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# PULMONARY DYSFUNCTION IN TYPE 2 DIABETES MELLITUS: INTEGRATED ANALYSIS OF GLYCEMIC BURDEN, ADIPOSITY, AND SPIROMETRIC IMPAIRMENT

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ABSTRACT

**Background:** Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder associated with multiple systemic complications. Emerging evidence suggests that the lung may represent an additional target organ; however, pulmonary involvement remains under-recognized. The objective is to evaluate pulmonary function in patients with Type 2 Diabetes Mellitus and to assess its association with glycemic parameters and anthropometric indices.

**Materials and Methods:** This analytical case–control study included 200 participants comprising 100 patients with T2DM and 100 age- and sex-matched healthy controls. Anthropometric parameters including body mass index (BMI), waist circumference, hip circumference, and waist–hip ratio (WHR) were recorded. Glycemic status was assessed using fasting blood sugar (FBS) and postprandial blood sugar (PPBS). Pulmonary function tests were performed using spirometry, measuring FVC, $FEV_1$, $FEV_1$/FVC ratio, FEF 25–75%, and PEF. Statistical analysis was conducted using Z-test and Pearson's correlation.

**Results:** Patients with T2DM exhibited significantly higher BMI, waist circumference, hip circumference, and WHR compared to controls ($p < 0.0001$). Glycemic parameters (FBS and PPBS) were markedly elevated in the diabetic group ($p < 0.0001$). Pulmonary function tests revealed significant reductions in FVC and $FEV_1$ in diabetic patients ($p < 0.0001$), with a predominantly restrictive pattern of lung impairment observed. Significant negative correlations were found between glycemic parameters and pulmonary function indices, indicating progressive decline in lung function with increasing glycemic burden.

**Conclusion:** Type 2 Diabetes Mellitus is associated with significant impairment in pulmonary function, predominantly of a restrictive nature. Chronic hyperglycemia and increased adiposity appear to play a central role in this dysfunction. These findings support the inclusion of pulmonary function testing in the routine assessment of patients with T2DM.

**Keywords:** Type 2 Diabetes Mellitus; Pulmonary Function; Spirometry; Glycemic Burden; Adiposity; Restrictive Lung Disease; Forced Vital Capacity; Insulin Resistance.

## INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from insulin resistance and relative insulin deficiency. The global burden of diabetes has reached epidemic proportions, with significant morbidity and mortality arising from its long-term complications. Traditionally, the complications of diabetes have been categorized into

microvascular (retinopathy, nephropathy, neuropathy) and macrovascular (cardiovascular disease) domains. However, emerging evidence suggests that the lung may represent an additional target organ affected by diabetes.[1]

The lung is a highly vascularized organ with an extensive microcapillary network and abundant connective tissue matrix, rendering it susceptible to the effects of chronic hyperglycemia. According to principles described in standard physiology, prolonged elevation of blood glucose leads to non-enzymatic glycation of proteins, resulting in structural alterations in collagen and elastin fibers. These changes reduce lung elasticity and impair pulmonary mechanics.[1]

In addition to glycation-induced structural damage, diabetes is associated with microangiopathy affecting small blood vessels throughout the body, including pulmonary capillaries. Thickening of the alveolar-capillary basement membrane may impair gas exchange and contribute to restrictive ventilatory defects.[2] These pathophysiological mechanisms support the concept of the lung as a "target organ" in diabetes.

Furthermore, chronic hyperglycemia is associated with oxidative stress and low-grade systemic inflammation, both of which contribute to tissue remodeling and functional impairment. These processes may further exacerbate pulmonary dysfunction in diabetic individuals.

Another important factor linking diabetes and pulmonary impairment is adiposity. Obesity, particularly central obesity, is commonly associated with T2DM and has independent effects on respiratory function. Increased abdominal fat limits diaphragmatic excursion, reduces chest wall compliance, and decreases lung volumes, thereby contributing to restrictive patterns of lung dysfunction.

Despite these mechanisms, pulmonary involvement in diabetes remains under-recognized in clinical practice. Most diabetic patients are not routinely evaluated for lung function, leading to underdiagnosis of early pulmonary impairment.

Previous studies have demonstrated reductions in Forced Vital Capacity (FVC) and Forced Expiratory Volume in one second ($FEV_1$) in patients with T2DM, suggesting a predominantly restrictive pattern of lung dysfunction. However, the relationship between glycemic burden, adiposity, and pulmonary impairment requires further clarification.

