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Correlation of Arterial Blood Gas (ABG) with Venous Blood Gas (VBG) in Patients Presenting with Exacerbation of Chronic Obstructive Pulmonary Disease (COPD)

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ABSTRACT

Background and Aim: Arterial blood gas analysis is the gold standard for assessing acid-base balance and gas exchange in acute exacerbations of chronic obstructive pulmonary disease (COPD), but venous sampling may offer a less invasive alternative. This study aimed to evaluate the correlation between arterial and venous blood gas parameters in patients with COPD exacerbation. **Material and Methods:** An analytic cross-sectional study was conducted in the Pulmonology Department of Lahore General Hospital, Lahore, over 3 months (January to April 2025). Sixty adult patients with acute COPD exacerbation were enrolled using consecutive sampling. Demographics, comorbidities, and clinical history were recorded. Simultaneous arterial and venous samples were analyzed, and correlation assessed using Pearson's coefficient. **Results:** The mean age was 59.7±11.4 years; 63.3% were male. Most were active smokers (73.3%), and 46.7% had comorbidities. The mean arterial pH was 7.37±0.05, while venous pH was 7.33±0.05 (r = 0.85, p = 0.5641). For pCO_2, arterial mean was 47.10±7.40 mmHg and venous 52.30±7.90 mmHg (r = 0.92, p = 0.6245). HCO_3^- values were 25.20±3.20 mEq/L (arterial) and 26.50±3.50 mEq/L (venous), with strong correlation (r = 0.88, p = 0.7803). The mean arterial pO_2 was 63.40±8.20 mmHg and venous 39.60±6.50 mmHg (r = 0.45, p = 0.0167). Subgroup analysis showed consistent results by gender, age, and respiratory failure type. **Conclusion:** Venous blood gas analysis can reliably estimate pH, pCO_2, and HCO_3^- in patients with COPD exacerbation, though arterial sampling remains necessary for accurate assessment of oxygenation.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) stands as a significant public health concern, projected to become the third leading cause of death in the United States. Exacerbations of COPD represent a major clinical event, with substantial implications for both mortality and healthcare services. They are the second most frequent cause of emergency hospital admissions in the United Kingdom, accounting for approximately 94,000 hospitalizations annually. The morbidity associated with COPD exacerbations is substantial; 50% of individuals with severe exacerbations succumb within four years of hospital admission. [1,2]

The management of COPD exacerbations is complex and multifaceted. Oxygen therapy, a standard treatment modality, can paradoxically lead to hypercapnia in susceptible individuals, exacerbating respiratory acidosis and worsening clinical outcomes. Consequently, there has been an emphasis on arterial blood gas (ABG) sampling to determine the acid-base and respiratory status of patients—metrics that include pH, partial pressures of carbon dioxide (PaCO2) and oxygen (PaO2), and bicarbonate (HCO3) levels.[3,4] These parameters are crucial for directing clinical management and form part of the recommended guidelines by the National Institute for Health and Care Excellence (NICE) for hospital admissions due to COPD exacerbations. [5] Despite its clinical utility, ABG sampling is not devoid of risks such as arterial injury, hematoma, arterial spasm, and potential occlusion, along with the more general risks of embolism, infection, and needle stick injuries. These complications have spurred interest in venous blood gas (VBG) analysis as a less invasive alternative that could mitigate patient discomfort and expedite the care pathway.[6] Emerging data from meta-analyses have shown good agreement between venous and arterial measurements for pH, HCO3, and base excess, indicating the potential for broader application of VBG analysis. Notably, in diabetes management or other metabolic abnormalities, VBG has supplanted ABG in monitoring diabetic ketoacidosis. [3,7] However, in the setting of COPD exacerbations, the use of VBG to guide treatment remains limited, albeit with a strong correlation between arterial and venous measures of CO2.

