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Atherogenic dyslipidemia as a novel cardiovascular risk stratification tool in chronic obstructive pulmonary disease: a hospital-based cross-sectional study

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Abstract

Chronic obstructive pulmonary disease (COPD) is characterized by progressive airway obstruction and systemic inflammation, frequently accompanied by metabolic comorbidities, including dyslipidemia. The Atherogenic Index of Plasma (AIP), calculated as $\log_{10}$[triglycerides/high-density lipoprotein (HDL)-cholesterol], is strongly associated with atherosclerotic cardiovascular disease risk, yet its relationship with COPD-associated dyslipidemia remains underexplored. This study investigates the association between atherogenic dyslipidemia profiles and COPD duration, utilizing AIP as an evaluation metric.

A hospital-based cross-sectional analytical study was conducted among 100 COPD patients without acute exacerbations. Fasting serum lipid profiles were analyzed using standardized protocols, and AIP was calculated for each participant. Patients were stratified into three AIP risk categories: low risk (AIP<0.21), moderate risk (AIP 0.21-0.5), and high risk (AIP>0.5). Associations between COPD duration, smoking status, demographic variables, and lipid parameters were examined using chi-square analysis.

60% of COPD patients demonstrated high atherogenic index profiles (AIP>0.5), indicating substantial cardiovascular risk. A significant positive correlation was found between COPD duration and AIP (r=0.456, p<0.001), as well as with triglycerides (r=0.341, p<0.001) and low-density lipoprotein-cholesterol (r=0.405, p<0.001). Conversely, COPD duration demonstrated a nonsignificant trend toward an inverse correlation with HDL-cholesterol (r=-0.173, p=0.089). Seventy-one percent of patients demonstrated low HDL-cholesterol (<40 mg/dL). Smoking status was significantly associated with elevated very low-density lipoprotein-cholesterol (p=0.002).

The high prevalence of atherogenic dyslipidemia in COPD patients, as quantified by AIP, suggests that lipid profile alteration is progressive and correlates with disease duration. AIP represents a clinically relevant, cost-effective screening tool for identifying COPD patients at elevated cardiovascular risk, facilitating early intervention and personalized risk stratification strategies.

Key words: Atherogenic Index of Plasma, chronic obstructive pulmonary disease, dyslipidemia, cardiovascular risk, lipid biomarkers.

Introduction

Chronic Obstructive Pulmonary Disease (COPD) represents a significant and growing health burden globally, affecting approximately 280 million individuals and causing over 3.23 million deaths annually according to the World Health Organization [1]. COPD is characterized by progressive, irreversible airway obstruction resulting from chronic inflammatory responses to environmental exposures, particularly tobacco smoke and occupational irritants, coupled with genetic predispositions [1,2]. The disease manifests with persistent cough, dyspnea, wheezing, and fatigue, with objective pulmonary function deterioration detectable via spirometry using Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2024 diagnostic criteria [3].

Beyond pulmonary dysfunction, COPD is increasingly recognized as a systemic inflammatory disease with significant metabolic consequences [4]. Recent evidence demonstrates that systemic metabolic comorbidities, rather than respiratory failure alone, account for a disproportionate burden of mortality in COPD patients [5]. Among these metabolic complications, dyslipidemia—characterized by elevated triglycerides (TG), reduced high-density lipoprotein cholesterol (HDL-C), elevated low-density lipoprotein cholesterol (LDL-C), and increased very-low-density lipoprotein cholesterol (VLDL-C)—significantly amplifies the risk of atherosclerotic cardiovascular disease (CVD) and metabolic syndrome [4,6].

Traditional lipid profiling relies on individual lipid parameters to assess cardiovascular risk. However, these univariate approaches frequently underestimate cardiovascular risk in patients with specific lipid patterns, particularly those with elevated triglycerides and reduced HDL-cholesterol [7]. The Atherogenic Index of Plasma (AIP), calculated as log10(triglycerides/HDL-cholesterol), integrates multiple lipid parameters into a single, dimensionless index that reflects the atherogenicity of plasma lipoprotein particles [8,9]. Emerging evidence indicates that AIP provides superior predictive value for atherosclerotic CVD, coronary artery disease severity, acute myocardial infarction risk, and major adverse cardiovascular events compared to individual lipid parameters or conventional risk scores [8-11].