Therefore, the present study was undertaken to comprehensively evaluate pulmonary function in patients with Type 2 Diabetes Mellitus and to analyze its association with glycemic parameters and anthropometric indices.[3]

## MATERIALS AND METHODS

**Study Design and Setting:** This study was designed as a hospital-based, analytical case–control study conducted to evaluate pulmonary function in patients with Type 2 Diabetes Mellitus (T2DM) and to investigate its relationship with glycemic burden and adiposity. The study was carried out in Pravara Institute of Medical Sciences, Loni over a defined study period after obtaining approval from the Institutional Ethics Committee.

The case–control design was chosen as it allows efficient comparison between diseased and non-diseased populations and is particularly suitable for evaluating associations between metabolic and physiological parameters.[4]

**Study Population**

**The study population consisted of a total of 200 participants, divided into:**

- **Case Group:** 100 patients diagnosed with Type 2 Diabetes Mellitus
- **Control Group:** 100 apparently healthy individuals

Controls were selected to match cases with respect to age and gender, thereby minimizing confounding variables.

**Inclusion Criteria**

**Cases:**

- Diagnosed Type 2 Diabetes Mellitus patients
- Age between 31–60 years
- Both males and females

**Controls:**

- Apparently healthy individuals
- No history of diabetes mellitus
- Age- and sex-matched with cases

**Exclusion Criteria**

Participants with the following conditions were excluded:

- History of smoking
- Chronic respiratory diseases (e.g., COPD, asthma, tuberculosis)
- Known cardiovascular disorders
- Acute illness at the time of study
- Occupational exposure to respiratory irritants

These criteria were applied to eliminate confounding factors that could independently influence pulmonary function.[5]

**Sample Size Consideration:** A total sample size of 200 participants was considered adequate to detect statistically significant differences in pulmonary function parameters between groups, based on prior studies evaluating spirometric changes in diabetic populations.[6]

**Data Collection and Clinical Evaluation**

All participants underwent detailed clinical evaluation, including:

- Medical history
- Duration of diabetes (for case group)
- Treatment history
- Physical examination

Anthropometric measurements were recorded using standardized techniques.

Anthropometric Measurements

**The following parameters were assessed:**

- Body Mass Index (BMI): Calculated as weight (kg) divided by height ($m^2$)

- Waist Circumference (WC): Measured at the midpoint between the lower rib and iliac crest
- Hip Circumference (HC): Measured at the widest part of the buttocks
- Waist–Hip Ratio (WHR): Calculated as WC/HC

These parameters are well-established indicators of overall and central adiposity and have significant implications for respiratory mechanics and lung volumes.[7]

Biochemical Analysis (Glycemic Parameters)

**Blood samples were collected under standardized conditions:**

- Fasting Blood Sugar (FBS): Measured after overnight fasting
- Postprandial Blood Sugar (PPBS): Measured 2 hours after meal

Hyperglycemia is known to induce biochemical and structural alterations in tissues through non-enzymatic glycation and oxidative stress pathways.[8]

**Pulmonary Function Testing**

Pulmonary function tests (PFTs) were performed using a calibrated spirometer under standardized conditions, following guidelines for respiratory function testing.

**The following parameters were recorded:**

- Forced Vital Capacity (FVC)
- Forced Expiratory Volume in 1 second ($FEV_1$)
- $FEV_1$/FVC ratio
- Forced Expiratory Flow (FEF 25–75%)
- Peak Expiratory Flow (PEF)

Each subject performed at least three acceptable maneuvers, and the best value was considered for analysis.

Spirometry is a fundamental tool for assessing lung function and provides reliable information regarding both obstructive and restrictive ventilatory defects.[5]

**Physiological Basis of Pulmonary Assessment**

Pulmonary function parameters such as FVC and $FEV_1$ reflect lung volumes, airway resistance, and elastic properties of lung tissue. According to established physiological principles, any alteration in lung compliance, airway caliber, or respiratory muscle function can lead to measurable changes in spirometric indices.[4]

In diabetes mellitus, chronic hyperglycemia leads to glycation of structural proteins such as collagen and elastin, resulting in decreased lung compliance and reduced lung volumes. Additionally, microvascular changes in pulmonary circulation may impair gas exchange and contribute to restrictive patterns of dysfunction.[7]

**Interpretation of Pulmonary Function**

Pulmonary function was categorized based on spirometric patterns:

- Normal
- Mild restriction
- Moderate restriction
- Severe restriction

A restrictive pattern was identified based on reduced FVC with relatively preserved or increased $FEV_1$/FVC ratio.

