



Association between Weight-adjusted Waist Index and Pulmonary Function: A Population-Based Study from the NHANES (2007–2012)

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Abstract

Background: The relationship between central obesity and pulmonary function remains a critical area of investigation. The Weight-Adjusted Waist Index (WWI) has emerged as a novel marker for assessing central obesity. This study aimed to investigate the association between WWI and various pulmonary function indices.

Methods: Data were extracted from the National Health and Nutrition Examination Survey (NHANES) 2007–2012, involving 8,361 participants. Participants were categorized into tertiles based on their WWI values. Pulmonary function measures included Forced Vital Capacity (FVC), Forced Expiratory Volume in one second (FEV₁), FEV₁/FVC ratio, and Peak Expiratory Flow rate at 25–75% of forced vital capacity (PEF_{25–75%}). Multivariate linear regression models were used to assess associations, adjusting for covariates such as gender, age, race, education level, hypertension, diabetes, smoking status, and poverty index ratio.

Results: Higher WWI was significantly associated with lower FVC, FEV₁, and PEF_{25–75%}, but not with FEV₁/FVC ratio. Specifically, each unit increase in WWI corresponded to reductions in FVC (–603.9 ml, 95% CI: –628.7, –579.2), FEV₁ (–527.7 ml, 95% CI: –547.7, –507.7), and PEF_{25–75%} (–550.5 ml/s, 95% CI: –581.7, –519.3). Sensitivity analyses confirmed these findings after adjusting for multiple covariates. Subgroup analyses indicated similar trends across different genders and age groups.

Conclusion: The Weight-Adjusted Waist Index (WWI) is negatively associated with lung volumes and mid-expiratory flow rates, suggesting its potential role in respiratory impairment. These results underscore the importance of considering central adiposity in clinical evaluations of pulmonary health. Future longitudinal studies are warranted to further elucidate the mechanisms underlying these associations and to validate these findings in diverse populations.

Keywords

Weight-Adjusted Waist Index, Pulmonary Function, NHANES, Obesity

Abbreviations

WWI: Weight-Adjusted Waist Index; WC: Waist Circumference; BMI: Body Mass Index; FRC: Functional Residual Capacity; FEV₁: Forced Expiratory Volume in One Second; NHANES: National Health and Nutrition Examination

Survey; PEF 25-75%: Mid-Exhalation Forced Expiratory Flow Rate

Introduction

The global obesity epidemic has evolved from a mere concern over weight gain to a complex public health crisis characterized by central obesity and its associated metabolic complications. The World Health Organization predicts that by 2025, one in every five adults worldwide will be obese or overweight [1]. Obesity not only impacts quality of life but also significantly increases the risk for numerous chronic diseases, including cardiovascular disease, type 2 diabetes mellitus (T2DM), cancer, and respiratory disorders [2].

Traditional measures such as Body Mass Index (BMI) and waist circumference (WC) have limitations in assessing obesity-related health risks. BMI, which reflects overall fat and muscle mass, fails to distinguish between adipose tissue and lean body mass, nor does it assess fat distribution patterns [3]. For instance, an athlete with high muscle mass may be classified as "obese," while an individual with normal weight but high body fat percentage might be underdiagnosed [4].

WC, although useful in indicating abdominal fat accumulation, does not account for height differences across individuals, complicating standardization [5]. In contrast, the weight-adjusted waist index (WWI), calculated as waist circumference (cm) divided by the square root of weight (kg), provides a more comprehensive assessment of fat distribution and body composition [6]. Studies have shown that WWI is more accurately reflective of visceral fat distribution, demonstrating a linear positive correlation with metabolic syndrome, cardiovascular diseases, and diabetes, thereby offering unique advantages in health risk prediction [2,7-9].