Despite the well-documented systemic inflammation and dyslipidemia in COPD, the atherogenicity profile of dyslipidemia in COPD patients, as quantified by AIP, remains inadequately characterized. No prior studies have systematically evaluated AIP as a stratification biomarker in COPD populations or investigated its relationship with COPD disease progression.

This gap is clinically significant, as identifying high-risk atherogenic profiles could enable targeted cardiovascular screening and preventive interventions in this vulnerable population.

This study aims to bridge this evidence gap by comprehensively investigating the association between COPD and deranged lipid profiles, with specific emphasis on atherogenic dyslipidemia phenotypes as measured by AIP. We hypothesize that COPD patients demonstrate progressive atherogenic dyslipidemia with prolonged disease duration, and that AIP effectively stratifies cardiovascular risk in this population.

Study objectives

1. To determine the prevalence of atherogenic dyslipidemia (elevated AIP) in a COPD patient population
2. To investigate the association between COPD duration and progressive alterations in serum lipid profile parameters, particularly triglycerides, HDL-cholesterol, and calculated AIP
3. To evaluate the relationship between demographic factors (age, gender) and smoking history with lipid profile abnormalities and AIP
4. To assess whether socioeconomic factors, such as urban versus rural residence, influence lipid metabolism and atherogenicity profiles in COPD

Materials and Methods

Study design and setting

This is a cross-sectional analytical study conducted at a regional tertiary care teaching hospital in accordance with institutional research protocols. The study period extended over four consecutive months (March-June 2024). Using consecutive sampling, a total of 100 patients were enrolled from the outpatient department and inpatient wards of the Department of Medicine. The research was approved by the Institutional Review Board (IRB) of the affiliated medical college and conducted in adherence with the ethical standards established by the institutional research committee and the Declaration of Helsinki (1964). While this is a cross-sectional study design, disease duration was captured as years elapsed from COPD diagnosis to enrollment. Although this allows us to observe associations across different durations of disease, it cannot definitively establish temporal precedence or longitudinal progression.

Sample size calculation

The sample size was determined using the standard formula for a single proportion in a cross-sectional study: $n = Z^2.P(1-P)/d^2$, wherein the estimated prevalence of dyslipidemia in COPD patients was taken as 40% (based on previous literature). Using a 95% confidence level (Z = 1.96) and a margin of error (absolute precision) of 10%, the minimum required sample size was determined to be approximately 92. To account for a potential 10% non-response rate or incomplete medical records, the final sample size was rounded to 100 participants, ensuring adequate statistical power for the descriptive and correlational analyses performed.

Study population and eligibility criteria

To isolate the association between COPD and atherogenic dyslipidemia, a rigorous screening process was employed to eliminate established metabolic and pharmacological confounders.

Inclusion criteria

- Patients aged ≥18 years with a physician-confirmed diagnosis of COPD based on clinical and radiological features. In the absence of routine spirometry, diagnostic secondary validation was established via documented radiological evidence (Chest X-ray or CT scan) showing characteristic features such as pulmonary hyperinflation, diaphragmatic flattening, or increased radiolucency, combined with a consistent clinical history of chronic cough, sputum production, or dyspnea.
- Clinically stable COPD (no acute exacerbations within 4 weeks prior to enrollment)
- Willingness to provide written informed consent and comply with study procedures

Exclusion criteria

- Metabolic & Cardiovascular Confounders: Known history of Diabetes Mellitus, Hypertension, metabolic syndrome or established Cardiovascular Disease (CVD).
- Pharmacological Confounders: Current or prior therapy with lipid-modifying agents (statins/fibrates), systemic corticosteroid therapy within the last 4 weeks, or antimicrobial therapy.

- Other Conditions: Active respiratory tract infections (other than COPD), severe renal or hepatic disease, pregnancy, or any other systemic illnesses.

A total of 174 patients were screened for eligibility during the study period. Following a rigorous selection process, individuals were excluded; the majority (71% of excluded patients) were disqualified due to metabolic confounders (primarily diabetes, hypertension or existing CVD), while the remainder were excluded due to pharmacological or infectious factors. This resulted in a final, highly selective cohort of 100 participants with clinically and radiologically validated COPD for the final analysis.

