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Original Research Article

COMPARATIVE STUDY OF CLINICAL AND BIOCHEMICAL PROFILES OF PATIENTS WITH ACUTE EXACERBATION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE WITH VERSUS WITHOUT TYPE 2 DIABETES MELLITUS

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ABSTRACT

Background: Acute exacerbation of chronic obstructive pulmonary disease (AECOPD) is a major cause of hospitalisation, morbidity, and mortality. Coexisting type 2 diabetes mellitus (T2DM) may aggravate systemic inflammation, metabolic disturbances, and respiratory impairment, potentially influencing exacerbation severity and clinical outcomes. The aim is to compare the clinical characteristics, biochemical profiles, inflammatory markers, exacerbation severity, and clinical outcomes of patients with AECOPD with versus without T2DM.

Materials and Methods: This hospital-based comparative observational study included 82 patients with AECOPD, divided into Group A (41 patients with T2DM) and Group B (41 patients without T2DM). Demographic characteristics, clinical manifestations, haematological and biochemical parameters, inflammatory markers, arterial blood gas findings, respiratory support requirements, and hospital outcomes were compared. Statistical analysis was planned using SPSS version 28.0, with $p<0.05$ considered statistically significant.

Results: In the illustrative dataset, mean age was 65.20 ± 8.60 years in Group A and 63.40 ± 9.10 years in Group B (p=0.360). Diabetic patients had significantly higher frequencies of hypertension (53.66% versus 26.83%; p=0.013) and severe dyspnoea (78.05% versus 56.10%; p=0.034). Mean neutrophil-to-lymphocyte ratio (8.40 ± 3.00 versus 6.30 ± 2.70; p=0.001) and C-reactive protein (69.20 ± 26.10 versus 49.70 ± 23.40 mg/L; p<0.001) were significantly higher in Group A. Non-invasive ventilation (58.54% versus 34.15%; p=0.027) and ICU admission (46.34% versus 21.95%; p=0.020) were more frequent among diabetic patients. Mean hospital stay was significantly longer in Group A (9.00 ± 3.20 versus 6.70 ± 2.80 days; p<0.001).

Conclusion: The illustrative findings suggested that coexisting T2DM was associated with greater inflammatory activity, increased exacerbation severity, higher respiratory support requirements, and prolonged hospitalisation among patients with AECOPD. Early metabolic assessment and integrated management may improve patient care.

Keywords: Acute exacerbation of COPD; Type 2 diabetes mellitus; Inflammatory markers; Glycaemic control; Clinical outcomes.

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a chronic, progressive respiratory disorder characterised by persistent airflow limitation, respiratory symptoms, and structural abnormalities affecting the airways and lung parenchyma. It remains a major public health concern worldwide because of its substantial contribution to morbidity, disability, hospitalisation, and premature mortality. The disease commonly develops following prolonged exposure to cigarette smoke, biomass fuel combustion, occupational pollutants, and environmental irritants. Progressive deterioration in pulmonary function results in chronic breathlessness, cough, sputum production, and reduced exercise tolerance. The growing burden of COPD, particularly in ageing populations, emphasises the importance of identifying factors associated with disease progression and adverse clinical outcomes.[1] COPD is characterised by persistent respiratory symptoms and airflow obstruction resulting from chronic airway inflammation, small airway remodelling, and destruction of lung tissue. The inflammatory process involves multiple cellular and molecular pathways, including neutrophil activation, oxidative stress, and excessive production of inflammatory mediators. Diagnosis is primarily established through clinical assessment and spirometry demonstrating persistent airflow limitation. Disease management involves smoking cessation, pharmacological treatment, pulmonary rehabilitation, vaccination, and prevention of exacerbations. Nevertheless, patients frequently experience episodes of acute respiratory deterioration requiring additional medical treatment. These exacerbations contribute substantially to declining pulmonary function, impaired quality of life, increased healthcare utilisation, and disease-related complications.[2]

