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Correlation Between Obesity and Lung Function: A Cross-Sectional Study

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Abstract: ***Background: Obesity globally impacts respiratory mechanics and increases obstructive sleep apnea (OSA) risk, particularly in Asian Indians who develop metabolic risks at lower BMI thresholds. This prospective cross-sectional study aimed to correlate obesity severity with pulmonary function test (PFT) parameters and OSA risk. Aim: To determine the correlation between obesity (BMI ≥25 kg/m²) and pulmonary function test values. Methods: Conducted at Sri Siddhartha Medical College, the study included 36 adults (18–60 years) with a BMI ≥ 25 kg/m². Participants underwent spirometry and STOP-BANG questionnaire screening. Results: Results indicated a predominantly restrictive ventilatory defect, with a mean pre-bronchodilator Forced Vital Capacity (FVC) of 75.4% and Forced Expiratory Volume in 1 second (FEV_1) of 78.9% of predicted values, alongside a preserved FEV_1/FVC ratio (82.3%). BMI demonstrated a strong negative dose-dependent correlation with FVC% ($r = -0.72$, $P < 0.001$) and FEV_1% ($r = -0.61$, $P < 0.001$). The 36.1% of participants categorized as high-risk for OSA exhibited significantly lower FVC% compared to low-risk subjects (65.7% vs. 84.1%, $P < 0.001$). Conclusion: Obesity causes a measurable restrictive impairment of lung volumes and exacerbates OSA risk. Early spirometric and clinical screening in obese individuals is essential to identify functional impairment and prevent progressive respiratory morbidity.***

Keywords: Obesity, Spirometry, Sleep Apnea Syndromes, Body Mass Index, Respiratory Function Tests

1. Introduction

Obesity has reached pandemic proportions, evolving into one of the most critical public health challenges globally [1]. In India, rapid urbanization, dietary transitions, and sedentary lifestyles have driven a sharp rise in obesity prevalence [2]. Unlike Western demographics, Asian Indians are predisposed to central adiposity and obesity-related metabolic risks at significantly lower Body Mass Index (BMI) thresholds. Consequently, international guidelines recommend a reduced BMI cutoff (≥ 25 kg/m²) to define obesity in this demographic [3].

Beyond cardiometabolic complications, excessive adiposity fundamentally alters pulmonary physiology. Fat deposition on the thoracic and abdominal walls mechanically loads the respiratory system, reducing chest wall compliance and limiting diaphragmatic excursion [4]. These structural alterations primarily manifest as a restrictive ventilatory defect, diminishing functional residual capacity (FRC) and expiratory reserve volume (ERV). Furthermore, the reduction in lung volumes compromises upper airway tethering, establishing obesity as the strongest modifiable risk factor for obstructive sleep apnea (OSA) [5]. Recognizing the integrated impact of these pathophysiological changes is vital for the early detection and mitigation of long-term respiratory morbidity.

2. Literature Survey

Epidemiological and clinical studies consistently link increasing BMI with declining pulmonary mechanics. A systematic review by Melo et al. demonstrated marked reductions in FEV_1 and FVC among obese adults with a preserved FEV_1/FVC ratio, confirming a predominantly restrictive pattern [6]. Banerjee et al. further highlighted negative correlations between BMI and mid-expiratory flow rates (FEF25-75%) in an Indian cohort, noting gender-specific variations in spirometric decline [7].

Regarding sleep-disordered breathing, Hao et al. evaluated pulmonary function parameters as predictors of OSA severity, identifying a stepwise increase in OSA severity with rising BMI and demonstrating that severe OSA strongly correlates with altered ventilatory parameters [8]. Despite this global consensus, data explicitly correlating incremental BMI, comprehensive spirometric variables, and clinical OSA risk—utilizing validated screening tools like the STOP-BANG questionnaire- remain limited within the Asian Indian phenotype, where restrictive respiratory mechanics occur at lower total body weights.

3. Problem Definition

Current literature lacks an integrated assessment of objective spirometric alterations and subjective OSA risk specifically tailored to the Asian Indian obesity criteria (BMI ≥ 25 kg/m²). Because restrictive mechanics occur at lower BMI thresholds in this demographic, clinicians frequently underdiagnose

impending respiratory dysfunction and sleep-disordered breathing.

This study addresses this specific clinical gap by formulating the following research question: *How does incremental obesity severity (measured via Asian Indian BMI cutoffs) correlate with objective spirometric indices and clinical OSA risk in an adult Indian cohort?*

4. Aim and Objectives

Aim of the study

To determine the correlation between people with obesity (BMI >25kg/m2) and their Pulmonary function test values.

Objectives of the Study

1) **To determine Pulmonary Function Test in people with obesity**
- To compare lung function values (FEV1, FVC, FEV1/FVC ratio, FEF25–75, etc.) across individuals with BMI ≥25 kg/m².
- To determine whether obesity is associated with a restrictive or obstructive pattern in spirometry.

2) **To determine Pulmonary Function Test in people with obesity and Obstructive Sleep apnoea (by STOP BANG questionnaires).**
- To correlate spirometric parameters with STOP-BANG questionnaire scores.
- To examine whether individuals at intermediate or high risk for obstructive sleep apnea demonstrate significant alterations in lung function compared to those at low risk.