**Statistical Analysis**

Data were entered and analyzed using appropriate statistical software.

- Quantitative variables were expressed as mean ± standard deviation (SD)
- Comparison between case and control groups was performed using Z-test
- Correlation analysis was conducted using Pearson's correlation coefficient (r)
- A p-value $< 0.05$ was considered statistically significant

Statistical methods were selected based on the nature of data and study design to ensure valid and reliable interpretation of results.[6]

## RESULTS

**1. Baseline Characteristics of Study Population**

The study included a total of 200 participants, comprising 100 patients with Type 2 Diabetes Mellitus (case group) and 100 healthy individuals (control group).

The distribution of participants according to age is presented in [Table 1].

**Table 1: Distribution of participants in the study groups according to age-group**

| Age-Group [In year] | Case Group | | Control Group | |
|---|---|---|---|---|
| | No | Percentage | No | Percentage |
| 31-40 | 30 | 30.0 | 32 | 32.0 |
| 41-50 | 33 | 33.0 | 31 | 31.0 |
| 51-60 | 36 | 36.0 | 37 | 37.0 |
| Total | 100 | 100% | 100 | 100% |
| Mean±SD | 45.74±8.66 | | 45.45±7.41 | |
| Z-value | 0.255 | | | |
| P-value | P=0.799 NS | | | |

The mean age of the case group was 45.74 ± 8.66 years, while that of the control group was 45.45 ± 7.41 years. The difference in mean age between the two groups was not statistically significant ($p = 0.799$), indicating that both groups were comparable with respect to age.

Similarly, gender distribution [Table 2] showed that both groups consisted of 45% males and 55% females, confirming appropriate matching and eliminating gender as a confounding factor.

**Table 2: Distribution of participants in the study groups according to Gender**

| Gender | Case Group | | Control Group | |
|---|---|---|---|---|
| | No | Percentage | No | Percentage |
| Male | 45 | 45.0 | 45 | 45.0 |
| Female | 55 | 55.0 | 55 | 55.0 |
| Total | 100 | 100% | 100 | 100% |

These findings establish that the case and control groups were comparable at baseline, thereby strengthening the validity of subsequent comparisons.[9]

**2. Anthropometric Profile (Adiposity Analysis)**

Anthropometric parameters showed significant differences between the case and control groups [Table 3].

**Table 3: Comparison of Mean Body Mass Index (BMI), Waist Circumference, Hip Circumference, and Waist–Hip Ratio (WHR) between Case and Control Groups**

| | Case Group Mean±SD | Control Group Mean±SD | Z-value | P-value |
|---|---|---|---|---|
| BMI | 29.89±3.87 | 24.95±3.95 | 8.91 | P<0.0001S |
| Waist (cm) | 96.07±9.63 | 84.57±10.06 | 8.25 | P<0.0001S |
| Hip (cm) | 110.23±8.92 | 98.24±10.16 | 8.87 | P<0.0001 S |
| WHR | 0.87±0.05 | 0.86±0.04 | 9.26 | P<0.0001S |

The mean Body Mass Index (BMI) was significantly higher in the case group (29.89 ± 3.87 kg/m²) compared to the control group (24.95 ± 3.95 kg/m²), with a highly significant difference ($p < 0.0001$).

Waist circumference and hip circumference were also significantly elevated in the case group. The mean waist circumference was 96.07 ± 9.63 cm in cases versus 84.57 ± 10.06 cm in controls ($p < 0.0001$). Similarly, hip circumference was significantly higher in cases (110.23 ± 8.92 cm) compared to controls (98.24 ± 10.16 cm).

Waist–Hip Ratio (WHR), an indicator of central obesity, was also significantly elevated in the diabetic group.

These findings indicate that patients with T2DM exhibited significantly higher levels of both generalized and central adiposity.

**3. Glycemic Parameters**

The comparison of glycemic indices is shown in [Table 4].

