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Prediction of Atherosclerosis by Observing CIMT According to GOLD Severity Assessment in COPD Patients

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ABSTRACT

Background: Atherosclerosis, a chronic vascular condition, involves plaque buildup in arterial walls, leading to restricted blood flow and cardiovascular diseases. Early detection of atherosclerosis is possible through carotid intima-media thickness (CIMT) measurement. Chronic Obstructive Pulmonary Disease (COPD) exacerbations increase systemic inflammation, worsening vascular damage. CIMT consistently correlates with COPD severity (GOLD stages), highlighting its value as a marker for cardiovascular risk and guiding early therapeutic interventions such as lifestyle changes and medication.

Methods: This cross-sectional study, conducted at MKCG MCH, Odisha (June 2022–June 2024), examined the correlation between CIMT and COPD severity. Stable COPD patients meeting criteria such as smoking history or biomass fuel exposure were included. CIMT was measured using B-mode ultrasonography, with ≥0.8 mm indicating increased thickness. Exclusion criteria covered comorbidities and other confounding conditions. Statistical analysis employed t-tests and chi-square tests, with significance set at $p<0.05$.

Results: Among 103 COPD patients, 38.83% exhibited atherosclerotic plaques with significantly higher CIMT values on the right (1.42±0.25 mm vs. 0.36±0.33 mm; p=0.001) and left sides (1.31±0.31 mm vs. 0.36±0.31 mm; p=0.001). Cholesterol levels were higher in the plaque group (134.32±33.00 mg/dl vs. 123.81±15.70 mg/dl; p=0.03). CIMT-right demonstrated superior diagnostic performance (AUC 0.988, sensitivity 97.06%, specificity 100%).

Conclusion: The study concludes that increasing COPD severity, as per GOLD criteria, strongly correlates with elevated CIMT, indicating progressive atherosclerosis.

Key-words: Carotid intima-media thickness (CIMT), COPD, Atherosclerosis, Vascular disease, Pulmonary

INTRODUCTION

Atherosclerosis is a chronic vascular condition which is characterized by lipid accumulation and thickening of the arterial wall which is a major reason for cardiovascular disorders, such as myocardial infarction and cerebrovascular accidents [1].

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Atherosclerosis is a chronic and progressive disease in which arteries become narrowed due to the buildup of plaques composed of lipids, cholesterol, calcium, and cellular wastes within their walls. This condition is a leading cause of cardiovascular diseases including atherosclerosis [2]. Atherosclerosis starts with endothelial dysfunction where endothelium is damaged by high blood pressure, increased low-density lipoprotein (LDL) cholesterol, smoking, or oxidative stress. This damage disrupts the endothelium's protective barrier, making it more permeable to lipoproteins like LDL cholesterol, which infiltrate the arterial wall. Then, LDL cholesterol particles become oxidized, triggering an inflammatory response. The immune system recognizes oxidized LDL as

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harmful and recruit's monocytes to the site of injury. These monocytes penetrate the arterial wall and differentiate into macrophages, which engulf the oxidized LDL, forming foam cells. The accumulation of foam cells leads to the formation of fatty streaks, the earliest visible lesions of atherosclerosis. Meanwhile, the presence of oxidized LDL and foam cells stimulates the release of inflammatory cytokines and growth factors, perpetuating a chronic inflammatory environment that further damages the arterial wall. Smooth muscle cells from the middle layer of the artery, known as the media, migrate to the intima (the inner layer), where they proliferate and produce extracellular matrix proteins like collagen, forming a fibrous cap over the growing plaque. This fibrous cap helps stabilize the plaque but also narrows the arterial lumen, restricting blood flow (Fig. 1) [3].