3) **Find correlation between obesity and higher scores of Sleep Apnea (by STOP BANG questionnaires).**
- To analyze whether the interaction of obesity (BMI) and risk for obstructive sleep apnea has an additive or independent impact on spirometric outcomes.

5. Methodology / Approach

Study Design and Setting

A prospective cross-sectional study was conducted at the Department of Respiratory Medicine, Sri Siddhartha Medical College and Hospital, Tumkur, India, spanning a 24-month period. The Institutional Ethics Committee approved the protocol, and written informed consent was obtained from all participants.

Participant Selection

A purposive sampling technique was utilized. The sample size was calculated using a known correlation coefficient ($r = 0.531$) between BMI and FEV_1; achieving 80% statistical power at a 99% confidence interval required a minimum of 36 subjects. Inclusion criteria mandated adults aged 18–60 years with a BMI ≥ 25 kg/m². Exclusion criteria eliminated individuals with acute respiratory infections, known chronic respiratory conditions (e.g., COPD, asthma), or contraindications to performing reliable spirometry (e.g., recent myocardial infarction, thoracic surgery).

Clinical and Spirometric Evaluation

Anthropometric measurements were recorded, and participants were categorized into Obese I (25–29.9 kg/m²) and Obese II (≥ 30 kg/m²) cohorts. The validated 8-item STOP-BANG questionnaire was administered to stratify OSA risk into low (0–2), intermediate (3–4), and high (≥ 5) risk categories. Standardized pulmonary function testing was performed adhering to American Thoracic Society/European Respiratory Society (ATS/ERS) guidelines. Three reproducible maneuvers were obtained, recording the best values for Forced Vital Capacity (FVC), Forced Expiratory Volume in 1 second (FEV_1), FEV_1/FVC ratio, and Forced Expiratory Flow (FEF25-75%).

Statistical Analysis

Data were analyzed using SPSS version 21.0. Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as frequencies. Pearson correlation coefficients assessed the linear associations between BMI and spirometric parameters. Chi-square tests evaluated categorical distribution across OSA risk groups. Statistical significance was established at $P < 0.05$.

6. Results & Discussion

6.1 Results

The cohort consisted of 36 participants (66.7% male) with a mean age of 42.17 ± 10.62 years and a mean BMI of 29.42 ± 2.31 kg/m². The majority of participants (72.2%) belonged to the Obese I category, while 27.8% were classified as Obese II. Subjective sleep disturbances were reported by 58.3% of the cohort.

Table 1: BMI Category Distribution

BMI category	n	%
Obese I (25–29.9 kg/m²)	26	72.2
Obese II (≥30 kg/m²)	10	27.8

Baseline spirometry revealed a predominantly restrictive ventilatory pattern. The mean pre-bronchodilator FEV_1 was 2.05 ± 0.48 L (78.9% ± 8.6% of predicted), and FVC was 2.45 ± 0.46 L (75.4% ± 9.2% of predicted). The FEV_1/FVC ratio remained preserved at 82.3% ± 4.1%. When stratified by BMI, individuals in the Obese II category demonstrated significantly lower percent predicted FEV_1 (71.8% vs. 82.6%, $P < 0.001$) and FVC (66.4% vs. 79.3%, $P < 0.001$) compared to the Obese I group.

Table 2: Overall Mean Anthropometric and Spirometric Parameters

Parameter	Mean ± SD
Age (years)	42.17 ± 10.62
BMI (kg/m²)	29.42 ± 2.31
Neck circumference (cm)	42.01 ± 3.12
Pre-FEV₁ (L)	2.05 ± 0.48
Pre-FEV₁ (% predicted)	78.9 ± 8.6
Pre-FVC (L)	2.45 ± 0.46
Pre-FVC (% predicted)	75.4 ± 9.2
Pre-FEV₁/FVC (%)	82.3 ± 4.1
Pre-FEF25–75% (L/s)	2.01 ± 0.37
MVV (L/min)	99.8 ± 14.9

Pearson analysis established a strong, dose-dependent inverse relationship between adiposity and lung volumes. BMI correlated negatively with predicted FVC% ($r = -0.72$, 95% CI [−0.85, −0.51], $P < 0.001$) and predicted FEV₁% ($r = -0.61$, 95% CI [−0.77, −0.37], $P < 0.001$). Conversely, a weak positive correlation was observed between BMI and the FEV₁/FVC ratio ($r = +0.32$, $P = 0.04$).

