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Assessment of pulmonary function test in type 2 diabetes mellitus and its relation to glycaemic control and duration of disease

Abstract

Background: Diabetes mellitus is a metabolic disease that affects many systems and is marked by extensive vascular problems. One large microvascular bed that may be vulnerable to diabetic microangiopathy is the pulmonary alveolar–capillary network. This study aimed to assess pulmonary function in patients with type 2 diabetes mellitus (T2DM) and to examine its association with glycaemic control and disease duration.

Methods: This cross-sectional observational study was conducted at Ain Shams University Hospitals between February 2025 and February 2026. A total of 150 participants were enrolled, including 50 patients with controlled T2DM (HbA1c ≤ 7%), 50 patients with uncontrolled T2DM (HbA1c > 7%), and 50 age-matched healthy controls. Participants were recruited from both outpatient and inpatient settings. Data collection included fasting blood glucose, 2-hour postprandial glucose, HbA1c levels, and pulmonary function tests (PFTs).

Results: Baseline demographic characteristics, including age, sex, and body mass index, were comparable among groups. Pulmonary function parameters (FEV1, FVC, FEV1/FVC, FEF25, FEF50, and FEF25–75) were significantly reduced in the uncontrolled group, indicating both restrictive and small airway impairment. HbA1c showed significant negative correlations with all spirometric indices, while longer diabetes duration was also associated with reduced pulmonary function. Multiple regression analysis identified HbA1c as the strongest independent predictor of pulmonary function decline.

Conclusion: Uncontrolled T2DM is associated with significant impairment in pulmonary function. These findings support incorporating pulmonary function assessment into routine diabetes care.

Key words: Type 2 diabetes mellitus; Glycaemic control; Pulmonary function tests; HbA1c; Spirometry.

Introduction:

Diabetes mellitus is a metabolic disease that affects many organs and is marked by extensive vascular problems. The pulmonary consequences of diabetes mellitus have not been sufficiently characterized, despite contradictory findings. One important microvascular unit in the lung, the alveolar capillary network, may be impacted by microangiopathy. The significant reserve of the microvascular bed makes it possible to tolerate significant loss without experiencing dyspnea. This could result in pulmonary diabetic microangiopathy being underdiagnosed clinically. Research on the pathogenesis of reduced pulmonary function is very fascinating. Normal lung mechanics and gas exchange are influenced by the health of the pulmonary connective tissue and microvasculature. The presence of nonenzymatic glycosylation, the acceleration of the aging process in connective tissue cross connections, and the modification of alveolar surfactant activity are the causes of the decline in pulmonary function tests (PFTs). [(1)].

Diabetes mellitus (DM) and reduced pulmonary function have been linked for many years. Numerous studies have shown a connection between diabetes and reduced lung function. There is growing evidence that chronic hyperglycemia may have an impact on pulmonary tissue. indicating that a significant contributing factor to type 2 diabetic patients' reduced lung function is their exposure to glucose. [(2)].

According to a meta-analysis of cross-sectional studies, individuals with diabetes had lower forced vital capacity (FVC) and forced expiratory volume in 1s (FEV1) than those without the disease, which is consistent with the link between diabetes and impaired lung function[(3)].

Research on Type 2 Diabetes Mellitus (T2DM) shows that it is also linked to decreased pulmonary function parameters, such as forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1), which often exhibit a restrictive ventilatory pattern, even though pulmonary dysfunction in Type 1 diabetes has been better described[(4)].

The expected causes include the accumulation of advanced glycation endproducts in lung tissue and persistent rise of blood glucose, which contributes to oxidative damage and inflammatory pathways that may compromise pulmonary structure and function [5].

13 The objective of this work was to assess PFTs in T2DM and its relation to glycaemic control and duration of illness

Patients and Methods:

This cross-sectional, observational study was executed in Ain Shams University Hospitals from February 2025 to February 2026, enrolling 50 patients with controlled type 2 DM (glycosylated haemoglobin less than or equal seven percent), 50 patients with uncontrolled type 2 DM (glycosylated haemoglobin more than 7%), and 50 age-matched individuals as controls. The patients and controls were recruited from outpatient and inpatient settings at Ain Shams University Hospitals.

Ethical approval

The study was approved by the Ethics Committee of Ain Shams University, and informed consent was obtained from all participants.

