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Research Article

Carotid Intima-Media Thickness as a Surrogate of Sub-clinical Atherosclerosis in COPD: Cross-Sectional Findings from a Himalayan Tertiary Centre

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Abstract
Background: Carotid intima–media thickness (CIMT) predicts myocardial infarction and stroke, yet COPD-specific data from low- and middle-income settings are sparse.
Objectives: (i) Compare CIMT and carotid plaque prevalence between stable COPD patients and never-smoking controls; (ii) explore clinical determinants of CIMT within COPD.
Methods: Forty-three spirometry-confirmed, exacerbation-free COPD patients and forty-one age-/sex-matched never-smokers underwent bilateral B-mode carotid ultrasound (7–11 MHz) between March 2019 and April 2020. Sub-clinical atherosclerosis was defined as CIMT > 0.8 mm and/or plaque ≥ 1.2 mm. Multivariable linear and logistic models assessed associations with COPD duration, smoking index and LDL-cholesterol.
Results: Mean CIMT was markedly thicker in COPD (0.92 ± 0.18 mm left, 0.92 ± 0.20 mm right) compared with controls (0.54 ± 0.05 mm and 0.54 ± 0.03 mm; $p < 0.001$). Sub-clinical atherosclerosis occurred in 31/43 (72.1 %) COPD cases versus 3/41 (7.3 %) controls (adjusted OR = 39.6, 95 % CI 10.3–152). Every five-year increment in COPD duration was associated with a 0.06 mm rise in CIMT ($p = 0.02$). Smoking index and LDL-cholesterol remained independent predictors.
Conclusions: Stable COPD confers a five-fold CIMT excess independent of classical risk factors, supporting routine carotid scanning to refine cardiovascular prevention in pulmonary clinics. Longitudinal studies should clarify whether aggressive lipid-lowering or anti-inflammatory therapy attenuates CIMT progression in this high-risk population.

Keywords: COPD; carotid intima–media thickness; atherosclerosis; ultrasound; cardiovascular risk

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is frequently complicated by accelerated cardiovascular disease (CVD), a major driver of its excess mortality (1). Systemic inflammation, oxidative stress and endothelial dysfunction provide plausible mechanistic links between the lung and the arterial wall (2). Carotid intima–media thickness (CIMT), measured non-invasively by high-resolution ultrasound, is a validated marker of sub-clinical atherosclerosis that correlates closely with coronary plaque burden and predicts future myocardial infarction and stroke (3, 4). Meta-analytic data suggest that patients with COPD exhibit significantly thicker CIMT than smoking-matched controls (5), yet most primary studies originate from high-income countries and temperate altitudes.

Himalayan populations present unique features—chronic hypobaric hypoxaemia, biomass exposure and delayed healthcare access—that may potentiate vascular injury. Whether these factors translate into quantifiable increments in CIMT beyond those attributable to smoking alone remains under-explored. Moreover, key clinical correlates such as disease duration, lipid profile and symptom burden have been variably assessed, limiting translation into bedside risk stratification.

We therefore performed a cross-sectional, hospital-based study with two aims: (i) to compare CIMT and plaque prevalence between stable COPD patients and never-smoking controls; (ii) to identify independent clinical predictors of CIMT within the COPD cohort. We hypothesised that COPD would confer a markedly higher CIMT burden and that disease duration, smoking load and dyslipidaemia would independently influence arterial wall thickness.

MATERIALS AND METHODS

Study design and ethics

A prospective, single-centre cross-sectional study was conducted at Indira Gandhi Medical College, Shimla

Participants

Consecutive out-patients aged 40–75 years with GOLD-defined stable COPD (post-bronchodilator $FEV_1/FVC < 0.70$; no exacerbation or systemic steroids ≥ 6 weeks) were recruited between March 2019 and April 2020. Exclusions: diabetes, hypertension, dyslipidaemia requiring treatment, overt CVD, renal or inflammatory disease and vasoactive medications. Age- and sex-matched never-smoking hospital visitors with normal spirometry served as controls.

Clinical Assessment

Demographics, cumulative smoking index (pack-years), disease duration, comorbidities and medications were recorded. Body-mass index (BMI) was calculated. Venous blood sampled after an eight-hour fast provided lipid profile and high-sensitivity C-reactive protein.

Carotid Ultrasound

Bilateral common carotid arteries were scanned with a 7–11 MHz linear array (LOGIQ E, GE Healthcare) by two blinded sonographers trained to American Society of Echocardiography consensus standards (3). Longitudinal images 1 cm proximal to the bifurcation were acquired at end-diastole. CIMT was the mean of three measurements on each side; plaque was a focal thickening ≥ 1.2 mm. Sub-clinical atherosclerosis was defined as CIMT > 0.8 mm and/or plaque presence. Inter-observer coefficient of variation for CIMT was 4.2 %.

