Original Article

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Spirometric Variations in Adults with Controlled Hypertension: A Cross-sectional Study from Eastern India

Physiology Section

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ABSTRACT

Introduction: Hypertension's impact on global health is vast, yet its link to Indian pulmonary function remains unclear. Despite maintaining controlled blood pressure, hypertensive individuals may harbour undetected pulmonary deficits, necessitating a closer look at the intersection of cardiovascular and respiratory health.

Aim: To compare spirometric parameters between subjects with controlled essential hypertension and normotensive healthy controls in Eastern India.

Materials and Methods: A cross-sectional study was conducted at Rampurhat Government Medical College and Hospital of eastern India from April to December 2024. Total 84 non-smoking participants {42 controlled essential hypertensives (≥1 year duration), 42 normotensive} aged 18-60 years underwent spirometric evaluation. Spirometric parameters like Forced Vital Capacity (FVC), Forced Expiratory Volume in 1 second (FEV_1), FEV_1/FVC ratio, Forced Expiratory Flow 25-75% (FEF_{25-75}%) and Peak Expiratory Flow Rate (PEFR) were measured using standardised American Thoracic Society (ATS)/European Respiratory Society (ERS) protocols. Data were statistically analysed by using Mann-Whitney U test.

Results: Participants were middle-aged adults, with a mean age of 43.38 years including both the genders. However, hypertensive participants were significantly older (50.6±8.81 vs. 36.17±15.03 years, p<0.0001) with higher Mean Arterial Pressure (MAP) (103.43±8.79 vs. 88.93±6.09 mmHg, p<0.0001). All spirometric parameters except FEV_1/FVC ratio were significantly impaired in hypertensives: FVC (68.52±12.77% vs. 100.6±15.69% predicted, p<0.0001), FEV_1 (71.17±16.93% vs. 107.12±14.62% predicted, p<0.0001), PEFR (44.86±28.04% vs. 95.57±32.7% predicted, p<0.0001), and FEF_{25-75}% (57.57±43.42% vs. 100.4±35.5% predicted, p<0.0001). Abnormal spirometric patterns occurred in 83.3% of hypertensives versus 9.5% of controls (OR=47.5, p<0.0001), with mixed defects predominating (59.5%). Strong negative correlations existed between MAP and spirometric indices (ρ=-0.75 for MAP-FVC).

Conclusion: The present study findings indicated that controlled essential hypertension significantly impairs lung function in Eastern Indians, necessitating routine spirometry for comprehensive risk assessment.

Keywords: Airway resistance, Cardiovascular complications, Pulmonary function tests, Respiratory mechanics

INTRODUCTION

Systemic arterial hypertension represents a leading global health challenge, affecting approximately one billion individuals worldwide and constituting a major modifiable risk factor for cardiovascular mortality [1]. The condition is defined in adults as Systolic Blood Pressure (SBP) ≥140 mmHg and/or Diastolic Blood Pressure (DBP) ≥90 mmHg, or any blood pressure level in patients receiving antihypertensive therapy [2,3].

Essential hypertension, characterised by elevated blood pressure without identifiable secondary causes, accounts for approximately 85% of all hypertensive cases [4,5]. Beyond its well-established cardiovascular and renal complications, emerging evidence suggests potential associations between hypertension and pulmonary function alterations, though this relationship remains incompletely characterised [6].

The physiological interplay between cardiovascular and respiratory systems has garnered increasing research attention [7]. Several studies have investigated the impact of hypertension on pulmonary function, yielding conflicting results. Some investigators have reported decreased pulmonary function parameters in hypertensive patients [8,9] while others have documented restrictive lung patterns, particularly in hypertensive women [7,10]. Conversely, certain studies have attributed respiratory function changes to antihypertensive medications rather than hypertension itself [8,11]. However, the literature has yet to establish a causal link between long-term antihypertensive use (up to 10 years) and the development of permanent bronchoconstriction.

Despite the high prevalence of hypertension in India, limited data exist regarding its effect on pulmonary function in this population [7]. Previous investigations [7,12,13] have primarily focused on patients with pre-existing pulmonary diseases rather than population-based assessments. Furthermore, ethnic variations in spirometric parameters across different geographical regions necessitate population specific studies.