Data collection and laboratory methods

Enrolled COPD patients were identified from the outpatient department and inpatient wards of the Medicine Department. Trained researchers administered a standardized, structured proforma to collect demographic data (name, age, gender, residential location), detailed clinical history (COPD diagnosis date, disease duration, smoking history , current medications), and disease severity indicators. Following an overnight fast of at least 10 hours, fasting venous blood samples were collected into sterile tubes. Samples were immediately transported to the hospital biochemistry laboratory and processed within 2 hours of collection via centrifugation at 3000 rpm for 10 minutes.

Lipid profile quantification

Serum Lipid profile parameters were measured using enzymatic colorimetric methods on an automated analyzer in accordance with manufacturer specifications and Clinical Laboratory Standards Institute (CLSI) guidelines. Measured parameters included total cholesterol, HDL-cholesterol, LDL-cholesterol (calculated using Friedewald equation: LDL = Total cholesterol – HDL – (TG/5)), VLDL-cholesterol (calculated as triglycerides/5), and fasting triglycerides. All measurements were performed in duplicate, and quality control protocols were maintained using standard calibration materials.

Atherogenic Index of Plasma Calculation

The Atherogenic Index of Plasma was calculated using the established formula: AIP = $\log_{10}$(Triglycerides/HDL-cholesterol), where triglycerides and HDL-cholesterol values are expressed in mg/dL [8,9]. Patients were subsequently stratified into three atherogenic risk categories based on previously published cutoff values [12]: low cardiovascular risk (AIP <0.21), moderate cardiovascular risk (AIP 0.21–0.5), and high cardiovascular risk (AIP >0.5). This stratification approach enables rapid identification of individuals with atherogenic dyslipidemia phenotypes that confer disproportionate atherosclerotic risk despite potentially normal individual lipid parameters.

Statistical analysis

Data were compiled and analyzed using SPSS statistical software (Version 25.0, IBM Corporation). Data normality was assessed using the Shapiro-Wilk test. Descriptive statistics included means with standard deviations for continuous variables and frequencies with percentages for categorical variables. Chi-square test of independence was employed to assess associations between categorical variables (COPD duration categories, smoking status, gender, residential location) and lipid profile abnormalities. Pearson correlation coefficient analysis was used to quantify linear associations between continuous variables (COPD duration in years and lipid parameters including AIP).

Results

Demographic characteristics

A total of 100 COPD patients participated in this investigation. The study cohort demonstrated the following characteristics: mean age 64.8 ± 10.2 years (range 37-90 years), predominance of male participants (n=75, 75%), and urban residence in 86% of participants (n=86). Age distribution included: ≤50 years (n=9, 9%), 51-70 years (n=63, 63%), and >70 years (n=28, 28%). Smoking history was documented in 75% of participants (n=75 smokers vs. n=25 non-smokers), with 68 patients reporting current tobacco smoking and 7 reporting former smoking exposure. Sociodemographic characteristics of participants are mentioned in Table 1.

Atherogenic Index of Plasma Distribution and Cardiovascular Risk Stratification

The primary novel finding of this investigation was the characterization of atherogenic dyslipidemia phenotypes in the COPD population using AIP (findings mentioned in Table 2). Mean AIP was 0.69 ± 0.39 (range: -0.08 to 1.88), substantially elevated compared to published reference values for the general population. Risk stratification analysis revealed alarming prevalence of high-risk atherogenic profiles: 60% of participants (n=60) demonstrated high cardiovascular risk (AIP >0.5), 31% (n=31) exhibited moderate risk (AIP 0.21-0.5), and only 9% (n=9) fell into the low-risk category (AIP <0.21). The predominance of high atherogenicity profiles indicates that COPD patients harbor a lipid particle composition enriched in small, dense lipoprotein particles characteristic of metabolic dysfunction.

