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Association Between Dyspnea Severity, Functional Exercise Capacity, and Carotid Intima-Media Thickness in Chronic Obstructive Pulmonary Disease

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ABSTRACT

Background: Dyspnea and reduced exercise capacity are hallmark features of chronic obstructive pulmonary disease (COPD). Emerging evidence suggests that systemic vascular changes, particularly increased carotid intima-media thickness (CIMT), may contribute to functional limitation.
Objective: To evaluate the association between dyspnea severity (mMRC), functional exercise capacity (6MWD), and CIMT in patients with COPD.
Methods: We conducted a cross-sectional study on 31 stable COPD patients. Demographics, spirometry, GOLD staging, CIMT (measured by high-resolution B-mode ultrasonography), mMRC dyspnea grade, and six-minute walk distance (6MWD) were recorded. Correlations between CIMT, mMRC, and 6MWD were analyzed using Pearson's correlation coefficient.
Results: Patients with higher dyspnea scores (mMRC ≥3) demonstrated significantly higher CIMT (2.1 ± 0.6 mm) and lower 6MWD (320 ± 80 m) compared with those with mMRC ≤1 (CIMT 0.7 ± 0.2 mm, 6MWD 500 ± 60 m; $p<0.01$). CIMT positively correlated with mMRC ($r = 0.61$, $p<0.01$) and inversely with 6MWD ($r = –0.58$, $p<0.01$).
Conclusion: Increased CIMT is strongly associated with higher dyspnea severity and reduced exercise capacity in COPD patients, suggesting systemic vascular remodeling as a contributor to functional limitation beyond airflow obstruction.

Keywords: COPD, dyspnea, CIMT, six-minute walk distance, systemic atherosclerosis

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a progressive condition characterized by irreversible airflow limitation and chronic inflammation. Beyond pulmonary involvement, COPD is increasingly recognized as a systemic disease with significant extrapulmonary manifestations, particularly cardiovascular morbidity and mortality.[1] Atherosclerosis is one of the leading comorbidities associated with COPD, sharing common pathophysiological pathways such as systemic inflammation, oxidative stress, and endothelial dysfunction.[2]

Carotid intima-media thickness (CIMT) measured by B-mode ultrasonography serves as a validated surrogate marker of

subclinical atherosclerosis and cardiovascular risk. Several studies have demonstrated increased CIMT in COPD patients compared to healthy controls, even after adjusting for traditional cardiovascular risk factors.[2,3]

Dyspnea and reduced exercise tolerance are central clinical features of COPD. The modified Medical Research Council (mMRC) dyspnea scale is widely used to grade breathlessness in COPD patients, while the six-minute walk distance (6MWD) test serves as a simple and reliable measure of functional exercise capacity. Both are strong predictors of morbidity, hospitalization, and mortality in COPD.

Despite well-documented associations of CIMT with COPD severity, limited evidence exists regarding its relationship with functional impairment as reflected by dyspnea and 6MWD. Understanding this association could provide insight into systemic vascular contributions to functional limitation in COPD.

This study was therefore designed to investigate the association between CIMT, dyspnea severity, and functional exercise capacity in patients with COPD.

MATERIALS & METHODS

Study Design and Population

This cross-sectional study was conducted in the Department of Respiratory Medicine at NITRD New Delhi. A total of 31 stable COPD patients were enrolled between June 2018 – May 2020. Prior to enrollment, written informed consent was obtained from all participants, and the study protocol received approval from the Institutional Ethics Committee (NITRD/PGEC/2018/6626). Patients with acute exacerbations within the preceding 4 weeks, known cardiovascular disease, diabetes, or renal failure were excluded to minimize confounding effects on CIMT.

Data Collection

- **Demographic and Clinical Data:** Age, sex, smoking history (pack-years), height, weight, and BMI were recorded.
- **Spirometry:** Performed according to ATS/ERS guidelines. FEV1, FVC, and FEV1/FVC ratio were documented, and patients were classified into GOLD stages I–IV.
- **CIMT Measurement:** CIMT was measured using high-resolution B-mode ultrasonography at the far wall of the common carotid artery, 1 cm proximal to the bifurcation. The mean of three readings from both carotids was taken.
- **Dyspnea Assessment:** The modified Medical Research Council (mMRC) scale (grades 0–4) was used to assess dyspnea severity.
- **Exercise Capacity:** The six-minute walk test (6MWT) was conducted according to ATS guidelines, with the distance covered (6MWD) recorded in meters.