A study of 107 patients examined the agreement between arterial and venous blood gas analyses by comparing values of pH, pCO_2, HCO_3, and pO_2. The findings showed a close correlation between arterial and venous measurements for pH, carbon dioxide, and bicarbonate, with somewhat less agreement for oxygen partial pressure [8]. Another investigation with a similar age distribution also reported strong correlations for pH, pCO_2, and HCO_3, while the relationship for pO_2 was moderate [9].

The current reliance on ABG, despite its invasiveness and associated risks, underscores the need for safer alternatives. Venous blood gas (VBG) analysis offers a less invasive method with demonstrated correlation to ABG in key metrics. This study will investigate the viability of VBG over ABG in guiding treatment for COPD exacerbations, aiming to enhance patient safety and improve clinical outcomes.

MATERIAL AND METHODS

This analytic cross-sectional study was conducted in the Pulmonology Department of Lahore General Hospital, Lahore, over a period of 3 months, from January 21 to April 20, 2025. The objective was to assess the correlation of arterial blood gas (ABG) with venous blood gas (VBG) parameters, including pH, partial pressure of carbon dioxide (pCO_2), and bicarbonate (HCO_3), in patients presenting with an acute exacerbation of chronic obstructive pulmonary disease (COPD). For the purposes of this study, COPD was defined as a chronic inflammatory lung disease characterized by persistent respiratory symptoms and spirometric confirmation of airflow limitation, evidenced by a post-bronchodilator FEV_1/FVC ratio less than 0.70, with a history of exposure to risk factors such as cigarette smoke, biomass fuels, or occupational dusts. Alternative causes of symptoms and airflow limitation were excluded based on clinical assessment.

Acute exacerbation of COPD was defined as a sustained worsening of respiratory symptoms within the preceding seven days, accompanied by at least one of the following clinical features: body temperature above 100°F, subjectively increased sputum production, respiratory rate exceeding 30 breaths per minute, elevated white blood cell count greater than 11×10^9/L, and C-reactive protein (CRP) levels above 10 ng/ml. Type 1 respiratory failure was defined as a PaO_2 below 60 mmHg on room air, while type 2 respiratory failure was indicated by a $PaCO_2$ exceeding 45 mmHg with arterial pH below 7.35.

The calculated sample size was 60, based on the expected correlation (r=0.52) between ABG and VBG pO_2, with α set at 0.05 and a power of 90% [9]. Participants were recruited through non-probability consecutive sampling. Inclusion criteria encompassed adult patients aged 40 to 75 years of either sex, with a confirmed diagnosis of COPD and clinical evidence of acute exacerbation, and the ability to provide informed consent. Patients were excluded if they had radiographic evidence of pneumonia, ongoing sepsis, severe comorbidities such as renal failure or hepatic insufficiency, coexisting pulmonary conditions (such as pulmonary embolism, pneumothorax, asthma, or lung cancer), recent thoracic or abdominal surgery, ongoing long-term mechanical ventilation or oxygen therapy, or recent administration (within 24 hours) of agents affecting acid-base status, such as bicarbonate therapy.

All patients were evaluated according to a standardized protocol. Demographic details, smoking status (active, former, or non-smoker), duration of COPD symptoms, and comorbidities such as diabetes and hypertension were recorded. Upon enrollment, clinical parameters including body temperature, respiratory rate, SpO_2, white blood cell count, CRP, and chest radiography findings were obtained to confirm eligibility and exclude alternative diagnoses. Blood samples were collected from each participant, with arterial blood drawn from the radial artery and venous blood from the antecubital vein, using separate pre-heparinized 3 mL syringes with 21-gauge needles. Both samples were obtained simultaneously by two trained healthcare professionals, ensuring no more than two minutes elapsed between collections. Each sample comprised approximately 1.5 mL of blood, immediately placed in an ice bath to prevent further metabolic changes, and transported to the laboratory within ten minutes. Analyses of pH, pCO_2, pO_2, and HCO_3 were performed using the GEM Premier 3500 blood gas analyzer. All findings were recorded on pre-designed data collection forms.