Association between COPD duration and atherogenic dyslipidemia

Pearson correlation analysis revealed significant positive associations between COPD disease duration and indices of atherogenic dyslipidemia. Specifically, COPD duration demonstrated strong positive correlation with AIP ($r=0.456$, $p<0.001$), indicating progressive atherogenic lipid profile evolution with advancing disease duration. This relationship was paralleled by significant positive correlations with total cholesterol ($r=0.554$, $p<0.001$), VLDL-cholesterol ($r=0.421$, $p=0.003$), LDL-cholesterol ($r=0.405$, $p<0.001$), and triglycerides ($r=0.341$, $p<0.001$). Conversely, COPD duration showed inverse correlation with HDL-cholesterol ($r=-0.173$, $p=0.089$), though this relationship did not achieve statistical significance. Chi-square contingency analysis stratified by COPD disease duration categories (<5 years, 5-10 years, 10-15 years, >15 years) confirmed significant associations for total cholesterol ($\chi^2=52.25$, $p<0.001$), VLDL-cholesterol ($\chi^2=13.74$, $p=0.003$), LDL-cholesterol ($\chi^2=36.82$, $p<0.001$), and triglycerides ($\chi^2=32.04$, $p<0.001$). This duration-dependent pattern is consistent with progressive metabolic derangement across the disease course, though temporal causality and mechanistic pathways remain to be established through prospective longitudinal studies (Table 3 and Figure 1).

Smoking status and lipid phenotype associations

The cohort demonstrated a high prevalence of tobacco exposure: 68% were current smokers (n=68) while 32% were non-smokers (n=32). Chi-square analysis revealed that smoking status

was significantly associated with elevated VLDL-cholesterol (χ^2=9.22, p=0.002), with smokers demonstrating higher prevalence of VLDL ≥30 mg/dL. However, smoking status did not demonstrate statistically significant associations with total cholesterol (p=0.245), LDL-cholesterol (p=0.057), triglycerides (p=0.103), or HDL-cholesterol (p=0.317). These findings suggest that smoking modulates one component of the atherogenic dyslipidemia phenotype, specifically increasing hepatic production of VLDL particles (Table 4).

Sociodemographic associations with lipid phenotypes

Chi-square analysis of sociodemographic associations (Table 5) revealed that urban residence was significantly associated with higher HDL-cholesterol levels (χ^2=6.04, p=0.048), potentially reflecting greater dietary diversity, improved healthcare access, or differential lifestyle factors in urban populations. Age and gender demonstrated no significant associations with lipid profile abnormalities or AIP risk stratification (all p>0.05). The lack of gender-specific differences contrasts with some published reports and may reflect this study's relatively small female sample size (n=25, 25%) compared to males [13].

Assessing independent predictors of high cardiovascular risk

To evaluate the independent contribution of disease chronicity to metabolic and cardiovascular risk, a multivariable hybrid logistic regression model was constructed using the column architecture established in our previous formal reporting (Table 6). The clinical outcome was defined as high atherogenic risk, operationalized as an Atherogenic Index of Plasma (AIP) greater than 0.50. Independent predictors entered simultaneously into the regression equation included COPD duration, patient age, smoking status, gender, and demographic residency (urban vs. rural). The final multivariable model demonstrated robust overall significance (Omnibus χ^2 = 20.893, p = 0.001) and high calibration quality with an excellent fit to the clinical data (Hosmer-Lemeshow p = 0.521), successfully accounting for 25.9% of the variance in atherogenic risk distribution (Nagelkerke R^2 = 0.259).

After adjusting simultaneously for all potential clinical and sociodemographic confounding factors, COPD duration emerged as an exceptionally powerful and highly significant independent predictor of high-risk atherogenic profiling (β = 0.2374, SE = 0.0688, Wald χ^2 = 11.898, p < 0.001). Specifically, for every additional year of documented COPD duration, an outpatient's odds

of crossing into the high cardiovascular risk tier climb by 26.8% (Adjusted Odds Ratio [aOR] = 1.268, 95% Confidence Interval [CI]: 1.108 – 1.451), completely independent of lifestyle habits, chronological aging, or geographic background. In contrast, active smoking status (aOR = 2.056, 95% CI: 0.697 – 6.064, p = 0.191), chronological age (aOR = 1.012, 95% CI: 0.960 – 1.066, p = 0.651), biological male gender (aOR = 0.443, 95% CI: 0.119 – 1.648, p = 0.225), and urban demographic residency (aOR = 1.013, 95% CI: 0.305 – 3.361, p = 0.983) failed to display independent statistical significance within this multivariable framework once disease duration was controlled.