Statistical Analysis

Data were analyzed using SPSS v25. Continuous variables were expressed as mean ± standard deviation. Comparisons between groups (mMRC ≤1 vs. ≥3) were made using Student's t-test. Correlation between CIMT, mMRC, and 6MWD was evaluated using Spearman's correlation test. A p-value <0.05 was considered statistically significant.

RESULTS

Participant Characteristics

A total of 31 patients diagnosed with chronic obstructive pulmonary disease (COPD) were included in the analysis. The mean age was 62.4 ± 8.7 years, with a male predominance (74.2%). The distribution of Modified Medical Research Council (mMRC) dyspnea grades ranged from 0 to 4, with the majority of patients falling within grades 2 to 3 as shown in Table 1.

Table 1; Baseline Characteristics of Participants

Characteristic	Value
Mean age	54.2 ± 9.1 years
Sex	All patients were male
Mean BMI	21.1 ± 3.6 kg/m²
Mean pack-years	32.5 ± 12.4
GOLD Stage I	3 patients
GOLD Stage II	5 patients
GOLD Stage III	13 patients
GOLD Stage IV	10 patients

Dyspnea Severity (mMRC GRADE) and Carotid Intima-Media Thickness (CIMT)
Relationship between mMRC and CIMT is shown in Table 2 Figure 1. As the mMRC grades increase, so does the mean CIMT values. Patients with higher CIMT values are more symptomatic. A positive correlation was observed between mMRC grade and CIMT (Spearman's $\rho = +0.61$, $p < 0.001$). Patients with higher dyspnea scores exhibited greater CIMT values, suggesting increased subclinical atherosclerosis. The mean CIMT in patients with mMRC 0–1 was 0.75 ± 0.12 mm, compared to 2.10 ± 0.28 mm in those with mMRC 3–4.

Table 2: mMRC grades and CIMT values

mMRC Score	Frequency (n)	Mean CIMT (mm)	Standard Deviation (SD)
0	2	0.50	0.14
1	7	0.77	0.10
2	11	1.37	0.49
3	11	2.05	0.78
4	0	–	–

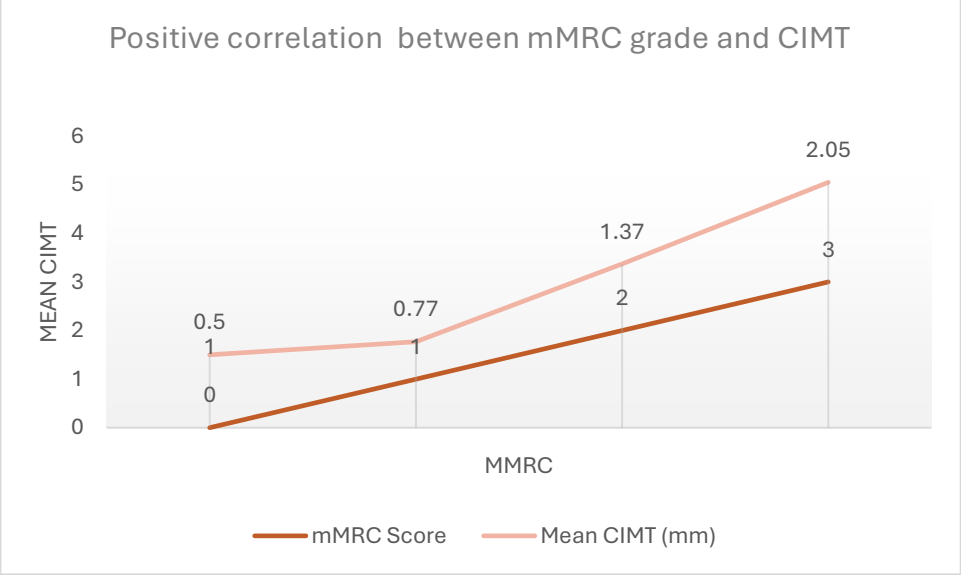

Figure 1; Line Diagram showing relationship between mean CIMT and mMRC grade.

