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Assessment of Insulin Resistance in Patients with Chronic Obstructive Pulmonary Disease Attending a Tertiary Care Hospital

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ABSTRACT

Introduction: Chronic Obstructive Pulmonary Disease (COPD) is increasingly recognized as a systemic inflammatory disorder with multiple extra-pulmonary manifestations. Insulin resistance (IR) is one such metabolic abnormality that contributes to cardiovascular morbidity and poor outcomes in COPD patients.

Aim: To assess insulin resistance in patients with COPD and to evaluate its association with disease severity.

Materials and Methods: This hospital-based cross-sectional study was conducted in a tertiary care hospital and included 100 participants: 50 clinically stable COPD patients diagnosed according to ATS/ERS guidelines and 50 age- and sex-matched healthy controls. Fasting blood glucose, fasting serum insulin, lipid profile parameters, and anthropometric measurements were recorded. Insulin resistance was assessed using the Homeostatic Model Assessment of Insulin Resistance (HOMA-IR). Surrogate markers such as triglyceride-to-HDL ratio (TG:HDL) and triglyceride-glucose (TyG) index were also calculated. Pulmonary function tests were performed using spirometry. Statistical analysis was carried out using SPSS version 25, and a p-value <0.05 was considered statistically significant.

Results: COPD patients had significantly higher fasting blood glucose, fasting insulin levels, HOMA-IR, TG:HDL ratio, and TyG index compared to controls ($p<0.001$). Insulin resistance showed a significant progressive increase with advancing stages of COPD. HOMA-IR demonstrated a strong association with disease severity.

Conclusion: Insulin resistance is significantly increased in patients with COPD and correlates positively with disease severity. Early identification of metabolic abnormalities may help reduce long-term cardiovascular and metabolic complications in COPD patients.

Keywords: Chronic Obstructive Pulmonary Disease; Insulin Resistance; HOMA-IR; TyG Index; Metabolic Dysfunction

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a common, preventable, and treatable respiratory disorder characterized by persistent airflow limitation that is usually progressive and associated with an enhanced chronic inflammatory response in the airways and lungs

to noxious particles or gases [1]. Globally, COPD represents a major public health burden and is currently one of the leading causes of morbidity and mortality worldwide [2]. In India, the prevalence of COPD is rising due to increasing tobacco consumption, exposure to biomass fuel smoke, environmental pollution, and occupational hazards [3]. Traditionally, COPD was considered a disease confined to the lungs. However, growing evidence has established COPD as a systemic inflammatory disorder with multiple extra-pulmonary manifestations [4]. Chronic low-grade systemic inflammation plays a central role in the pathogenesis of these manifestations and contributes to the development of various comorbid conditions, including cardiovascular disease, skeletal muscle dysfunction, osteoporosis, metabolic syndrome, and diabetes mellitus [5,6]. These comorbidities significantly impact disease progression, quality of life, healthcare utilization, and mortality among COPD patients [7].

Insulin resistance (IR), defined as a reduced biological response of peripheral tissues to insulin, is a key metabolic abnormality underlying type 2 diabetes mellitus, metabolic syndrome, and cardiovascular disease [8]. Increasing evidence suggests that insulin resistance is prevalent among patients with COPD, even in the absence of overt diabetes mellitus [9]. The pathophysiological mechanisms linking COPD and insulin resistance are complex and multifactorial. Chronic systemic inflammation, oxidative stress, hypoxia, physical inactivity, and altered adipokine secretion have all been implicated in the development of insulin resistance in COPD patients [10,11].

Inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and C-reactive protein (CRP), which are elevated in COPD, interfere with insulin signaling pathways and promote peripheral insulin resistance [12]. Additionally, chronic hypoxemia seen in advanced COPD may impair glucose metabolism by inducing mitochondrial dysfunction and altering skeletal muscle fiber composition [13]. Reduced physical activity and corticosteroid exposure further exacerbate metabolic derangements in these patients [14].

Several epidemiological studies have demonstrated an inverse relationship between lung function parameters and glucose metabolism. Reduced forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) have been associated with an increased risk of developing insulin resistance and type 2 diabetes mellitus [15]. Conversely, diabetes mellitus has also been shown to adversely affect lung function, suggesting a bidirectional relationship between pulmonary dysfunction and metabolic abnormalities [16].

The Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) is a widely accepted and validated method for estimating insulin resistance using fasting glucose and insulin levels [17]. In recent years, surrogate markers such as the triglyceride-to-high-density lipoprotein cholesterol ratio (TG:HDL) and the triglyceride-glucose (TyG) index have emerged as simple, cost-effective indicators of insulin resistance and metabolic risk [18,19]. These indices are particularly useful in resource-limited settings and may help identify subclinical metabolic abnormalities in COPD patients.

Although several international studies have reported a higher prevalence of insulin resistance among COPD patients, data from the Indian population remain limited and inconsistent [9,20]. Furthermore, the association between insulin resistance and the severity of COPD has not been adequately explored in Indian settings. Understanding this relationship is crucial, as insulin resistance may contribute to increased cardiovascular morbidity and poorer clinical outcomes in COPD patients.

Therefore, the present study was undertaken to assess insulin resistance in patients with COPD using HOMA-

IR and surrogate metabolic indices such as TG:HDL ratio and TyG index, and to compare these parameters with age- and sex-matched healthy controls. The study also aimed to evaluate the association between insulin resistance and the severity of COPD in a tertiary care hospital setting.

MATERIALS AND METHODS

Study Design and Study Population

This hospital-based cross-sectional study was conducted in the Department of Biochemistry in collaboration with the Department of Pulmonary Medicine at a tertiary care hospital. A total of 100 participants were enrolled in the study, comprising 50 patients diagnosed with Chronic Obstructive Pulmonary Disease (COPD) and 50 age- and sex-matched healthy controls.

Patients with COPD were diagnosed according to the updated American Thoracic Society/European Respiratory Society (ATS/ERS) guidelines [1]. Only clinically stable COPD patients with a disease duration of more than one year were included. The control group consisted of apparently healthy individuals without any known respiratory or metabolic disorders. Inclusion criteria included patients aged ≥30 years with stable COPD.

Exclusion criteria were the presence of other obstructive airway diseases, diabetes mellitus, thyroid disorders, hyperlipidaemia, ischemic heart disease, renal, hepatic or cardiac failure, autoimmune diseases, malignancy, or any acute or chronic infectious condition.

The study was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to enrollment.

Clinical Evaluation and Laboratory Investigations

A detailed clinical history was obtained from all participants, followed by anthropometric measurements including body mass index (BMI), waist circumference, and waist–hip ratio. After an overnight fast, venous blood samples were collected under aseptic conditions and analyzed for biochemical parameters.

Fasting blood glucose was measured using the hexokinase method. Fasting serum insulin levels were estimated using chemiluminescent immunoassay (CLIA). Insulin resistance was assessed using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), calculated using the formula:

HOMA-IR = [Fasting glucose (mg/dL) × Fasting insulin (µIU/mL)] / 405 [17].

Serum triglycerides and high-density lipoprotein cholesterol (HDL-C) were measured using standard enzymatic methods. The triglyceride-to-HDL cholesterol ratio (TG:HDL) was calculated. The triglyceride-glucose (TyG) index was derived using the formula:

TyG index = ln [fasting triglycerides (mg/dL) × fasting glucose (mg/dL) / 2] [18].

Pulmonary function testing was performed for all participants using a computer-assisted spirometer in accordance with European Respiratory Society guidelines. Forced expiratory volume in one second (FEV_1), forced vital capacity (FVC), and FEV_1/FVC ratio were recorded. The best of three acceptable maneuvers was considered for analysis.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using Statistical Package for Social Sciences (SPSS) software version 25. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were expressed as frequencies and percentages. Normality of data distribution was assessed using the Kolmogorov–Smirnov test.

Comparisons between two independent groups were performed using the unpaired Student's t-test for normally distributed variables and the Mann–Whitney U test for non-normally distributed data. For comparison

across more than two groups, one-way analysis of variance (ANOVA) or Kruskal–Wallis test was applied as appropriate. Categorical variables were compared using the chi-square test. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 100 participants were included in the study, comprising 50 patients with COPD and 50 age- and sex-matched healthy controls. The results are presented under appropriate subheadings with a maximum of four tables.

Baseline Characteristics of Study Participants

The mean age of patients in the COPD group was 49.20 ± 12.70 years, while that of the control group was 46.28 ± 12.71 years. The difference in age between the two groups was not statistically significant (p = 0.25). Males constituted the majority in both groups (COPD: 62%; controls: 64%), and gender distribution was comparable (p = 0.83). Anthropometric parameters, including body mass index (BMI), waist circumference, and waist–hip ratio, were slightly higher in the COPD group but did not differ significantly from controls.