Spirometry remains the gold standard for functional assessment of airways, offering a relatively simple, quick, and widely accessible diagnostic tool [14]. However, the association between blood pressure and spirometric parameters in the Indian population, particularly in Eastern India, remains largely unexplored. Hence, the present study aims to compare spirometric parameters between subjects with controlled essential hypertension and normotensive healthy controls in eastern India.

Null Hypothesis (H_0): There will be no significant difference in spirometric parameters between individuals with essential hypertension and normotensive controls in the Eastern Indian population.

Alternative Hypothesis (H_1): Significant differences exist in spirometric parameters between individuals with essential hypertension and normotensive controls.

MATERIALS AND METHODS

This cross-sectional study was conducted at Rampurhat Government Medical College and Hospital of West Bengal, India from April to December 2024. The study protocol received approval

from the Institutional Ethics Committee (IEC) {IEC Memo NO: IEC/2024/04/05, IEC registration no: ECR/1438/Inst/WB/2020 on 18.08.2020 (CDSCO), EC/NEW/INST/2021/2475 on 23.05.2022 (DHR)} and adhered to the ethical guidelines of the World Health Assembly and Helsinki Declaration (2013 amendment) [15]. Written informed consent was obtained from all participants in their preferred language (Bengali/Hindi/English).

Participants were recruited through purposive sampling from patients referred to various outpatient departments (General Medicine and Chest) for spirometric evaluation.

Inclusion criteria: Participants eligible for this current study were aged 18-60 years including both genders with either controlled essential hypertension of minimum one-year duration or normotensive status and subjects on antihypertensive medication(s) for not more than 10 years. All participants were required to be free from anatomical or clinical disorders that might affect respiratory system function and maintain legal residence in Eastern India. The age range was specifically selected to focus on the working-age population, thereby minimising age-related alterations in pulmonary mechanics that could confound the study results.

Exclusion criteria: Subjects with any history of smoking or substance abuse, pre-existing respiratory disorders, active haemoptysis, or any acute illness within the three weeks prior to the study; those who used drugs that influence bronchomotor tone, had a history of hypertension exceeding ten years, or presenting with uncontrolled or newly diagnosed cases additionally, systemic conditions like diabetes mellitus, hypothyroidism, and non-hypertensive cardiovascular abnormalities were excluded. Finally subjects who were, non-residents of Eastern India, trained endurance athletes, pregnant, lactating, postmenopausal, or experiencing dysfunctional uterine bleeding were excluded.

Sample size calculation: Based on the reported hypertension prevalence of 29.8% in India [16], with a rounded prevalence of 30% (p=0.30, q=0.70), margin of error of 10%, and 95% Confidence Interval (CI), the calculated sample size was:

$$n=4pq/L^2=(4\times30\times70)/(10\times10)=84$$

The study included 42 essential hypertensive subjects and 42 normotensive controls to maintain homogeneity.

Data Collection Procedures

Before appointing the subjects for this current study, they were instructed not to take any medication 24 hours prior to study after they were referred from Outpatient Department (OPD) for doing spirometry test (as few groups antihypertensive drugs like Angiotensin Converting Enzyme (ACE) inhibitors, non-selective beta blockers, calcium channel blockers are well known to cause acute bronchospasm).

Blood pressure measurement: Blood pressure was measured in the sitting position using standardised procedures with a digital sphygmomanometer (Dr. Odin OLS103 LCD Mercury Free Sphygmomanometer). MAP was calculated based on the SBP and DBP. Hypertension was defined as average SBP ≥140 mmHg or average DBP ≥90 mmHg (calculated from three measurements) or self-reported hypertension diagnosis [3].

Anthropometric measurements: Body weight was measured to the nearest 0.1 kg using a calibrated balance scale (Detecto, Webb City, MO). Height was measured without shoes using a stadiometer. Body Mass Index (BMI) was calculated as weight (kg)/height^2 (m^2) [17].

Spirometric testing: All spirometric evaluations were conducted between 10:00 AM and 1:00 PM in a controlled laboratory environment maintained at 18-28°C to ensure standardised testing conditions. Participants were instructed to empty their bladder prior to testing to minimise reflex sympathetic activation and transient blood pressure elevation induced by bladder distension [18]. A female attendant was present throughout the procedure when testing female participants to ensure comfort and compliance.

