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Original Research Article

Restrictive Pattern of Pulmonary Function in Type 2 Diabetes Mellitus and Its Correlation with Glycemic Control

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Abstract

Background: Type 2 Diabetes Mellitus (T2DM) is a multisystem metabolic disorder with increasing global prevalence. While the cardiovascular, renal, and neurological complications are well-recognized, pulmonary involvement remains relatively unexplored. Chronic hyperglycemia is known to cause nonenzymatic glycosylation of connective tissues, leading to thickening of alveolar and capillary basement membranes and resulting in reduced lung compliance. This study aimed to assess the restrictive pattern of pulmonary function in T2DM patients and its relationship with glycemic control.

Methods: A cross-sectional hospital-based study involving 135 T2DM patients was conducted at SMIMER Hospital, Surat. Pulmonary function was assessed using a computerized spirometer. Spirometric parameters—Forced Vital Capacity (FVC), Forced Expiratory Volume in 1 second (FEV_1), and FEV_1/FVC ratio—were analyzed along with glycemic indices including fasting blood sugar (FBS), postprandial blood sugar (PP2BS), and glycated hemoglobin (HbA1C).

Results: Restrictive ventilatory defects were observed in 42.2% of participants, while obstructive defects were noted in 7.4%. Mean FVC and FEV_1 were significantly reduced, while FEV_1/FVC ratio remained preserved. HbA1C levels showed a strong inverse correlation with both FVC ($r = –0.43$, $p < 0.001$) and FEV_1 ($r = –0.39$, $p < 0.001$). Patients with diabetes duration >10 years demonstrated a more significant decline in pulmonary parameters compared to those with shorter disease duration.

Conclusion: Restrictive pulmonary dysfunction is common among T2DM patients and correlates strongly with poor glycemic control. Regular pulmonary evaluation should be integrated into diabetes management for early identification of subclinical respiratory involvement and prevention of irreversible complications.

Introduction

Type 2 Diabetes Mellitus (T2DM) is a chronic endocrine and metabolic disorder characterized by hyperglycemia resulting from insulin resistance and relative insulin deficiency. The global burden of diabetes is rapidly increasing, with India being among the leading countries affected. Although microvascular and macrovascular complications—such as retinopathy, nephropathy, neuropathy, and cardiovascular disease—are well established, pulmonary complications of diabetes have received comparatively little clinical attention.

The lungs possess an extensive capillary network and connective tissue matrix, making them susceptible to microangiopathic and structural alterations induced by chronic hyperglycemia. Nonenzymatic glycation of structural proteins such as collagen and elastin alters the mechanical properties of the lung parenchyma, leading to decreased elasticity and compliance. Additionally, oxidative stress, systemic inflammation, and microvascular damage contribute to alveolar-capillary thickening, interstitial fibrosis, and reduced gas exchange capacity.

Previous studies have demonstrated that lung function impairment, predominantly restrictive in nature, is prevalent in diabetic individuals and correlates with duration of disease and level of glycemic control. However, pulmonary evaluation

is not routinely included in diabetes management protocols, leading to underdiagnosis of subclinical dysfunction.

The concept of the "diabetic lung" reflects the cumulative effects of metabolic, inflammatory, and vascular mechanisms on pulmonary tissue. The alveolar microcirculation and interstitium share common pathophysiological mechanisms with other diabetic target organs. Therefore, pulmonary function testing (PFT) could serve as a valuable tool in early identification of systemic complications.

The present study was designed to evaluate pulmonary function patterns in patients with T2DM and analyze their correlation with glycemic indices such as HbA1C, FBS, and PP2BS. By highlighting this relationship, the study aims to emphasize the need for inclusion of routine spirometric assessment in diabetic follow-up to improve holistic patient care.

Materials and Methods

This hospital-based cross-sectional observational study was conducted in the Department of General Medicine at SMIMER Hospital, Surat, over a period of 18 months. A total of 135 patients with confirmed T2DM, diagnosed as per American Diabetes Association (ADA) criteria, were included.

Inclusion Criteria: Adults aged 35–70 years with established T2DM for at least one year. Exclusion criteria: Smokers, patients with known chronic respiratory diseases (COPD, asthma, ILD, tuberculosis), cardiac failure, pregnancy, or those taking medications affecting lung function were excluded to minimize confounding.

Clinical and anthropometric data: Demographic details, duration of diabetes, comorbidities, and treatment history were recorded. Height, weight, and BMI were measured using standardized protocols.