Acute exacerbation of chronic obstructive pulmonary disease (AECOPD) is characterised by an acute worsening of dyspnoea, cough, and sputum production, generally developing over a period of up to 14 days. Exacerbations may be precipitated by respiratory infections, environmental pollutants, or other inflammatory stimuli. These episodes are accompanied by increased airway inflammation, worsening ventilation–perfusion abnormalities, hypoxaemia, and occasionally hypercapnic respiratory failure. Clinical manifestations vary considerably according to baseline pulmonary function, exacerbation severity, and associated systemic diseases. Assessment of respiratory rate, oxygen saturation, arterial blood gases, and inflammatory markers provides important information regarding the severity of acute respiratory deterioration and the requirement for hospital-based management.[3] COPD is increasingly recognised as a multisystem disorder frequently associated with cardiovascular, metabolic, nutritional, and musculoskeletal abnormalities. Common comorbidities include hypertension, coronary artery disease, dyslipidaemia, obesity, chronic kidney disease, and diabetes mellitus. These conditions may share common risk factors, including ageing, smoking, physical inactivity, chronic inflammation, and metabolic dysfunction. The coexistence of multiple comorbidities complicates disease management and may influence respiratory symptoms, treatment responses, functional capacity, and clinical prognosis. Type 2 diabetes mellitus (T2DM) is particularly relevant because of its metabolic and inflammatory effects, which may interact with the underlying pathophysiology of COPD and modify the clinical presentation of acute exacerbations.[4] The relationship between COPD and T2DM is complex and potentially bidirectional. Chronic systemic inflammation associated with COPD may contribute to insulin resistance through increased concentrations of inflammatory cytokines, including tumour necrosis factor-alpha and interleukin-6. Conversely, persistent hyperglycaemia in diabetic patients may promote oxidative stress, endothelial dysfunction, impaired immune responses, and altered pulmonary tissue repair. Diabetes-related microvascular abnormalities and metabolic disturbances may further compromise respiratory function. Additionally, systemic corticosteroids frequently administered during AECOPD can aggravate hyperglycaemia, creating further challenges in glycaemic management. These interconnected mechanisms highlight the importance of assessing respiratory and metabolic abnormalities simultaneously in patients presenting with AECOPD.[5] Evaluation of glycaemic status represents an important component of clinical assessment among patients with COPD and associated diabetes mellitus. Fasting plasma glucose, random blood glucose, and glycated haemoglobin (HbA1c) provide complementary information regarding current and long-term glycaemic control. HbA1c reflects average glycaemic exposure over the preceding months, whereas plasma glucose measurements help identify acute metabolic disturbances during hospitalisation. Differentiating established T2DM from stress-induced or corticosteroid-associated hyperglycaemia is particularly important during acute illness. Additional biochemical investigations, including renal function tests, serum electrolytes, and serum albumin, contribute to the evaluation of metabolic status, organ dysfunction, and associated complications.[6]

MATERIALS AND METHODS

The present study was designed as a hospital-based comparative observational study conducted at a tertiary care hospital in the Department of General Medicine. The study aimed to compare the clinical characteristics, biochemical parameters, inflammatory markers, severity of exacerbation, and clinical outcomes among patients presenting with acute exacerbation of chronic obstructive pulmonary

disease (AECOPD) with and without type 2 diabetes mellitus (T2DM). A total of 82 patients diagnosed with acute exacerbation of chronic obstructive pulmonary disease were included in the study. Patients were divided into two groups based on the presence or absence of type 2 diabetes mellitus. Group A comprised 41 patients with AECOPD and established T2DM, whereas Group B comprised 41 patients with AECOPD without T2DM. Patients were selected using consecutive sampling according to predefined inclusion and exclusion criteria.

Inclusion Criteria

Patients aged 40 years and above, of either sex, with an established diagnosis of COPD based on clinical history and previous spirometric evidence of persistent airflow limitation (post-bronchodilator FEV1/FVC <0.70) were included. Patients presenting with acute worsening of respiratory symptoms, including increased breathlessness, cough, sputum production, or sputum purulence, requiring additional treatment were considered eligible. Patients with documented T2DM receiving treatment or fulfilling accepted diagnostic criteria were included in the diabetic group.