Numerous prospective studies have confirmed significant associations between WWI and metabolic diseases. A large cohort study involving 6,506 patients with metabolic syndrome found that each unit increase in WWI was associated with a 48% increase in all-cause mortality and a 67% increase in cardiovascular disease mortality [8]. Compared to BMI and WC, WWI exhibited superior predictive performance for all-cause

and cardiovascular mortality, with areas under the curve (AUCs) of 0.596 and 0.589, respectively, compared to BMI (0.576) and WC (0.523) [8].

Additionally, WWI has been linked to elevated urinary albumin-to-creatinine ratios even in individuals with normal BMI, suggesting its potential role in early detection of renal dysfunction [2]. In diabetes research, WWI has emerged as an independent predictor of diabetic kidney disease, with an AUC of 0.664, significantly higher than that of BMI (0.555), indicating its ability to identify diabetes complications earlier [7].

These findings collectively underscore the scientific foundation of WWI as a novel obesity assessment metric, capable of more precisely capturing the adverse effects of central obesity on metabolic health.

Concurrently, the impact of obesity on pulmonary function has become a critical area of respiratory research. Obesity influences lung function through multiple pathophysiological mechanisms, including mechanical compression, inflammatory responses, and sleep-disordered breathing [10].

Mechanical compression occurs when abdominal fat exerts upward pressure on the diaphragm, restricting its downward movement and reducing lung expansion capacity, leading to a significant decrease in functional residual capacity [11]. Studies have demonstrated reductions in FRC by up to 10%, 22%, and 33% among overweight, mildly obese, and severely obese individuals without asthma, respectively [12]. Increased thoracic wall fat further restricts rib cage expansion, diminishing lung compliance and ventilation efficiency [12]. Clinical data show that in obese individuals with a BMI ≥ 30 , both FEV₁ and FVC are significantly reduced, indicating impaired gas exchange and retention [13].

Despite extensive research on the relationship between obesity and lung function, no prior research has directly examined the relationship between WWI and lung function indices, nor verified whether WWI outperforms traditional metrics like BMI and WC in predicting lung function impairment. Given existing

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evidence, we hypothesize that WWI, as an independent indicator of central obesity, may uniquely influence lung function.

This study sought to explore the association between WWI and pulmonary function among the general adult population in the United States using the data from the National Health and Nutrition Examination Survey (NHANES).

Materials and Methods

Study Population:

This study utilized data from the National Health and Nutrition Examination Survey (NHANES) conducted between 2007 and 2012. Detailed descriptions and protocols about the NHANES can be found online [14].

A total of 30,443 individuals were initially enrolled. After excluding participants with missing weight-adjusted waist index (WWI) data ($n = 7,102$), pregnancy status ($n = 161$), missing pulmonary function data or low-quality data ($n = 564 + 2,917$), aged under 18 years ($n = 4,024$), with respiratory problems ($n = 1,726$), or missing education, hypertension, diabetes, and smoking status data, a final sample of 8,361 participants was included in the analysis. The selection process is illustrated in (Fig-1).

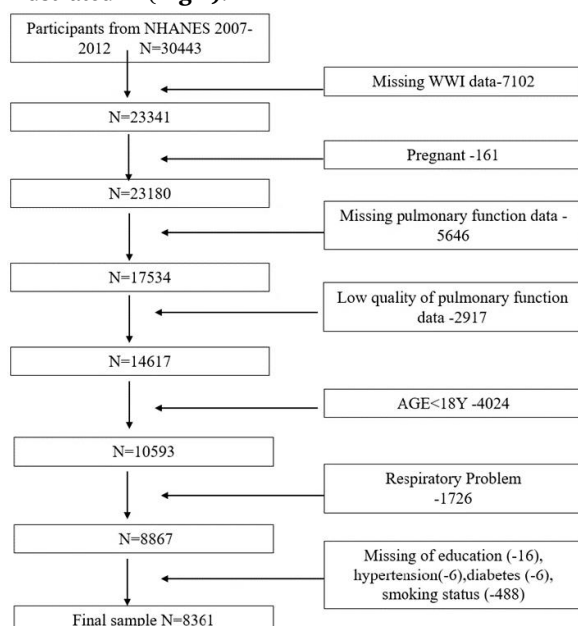


Fig-1: Flow Diagram of the Sample Selection

Assessment of WWI and Covariates:

WWI was calculated as waist circumference (cm)

divided by the square root of weight (kg). Demographic covariates in this study included gender (male/female), age (years), race (Mexican American/other Hispanic/non-Hispanic White/non-Hispanic Black/other races), education level (high school or below/college or above), hypertension, diabetes status (yes/no/borderline), smoking status (yes/no), and poverty index ratio.

