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Original Research Article

Investigating The Pulmonary Function Measurements using Spirometry and their Correlation with BMI and HbA1c Levels in Individuals with Type 2 Diabetes Mellitus

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Abstract:

Background: Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder associated with systemic complications. The lung, though susceptible to hyperglycaemia-induced structural changes, remains an underappreciated target organ. Spirometry offers a simple, non-invasive approach to evaluate early pulmonary impairment in diabetic individuals.

Aims and Objectives: To assess pulmonary function using spirometry in patients with T2DM and evaluate its association with glycaemic control (HbA1c) and Body Mass Index (BMI).

Setting and Design: A hospital-based comparative cross-sectional study conducted in the Department of Physiology, P.D.U. Government Medical College and Hospital, Rajkot, Gujarat, India.

Materials and Methods: One hundred participants aged 30–60 years were enrolled: 50 individuals with confirmed T2DM (HbA1c ≥6.5%) and 50 age- and BMI-matched normoglycaemic healthy controls. Spirometric parameters — Forced Vital Capacity (FVC), Forced Expiratory Volume in one second (FEV_1), and FEV_1/FVC ratio — were recorded using the RMS Helios 401 spirometer, with percentage predicted values derived from GLI-2012 reference equations. Pearson correlation analysis was performed to assess associations with BMI and HbA1c.

Results: Percentage predicted FVC was significantly lower in diabetics compared to controls (87.98 ± 6.45% vs 91.40 ± 5.19%; $p<0.05$). Percentage predicted FEV_1 showed a numerical reduction but did not reach statistical significance (89.68 ± 5.39% vs 91.52 ± 5.21%; $p>0.05$). The FEV_1/FVC ratio was preserved in both groups (0.77 ± 0.03 vs 0.76 ± 0.05; $p>0.05$), indicating a restrictive ventilatory tendency. BMI demonstrated significant inverse correlations with both FVC% ($r = -0.38$, $p<0.05$) and FEV_1% ($r = -0.31$, $p<0.05$) within the diabetic subgroup. No statistically significant correlation was identified between HbA1c and any spirometric parameter.

Conclusion: Subclinical restrictive pulmonary impairment is detectable in T2DM patients, with BMI demonstrating a stronger association with spirometric impairment than HbA1c. Routine spirometric screening may be warranted in T2DM patients, particularly those with higher BMI.

Keywords: Type 2 Diabetes Mellitus, Spirometry, Pulmonary Function Tests, FVC, FEV_1, Body Mass Index, HbA1c, Restrictive Ventilatory Pattern.

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Introduction

Diabetes Mellitus is a major global public health challenge, with Type 2 Diabetes Mellitus (T2DM) representing the predominant form, characterised by peripheral insulin resistance and relative insulin deficiency. [1,2] According to the International Diabetes Federation Diabetes Atlas (10th edition, 2021), approximately 537 million adults worldwide were living with diabetes, with India alone accounting for more than 74 million individuals. [3] Recent national data from the ICMR-INDIAB study further report a diabetes prevalence exceeding 11.4% within the Indian population. [4] Long-standing hyperglycaemia drives both macrovascular and microvascular complications, and early diagnosis with strict glycaemic control remains the cornerstone of clinical management.

While renal, retinal, and cardiovascular complications of T2DM are well-recognised, the lung has historically been an overlooked target organ. [5,6] The pulmonary capillary bed is among the most extensive microvascular networks in the body, rendering it inherently susceptible to diabetic microangiopathy. Chronic hyperglycaemia promotes non-enzymatic glycosylation of structural proteins — particularly collagen and elastin — through the Maillard reaction, resulting in the formation of Advanced Glycation End-products (AGEs). [7] Accumulation of AGEs induces cross-linking of pulmonary connective tissue, increasing parenchymal stiffness, reducing elastic recoil, and leading to progressive loss of lung volumes and capacities. [6,7]

Spirometry is the most widely used, non-invasive method to objectively evaluate pulmonary mechanics. [8] In T2DM, these structural alterations typically manifest as a restrictive ventilatory pattern, characterised by reduced FVC and FEV_1, with a preserved FEV_1/FVC ratio. Both Body Mass Index (BMI) and HbA1c are closely implicated: BMI exerts mechanical restriction on thoracic excursion through adipose tissue deposition, while HbA1c reflects cumulative glycaemic burden and hence the degree of connective tissue glycation. [7,9]

Despite accumulating evidence, the relative independent contributions of BMI and HbA1c to pulmonary impairment in T2DM remain incompletely characterised, particularly in the Indian population where individuals are predisposed to metabolic risk at comparatively lower BMI thresholds. [4] The present study was designed to assess spirometric pulmonary function parameters in T2DM patients, compare them with age- and BMI-matched healthy controls, and evaluate their correlation with BMI and HbA1c, to provide localised clinical evidence supporting integration of spirometric screening into routine diabetes management.