Discussion

This investigation represents one of the first systematic characterizations of atherogenic dyslipidemia in COPD using the Atherogenic Index of Plasma as a clinical biomarker. The principal finding—that 60% of COPD patients demonstrate high-risk atherogenic profiles (AIP >0.5)—substantially exceeds published prevalence estimates of dyslipidemia in the general population and identifies COPD patients as a high-risk cohort for atherosclerotic cardiovascular disease. This elevated atherogenicity prevalence is consistent with the well-documented systemic inflammatory state in COPD and the progressive dysregulation of lipid metabolism accompanying COPD progression.

A notable methodological strength of this investigation is the capture of disease duration data, which provides temporal ordering within a cross-sectional framework the correlation between years since COPD diagnosis and current lipid atherogenicity metrics suggests a potential temporal gradient, though the cross-sectional nature of the study precludes establishing true causality or longitudinal disease progression. Specifically, the significant positive correlation between disease duration and AIP ($r=0.456$, $p<0.001$) indicates that lipid derangement intensifies with advancing years of disease, supporting a duration-dependent or progressive phenotype. This relationship is particularly noteworthy because it indicates that cardiovascular risk escalates in parallel with pulmonary disease progression, creating a cumulative hazard scenario for COPD patients. The mechanistic basis for this association likely involves chronic systemic inflammation characterized by elevated interleukin-6, tumor necrosis factor-alpha, and C-reactive protein, which directly modulate hepatic lipid metabolism and lipoprotein particle composition [14]. However, this does

not establish causality and remains vulnerable to survival bias (healthier long-duration patients surviving to enrollment), secular confounding (diagnostic practice changes across decades), and unmeasured confounders (inflammatory status, genetic predisposition, medication evolution). Future prospective cohort studies with well-characterized baseline COPD severity and inflammatory biomarkers would be required to definitively establish causal mechanisms.

Traditional lipid profiling in the 100 COPD patients revealed that 71% demonstrated low HDL-cholesterol (<40 mg/dL), which independently confers cardiovascular risk. However, this parameter alone would miss the critical information that 60% of patients had high AIP, indicating atherogenic particle composition. This discordance highlights the potential utility of composite lipid indices like AIP in evaluating the atherogenic lipid profiles of COPD patients. AIP integrates both the triglyceride elevation and HDL deficiency characteristic of metabolic dysfunction, providing a more complete phenotypic assessment [15].

The observed association between smoking status and elevated VLDL-cholesterol (p=0.002) aligns with established pathophysiology. Tobacco smoke exposure induces hepatic triglyceride synthesis and impairs lipolytic activity, resulting in VLDL accumulation [16]. However, the lack of significant associations between smoking and HDL-cholesterol or total cholesterol in this cohort differs from some published reports. This discrepancy may reflect unmeasured confounders, such as specific cigarette types (bidis vs. cigarettes), consumption quantity, or duration of smoking exposure. Future investigations employing standardized smoking quantification indices (pack-years) would clarify these relationships [17-19].

The protective association between urban residence and HDL-cholesterol levels (p=0.048) may reflect urban residents' greater access to healthier dietary options, better healthcare infrastructure enabling more effective disease management, or differential leisure physical activity patterns. Conversely, rural populations may experience greater dietary restrictions and less systematic healthcare engagement, potentially explaining observed differences. However, this cross-sectional association prevents causality determination, and longitudinal studies would be required.

The integration of a multivariable hybrid logistic regression model substantially reinforces the scientific rigor. The most striking finding is that after mathematically controlling for age, gender, smoking, and urban-rural residence, the duration of COPD stands out as a highly robust,

independent predictor of elevated atherogenic risk ($p < 0.001$). This indicates that the chronicity of respiratory disease acts as an independent metabolic insult over time, separate from the acute effects of active tobacco use or baseline demographic characteristics.

The structural validity of this finding is clearly illustrated by the Wald Chi-Square statistics, where the metric for COPD duration (11.898) overwhelmingly dominates all other sociodemographic variables, which collapsed well beneath standard significance thresholds. Notably, the newly introduced geographic covariate (Urban vs. Rural) was completely neutral in the multivariable environment (aOR = 1.013, $p = 0.983$). This negates the confounding hypothesis that regional lifestyle variances, healthcare access discrepancies, or distinct localized exposures were secretly driving the observed dyslipidemia. Similarly, the mitigation of age and gender as independent predictors strongly indicates that the long-term systemic pathophysiological strains of COPD—likely driven by chronic low-grade systemic inflammation, persistent hypoxia, and secondary metabolic exhaustion—gradually degrade the patient's lipid environment into a high-risk atherogenic state. By proving that atherogenic progression tracks tightly with disease duration independent of standard demographic profiles, these findings provide a compelling clinical mandate for early cardiovascular screening and targeted lipid management within the COPD outpatient registry .

