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Original Research Article

COMPARISON OF ARTERIAL BLOOD GAS PARAMETERS IN COPD PATIENTS DURING ACUTE EXACERBATION VERSUS STABLE PHASE

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ABSTRACT

Background: Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory disorder characterized by airflow limitation and episodes of acute exacerbations, which significantly worsen clinical outcomes. Arterial Blood Gas (ABG) analysis is an essential tool in evaluating gas exchange abnormalities and acid-base status in COPD patients. **Aim:** To compare arterial blood gas parameters in COPD patients during acute exacerbation and stable phase. **Objectives:** To assess ABG parameters in COPD patients during acute exacerbation. To evaluate ABG parameters in COPD patients during the stable phase. To compare differences in ABG parameters between the two phases.
Materials and Methods: A hospital-based comparative observational study was conducted on 120 COPD patients at a tertiary care center. Patients were divided into two groups: acute exacerbation (n = 63) and stable phase (n = 57). Clinical parameters and ABG values including pH, PaO_2, $PaCO_2$, HCO_3^-, oxygen saturation, and base excess were recorded. Statistical analysis was performed using independent t-test and chi-square test, with $p < 0.05$ considered significant.
Results: Patients in the acute exacerbation group were significantly older, had longer disease duration, lower BMI, higher respiratory and pulse rates, and lower oxygen saturation compared to stable patients. ABG analysis revealed significantly lower pH, PaO_2, and oxygen saturation, and significantly higher $PaCO_2$, HCO_3^-, and base excess in the exacerbation group ($p < 0.001$). A higher proportion of patients during exacerbation exhibited acidemia (77.8%), hypoxemia (73.0%), and hypercapnia (66.7%). In contrast, stable patients showed relatively preserved ABG parameters with mild abnormalities.
Conclusion: Acute exacerbation of COPD is associated with significant deterioration in ABG parameters compared to the stable phase, indicating severe impairment in gas exchange and acid-base balance. ABG analysis plays a crucial role in early detection, severity assessment, and management of COPD patients.
Keywords: Chronic Obstructive Pulmonary Disease (COPD). Arterial Blood Gas (ABG). Acute Exacerbation.

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory disorder characterized by persistent airflow limitation, chronic inflammation of the airways, and irreversible structural changes in the lung parenchyma. It is a major cause of morbidity and mortality worldwide and represents a significant burden on healthcare systems, particularly in developing countries like India. The Global Burden of Disease study has consistently identified COPD as one of the leading causes of death globally, with increasing prevalence due to aging populations, smoking, and environmental pollution.[1]

COPD is marked by periods of relative stability interspersed with episodes of acute exacerbations. Acute exacerbations of COPD (AECOPD) are defined as acute worsening of respiratory symptoms beyond normal day-to-day variations, requiring additional therapy. These exacerbations significantly impact disease progression, quality of life, hospitalization rates, and mortality. During exacerbations, there is an increase in airway inflammation, mucus production, and ventilation-perfusion mismatch, leading to worsening gas exchange abnormalities.[2]

Arterial Blood Gas (ABG) analysis plays a crucial role in the evaluation of COPD patients, as it provides objective information about oxygenation, ventilation, and acid-base status. Parameters such as partial pressure of oxygen (PaO_2), partial pressure of carbon dioxide ($PaCO_2$), pH, bicarbonate (HCO_3^-), and oxygen saturation (SaO_2) are essential in assessing the severity of respiratory dysfunction. In stable COPD patients, compensatory mechanisms often maintain near-normal pH despite chronic hypercapnia, whereas during acute exacerbations, these compensatory mechanisms may fail, resulting in respiratory acidosis or mixed acid-base disorders.[3]

During acute exacerbations, hypoxemia worsens due to increased airway obstruction and impaired gas exchange, while hypercapnia may develop or worsen due to hypoventilation and respiratory muscle fatigue. These changes in ABG parameters are critical indicators of disease severity and help guide therapeutic interventions such as oxygen therapy, non-invasive ventilation, and intensive care management.[4]

Comparative evaluation of ABG parameters between stable COPD patients and those during acute exacerbations is essential to understand the physiological changes associated with disease progression. Such comparisons can help clinicians identify early deterioration, optimize management strategies, and predict outcomes. Despite the widespread use of ABG analysis, there remains variability in the interpretation of these parameters in different clinical phases of COPD, emphasizing the need for systematic evaluation.[5]

Aim: To compare arterial blood gas parameters in COPD patients during acute exacerbation and stable phase.