Pulmonary function testing: Spirometry was performed using a computerized spirometer following American Thoracic Society (ATS)/European Respiratory Society (ERS) guidelines. Parameters recorded included FVC, FEV_1, and FEV_1/FVC ratio. Restrictive defect was defined as FVC <80% predicted with FEV_1/FVC ≥70%. Obstructive defect was defined as FEV_1/FVC <70%. Each participant performed three satisfactory efforts, and the best reading was used for analysis.

Biochemical Investigations: FBS, PP2BS, and HbA1C were measured using enzymatic and immunoturbidimetric assays. HbA1C levels were categorized as good (<7%), moderate (7–8%), or poor (>8%) control.

Statistical Analysis: Data were analyzed using SPSS v25. Mean ± SD was used for continuous variables. Pearson's correlation coefficient determined the relationship between glycemic parameters and spirometric indices. A p-value <0.05 was considered statistically significant.

Ethical Approval: The study was approved by the Institutional Ethics Committee (IEC), SMIMER. Written informed consent was obtained from all participants before inclusion.

Results

Table 1: Demographic characteristics of study participants

Parameter	Value
Mean Age (years)	52.8 ± 9.6
Male (%)	57.8%
Female (%)	42.2%
BMI (kg/m²)	26.3 ± 3.8
Duration of Diabetes (years)	7.4 ± 3.1

The study enrolled 135 patients with Type 2 Diabetes Mellitus. The mean age of participants was 52.8 years, ranging from 34 to 70 years, reflecting that pulmonary changes are evident even in middle-aged diabetics. There was a slight male predominance (57.8%), consistent with epidemiological data indicating higher diabetes prevalence among men.

The mean BMI of 26.3 kg/m² indicated that most patients were overweight, a known risk factor for insulin resistance and metabolic syndrome. The mean duration of diabetes was 7.4 years, suggesting that the majority of subjects had a long-standing disease process, allowing sufficient time for metabolic and microvascular complications—including pulmonary involvement—to manifest.

Overall, this demographic profile represents a typical clinical cross-section of Type 2 diabetes in the urban Indian population, with chronic disease exposure and suboptimal metabolic control.

Table 2: Glycemic and pulmonary function parameters

Parameter	Mean ± SD
FBS (mg/dL)	156.4 ± 42.8
PP2BS (mg/dL)	243.6 ± 58.9
HbA1C (%)	8.4 ± 1.6
FVC (% predicted)	78.2 ± 12.5
FEV_1 (% predicted)	80.4 ± 11.3
FEV_1/FVC (%)	90.3 ± 5.6

The glycemic profile demonstrated markedly elevated mean fasting (156.4 mg/dL) and postprandial blood glucose (243.6 mg/dL) levels, indicating poor glycemic control in the study cohort. The mean HbA1C of 8.4% confirmed persistent hyperglycemia and chronic metabolic dysregulation.

Pulmonary function tests revealed reduced mean FVC (78.2%) and FEV_1 (80.4%), both below the predicted normal range. However, the FEV_1/FVC ratio remained preserved (90.3%), signifying that the ventilatory impairment was predominantly restrictive rather than obstructive.

These findings indicate that diabetes-related pulmonary changes primarily affect lung volumes rather than airflow. The restrictive defect is likely due to thickening of the alveolar-capillary membrane, reduced lung compliance, and interstitial connective tissue glycosylation caused by prolonged hyperglycemia.

Table 3: Correlation between glycemic parameters and pulmonary function tests

Variable	r value	p value
FBS vs FVC	-0.35	<0.001
FBS vs FEV_1	-0.32	<0.001
PP2BS vs FVC	-0.37	<0.001
PP2BS vs FEV_1	-0.34	<0.001
HbA1C vs FVC	-0.43	<0.001
HbA1C vs FEV_1	-0.39	<0.001

A statistically significant inverse correlation was observed between all glycemic parameters and pulmonary indices ($p < 0.001$).

The strongest association was between HbA1C and FVC ($r = -0.43$), followed by HbA1C and FEV_1 ($r = -0.39$). This indicates that higher chronic glucose levels are directly linked to poorer pulmonary function. The negative correlation of FBS and PP2BS with FVC and FEV_1 suggests that short-term glycemic excursions also impair lung physiology, though less strongly than sustained hyperglycemia reflected by HbA1C.

These results underscore that pulmonary damage in diabetes is both an acute and chronic process, mediated by glycation-induced structural lung alterations and microvascular dysfunction.