Inclusion criteria: Participants were adults above 18 years old, including patients with controlled type 2 DM (HbA1c ≤7%), patients with uncontrolled type 2 DM (HbA1c >7%), and age-matched healthy controls.

Exclusion criteria: current or former smokers, participants with cough, sputum, or dyspnea, and those with known respiratory diseases such as COPD, bronchial asthma, bronchiectasis, interstitial lung disease, prior pulmonary tuberculosis, or other major systemic or cardiac illnesses that could affect pulmonary function.

All patients were subjected to a full detailed medical history and clinical examination, measurement of HbA1c (glycosylated hemoglobin), fasting and postprandial blood glucose,

and spirometry for FEV1, FVC, FEV1/FVC, FEF25, FEF50, and FEF25–75, while the control group recruited after confirm normal HbA1c then underwent only spirometry.

Spirometry was performed according to American Thoracic Society / European Respiratory Society (ATS/ERS) recommendations using calibrated equipment by trained personnel. At least three acceptable manoeuvres were obtained, and the best reproducible values were recorded. Predicted values were generated using manufacturer reference equations based on age, sex, and height.

Pre-bronchodilator spirometry was used for study comparisons. Individuals with known asthma, COPD, smoking history, or respiratory symptoms were excluded during screening.

Restrictive tendency was interpreted as reduced FVC with preserved/increased FEV1/FVC ratio, whereas obstructive tendency was considered when FEV1/FVC was reduced

Statistical analysis:

All collected data were organized, coded, and initially tabulated using Microsoft Excel 2016 (Microsoft Corporation, USA). Statistical processing was carried out utilizing IBM SPSS Statistics for Windows, version 27.0 (IBM Corp., Armonk, NY, USA).

The distribution of continuous variables was assessed for normality utilizing the Kolmogorov–Smirnov test. Normally disseminated quantitative variables were summarized as mean ± SD, along with minimum and maximum values. For variables that deviated from normality, data were expressed as interquartile range (IQR)and median. Categorical variables were described utilizing percentages and frequencies. Statistical significance was determined at a two-tailed p-value < 0.05. Comparisons between groups were performed utilizing one-way ANOVA or Kruskal–Wallis tests as appropriate, with post hoc analyses (Tukey HSD or Bonferroni correction). Correlation analysis was conducted utilizing Spearman's rank correlation coefficient. Multiple linear regression analysis was performed to classify independent predictors of pulmonary function.

Results:

This study conducted in Ain shams university hospitals either outpatients or inpatients from February 2025 to February 2026,The study included three groups, each comprising 50 participants: controlled T2DM, uncontrolled type 2 DM, and healthy controls.

Table (1) shows the demographic, characteristics among controlled type 2 diabetes mellitus (T2DM), uncontrolled T2DM, and healthy controls. There were no statistically significant differences among the three groups regarding baseline characteristics. The distribution of gender was non-significant (p = 0.723), with males constituting 40%, 48%, and 44% in the three groups, respectively. Age was also non-significant between groups (p = 0.162), with mean of 53.16 ± 5.89 years in the controlled group, 50.91 ± 6.02 years in the uncontrolled group, and 52.62 ± 6.46 years in controls. When categorized, age groups (<50 and >50 years) remained non-significant (p = 0.098).

Body mass index (BMI) values were also non-significant (p = 0.185). The controlled T2DM group showed the highest median BMI [27.35 (25.4–30.1) kg/m²], followed by the uncontrolled group [26.55 (24.4–29.1) kg/m²], and controls [25.85 (24.4–28.2) kg/m²].

Table (1): Demographic characteristics of the studied groups.

		Controlled type 2 DM (N=50)	Uncontrolled type 2 DM (N=50)	Controls (N=50)	p- value
Gender	**Male**	20 (40.0%)	24 (48.0%)	22 (44.0%)	0.723‡
	Female	30 (60.0%)	26 (52.0%)	28 (56.0%)	
Age (years)	**Mean± SD**	53.16 ± 5.89	50.91 ± 6.02	52.62 ± 6.46	0.162*
	Min.- Max	40–65	40–62	40–65	
Age group	**<50 years**	13 (26.0%)	23 (46.0%)	16 (32.0%)	0.098‡
	>50 years	37 (74.0%)	27 (54.0%)	34 (68.0%)	
BMI (Kg/m²)	**Median (IQR)**	27.35 (25.4–30.1)	26.55 (24.4–29.1)	25.85 (24.4–28.2)	0.185#
	Min.- Max	20.4–38.4	21.2–36.9	18.8–37.0	

p>0.05 is non-significant; p≤0.05 is significant. *One- Way ANOVA test, ‡ Chi-square test,
p1: p-value between controlled and uncontrolled type 2 DM, p2: p-value between controlled type 2 DM and controls, p3: p-value between uncontrolled type 2 DM and controls