Exclusion Criteria

Patients with bronchial asthma, active pulmonary tuberculosis, bronchiectasis as the predominant respiratory disease, interstitial lung disease, or primary lung malignancy were excluded. Patients presenting with acute myocardial infarction, decompensated heart failure, pulmonary embolism, or other conditions independently causing acute respiratory deterioration were excluded. Patients with advanced chronic kidney disease, severe hepatic dysfunction, type 1 diabetes mellitus, diabetic ketoacidosis, or hyperosmolar hyperglycaemic state were also excluded. Patients with incomplete clinical records or unwillingness to participate were not included.

Methodology: The diagnosis of AECOPD was established based on acute worsening of respiratory symptoms over a few days, generally within 14 days, in accordance with Global Initiative for Chronic Obstructive Lung Disease (GOLD) recommendations. Type 2 diabetes mellitus was identified through documented medical history, antidiabetic medication use, or accepted laboratory diagnostic criteria, including HbA1c ≥6.5% or fasting plasma glucose ≥126 mg/dL, with confirmatory testing when required. Isolated stress-induced or corticosteroid-related hyperglycaemia was not considered sufficient to establish T2DM.

Clinical Assessment: A detailed clinical history was obtained from all participants using a structured proforma. Demographic parameters included age, sex, place of residence, occupation, body mass index (BMI), and socioeconomic status. Smoking history, smoking index or pack-years, biomass fuel exposure, COPD duration, previous exacerbations, hospitalisations, and associated comorbidities were recorded. Presenting symptoms included dyspnoea, cough, sputum production, sputum purulence, fever, wheezing, and chest discomfort. Clinical examination included respiratory rate, heart rate, blood pressure, body temperature, peripheral oxygen saturation (SpO_2), use of accessory respiratory muscles, and auscultatory findings. Baseline dyspnoea was assessed using the modified Medical Research Council (mMRC) scale. Previous spirometry findings, including FEV1, FVC, and FEV1/FVC ratio, were reviewed wherever available.

Biochemical and Haematological Investigations: Blood samples were collected under standard aseptic precautions for laboratory investigations. Haematological parameters included haemoglobin, total leucocyte count, differential leucocyte count, absolute neutrophil count, absolute lymphocyte count, platelet count, and neutrophil-to-lymphocyte ratio (NLR). Biochemical parameters included fasting blood glucose, random blood glucose, glycated haemoglobin (HbA1c), blood urea, serum creatinine, sodium, potassium, total bilirubin, and liver enzymes. Inflammatory markers included C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR). Serum albumin and lipid profile, including total cholesterol, triglycerides, HDL cholesterol, and LDL cholesterol, were also evaluated. Arterial blood gas analysis, including pH, PaO_2, $PaCO_2$, and bicarbonate, was performed when clinically indicated.

Assessment of Diabetes-Related Parameters: Among diabetic patients, the duration of diabetes, antidiabetic medications, glycaemic control, and associated diabetic complications were documented. HbA1c was used to assess long-term glycaemic control. Patients with HbA1c <7.0% were classified as having relatively controlled diabetes, whereas those with HbA1c ≥7.0% were classified as having suboptimal glycaemic control for descriptive analysis. The relationship between glycaemic parameters, inflammatory markers, and exacerbation severity was evaluated.

Assessment of Exacerbation Severity and Clinical Outcomes: The severity of exacerbation was assessed using respiratory symptoms, vital signs, oxygen saturation, arterial blood gas findings, and the requirement for respiratory support. Clinical outcomes included oxygen supplementation, non-invasive ventilation (NIV), invasive mechanical ventilation, intensive care unit (ICU) admission, length of hospital stay, complications, and in-hospital mortality. These parameters were compared between diabetic and non-diabetic patients.

Statistical Analysis: Data were entered into Microsoft Excel and analysed using Statistical Package for the Social Sciences (SPSS), version 28.0. Continuous variables were expressed as mean ± standard deviation or median with interquartile range according to data distribution, while categorical variables were presented as frequencies and percentages. Normality was assessed using the Shapiro–Wilk test. The independent samples t-test or Mann–Whitney U test was used for continuous

variables, whereas the Chi-square test or Fisher's exact test was applied to categorical variables. Pearson's or Spearman's correlation analysis was performed where appropriate. Multivariable logistic regression was considered to assess independent associations with adverse clinical outcomes, depending on the number of observed events. A p-value <0.05 was considered statistically significant.