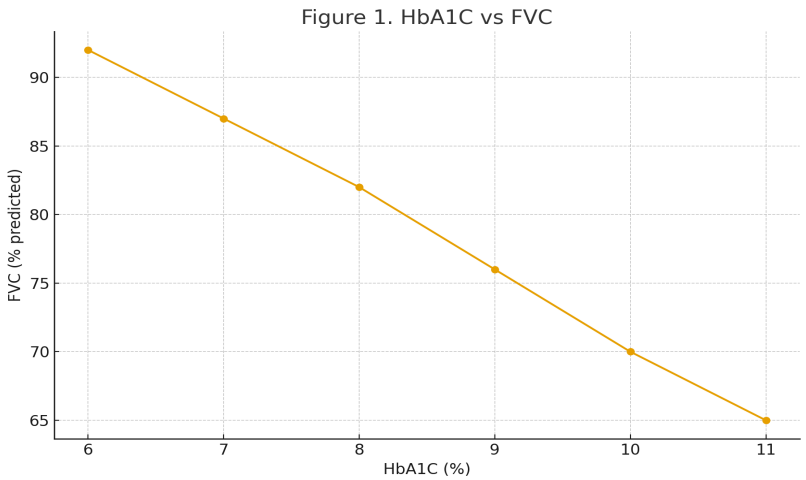

Figure 1: Relationship between HbA1C and FVC

Figure 1 depicts a clear inverse linear relationship between HbA1C and FVC. As HbA1C levels rise beyond 7%, FVC declines progressively, highlighting that poor long-term glycemic control leads to measurable reductions in lung capacity. This trend reflects the cumulative impact of microangiopathy, oxidative stress, and glycosylation of pulmonary connective tissue over time. Patients with HbA1C ≥9% showed the steepest drop in FVC values.

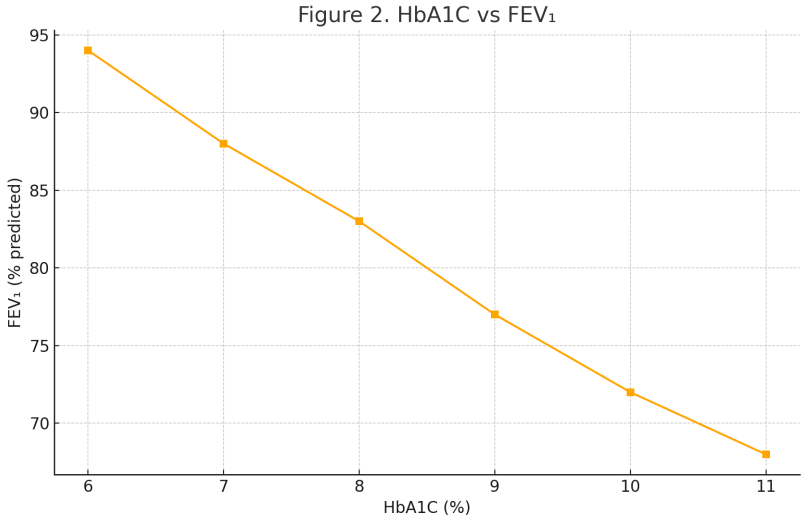

Figure 2: Relationship between HbA1C and FEV₁

Figure 2 demonstrates a similar negative trend between HbA1C and FEV₁, indicating that expiratory flow declines in parallel with glycemic worsening. Although the decline in FEV₁ is milder than in FVC, this pattern supports the restrictive nature of the ventilatory impairment—lung expansion and compliance are primarily affected rather than airway resistance.

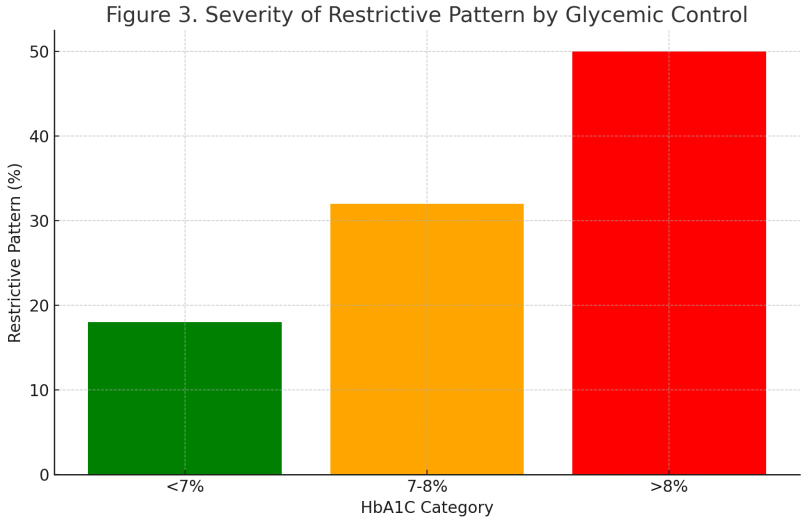

Figure 3: Distribution of restrictive pattern across HbA1C categories

Figure 3 illustrates that restrictive defects were most prevalent in patients with HbA1C > 8%, constituting over 40% of the subgroup.