Table (2) show analysis of glycaemic control and disease duration. The results revealed significant differences between controlled and uncontrolled type 2 DM patients. As expected, the uncontrolled group had higher HbA1c levels, with a median of 9.5% (IQR 9–10.1) compared to 6.7% (IQR 6.5–6.9) in the controlled group ($p < 0.001$). Similarly, fasting blood glucose (FBS) and 2-hour postprandial glucose (2-hr PP) were significantly elevated in uncontrolled diabetics, with median values of 192.5 mg/dL (IQR 178–207) and 282.5 mg/dL (IQR 264–300), respectively, compared to 113 mg/dL (IQR 109–121) and 170 mg/dL (IQR 136–197) in the controlled group ($p < 0.001$ for both). Moreover, the duration of diabetes was longer in the uncontrolled group, with a mean of 10.50 ± 4.03 years versus 7.36 ± 2.97 years in the controlled group ($p < 0.001$).

Table (2): Clinical characteristics of the studied groups.

		Controlled type 2 DM (N=50)	Uncontrolled type 2 DM (N=50)	p-value
HbA1c (%)	**Median (IQR)**	6.7 (6.5–6.9)	9.5 (9.0–10.1)	<0.001#
	Min.- Max	5.9–7.4	7.9–11.2	
FBS (mg/dL)	**Median (IQR)**	113 (109–121)	192.5 (178–207)	<0.001#
	Min.- Max	88–132	139–245	
2-hr PP (mg/dL)	**Median (IQR)**	170 (155–177)	282.5 (264–300)	<0.001#
	Min.- Max	136–197	234–339	
Duration (years)	**Mean ± SD**	7.36 ± 2.97	10.50 ± 4.03	<0.001‡
	Min.- Max	1–16.30	1.40–20	

Mann-Whitney U test. ‡ Independent Samples T test

PFTs showed significant differences among the three study groups. FEV1 (%) was significantly reduced in the uncontrolled T2DM group (82.33 ± 9.02) compared to both the controlled group (91.14 ± 8.37) and healthy controls (97.15 ± 8.19) ($p < 0.001$). Post hoc analysis confirmed significant differences between all groups indicating a progressive decline from healthy controls to controlled and further to uncontrolled patients. A similar pattern was observed for FVC (%), which was significantly lower in the uncontrolled group (79.82 ± 8.35) compared to controlled patients (92.01 ± 7.63) and healthy controls (96.69 ± 7.11) ($p < 0.001$), with significant pairwise differences. The FEV1/FVC ratio showed a statistically significant overall

difference ($p < 0.001$), with lower median values in the uncontrolled group [78.4 (75.6–81.2)] compared to healthy controls [82.0 (78.5–84.7)], while the controlled group [79.5 (77.5–81.9)] was intermediate. Post hoc analysis revealed non-significant difference between controlled and uncontrolled groups ($p2 = 0.820$), whereas both differed significantly from healthy controls ($p1 < 0.001$, $p3 < 0.001$).

Regarding small airway function, FEF25 (%), FEF50 (%), and FEF25–75 (%) were all significantly reduced in the uncontrolled T2DM group compared to both controlled patients and healthy controls ($p < 0.001$ for all). For FEF25 (%), all pairwise comparisons were statistically significant ($p < 0.001$). For FEF50 (%) and FEF25–75 (%), although all comparisons were significant, the difference between controlled and uncontrolled groups was less pronounced ($p1 = 0.024$ and $p1 = 0.012$, respectively) compared to comparisons with healthy controls ($p2$ and $p3 < 0.001$) as shown in **Table (3). Figure (1)** show PFTs among studied groups.

Table (3): Pulmonary function tests among studied groups.