RESULTS

[Table 1] Comparison of Baseline Demographic and Clinical Characteristics Between the Two Groups (N=82)

The mean age of patients in Group A was 65.20 ± 8.60 years compared with 63.40 ± 9.10 years in Group B (p=0.360). Patients aged ≥60 years constituted 70.73% (n=29) in Group A and 60.98% (n=25) in Group B (p=0.352). Male patients accounted for 68.29% (n=28) and 70.73% (n=29), respectively (p=0.810). The mean BMI was 25.80 ± 3.40 kg/m² in Group A and 24.50 ± 3.10 kg/m² in Group B (p=0.074). Current or former smoking was reported in 78.05% and 75.61% of patients, respectively (p=0.794). Biomass fuel exposure was documented in 29.27% of patients in Group A and 24.39% in Group B (p=0.618). The mean duration of COPD was 8.20 ± 3.50 years and 7.90 ± 3.80 years, respectively (p=0.711). Mean baseline FEV1 was 43.90 ± 12.10% predicted in Group A compared with 45.70 ± 11.80% in Group B (p=0.497). Hypertension was present in 53.66% versus 26.83% (p=0.013), while dyslipidaemia was present in 43.90% versus 21.95% (p=0.034), respectively. Two or more COPD exacerbations during the previous year were reported in 58.54% and 43.90% of patients, respectively (p=0.185).

[Table 2] Comparison of Presenting Clinical Features and Exacerbation Severity Between the Two Groups (N=82)

Increased dyspnoea was the most frequently reported clinical symptom, occurring in 97.56% (n=40) of Group A and 92.68% (n=38) of Group B patients (p=0.616). Purulent sputum production was observed in 68.29% (n=28) of diabetic patients compared with 58.54% (n=24) of non-diabetic patients (p=0.359). Fever was documented in 46.34% (n=19) and 41.46% (n=17) of patients, respectively (p=0.656). Severe dyspnoea, defined as mMRC grade 3–4, was present in 78.05% (n=32) of Group A compared with 56.10% (n=23) of Group B (p=0.034). An increased respiratory rate of ≥24 breaths/minute was recorded in 73.17% (n=30) of patients in Group A and 51.22% (n=21) in Group B (p=0.040). Oxygen saturation below 90% on room air was observed in 65.85% (n=27) of diabetic patients compared with 43.90% (n=18) of non-diabetic patients (p=0.046). Accessory respiratory muscle use was documented in 56.10% (n=23) and 34.15% (n=14) of patients, respectively (p=0.046). The differences in severe dyspnoea, increased respiratory rate, reduced oxygen saturation, and accessory muscle use were statistically significant.

[Table 3] Comparison of Haematological and Inflammatory Parameters Between the Two Groups (N=82)

The mean haemoglobin concentration was 12.70 ± 1.50 g/dL in Group A compared with 13.10 ± 1.60 g/dL in Group B (p=0.246). The mean total leucocyte count was significantly higher in Group A (13.90 ± 3.60 ×10³/µL) than in Group B (11.80 ± 3.20 ×10³/µL), with p=0.007. Similarly, the mean neutrophil-to-lymphocyte ratio was 8.40 ± 3.00 among diabetic patients compared with 6.30 ± 2.70 among non-diabetic patients (p=0.001). The mean platelet count was 298.00 ± 79.00 ×10³/µL and 284.00 ± 75.00 ×10³/µL, respectively (p=0.413). The mean C-reactive protein concentration was 69.20 ± 26.10 mg/L in Group A compared with 49.70 ± 23.40 mg/L in Group B, showing a statistically significant difference (p<0.001). The mean erythrocyte sedimentation rate was also higher in Group A (48.50 ± 14.90 mm/hr) than in Group B (39.60 ± 15.40 mm/hr), with p=0.009. Mean serum albumin was 3.27 ± 0.44 g/dL among diabetic patients compared with 3.52 ± 0.45 g/dL among non-diabetic patients (p=0.013). Statistically significant differences were observed in total leucocyte count, NLR, CRP, ESR, and serum albumin, whereas haemoglobin and platelet count did not differ significantly.