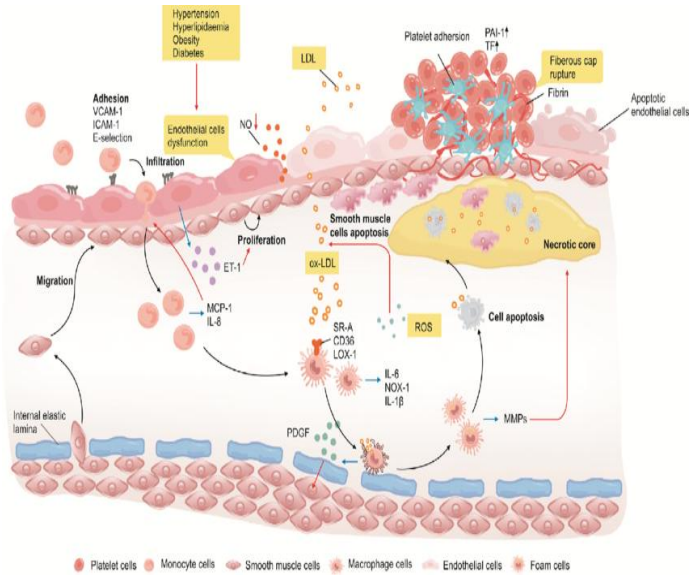

Fig. 1: Mechanism of the development of atherosclerosis

Over time, plaques can grow larger as more lipids and inflammatory cells accumulate which in terms forms atherosclerosis. The rupture of a plaque exposes its contents to the bloodstream, and the aggregation of platelets results in the formation of a thrombus, or blood clot, which may partially or completely obstruct blood flow and result in acute events like heart attacks or strokes [4]. In the later stages of atherosclerosis, chronic hypoxia, and oxidative stress further promote vascular remodeling with enhanced arterial stiffness and increased lumen narrowing. Early atherosclerotic changes can be identified by non-invasive measuring like CIMT [5]. COPD is a chronic lung disease mainly caused by airflow limitation which causes breathing difficulties. The most common cause is long-term exposure to noxious particles and gases but the leading cause remains smoking. COPD includes two major conditions such as chronic bronchitis and emphysema. Chronic bronchitis is defined as the thickening of the bronchial tubes due to the increased production of mucus, which causes airway restriction. Emphysema involves the destruction of the alveolar walls, which causes the lungs' ability to transfer oxygen into the bloodstream. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) created several guidelines for labelling different stages of chronic obstructive pulmonary disease (COPD). It classifies COPD based on its lung function, and symptoms. There are currently four stages based on lung function [6] (Table 1).

Table 1: Stages of Lung Functions according to GOLD criteria

GOLD Stage	FEV1 (% Predicted)	Symptom Burden	Risk of Exacerbations	Management Recommendations
GOLD 1	≥80%	Mild	Low	Smoking cessation, influenza vaccines

GOLD 2	50–79%	Moderate	Low	Bronchodilator therapy
GOLD 3	30–49%	Severe	High	Inhaled corticosteroids, combination therapy
GOLD 4	<30%	Very Severe	High	Advanced therapies, long-term oxygen

CIMT is a non-invasive diagnostic procedure that measures the thickness of the intimal and medial layers of the carotid artery wall and can potentially be used as an important marker for detecting atherosclerosis. It is measured by using high-resolution B-mode ultrasonography, which gives detailed imaging of the carotid arteries situated in the neck. This measurement shows early structural changes in the arterial wall, like thickening due to lipid accumulation, inflammatory cell infiltration, and vascular remodeling [7]. In COPD, CIMT has recently been an important tool for identifying cardiovascular risk. GOLD classification is based on FEV (forced respiratory volume) values, symptoms, and exacerbation risk, which provides a way by which COPD relates to CVD [8]. COPD exacerbations increase systemic inflammation, which increases the levels of pro-inflammatory cytokines such as IL-6 and TNF-α as well as oxidative stress. These trigger endothelial dysfunction and vascular damage. CIMT in patients with COPD shows a consistent increase in the thickness of the arterial wall with progressing GOLD stages [9]. Therefore, it shows a clear relationship between COPD & cardiovascular diseases. Patients of GOLD stage 1 or 2, who have mild or moderate COPD symptoms, may show a slight increase in CIMT. Patients within GOLD stages 3 and 4, which are severe or very severe COPD, may show significantly higher levels of CIMT values. The association observed between CIMT and the degree of GOLD provides us with a link that can identify the early development of atherosclerosis. CIMT acts as a marker, which helps identify cardiovascular risk and helps us develop early therapeutic measures such as lipid-lowering therapy, anti-hypertensive treatment, and lifestyle changes [10].