		Controlled type 2 DM (N=50)	Uncontrolled type 2 DM (N=50)	Controls (N=50)	p-value	Post hoc analysis
FEV1 (%)	**Mean ± SD**	91.14 ± 8.37	82.33 ± 9.02	97.15 ± 8.19	<0.001*	p1<0.001, p2=0.002, p3<0.001
	Min–Max	72.0–107.0	59.3–101.4	80.6–115.3		
FVC (%)	**Mean ± SD**	92.01 ± 7.63	79.82 ± 8.35	96.69 ± 7.11	<0.001*	p1<0.001, p2=0.008, p3<0.001
	Min–Max	74.5–108.2	61.5–100.9	81.0–110.7		
FEV1/FVC	**Median (IQR)**	79.5 (77.5–81.9)	78.4 (75.6–81.2)	82.0 (78.5–84.7)	<0.001*	p1<0.001, p2=0.820, p3<0.001
	Min–Max	68.9–89.2	61.2–87.1	71.5–89.6		
FEF25 (%)	**Mean ± SD**	90.27 ± 9.52	75.88 ± 9.63	99.96 ± 8.52	<0.001*	p1<0.001, p2<0.001, p3<0.001
	Min–Max	71.3–114.0	56.6–92.5	85.4–125.4		
FEF50 (%)	**Mean ± SD**	84.77 ± 12.32	78.85 ± 11.69	95.83 ± 9.36	<0.001*	p1=0.024, p2<0.001, p3<0.001
	Min–Max	51.2–111.6	48.5–105.7	74.6–113.1		
FEF25–75 (%)	**Mean ± SD**	83.54 ± 10.32	77.65 ± 10.82	92.46 ± 9.42	<0.001*	p1=0.012, p2<0.001, p3<0.001
	Min–Max	63.6–105.8	56.3–103.3	77.8–117.6		

p>0.05 is non-significant; p≤0.05 is significant. *One- Way ANOVA test, * Kruskal Wallis test
p1: p-value between controlled and uncontrolled type 2 DM, p2: p-value between controlled type 2 DM and controls, p3: p-value between uncontrolled type 2 DM and controls

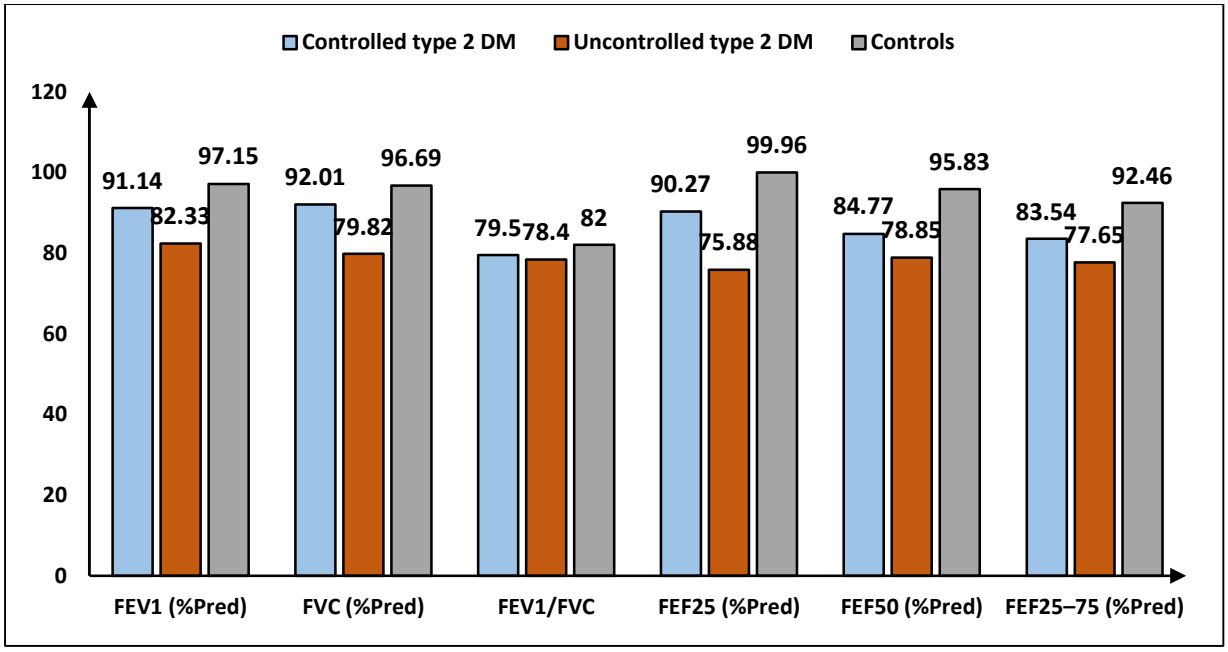

Figure 1: Pulmonary function tests among studied groups.