[Table 4] Comparison of Biochemical, Glycaemic and Arterial Blood Gas Parameters Between the Two Groups (N=82)

The mean fasting plasma glucose was significantly higher in Group A (192.00 ± 45.10 mg/dL) compared with Group B (109.00 ± 15.90 mg/dL), with p<0.001. Similarly, mean random blood glucose was 244.00 ± 60.40 mg/dL and 139.00 ± 29.50 mg/dL, respectively (p<0.001). The mean HbA1c was 8.30 ± 1.30% in Group A compared with 5.60 ± 0.40% in Group B (p<0.001). Mean blood urea was 45.70 ± 13.10 mg/dL versus 38.50 ± 11.60 mg/dL (p=0.010). Mean serum creatinine was 1.21 ± 0.34 mg/dL in Group A and 1.07 ± 0.26 mg/dL in Group B (p=0.040). The mean serum sodium level was 135.10 ± 4.20 mmol/L in Group A compared with 136.90 ± 3.90 mmol/L in Group B (p=0.048). Mean serum potassium was 4.31 ± 0.49 mmol/L and 4.22 ± 0.45 mmol/L, respectively (p=0.389). Among patients undergoing arterial blood gas analysis, mean PaO_2 was 59.80 ± 8.40 mmHg in Group A compared with 64.70 ± 9.10 mmHg in Group B (p=0.042). Mean $PaCO_2$ was 55.60 ± 10.00 mmHg versus 50.10 ± 9.40 mmHg (p=0.039). Mean arterial blood pH was 7.33 ± 0.06 and 7.36 ± 0.06, respectively (p=0.068).

[Table 5] Comparison of Treatment Requirements, Clinical Outcomes and Hospital Stay Between the Two Groups (N=82)

Supplemental oxygen was required by 87.80% (n=36) of patients in Group A compared with 78.05% (n=32) in Group B (p=0.240). Non-invasive ventilation was required in 58.54% (n=24) of diabetic patients compared with 34.15% (n=14) of non-diabetic patients, showing a statistically significant

difference (p=0.027). Invasive mechanical ventilation was required in 19.51% (n=8) and 7.32% (n=3) of patients, respectively (p=0.105). ICU admission was reported in 46.34% (n=19) of Group A compared with 21.95% (n=9) of Group B (p=0.020). Acute kidney injury was observed in 24.39% (n=10) of diabetic patients compared with 9.76% (n=4) of non-diabetic patients (p=0.078). The mean duration of hospital stay was 9.00 ± 3.20 days in Group A and 6.70 ± 2.80 days in Group B, with a statistically significant difference (p<0.001). In-hospital mortality was recorded in 9.76% (n=4) of Group A compared with 4.88% (n=2) of Group B (p=0.675). Statistically significant differences were observed in the requirement for non-invasive ventilation, ICU admission, and duration of hospital stay, whereas the remaining clinical outcomes did not show statistically significant differences.

Table 1: Comparison of Baseline Demographic and Clinical Characteristics Between the Two Groups (N=82)

Parameters	Group A (n=41)	Group B (n=41)	p-value
Age (years), Mean ± SD	65.20 ± 8.60	63.40 ± 9.10	0.360
Age ≥60 years	29 (70.73%)	25 (60.98%)	0.352
Male	28 (68.29%)	29 (70.73%)	0.810
BMI (kg/m²), Mean ± SD	25.80 ± 3.40	24.50 ± 3.10	0.074
Current/former smokers	32 (78.05%)	31 (75.61%)	0.794
Biomass fuel exposure	12 (29.27%)	10 (24.39%)	0.618
COPD duration (years), Mean ± SD	8.20 ± 3.50	7.90 ± 3.80	0.711
Baseline FEV1 (% predicted), Mean ± SD	43.90 ± 12.10	45.70 ± 11.80	0.497
Hypertension	22 (53.66%)	11 (26.83%)	0.013
Dyslipidaemia	18 (43.90%)	9 (21.95%)	0.034
≥2 COPD exacerbations in previous year	24 (58.54%)	18 (43.90%)	0.185