MATERIALS AND METHODS

Research Design- This is a cross-sectional design that investigates the correlation between CIMT and the severity of COPD, as well as associated blood markers. This study was conducted at the Department of Respiratory Medicine, MKCG MCH, Berhampur, Odisha, from June 2022 to June 2024. The study enrolled stable COPD patients meeting clear inclusion criteria, such as smoking history or biomass fuel exposure, and excluded those with confounding comorbidities or conditions. COPD diagnosis was established by employing spirometry, followed by vast clinical evaluations, blood marker analyses, and imaging studies. The CIMT was measured employing carotid B-mode duplex ultrasonography, with a threshold of ≥0.8 mm showing increased thickness. The study adhered to standardized protocols to provide reproducibility and reliability, analyzing data to evaluate the relationship between CIMT, COPD severity, and biomarkers, thereby providing an understanding of cardiovascular risks in COPD patients.

Inclusion Criteria

- All COPD patients without exacerbation within the last 6 weeks.
- All patients with a history of smoking for at least 10 pack years.
- All COPD patients with exposure to biomass fuels.
- All patients who give informed consent.

Exclusion Criteria

- Patients with a history of hypertension and diabetes mellitus before the onset of COPD.
- All COPD patients with obstructive lung diseases and parenchymal disorders.
- All COPD patients with pregnancy, hepatic or renal abnormalities, on anticoagulants or statins,
- All COPD patients with a history of myocardial infarction, congenital heart disease, valvular heart disease, atrial fibrillation, vasculitis, coronary catheterization, and carotid artery surgery.
- All COPD patients with extrapulmonary infective, inflammatory, and malignancy disorders.
- All COPD patients with HIV.

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Statistical Analysis- Data were analyzed using descriptive and inferential statistics. Comparisons between groups were made using t-tests for continuous variables and chi-square tests for categorical data. A p-value < 0.05 was considered statistically significant. Analyses were performed using SPSS software.

Ethical Approval- The study was approved by the Ethical Committee of the hospital.

RESULTS

Table 2 presents the study's demographic and socioeconomic profile of 103 COPD patients. It shows that most patients were aged between 51 and 70 (58.26%), with the highest ratio in the 61-70 age group (30.10%). Males accounted for 62.14% of the participants, while females made up 37.86%. Most patients (60.19%) belonged to the low socioeconomic status category, with the remainder in the middle class (39.81%). About the occupations, homemakers (26.21%), businessmen (25.24%), and farmers (24.27%) described the largest groups, followed by daily laborers, cooks, and masons. Smoking habits showed that 62.14% of patients had a history of smoking, with 34.95% being present smokers and 27.18% being ex-smokers, while 37.86% were non-smokers. These results show that COPD predominantly impacts middle-aged to older adults, is more familiar among males, is associated with lower socioeconomic status, and is significantly linked to smoking history.

Table 2: Demographic and socioeconomic details of the patient

Age group	No. of cases	Percentage (%)
31 to 40	2	1.94
41 to 50	12	11.65
51 to 60	29	28.16
61 to 70	31	30.10
71 to 80	21	20.39
81 to 90	7	6.80
91 to 100	1	0.97
Total	103	100
Gender		
Male	64	62.14
Female	39	37.86
Total	103	100
Socioeconomic status		
Low	62	60.19
Middle	41	39.81
Total	103	100
Occupation		
Home maker	27	26.21
Businessman	26	25.24
Farmer	25	24.27
Daily labourer	15	14.56
Cook	7	6.80
Mason	3	2.91
Total	103	100
Smoking		
Non-smoker	39	37.86
Current smoker	36	34.95
Ex-smoker	28	27.18
Total	103	100

