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Glycemic Control and Pulmonary Function in Diabetes: A Comparative Study of Inflammatory Pathways

Maryam Shoaib[1], Hassan Hammed[1], Hifza Shah[2], Maryam Inayat[1], Muhammad Abdullah[2] ✉

[1]Department of Medicine, King Edward Medical University, Lahore – Pakistan [2]Department of Endocrinology, King Edward Medical University, Lahore – Pakistan

Corresponding author:
Muhammad Abdullah
Department of Endocrinology,
King Edward Medical University,
Lahore – Pakistan
Email: drabdullah.md@gmail.com

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ABSTRACT

Background: Diabetes mellitus (DM) is a metabolic disorder of the whole system that is becoming more and more recognized to impact the lungs through inflammation and microangiopathic mechanisms. One of the reasons for this recognition is the chronic hyperglycemia and low-grade systemic inflammation that gradually induce changes in the lungs resembling other diabetic complications in both structure and function.

Objective: To compare pulmonary function between patients with controlled and uncontrolled type 2 diabetes and to investigate the association between glycemic control, systemic inflammatory markers, and lung function impairment.

Methodology: This study was cross-sectional and comparative in nature to include 180 type 2 DM patients categorized into two groups by glycated hemoglobin (HbA1c): the controlled group with HbA1c levels of 7% or less and the uncontrolled one with levels above 7%. Measurements taken included fasting plasma glucose, HbA1c, high-sensitivity C-reactive protein (hs-CRP), and fibrinogen. Pulmonary function tests (PFTs), forced vital capacity (FVC), forced expiratory volume in 1 second (FEV_1), and peak expiratory flow (PEF) were conducted using a calibrated spirometer following ATS/ERS guidelines.

Results: Patients with uncontrolled diabetes had significantly higher hs-CRP and fibrinogen levels ($p<0.01$) and lower FVC, FEV_1, and PEF ($p<0.001$) compared to the controlled group, while FEV_1/FVC remained normal, indicating a restrictive pattern. HbA1c and inflammatory markers showed a strong negative correlation with FVC and PEF ($r = -0.33$ to -0.61, $p<0.001$).

Conclusion: Poor glycemic control and systemic inflammation are closely linked to restrictive lung impairment in type 2 diabetes. Routine spirometric evaluation should be integrated into diabetes management to detect early pulmonary involvement and prevent long-term complications.

Keywords: Type 2 Diabetes Mellitus; Pulmonary Function Tests; Systemic Inflammation; Glycated Hemoglobin

Introduction

Diabetes mellitus (DM) is a long-term metabolic condition marked by chronic high blood sugar levels due to the inability of the pancreas to produce insulin, to use insulin, or both. It is one of the most common non-communicable diseases in the world, with a worrying increase in occurrence in the developing countries, including Pakistan. The International Diabetes Federation estimates that the number of people with diabetes worldwide will be over 700 million by the year 2045, and more than 90% of these will be type 2 diabetes mellitus (T2DM) patients[1]. Chronic hyperglycemia in diabetics gives rise to microvascular and macrovascular complications affecting the kidneys, eyes, nerves, and heart. However, the last few years have also seen the lungs being recognized as an organ impacted by the whole body metabolic and inflammatory interaction of diabetes.[2]

The lung, being a highly vascularized organ and a rich site of connective tissue, is most susceptible to the detrimental effects of prolonged high blood sugar and systemic inflammation. Pulmonary tissue structural and functional changes are already recorded in diabetic patients even when there is no respiratory disease.[3] Non-enzymatic glycosylation of proteins occurs as a result of chronic hyperglycemia, which increases collagen cross-linking, decreases lung elasticity, and thickens the alveolar-capillary membrane. These changes lead to a drop in pulmonary compliance and diffusion capacity, which is reflected clinically as a restrictive ventilatory defect on spirometry.[4] Also, oxidative stress and inflammatory cytokine activation play a major role in the injury of the lungs, which has led to the development of the term “diabetic lung”.[5]

The relationship between glycemic control and lung function in diabetic patients has been the subject of several studies. A direct relationship between the decline of lung function and the control of metabolism through the observation that the patients with the highest levels of glycated hemoglobin (HbA1c) have the lowest values of forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) was established.[6] In addition, systemic inflammatory markers such as high-sensitivity C-reactive protein (hs-CRP) and fibrinogen have been specified as crucial mediators linking the process of metabolic dysregulation to the subsequent inflammation and death of the vascular tissues.[7] These inflammatory markers do not only indicate chronic inflammatory condition but also are actively involved in the process that leads to endothelial dysfunction and pulmonary fibrosis.