Spirometry was performed using a calibrated spirometer (Spirotech, Serial No: ST-ETSP-0067) following American Thoracic Society/ European Respiratory Society (ATS/ERS) 2005 guidelines [19].

Daily calibration was performed using a 3-L calibrated syringe according to ATS/ERS protocols, with the syringe checked for leaks and damage prior to each testing session. Participants received adequate coaching about the testing maneuvers prior to the procedure, and pulse oximetry was attached to monitor oxygen saturation throughout testing.

The following spirometric parameters were measured: FVC, FEV_1, FEV_1/FVC ratio, FEF_{25-75}% and PEFR. At least three acceptable and reproducible tests were performed for each participant, with the largest observed FEV_1 and FVC values used for final analysis. Bronchodilator reversibility testing was not performed as the study included only non-smoking participants with good respiratory health. Interpretation of spirometric indices and pattern classification were performed in accordance with established Indian guidelines to minimise ethnic and population-related bias [20].

Spirometric Pattern Classification

Results were classified according to established criteria to identify different patterns of pulmonary dysfunction. Normal spirometric results were defined as all parameters falling within normal limits according to established reference values. Obstructive patterns were characterised by FEV_1/FVC ratio less than 70% [21]. Restrictive Spirometric Pattern (RSP) was identified when FVC fell below 80% predicted, FEV_1 was 80% predicted or lower (normal or decreased), and FEV_1/FVC ratio remained at or above 0.7 (normal or increased) [22]. Small Airway Obstruction (SAO) was diagnosed when FEF_{25-75}% measured less than 50% of predicted values, indicating dysfunction in the peripheral airways. FEF_{25-75}% has been shown to reflect early bronchial and peripheral airway impairment even in the absence of overt obstructive spirometric changes, supporting its utility as a marker of small airway dysfunction [23].

STATISTICAL ANALYSIS

Data was entered into Microsoft Excel (version 2010) with double-entry verification and presented as mean±standard deviation for continuous variables and frequencies (percentages) for categorical variables. Normality testing was conducted subsequently using the Shapiro-Wilk test to determine appropriate statistical methods. Comparisons of continuous variables in between groups were analysed using the Mann-Whitney U test (two-tailed) and categorical variables were assessed using Fisher's-exact test for small cell counts and Chi-square test with Yates' correction for larger samples. To determine the association between blood pressure indices and spirometric metrics, non-parametric Spearman's rank correlation coefficient (ρ) was applied. Clinical effect size for non-parametric continuous distributions was quantified using Cohen's d or equivalent rank correlation ρ (small: 0.1, medium: 0. 3, large: 0.5). Relative risks were calculated via Odds Ratios (OR) with 95% CI, alongside the Number Needed to Harm (NNH). To control for the age imbalance between the two main groups, a secondary age-stratified sub-analysis was executed using independent Mann-Whitney U tests. The threshold for statistical significance was established at $p<0.05$. This Statistical analysis was performed using GraphPad QuickCalcs software (California, USA) and SPSS version 28.0 (IBM Corp., Armonk, NY, USA).

RESULTS

A total of 84 participants were enrolled and completed the study protocol without dropouts, comprising 42 individuals with essential hypertension and 42 normotensive controls. All participants met the inclusion criteria and underwent successful spirometric evaluation according to ATS/ERS guidelines.

In the present study, the HTN group is substantially older (50.6 vs. 36.17 years) and has a slightly higher BMI than the controls. All cardiovascular parameters- SBP, DBP, and MAP are significantly elevated in the HTN group with large effect sizes (r≥ 0.72), confirming a clear physiological distinction between the cohorts. Overall, the data indicates that age and blood pressure are the primary differentiating factors, with all comparisons reaching statistical significance (p<0.05), although gender distribution was found insignificant in study groups [Table/Fig-1].