Assessment of Pulmonary Function:

Participants aged 18 years and older were eligible for spirometry testing. Specific exclusion criteria included chest pain at the time of examination, recent surgery of the eye, chest, or abdomen, tuberculosis exposure, or physical problems affecting forceful expiration [15].

Spirometry was performed using a standardized protocol according to the recommendations of the American Thoracic Society (ATS) and the European Respiratory Society (ERS). The quality of the FEV₁ and FVC values was rated on a scale from A to F [15]. Participants stood (or sat if unable), wore a nose clip, and performed maximal forced exhalations. Individuals aged 18–79 were instructed to exhale for ≥ 6 seconds. Testing continued until three acceptable curves were obtained, two of which met reproducibility criteria (≤ 150 mL difference in FVC and FEV₁). A post-bronchodilator test followed the same procedure.

Quality control included NIOSH-certified training, ongoing expert review, direct performance monitoring, and regular equipment calibration. The spirometry dataset contained raw values of the highest overall value of FVC estimated in milliliters, FEV₁ in milliliters, and mid-exhalation forced expiratory flow rate (FEF 25–75%) in milliliters per second from reproducible curves that met or exceeded ATS standards. The ratio of FEV₁ to FVC was derived by dividing FEV₁ by FVC.

Statistical Analysis:

The statistical analysis was conducted using the statistical computing and graphics software R (version 4.2.3) and EmpowerStats (version 4.1). Baseline tables for the study population were statistically described by WWI tertiles; continuous variables are described using Mean $\pm$ SD and weighted linear regression models.

The beta values and 95% confidence intervals were calculated using multivariate linear regression analysis between the WWI and pulmonary function measures including FVC, FEV₁, PEF_{25–75%}, and FEV₁/FVC. The multivariate test was built using three models: Model 1: no variables adjusted; Model 2: adjusted for gender, age, and race; Model 3: adjusted for all covariates (Gender; Age; Race; Education; Poverty index ratio; Hypertension status; Diabetes status; Smoking status). By adjusting the variables, smoothed curve fits were done simultaneously. It was determined that $P < 0.05$ was statistically significant. We used a weighting approach to reduce the significant volatility of our dataset.

Results

Baseline Characteristics of Participants:

Table-1 summarizes the demographic characteristics of the 8,361 eligible participants stratified by WWI tertile. The mean ($\pm$ SD) age was 46.0 ($\pm$ 16.1) years, and 50.5% of the participants were male. Mean ($\pm$ SD) WWI values for low, middle, and high tertiles were 10 ($\pm$ 0.4), 10.9 ($\pm$ 0.4), and 11.8 ($\pm$ 0.4), respectively. Mean ($\pm$ SD) values for FVC, FEV₁, PEF_{25–75%}, and FEV₁/FVC were 3989.3 ($\pm$ 1067.1), 3140.8 ($\pm$ 878.2), 3015.3 ($\pm$ 1269.1), and 78.8 ($\pm$ 7.3), respectively.

Participants in the highest WWI tertile were significantly older, more likely to be female, Mexican American, or other Hispanic, had lower educational levels and poverty index ratios, and higher rates of hypertension and diabetes compared to those in lower tertiles. Higher WWI was associated with significantly lower FEV₁, FVC, and PEF_{25–75%}, but not FEV₁/FVC (**Table-1**).