Materials and Methods

Study Design and Setting: This hospital-based comparative cross-sectional study was conducted in the Department of Physiology at P.D.U. Government Medical College and Hospital, Rajkot, Gujarat, India, in collaboration with the Departments of Tuberculosis & Respiratory Medicine and Biochemistry. The study was conducted over one year from August 1, 2024 to July 31, 2025. The study protocol received formal approval from the Institutional Ethics Committee, and written informed consent was obtained from all participants.

Study Population: A total of 100 participants aged 30–60 years were enrolled: Group A (Cases) comprised 50 individuals with confirmed T2DM; Group B (Controls) comprised 50 age- and BMI-matched normoglycaemic healthy individuals. [10] The sample size was calculated using the formula for comparison of two independent means, yielding n = 50 per group at 95% confidence and 80% power, based on a standardised effect size derived from published Indian spirometric data in T2DM.

Inclusion and Exclusion Criteria: Adults of either gender aged 30–60 years were eligible. Cases were required to have confirmed T2DM with HbA1c ≥6.5% per ADA diagnostic criteria. [10] Controls were normoglycaemic (HbA1c <6.5%) with no history of chronic illness. Exclusion criteria included: known chronic respiratory diseases (asthma, tuberculosis, COPD); current or former smoking or occupational exposure to respiratory irritants; known cardiac disease, connective tissue disorders, neuromuscular disorders, or chronic kidney disease; haemoglobinopathies affecting HbA1c reliability; known or suspected Type 1 Diabetes Mellitus; significant alcohol use; acute respiratory tract infection within four weeks prior to testing; pregnancy or lactation; recent thoracic or abdominal surgery; and inability to perform technically acceptable spirometric manoeuvres as per ATS/ERS guidelines. [8]

Anthropometric Measurements: Height was recorded using a standardised wall-mounted stadiometer to the nearest 0.1 cm. Weight was measured on a calibrated digital scale to the nearest 0.1 kg. BMI was calculated as weight (kg) / height (m^2).

Biochemical Analysis: Venous blood samples were collected under aseptic precautions. HbA1c was quantitatively measured using High-Performance Liquid Chromatography (HPLC) on an automated analyser.

Spirometry: Pulmonary function testing was performed using the RMS Helios 401 calibrated computerised spirometer, following ATS/ERS 2019 technical standards. [8] The instrument was calibrated daily using a standard calibration syringe. The test was performed in the sitting position with a nose clip applied; participants exhaled forcefully and completely for a minimum of six seconds following maximal inspiration. A minimum of three acceptable manoeuvres were recorded and the best technically valid effort selected, in conformity with ATS/ERS 2019 acceptability and reproducibility criteria. [8]

Parameters recorded included FVC, FEV_1, and FEV_1/FVC ratio. Values were expressed as absolute measurements and as percentage predicted values, with predicted reference values derived from Global Lung Function Initiative 2012 (GLI-2012) reference equations, as recommended by ATS/ERS guidelines. [8]

Statistical Analysis: Data were analysed using IBM SPSS Statistics (Version 28.0). Continuous variables were expressed as mean ± Standard Deviation (SD). An unpaired Student's t-test was used to compare spirometric parameters between groups. Pearson's correlation coefficient was applied to evaluate relationships between spirometric parameters, HbA1c, and BMI. A p-value of <0.05 was considered statistically significant.