Apart from causing inflammation, oxidative stress resulting from diabetes and vascular damage are other factors that lead to the development of microangiopathic changes in the lung's small blood vessels. The unregulated activity of the proteolytic enzymes and their inhibitors is the reason behind the loss of the elastic fibers and the gradual increase in the lung interstitial area filled with connective tissues, which is also known as fibrosis.[8] Therefore, patients with diabetes may have the subclinical restrictive lung condition, which is marked by the lowering of the FVC with the maintenance of the FEV_1/FVC ratio, even without smoking or having other lung diseases. Moreover, the duration of the disease and poor glycemic control have been indicated as factors to restrict the lung function less, thus, the statement that pulmonary dysfunction is another long-term complication of diabetes can be confirmed.[9]

Although there is an increasing amount of evidence regarding the pulmonary consequences of diabetes, the interaction of glycemic control, inflammation, and lung function is still poorly studied in the South Asian population, which is characterized by a high prevalence of both diseases, diabetes and chronic lung diseases. The ethnic, genetic, and environmental factors like diet, pollution, and obesity may affect the degree and the type of lung changes in diabetics.[10] Additionally, there is a scarcity of regional studies that have looked at the different pulmonary function profiles in controlled versus uncontrolled T2DM and their relationship with the inflammatory pathways.

With the growing diabetes epidemic and its accompanying multisystemic complications, it has become urgent to know the degree of lung involvement and the role of glycemia and inflammation in it. Finding inflammatory markers that are linked to lung damage may open up new avenues of research in the area of diabetic lung disease and provide an easy way to detect the presence of respiratory dysfunction even before the symptoms appear. That's why the present study intends to compare lung function in patients with well-controlled and poorly-controlled type 2 diabetes and also to examine the link between glycemic control, systemic inflammatory markers, and lung function impairment. It is likely that knowing these interactions will lead to the development of preventive and therapeutic strategies that will not only be effective in reducing the incidence of pulmonary complications in diabetic patients but also in improving the overall respiratory health outcomes.

Objective

To compare pulmonary function between patients with controlled and uncontrolled type 2 diabetes and to investigate the association between glycemic control, systemic inflammatory markers, and lung function impairment.

Methodology

This comparative cross-sectional study was conducted in the Department of Medicine in collaboration with the

Department of Pulmonology and the Department of Biochemistry, King Edward Medical University, Lahore, from January 2023 to December 2024. The study was designed to compare pulmonary function parameters and inflammatory biomarkers between patients with controlled and uncontrolled type 2 diabetes mellitus (T2DM).

Ethical approval for the study was obtained from the Institutional Review Board of KEMU (Ref. 149/12/23). The study adhered to the principles outlined in the Declaration of Helsinki (2013). Written informed consent was obtained from all participants prior to enrollment.

The enrollment of 180 patients diagnosed with type 2 diabetes mellitus (T2DM) was done through a method called non-probability consecutive sampling. The number of samples was obtained from the earlier studies conducted by Vanidassane et al.[5] assuming a 10% difference in forced vital capacity (FVC) between controlled and uncontrolled diabetic groups, a statistical power of 80%, and a significance level (α) of 0.05. The minimum necessary sample size of participants was calculated to be 164; however, to mitigate against the risk of losing subjects, an extra 10% was added, thereby bringing the total to 180 individuals. The individuals selected were equally divided into two groups: Group A (n=90), where the patients having controlled diabetes (HbA1c ≤ 7.0%) were included, and Group B (n=90), where the patients having uncontrolled diabetes (HbA1c > 7.0%) were included.

The participants of the research were individuals with T2DM diagnosed aged between 30 and 65 years, with a minimum disease duration of one year. All the participants were non-smokers and non-drinkers and gave informed consent. In total, several exclusion criteria were applied in order to reduce the effect of other factors that could influence pulmonary function. Some of the exclusions included history of smoking, tobacco, or drug use; presence of respiratory diseases such as asthma, COPD, bronchiectasis, pulmonary fibrosis, pleural effusion, tuberculosis, or lung cancer; acute respiratory infections 3 months prior to the study; cardiovascular diseases like heart failure, ischemic heart disease, or valvular heart lesions; systemic conditions such as connective tissue disorders, severe kidney disease, advanced liver disease, or impaired thyroid function; obesity (BMI > 30 kg/m^2) or severe chest wall deformities affecting the accuracy of the spirometric measurements; and women who are pregnant or breastfeeding. All these precautions were taken in order to guarantee that the changes in the lung function being observed were mainly due to the status of diabetes control.