Functional Exercise Capacity (6 MWD) and CIMT;
The 6-minute walk distance (6 MWD) provides a measure for integrated global response of multiple cardiopulmonary and musculoskeletal systems involved in exercise. The six minute walk test provides information regarding functional capacity, response to therapy and prognosis across a broad range of chronic cardiopulmonary conditions. Relationship between 6MWD with mean CIMT is shown in Table 3 Figure 2 and 3. An inverse relationship was also found between 6MWD and CIMT

(Spearman's ρ = −0.59, p < 0.001). Patients with preserved functional capacity (6MWD > 500 m) had significantly lower CIMT values (mean 0.85 ± 0.14 mm), while those with reduced capacity (< 350 m) showed elevated CIMT (mean 2.00 ± 0.26 mm).

Table 3: Categories of 6MWD and CIMT values

6MWD (meters)	Frequency (n)	Mean CIMT (mm)	Standard Deviation (SD)
200–300	2	2.07	1.87
300–400	15	1.39	0.77
400–500	7	1.56	0.64
500–600	5	1.19	0.60
600–700	2	1.12	0.53

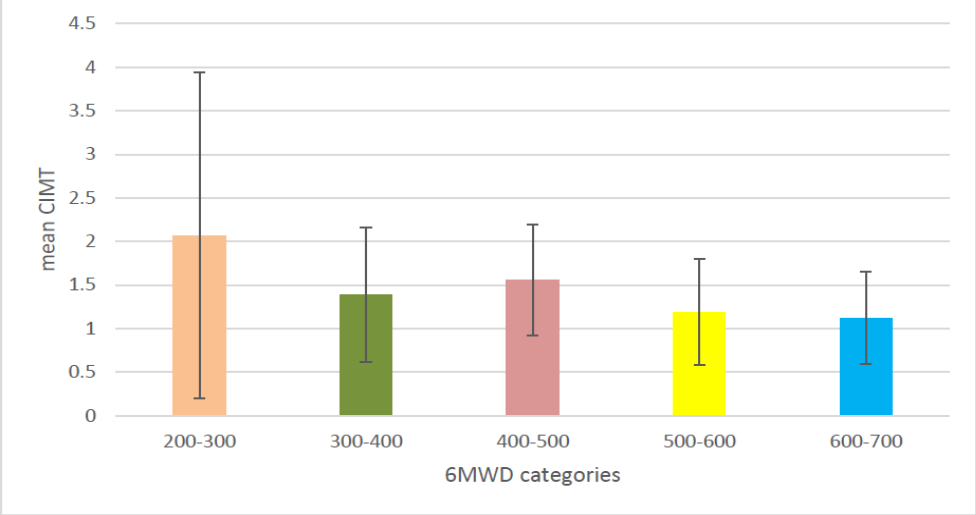

Figure 2; Bar diagram of 6MWD versus CIMT

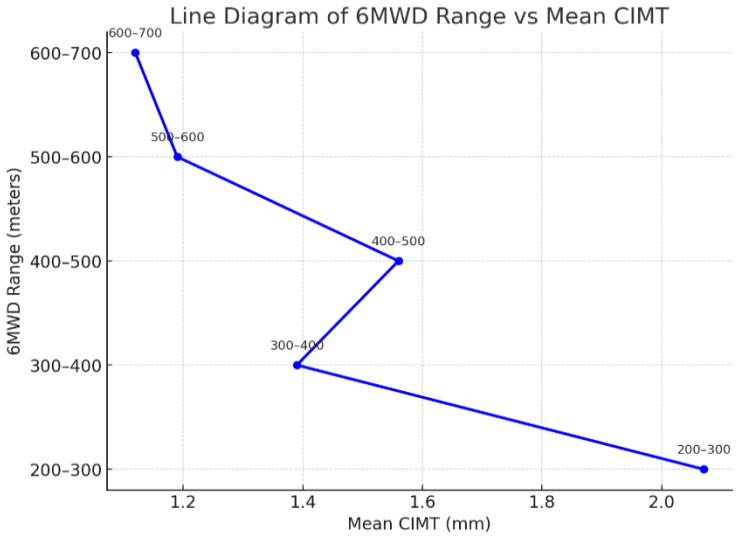

Figure 3; *Line diagram depicting the inverse relationship between 6-minute walk distance (6MWD) and mean carotid intima-media thickness (CIMT). Each point represents a 6MWD range (200–700 meters) plotted against the corresponding mean CIMT (mm). The downward trend suggests that lower functional capacity, as indicated by shorter walking distance, is associated with increased vascular thickness, potentially reflecting greater cardiovascular risk.*

Group Comparisons by mMRC Grade

Box plot analysis revealed significant differences in both 6MWD and CIMT across mMRC categories. ANOVA testing confirmed that both 6MWD (F = 12.4, p <

0.001) and CIMT ($F = 10.7$, $p < 0.001$) varied significantly with dyspnea severity.

Figure 4A and Figure 4B show box plots of 6MWD and CIMT respectively, stratified by mMRC grade.

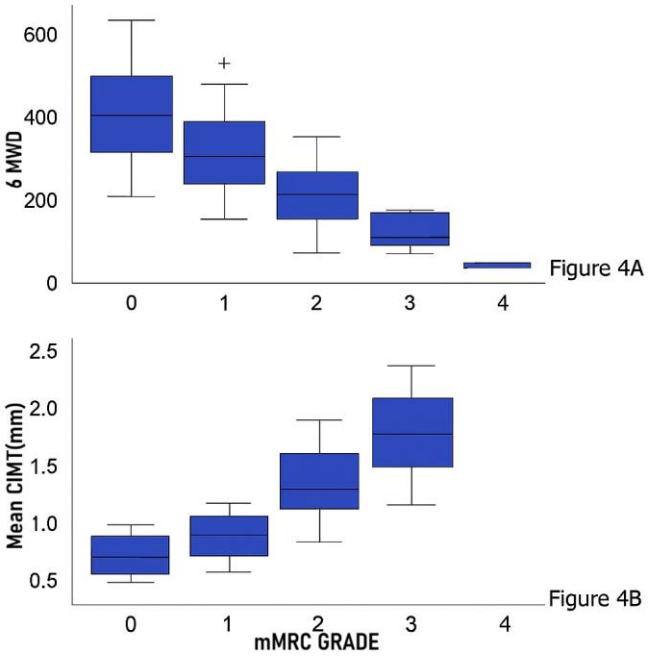