Table 3: Comparison of Mean Spirometric Parameters by BMI Category

Parameter	Obese I (n = 26) Mean ± SD	Obese II (n = 10) Mean ± SD	F value	p-value
Pre-FEV₁ (L)	2.22 ± 0.31	1.78 ± 0.34	18.62	<0.001
Pre-FEV₁ (% predicted)	82.6 ± 6.4	71.8 ± 7.2	21.14	<0.001
Pre-FVC (L)	2.62 ± 0.29	2.02 ± 0.33	29.87	<0.001
Pre-FVC (% predicted)	79.3 ± 7.1	66.4 ± 8.1	26.41	<0.001
Pre- FEV₁/FVC (%)	81.9 ± 2.8	84.1 ± 3.2	6.54	0.01
Pre-FEF25– 75% (L/s)	2.18 ± 0.21	1.78 ± 0.26	24.96	<0.001

Table 4: Correlation Between BMI and Spirometric Parameters

Parameter	Pearson r	p- value	95% CI (Lower)	95% CI (Upper)
BMI vs Pre-FEV₁ (L)	−0.56	<0.001	−0.73	−0.30
BMI vs Pre-FEV₁ (% predicted)	−0.61	<0.001	−0.77	−0.37
BMI vs Pre-FVC (L)	−0.68	<0.001	−0.82	−0.46
BMI vs Pre-FVC (% predicted)	−0.72	<0.001	−0.85	−0.51
BMI vs Pre- FEV₁/FVC (%)	+0.32	0.04	+0.02	+0.57
BMI vs Pre-FEF25– 75% (L/s)	−0.48	0.003	−0.68	−0.20

STOP-BANG stratification categorized 41.7% of subjects as low risk, 22.2% as intermediate risk, and 36.1% as high risk for OSA. High-risk participants exhibited severe pulmonary impairment, recording an average predicted FVC of 65.7%, compared to 84.1% in the low-risk group ($P < 0.001$).

Table 5: Comparison of Mean Spirometric Parameters by OSA Risk Category

Parameter	High risk (n=13) Mean ± SD	Intermediate (n=8) Mean ± SD	Low risk (n=15) Mean ± SD	F value	p- value
Pre-FEV₁ (L)	1.62 ± 0.34	2.01 ± 0.26	2.48 ± 0.28	28.73	<0.001
Pre-FEV₁ (% predicted)	70.4 ± 6.8	79.3 ± 6.1	86.9 ± 5.4	34.21	<0.001
Pre-FVC (L)	2.05 ± 0.31	2.33 ± 0.19	2.87 ± 0.34	31.66	<0.001
Pre-FVC (% predicted)	65.7 ± 7.4	75.6 ± 6.8	84.1 ± 6.9	29.94	<0.001
Pre- FEV₁/FVC (%)	83.1 ± 2.6	82.0 ± 2.1	80.4 ± 1.9	8.92	<0.001
Pre- FEF25– 75% (L/s)	1.74 ± 0.18	1.98 ± 0.17	2.36 ± 0.23	42.17	<0.001

6.2 Discussion

The data indicate that obesity severity directly dictates the degree of restrictive pulmonary impairment. The concurrent decline in FVC and FEV₁, alongside a preserved FEV₁/FVC ratio, confirms findings by Melo et al. [6]. Pathophysiologically, excess visceral and thoracic adiposity structurally impedes diaphragmatic descent and drastically reduces chest wall compliance [9].

This mechanical loading leads to premature small airway closure at lower lung volumes, driving the significant reduction in FEF25-75% observed in this cohort. The loss of FRC fundamentally destabilizes upper airway tethering. As demonstrated, participants with the most severe restrictive deficits (Obese II) were overwhelmingly categorized into the high-risk STOP-BANG group. This confirms that obesity-induced restrictive physiology is not an isolated mechanical defect but a direct catalyst for nocturnal airway collapse and heightened OSA severity.

The study population was predominantly middle-aged, with a higher proportion of males and urban residents, consistent with profiles in prior literature [8]. Male predominance among high-risk OSA participants aligns with the recognized sex-specific anatomical and physiological factors in airway

collapsibility. Urban residence and sedentary occupation profiles further support the role of lifestyle in compounding obesity-related respiratory risk.

Association analyses demonstrated that increasing age, male sex, higher BMI category, urban residence, and disturbed sleep quality were each significantly associated with elevated OSA risk (all $P < 0.001$). These multivariate determinants reinforce that OSA risk in obese individuals is not a function of BMI alone but reflects an interplay of metabolic, anatomical, and behavioral factors.

7. Conclusion

Obesity induces a marked, dose-dependent restrictive impairment in pulmonary mechanics, quantified by simultaneous declines in FVC and FEV_1 with preserved FEV_1/FVC ratios. This mechanical restriction strongly correlates with an elevated clinical risk for obstructive sleep apnea. Increasing BMI category and OSA risk are independently and combinatorially associated with worsening spirometric parameters. Routine early integration of spirometry and STOP-BANG screening in obese patients offers critical clinical value, allowing providers to identify functional impairment prior to the onset of irreversible cardiovascular and respiratory morbidities.

8. Future Scope

This study acknowledges limitations inherent to its cross-sectional design, which precludes drawing definitive causal inferences regarding weight gain timelines and PFT decline. Additionally, the sample size of 36 participants restricts broad epidemiological generalization, and reliance on the STOP-BANG questionnaire lacks the diagnostic finality of gold-standard polysomnography (PSG). Future multicentric studies utilizing large longitudinal cohorts should incorporate comprehensive PSG and whole-body plethysmography to map exact respiratory mechanical trajectories and quantify recovery following targeted weight loss interventions.

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