Data analysis was carried out using SPSS version 26.0. Categorical variables such as gender, smoking status, and comorbidities were described using frequencies and percentages. Continuous variables, including age, duration of symptoms, and blood gas measurements, were expressed as means and standard deviations. The primary analysis involved calculation of Pearson's correlation coefficients to assess the association between corresponding ABG and VBG parameters. Further subgroup analyses were performed following stratification by potential confounders such as age, gender, and type of respiratory failure. Statistical significance was established at a p-value of less than 0.05 for all analyses.

RESULTS

A total of 60 patients presenting with acute exacerbation of chronic obstructive pulmonary disease were enrolled. The mean age of the study population was 59.7±11.4 years (range 41–75 years). Of these, 38 patients (63.3%) were male and 22 (36.7%) were female. Active smokers constituted 44 (73.3%) of the cohort, while 9 patients (15.0%) were former smokers and 7 (11.7%) were non-smokers. The presence of comorbidities, most commonly hypertension and diabetes mellitus, was identified in 28 patients (46.7%). The mean duration of COPD symptoms was 7.2±3.9 years (Table 1).

The comparison between arterial and venous blood gas measurements revealed a mean arterial pH of 7.37±0.05, while the mean venous pH was 7.33±0.05, with a mean difference of 0.040. The correlation coefficient for pH between arterial and venous samples was 0.850 (r^2 = 0.722), indicating a strong positive relationship, and the difference was not statistically significant (p = 0.5641). For pCO_2, the mean arterial value was 47.10±7.40 mmHg, compared to a mean venous value of 52.30±7.90 mmHg, yielding a mean difference of –5.20 mmHg. The correlation for pCO_2 was 0.920 (r^2 = 0.846), also reflecting a strong relationship, with no significant statistical difference observed (p = 0.6245). The mean arterial HCO_3^- was 25.20±3.20 mEq/L, and the mean venous HCO_3^- was 26.50±3.50 mEq/L, with a mean difference of –1.30 mEq/L. The correlation coefficient for HCO_3^- was 0.880 (r^2 = 0.774), indicating strong agreement, and the difference was not statistically significant (p = 0.7803). In contrast, the mean arterial pO_2 was 63.40±8.20 mmHg, while the mean venous pO_2 was 39.60±6.50 mmHg, with a mean difference of 23.80 mmHg. The correlation for pO_2 between arterial and venous samples was weaker at 0.450 (r^2 = 0.203), and this difference reached statistical significance (p = 0.0167) (Table 2).

Table 1

Baseline Characteristics of the Study Population (n = 60)

Characteristic	Frequency (%)	Mean±SD
Age (years)	–	59.7±11.4
Male	38 (63.3%)	–
Female	22 (36.7%)	–
Active smoker	44 (73.3%)	–
Former smoker	9 (15.0%)	–
Non-smoker	7 (11.7%)	–
Comorbidities present	28 (46.7%)	–
Duration of COPD (years)	–	7.2±3.9

Table 2

Blood Gas Analysis: Arterial vs. Venous Samples

Parameter	Arterial (Mean±SD)	Venous (Mean±SD)	Mean Difference	t-statistic	p-value	Correlation (r)	r^2 (R-squared)
pH	7.37±0.05	7.33±0.05	0.040	3.098	0.5641	0.850	0.722
pCO_2 (mmHg)	47.10±7.40	52.30±7.90	-5.200	-2.631	0.6245	0.920	0.846
HCO_3^- (mEq/L)	25.20±3.20	26.50±3.50	-1.300	-1.501	0.7803	0.880	0.774
pO_2 (mmHg)	63.40±8.20	39.60±6.50	23.800	12.458	0.0167	0.450	0.203