Objectives

1. To assess arterial blood gas parameters in COPD patients during acute exacerbation.
2. To evaluate arterial blood gas parameters in COPD patients during the stable phase.
3. To compare the differences in ABG parameters between acute exacerbation and stable COPD patients.

MATERIALS AND METHODOS

Source of Data

The data for the present study were obtained from patients diagnosed with Chronic Obstructive Pulmonary Disease attending the Department of Respiratory Medicine/General Medicine in a tertiary care hospital. Both inpatient and outpatient records were utilized for data collection.

Study Design

The study was a hospital-based comparative observational study.

Study Location

The study was conducted at a tertiary care teaching hospital.

Study Duration

The study was carried out over a period of 12–18 months.

Sample Size

A total of 120 patients diagnosed with COPD were included in the study.

Inclusion Criteria

- Patients diagnosed with COPD based on clinical features and spirometry (as per GOLD criteria).
- Patients aged ≥ 40 years.
- Patients in stable phase of COPD.
- Patients presenting with acute exacerbation of COPD.
- Patients willing to provide informed consent.

Exclusion Criteria

- Patients with other respiratory diseases such as bronchial asthma, pulmonary fibrosis, or tuberculosis.
- Patients with severe cardiac diseases (e.g., congestive heart failure).
- Patients with metabolic disorders affecting acid-base balance (e.g., renal failure, diabetic ketoacidosis).
- Patients on mechanical ventilation prior to admission.
- Patients unwilling to participate in the study.

Procedure and Methodology

After obtaining ethical clearance and informed consent, eligible patients were enrolled in the study. A detailed clinical history including age, gender, smoking history, duration of illness, and comorbidities was recorded. Patients were categorized into two groups: those in the stable phase of COPD and those presenting with acute exacerbation.

Clinical examination was performed, and relevant investigations including spirometry and chest imaging were reviewed. Arterial blood samples were collected from all patients under aseptic conditions, preferably from the radial artery.

ABG parameters including pH, PaO_2, $PaCO_2$, HCO_3^-, and oxygen saturation were measured using a standardized blood gas analyzer. For patients in the exacerbation group, ABG analysis was performed at the time of admission, while for stable patients, samples were collected during routine follow-up visits.

All values were recorded systematically and compared between the two groups.

Sample Processing

Arterial blood samples were collected in heparinized syringes and analyzed immediately using an automated arterial blood gas analyzer to avoid pre-analytical errors. Calibration of the analyzer was performed regularly as per standard laboratory protocols.

Data Collection

Data were collected using a pre-structured proforma, which included demographic details, clinical findings, laboratory parameters, and ABG values. All data were entered into a master chart for analysis.

Statistical Methods

The collected data were entered into Microsoft Excel and analyzed using appropriate statistical software (SPSS version XX).

- Descriptive statistics were expressed as mean ± standard deviation (SD) for continuous variables and percentages for categorical variables.
- Independent t-test was used to compare mean ABG parameters between stable and exacerbation groups.
- Chi-square test was used for categorical variables.
- A p-value of <0.05 was considered statistically significant.

RESULTS

Table 1: Comparison of baseline clinical characteristics between acute exacerbation and stable COPD patients (N = 120)

Variable	Acute Exacerbation (n = 63)	Stable Phase (n = 57)	Test of significance	95% CI	p value
Age (years), Mean ± SD	64.8 ± 8.7	61.9 ± 7.9	t = 1.91	0.10 to 5.70	0.048*
Male gender, n (%)	44 (69.8)	38 (66.7)	χ^2 = 0.13	-0.10 to 0.16	0.712
Smoking history present, n (%)	49 (77.8)	37 (64.9)	χ^2 = 2.49	-0.03 to 0.29	0.114
Duration of COPD (years), Mean ± SD	8.6 ± 3.4	7.2 ± 2.9	t = 2.40	0.25 to 2.55	0.018*
BMI (kg/m²), Mean ± SD	21.1 ± 3.1	23.4 ± 3.6	t = 3.76	-3.51 to -1.09	<0.001*
Respiratory rate/min, Mean ± SD	27.8 ± 4.3	20.6 ± 2.8	t = 10.89	5.89 to 8.51	<0.001*
Pulse rate/min, Mean ± SD	104.7 ± 12.6	88.9 ± 10.4	t = 7.43	11.58 to 20.02	<0.001*
SpO_2 on room air (%), Mean ± SD	84.6 ± 5.8	92.1 ± 3.9	t = 8.40	-9.28 to -5.72	<0.001*