Parameters		HTN group (n=42)	Control group (n=42)	p-value	Effect size (r)
Demographics					
Age (years)		50.6±8.81	36.17±15.03	<0.0001*	0.47 (large)
BMI (kg/m^2)		23.26±4.04	21.57±3.96	0.042*	0.22 (small)
Gender distribution	Male	30 (71.42%)	25 (59.52%)	0.358	-
	Female	12 (28.58%)	17 (40.48%)		
Blood pressure parameters					
SBP (mmHg)		137.6±11.46	117.86±10.78	<0.0001*	0.72 (large)
DBP (mmHg)		86.36±9.1	74.5±4.38	<0.0001*	0.72 (large)
MAP (mmHg)		103.43±8.79	88.93±6.09	<0.0001*	0.79 (large)

[Table/Fig-1]: Baseline characteristics of study participants.
HTN: Hypertension; SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure; BMI: Body mass index. Data are expressed as Mean±Standard Deviation (SD). *Indicates statistical significance at the p<0.05 level. The Mann-Whitney U test was used for all comparisons as the data distribution significantly deviated from normality. Effect size (r) is interpreted as small (0.1), medium (0.3), or large (0.5)
Effect size is expressed as r (correlation-based effect size), not Cohen's d

The HTN group had a higher prevalence of overweight (21.42%) and obesity (7.14%) compared to the control group (11.90% and 2.38%, respectively). However, with a p-value of 0.10 and a small effect size (0.12), these differences in BMI distribution between the two groups were not statistically significant [Table/Fig-2].

BMI categories*	HTN group (n=42)	Control group (n=42)	p-value	Effect size
Normal (18.5-24.9 kg/m^2)	30 (71.42%)	36 (85.71%)	0.10	0.12 (small)
Overweight (25-29.9)	9 (21.42%)	5 (11.90 %)		
Obese (≥30)	3 (7.14%)	1 (2.38 %)		

[Table/Fig-2]: BMI Classification comparison among groups.
BMI: Body mass index; HTN: Hypertension. Data are presented as frequency and percentage n (%) *Pearson's Chi-square test was applied with Yates' continuity correction due to small cell frequencies in the "Obese" category (expected frequency <5)

Participants with hypertension demonstrated significantly reduced pulmonary function compared with healthy controls. Both FVC and FEV_1 (% predicted) were markedly lower in the hypertension group, with large effect sizes indicating substantial impairment in ventilatory capacity. Flow-related indices, including PEFR and FEF_{25-75}%, were also significantly diminished, reflecting compromised large and small airway function. In contrast, the absolute FEV1/FVC ratio showed no significant difference between groups, suggesting preservation of the expiratory proportion and pointing to a restrictive or mixed ventilatory pattern rather than an obstructive defect [Table/Fig-3].

All spirometric parameters (FVC, FEV_1, PEFR, and FEF_{25-75}%) showed negative r values. This indicates that higher SBP, DBP, and MAP are correlated with lower lung volumes and flow rates. The strongest correlations were seen with MAP and FVC (ρ=-0.75). This suggests that overall average blood pressure has a particularly powerful inverse relationship with the total volume of air a person can exhale (FVC). The negative impact of high blood pressure was seen across both volume parameters (FVC, FEV_1) and flow parameters (PEFR, FEF_{25-75}%), though the correlation was slightly weaker for the small airways (FEF_{25-75}%) [Table/Fig-4].

Spirometric parameter	SBP (ρ) (p-value)	DBP (ρ) (p-value)	MAP (ρ) (p-value)
FVC (% pred)	-0.72 (<0.001)*	-0.68 (<0.001)*	-0.75 (<0.001)*
FEV_1 (% pred)	-0.69 (<0.001)*	-0.65 (<0.001)*	-0.73 (<0.001)*
PEFR (% pred)	-0.63 (<0.001)*	-0.59 (<0.001)*	-0.67 (<0.001)*
FEF_{25-75}% (% pred)	-0.45 (<0.001)*	-0.42 (0.002)*	-0.48 (<0.001)*

[Table/Fig-4]: Correlation between blood pressure and spirometric parameters among hypertensive subjects.
SBP: Systolic blood pressure; DBP: Diastolic blood pressure; MAP: Mean arterial pressure; FVC: Forced vital capacity; FEV_1: Forced expiratory volume in 1 second; PEFR: Peak expiratory flow rate; FEF_{25-75}%: Forced expiratory flow at 25–75%; % pred: percentage of predicted value. *Correlation is significant at the 0.01 level (2-tailed). Values represent Spearman's rank correlation coefficient (ρ)

An overwhelming majority 83.33% of the HTN group shows some form of lung abnormality, compared to only 9.5% of the control group. The OR of 47.5 is exceptionally high. This suggests that individuals with hypertension are roughly 48 times more likely to have a lung abnormality than those with normal blood pressure. The most common issue in the HTN group was a mixed defect (Restrictive + SAO), affecting 59.5% of the subjects. Nearly a quarter (23.8%) exhibited a pure restrictive pattern [Table/Fig-5].