Table-1: Baseline Characteristics of Participants Across WWI Tertiles

WWI Characteristic	Overall N=8361	Low (10±0.4) n=2784	Middle (10.9±0.4) n=2783	High (11.8±0.4) n=2784	P-value
Age (Years)	46.0 ± 16.1	37.33 ± 13.33	46.68 ± 14.83	53.90 ± 15.52	<0.001
Gender (%)					<0.001
Male	4219 (50.5%)	57.54	51.1	38.62	
Female	4132 (49.5%)	42.46	48.9	61.38	
Ethnicity (%)					<0.001
Mexican American	1462 (17.5%)	5.29	9.82	12.54	
Other Hispanic	943 (11.3%)	4.63	5.79	6.63	
Non-Hispanic White	3612 (43.3%)	69.84	68.55	67.2	
Non-Hispanic Black	1670 (20.0%)	12.51	9.17	8.59	
Other race	664 (8.0%)	7.73	6.68	5.05	
Education level(%)					<0.001
High school or below	3867 (46.3%)	28.44	37.99	46.79	
College or above	4484 (53.7%)	71.56	62.01	53.21	
Hypertension(%)					
Yes	2372 (28.4%)	11.87	26.45	42.04	
No	5979 (71.6%)	88.13	73.55	57.96	
Diabetes(%)					<0.001
Yes	768 (9.2%)	1.85	4.48	15.13	
No	7440 (89.1%)	97.36	93.75	82.22	
Borderline	143 (1.7%)	0.79	1.77	2.66	
Smoking (%)					<0.001
Yes	3514 (42.1%)	37.68	43.85	45.17	
No	4837 (57.9%)	62.32	56.15	54.83	
Poverty index ratio	2.7 ± 1.7	2.83 ± 1.69	2.70 ± 1.65	2.44 ± 1.60	<0.001
FVC	3989.3 ± 1067.1	4504.94 ± 1049.50	4029.32 ± 964.83	3433.54 ± 898.77	<0.001
FEV ₁	3140.8 ± 878.2	3595.25 ± 854.62	3159.97 ± 783.99	2667.27 ± 733.57	<0.001
PEF _{25–75%}	3015.3 ± 1269.1	3492.57 ± 1255.78	3012.81 ± 1206.31	2540.43 ± 1160.90	<0.001
FEV ₁ /FVC(%)	78.8 ± 7.3	79.99 ± 7.27	78.52 ± 6.95	77.75 ± 7.51	<0.001

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Table-2: Association Between WWI and Pulmonary Function Measures

WWI	FVC	FEV1	PEF25%-75%	FEV1/FVC(%)
Model 1				
Total	-603.9 (-628.7, -579.2) <0.001	-527.7 (-547.7, -507.7) <0.001	-550.5 (-581.7, -519.3) <0.001	-1.3 (-1.5, -1.1) <0.001
Q1	Ref.	Ref.	Ref.	Ref.
Q2	-475.6 (-528.6, -422.7) <0.001	-435.3 (-478.4, -392.2) <0.001	-479.8 (-544.4, -415.1) <0.001	-1.5 (-1.8, -1.1) <0.001
Q3	-1071.4 (-1122.7, -1020.1) <0.001	-928.0 (-969.8, -886.2) <0.001	-952.1 (-1015.7, -888.6) <0.001	-2.2 (-2.6, -1.9) <0.001
P for trend	-692.9 (-726.2, -659.7) <0.001	-600.3 (-627.4, -573.2) <0.001	-616.0 (-657.2, -574.9) <0.001	-1.5 (-1.7, -1.2) <0.001
Model 2				
Total	-282.4 (-302.7, -262.1) <0.001	-209.4 (-225.6, -193.1) <0.001	-93.5 (-123.9, -63.1) <0.001	0.4 (0.2, 0.6) <0.001
Q1	Ref.	Ref.	Ref.	Ref.
Q2	-201.2 (-236.4, -166.0) <0.001	-147.3 (-175.5, -119.2) <0.001	-39.8 (-91.9, 12.4) 0.135	0.4 (0.0, 0.7) 0.024
Q3	-474.2 (-511.6, -436.7) <0.001	-336.6 (-366.8, -306.3) <0.001	-113.6 (-171.3, -55.9) <0.001	0.9 (0.5, 1.3) <0.001
P for trend	-307.0 (-331.2, -282.9) <0.001	-217.9 (-237.5, -198.4) <0.001	-73.6 (-111.0, -36.3) <0.001	0.6 (0.3, 0.8) <0.001
Model 3				
Total	-248.4 (-270.2, -226.7) <0.001	-183.2 (-200.5, -165.9) <0.001	-72.7 (-105.1, -40.3) <0.001	0.4 (0.2, 0.6) <0.001
Q1	Ref.	Ref.	Ref.	Ref.
Q2	-180.5 (-216.9, -144.1) <0.001	-124.2 (-153.1, -95.3) <0.001	-0.7 (-54.7, 53.2) 0.979	0.6 (0.2, 0.9) 0.001
Q3	-415.5 (-455.0, -376.0) <0.001	-288.9 (-320.5, -257.3) <0.001	-70.2 (-131.2, -9.2) 0.024	1.0 (0.6, 1.4) <0.001
P for trend	-268.6 (-294.2, -243.0) <0.001	-186.8 (-207.3, -166.3) <0.001	-45.2 (-84.7, -5.7) 0.025	0.6 (0.4, 0.9) <0.001