The correlation analysis demonstrated that glycaemic control, as reflected by HbA1c, showed a statistically significant negative associations with pulmonary function parameters. Specifically, HbA1c was significantly inversely correlated with FEV1 ($r = -0.385$, $p < 0.001$), FVC ($r = -0.580$, $p < 0.001$), FEF25% ($r = -0.530$, $p < 0.001$), FEF50% ($r = -0.211$, $p = 0.035$), a and non-significant correlation with FEV1/FVC ratio ($r = -0.166$, $p = 0.099$), and FEF25–75% ($r = -0.191$, $p = 0.057$), indicating that poorer long-term glycaemic control is associated with a decline in both large and small airway function.

Table (4): Correlation between glycaemic control and pulmonary function tests in controlled and uncontrolled type 2 DM.

Pulmonary Parameter	HbA1c (r)	p-value
FEV1 (%)	-0.385	<0.001
FVC (%)	-0.580	<0.001
FEV1/FVC	-0.166	0.099
FEF25 (%)	-0.530	<0.001
FEF50 (%)	-0.211	0.035

Pulmonary Parameter	HbA1c (r)	p-value
FEF25–75 (%)	-0.191	0.057

p>0.05 is non-significant; p≤0.05 is significant. r: Spearman correlation coefficient

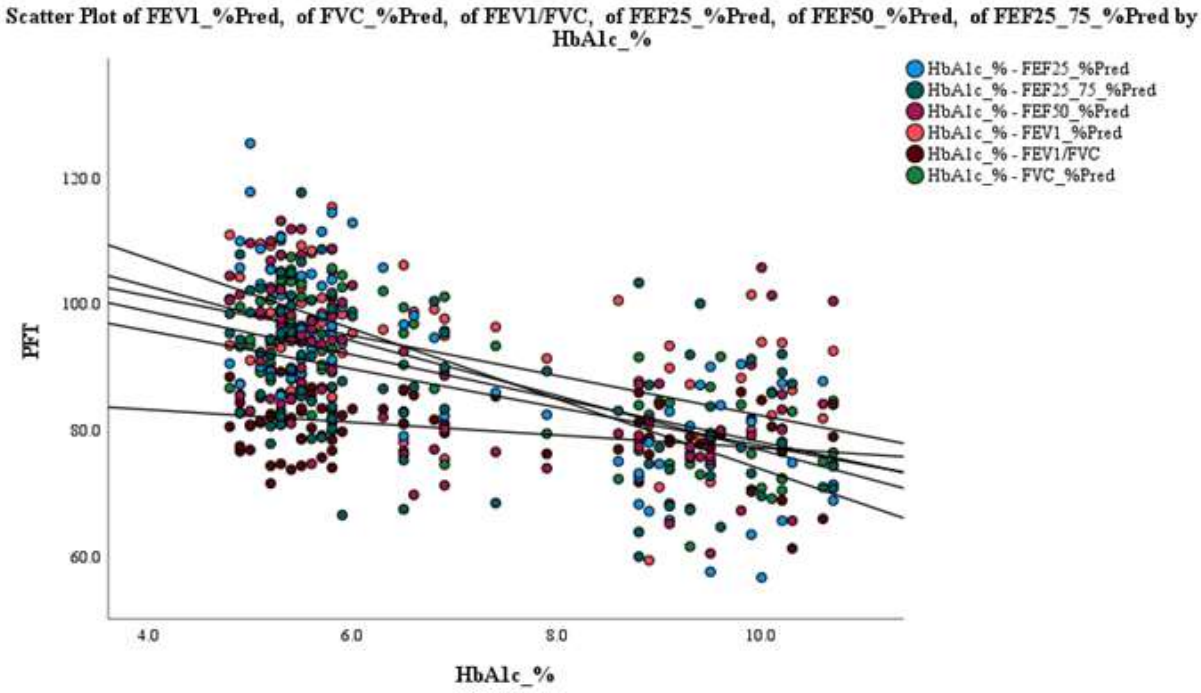

Figure 2: Correlation between HbA1c levels and PFT parameters in Controlled and uncontrolled type 2 DM patients.