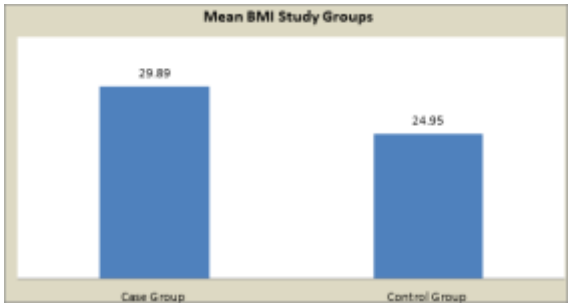


**Figure 1: Comparative Analysis of Body Mass Index in Type 2 Diabetes Mellitus and Control Groups.**

**Table 4: Comparison of mean FBS (mg/dl) and PPBS (mg/dl) in study Groups**

| | Case Group Mean±SD | Control Group Mean±SD | Z-value | P-value |
|---|---|---|---|---|
| FBS (mg/dl) | 176.63±24.92 | 89.13±8.17 | 33.35 | P<0.0001S |
| PPBS (mg/dl) | 255.06±36.69 | 124.0±7.36 | 34.75 | P<0.0001S |

The mean fasting blood sugar (FBS) level in the case group was 176.63 ± 24.92 mg/dl, significantly higher than the control group (89.13 ± 8.17 mg/dl) ($p < 0.0001$).

Similarly, the mean postprandial blood sugar (PPBS) level was markedly elevated in the case group (255.06 ± 36.69 mg/dl) compared to controls (124.0 ± 7.36 mg/dl), with a highly significant difference ($p < 0.0001$).

These results confirm the presence of significant hyperglycemia in the diabetic group.

**4. Pulmonary Function Tests:** Pulmonary function parameters demonstrated significant impairment in patients with T2DM compared to controls.

**Forced Vital Capacity (FVC):** As shown in [Table 5], the mean recorded FVC was significantly lower in the case group (3.83 ± 1.15 L) compared to the control group (4.38 ± 0.59 L), with a highly significant difference ($p < 0.0001$).

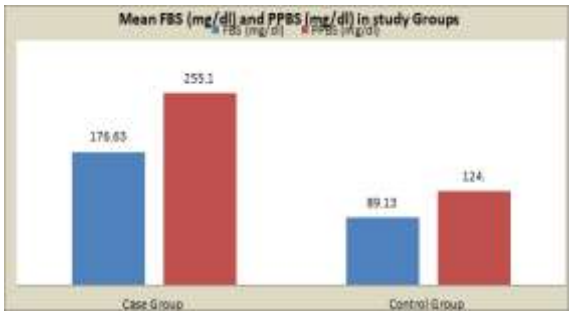


**Figure 2: Comparative Evaluation of Fasting and Postprandial Blood Glucose Levels in Study Groups**

**Table 5: Comparison of Mean Forced Vital Capacity (FVC) between Case and Control Groups**

| FVC | Case Group Mean±SD | Control Group Mean±SD | Z-value | P-value |
|---|---|---|---|---|
| Recorded FVC | 3.83±1.15 | 4.38±0.59 | 4.27 | P<0.0001S |
| Predicted FVC | 4.53±1.06 | 4.67±0.55 | 1.15 | P=0.251NS |
| Recorded FVC/ Predicted FVC | 83.97±10.26 | 93.68±2.25 | 9.22 | P<0.0001S |