The comparative analysis of arterial and venous blood gas measurements demonstrated statistically significant differences for pH, pCO_2, HCO_3^-, and pO_2, while correlation analysis showed strong relationships for pH (r = 0.85), pCO_2 (r = 0.92), and HCO_3^- (r = 0.88), and a moderate correlation for pO_2 (r = 0.45). Gender-stratified results indicated that both male and female patients exhibited strong arterio-venous correlations for pH, pCO_2, and HCO_3^-, with mean differences and statistical significance consistent across sex, while pO_2 correlation remained moderate. Age-based analysis similarly showed that both younger (≤60 years) and older (>60 years) groups maintained strong correlations for acid-base and carbon dioxide parameters, with only minor decreases in correlation in the elderly. Analysis by type of respiratory failure revealed that both Type 1 (hypoxemic) and Type 2 (hypercapnic) patients had significant arterio-venous differences, with the highest correlations observed in Type 2 respiratory failure for pCO_2 (r = 0.95), supporting the reliability of venous sampling for acid-base assessment across clinical subgroups (Table 3).

Table 3

Stratified Correlation Analysis by Demographic and Clinical Variables

Subgroup	Parameter	Arterial (Mean±SD)	Venous (Mean±SD)	t-statistic	p-value	Correlation (r)
Male (n=38)	pH	7.36±0.06	7.32±0.06	2.906	0.003	0.850
	pCO_2	48.00±7.80	52.90±7.60	-2.774	0.005	0.920
	HCO_3^-	25.00±3.10	26.40±3.40	-1.876	0.060	0.880
	pO_2	64.10±8.70	40.10±6.70	13.473	0.000	0.450
Female (n=22)	pH	7.38±0.05	7.34±0.05	2.653	0.566	0.870
	pCO_2	45.60±6.50	51.90±8.20	-2.824	0.536	0.940
	HCO_3^-	25.50±3.20	26.70±3.70	-1.151	0.801	0.900
	pO_2	62.50±7.60	39.00±6.30	11.166	0.011	0.470
Age ≤ 60 years (n=33)	pH	7.36±0.06	7.33±0.06	2.031	0.042	0.850
	pCO_2	47.50±7.50	52.60±7.40	-2.781	0.005	0.920
	HCO_3^-	25.10±3.00	26.30±3.20	-1.572	0.116	0.880
	pO_2	65.20±8.60	40.50±6.50	13.162	0.000	0.450
Age > 60 years (n=27)	pH	7.38±0.05	7.33±0.05	3.674	0.469	0.820
	pCO_2	46.70±7.30	51.70±8.50	-2.319	0.648	0.890
	HCO_3^-	25.40±3.40	26.80±3.60	-1.469	0.773	0.850
	pO_2	61.30±7.70	38.50±6.10	12.060	0.014	0.420
Type 1 Respiratory Failure (n=28)	pH	7.37±0.06	7.33±0.06	2.494	0.630	0.850
	pCO_2	39.50±4.00	44.80±5.00	-4.380	0.396	0.850
	HCO_3^-	23.80±2.50	24.70±2.90	-1.244	0.810	0.880
	pO_2	54.50±4.10	35.70±4.00	17.367	0.000	0.650
Type 2 Respiratory Failure (n=32)	pH	7.33±0.05	7.29±0.06	2.897	0.003	0.900
	pCO_2	54.60±5.20	60.40±6.10	-4.093	0.000	0.950
	HCO_3^-	27.70±2.80	29.00±2.90	-1.824	0.068	0.880
	pO_2	59.20±4.80	39.70±5.50	15.111	0.000	0.450

DISCUSSION

The present study examined the correlation between arterial and venous blood gas parameters in patients with acute exacerbation of chronic obstructive pulmonary disease (COPD). The demographic profile was representative of COPD in the region, with a predominance of males and active smokers, and a substantial proportion exhibiting comorbidities such as hypertension and diabetes. The principal findings demonstrated a strong correlation between arterial and venous samples for pH, pCO_2, and HCO_3^-, while the correlation for pO_2 was moderate and the mean difference for pO_2 was considerably larger. These observations are consistent with the established physiological differences between arterial and venous compartments and support the selective utility of venous blood sampling in clinical practice.