Table 1: Baseline demographic and anthropometric characteristics of study subjects

Parameter	COPD group (n=50)	Control group (n=50)	p-value
Age (years)	49.20 ± 12.70	46.28 ± 12.71	0.25
Male/Female	31 / 19	32 / 18	0.83
BMI (kg/m²)	24.52 ± 3.28	23.41 ± 3.00	0.08
Waist circumference (cm)	92.04 ± 10.90	88.16 ± 9.85	0.06
Waist–hip ratio	0.96 ± 0.20	0.89 ± 0.10	0.06

Comparison of Glycemic and Insulin Resistance Parameters

Fasting blood glucose and fasting serum insulin levels were significantly higher in the COPD group compared to controls (p < 0.001). Insulin resistance assessed by HOMA-IR was markedly elevated in COPD patients, indicating a significantly higher degree of insulin resistance in this group.

Table 2: Comparison of glycemic and insulin resistance parameters between groups

Parameter	COPD group (n=50)	Control group (n=50)	p-value
Fasting blood glucose (mg/dL)	106.66 ± 13.56	92.84 ± 6.87	<0.001
Fasting serum insulin (mIU/L)	12.52 ± 4.12	9.26 ± 1.86	<0.001
HOMA-IR	3.36 ± 1.68	1.77 ± 0.86	<0.001

Comparison of Lipid-Based Insulin Resistance Indices

Serum triglyceride levels were significantly higher in the COPD group compared to controls (p < 0.001). Although HDL cholesterol levels were slightly lower in COPD patients, the difference was not statistically significant. Both TG:HDL ratio and TyG index were significantly elevated in COPD patients, reflecting increased metabolic and insulin resistance risk.

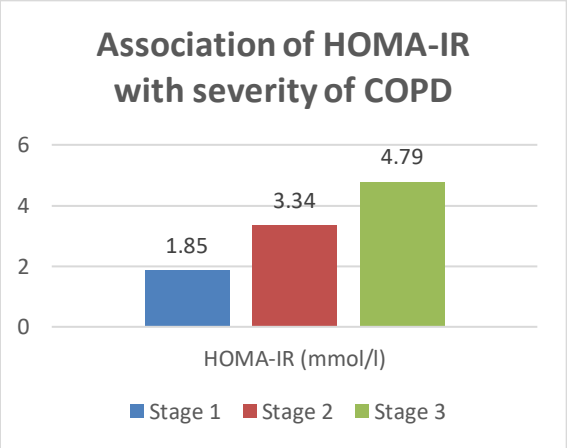
Table 3: Comparison of lipid profile and surrogate insulin resistance indices

Parameter	COPD group (n=50)	Control group (n=50)	p-value
Triglycerides (mg/dL)	117.22 ± 15.41	98.08 ± 10.23	<0.001
HDL cholesterol (mg/dL)	42.50 ± 6.56	44.82 ± 6.65	0.08
TG:HDL ratio	2.82 ± 0.58	2.23 ± 0.40	<0.001
TyG index	8.72 ± 0.19	8.41 ± 0.13	<0.001

Association of Insulin Resistance with COPD Severity

Based on spirometric assessment, 32% of patients had Stage 1 COPD, while 34% each had Stage 2 and Stage 3 disease. A progressive and statistically significant increase in insulin resistance was observed with increasing COPD severity. Mean HOMA-IR values rose from 1.85 ± 0.35 in Stage 1 to 3.34 ± 1.60 in Stage 2 and 4.79 ± 1.22 in Stage 3 ($p < 0.001$), indicating a strong association between insulin resistance and disease severity (figure 1).

Figure 1: Association of HOMA-IR with severity of COPD



DISCUSSION

The present hospital-based cross-sectional study was conducted to assess insulin resistance in patients with Chronic Obstructive Pulmonary Disease (COPD) and to evaluate its association with disease severity using established biochemical and surrogate metabolic markers. The key findings of this study demonstrate that insulin resistance is significantly higher in COPD patients compared to age- and sex-matched healthy controls, and that the degree of insulin resistance increases progressively with advancing severity of COPD.

Demographic and Anthropometric Profile

In the present study, the mean age and gender distribution of COPD patients were comparable to those of the control group, minimizing the confounding effect of age and sex on metabolic parameters. The predominance of male patients observed in this study is consistent with earlier reports from India and other developing countries, where smoking and occupational exposure remain more prevalent among men [3,7]. However, a substantial proportion of female patients was also noted, reflecting increasing exposure to biomass fuel smoke and indoor air pollution among women [3].