Results

Table 1: Baseline Demographic and Clinical Characteristics of Participants

Parameter	Controls (n=50) Mean ± SD	Diabetics (n=50) Mean ± SD	p-value	Significance
Age (years)	45.88 ± 7.48	46.84 ± 7.41	>0.05	NS
Height (cm)	165.16 ± 8.34	164.14 ± 8.59	>0.05	NS
Weight (kg)	70.50 ± 9.14	71.28 ± 9.79	>0.05	NS
BMI (kg/m²)	25.73 ± 1.43	26.42 ± 2.70	>0.05	NS
HbA1c (%)	5.28 ± 0.31	8.94 ± 1.93	**<0.05**	**Significant**

BMI: Body Mass Index; HbA1c: Glycated Haemoglobin; NS: Not Significant. p<0.05 — Significant; p>0.05 — Not Significant

The two groups were well-matched for age, height, weight, and BMI (p>0.05 for all), ensuring anthropometric comparability and minimising confounding of spirometric values. HbA1c was, as expected, markedly elevated in the diabetic group (8.94 ± 1.93% vs 5.28 ± 0.31%; p<0.05), confirming metabolic divergence between groups.

Table 2: Comparison of Percentage Predicted Pulmonary Function Parameters between Controls and Diabetics

Parameter (% Predicted)	Controls (n=50) Mean ± SD	Diabetics (n=50) Mean ± SD	p-value	Significance
FVC (%)	91.40 ± 5.19	87.98 ± 6.45	**<0.05**	**Significant**
FEV_1 (%)	91.52 ± 5.21	89.68 ± 5.39	>0.05	NS
FEV_1/FVC	0.76 ± 0.05	0.77 ± 0.03	>0.05	NS

FVC: Forced Vital Capacity; FEV_1: Forced Expiratory Volume in 1 second; NS: Not Significant. p<0.05 — Significant; p>0.05 — Not Significant

Percentage predicted FVC was significantly lower in the diabetic group (87.98 ± 6.45% vs 91.40 ± 5.19%; p<0.05). Percentage predicted FEV_1 showed a numerical reduction in diabetics but did not achieve statistical significance (89.68 ± 5.39% vs 91.52 ± 5.21%; p>0.05). The FEV_1/FVC ratio was virtually identical between groups (0.77 ± 0.03 vs 0.76 ± 0.05; p>0.05). The pattern of reduced FVC% with a preserved FEV_1/FVC ratio is suggestive of a restrictive ventilatory pattern.

Table 3: Pearson Correlation of Pulmonary Function Parameters with BMI (Diabetic Group, n=50)

Parameter (% Predicted)	r value	p-value	Significance
FVC (%)	−0.38	**<0.05**	**Significant**
FEV_1 (%)	−0.31	**<0.05**	**Significant**
FEV_1/FVC	0.03	>0.05	NS

r: Pearson correlation coefficient; NS: Not Significant. p<0.05 — Significant; p>0.05 — Not Significant

Within the diabetic group, BMI demonstrated significant inverse correlations with both FVC% (r = −0.38, p<0.05) and FEV_1% (r = −0.31, p<0.05). No significant correlation was identified between BMI and the FEV_1/FVC ratio (r = 0.03, p>0.05).

Stratification of diabetic subjects by glycaemic control (HbA1c <7%, n=10; HbA1c ≥7%, n=40) revealed no statistically significant difference in any spirometric parameter: FVC% (90.05 ± 6.56% vs 87.47 ± 6.40%; p>0.05), FEV_1% (92.12 ± 4.32% vs 89.07 ± 5.50%; p>0.05), and FEV_1/FVC (0.77 ± 0.02 vs 0.77 ± 0.03; p>0.05). Pearson correlation analysis within the diabetic group also revealed no statistically significant association between HbA1c and FVC% (r = −0.24, p>0.05), FEV_1% (r = −0.19, p>0.05), or FEV_1/FVC (r = −0.04, p>0.05).

Table 4: Pearson Correlation of Pulmonary Function Parameters with HbA1c (Diabetic Group, n=50)

Parameter (% Predicted)	r value	p-value	Significance
FVC (%)	−0.24	>0.05	NS
FEV_1 (%)	−0.19	>0.05	NS
FEV_1/FVC	−0.04	>0.05	NS

r: Pearson correlation coefficient; NS: Not Significant. HbA1c: Glycated Haemoglobin. p<0.05 — Significant; p>0.05 — Not Significant

Discussion

The present study assessed spirometric pulmonary function in patients with Type 2 Diabetes Mellitus in comparison with age- and BMI-matched healthy controls. The findings demonstrate subclinical restrictive pulmonary impairment in T2DM, with BMI demonstrating a stronger association with spirometric impairment than HbA1c.