Figure 4. Relationship between mMRC Dyspnea Grade and Functional/Vascular Parameters in COPD Patients. *(A) Box plot showing the distribution of Six-Minute Walk Distance (6MWD, meters) across modified Medical Research Council (mMRC) dyspnea grades (0–4). A progressive decline in median 6MWD is observed with increasing mMRC grade, indicating reduced exercise tolerance and functional capacity. (B) Box plot depicting Carotid Intima-Media Thickness (CIMT, mm) across mMRC grades. CIMT increases with higher dyspnea severity, suggesting a potential link between respiratory impairment and subclinical atherosclerosis. Boxes represent interquartile ranges (IQR), horizontal lines indicate medians, and whiskers denote data spread excluding outliers.*

Summary of Correlations

Table 4 Figure 5 illustrates the Spearman correlations among three clinical variables: mMRC (Modified Medical Research Council dyspnea scale), 6MWD (Six-Minute Walk Distance), and CIMT (Carotid Intima-Media Thickness). Each variable is represented by a distinct geometric shape - rectangles for mMRC and CIMT, and a circle for 6MWD - highlighting their roles in assessing respiratory function, physical performance, and vascular health, respectively. The arrows connecting these variables denote the direction and strength of their relationships, quantified by Spearman's ρ. A strong inverse correlation ($\rho = -0.68$) exists between mMRC and 6MWD, indicating that higher dyspnea scores are associated with reduced walking capacity. A moderate positive correlation ($\rho = +0.61$) between mMRC and CIMT suggests that worsening dyspnea may parallel increased vascular thickness, potentially reflecting systemic disease burden. Lastly, the moderate inverse correlation ($\rho = -0.59$) between 6MWD and CIMT implies that reduced physical endurance is linked to greater vascular compromise. Together, these relationships underscore the interconnected nature of respiratory symptoms, functional capacity, and vascular pathology in chronic disease states.