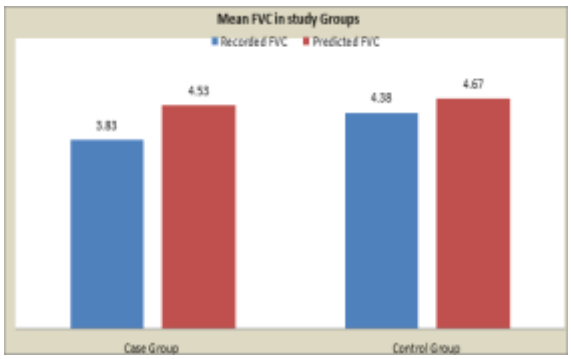


**Figure 3: Comparative Analysis of Forced Vital Capacity (FVC) Between Type 2 Diabetes Mellitus and Control Groups**

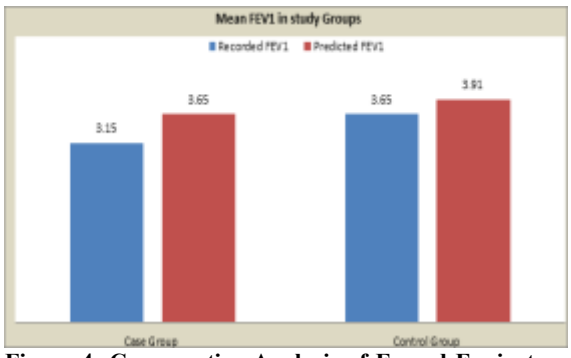


**Figure 4: Comparative Analysis of Forced Expiratory Volume in One Second ($FEV_1$) Between Type 2 Diabetes Mellitus and Control Groups**

**4.2 Forced Expiratory Volume in 1 Second ($FEV_1$)**
[Table 5] shows that the mean recorded $FEV_1$ was significantly reduced in the case group (3.15 ± 0.93 L) compared to controls (3.65 ± 0.50 L), with statistical significance ($p < 0.0001$).

**4.3 $FEV_1$/FVC Ratio**
The mean $FEV_1$/FVC ratio also showed statistically significant differences between groups [Table 6], indicating altered ventilatory dynamics in diabetic patients.

**Table 6: Comparison of mean FEV1/ FVC in study Groups**

| FEV1/ FVC | Case Group Mean±SD | Control Group Mean±SD | Z-value | P-value |
|---|---|---|---|---|
| Recorded FEV1/ FVC | 82.48±2.93 | 83.37±1.57 | 2.69 | P=0.008S |
| Predicted FEV1/ FVC | 80.56±2.67 | 84.09±1.50 | 11.51 | P<0.0001S |
| Recorded FEV1/ FVC / Predicted FEV1/ FVC | 102.37±2.87 | 99.23±1.61 | 9.54 | P<0.0001S |

**4.4 Forced Expiratory Flow (FEF 25–75%):** As shown in [Table 7], the mean FEF 25–75% was significantly reduced in the case group compared to controls ($p < 0.0001$), suggesting involvement of small airways.

**Table 7: Comparison of mean FEF 25-75% in study Groups**

| FEF25-75% | Case Group Mean±SD | Control Group Mean±SD | Z-value | P-value |
|---|---|---|---|---|
| Recorded FEF25-75% | 3.44±0.73 | 3.93±0.45 | 5.89 | P<0.0001S |
| Predicted FEF25-75% | 3.88±0.51 | 4.12±0.43 | 3.45 | P<0.0001S |
| Recorded FEF25-75% / Predicted FEF25-75% | 87.95±8.08 | 95.54±2.03 | 9.11 | P<0.0001S |

**4.5 Peak Expiratory Flow (PEF):** The mean PEF values were significantly lower in diabetic patients [Table 8], indicating reduced expiratory flow capacity.

**Table 8: Comparison of mean PEF (L/min) in study Groups**

| PEF (L/min) | Case Group Mean±SD | Control Group Mean±SD | Z-value | P-value |
|---|---|---|---|---|
| Recorded PEF (L/min) | 437.45±93.11 | 493.04±92.14 | 4.22 | P<0.0001S |
| Predicted PEF (L/min) | 512.09±79.70 | 522.15±85.00 | 0.863 | P=0.398NS |
| Recorded PEF (L/min) / Predicted PEF (L/min) | 85.24±9.34 | 94.13±2.85 | 9.10 | P<0.0001S |

Overall, spirometric evaluation revealed a consistent pattern of reduced pulmonary function in T2DM patients.

**5. Pattern of Pulmonary Dysfunction:** The distribution of pulmonary function patterns is presented in [Table 9].

**Table 9: Distribution of Participants in the Case and Control Groups According to Interpretation of Pulmonary Function Test (Restriction Pattern)**

| Interpretation Restriction | Case Group | | Control Group | |
|---|---|---|---|---|
| | No | Percentage | No | Percentage |
| Normal | 65 | 65.0 | 100 | 100.0 |
| Mild Restriction | 27 | 27.0 | 00 | 00 |
| Moderate Restrction | 08 | 08.0 | 00 | 00 |
| Severe Restrction | 00 | 00 | 00 | 00 |
| Total | 100 | 100% | 100 | 100% |

**In the case group:**

- 65% of participants had normal pulmonary function
- 27% showed mild restriction
- 8% showed moderate restriction

In contrast, all participants in the control group had normal pulmonary function.