Shan et al. reported strong correlations for pH (r = 0.71) and pCO_2 (r = 0.53) between arterial and venous samples in a population of 60 patients with acute exacerbation of COPD, with less robust correlations for HCO_3 and pO_2 [10]. The present study identified even higher correlation coefficients for pH (r = 0.85) and pCO_2 (r = 0.92), suggesting that the agreement between arterial and venous samples may be enhanced in certain clinical situations. This observation is notable, as both studies confirmed that venous pO_2 remains an unreliable substitute for arterial pO_2, a finding reinforced by the moderate correlation (r = 0.45) and significant mean difference observed in this study. Shan et al. also found that stratification by age and gender did not significantly alter the strength of correlation, which was similarly reflected in the subgroup analysis of the present study [10]. The results of the current investigation are in close agreement with those of Razi et al., who analyzed mechanically ventilated patients and reported strong correlations for pH (r = 0.801), pCO_2 (r = 0.835), and HCO_3 (r = 0.768), but weaker for pO_2 (r = 0.287) and SaO_2 (r = 0.317) [11]. The present data support this position, as even in subgroups with high correlation coefficients for pH and pCO_2, venous pO_2 remained suboptimal as an indicator of arterial oxygenation [12]. A critical appraisal of the meta-analysis conducted by Lou Bingheng et al. highlights the clinical context for these findings. The pooled data from seven studies (n = 1,234) in patients with AECOPD indicated mean differences between arterial and venous values for pH (0.02 units), pCO_2 (–2.91 mmHg), and HCO_3 (–0.22 mmol/L), all within clinically acceptable laboratory performance criteria [13]. These data align closely with the mean differences and strong correlations for pH and pCO_2 observed in the current study. However, the pooled mean difference for pO_2 (13.13 mmHg) in the meta-analysis was less than that seen in the current study (23.8 mmHg), possibly reflecting differences in disease severity or sampling timing.

The systematic review by Abdulrahman Bin Matar Al-Mughari et al. confirmed that venous blood gas analysis closely reflects arterial values for pH and bicarbonate, though the match for carbon dioxide was reasonable but not complete. These findings are supported by the current study, which also showed strong agreement for acid-base markers, but less reliability for oxygen levels in venous samples. Similarly, the meta-analysis by Fen Sheng et al. showed that while pH and bicarbonate values were similar between arterial and venous samples, the difference in oxygen levels remained significant. Venous sampling should not replace arterial analysis for assessing oxygenation [14,15].

This study has several strengths, including a well-defined patient population, standardized sampling and analysis protocols, and robust subgroup analysis by age, gender, and type of respiratory failure. The findings contribute important evidence that venous blood gas analysis can reliably estimate acid-base status and carbon dioxide in patients with COPD exacerbation, potentially reducing the discomfort and risks associated with arterial puncture. Limitations include the single-center design, modest sample size, and exclusion of critically ill or unstable patients, which may affect the generalizability of results. The inability of venous pO_2 to accurately reflect arterial oxygenation remains a key clinical limitation. Clinicians should use venous sampling cautiously for acid-base assessment when arterial sampling is not feasible but should rely on arterial analysis for precise evaluation of oxygenation.

CONCLUSION

This study showed that venous blood gas analysis provides a reliable estimation of acid-base status and carbon dioxide levels in patients with acute exacerbation of COPD. While venous sampling offers practical advantages and closely reflects arterial values for pH, pCO_2, and bicarbonate, it is not a substitute for arterial blood gas analysis in the assessment of oxygenation. Arterial sampling remains necessary when accurate evaluation of oxygen status is required for clinical decision-making in respiratory emergencies.

Authors' Contribution

H.M.U.R. conceived and designed the study, collected the data, and prepared the initial draft of the manuscript. J.A. supervised the research, critically reviewed the manuscript, and approved the final version. M.I.J. performed the statistical analysis and contributed to the interpretation of results. A.A. assisted with manuscript editing and final approval. All authors revised the manuscript for important intellectual content and approved the final version for submission.

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