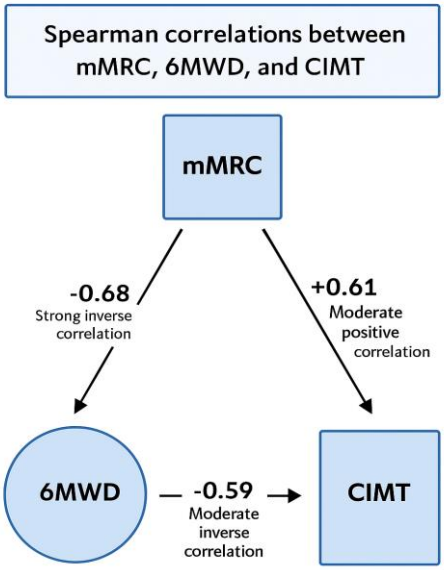

Figure 5; Summary of Correlations

Table 4; Summary of correlation of Variables and statistical significances.

Variable Pair	Spearman's ρ	Interpretation	p-value
mMRC vs. 6MWD	−0.68	Strong inverse correlation	< 0.001
mMRC vs. CIMT	+0.61	Moderate positive correlation	< 0.001
6MWD vs. CIMT	−0.59	Moderate inverse correlation	< 0.001

These findings suggest a triad of worsening dyspnea, declining functional capacity, and increasing vascular thickness in COPD patients, underscoring the systemic nature of the disease and its cardiovascular implications.

DISCUSSION

Chronic Obstructive Pulmonary Disease (COPD) is a preventable and treatable condition characterized by persistent airflow limitation that is typically progressive and not fully reversible.

This limitation is associated with an enhanced chronic inflammatory response in the airways and lungs to noxious particles and gases, most commonly from tobacco smoke. The disease encompasses both structural and functional alterations, contributing to systemic manifestations and significant morbidity.[4]

Chronic Obstructive Pulmonary Disease (COPD) is increasingly recognized as a systemic disorder with significant extrapulmonary manifestations. Its pathogenesis reflects a complex interplay between genetic predisposition and environmental exposures—most notably tobacco smoke, occupational pollutants, and biomass fuel. These factors contribute not only to persistent airflow limitation but also to systemic inflammation, skeletal muscle dysfunction, cardiovascular comorbidities, and metabolic disturbances.[5] Comorbidities are integral to the clinical burden of Chronic Obstructive Pulmonary Disease (COPD), contributing substantially to both morbidity and mortality. Among these, cardiovascular comorbidities are particularly prevalent and impactful. Conditions such as angina pectoris, cerebrovascular accidents, cardiac arrhythmias, left ventricular hypertrophy, and myocardial infarction frequently coexist with COPD and are associated with significantly reduced survival. Their presence underscores the systemic nature of

COPD and necessitates comprehensive risk stratification and multidisciplinary management.[5,6] Emerging evidence indicates a heightened risk of cardiovascular disease among patients with Chronic Obstructive Pulmonary Disease (COPD), contributing substantially to overall mortality.

Multiple studies have demonstrated that cardiovascular complications - including ischemic heart disease, arrhythmias, and cerebrovascular events - are prevalent in this population. Notably, up to 40% of deaths in COPD patients have been attributed to cardiovascular causes.[7,8] This underscores the need for integrated cardiopulmonary care and early identification of vascular risk in COPD management.

Large-scale epidemiological data indicate that individuals with severe to very severe Chronic Obstructive Pulmonary Disease (COPD) exhibit more than a twofold increased risk of cardiovascular disease, a 1.6-fold higher prevalence of hypertension, and an elevated likelihood of hospitalization.