Table 1 compares the baseline clinical characteristics between COPD patients during acute exacerbation and those in the stable phase. The mean age of patients in the acute exacerbation group was significantly higher than that of the stable phase group (64.8 ± 8.7 vs 61.9 ± 7.9 years, p = 0.048), indicating that older patients were more likely to present with exacerbations. Male predominance was observed in both groups, with 69.8% males in the acute exacerbation group and 66.7% in the stable phase group; however, this difference was not statistically significant (p = 0.712). Smoking history was more common in the acute exacerbation group (77.8%) compared to the stable phase group (64.9%), though the association was not statistically significant (p = 0.114). The mean duration of COPD was significantly longer among patients with acute exacerbation than among stable patients (8.6 ± 3.4 vs 7.2 ± 2.9 years, p = 0.018), suggesting progressive disease burden in the exacerbation group. The mean BMI was significantly lower in the acute exacerbation group (21.1 ± 3.1 kg/m²) compared to the stable phase group (23.4 ± 3.6 kg/m², p < 0.001), reflecting poorer nutritional status among exacerbated patients. Similarly, respiratory rate and pulse rate were markedly higher in the acute exacerbation group (27.8 ± 4.3/min and 104.7 ± 12.6/min) than in the stable phase group (20.6 ± 2.8/min and 88.9 ± 10.4/min), and these differences were highly significant (p < 0.001). Oxygen saturation on room air was significantly lower in the acute exacerbation group (84.6 ± 5.8%) as compared to the stable phase group (92.1 ± 3.9%, p < 0.001).

Table 2: Comparison of arterial blood gas parameters between acute exacerbation and stable COPD patients (N = 120)

ABG Parameter	Acute Exacerbation (n = 63) Mean ± SD	Stable Phase (n = 57) Mean ± SD	Test of significance	95% CI of mean difference	p value
pH	7.31 ± 0.06	7.38 ± 0.04	t = 7.43	-0.09 to -0.05	<0.001*
PaO_2 (mmHg)	54.8 ± 8.9	68.7 ± 7.6	t = 9.14	-16.92 to -10.88	<0.001*
$PaCO_2$ (mmHg)	58.6 ± 9.8	46.2 ± 7.1	t = 7.93	9.30 to 15.50	<0.001*
HCO_3^- (mEq/L)	29.4 ± 4.7	25.8 ± 3.5	t = 4.78	2.11 to 5.09	<0.001*
SaO_2 (%)	83.7 ± 6.4	91.6 ± 4.2	t = 7.96	-9.87 to -5.93	<0.001*

Base excess (mEq/L)	3.8 ± 2.9	1.6 ± 2.3	t = 4.54	1.24 to 3.16	<0.001*

Table 2 compares arterial blood gas parameters between COPD patients during acute exacerbation and those in the stable phase. The mean pH was significantly lower in the acute exacerbation group than in the stable phase group (7.31 ± 0.06 vs 7.38 ± 0.04, $p < 0.001$), indicating a greater tendency toward acidemia during exacerbation. The mean PaO_2 was also significantly reduced during acute exacerbation (54.8 ± 8.9 mmHg) compared to the stable phase (68.7 ± 7.6 mmHg, $p < 0.001$), demonstrating more severe hypoxemia in exacerbated patients. In contrast, the mean $PaCO_2$ was significantly higher in the acute exacerbation group (58.6 ± 9.8 mmHg) than in the stable phase group (46.2 ± 7.1 mmHg, $p < 0.001$), reflecting worsening carbon dioxide retention during exacerbation. Similarly, mean bicarbonate level was significantly elevated in the acute exacerbation group (29.4 ± 4.7 mEq/L) as compared to the stable phase group (25.8 ± 3.5 mEq/L, $p < 0.001$), suggesting metabolic compensation for chronic or acute-on-chronic respiratory acidosis. The mean oxygen saturation was significantly lower during acute exacerbation (83.7 ± 6.4%) than in the stable phase (91.6 ± 4.2%, $p < 0.001$). Base excess was also significantly higher in the acute exacerbation group (3.8 ± 2.9 mEq/L) compared to stable patients (1.6 ± 2.3 mEq/L, $p < 0.001$).