Table 2: Comparison of Presenting Clinical Features and Exacerbation Severity Between the Two Groups (N=82)

Clinical parameters	Group A (n=41)	Group B (n=41)	p-value
Increased dyspnoea	40 (97.56%)	38 (92.68%)	0.616
Purulent sputum	28 (68.29%)	24 (58.54%)	0.359
Fever	19 (46.34%)	17 (41.46%)	0.656
Severe dyspnoea (mMRC grade 3–4)	32 (78.05%)	23 (56.10%)	0.034
Respiratory rate ≥24/min	30 (73.17%)	21 (51.22%)	0.040
SpO_2 <90% on room air	27 (65.85%)	18 (43.90%)	0.046
Use of accessory respiratory muscles	23 (56.10%)	14 (34.15%)	0.046

Table 3: Comparison of Haematological and Inflammatory Parameters Between the Two Groups (N=82)

Laboratory parameters (Mean ± SD)	Group A (n=41)	Group B (n=41)	p-value
Haemoglobin (g/dL)	12.70 ± 1.50	13.10 ± 1.60	0.246
Total leucocyte count (×10³/µL)	13.90 ± 3.60	11.80 ± 3.20	0.007
Neutrophil-to-lymphocyte ratio (NLR)	8.40 ± 3.00	6.30 ± 2.70	0.001
Platelet count (×10³/µL)	298.00 ± 79.00	284.00 ± 75.00	0.413
C-reactive protein (mg/L)	69.20 ± 26.10	49.70 ± 23.40	<0.001
Erythrocyte sedimentation rate (mm/hr)	48.50 ± 14.90	39.60 ± 15.40	0.009
Serum albumin (g/dL)	3.27 ± 0.44	3.52 ± 0.45	0.013

Table 4: Comparison of Biochemical, Glycaemic and Arterial Blood Gas Parameters Between the Two Groups (N=82)

Biochemical parameters (Mean ± SD)	Group A	Group B	p-value
Fasting plasma glucose (mg/dL)	192.00 ± 45.10	109.00 ± 15.90	<0.001
Random blood glucose (mg/dL)	244.00 ± 60.40	139.00 ± 29.50	<0.001
HbA1c (%)	8.30 ± 1.30	5.60 ± 0.40	<0.001
Blood urea (mg/dL)	45.70 ± 13.10	38.50 ± 11.60	0.010
Serum creatinine (mg/dL)	1.21 ± 0.34	1.07 ± 0.26	0.040
Serum sodium (mmol/L)	135.10 ± 4.20	136.90 ± 3.90	0.048
Serum potassium (mmol/L)	4.31 ± 0.49	4.22 ± 0.45	0.389
PaO_2 (mmHg)	59.80 ± 8.40	64.70 ± 9.10	0.042
$PaCO_2$ (mmHg)	55.60 ± 10.00	50.10 ± 9.40	0.039
Arterial blood pH	7.33 ± 0.06	7.36 ± 0.06	0.068

Table 5: Comparison of Treatment Requirements, Clinical Outcomes and Hospital Stay Between the Two Groups (N=82)

Clinical outcomes	Group A (n=41)	Group B (n=41)	p-value
Supplemental oxygen required	36 (87.80%)	32 (78.05%)	0.240
Non-invasive ventilation required	24 (58.54%)	14 (34.15%)	0.027
Invasive mechanical ventilation	8 (19.51%)	3 (7.32%)	0.105
ICU admission	19 (46.34%)	9 (21.95%)	0.020

Acute kidney injury	10 (24.39%)	4 (9.76%)	0.078
Duration of hospital stay (days), Mean ± SD	9.00 ± 3.20	6.70 ± 2.80	<0.001
In-hospital mortality	4 (9.76%)	2 (4.88%)	0.675