This heightened risk profile is thought to be mediated, at least in part, by persistent low-grade systemic inflammation, a shared pathophysiological mechanism underlying both COPD and atherosclerotic cardiovascular disease.[7,9,10] The inflammatory milieu may contribute to endothelial dysfunction, vascular remodeling, and accelerated atherogenesis, reinforcing the need for integrated cardiopulmonary surveillance and intervention.

Low-grade systemic inflammation has been documented even in non-smokers with Chronic Obstructive Pulmonary Disease (COPD), suggesting that inflammatory pathways in COPD are not solely attributable to tobacco exposure.[11] This finding reinforces the concept of COPD as a heterogeneous and systemic disorder, potentially driven by genetic susceptibility, environmental pollutants, infections, or dysregulated immune responses independent of smoking history. Carotid Intima-Media Thickness (CIMT) is a well-established surrogate marker for subclinical atherosclerosis and plays a pivotal role in cardiovascular risk stratification.[12,13]

COPD patients exhibiting both metabolic abnormalities and elevated systemic inflammation. COPD patients with significant weight loss and muscle wasting had increased circulating levels of inflammatory mediators such as TNF-α.[14] These findings suggest a mechanistic link between systemic inflammation and metabolic derangements including cachexia in COPD.

Peripheral muscle dysfunction is a recognized extrapulmonary manifestation of COPD, with significant implications for exercise capacity and symptom burden. Bernard et al. demonstrated that quadriceps weakness in COPD patients occurs independently of spirometric severity and is closely associated with reduced physical performance.[15] This aligns with the present study's findings, where Six-Minute Walk Distance (6MWD) declined progressively across increasing mMRC grades, reflecting worsening functional limitation. The modified Medical Research Council (mMRC) dyspnoea scale offers a simple, validated tool for categorizing COPD patients based on functional disability and symptom burden. While spirometric indices such as FEV_1 remain central to COPD staging, mMRC grading provides complementary insight into the patient's real-world limitations.[16] The mMRC dyspnea scale, while subjective, appears to correlate not only with ventilatory impairment but also with peripheral muscle strength and endurance. These observations underscore the systemic nature of COPD and support the integration of muscle assessment and rehabilitation strategies into routine care, particularly for patients with higher mMRC grades and reduced 6MWD.

Longitudinal evidence suggests that functional performance in COPD patients declines progressively over time, independent of spirometric deterioration.[17]

This decline is driven by a combination of worsening dyspnea, fatigue, and reduced physical activity, which collectively impair daily functioning. In the present study, this trajectory is reflected in the inverse relationship between mMRC grade and Six-Minute Walk Distance (6MWD), where higher dyspnea scores corresponded with significantly reduced exercise capacity. These findings support the notion that mMRC grading captures more than just symptom severity - it reflects underlying muscular and behavioral deconditioning. When considered alongside peripheral muscle weakness and vascular changes such as increased Carotid Intima-Media Thickness (CIMT), the mMRC scale emerges as a valuable clinical tool for stratifying systemic burden in COPD.[2,7,15] This underscores the need for early intervention strategies targeting physical rehabilitation, nutritional support, and cardiovascular risk reduction.

The multidimensional nature of functional decline in COPD is well illustrated by the functional status model proposed by Yeh et al., which emphasizes the interplay between physical limitations, psychological distress, and social factors in shaping patient outcomes.[18] In the present study, this framework is reflected in the progressive reduction in Six-Minute Walk Distance (6MWD) and increasing mMRC dyspnea grades, both of which capture not only ventilatory impairment but also systemic deconditioning and behavioral adaptation. Furthermore, the observed association between higher mMRC scores and elevated Carotid Intima-Media Thickness (CIMT) suggests that functional disability may also mirror underlying vascular pathology. These findings support a more holistic approach to COPD assessment—one that integrates symptom burden, physical performance, and cardiovascular risk—to guide individualized rehabilitation and risk mitigation strategies. A recent cross-sectional study published in *Rehabilitation* (2021) confirmed that patients with COPD exhibit significantly reduced physical activity and functional performance compared to healthy controls.[19] These limitations correlate with higher dyspnea scores and diminished exercise tolerance. In our cohort, declining 6MWD across mMRC grades reflects this trend. The association with elevated CIMT further suggests that functional impairment may signal underlying vascular pathology, reinforcing the need for multidimensional assessment. A study by Köseoğlu et al. (2016) demonstrated that Carotid Intima-Media Thickness (CIMT) is significantly elevated in COPD patients with angiographically confirmed coronary artery disease.[20] Firincioglulari et al. (2019) found that carotid intima-media thickness (CIMT) was significantly higher in COPD patients compared to healthy controls, with strong associations between CIMT and disease severity, hypoxemia, and hypercapnia. Their findings suggest that atherosclerosis begins early in COPD and progresses with worsening respiratory dysfunction.[21] This supports the use of CIMT as a non-invasive marker for vascular risk in COPD patients.