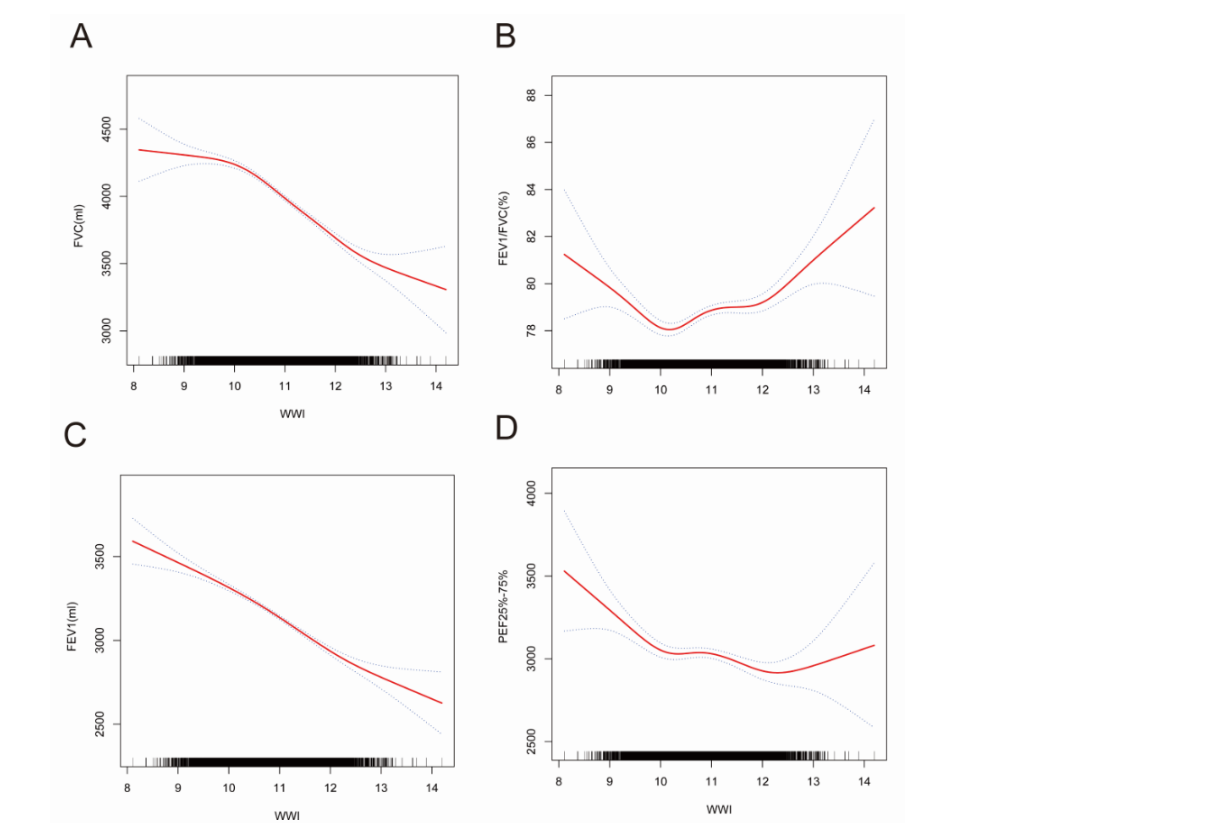


Fig-2: Smooth curve fitting for WWI and pulmonary function. Adjust for: Gender; Age; Race; Education; Poverty index ratio; Hypertension status; Diabetes status, Smoking status

Association Between Weight-Adjusted Waist Index and Pulmonary Function:

Association Between WWI and Pulmonary Function:

Table-2 presents the association between WWI and pulmonary function measures. When treated as continuous variables, WWI was negatively associated with FVC ($\beta = -603.9$, 95% CI: -628.7, -579.2), FEV₁ ($\beta = -527.7$, 95% CI: -547.7, -507.7), PEF_{25-75%} ($\beta = -550.5$, 95% CI: -581.7, -519.3), and FEV₁/FVC ($\beta = -1.3$, 95% CI: -1.5, -1.1). Similar results were observed when WWI was categorized, with significant negative trends for FEV₁, FVC, and PEF_{25-75%} (not for FEV₁/FVC). Sensitivity analyses adjusting for different covariates confirmed these findings (**Table-2**).

The curve fitting analysis (**Fig-2**) similarly illustrates significant correlations between Weight-Adjusted Waist Index (WWI) and pulmonary function parameters. For Forced Vital Capacity (FVC) and Forced Expiratory Volume in one second (FEV₁), the curves show a clear negative trend, indicating reduced lung volumes with increasing WWI, suggesting restrictive patterns. Conversely, the FEV₁/FVC ratio curve remains relatively flat, implying minimal effect of central obesity on airflow obstruction.

