidney failure can impair tissue perfusion, resulting in metabolic acidosis. This acidosis may then trigger hypocapnia as a compensatory respiratory mechanism. Several studies have documented that altered PaCO_2 levels are common in acute heart failure, with hypocapnia being associated with increased in-hospital and one-year mortality.^{15,16} Notably, PaCO_2 levels below 32.3 mmHg have been correlated with the highest in-hospital mortality rates.¹⁷ These observations are consistent with data from patients with cardiac-related respiratory failure, in whom hypocapnia similarly portends a poorer prognosis.

Consistent with previous research, lactate levels were independently associated with ICU transfer and mortality. This indicates that lactate levels, particularly when within a normal range, can be valuable for ruling out impending clinical deterioration. Previous studies have established a strong association between hyperlactatemia and increased mortality in patients with severe sepsis and septic shock presenting to the emergency department.¹⁸ Further supporting this, a lactate level exceeding 2.5 mmol/L upon admission has been linked to a significantly higher 28-day mortality in patients with

severe sepsis and septic shock (16.5% vs 5.8%, $p < 0.001$), suggesting this cutoff value holds predictive power for mortality risk.¹⁹ Moreover, In the study conducted by Ozaydin et al, elevated initial lactate levels were associated with increased 28-day mortality.²⁰ Our study, in line with previous studies, demonstrated that lactate levels are associated with both mortality and the requirement for intensive care.

This study has several limitations that must be acknowledged. Its retrospective and single-center design may limit the generalizability of the findings. As patients hospitalized in internal medicine wards often have complex comorbid disease profiles, variations in disease severity, concomitant conditions, and differences in treatment regimens during hospitalization may have acted as confounding factors influencing the outcomes. Furthermore, admission to the intensive care unit cannot be determined solely by physiological severity. Institutional protocols, physician assessment, and bed availability can also influence decisions regarding transfer to the intensive care unit. This can be considered a limitation of the study. Although the substantial sample size bolsters the reliability of our results, external validation through prospective, multicenter studies is warranted to confirm their broader applicability.

Conclusion

In this large cohort of internal medicine patients, arterial blood gas parameters, particularly lactate and $p\text{CO}_2$, were associated with ICU transfer and in-hospital mortality. While these findings highlight the potential clinical relevance of blood gas abnormalities, prospective studies are needed to determine whether these parameters can contribute meaningfully to future risk assessment strategies.

Data Sharing Statement

Due to data privacy and ethical restrictions, the private dataset is not publicly available, but data sharing may be permitted upon request. The data are available from the corresponding author upon reasonable request.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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