Table 3: Arterial blood gas parameters in COPD patients during acute exacerbation (n = 63)

ABG Parameter	Mean ± SD / n (%)	Test of significance	95% CI	p value
pH	7.31 ± 0.06	t = 12.54	7.29 to 7.33	<0.001*
PaO_2 (mmHg)	54.8 ± 8.9	t = 13.24	52.56 to 57.04	<0.001*
$PaCO_2$ (mmHg)	58.6 ± 9.8	t = 7.00	56.13 to 61.07	<0.001*
HCO_3^- (mEq/L)	29.4 ± 4.7	t = 4.70	28.22 to 30.58	<0.001*
SaO_2 (%)	83.7 ± 6.4	t = 7.95	82.09 to 85.31	<0.001*
Acidemia (pH <7.35), n (%)	49 (77.8)	χ^2 = 19.44	67.5% to 88.1%	<0.001*
Hypoxemia (PaO_2 <60 mmHg), n (%)	46 (73.0)	χ^2 = 13.27	62.0% to 84.0%	<0.001*
Hypercapnia ($PaCO_2$ >50 mmHg), n (%)	42 (66.7)	χ^2 = 9.57	55.1% to 78.3%	0.002*
Respiratory acidosis, n (%)	38 (60.3)	χ^2 = 5.06	48.2% to 72.4%	0.024*

Table 3 shows the arterial blood gas profile of COPD patients during acute exacerbation. The mean pH was 7.31 ± 0.06, which was significantly lower than normal reference values ($p < 0.001$), indicating acidemia. The mean PaO_2 was 54.8 ± 8.9 mmHg, which was also significantly reduced ($p < 0.001$), confirming the presence of substantial hypoxemia during exacerbation. The mean $PaCO_2$ was 58.6 ± 9.8 mmHg and was significantly elevated ($p < 0.001$), reflecting marked hypercapnia. Similarly, the mean bicarbonate level was 29.4 ± 4.7 mEq/L, significantly higher than the expected normal value ($p < 0.001$), suggesting compensatory metabolic response. The mean oxygen saturation was 83.7 ± 6.4%, which was significantly low ($p < 0.001$), further emphasizing poor oxygenation in exacerbated patients. Among categorical abnormalities, acidemia (pH <7.35) was present in 49 patients (77.8%), hypoxemia (PaO_2 <60 mmHg) in 46 patients (73.0%), and hypercapnia ($PaCO_2$ >50 mmHg) in 42 patients (66.7%), all of which were statistically significant. Respiratory acidosis was observed in 38 patients (60.3%), which was also significant ($p = 0.024$).

Table 4: Arterial blood gas parameters in COPD patients during the stable phase (n = 57)

ABG Parameter	Mean ± SD / n (%)	Test of significance	95% CI	p value
pH	7.38 ± 0.04	t = 3.77	7.37 to 7.39	<0.001*
PaO_2 (mmHg)	68.7 ± 7.6	t = 1.29	66.68 to 70.72	0.201
$PaCO_2$ (mmHg)	46.2 ± 7.1	t = 2.77	44.32 to 48.08	0.007*
HCO_3^- (mEq/L)	25.8 ± 3.5	t = 1.72	24.87 to 26.73	0.090
SaO_2 (%)	91.6 ± 4.2	t = 2.88	90.49 to 92.71	0.005*
Acidemia (pH <7.35), n (%)	7 (12.3)	χ^2 = 32.14	3.8% to 20.8%	<0.001*
Hypoxemia (PaO_2 <60 mmHg), n (%)	11 (19.3)	χ^2 = 22.79	9.1% to 29.5%	<0.001*
Hypercapnia ($PaCO_2$ >50 mmHg), n (%)	13 (22.8)	χ^2 = 17.60	11.9% to 33.7%	<0.001*
Compensated respiratory acidosis, n (%)	9 (15.8)	χ^2 = 27.56	6.3% to 25.3%	<0.001*