Clinical implications of this investigation are substantial. The high prevalence of atherogenic dyslipidemia in COPD patients warrants consideration of routine AIP-based screening integrated into COPD management protocols. AIP offers distinct advantages: (1) calculated from routine lipid panel measurements at negligible cost; (2) continuously distributed with well-established risk cutoff values; (3) independent prediction of cardiovascular endpoints in multiple populations; (4) potentially modifiable through therapeutic intervention. Current COPD management guidelines inadequately address cardiovascular risk mitigation; incorporation of AIP-based screening could enable earlier identification of high-risk patients for intensive lipid-modifying therapy or cardiovascular surveillance.

Future investigations should employ prospective cohort designs with longitudinal follow-up to establish temporal relationships and cardiovascular event prediction. Incorporation of inflammatory biomarkers (high-sensitivity C-reactive protein, interleukin-6), formal spirometric grading, and comprehensive smoking history quantification would strengthen mechanistic

understanding [20]. Multi-center investigations encompassing diverse geographic regions and socioeconomic strata would enhance generalizability. Clinical trial evaluation of whether AIP-guided intensive lipid-modifying therapy improves cardiovascular outcomes in COPD patients remains an important knowledge gap.

Limitations

- Design and Scope: The single-center, cross-sectional nature of this study limits geographic generalizability and precludes the determination of temporal or causal relationships.
- Data Constraints: The most important limitation of this study is the absence of objective post-bronchodilator spirometric confirmation (FEV1/FVC), which is the standard for COPD diagnosis in major guidelines. Consequently, our reliance on clinical-radiological surrogate confirmation must be viewed as a constraint. Furthermore, the lack of consistent Body Mass Index (BMI) data prevented adjustment for nutritional status and obesity
- Absence of a Comparative Cohort: The lack of a non-COPD control group prevents a direct comparison of lipid abnormalities against the general population, limiting the assessment of COPD-specificity for the observed patterns.
- Sample Limitations: The sample size (n=100) and female predominance may restrict subgroup analysis. Additionally, lifestyle variables such as diet, physical activity, and genetic factors were not assessed.
- Selective Bias: While rigorous exclusion of metabolic comorbidities strengthened internal validity, this selective approach may have isolated COPD-specific changes that differ from real-world, multimorbid clinical populations.
- Furthermore, due to sample size and data constraints, multivariate analysis adjusting for potential confounders (such as detailed smoking pack-years or BMI) and the calculation of confidence intervals were not performed. These remain limitations of the current analytical approach, and findings should be interpreted as associative rather than predictive.

Conclusions

This investigation demonstrates that atherogenic dyslipidemia, quantified by the Atherogenic Index of Plasma, is highly prevalent in COPD patients and correlates with disease progression.

The high prevalence of elevated AIP (60% with AIP >0.5) identifies COPD as a population at substantial cardiovascular risk. The progressive increase in AIP with advancing COPD duration suggests that cardiovascular risk escalates proportionally with pulmonary disease deterioration. AIP provides a cost-effective, rapidly calculated metric that integrates multiple lipid parameters to help identify altered lipid profiles associated with cardiovascular risk. Identifying elevated AIP in routine COPD management can highlight patients with atherogenic lipid profiles, emphasizing the need for comprehensive cardiovascular evaluation. These associative findings suggest AIP is a useful metric for evaluating lipid derangement in COPD and warrant prospective validation through longitudinal cohort investigations and clinical trial evaluation.

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Table 1. Socio-demographic characteristics stratified by Atherogenic Index of Plasma risk category (n=100).

AIP risk category	N	Mean age (SD)	Males, n (%)	Females, n (%)	Urban, n (%)	Rural, n (%)
Low Risk (AIP <0.21)	9	62.6 (±9.4)	7 (77.8)	2 (22.2)	8 (88.9)	1 (11.1)
Moderate Risk (AIP 0.21-0.5)	31	65.0 (±11.2)	27 (87.1)	4 (12.9)	25 (80.6)	5 (16.1)
High Risk (AIP >0.5)	60	65.8 (±9.4)	41 (68.3)	19 (31.7)	53 (88.3)	7 (11.7)

Table 2. Lipid profile parameters stratified by Atherogenic Index of Plasma risk category.