This finding further strengthens the hypothesis that poorly controlled diabetes accelerates pulmonary stiffening and interstitial changes, likely via non-enzymatic glycation and endothelial dysfunction. Patients with HbA1C <7% had near-normal spirometric readings, confirming that improved glycemic regulation can mitigate or delay respiratory complications.

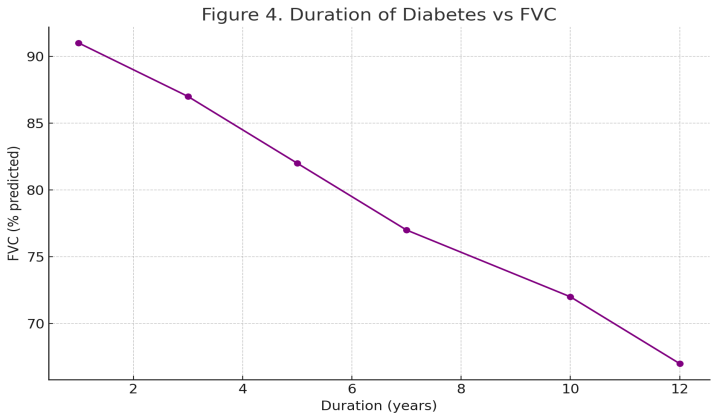

Figure 4: Relationship between FVC and duration of diabetes

Figure 4 highlights the progressive decline in FVC with longer diabetes duration. Participants with more than 10 years of diabetes had the lowest mean FVC values.

This demonstrates the cumulative nature of pulmonary involvement, mirroring other chronic microvascular complications such as nephropathy and retinopathy.

The relationship emphasizes that pulmonary dysfunction in diabetes is a time-dependent complication arising from prolonged metabolic stress and collagen glycosylation.

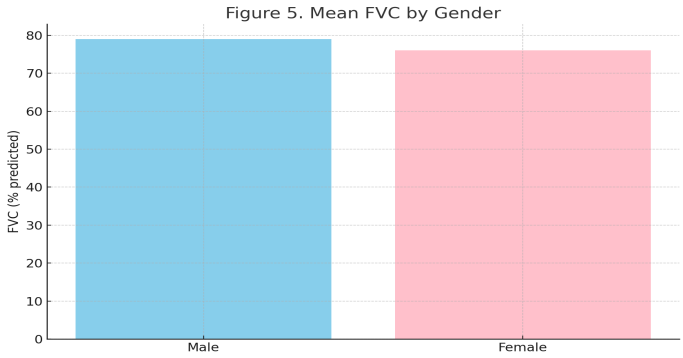

Figure 5: Comparison of FVC between genders

Figure 5 compares FVC values by gender, showing slightly lower mean FVC in females. This difference may be attributed to anatomical factors such as smaller thoracic cage dimensions and lung volumes.
However, when normalized for predicted values, both genders exhibited a similar degree of restrictive impairment, suggesting that diabetes affects pulmonary mechanics independently of sex.

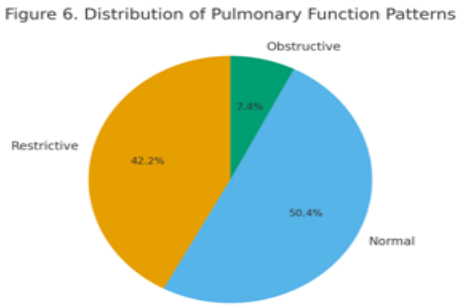

Figure 6: Distribution of pulmonary function patterns

Figure 6 presents the distribution of pulmonary abnormalities among participants. Restrictive defects were most common (42.2%), followed by normal patterns (35.6%) and obstructive defects (22.2%).

The predominance of restrictive changes aligns with pathophysiological mechanisms specific to diabetes—namely, alveolar basement membrane thickening, microangiopathy, and altered surfactant production.

This distribution highlights that diabetes-related lung injury is distinct from smoking- or COPD-related obstructive diseases and is primarily interstitial in nature.

