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**Research Article**

# High Ankle–Brachial Index in COPD — Marker of Arterial Stiffness or Sampling Artifact? A Clinicoradiological Correlation Study

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**Abstract**
**Background:** An ankle–brachial index (ABI) > 1.40 denotes non-compressible, calcified arteries and predicts excess cardiovascular mortality, yet its prevalence and determinants in chronic obstructive pulmonary disease (COPD) remain unclear.
**Objectives:** (i) Compare the frequency of high (> 1.40) and low (< 0.90) ABI between stable COPD patients and healthy never-smokers; (ii) identify clinical and radiographic correlates of high ABI within COPD.
**Methods:** Forty-three spirometry-confirmed, exacerbation-free COPD patients and forty-one age-/sex-matched never-smoking volunteers were studied (March 2019 – April 2020). After 10 min supine rest, bilateral ankle and brachial systolic pressures were measured by continuous-wave Doppler; ABI was the higher ankle pressure divided by the higher brachial pressure. Categories: low < 0.90, normal 1.00–1.40, high > 1.40. Chest radiographs were scored for hyperinflation; symptom burden captured with the COPD Assessment Test (CAT). Multivariable logistic regression examined predictors of high ABI.
**Results:** High ABI was more common in COPD (14/43, 32.6 %) than controls (3/41, 7.3 %; $p = 0.005$). Only one COPD subject exhibited low ABI. COPD patients with high ABI showed more radiographic hyperinflation (71 % vs 23 %, $p = 0.01$) and higher CAT scores (34 ± 6 vs 28 ± 8, $p = 0.03$). In adjusted analysis, dyslipidaemia (OR 3.4, 95 % CI 1.2–9.5) and hyperinflation (OR 4.1, 95 % CI 1.3–12.9) independently predicted high ABI, supporting a true arterial-stiffness phenotype rather than cuff artifact.
**Conclusions:** One-third of Himalayan COPD patients harbour abnormally stiff, non-compressible peripheral arteries linked to hyperinflation and metabolic risk. Given its speed, low cost and prognostic power, routine ABI measurement should accompany spirometry for comprehensive cardiovascular stratification in COPD clinics.

**Keywords:** COPD; ankle–brachial index; arterial stiffness; hyperinflation; cardiovascular risk

## INTRODUCTION

Cardiovascular disease (CVD) accounts for up to one-third of deaths in chronic obstructive pulmonary disease (COPD) (1) . Beyond shared risk factors, systemic inflammation, oxidative stress and premature vascular ageing are thought to accelerate atherogenesis and arterial stiffness in COPD (2) .The ankle–brachial index (ABI) — the ratio of ankle to brachial systolic pressure — is an established bedside test for peripheral arterial disease and a strong predictor of myocardial infarction, stroke and mortality even after adjustment for Framingham risk scores (3) . Whereas a low ABI (< 0.90) reflects obstructive plaque, a *high* ABI (> 1.40) signifies non-compressible, calcified arteries typical of stiffened media; both extremes independently double CVD mortality in population studies such as the Strong Heart Study (4) .Data on ABI distribution in COPD are sparse and conflicting. European cohorts report excess low ABI, whereas the only North-American analysis found prevalent high ABI with arterial stiffness indices elevated in COPD (2) . No Indian or high-altitude data exist despite hypobaric hypoxaemia potentially potentiating vascular calcification. Importantly, radiographic hyperinflation, dyslipidaemia and symptom burden — easily captured in outpatient practice — have not been jointly examined as predictors of aberrant ABI. We therefore conducted a clinicoradiological study in a Himalayan tertiary centre to (i) quantify the prevalence of high and low ABI in stable COPD

versus never-smokers and (ii) explore clinical, biochemical and imaging correlates of high ABI within COPD. We hypothesised that COPD would display an excess of high ABI consistent with medial arterial stiffness and that hyperinflation and metabolic factors would independently predict this phenotype.

## Materials and Methods

### Study Design and Ethics

This prospective case-control study was performed at Indira Gandhi Medical College, Shimla.

### Participants

Cases were adults aged 40–75 years with GOLD-defined stable COPD (post-bronchodilator $FEV_1/FVC < 0.70$, no exacerbation ≥ 6 weeks). Exclusions: diabetes, hypertension, chronic kidney disease, established CVD, lipid-lowering therapy or vasoactive drugs. Age- and sex-matched healthy never-smokers with normal spirometry served as controls.

### Clinical Assessment

Demographics, pack-years, COPD duration, medications and comorbidities were recorded. Body-mass index (BMI) was calculated. CAT quantified symptoms; resting oxygen saturation ($SpO_2$) and respiratory rate were measured.

### Ankle–Brachial Index

After 10 min supine rest, systolic pressures at brachial, posterior tibial and dorsalis pedis arteries were measured with an 8 MHz Doppler probe (Huntleigh Dopplex). ABI for each leg was ankle (higher of two sites) divided by higher brachial pressure; the lower ABI of two legs represented the subject. Categories: low < 0.90; normal 1.00–1.40; high > 1.40 as per AHA guidelines (3). Two observers performed duplicate readings; coefficient of variation was 3.8 %.

### Chest Radiography

Postero-anterior films were scored for hyperinflation (flattened diaphragms + increased retrosternal airspace) by a radiologist blinded to ABI.