These findings indicate that a significant proportion of diabetic patients exhibited a restrictive pattern of lung dysfunction.

6. Correlation Between Glycemic Parameters and Pulmonary Function

Correlation analysis between glycemic indices (FBS and PPBS) and pulmonary function parameters is shown in [Table 10].

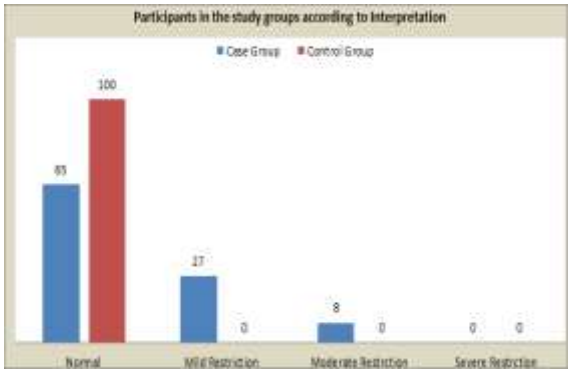


**Figure 5: Distribution of Restrictive Ventilatory Patterns in Type 2 Diabetes Mellitus and Control Groups.**

**Table 10: Correlation between FBS, PPBS and Pulmonary Function Test**

| | | FVC | | | FEV1 | | |
|---|---|---|---|---|---|---|---|
| | | **Recorded** | **Predicted** | **R/P%** | **Recorded** | **Predicted** | **R/P%** |
| FBS (mg/dl) | r-value | -0.246 | -0.049 | -0.511 | -0.272 | -0.143* | -0.433 |
| | P-value | <0.0001 | 0.487 | <0.0001 | 0.000 | 0.044 | <0.0001 |
| PPBS (mg/dl) | r-value | -0.214 | -0.005 | -0.514 | -0.24* | -0.100 | -0.448 |
| | P-value | 0.002 | 0.946 | <0.0001 | 0.001 | 0.160 | <0.0001 |

**Significant negative correlations were observed between:**

- FBS and FVC ($r = -0.246$, $p < 0.0001$)
- FBS and $FEV_1$ ($r = -0.272$, $p < 0.0001$)
- PPBS and FVC ($r = -0.214$, $p = 0.002$)
- PPBS and $FEV_1$ ($r = -0.240$, $p = 0.001$)

Similarly, strong negative correlations were observed with FEF 25–75% and PEF.

These findings indicate that increasing glycemic burden is associated with progressive decline in pulmonary function.

**Summary of Key Findings**

- Case and control groups were comparable at baseline
- Diabetic patients had significantly higher adiposity
- Glycemic parameters were markedly elevated in T2DM
- Pulmonary function was significantly reduced in diabetic patients
- A restrictive pattern of lung dysfunction was observed
- Glycemic burden showed significant negative correlation with lung function.

## DISCUSSION

The present study demonstrates that Type 2 Diabetes Mellitus (T2DM) is associated with significant impairment in pulmonary function, predominantly manifesting as a restrictive ventilatory pattern. The observed reductions in FVC and $FEV_1$, along with preservation of the $FEV_1$/FVC ratio, suggest that pulmonary involvement in diabetes reflects parenchymal and mechanical alterations rather than airway obstruction. These findings support the concept that the lung functions as a target organ in diabetes, influenced by both metabolic and structural factors.

The reduction in lung volumes observed in this study is consistent with contemporary epidemiological evidence. Large-scale analyses have demonstrated that individuals with diabetes exhibit lower baseline lung function and accelerated decline over time, independent of smoking and other confounders. Notably, studies led by Elizabeth Selvin have shown that higher glycemic markers are associated with reduced pulmonary function and progressive decline, even in non-diabetic populations.[10] Similarly, Ravi Kalhan et al. reported longitudinal reductions in spirometric indices among diabetic individuals, reinforcing the concept of diabetes-related pulmonary dysfunction as a progressive phenomenon.[11]

The pathophysiological basis of this impairment is multifactorial. Chronic hyperglycemia leads to the formation of advanced glycation end-products (AGEs), which accumulate in connective tissue and result in cross-linking of collagen and elastin fibers, thereby reducing lung compliance. In addition, hyperglycemia induces oxidative stress and inflammatory signaling pathways that contribute to tissue remodeling. The mechanistic insights provided by Michael Brownlee highlight the central role of metabolic stress in driving structural changes across multiple organ systems, including the lung.[12]