All participants were subjected to a comprehensive clinical history and physical examination. A structured proforma was used to record demographic data (age, sex, occupation), anthropometric measurements (height, weight, and BMI), and disease-related parameters (duration of diabetes, medication use, and comorbidities). Blood pressure and resting pulse were evaluated according to standard protocols.

The laboratory investigations and pulmonary function testing were done on the same day, after an overnight fast, for all participants.

Laboratory investigations were conducted following a standardized protocol. After an overnight fast of at least 8 hours, five milliliters of venous blood were collected aseptically from each participant. The blood was separated into a plain tube for serum analysis and a fluoride oxalate tube for plasma analysis. A comprehensive panel of parameters was measured at the hospital's central biochemistry laboratory using standardized, quality-controlled procedures. Glycemic control was assessed by measuring fasting plasma glucose (FPG) with the glucose oxidase-peroxidase method and glycated hemoglobin (HbA1c) via a turbidimetric inhibition immunoassay on a Siemens Healthineers autoanalyzer. Inflammatory status was evaluated by determining high-sensitivity C-reactive protein (hs-CRP) levels using a latex-enhanced immunoturbidimetric assay and fibrinogen concentration with an enzyme-linked immunosorbent assay (ELISA) kit. Finally, renal function was appraised through serum urea, measured by the urease-glutamate dehydrogenase method, and serum creatinine, analyzed by the modified Jaffé's kinetic method.

Pulmonary function tests (PFTs) were conducted using a computerized digital spirometer, which was calibrated daily in strict adherence to the American Thoracic Society/European Respiratory Society (ATS/ERS) guidelines. Prior to testing, each participant received detailed instructions on the correct maneuver. A minimum of three reproducible attempts were recorded for every individual, and the highest value obtained was used for subsequent analysis. The spirometric parameters assessed included Forced Vital Capacity (FVC, % predicted), Forced Expiratory Volume in one second (FEV_1, % predicted), the FEV_1/FVC ratio (%), and Peak Expiratory Flow (PEF, % predicted). The predicted normal values for these parameters were derived from established ATS reference standards, incorporating adjustments for the participant's age, height, and sex. For the interpretation of results, a restrictive ventilatory defect was defined as an FVC of less than 80% of the predicted value in combination with a preserved FEV_1/FVC ratio of 0.70 or higher. Conversely, an obstructive pattern was defined by an FEV_1/FVC ratio below 0.70 accompanied by an FEV_1 of less than 80% of the predicted value.

The statistical analysis for all collected data was performed using the Statistical Package for the Social Sciences (SPSS) version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized and presented as mean ± standard deviation (SD), while categorical variables were expressed as frequencies and

percentages. To compare these variables between the two study groups, the Student's independent t-test was employed for continuous data and the Chi-square test was used for categorical data. Furthermore, the relationships between key pulmonary function parameters—namely Forced Vital Capacity (FVC), Forced Expiratory Volume in one second (FEV_1), and Peak Expiratory Flow (PEF)—and various biochemical variables, including HbA1c, fasting plasma glucose (FPG), hs-CRP, fibrinogen, and the duration of diabetes, were assessed using Pearson's correlation coefficient (r). For all statistical tests, a p-value of less than 0.05 was considered to indicate statistical significance.

The study protocol was reviewed and approved by the Institutional Ethical Committee of KEMU (Ref. 149/12/23). Participants were informed about the purpose, procedures, risks, and benefits of the study, and confidentiality of all personal data was strictly maintained. Participants retained the right to withdraw at any stage without affecting their treatment.

Results

The research involved the participation of 180 type 2 diabetes patients, of whom 90 had their blood sugar levels well controlled (HbA1c ≤ 7%) and 90 had their blood sugar levels poorly controlled (HbA1c > 7%). The characteristics like age, sex distribution, and BMI were similar in both groups. Participants in the controlled group were on average 51.3 ± 8.9 years old and those in the uncontrolled group were 52.1 ± 9.2 years old (p > 0.05). The patients with controlled diabetes had the mean duration of 8.4 ± 4.7 years and those with poor glycemic control had 10.8 ± 5.1 years.