Table 3 summarizes the clinical variables observed among 103 COPD patients. It highlights both general and respiratory symptoms and signs. General symptoms included swelling of the ankle in 50.49% of cases, fever in 30.10%, and raised jugular venous pressure (JVP) in 37.86%. Tachycardia was noticed in 54 patients (52.42%), with mean systolic and diastolic blood pressure at 117.75±5.26 mmHg and 75.90±11.07 mmHg. Among respiratory symptoms, cough and decreased vesicular breath sounds (VBS) were universal (100%), followed closely by breathlessness (99.03%) and wheezing (99.03%). Sputum expectoration was reported in 51.46%, while chest pain was less frequent (24.27%). Respiratory signs had intercostal indrawing in 62.14%, accessory muscle use in 87.38%, barrel chest in 49.51%, and obliterated cardiac/liver dullness in 43.69%. Crepitations and rhonchi were present in 24.27% and 65.05% of cases, which reflects significant respiratory compromise typical in COPD patients.

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Table 3: General and respiratory symptoms among patients

Clinical variables	No. of cases	Percentage	Clinical variables	No. of cases or Mean±SD	Percentage
General symptoms			General signs		
			Raised JVP	39	37.86%
Fever Swelling of Ankle	31 52	30.10% 50.49%	Tachycardia systolic BP diastolic BP Hypertension	54 117.75±5.26 75.90±11.07 28	52.42% 27.18%
			Tachypnea	99	96.11%
Respiratory symptoms			Respiratory signs		
			Intercostal indrawing	64	62.14%
Cough	103	100%	Accessory	90	87.38%
Breathlessness	102	99.03%	Muscle Use		
Sputum	53	51.46%	Barrel Chest	51	49.51%
Expectoration	53	51.46%	Obliterated		
Chest Pain	25	24.27%	Cardiac/Liver	45	43.69%
Wheeze	102	99.03%	Dullness		
-			Decreased VBS	103	100%
			Crepts	25	24.27%
			Ronchi	67	65.05%

Table 4 illustrates the study population's clinical parameters connected to atherosclerotic plaque, CIMT measurements, and GOLD staging. The atherosclerotic plaque was seen in 38.83% of cases, with mean CIMT values of 0.71±0.58 mm on the right and 0.67±0.54 mm on the left. GOLD staging showed that most participants were in Stage 2 (43.69%) or Stage 3 (52.43%), with only a small fraction in Stage 4 (3.88%). These results show a notable prevalence of atherosclerotic changes and advanced GOLD stages, which highlights the importance of considering CIMT and disease severity in the population.

Table 4: Clinical parameters connected to atherosclerotic plaque, CIMT measurements, and GOLD staging

Parameters	No. of cases	Percentage	Mean±SD	Atherosclerotic plaque
CIMT Right (mm)	40	38.83%	0.71±0.58	28
CIMT Left (mm)	38	36.89%	0.67±0.54	6
GOLD Staging	No. of cases	Percentage		

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2	45	43.69%
3	54	52.43%
4	4	3.88%
Total	103	100%

Table 5 compares clinical and biomarker characteristics between patients with atherosclerotic plaque (33.01%) and those without (66.99%). Statistically significant differences were observed in CIMT values, with higher mean CIMT for the right (1.42±0.25 mm vs. 0.36±0.33 mm; p=0.001) and left sides (1.31±0.31 mm vs. 0.36±0.31 mm; p=0.001) among patients with plaques. Cholesterol levels were also significantly higher in the plaque group (134.32±33.00 mg/dl vs. 123.81±15.70 mg/dl; p=0.03). Other variables, including age, gender distribution, smoking status, blood pressure, oxygen saturation (SpO_2), mMRC scale, CAT score, post-bronchodilator FEV1, GOLD staging, and ABE assessment, showed no significant differences between the two groups. Biomarkers such as CRP, LDH, RBS, urea, and LDL were also not significantly different, though CRP and RBS trended higher in the plaque group (p=0.06). These findings emphasize the association of higher CIMT and cholesterol levels with the presence of atherosclerotic plaques.