Pattern	HTN group n=42	Control group n=42	OR (95% CI)	p-value	NNH
Normal pattern	7 (16.7%)	38 (90.5%)	1.0 (reference)	-	-
Any abnormality	35 (83.3%)	4 (9.5%)	47.5 (12.8 to 175.6)	<0.0001*	1.35
Mixed defect (RSP+SAO)	25 (59.5%)	0 (0.0%)	∞ (25.4-∞)	<0.001*	1.7
Pure RSP	10 (23.8%)	0 (0.0%)	∞ (5.9-∞)	<0.001*	4.2
Pure SAO	0 (0.0%)	4 (9.5%)	0.1 (0.01-1.93)	0.115	-

[Table/Fig-5]: Distribution of spirometric abnormalities with risk assessment.
HTN: Hypertension; OR: Odds ratio; CI: Confidence interval; NNH: Number needed to harm; RSP: Restrictive spirometric pattern; SAO: Small airway obstruction. Data are presented as frequency n (%)

Only 16.67% of the HTN group maintained normal FVC levels, whereas 100% of the control group was normal. The most frequent finding in the HTN group is Moderate Restriction (45.24%), affecting nearly half of the hypertensive participants. Over 21% of hypertensive subjects reached the moderately severe category (50%-59% predicted), a degree of impairment entirely absent in the control group [Table/Fig-6].

Note on cohort consistency: All 42 subjects in the control group demonstrated normal lung volumes (FVC ≥80% predicted). This

Parameters	HTN group (n=42)	Control group (n=42)	Mean difference (95% CI)	p-value	Effect size (Cohen's d)
FVC (% pred)	68.52±12.77	100.6±15.69	-32.08 (-38.29 to -25.87)	<0.001*	2.24 (large)
FEV_1 (% pred)	71.17±16.93	107.12±14.62	-35.95 (-42.82 to -29.08)	<0.001*	2.27 (large)
FEV_1/FVC raw ratio	0.81±0.06	0.83±0.04	-0.02(-0.04 to 0.00)	0.112	0.39 (negligible)
Flow parameters					
PEFR (% pred)	44.86±28.04	95.57±32.7	-50.71 (-63.93 to -37.49)	<0.001*	1.66 (large)
FEF_{25-75}% (% pred)	57.57±43.42	100.4±35.5	-42.83 (-60.05 to -25.61)	<0.001*	1.08 (large)

[Table/Fig-3]: Spirometric parameters with clinical significance.
HTN: Hypertension; FVC: Forced vital capacity; FEV_1: Forced expiratory volume in 1 second; PEFR: Peak expiratory flow rate; FEF_{25-75}%: Forced expiratory flow at 25–75% of vital capacity; % pred: percentage of predicted value; CI: Confidence interval. Data are presented as Mean±SD

Severity category	FVC criteria	HTN group	Control group	p-value
Normal	≥80% predicted	7 (16.67%)	42 (100%)	<0.0001*
Mild restriction	70-79% predicted	7 (16.67%)	0 (0%)	<0.0001*
Moderate restriction	60-69% predicted	19 (45.24%)	0 (0.0%)	<0.0001*
Moderately severe	50-59% predicted	9 (21.42%)	0 (0.0%)	<0.001*

[Table/Fig-6]: Severity distribution of restrictive pulmonary impairment based on FVC Criteria.
HTN: Hypertension; FVC: Forced vital capacity; % pred: percentage of predicted value. Data are expressed as frequency n (%); *Indicates statistical significance at the p < 0.05. Level using Pearson's Chi-square or Fisher's-exact test where appropriate

includes the four control participants (9.5%) identified in [Table/Fig-5] with Pure SAO, as their impairment was strictly limited to mid-expiratory flow rates ($FEF_{25-75\%}$ <50%predicted) without altering FVC.