### Biochemistry

Fasting lipids and high-sensitivity C-reactive protein were analysed by enzymatic colorimetry.

### Statistical Analysis

Continuous variables are mean ± SD or median [IQR]; categorical variables n (%). Group differences used Student's *t*, Mann-Whitney *U* or $\chi^2$ tests. Predictors of high ABI were examined with multivariable logistic regression (enter method); covariates with $p < 0.10$ on bivariable testing were entered; multicollinearity checked (VIF < 2). Hosmer–Lemeshow assessed calibration. Analyses used Stata 17; two-tailed $p < 0.05$ was significant.

## RESULTS

### Baseline characteristics

Eighty-four participants (43 COPD, 41 controls) were included (Table 1). Groups were similar in age and BMI; COPD subjects had heavier smoking exposure, lower $FEV_1$ and higher dyslipidaemia prevalence.

Table 1 Baseline characteristics

| Parameter | COPD (n = 43) | Controls (n = 41) | *p* |
|---|---|---|---|
| Age, years | 59 ± 8 | 57 ± 9 | 0.25 |
| Male sex, n (%) | 43 (100) | 41 (100) | – |
| BMI, kg $m^{-2}$ | 23.2 ± 4.4 | 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 |
| Dyslipidaemia, n (%) | 17 (39.5) | 4 (9.8) | 0.002 |

### ABI Distribution

ABI categories differed markedly between groups (Table 2). High ABI (> 1.40) affected 14/43 COPD patients (32.6 %) versus 3/41 controls (7.3 %; $\chi^2 = 7.9$, $p = 0.005$). Low ABI (< 0.90) occurred in one COPD subject and none of the controls.

Table 2 Ankle–brachial index categories

| ABI category | COPD (n = 43) | Controls (n = 41) |
|---|---|---|
| Low < 0.90 | 1 (2.3 %) | 0 |
| Normal 1.00–1.40 | 28 (65.1 %) | 38 (92.7 %) |
| High > 1.40 | 14 (32.6 %) | 3 (7.3 %) |

**Correlates of high ABI within COPD**

Compared with COPD patients with normal ABI, those with high ABI exhibited more chest hyperinflation (71 % vs 23 %, $p$ = 0.01) and higher CAT scores (34 ± 6 vs 28 ± 8, $p$ = 0.03). Dyslipidaemia was also more frequent (59 % vs 29 %, $p$ = 0.04).

Multivariable logistic regression (Table 3) identified hyperinflation and dyslipidaemia as independent predictors of high ABI; model discrimination c-statistic = 0.81.

Table 3 Predictors of high ABI (> 1.40) in COPD (n = 43)

| Predictor | Adjusted OR (95 % CI) | *p* |
|---|---|---|
| Chest hyperinflation (yes) | 4.1 (1.3–12.9) | 0.02 |
| Dyslipidaemia (yes) | 3.4 (1.2–9.5) | 0.03 |
| Smoking index (per 20 pack-years) | 1.2 (0.9–1.7) | 0.18 |

## DISCUSSION

This study is the first from a high-altitude Indian population to demonstrate a striking excess of high ABI in stable COPD. One-third of patients exhibited non-compressible (> 1.40) indices, quadruple the prevalence in matched never-smokers and surpassing European primary-care estimates (5) . High ABI reflects medial calcification and arterial stiffness rather than obstructive plaque; its independent association with CVD mortality rivals that of low ABI (4) .

Two findings support a *true* stiffness phenotype rather than cuff artifact. First, radiographic hyperinflation independently predicted high ABI, echoing mechanistic work linking lung over-inflation to increased intrathoracic pressure, altered pulsatile flow and aortic stiffening (2) . Second, dyslipidaemia remained a significant correlate after controlling for smoking, suggesting metabolic injury contributes to arterial calcification in COPD beyond inflammatory pathways.

Our results extend limited COPD-ABI literature. Houben-Wilke et al. reported 8.8 % peripheral artery disease (low ABI < 0.90) but did not examine high ABI (1) , whereas Mills et al. documented elevated pulse-wave velocity and augmentation index, surrogate markers of stiffness (2). We show that simple Doppler measurement captures the same vascular abnormality. The low frequency of ABI < 0.90 in our cohort may reflect exclusion of overt CVD and relatively young age.

Strengths include standardised Doppler protocol, blinding, and comprehensive radiographic and metabolic profiling. Limitations are the male-only sample, modest size precluding analysis by GOLD stage, and cross-sectional design that cannot confirm causality or prognostic impact. Nonetheless, the c-statistic of 0.81 for our model suggests clinical utility.

Given its low cost, reproducibility and guideline endorsement, ABI measurement could be integrated with spirometry at first COPD diagnosis. Prospective studies should test whether targeted interventions (statins, vitamin K antagonists, exercise) reduce arterial stiffness and subsequent cardiovascular events in COPD with high ABI.

## CONCLUSION

Approximately one-third of stable Himalayan COPD patients exhibit a high ABI indicative of medial arterial stiffness, independently associated with chest hyperinflation and dyslipidaemia. Routine ABI screening alongside lung function testing may enhance cardiovascular risk stratification and support early preventive therapy in COPD clinics.

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