Table 5: Clinical and biomarker characteristics between patients with atherosclerotic plaque (33.01%) and those without

Measures	**Atherosclerotic Plaque n=34 (33.01%)**	**No Atherosclerotic plaque n=69 (66.99%)**	**t-test**	**p-value**
CIMT Right (mm)	1.42±0.25	0.36±0.33	16.52	0.001*
CIMT Left (mm)	1.31±0.31	0.36±0.31	14.89	0.001*
Age	64.62±11.12	63.36±12.17	0.51	0.61
Female	12 (30.77%)	27 (69.23%)	0.03	0.87
Male	22 (34.38%)	42 (65.62%)	0.03	0.87
Smoker	22 (34.37%)	42 (65.62%)	0.9	0.66
systolic BP	117.35±5.41	117.94±5.21	-0.53	0.60
diastolic BP	75.24±11.25	76.23±11.05	-0.43	0.67
SpO_2	89.68±2.32	89.65±2.45	0.05	0.96
mMRC scale	2.47±0.56	2.45±0.58	0.18	0.86
CAT score	27.97±3.82	29.78±5.00	-1.86	0.07
POST Bronchodilator FEV1(%)	52.47±7.89	51.61±8.16	0.51	0.61
GOLD staging			1.78	0.41
Stage 2 (n=45)	18 (40.00%)	27 (60.00%)	N/A	
Stage 3 (n=54)	15 (27.78%)	39 (72.22%)		
Stage 4 (n=4)	1 (25.00%)	3 (75.00%)		

ABE assessment			0.49	0.49
B	16 (38.10%)	26 (61.90%)	N/A	
E	18 (29.51%)	43 (70.49%)	-	-
Biomarkers				
TLC	8447.44±4619.99	9383.67±3725.77	-1.11	0.27
CRP (mg/dl)	13.63±13.36	10.22±4.81	1.89	0.06
LDH U/L	424.29±149.60	429.09±145.01	-0.16	0.88
CHOLESTEROL (mg/dl)	134.32±33.00	123.81±15.70	2.2	0.03*
LDL (mg/dl)	87.88±28.73	82.35±24.48	1.02	0.31
RBS (mg/dl)	107.50±11.46	102.61±12.77	1.89	0.06
UREA (mg/dl)	41.68±9.21	40.71±7.35	0.58	0.57

Table 6 evaluates diagnostic parameters for CIMT (right and left), GOLD staging, and ABE assessment in detecting specific clinical outcomes. CIMT-right demonstrated excellent diagnostic performance with an AUC of 0.988 (95% CI: 0.943–0.999), sensitivity of 97.06% (84.7–99.9%), and specificity of 100% (94.8–100%), alongside perfect positive predictive value (PPV: 100%) and a high negative predictive value (NPV: 98.57%). CIMT-left also performed well, with sensitivity and specificity at 97.06% (84.7–99.9%) and 98.55% (80.2–95.8%), respectively, and high PPV (97.06%) and NPV (98.55%). GOLD staging showed lower sensitivity (67%) and specificity (69%), with moderate PPV (78%) and low NPV (60%). ABE assessment performed slightly better, with sensitivity at 72%, specificity at 78%, and moderate predictive values (PPV: 68%, NPV: 70%). These findings highlight the superior diagnostic value of CIMT measures, especially the right side, compared to GOLD and ABE assessments.

Table 6: Diagnostic parameters for CIMT (right and left), GOLD staging, and ABE assessment

Parameter	CIMT Right	CIMT Left	GOLD Staging	ABE Assessment
Area under curve (AUC)	0.988 (0.943-0.999)	-	-	-
Youden Index	0.97	-	-	-
Sensitivity	97.06% (84.7-99.9%)	97.06% (84.7-99.9%)	67% (31-83%)	72% (31-84%)
Specificity	100% (94.8-100%)	98.55% (80.2-95.8%)	69% (28-71%)	78% (51-84%)
Positive Predictive Value (PPV)	100% (94.8-100%)	97.06% (85.0-99.4%)	78% (18-80%)	68% (25-73%)
Negative Predictive Value (NPV)	98.57% (92.3-99.7%)	98.55% (92.2-99.7%)	60% (45-73%)	70% (58-80%)
Positive Likelihood Ratio (PLR)	0	-	-	-
Negative Likelihood Ratio (NLR)	0.02	-	-	-