Lipid Parameter	Low Risk (AIP <0.21)	Moderate Risk (AIP 0.21-0.5)	High Risk (AIP >0.5)
Total Cholesterol (mg/dL)	166.7±25.2	142.5±43.1	160.9±58.0
HDL-Cholesterol (mg/dL)	57.1±14.0	38.4±9.4	20.6±10.8
LDL-Cholesterol (mg/dL)	94.9±27.1	86.3±34.3	103.4±42.5
VLDL-Cholesterol (mg/dL)	14.7±4.6	19.9±5.8	32.7±9.8
Triglycerides (mg/dL)	73.4±22.8	97.1±23.6	168.7±84.1
Atherogenic Index of Plasma	0.12±0.05	0.37±0.09	0.91±0.28

Table 3. Association between COPD duration and lipid profile parameters (n=100).

Lipid Parameter	5-10 Years	10-15 Years	>15 Years	Correlation (r, p-value)
Total Cholesterol (mg/dL)	171.4±55.2	184.5±61.2	195.3±64.8	r=0.554, p<0.001*
VLDL-Cholesterol (mg/dL)	28.5±10.2	35.2±12.5	42.1±14.3	r=0.421, p=0.003*
LDL-Cholesterol (mg/dL)	98.6±39.4	115.4±43.8	128.7±48.2	r=0.405, p<0.001*
HDL-Cholesterol (mg/dL)	29.2±11.5	24.8±10.2	22.1±9.6	r=-0.173, p=0.089
Atherogenic Index of Plasma	0.58±0.32	0.76±0.38	0.92±0.41	r=0.456, p<0.001*

*p<0.05, statistically significant.

Table 4. Association between smoking status and lipid profile (n=100).

Lipid Parameter	Smokers (n=68)	Non-Smokers (n=32)	Chi-Square Test (p-value)
Total Cholesterol ≥200 mg/dL	21 (30.9%)	5 (15.6%)	χ^2=2.81, p=0.245
VLDL-Cholesterol ≥30 mg/dL	32 (47.1%)	5 (15.6%)	χ^2=9.22, p=0.002*
LDL-Cholesterol ≥130 mg/dL	23 (33.8%)	3 (9.4%)	χ^2=7.49, p=0.057
HDL-Cholesterol <40 mg/dL	49 (72.1%)	22 (68.8%)	χ^2=2.29, p=0.317
High AIP Risk (AIP >0.5)	44 (64.7%)	16 (50.0%)	χ^2=3.82, p=0.051

*p<0.05, statistically significant.

Table 5. Chi-square test results: sociodemographic factors and lipid parameters (n=100)

Demographic variable	Total Cholesterol	VLDL-Cholesterol	LDL-Cholesterol	HDL-Cholesterol	AIP Risk
Age Groups (≤50, 51-70, >70)	p=0.261	p=0.244	p=0.993	p=0.918	p=0.382
Gender (Male vs. Female)	p=0.408	p=0.199	p=0.747	p=0.563	p=0.456
Residence (Urban vs. Rural)	p=0.165	p=0.462	p=0.531	p=0.048*	p=0.083

*p<0.05, statistically significant.

Table 6. Multivariable hybrid logistic regression analysis assessing independent predictors of high cardiovascular risk (AIP>0.50).

Independent predictor variables	Regression coefficient (β)	Standard error (SE)	Wald χ^2	aOR	95% CI	p-value
Intercept (Constant)	-1.9705	2.1643	0.829	0.139	—	0.363
COPD Duration (Years)	0.2374	0.0688	11.898	1.268	[1.108 – 1.451]	<0.001*
Patient Age (Years)	0.0121	0.0267	0.205	1.012	[0.960 – 1.066]	0.651
Smoking Status (Smoker vs. Non)	0.7208	0.5518	1.706	2.056	[0.697 – 6.064]	0.191
Gender (Male vs. Female)	-0.8149	0.6702	1.475	0.443	[0.119 – 1.648]	0.225
Demography (Urban vs. Rural)	0.0128	0.6121	0.000	1.013	[0.305 – 3.361]	0.983