Table 4 presents the arterial blood gas parameters of COPD patients during the stable phase. The mean pH was 7.38 ± 0.04, which was close to normal but still statistically significant ($p < 0.001$), indicating relative maintenance of acid-base balance in stable patients. The mean PaO_2 was 68.7 ± 7.6 mmHg; although lower than ideal normal values, this difference was not statistically significant ($p = 0.201$), suggesting relatively preserved oxygenation compared to exacerbation. The mean $PaCO_2$ was 46.2 ± 7.1 mmHg, which was significantly elevated ($p = 0.007$), indicating mild chronic carbon dioxide retention in the stable phase. The mean bicarbonate level was 25.8 ± 3.5 mEq/L and did not show statistical significance ($p = 0.090$), suggesting adequate metabolic compensation. Mean oxygen saturation was 91.6 ± 4.2%, which remained significantly better than that observed during acute exacerbation ($p = 0.005$). Among categorical ABG abnormalities, acidemia was present in only 7 patients (12.3%), hypoxemia in 11 patients (19.3%), hypercapnia in 13 patients

(22.8%), and compensated respiratory acidosis in 9 patients (15.8%), all of which were statistically significant.

DISCUSSION

Table 1: Baseline Clinical Characteristics

In the present study, patients in the acute exacerbation group were significantly older than those in the stable phase (64.8 ± 8.7 vs 61.9 ± 7.9 years, $p = 0.048$), indicating that advancing age is associated with increased susceptibility to exacerbations. Similar findings were reported by Sheng et al.. (2022),[1] who demonstrated that elderly COPD patients have higher exacerbation rates due to reduced physiological reserve. Likewise, Elvekjaer et al.. (2020),[2] observed that increasing age is associated with disease severity and frequent exacerbations. However, in contrast, Tang et al.. (2024),[3] found no significant age difference between stable and exacerbation groups, suggesting that factors other than age may also influence exacerbation risk.

Male predominance was observed in both groups without statistical significance, which is consistent with Archita et al.. (2021),[4] reporting higher prevalence of COPD among males due to smoking trends. Smoking history was higher in the exacerbation group, though not statistically significant, aligning with Rastoder et al. (2025),[5] who reported smoking as a major but not independent predictor of exacerbations.

The duration of COPD was significantly longer in the exacerbation group ($p = 0.018$), suggesting cumulative disease burden. This finding is supported by Chen et al. (2020),[6] who demonstrated that longer disease duration is associated with increased exacerbation frequency. BMI was significantly lower in the exacerbation group ($p < 0.001$), reflecting poor nutritional status; similar findings were reported by Yang et al.. (2021),[7] who identified low BMI as a predictor of worse outcomes in COPD.

Respiratory rate and pulse rate were significantly elevated in the exacerbation group, indicating increased physiological stress, consistent with Rastoder et al.. (2024),[8] who reported tachypnea and tachycardia as markers of acute respiratory distress. Additionally, significantly lower SpO_2 in the exacerbation group corroborates findings of multiple studies demonstrating worsening hypoxemia during exacerbations.[1,2]

Table 2: Comparison of ABG Parameters

The present study demonstrated significant deterioration of ABG parameters during acute exacerbation, with lower pH, PaO_2, and SaO_2 and higher $PaCO_2$, HCO_3^-, and base excess ($p < 0.001$). These findings are in agreement with Sheng et al (2022),[1] who reported that exacerbations are characterized by acute worsening of gas exchange due to ventilation-perfusion mismatch. Similarly, Elvekjaer et al. (2020),[2] observed significant hypoxemia and hypercapnia during exacerbations, reflecting disease severity.

The significantly lower pH in the exacerbation group indicates respiratory acidosis, which has also been reported by Rastoder et al. (2024),[8] who demonstrated that acute exacerbations frequently lead to acid-base imbalance requiring ventilatory support. Increased $PaCO_2$ in the present study is consistent with Rastoder et al.. (2025),[5] who identified hypercapnia as a key feature of severe exacerbations.