Lastly, Peak Expiratory Flow rate at 25–75% (PEF_{25-75%}) also displays a negative correlation with WWI, signaling potential small airway dysfunction. These findings collectively highlight the adverse impact of central obesity on lung volumes and mid-expiratory flow rates, underscoring its role in respiratory impairment. Confidence intervals support these trends, confirming the robustness of the observed associations.

Discussion

The present study investigates the association between Weight-Adjusted Waist Index (WWI) and pulmonary function indices, specifically Forced Vital Capacity (FVC), Forced Expiratory Volume in one second (FEV₁), Peak Expiratory Flow Rate (PEF_{25-75%}), and FEV₁/FVC ratio. Our findings indicate that higher WWI is associated with significantly lower values of FVC, FEV₁, and PEF_{25-75%}, but not FEV₁/FVC. These results suggest that central obesity, as measured by WWI, may have a detrimental effect on lung volumes and airflow rates, independent of overall body mass.

Central obesity, characterized by excessive visceral fat accumulation around the abdomen, has been shown to adversely affect pulmonary function through multiple mechanisms [9]. The mechanical compression exerted by abdominal fat can restrict diaphragmatic movement, leading to reduced lung expansion and decreased functional residual capacity (FRC) and expiratory reserve volume (ERV) [16]. Our data support this notion, as participants with higher WWI exhibited significantly lower FVC and FEV₁ values compared to those with lower WWI. This finding aligns with previous studies demonstrating that central obesity is more strongly associated with restrictive ventilatory impairment than overall obesity [17].