Table (5) show Analysis of the relationship among the duration of T2DM and pulmonary function. Diabetes duration showed significant correlations withFEV1%,FVC,FEF25%. longer disease duration was significantly associated with lower FEV1 ($r = -0.338$, $p<0.001$), FVC ($r = -0.261$, $p = 0.009$), FEF25% ($r = -0.265$, $p = 0.008$).

Table (5): Correlation between pulmonary function and duration of diabetes.

Pulmonary Parameter	Duration (r)	p-value
FEV1 (%)	-0.338	<0.001
FVC (%)	-0.261	0.009
FEV1/FVC	-0.050	0.619
FEF25 (%)	-0.265	0.008
FEF50 (%)	-0.161	0.111

Pulmonary Parameter	Duration (r)	p-value
FEF25–75 (%)	-0.026	0.798

p>0.05 is non-significant; p≤0.05 is significant. r: Pearson/Spearman correlation coefficient

Table (6) show Analysis of the relationship among the BMI and pulmonary function in both controlled and uncontrolled T2DM groups. In the controlled group, FEV1%, FVC%, FEF25%, FEF50%, and FEF25–75% showed no significant correlations with BMI ($p > 0.05$). However, a statistically significant weak negative correlation was observed between BMI and FEV1/FVC ratio ($r = -0.313$, $p = 0.027$). In the uncontrolled T2DM group, all pulmonary parameters, including FEV1%, FVC%, FEV1/FVC, and small airway indices (FEF25%, FEF50%, FEF25–75%), showed no significant correlations with BMI ($p > 0.05$).

Table (6): Correlation between pulmonary function and BMI.

Pulmonary parameter	BMI (Kg/m^2)			
	Controlled type 2 DM (N=50)		Uncontrolled type 2 DM (N=50)	
	r	P-value	r	P-value
FEV1 (%)	0.054	0.708	0.235	0.100
FVC (%)	-0.213	0.137	-0.110	0.447
FEV1/FVC	-0.313	**0.027**	0.040	0.781
FEF25 (%)	-0.052	0.719	-0.023	0.877
FEF50 (%)	0.220	0.125	-0.007	0.963
FEF25–75 (%)	0.089	0.538	-0.001	0.995

p>0.05 is non-significant; p≤0.05 is significant. r: Spearman correlation coefficient

Table (7) show Multiple regression analysis that was conducted to recognise independent predictors of pulmonary function. Multiple regression analysis revealed that HbA1c was the only consistent independent predictor of pulmonary function impairment across most parameters. For FEV1%, HbA1c showed a significant negative association ($B = -2.188$, $\beta = -0.346$, $p < 0.001$), while age, gender, BMI, and diabetes duration were non-significant. Similarly, for FVC%, HbA1c remained a strong negative predictor ($B = -4.126$, $\beta = -0.631$, $p < 0.001$), with all other variables showing non-significant effects. Regarding the FEV1/FVC ratio, HbA1c also demonstrated a significant negative association ($B = -0.783$, $\beta = -0.250$, $p = 0.024$), while age, gender, BMI, and duration were non-significant. For FEF25 , HbA1c (B

226 = −4.165, β = −0.534, p < 0.001) were significant predictors, indicating higher HbA1c was
227 associated with lower values.

228 In contrast, no variables were significant predictors of FEF50%. For FEF25–75%, HbA1c
229 again showed a significant negative association (B = −1.932, β = −0.271, p = 0.015), while age,
230 gender, BMI, and duration were non-significant.

231 **Table (7): Multiple regression analysis to identify independent predictors of pulmonary**
232 **function.**