Demographic Validity: The two groups were well-matched for age, height, weight, and BMI (p>0.05 for all), ensuring that observed spirometric differences are attributable to the metabolic milieu of diabetes rather than constitutional variables. [8] The markedly elevated HbA1c in diabetics (8.94 ± 1.93% vs 5.28 ± 0.31%; p<0.05) confirmed appropriate metabolic separation between groups.

Forced Vital Capacity: Percentage predicted FVC was significantly lower in diabetics (87.98 ± 6.45% vs 91.40 ± 5.19%; p<0.05). The reduction in % predicted FVC — which adjusts for age, sex, and height — points to genuine impairment in lung volume reflecting glycation-induced cross-linking of pulmonary connective tissue, reducing elastic distensibility. [6,7] The Fremantle Diabetes Study by Davis et al. demonstrated that cumulative glycaemic exposure is independently associated with progressive decline in FVC in T2DM, [11] while the meta-analysis by Díez-Manglano and Asín Samper established a consistent, albeit modest, reduction in FVC across diverse diabetic populations. [12] Indian hospital-based studies by Shah et al., Mittal et al., and El-Habashy et al. have reported similar findings. [13,14,15]

FEV_1 and Pattern of Ventilatory Impairment: Percentage predicted FEV_1 showed a numerical reduction in diabetics but did not achieve statistical significance (89.68 ± 5.39% vs 91.52 ± 5.21%; p>0.05). The FEV_1/FVC ratio was preserved in both groups (0.77 ± 0.03 vs 0.76 ± 0.05; p>0.05). This pattern of parallel proportionate reduction in both FEV_1 and FVC — without reduction in their ratio — is consistent with a restrictive rather than obstructive pattern of ventilatory dysfunction, a finding corroborated by multiple prior studies in diabetic cohorts.[12,13] It reflects parenchymal stiffening secondary to AGE-mediated collagen cross-linking and thoracic adiposity, rather than obstructive airway disease. [6,7]

Relationship with BMI: Within the diabetic subgroup, significant inverse correlations were identified between BMI and both FVC% (r = −0.38, p<0.05) and FEV_1% (r = −0.31, p<0.05). The mechanical basis is well established: excess adipose tissue restricts diaphragmatic descent, reduces chest wall compliance, and diminishes functional residual capacity. [9] In T2DM, these mechanical constraints may be compounded by hyperglycaemia-related pulmonary connective tissue changes, creating a synergistic adverse effect.[9] These findings align with those of Hayfron-Benjamin et al., who demonstrated that T2DM and obesity compound spirometric impairment,[15] and with Indian studies by Shah et al. showing central adiposity significantly associates with reduced FVC and FEV_1 in T2DM. [13]

Relationship with HbA1c: No statistically significant correlation was observed between HbA1c and any spirometric parameter (r values: −0.04 to −0.24; all p>0.05). Glycaemic stratification similarly produced no significant spirometric differences. These observations are consistent with prior cross-sectional studies by Mittal et al. and El-Habashy et al., [14,15] and with Shah et al., who attributed non-significance to the large physiological reserve of the human lung masking early sub-clinical changes. [13] Fundamentally, HbA1c reflects glycaemic exposure over only the preceding 8–12 weeks and cannot capture the cumulative glycaemic burden required to drive significant pulmonary connective tissue changes.[11] Longitudinal studies incorporating cumulative glycaemic indices and disease duration are warranted to more precisely characterise the independent contribution of glycaemic control to pulmonary impairment in T2DM.

Conclusion

This hospital-based comparative cross-sectional study demonstrates that patients with Type 2 Diabetes Mellitus exhibit subclinical pulmonary impairment characterised by a statistically significant reduction in percentage predicted FVC, with preservation of the FEV_1/FVC ratio, suggestive of a restrictive ventilatory pattern. BMI demonstrated significant inverse correlations with both FVC% and FEV_1% in the diabetic group, while HbA1c showed no statistically significant association with any spirometric parameter. These findings indicate that BMI demonstrated a stronger association with spirometric impairment than

HbA1c. Although the observed changes remain subclinical and do not cross formal diagnostic thresholds for restrictive lung disease, their detection in asymptomatic individuals underscores the potential value of integrating routine spirometric screening into comprehensive diabetes management, particularly in patients with higher BMI. Larger prospective longitudinal studies incorporating cumulative glycaemic indices, disease duration, and multivariate modelling are required to precisely define the trajectory of diabetes-related pulmonary impairment and determine whether early glycaemic optimisation or weight reduction can attenuate its progression.