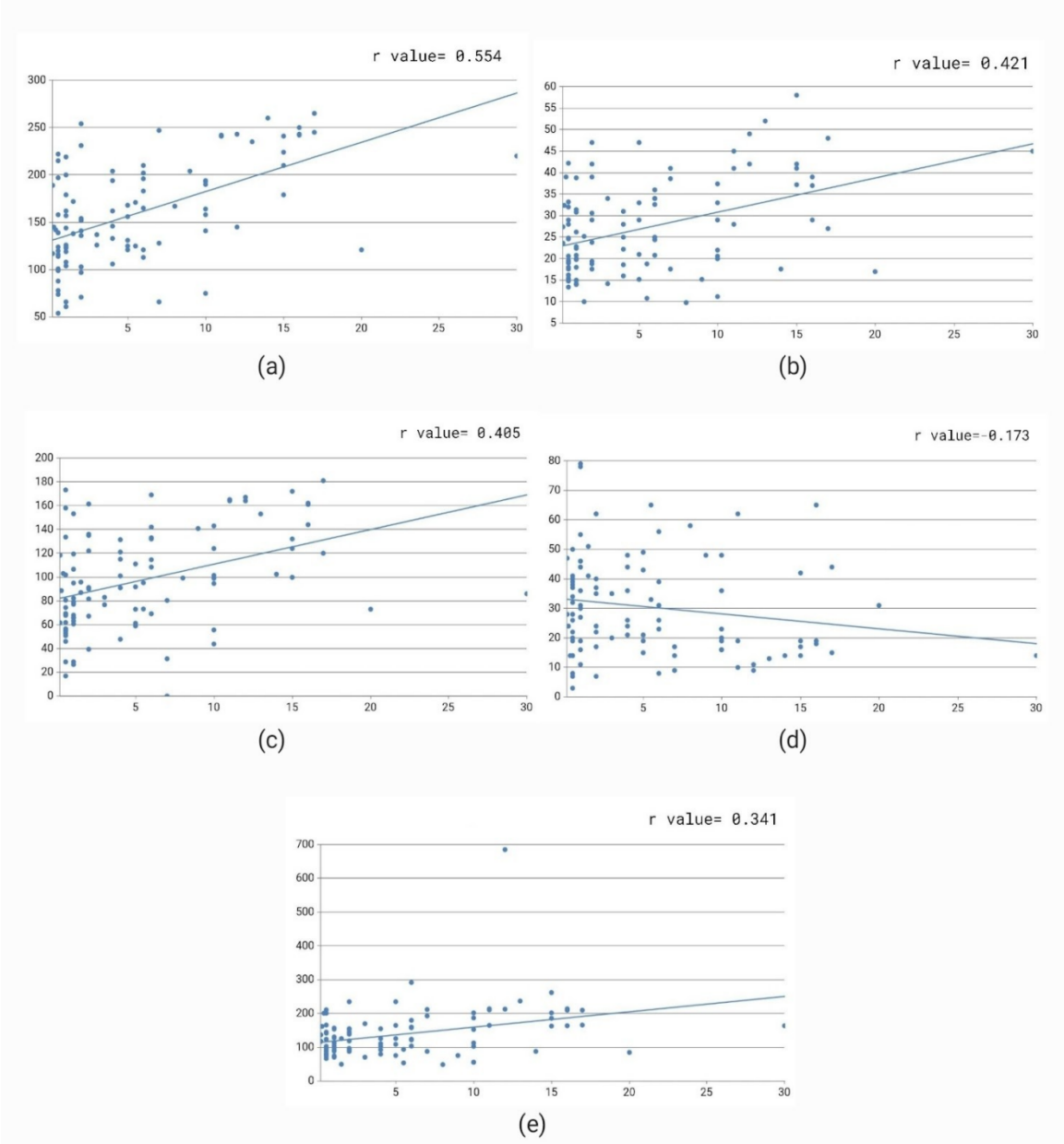

Figure 1. Pearson correlation graphs (with r values) showing association between COPD duration (in X axis) and lipid parameters (in Y axis) (a) total cholesterol, (b) VLDL-cholesterol, (c) LDL-cholesterol, (d) HDL-cholesterol, (e) triglycerides.