	Unstandardized Coefficients		Standardized Coefficients	t	P-value
	B	Std. Error	Beta		
Age (years)	0.116	0.146	0.072	0.793	0.430
Gender	1.949	1.793	0.100	1.087	0.280
BMI	0.209	0.244	0.079	0.856	0.394
HBA1c	-2.188	0.628	-0.346	-3.484	**<0.001**
Duration (years)	-0.421	0.249	-0.167	-1.687	0.095
a. Dependent Variable: FEV1%					
	Unstandardized Coefficients		Standardized Coefficients	t	P-value
	B	Std. Error	Beta		
Age (years)	-0.185	0.135	-0.111	-1.375	0.172
Gender	0.883	1.649	0.044	0.536	0.593
BMI	-0.383	0.224	-0.140	-1.708	0.091
HBA1c	-4.126	0.578	-0.631	-7.139	**<0.001**
Duration (years)	0.013	0.229	0.005	0.058	0.954
a. Dependent Variable: FVC%					
	Unstandardized Coefficients		Standardized Coefficients	t	P-value
	B	Std. Error	Beta		
Age (years)	-0.051	0.080	-0.064	-0.640	0.524
Gender	-1.042	0.976	-0.108	-1.068	0.288
BMI	-0.151	0.133	-0.115	-1.136	0.259
HBA1c	-0.783	0.342	-0.250	-2.291	**0.024**
Duration (years)	0.030	0.136	0.024	0.224	0.823
a. Dependent Variable: FEV1/FVC					
	Unstandardized Coefficients		Standardized Coefficients	t	P-value
	B	Std. Error	Beta		
Age (years)	0.415	0.161	0.209	2.574	**0.012**
Gender	-2.977	1.973	-0.124	-1.509	0.135
BMI	0.007	0.268	0.002	0.026	0.979
HBA1c	-4.165	0.691	-0.534	-6.023	**<0.001**
Duration (years)	-0.131	0.275	-0.042	-0.477	0.634
a. Dependent Variable: FEF25 (%)					

	Unstandardized Coefficients		Standardized Coefficients	t	P-value
	B	Std. Error	Beta		
Age (years)	0.228	0.198	0.112	1.150	0.253
Gender	-3.595	2.428	-0.146	-1.481	0.142
BMI	-0.383	0.224	-0.140	-1.708	0.091
HBA1c	-1.189	0.851	-0.148	-1.398	0.165
Duration (years)	-0.290	0.338	-0.091	-0.860	0.392
a. Dependent Variable: FEF50 (%)					
	Unstandardized Coefficients		Standardized Coefficients	t	P-value
	B	Std. Error	Beta		
Age (years)	-0.061	0.182	-0.034	-0.334	0.739
Gender	0.021	2.227	0.001	0.009	0.993
BMI	0.246	0.303	0.083	0.814	0.418
HBA1c	-1.932	0.780	-0.271	-2.475	**0.015**
Duration (years)	0.373	0.310	0.132	1.204	0.231
a. Dependent Variable: FEF25–75 (%)					

Discussion:

In the present study, baseline characteristics were comparable among the three groups, as age, sex distribution, and BMI did not differ significantly. This reduces the likelihood that differences in spirometric indices were related to demographic imbalance rather than diabetes itself. Matching these variables is important because age, obesity, and sex are recognized determinants of lung function.

The present study demonstrated that pulmonary function is significantly impaired in patients with type 2 diabetes mellitus (T2DM), particularly in those with poor glycaemic control. Patients with uncontrolled T2DM had significantly lower spirometric indices, including FEV1%, FVC%, FEV1/FVC ratio, FEF25%, FEF50%, and FEF25–75%, compared with controlled diabetic patients and healthy controls. These findings suggest that chronic hyperglycaemia adversely affects both large and small airway function and may induce a mixed restrictive and obstructive ventilatory defect.

In the current study, gender distribution did not differ significantly among groups, indicating that sex is unlikely to confound the relationship between diabetes and pulmonary function. This

agrees with Davis et al.(6), who found no consistent association between gender and lung function decline in T2DM patients. Age also did not differ significantly between groups. Although ageing is known to reduce lung elasticity and function, regression analysis in this study demonstrated that age was not an independent predictor of PFT, suggesting that hyperglycaemia exerts a greater effect than age alone. Similar findings were reported by Klein et al.(7).

A major finding of this study is the reduction in pulmonary function parameters including FEV1, FVC, and mid-expiratory flow rates in uncontrolled diabetic patients compared with controlled patients and healthy controls. These findings are consistent with Meo et al.(2), who reported reduced lung volumes and airflow limitation in T2DM. Our results also agree with Benbassat et al.(8), who described similar pulmonary impairment in diabetic patients.

The pattern observed in the present study suggests both restrictive (↓FVC) and obstructive/small airway involvement (↓FEF25–75). This mixed pattern was also reported by Klein et al.(7), who concluded that diabetes affects lung mechanics through multiple pathophysiological pathways.