Additionally, central obesity is linked to systemic inflammation, which can contribute to airway hyperresponsiveness and impaired gas exchange [9,18]. Pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF- α) and interleukin-6 (IL-6), secreted by adipose tissue, can activate pathways leading to alveolar damage and ventilation/perfusion mismatch [19]. Our results show that higher WWI was associated with lower PEF_{25-75%}, indicating potential airway obstruction or increased airway resistance. These findings are consistent with prior research showing that obese individuals, particularly those with central obesity, exhibit signs of small airway dysfunction [9,17,20].

While BMI is widely used as an indicator of obesity, it fails to distinguish between fat mass and lean body mass and does not account for fat distribution patterns [3]. In contrast, WWI integrates waist circumference, reflecting visceral fat accumulation, and weight, distinguishing fat from muscle mass. Our study found that WWI outperformed BMI in predicting reductions in lung volumes and airflow rates. For instance, although BMI showed some negative correlations with FVC and FEV₁, these associations were weaker compared to those observed with WWI [21]. This suggests that WWI may be a more sensitive metric for assessing the impact of central obesity on pulmonary function.

Moreover, our findings are supported by other studies that have investigated the relationship between different obesity indices and lung function [22]. Overall,

the present study provides compelling evidence that higher WWI values are associated with lower FVC, FEV₁, and PEF_{25–75%}, indicating potential restrictive ventilatory impairment and small airway dysfunction. However, the FEV₁/FVC ratio remains relatively stable across different levels of central obesity, suggesting that the impact of WWI on lung function is more pronounced in terms of volume and flow rate rather than the pattern of airflow limitation.

The clinical implications of our findings are significant. Clinicians should consider using WWI alongside traditional measures like BMI when assessing respiratory health, especially in patients with suspected obesity-related lung dysfunction. Early identification of individuals at risk for pulmonary impairment due to central obesity could facilitate timely interventions aimed at reducing abdominal fat and improving lung function. Furthermore, our results highlight the importance of addressing central obesity in the management of chronic respiratory conditions such as asthma and chronic obstructive pulmonary disease (COPD). Interventions targeting weight loss and reduction of abdominal fat may help alleviate symptoms and improve quality of life in affected individuals.

Despite the robust findings, our study has several limitations. First, the cross-sectional design precludes causal inference, and longitudinal studies are needed to establish temporal relationships between WWI and changes in pulmonary function over time. Second, although we adjusted for various covariates, residual confounding cannot be entirely ruled out. Third, our study population primarily consisted of adults aged 18 years and older, limiting generalizability to younger populations or children.

Future research should focus on validating our findings in diverse populations and exploring the underlying mechanisms linking central obesity to pulmonary function impairment. Additionally, investigating the impact of lifestyle interventions targeting abdominal fat reduction on lung function outcomes would provide valuable insights into potential therapeutic strategies.

Conclusion

Weight-Adjusted Waist Index (WWI) demonstrates a significant negative association with key pulmonary function metrics, including Forced Vital Capacity (FVC), Forced Expiratory Volume in one second (FEV₁), and Peak Expiratory Flow rate at 25–75% of forced vital capacity (PEF_{25–75%}). While the FEV₁/FVC ratio remains unaffected by WWI, this indicates that central obesity primarily impacts lung volumes rather than airflow obstruction.

These findings highlight the adverse effects of central adiposity on respiratory health, underscoring the necessity for incorporating measures of central obesity like WWI into clinical evaluations. Future longitudinal studies are essential to further investigate the underlying mechanisms and validate these observations across different populations. This research provides critical insights into how central obesity contributes to respiratory impairment, emphasizing the importance of comprehensive assessments in managing pulmonary health.

Consent for Publication

Written informed consent was obtained from the patient's son for the publication of this case report and related images.

Ethics Approval and Consent to Participate

The Research Ethics Review Board of the National Center for Health Statistics reviewed and approved the study protocol. Every selected participant signed written informed consent.

Data Availability Statement

The original contributions presented in this study are included in the article. Further inquiries can be directed to the corresponding authors.

Conflict of Interest

The author has read and approved the final version of the manuscript. The author declares no conflicts of interest.

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