Age-stratified analysis was performed to control the confounding effect of age on pulmonary function. The results demonstrate that the significant reduction in FVC and FEV_1 in the HTN group persists across both younger (18-40) and middle-aged (41-60) cohorts, suggesting that hypertensive status, rather than age alone, is the primary driver of the observed restrictive pattern. In both the 18-40 and 41-60 age groups, the HTN group showed significantly lower FVC and FEV_1 percentages compared to their respective age-matched controls (p<0.001). This indicates that hypertension-associated lung impairment is not merely a byproduct of aging but appears to be associated with the hypertensive state itself. Interestingly, the younger hypertensive group (18-40 years) showed slightly lower mean FVC (66.3%) and FEV_1 (66.9%) than the older hypertensive group (68.9% and 72.5%, respectively). This highlights that even in younger individual, hypertension can be associated with marked restrictive lung changes [Table/Fig-7].

Age group	Parameter	HTN group	Control group	Age-matched p-value
18-40 years	n	10	24	
	FVC (% pred)	66.3±12.46	98.38±15.35	<0.001*
	FEV_1 (% pred)	66.9±12.61	106.00±14.42	<0.001*
41-60 years	n	32	18	-
	FVC (% pred)	68.90±13.04	103.56±16.074	<0.001*
	FEV_1 (% pred)	72.5±18.04	108.61±14.42	<0.001*

[Table/Fig-7]: Age-stratified spirometric parameters.
HTN: Hypertension; FVC: Forced vital capacity; FEV_1: Forced expiratory volume in 1 second; % pred: percentage of predicted value. Data are expressed as Mean±SD; *Indicates statistical significance at p<0.05.

DISCUSSION

Hypertension in Eastern Indian adults is strongly linked to impaired lung function, with FVC and FEV_1 reduced by 32% and 33.6%, respectively supporting the alternate hypothesis. High ORs (47.5) and negative correlations with MAP (ρ= -0.75) suggest a mechanistic dose-response relationship, necessitating the integration of pulmonary screening into routine hypertensive care [24,25]. Chronic hypertension likely triggers left ventricular dysfunction, elevating left atrial and pulmonary capillary wedge pressures. This causes interstitial fluid accumulation and subclinical oedema, manifesting as restrictive patterns in 83.3% of hypertensive participants. Notably, 59.5% demonstrated mixed defects. These spirometric impairments appear early in disease progression, likely independent of long-term antihypertensive medication effects or secondary environmental exposures [20,26,27]. Findings from the normative aging study support this bidirectional relationship, where lower FVC independently predicts hypertension risk in normotensive adults [28]. However, causality cannot be established between antihypertensive use and spirometric outcomes.

The present study findings align with previous international studies while demonstrating considerable severity of impairment [29]. Margretardottir OB et al., (2009) demonstrated independent associations between hypertension and reduced FEV_1 and FVC values, but the magnitude of reduction in our Eastern Indian population (FVC: 68.52% vs. 100.6% predicted; FEV_1: 71.17% vs. 107.12% predicted) represents clinically significant impairment requiring intervention [30]. Similarly, Patel FO et al., (2022) reported comparable reductions in Indian populations, supporting the generalisability of the present findings within the Indian subcontinent [31].

The predominance of RSPs in our study (83.33% abnormal patterns) contrasts with Shah S et al., (2014), who primarily observed obstructive patterns [7]. However, their study did acknowledge restrictive changes, and the discrepancy may reflect differences in hypertension severity, duration, or population characteristics. The present age-stratified analysis confirming significant differences even after controlling age strengthens the argument for a true association independent of demographic confounders.

The severity distribution reveals 66.66% of hypertensive patients had moderate to moderately severe restriction (FVC: 50-69%), while only 16.67% remained normal. The preserved absolute FEV1/FVC ratio (0.81±0.06) confirms a primary restrictive pattern [Table/Fig-3]. This profile suggests hypertension-associated pulmonary dysfunction is a clinically significant complication driven by parenchymal and vascular changes rather than traditional obstructive airway disease. FVC < 80% predicted independently predicts increased cardiovascular morbidity and mortality, making it a critical high-risk biomarker [32,33]. A total of 83.33% of hypertensive patients fell below this threshold, necessitating intensive monitoring.