The strong negative correlations observed between glycaemic control, as indicated by HbA1c, and pulmonary function parameters further reinforce the impact of hyperglycaemia on lung health. These findings are in agreement with van den Borst et al.(3) and Yeh et al.(10), who demonstrated an inverse relationship between HbA1c levels and lung function. Proposed mechanisms include non-enzymatic glycation of collagen and elastin in lung tissue, reduced elasticity, oxidative stress, chronic low-grade inflammation, and diabetic microangiopathy affecting the pulmonary capillary network (1,11).

The current investigation also identified a significant negative correlation between duration of diabetes and pulmonary function parameters, indicating progressive deterioration over time. This supports the hypothesis that prolonged exposure to hyperglycaemia results in cumulative

structural damage in lung tissue. Similar observations were reported by Davis et al.(12). Importantly, multiple regression analysis identified HbA1c as the only independent predictor of FEV1%, while age, gender, BMI, and disease duration were not significant after adjustment. This underscores the central role of glycaemic control as a modifiable risk factor in pulmonary dysfunction. Comparable results were reported by Litonjua et al.(13).

In our study, correlation analysis revealed a strong and significant inverse relationship between glycaemic control and pulmonary function parameters. HbA1c was negatively correlated with FEV1 (%), FVC (%), FEF25 (%), and FEF50 (%). These findings indicate that poorer glycaemic control is closely associated with greater impairment of pulmonary function in patients with T2DM. This agrees with Nao Sonoda et al.(14), who found that HbA1c ≥8.0% was associated with a 2.4-fold increased risk of restrictive lung impairment compared with HbA1c <6.9%, highlighting the importance of good glycaemic control.

Debasish Barik and Sharath Babu(15) also demonstrated a significant negative correlation between HbA1c and pulmonary function parameters in patients with T2DM. Poor glycaemic control was associated with reduced FVC, FEV1, and PEFR, predominantly showing a restrictive pattern of dysfunction. Their findings support the concept that the lung may be an important target organ in diabetic complications.

Our results are in strong agreement with Mittal et al.(16), who demonstrated a significant inverse correlation between disease duration and spirometric parameters, indicating that longer diabetes duration is associated with progressive pulmonary impairment. Likewise, in our study, longer diabetes duration was significantly associated with lower pulmonary function values, including FEV1, FVC, and FEF25%. These comparable findings reinforce the concept that the lung may represent another target organ affected by chronic diabetic microangiopathy and glycation-related connective tissue changes.

Notably, our study extends the findings of Mittal et al. by demonstrating that HbA1c was the strongest independent predictor of pulmonary dysfunction on multivariate regression analysis, suggesting that glycaemic control may have an even greater influence than disease duration alone. Therefore, while both studies confirm the cumulative harmful effect of prolonged diabetes on lung function, our results additionally emphasize the importance of strict metabolic control in preventing respiratory decline.

Our findings are also consistent with Maan et al.(17), who investigated the effect of HbA1c and disease duration on lung function in patients with T2DM. Similar to our results, they demonstrated that poor glycaemic control was significantly associated with impaired pulmonary function and concluded that high HbA1c had a more deleterious effect than disease duration alone. In our study, the uncontrolled diabetic group showed significantly lower FEV1, FVC, FEF25, FEF50, and FEF25–75 compared with both controlled diabetics and healthy controls. Although Maan et al. reported no significant overall correlation between disease duration and lung function, our study demonstrated a significant negative association between longer diabetes duration and most spirometric parameters. Nevertheless, both studies consistently highlight poor glycaemic control as a major determinant of pulmonary dysfunction in T2DM.

Conclusion:

Uncontrolled T2DM is associated with significant impairment in pulmonary function. Poor glycaemic control and longer disease duration are strongly linked to reduced lung function, with HbA1c emerging as the most important independent predictor. These findings highlight the position of optimal glycaemic control and support incorporating pulmonary function assessment into routine diabetes care.

In spite of its strengths, this study has some limitations. The generalizability of the findings may be restricted by the relatively modest sample size. Additionally, the cross-sectional design

renders it impossible to establish a causal relationship between hyperglycaemia and pulmonary dysfunction. Future longitudinal studies with larger populations and adding lung volumes and diffusion lung capacity are recommended to confirm these findings.

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