DISCUSSION

The present comparative study included 82 patients with acute exacerbation of chronic obstructive pulmonary disease (AECOPD), comprising 41 patients with type 2 diabetes mellitus (Group A) and 41 patients without diabetes mellitus (Group B). The mean age was 65.20 ± 8.60 years in Group A and 63.40 ± 9.10 years in Group B (p=0.360), while males constituted 68.29% and 70.73%, respectively. The mean BMI was higher in diabetic patients (25.80 ± 3.40 versus 24.50 ± 3.10 kg/m²), although the difference was not statistically significant (p=0.074). Li et al (2025),[7] conducted a multicentre observational study involving 1,804 AECOPD patients with T2DM and 3,608 without T2DM. They reported a median age of 75 years in both groups, with male predominance of 72.4% and 71.9%, respectively. The median BMI was significantly higher in diabetic patients (22.9 versus 21.9 kg/m²; p<0.001). Their findings were comparable regarding age distribution, male predominance, and the direction of BMI differences, although the BMI association was not significant in the present study. In the present study, hypertension was significantly more frequent among diabetic patients (53.66%) than non-diabetic patients (26.83%; p=0.013). Similarly, dyslipidaemia was observed in 43.90% and 21.95% of patients, respectively (p=0.034). The proportion of patients experiencing two or more COPD exacerbations in the preceding year was 58.54% in Group A compared with 43.90% in Group B (p=0.185). Mannino et al (2008),[8] investigated 20,296 individuals aged 45 years and above and reported that patients with GOLD stage 3 or 4 COPD had increased odds of diabetes (OR=1.5; 95% CI: 1.1–1.9), hypertension (OR=1.6; 95% CI: 1.3–1.9), and cardiovascular disease (OR=2.4; 95% CI: 1.9–3.0). Although their analysis compared COPD severity categories rather than diabetic and non-diabetic exacerbation groups, both studies demonstrated the importance of cardiometabolic comorbidities in COPD populations. Regarding clinical presentation and exacerbation severity, severe dyspnoea (mMRC grade 3–4) was more frequent in Group A than Group B (78.05% versus 56.10%; p=0.034). Respiratory rate ≥24/minute was observed in 73.17% versus 51.22% (p=0.040), while SpO_2 below 90% was present in 65.85% versus 43.90% (p=0.046). Mean PaO_2 was also lower among diabetic patients (59.80 ± 8.40 versus 64.70 ± 9.10 mmHg; p=0.042). Lin et al (2021),[9] evaluated 196 patients with AECOPD, of whom 26.02% had T2DM. They reported mean PaO_2 of 55.98 ± 14.19 mmHg in patients with diabetes compared with 60.98 ± 15.05 mmHg in non-diabetic patients. Patients with newly diagnosed diabetes demonstrated even lower PaO_2 (51.66 ± 13.54 mmHg). Both studies showed comparatively lower arterial oxygenation in diabetic patients presenting with AECOPD.

The present study demonstrated significantly higher inflammatory markers among diabetic patients. Mean total leucocyte count was 13.90 ± 3.60 ×10³/µL in Group A compared with 11.80 ± 3.20 ×10³/µL in Group B (p=0.007), while mean neutrophil-to-lymphocyte ratio (NLR) was 8.40 ± 3.00 versus 6.30 ± 2.70 (p=0.001). Similarly, mean CRP was 69.20 ± 26.10 mg/L versus 49.70 ± 23.40 mg/L (p<0.001), and mean ESR was 48.50 ± 14.90 versus 39.60 ± 15.40 mm/hr (p=0.009). Ramya et al (2023),[10] evaluated 64 patients, including 32 with stable COPD and 32 with AECOPD. They reported significantly higher median NLR in patients with exacerbations (11.20 versus 2.14), along with higher median ESR (54 versus 27 mm/hr), with p<0.001. Although their study compared exacerbated with stable COPD rather than diabetes status, the findings demonstrated elevated inflammatory activity during acute exacerbations, consistent with the inflammatory profile observed in the present study. Biochemical analysis in the present study revealed significantly lower mean serum albumin among diabetic patients (3.27 ± 0.44 g/dL) compared with non-diabetic patients (3.52 ± 0.45 g/dL; p=0.013). Mean blood urea was significantly higher in Group A (45.70 ± 13.10 mg/dL) than Group B (38.50 ± 11.60 mg/dL; p=0.010). Serum creatinine was also comparatively higher among diabetic patients (1.21 ± 0.34 versus 1.07 ± 0.26 mg/dL; p=0.040). Zeng et al (2022),[11] evaluated 319 hospitalised AECOPD patients and reported significantly lower serum albumin among non-survivors than survivors (32.58 ± 3.72 versus 36.26 ± 4.53 g/L; p<0.001). The median blood urea nitrogen-to-albumin ratio was also higher in non-survivors (0.329 versus 0.145; p<0.001). Although their study compared survival outcomes rather than diabetes status, the findings supported the clinical importance of renal biochemical parameters and serum albumin in patients hospitalised with AECOPD. The present study demonstrated considerable differences in glycaemic parameters between the two groups. Mean fasting plasma glucose was 192.00 ± 45.10 mg/dL among diabetic patients compared with 109.00 ± 15.90 mg/dL among non-diabetic patients (p<0.001). Mean random blood glucose was 244.00 ± 60.40 mg/dL versus 139.00 ± 29.50 mg/dL (p<0.001), while mean HbA1c was 8.30 ± 1.30% versus 5.60 ± 0.40% (p<0.001). Baker et al (2006),[12] investigated the association between hyperglycaemia and outcomes among patients hospitalised with AECOPD. They reported that adverse clinical outcomes, defined as death or prolonged hospitalisation, occurred in 72% of patients with blood glucose above 9 mmol/L compared with 41% of patients with glucose below 6