Microvascular involvement further contributes to pulmonary dysfunction in T2DM. The pulmonary capillary network is susceptible to diabetic microangiopathy, leading to thickening of the alveolar–capillary membrane and impaired gas exchange. Imaging and structural studies by Harvey O. Coxson and colleagues have demonstrated alterations in lung parenchyma associated with systemic metabolic disease, supporting the hypothesis of microvascular injury within the lung.[13]

An important finding of the present study is the significant association between adiposity and pulmonary impairment. Increased BMI and WHR in the diabetic group indicate the presence of both generalized and central obesity, which exert mechanical and metabolic effects on respiratory function. Central obesity reduces diaphragmatic excursion and chest wall compliance, while adipose tissue-derived inflammatory mediators contribute to systemic inflammation. The interaction between obesity, insulin resistance, and organ dysfunction has been explored by Aryeh D. Shuldiner, emphasizing the integrated nature of metabolic and physiological disturbances.[14]

The observed inverse correlation between glycemic parameters (FBS and PPBS) and pulmonary function indices provides further evidence of a dose-dependent relationship between hyperglycemia and lung dysfunction. This relationship suggests that poor glycemic control may accelerate pulmonary decline. Longitudinal data support this observation, with studies demonstrating that elevated glucose levels are associated with progressive reductions in lung function over time.[10,15] These findings underscore the importance of glycemic control not only in preventing classical complications but also in preserving pulmonary health.

The reduction in FEF 25–75% observed in this study suggests early involvement of small airways, which may represent an initial stage of diabetic lung disease. Small airway dysfunction is increasingly recognized as an early marker of pulmonary impairment. Research by MeiLan K. Han has highlighted the importance of small airway pathology in systemic diseases, suggesting that early detection may have prognostic significance.[16]

Clinically, the findings of this study have important implications. Pulmonary function is not routinely assessed in patients with T2DM, despite growing evidence of its involvement. The identification of restrictive lung impairment in a significant proportion of diabetic patients suggests that spirometric evaluation may serve as a useful adjunct in the comprehensive assessment of diabetes-related complications. Furthermore, early identification of pulmonary dysfunction may facilitate timely interventions aimed at improving overall outcomes.

At a broader level, these findings reinforce the concept of T2DM as a multisystem disorder, affecting not only classical target organs but also the respiratory system. The integration of metabolic, structural, and functional changes observed in this study highlights the need for a more holistic approach to the management of diabetes.

While the present study provides valuable insights, certain limitations should be acknowledged. The cross-sectional design limits causal inference, and longitudinal studies are required to establish temporal relationships. Additionally, advanced pulmonary assessments such as diffusion capacity were not included, which could provide further insight into microvascular involvement.

In conclusion, Type 2 Diabetes Mellitus is associated with significant impairment of pulmonary function, predominantly of a restrictive nature, mediated by a combination of metabolic, structural, and microvascular mechanisms. Recognition of the lung as a target organ in diabetes may have important implications for both clinical practice and future research.

## CONCLUSION

The present study demonstrates that Type 2 Diabetes Mellitus is associated with significant impairment in pulmonary function, predominantly manifesting as a restrictive ventilatory defect. The observed reductions in FVC and $FEV_1$, along with the presence of restrictive patterns on spirometric evaluation, indicate that pulmonary involvement is a clinically relevant but under-recognized complication of diabetes.

The strong inverse relationship between glycemic parameters and pulmonary function highlights the role of chronic hyperglycemia as a key determinant of lung dysfunction. In addition, the contribution of increased adiposity, particularly central obesity, suggests that both metabolic and mechanical factors synergistically influence respiratory physiology in diabetic individuals.

These findings support the concept that the lung should be considered a target organ in Type 2 Diabetes Mellitus, similar to the retina, kidney, and peripheral nerves. Early identification of pulmonary impairment through spirometry may facilitate timely intervention and improve overall disease management.

In conclusion, incorporation of pulmonary function assessment into routine evaluation of patients with Type 2 Diabetes Mellitus may enhance early detection of subclinical dysfunction and contribute to a more comprehensive approach to diabetes care.

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