Significant FEF25-75% reduction (p<0.001) suggests hypertension-induced small airway dysfunction, with 59.5% of patients demonstrating mixed restrictive-obstructive defects [Table/Fig-4] [34]. The elevated MAP values in our hypertensive group likely indicate increased peripheral vascular resistance, potentially compromising blood supply to muscles surrounding small airways and impairing ventilatory function [23,34]. This finding suggests that hypertension's effects on pulmonary circulation mirror its systemic vascular impact, providing evidence for a unified pathophysiological mechanism.

Hypertensive patients showed severe PEFR reduction, reflecting significant functional airflow limitation beyond FEV1 measurement [35,36]. Zhang Y et al., prospective study from Eastern China demonstrated that reduced peak expiratory flow predicts increased cardiovascular disease incidence, all-cause mortality, and cardiovascular mortality [37]. These findings suggest that hypertensive patients in Eastern India may face similar prognostic risks, emphasising the need for integrated cardiopulmonary risk assessment.

The persistence of significant differences across age strata (18-40 years and 41-60 years) strengthens the argument for a causal relationship between hypertension and pulmonary dysfunction [38]. Even in younger hypertensive patients (18-40 years), significantly reduced FVC%and FEV1% (p<0.001) indicate that pulmonary impairment is a direct pathophysiological consequence, not merely aging.

This first comprehensive study in Eastern India reveals a predominantly restrictive pattern, potentially reflecting local genetic or lifestyle factors. While the hypertensive group was older and had higher BMI, age-stratified analysis confirms that pulmonary impairment remains independently associated with hypertensive status.

High clinical impact (OR=47.5 and NNH=1.35) supports routine pulmonary screening for hypertensive patients. Strong correlations suggest aggressive blood pressure control could preserve lung function, though prospective studies are required for confirmation.

Furthermore, the presence of pure SAO in 9.5% of the control group contrasted with its complete absence in the hypertensive group (0%). While we tentatively label this observation a 'Small Airway Paradox'-hypothesising that distal airway dysfunction in hypertensive patients may progress rapidly to, or co-exist with, an overt restrictive pattern-it is critical to note that this specific distribution difference was statistically non-significant (p=0.115, [Table/Fig-5]). Therefore, this baseline trend requires validation in larger, longitudinal cohorts to determine if hypertension actively masks or accelerates early small airway remodeling.

Primary care physicians should integrate pulmonary testing into cardiovascular risk assessments for hypertensive patients, facilitating early risk stratification and intervention. The identification of severe restrictive patterns in controlled hypertensives suggests the need for more intensive cardiovascular management and possible pulmonary rehabilitation referrals.

Limitation(s)

Study limitations include cross-sectional design and single-centre sampling. While age differences were addressed via stratification and homogeneous gender distribution was observed in study groups, gender-wise comparative spirometric analysis and specific antihypertensive medication effects remain unexamined. The wide 95% confidence interval around the odds ratio indicates imprecision in the estimate, which may be attributed to the relatively small sample size and limited number of events.

CONCLUSION(S)

This study demonstrates a profound association between controlled essential hypertension and severely impaired pulmonary function in Eastern Indian adults, characterised by RSPs with concurrent small airway dysfunction. The 47.5-fold increased odds of abnormal spirometry, combined with large effect sizes and strong dose-response relationships, suggest this association as clinically significant and potentially life-threatening. The most severely affected parameter, PEFR (reduced to 44.86% of predicted), along with substantial reductions in FVC and FEV_1, mandate integration of pulmonary function assessment into routine hypertension management and highlight the need for comprehensive cardiopulmonary risk stratification in clinical practice. Future longitudinal research is required to confirm causality and evaluate whether pulmonary impairment stems from hypertension itself or its long-term pharmacological management. Further studies should focus on intensive blood pressure control, cellular mechanisms, and molecular biomarkers. Additionally, investigating why the FEV1/FVC ratio remains preserved despite significant impairment is crucial to understanding the underlying pathophysiology of hypertension-associated restrictive lung disease.

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