This supports CIMT as a non-invasive marker for detecting subclinical atherosclerosis and stratifying cardiovascular risk in COPD. In our cohort, higher mMRC grades and reduced 6MWD were similarly associated with increased CIMT, reinforcing its clinical utility in integrated cardiopulmonary assessment.

The findings of this study demonstrate a strong association between CIMT and functional impairment in COPD, as reflected by dyspnea severity and exercise capacity. Patients with more severe dyspnea had thicker CIMT and reduced 6MWD, indicating that systemic vascular changes may contribute to the functional limitations traditionally attributed to airflow obstruction and lung hyperinflation.

Our findings are consistent with previous studies that reported increased CIMT in COPD patients, independent of smoking history and conventional cardiovascular risk factors. Systemic inflammation, oxidative stress, and endothelial dysfunction in COPD

likely accelerate atherosclerosis, contributing to vascular remodeling.

Importantly, our study highlights that CIMT correlates not only with spirometric severity but also with patient-centered functional outcomes such as dyspnea and 6MWD. This underscores the systemic nature of COPD and the importance of assessing cardiovascular risk alongside pulmonary function.

Clinically, CIMT measurement could be incorporated into COPD assessment to identify patients at risk of both cardiovascular events and functional decline. Targeted interventions such as lifestyle modification, exercise rehabilitation, and cardiovascular risk reduction strategies may therefore improve overall prognosis.

Limitations

- Small sample size (n=31), all male patients.
- Cross-sectional design precludes causal inference.
- Lack of a control group.
- Potential residual confounding from unmeasured cardiovascular risk factors.

Future Directions

Larger, longitudinal studies are needed to confirm these associations and evaluate whether interventions that reduce CIMT can improve functional outcomes in COPD.

CONCLUSION

In COPD patients, increased CIMT is significantly associated with greater dyspnea severity and reduced exercise capacity. CIMT may serve as a non-invasive marker linking systemic vascular changes with functional impairment in COPD. Integrating vascular assessment into COPD management could enhance risk stratification and guide holistic treatment strategies.

Declaration by Authors

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REFERENCES

1. "https://goldcopd.org/2025-gold-report/."
2. A. G. N. Agusti, "Systemic Effects of Chronic Obstructive Pulmonary Disease," Proc Am Thorac Soc, vol. 2, no. 4, pp. 367–370, Nov. 2005, doi: 10.1513/pats.200504-026SR.
3. A. Agustí "Persistent Systemic Inflammation is Associated with Poor Clinical Outcomes in COPD: A Novel Phenotype," PLoS One, vol. 7, no. 5, p. e37483, May 2012, doi: 10.1371/journal.pone.0037483.
4. "GOLD (Global Initiative for Chronic Obstructive Lung Disease) 2017," Pneumologie, vol. 71, no. 01, pp. 9–14, 2017, doi: 10.1055/s-0042-121903.
5. D. Singh "Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Lung Disease: the GOLD science committee report 2019," European Respiratory Journal, vol. 53, no. 5, p. 1900164, May 2019, doi: 10.1183/13993003.00164-2019.
6. O. Okutan and Ö. Ayten, "İstanbul Chronic Obstructive Pulmonary Disease and Comorbidities Respiratory," Jl Respir, vol. 14, no. 3, pp. 181–183, 2012.
7. P. J. Barnes and B. R. Celli, "Systemic manifestations and comorbidities of COPD," European Respiratory Journal, vol. 33, no. 5, pp. 1165–1185, May 2009, doi: 10.1183/09031936.00128008.
8. S. P. Bhatt, J. M. Wells, and M. T. Dransfield, "cardiovascular disease in COPD: a call for action," Lancet Respir Med, vol. 2, no. 10, pp. 783–785, Oct. 2014, doi: 10.1016/S2213-2600(14)70197-3.
9. S. Scarlata, "Mechanisms of atherothrombosis in chronic obstructive pulmonary disease," Int J Chron Obstruct Pulmon Dis, vol. Volume 3, pp. 89–96, Mar. 2008, doi: 10.2147/COPD.S1401.
10. J. D. Maclay, d. A. Mcallister, and w. Macnee, "Cardiovascular risk in chronic obstructive pulmonary disease," Respirology, vol. 12, no. 5, pp. 634–641, Sep. 2007, doi: 10.1111/j.1440-1843.2007.01136. x.