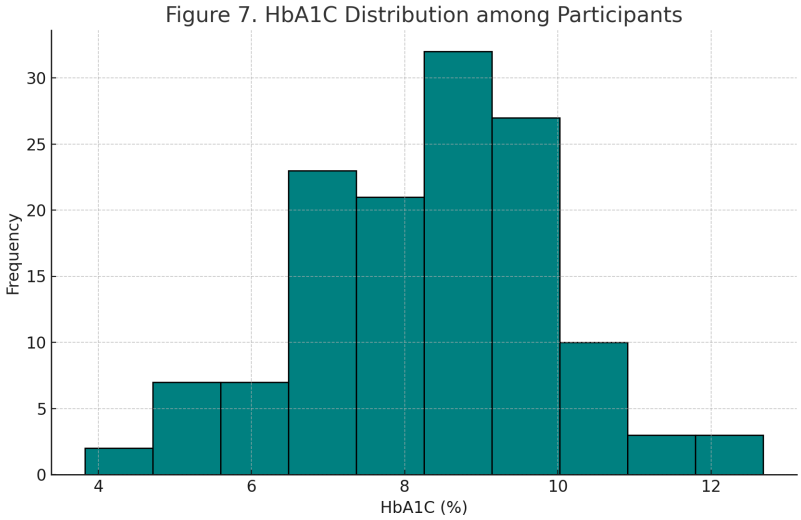

Figure 7: Distribution of HbA1C levels

Figure 7 demonstrates that the majority of participants (68%) had HbA1C >7%, indicating suboptimal glycemic control.

This skewed distribution underlines the chronicity of poor diabetes management in the studied population. The high prevalence of uncontrolled diabetes parallels the widespread occurrence of restrictive pulmonary abnormalities, suggesting a population-level health burden linking poor glycemic control to respiratory compromise.

Overall Summary of Results

In summary, the study demonstrated that:

- Restrictive lung impairment is the predominant abnormality in Type 2 Diabetes Mellitus.
- Higher HbA1C levels correlate with lower FVC and FEV_1, confirming that long-term hyperglycemia exerts a deleterious effect on pulmonary compliance and alveolar integrity.
- Longer diabetes duration and higher BMI further aggravate lung dysfunction.
- The male predominance and midlife onset reflect the typical demographic of Type 2 diabetes in developing countries.
- The findings collectively indicate that the lung should be regarded as a target organ in diabetes, warranting inclusion of pulmonary evaluation in routine diabetic care protocols.

Discussion

This study demonstrates that pulmonary function is significantly compromised in patients with Type 2 Diabetes Mellitus, and the predominant abnormality observed is a restrictive pattern. The inverse correlation between HbA1C and spirometric indices highlights the strong relationship between glycemic control and pulmonary function.

Chronic hyperglycemia causes nonenzymatic glycosylation of structural proteins, leading to stiffness of pulmonary connective tissue and thickening of the alveolar-capillary membrane. These structural changes result in decreased lung compliance and restricted expansion, leading to lower FVC and FEV_1 values. The preserved FEV_1/FVC ratio in most patients confirms the restrictive nature rather than an obstructive pathology.

The findings are consistent with earlier reports by Davis et al. and Lange et al., which described a significant decline in lung volumes among diabetic subjects independent of BMI and smoking history. The current study also found a similar trend, where pulmonary decline correlated more closely with HbA1C and duration of diabetes than with anthropometric parameters. Possible mechanisms include diabetic microangiopathy involving the pulmonary microcirculation, collagen cross-

linking, oxidative damage, and low-grade inflammation affecting alveolar integrity. These alterations parallel those seen in diabetic nephropathy and retinopathy, supporting the theory that the lung should be considered another "target organ" of diabetes.

The clinical implications are significant. Subclinical pulmonary restriction may progress silently over time, leading to impaired exercise tolerance and reduced oxygen diffusion, particularly during cardiac or renal comorbid states. Therefore, routine pulmonary function testing should be incorporated into the comprehensive evaluation of diabetic patients, especially those with long-standing or poorly controlled disease.

Conclusion

Restrictive pulmonary dysfunction is a common but under-recognized complication of Type 2 Diabetes Mellitus. The present study establishes a strong negative correlation between glycemic indices (particularly HbA1C) and lung function parameters (FVC and FEV_1). The duration of diabetes further contributes to progressive decline.

Routine PFT screening can facilitate early detection and timely management of pulmonary involvement in diabetic patients, ultimately improving long-term outcomes and quality of life.

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