Anthropometric parameters such as BMI, waist circumference, and waist–hip ratio were slightly higher in COPD patients compared to controls, but the differences were not statistically significant. Similar observations have been reported by Sagun et al. and Minas et al., who demonstrated that insulin resistance in COPD may occur independent of generalized obesity [9,20]. These findings suggest that metabolic abnormalities in COPD are not solely attributable to excess adiposity, highlighting the role of systemic inflammation and disease-related metabolic dysregulation.

Glycemic Status and Insulin Levels

COPD patients in the present study exhibited significantly higher fasting blood glucose and fasting serum insulin levels compared to healthy controls. Importantly, patients with known diabetes mellitus were excluded, indicating that these abnormalities represent subclinical disturbances in glucose–insulin homeostasis. These findings are in agreement with Bolton et al., who demonstrated elevated fasting glucose and hyperinsulinemia in COPD patients without overt diabetes [10]. Similar results have been reported by Minas et al and Sagun et al., reinforcing the concept that COPD itself predisposes patients to impaired glucose metabolism [9,20].

Chronic systemic inflammation, hypoxia, oxidative stress, and reduced physical activity are proposed mechanisms underlying these alterations [11,12]. Elevated inflammatory cytokines such as TNF-α and IL-6 interfere with insulin signaling pathways, resulting in

peripheral insulin resistance [12].

Insulin Resistance Assessed by HOMA-IR

A major finding of the present study is the significantly higher HOMA-IR values observed in COPD patients compared to controls. This finding clearly establishes insulin resistance as an important systemic manifestation of COPD. The magnitude of insulin resistance observed in this study is comparable to that reported by Minas et al., and Sagun et al., all of whom demonstrated significantly elevated HOMA-IR values in COPD patients even in the absence of diabetes mellitus [9,20].

The consistent demonstration of insulin resistance across different populations suggests that it is an intrinsic component of COPD pathophysiology rather than a coincidental finding. Chronic low-grade systemic inflammation, hypoxia-induced metabolic stress, altered skeletal muscle metabolism, and physical inactivity are likely contributors to this phenomenon [10–13].

Lipid Profile and Surrogate Markers of Insulin Resistance

In addition to HOMA-IR, surrogate markers of insulin resistance such as TG:HDL ratio and TyG index were significantly elevated in COPD patients in the present study. Elevated serum triglyceride levels and increased TG:HDL ratio indicate the presence of atherogenic dyslipidemia, which has been linked to insulin resistance and increased cardiovascular risk [18]. These findings are consistent with previous studies that have reported an increased prevalence of metabolic syndrome components in COPD patients [19,20].

The TyG index, a validated surrogate marker of insulin resistance, was also significantly higher in COPD patients. Wu et al. demonstrated a strong association between TyG index and impaired lung function, supporting its utility as a simple and cost-effective marker of metabolic risk in COPD [19]. The use of lipid-based indices in the present study adds strength to the findings and provides practical tools for early metabolic screening in routine clinical practice.

Association Between Insulin Resistance and COPD **Severity**

An important observation of this study is the progressive increase in insulin resistance with advancing severity of COPD. HOMA-IR values increased significantly from Stage 1 to Stage 3 disease, indicating a strong association between declining lung function and worsening metabolic derangement. Similar findings have been reported by Bolton et al. and Watz et al., who demonstrated higher levels of insulin resistance and systemic inflammation in patients with more severe airflow obstruction [10,15].

This severity-dependent relationship supports the concept of COPD as a progressive systemic inflammatory disorder. Hypoxia, increased inflammatory burden, and reduced physical activity in advanced disease stages may further exacerbate insulin resistance. Although fasting glucose levels and TG:HDL ratio showed an increasing trend with disease severity, these changes did not reach statistical significance, possibly due to the relatively smaller sample size within each COPD stage.

The findings of the present study have important clinical implications. Insulin resistance in COPD patients may contribute to increased cardiovascular morbidity, metabolic syndrome, and poor overall prognosis. Early identification of insulin resistance using simple and cost-effective markers such as HOMA-IR, TG:HDL ratio, and TyG index may allow timely intervention and risk stratification, particularly in patients with moderate to severe disease.

CONCLUSION

Patients with Chronic Obstructive Pulmonary Disease exhibit significantly higher insulin resistance compared to healthy controls, even in the absence of diabetes

mellitus. Insulin resistance increases with advancing severity of COPD, indicating a close association between disease progression and metabolic dysfunction. Simple indices such as HOMA-IR and TyG index may be useful, cost-effective tools for early detection of metabolic risk in COPD patients. Early recognition and management of insulin resistance may help reduce associated cardiovascular and metabolic complications.

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