11. W. Q. Gan, "Association between chronic obstructive pulmonary disease and systemic inflammation: a systematic review and a meta-analysis," Thorax, vol. 59, no. 7, pp. 574–580, Jul. 2004, doi: 10.1136/thx.2003.019588.
12. S. C. H. van den Oord, "Carotid intima-media thickness for cardiovascular risk assessment: Systematic review and meta-analysis," Atherosclerosis, vol. 228, no. 1, pp. 1–11, May 2013, doi: 10.1016/j.atherosclerosis.2013.01.025.
13. P.-J. Touboul., "Mannheim Carotid Intima-Media Thickness and Plaque Consensus (2004–2006–2011)," Cerebrovascular Diseases, vol. 34, no. 4, pp. 290–296, 2012, doi: 10.1159/000343145.
14. A. M. Schols, W. A. Buurman, A. J. S. Van den Brekel et al., "Evidence for a relation between metabolic derangements and increased levels of inflammatory mediators in a subgroup of patients with chronic obstructive pulmonary disease.," Thorax, vol. 51, no. 8, pp. 819–824, 1996.
15. S. Bernard, "Peripheral muscle weakness in patients with chronic obstructive pulmonary disease," Am J Respir Crit Care Med, vol. 158, no. 2, pp. 629–634, 1998.
16. J. C. Bestall, E. A. Paul, R. Garrod, R et al., "Usefulness of the Medical Research Council (MRC) dyspnoea scale as a measure of disability in patients with chronic obstructive pulmonary disease," Thorax, vol. 54, no. 7, pp. 581–586, Jul. 1999, doi: 10.1136/thx.54.7.581.
17. M. C. Kapella, J. L. Larson, M. K. Covey et al., "Functional performance in chronic obstructive pulmonary disease declines with time," Med Sci Sports Exerc, vol. 43, no. 2, p. 218, 2011.
18. M. Yeh, H. Chen, Y. Liao et al., "Testing the functional status model in patients with chronic obstructive pulmonary disease," J Adv Nurs, vol. 48, no. 4, pp. 342–350, 2004.
19. L. Delgado-Ortiz, "Physical activity and cardiac autonomic dysfunction in patients with chronic obstructive pulmonary disease: A cross-sectional analysis," Ann Phys Rehabil Med, vol. 65, no. 3, p. 101501, May 2022, doi: 10.1016/j.rehab.2021.101501.
20. C. Köseoğlu, "Association between carotid intima-media thickness and presence of coronary artery disease in chronic obstructive pulmonary disease patients.," Anatolian Journal of Cardiology/Anadolu Kardiyoloji Dergisi, vol. 16, no. 8, 2016.
21. A. Firincioglulari, H. Erturk, M. Firincioglulari et al., "Evaluation of atherosclerosis as a risk factor in COPD patients by measuring the carotid intima-media thickness," Cardiovasc Ultrasound, vol. 22, no. 1, p. 2, Jan. 2024, doi: 10.1186/s12947-023-00322-8.

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