References

1 Galicia-Garcia U, Benito-Vicente A, Jebari S, Larrea-Sebal A, Siddiqi H, Uribe KB, et al. Pathophysiology of type 2 diabetes mellitus. Int J Mol Sci. 2020;21(17):6275. doi:10.3390/ijms21176275.

2 Kharroubi AT, Darwish HM. Diabetes mellitus: the epidemic of the century. World J Diabetes. 2015;6(6):850–867. doi:10.4239/wjd.v6.i6.850.

3 International Diabetes Federation. IDF diabetes atlas. 10th ed. Brussels: International Diabetes Federation; 2021.

4 Anjana RM, Unnikrishnan R, Deepa M, Pradeepa R, Tandon N, Das AK, et al. Metabolic non-communicable disease health report of India: the ICMR-INDIAB national cross-sectional study. Lancet Diabetes Endocrinol. 2023;11(7):474–489. doi:10.1016/S2213-8587(23)00119-5.

5 Kopf S, Kumar V, Kender Z, Han Z, Fleming T, Herzig S, et al. Diabetic pneumopathy — a new diabetes-associated complication: mechanisms, consequences and treatment considerations. Front Endocrinol (Lausanne). 2021;12:765201. doi:10.3389/fendo.2021.765201.

6 Pitocco D, Fuso L, Conte EG, Zaccardi F, Condoluci C, Scavone G, et al. The diabetic lung: a new target organ? Rev Diabet Stud. 2012;9(1):23–35. doi:10.1900/RDS.2012.9.23.

7 Khalid M, Petroianu G, Adem A. Advanced glycation end products and diabetes mellitus: mechanisms and perspectives. Biomolecules. 2022;12(4):542. doi:10.3390/biom12040542.

8 Graham BL, Steenbruggen I, Miller MR, Barjaktarevic IZ, Cooper BG, Hall GL, et al. Standardization of spirometry 2019 update: an official American Thoracic Society and European Respiratory Society technical statement. Am J Respir Crit Care Med. 2019;200(8):e70–e88. doi:10.1164/rccm.201908-1590ST.

9 Salome CM, King GG, Berend N. Physiology of obesity and effects on lung function. J Appl Physiol. 2010;108(1):206–211. doi:10.1152/japplphysiol.00678.2009.

10 American Diabetes Association Professional Practice Committee. Diagnosis and classification of diabetes: Standards of care in diabetes—2024. Diabetes Care. 2024;47(Suppl 1):S20–S42. doi:10.2337/dc24-S002.

11 Davis WA, Knuiman M, Kendall P, Grange V, Davis TME. Glycemic exposure is associated with reduced pulmonary function in type 2 diabetes: the Fremantle Diabetes Study. Diabetes Care. 2004;27(3):752–757. doi:10.2337/diacare.27.3.752.

12 Díez-Manglano J, Asín Samper U. Pulmonary function tests in type 2 diabetes: a meta-analysis. ERJ Open Res. 2021;7(1):00371-2020. doi:10.1183/23120541.00371-2020.

13 Shah SH, Sonawane P, Nahar P, Vaidya S, Salvi S. Pulmonary function tests in type 2 diabetes mellitus and their association with glycemic control and duration of the disease. Lung India. 2013;30(2):108–112. doi:10.4103/0970-2113.110417.

14 Mittal S, Jindal M, Srivastava S, Sinha S. Evaluation of pulmonary functions in patients with type 2 diabetes mellitus: a cross-sectional study. Cureus. 2023;15(3):e35628. doi:10.7759/cureus.35628.

15 El-Habashy MA, Shakweer MM, Hassan MA, El-Adawy AR. Effect of glycemic control on pulmonary function in type 2 diabetic patients. Egypt J Chest Dis Tuberc. 2014;63(3):685–691.