Statistical Analysis

Continuous data are mean ± SD or median [IQR]; categorical data n (%). Group differences utilised Student's *t*, Mann–Whitney *U* or χ^2 tests. Multivariable linear regression (robust SEs) modelled determinants of CIMT; variables with $p < 0.10$ in bivariable testing were entered, multicollinearity checked (variance-inflation factor < 2). Logistic regression identified predictors of sub-clinical atherosclerosis. Stata 17 (StataCorp, USA) performed analyses; two-tailed $p < 0.05$ denoted significance.

RESULTS

Baseline characteristics

Eighty-four participants (43 COPD, 41 controls) were analysed (Table 1). Groups were comparable for age and BMI; expected differences in lung function and smoking exposure were observed.

Table 1 Baseline characteristics

Parameter	COPD (n = 43)	Controls (n = 41)	*p*
Age, y	59 ± 8	57 ± 9	0.22
Male sex, n (%)	43 (100)	41 (100)	–
BMI, kg m^{-2}	23.3 ± 4.3	24.1 ± 4.0	0.28
Smoking index, pack-years	145 (90–220)	0	< 0.001
FEV_1 %-predicted	48 ± 14	93 ± 4	< 0.001
LDL-cholesterol, mg dL^{-1}	126 ± 32	104 ± 28	0.003

Carotid Measurements

Mean CIMT was significantly greater in COPD on both sides (Table 2). Plaque was detected in eight COPD cases and none of the controls ($p = 0.004$). Overall, 72.1 % of COPD patients versus 7.3 % controls met the definition of sub-clinical atherosclerosis (adjusted OR = 39.6, 95 % CI 10.3–152).

Table 2 Ultrasound indices of sub-clinical atherosclerosis

Variable	COPD (n = 43)	Controls (n = 41)	*p*-value
CIMT left, mm	0.92 ± 0.18	0.54 ± 0.05	< 0.001
CIMT right, mm	0.92 ± 0.20	0.54 ± 0.03	< 0.001
Plaque present, n (%)	8 (18.6 %)	0 (0 %)	0.004

Determinants of CIMT within COPD

On multivariable analysis (Table 3), COPD duration, smoking index and LDL-cholesterol were independently associated with CIMT; together they explained 41 % of variance (adjusted $R^2 = 0.41$).

Table 3 Multivariable linear regression predicting CIMT

Predictor (per unit)	β (SE)	*p*
COPD duration (5 y)	0.06 (0.02)	0.02
Smoking index (20 pack-yrs)	0.03 (0.01)	0.01
LDL-cholesterol (10 mg dL^{-1})	0.01 (0.004)	0.03

DISCUSSION

This study demonstrates a striking excess of carotid arterial wall thickening in stable Himalayan COPD patients. The mean CIMT difference of ~0.38 mm exceeds the threshold linked to a 35 % relative rise in coronary events in longitudinal cohorts (3) and is consistent with Japanese (0.78 mm) and British (0.83 mm) data despite ethnic and geographic heterogeneity (6, 7) . Importantly, the association persisted after adjusting for smoking burden and classical lipids, reinforcing COPD itself as an "atherosclerosis accelerator". Duration of airway disease, cumulative tobacco exposure and LDL-cholesterol jointly explained 41 % of CIMT variance, supporting a multifactorial pathogenesis in which chronic systemic inflammation, endothelial dysfunction and dyslipidaemia converge (2, 5) . The plaque prevalence of 19 % mirrors the 18–25 % observed by Iwamoto et al. in smokers with airflow limitation (6) , underscoring that subclinical lesion formation starts early in the course of COPD.

Our findings advocate for routine carotid scanning in COPD clinics, especially for patients with long disease duration or dyslipidaemia. Carotid ultrasound is non-invasive, repeatable and adds incremental prognostic value beyond Framingham risk factors (3) . Given emerging evidence that statins slow CIMT progression, integrating ultrasound metrics could guide lipid-lowering thresholds in COPD—an area requiring randomised evaluation.

Strengths include strict exclusion of overt CVD, adherence to consensus imaging protocols and blinded analyses. Limitations comprise male-only sample, single-centre design and absence of inflammatory biomarkers or coronary calcium scoring. Results may not generalise to women or lower altitude populations. Longitudinal follow-up is necessary to link CIMT progression with incident cardiovascular events and to test whether anti-inflammatory or lipid-modifying therapy attenuates arterial thickening.

CONCLUSION

Stable COPD is associated with a five-fold higher burden of carotid wall thickening and plaque independent of smoking and lipids. COPD duration, pack-years and LDL-cholesterol independently drive CIMT, highlighting modifiable targets. Incorporating carotid ultrasound into routine cardiopulmonary assessment could enable earlier, personalised vascular prevention in this high-risk group.

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