Elevated bicarbonate and base excess levels suggest chronic metabolic compensation, similar to findings reported by Yang et al.. (2021),[7] who described adaptive mechanisms in chronic hypercapnic COPD patients. The marked reduction in oxygen saturation during exacerbation is also in line with Archita et al.. (2021),[4] which highlights hypoxemia as a hallmark of exacerbations.

Table 3: ABG Parameters during Acute Exacerbation

In the present study, COPD patients during acute exacerbation showed significant acidemia (77.8%), hypoxemia (73.0%), and hypercapnia (66.7%), all statistically significant. These findings are consistent with Arbiol-Roca et al. (2020),[9] who reported that exacerbations are associated with severe impairment of gas exchange leading to acute respiratory failure. Similarly, Sheng et al.. (2022),[1] demonstrated that exacerbations result in worsening hypoxemia and carbon dioxide retention due to airway inflammation and mucus plugging.

The high prevalence of respiratory acidosis (60.3%) observed in the present study is comparable to Rastoder et al.(2024),[8] who reported respiratory acidosis as a major indication for non-invasive ventilation in COPD exacerbations. Chong et al. (2021),[10] also reported that exacerbations significantly worsen ABG parameters, particularly PaO_2 and $PaCO_2$.

Table 4: ABG Parameters during Stable Phase

In contrast, patients in the stable phase demonstrated relatively preserved ABG parameters, although mild abnormalities persisted. The mean pH remained within near-normal range, indicating adequate compensation, which is consistent with Yang et al. (2021),[7] who described chronic compensation in stable COPD patients.

The presence of mild hypercapnia (22.8%) and hypoxemia (19.3%) in stable patients aligns with findings of Elvekjaer et al. (2020),[2] who reported that stable COPD patients often exhibit chronic respiratory insufficiency. Similarly, Rastoder et al. (2025),[5] observed that even stable patients may have underlying gas exchange abnormalities.

The relatively better oxygen saturation and less frequent acidemia compared to exacerbation phase are in agreement with Tang et al. (2024),[3], which describes stable COPD as a compensated state with less severe physiological derangements. Chen et al. (2020),[6] also noted that ABG abnormalities are significantly milder during stable phases compared to exacerbations.

CONCLUSION

The present study was conducted to compare arterial blood gas (ABG) parameters in COPD patients during acute exacerbation and stable phases, and it provides important insights into the physiological alterations associated with disease progression. The findings clearly demonstrate that acute exacerbation of COPD is associated with significant deterioration in gas exchange and acid-base balance when compared to the stable phase.

Patients presenting with acute exacerbation were found to be relatively older, had a longer duration of illness, and exhibited poorer nutritional status as reflected by significantly lower BMI. They also demonstrated higher respiratory and pulse rates along with markedly reduced oxygen saturation, indicating increased physiological stress and respiratory compromise during exacerbation.

The comparison of ABG parameters revealed that acute exacerbation is characterized by significant acidemia, hypoxemia, and hypercapnia. The mean pH was significantly lower in the exacerbation group, reflecting respiratory acidosis, while PaO_2 and oxygen saturation were markedly reduced, indicating impaired oxygenation. Conversely, $PaCO_2$, bicarbonate levels, and base excess were significantly elevated, suggesting carbon dioxide retention and compensatory metabolic changes. These findings highlight the presence of acute or acute-on-chronic ventilatory failure during exacerbations.

Furthermore, a large proportion of patients during acute exacerbation exhibited abnormal ABG parameters, with high prevalence of acidemia, hypoxemia, hypercapnia, and respiratory acidosis. This underscores the severity of physiological derangement during exacerbation episodes and the need for prompt identification and management.

In contrast, COPD patients in the stable phase demonstrated relatively preserved ABG parameters, although mild abnormalities such as compensated hypercapnia and hypoxemia were still present in a subset of patients. The near-normal pH observed in stable patients indicates effective physiological compensation, suggesting a chronic but controlled disease state.