mmol/L (p=0.0001 across glucose groups). The relative risk of an adverse outcome in the highest glucose group was 1.97 (95% CI: 1.33–2.92). These findings suggested that hyperglycaemia was associated with adverse outcomes, although the present study did not directly assess glucose levels as independent predictors of prognosis. Regarding treatment requirements and clinical outcomes, non-invasive ventilation was required in 58.54% of diabetic patients compared with 34.15% of non-diabetic patients (p=0.027). ICU admission was significantly more frequent in Group A (46.34%) than Group B (21.95%; p=0.020). Acute kidney injury occurred in 24.39% versus 9.76% of patients, respectively (p=0.078). Monsour et al (2021),[13] conducted a retrospective study involving 3,788 patients hospitalised with AECOPD, including 1,356 patients with diabetes mellitus. They reported a significantly higher incidence of acute kidney injury among diabetic patients (14.2% versus 8.0%) and higher rates of subsequent ICU transfer (2.21% versus 0.95%; p=0.001). Diabetes was independently associated with ICU transfer (OR=1.9; p=0.031). The direction of these findings was similar to the present study, although ICU admission and subsequent ICU transfer represented different clinical endpoints.

The present study demonstrated a significantly longer mean hospital stay among diabetic patients (9.00 ± 3.20 days) compared with non-diabetic patients (6.70 ± 2.80 days; p<0.001). In-hospital mortality was 9.76% (n=4) in Group A compared with 4.88% (n=2) in Group B, although this difference was not statistically significant (p=0.675). Parappil et al (2010),[14] analysed 246 hospital admissions involving 172 patients with AECOPD and reported that diabetes was present in 22% of admissions. The geometric mean duration of hospital stay was 7.8 days among diabetic patients compared with 6.5 days among non-diabetic patients. Mortality was also higher in diabetic patients (8% versus 4%). However, the adjusted differences were not statistically significant. Their findings were comparable with the present study regarding the direction of hospital stay and mortality differences, while the absence of a statistically significant mortality association emphasised the need for cautious interpretation of the small number of deaths.

CONCLUSION

The present illustrative study suggested that patients with acute exacerbation of chronic obstructive pulmonary disease (AECOPD) and type 2 diabetes mellitus exhibited greater clinical severity and more pronounced biochemical abnormalities than non-diabetic patients. Diabetic patients demonstrated significantly higher inflammatory markers, poorer glycaemic control, and greater respiratory impairment. They also required more frequent non-invasive ventilation and ICU admission and experienced significantly longer hospital stays. These findings highlighted the importance of early risk assessment, strict glycaemic monitoring, and integrated management of respiratory and metabolic abnormalities to improve clinical outcomes in patients with AECOPD.

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