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DISCUSSION

COPD severity, which is defined by GOLD criteria, shows a clear connection for the progression of atherosclerosis by measuring CIMT levels. The increase in CIMT values along with advancing GOLD stages shows us the development of atherosclerosis in patients. As the GOLD stage progresses, CIMT values are also increased along with them which serves as a major marker for the development of atherosclerosis. Several clinical studies provided us with those indications.

The study from Firincioglulari *et al.* shows a significant difference between GOLD stages of COPD in carotid thickness. Levels of CIMT were high in the patients who were in the early stages of COPD [11]. Another study by Trilochan et al showed that 82.35% of cases from the GOLD stages of COPD had increased levels of CIMT, in which 81.82% of cases were from the GOLD-2 stages and 66% of cases were from the GOLD-1 STAGE. GOLD-4 stage also showed 62.5% of cases had increased CIMT. They also found that the CIMT level is the highest in the GOLD-3 stage [12]. Chindhi *et al.* also concluded a similar outcome in their study, where 69.7% were from GOLD-1, 58.8% were from GOLD-2, 70.5% were from GOLD-3, and 71% were from GOLD-4 patients had increased CIMT [13]. A study on 610 patients by Gülbas *et al.* also revealed positive correlations between increasing CIMT levels and GOLD stages. Thus, it supports atherosclerosis risk in COPD patients [14]. The study from Dursunoglu *et al.* also shows the mean Gensini score in patients with COPD was higher in COPD patients & it is progressively higher along with GOLD stages which can further support CIMT values for detecting atherosclerosis [15]. Nevzorova et al studies show us that Symptoms of atherosclerosis are revealed at all stages of COPD & the highest level of CIMT is noted at the GOLD IV with increasing correlation throughout the GOLD stages [16]. However, the studies from Pobeha *et al.* and Hafez *et al.* show a null difference in CIMT with COPD severity across all the GOLD stages which was probably due to the build-up of atherosclerosis on the carotid artery that might have started earlier during the development of COPD [13,17].

Chronic hypoxia and hypercapnia in GOLD 3 and 4 patients increase vascular smooth muscle proliferation and narrowing arteries, which further elevates CIMT values. The use of CIMT as a predictive marker for the development of atherosclerosis in COPD populations is strongly evident. Its non-invasive nature along with its ability to detect vascular changes proves that CIMT can be used as an invaluable tool for the detection of atherosclerosis.

This study substantiates the association between increasing COPD severity and elevated CIMT, highlighting the interdependence of pulmonary and vascular health. Recognizing atherosclerosis as a critical comorbidity in COPD necessitates a paradigm shift toward multidisciplinary care models that address both respiratory and cardiovascular risks. By adopting such approaches, clinicians can enhance patient outcomes and mitigate the substantial burden of COPD and its systemic complications.

CONCLUSIONS

The study has concluded, there is a strong association between the severity of COPD, as classified by the GOLD criteria, and the progression of atherosclerosis, evidenced by increased CIMT. The findings reveal that CIMT is a reliable, non-invasive marker for early detection of vascular changes in COPD patients, highlighting its utility in predicting cardiovascular risks. As COPD progresses to more severe stages, systemic inflammation, endothelial dysfunction, and oxidative stress significantly contribute to the development of atherosclerosis. These results reinforce the importance of integrating cardiovascular risk assessment into the management of COPD to improve patient outcomes.

Future clinical practices should focus on multidisciplinary approaches that address both respiratory and cardiovascular health. Early interventions, including lifestyle changes, pharmacological treatments, and regular monitoring of CIMT, can play a crucial role in mitigating the systemic complications of COPD. This comprehensive care model could ultimately reduce the overall burden of disease and enhance the quality of life for affected individuals.

CONTRIBUTION OF AUTHORS

Research concept- N. Akshaya, Madhumita Nayak
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