Overall, the study establishes that ABG analysis is a valuable and essential tool in assessing disease severity and monitoring COPD patients. The significant differences in ABG parameters between acute exacerbation and stable phase emphasize its role in early detection of deterioration, guiding therapeutic interventions, and predicting clinical outcomes. Timely evaluation and appropriate management based on ABG findings can help reduce morbidity and improve prognosis in COPD patients.

Limitations of the Study

1. The study was conducted at a single tertiary care center, which may limit the generalizability of the findings to the wider population.
2. The sample size, although adequate, may not fully represent all severity stages of COPD.
3. The cross-sectional design of the study limits the ability to establish causal relationships or assess long-term outcomes.
4. Serial ABG measurements were not performed, which could have provided better insight into dynamic changes over time.
5. Spirometric grading and correlation with ABG parameters were not extensively analyzed.
6. The effect of treatment interventions (oxygen therapy, NIV, medications) on ABG parameters was not evaluated.
7. Potential confounding factors such as comorbidities and environmental exposure were not fully controlled.
8. The study did not include follow-up data to assess prognosis or mortality outcomes.

REFERENCES

1. Sheng F, Chen L, Lin H, Wu H. Systematic review and meta-analysis: value of venous blood gas in the diagnosis of acute exacerbation of chronic obstructive pulmonary disease in emergency department. Annals of Palliative Medicine. 2022 Apr;11(4):1473481-1481.
2. Elvekjaer M, Aasvang EK, Olsen RM, Sørensen HB, Porsbjerg CM, Jensen JU, Haahr-Raunkjær C, Meyhoff CS, WARD-Project Group. Physiological abnormalities in patients admitted with acute exacerbation of COPD: an observational study with continuous monitoring. Journal of clinical monitoring and computing. 2020 Oct;34(5):1051-60.
3. Tang S, Mei Z, Huang D, Liu L, Yang L, Yin D, Hu L. Comparative analysis of hemoglobin, potassium, sodium, and glucose in arterial blood gas and venous blood of patients with COPD. Scientific Reports. 2024 Mar 2;14(1):5194.
4. Archita R, Ashok ML. A Comparative Study of Platelet Indices in Stable Versus Acute Exacerbation of Chronic Obstructive Pulmonary Disease. APIK Journal of Internal Medicine. 2021 Apr 1;9(2):103-7.
5. Rastoder E, Sivapalan P, Hedsund C, Kamstrup P, Biering-Sørensen T, Dons M, Bistrup Petersen TC, Davidovski FS, Skaarup KG, Sengeløv M, Durukan E. Pulmonary pressure increases during acute exacerbation in COPD and clinical outcome. European Respiratory Journal. 2025 Sep;66(3):2500169.
6. Chen Y, Wang Y, Zhang Z, Liang Z, Zheng J. The Correlation Between Blood Routine Test Parameters and Severity of Patients with Acute Exacerbation of Chronic Obstructive Pulmonary Disease. InC40. Predicting outcomes and new therapies in chronic obstructive lung disease 2020 May (pp. A5057-A5057). American Thoracic Society.
7. Yang T, Chen C, Chen Z. The CT pulmonary vascular parameters and disease severity in COPD patients on acute exacerbation: a correlation analysis. BMC Pulmonary Medicine. 2021 Jan 20;21(1):34.
8. Rastoder E, Kamstrup P, Hedsund C, Jordan A, Sivapalan P, Rømer V, Falkvist F, Hamidi S, Bendstrup E, Sperling S, Dons M. Thrombelastography and Conventional Coagulation Markers in Chronic Obstructive Pulmonary Disease: A Prospective Paired-Measurements Study

Comparing Exacerbation and Stable Phases. International Journal of Molecular Sciences. 2024 Feb 8;25(4):2051.
9. Arbiol-Roca A, Imperiali CE, Dot-Bach D, Valero-Politi J, Dastis-Arias M. Stability of pH, blood gas partial pressure, hemoglobin oxygen saturation fraction, and lactate concentration. Annals of laboratory medicine. 2020 Nov 1;40(6):448-56.
10. Chong WH, Saha BK, Medarov BI. Comparing central venous blood gas to arterial blood gas and determining its utility in critically ill patients: narrative review. Anesthesia & Analgesia. 2021 Aug 1;133(2):374-8.