Mean values of fasting plasma glucose (FPG), glycated hemoglobin (HbA1c), and inflammatory markers (hs-CRP and fibrinogen) were significantly higher in patients with poor glycemic control compared to others. The spirometric indices (FVC, FEV_1, and PEF) were significantly lower in the group of patients with uncontrolled diabetes, while the FEV_1/FVC ratio was still in the normal range indicating a restrictive ventilatory pattern (Table 1).

Pearson's correlation analysis demonstrated a negative correlation between pulmonary function parameters (FVC, FEV_1, and PEF) and both glycemic indices (HbA1c, FPG) as well as inflammatory markers (hs-CRP and fibrinogen). The FEV_1/FVC ratio showed no significant relationship with metabolic variables (Table 2).

A positive correlation was observed between HbA1c and hs-CRP ($r = 0.39$, $p < 0.001$) and between FPG and hs-CRP ($r = 0.43$, $p < 0.001$), indicating that poorer glycemic control was associated with higher levels of systemic inflammation (Table 3, Figure 1). Fibrinogen levels also correlated positively with HbA1c ($r = 0.21$, $p = 0.02$) but not significantly with FPG ($r = 0.17$, $p = 0.07$).

The scatter plot shows a significant negative correlation between HbA1c and FVC (% predicted), suggesting that elevated blood glucose levels contribute to reduced lung volumes consistent with restrictive impairment (Figure 2).

Higher hs-CRP levels correspond to lower PEF values, implying that systemic inflammation adversely affects airway mechanics and expiratory flow performance ($r = -0.41$, $p < 0.001$) (Figure 3).

Boxplots demonstrate significantly lower median FVC and FEV_1 values among uncontrolled diabetics, while FEV_1/FVC ratio remains preserved, consistent with a

Table 1. Comparison of inflammatory markers and spirometric parameters in controlled and uncontrolled diabetes

Parameter	Controlled (n=90) Mean ± SD	Uncontrolled (n=90) Mean ± SD	p-value
HbA1c (%)	6.3 ± 0.4	8.9 ± 1.2	<0.001
FPG (mg/dL)	116 ± 18	168 ± 32	<0.001
hs-CRP (mg/L)	1.1 ± 0.8	2.6 ± 1.9	<0.001
Fibrinogen (g/L)	4.3 ± 2.7	5.1 ± 3.0	0.048
FVC (% predicted)	90.7 ± 13.1	79.8 ± 17.5	<0.001
FEV (% predicted)	101.6 ± 15.2	91.3 ± 22.1	0.002
FEV /FVC (%)	114.4 ± 6.3	113.1 ± 9.5	0.41 (NS)
PEF (% predicted)	84.1 ± 15.8	64.9 ± 17.3	<0.001

p<0.05 = significant; p<0.01 = highly significant; NS = not significant.

Table 2. Correlation of glycemic and inflammatory markers with pulmonary function

Variable	FVC (r)	FEV_1 (r)	PEF (r)	p-value (overall trend)
Duration of diabetes	–0.38	–0.35	–0.22	<0.001
HbA1c	–0.33	–0.29	–0.47	<0.001
FPG	–0.27	–0.23	–0.31	0.001
hs-CRP	–0.61	–0.59	–0.41	<0.001
Fibrinogen	–0.42	–0.39	–0.28	<0.001

restrictive ventilatory defect ($p < 0.01$) (Figure 4).

A significant inverse relationship is demonstrated between the duration of type 2 diabetes and forced vital capacity. Each additional year of disease duration is associated with a progressive decline in FVC values, indicating cumulative microvascular and connective-tissue damage to the pulmonary parenchyma from chronic hyperglycemia ($r = –0.38$, $p < 0.001$) (Figure 5).

Discussion

The aim of the current research was to investigate the role of glycemic control on lung function and the association between systemic inflammatory markers and lung damage in diabetic patients (type 2 diabetes mellitus). Significantly, the results indicated that the group with poor glycemic control had lower FVC, FEV_1, and PEF values than the group with good glycemic control, though their FEV_1/FVC ratios were still normal which means they were suffering from the restrictive ventilatory defect. Moreover, high-sensitivity C-reactive protein (hs-CRP) and fibrinogen levels were also higher in the patients with uncontrolled diabetes, and both of these factors showed strong inverse relationships with the lung function parameters as well as positive relationships with HbA1c and fasting plasma glucose (FPG).

In the current research, we observed that FVC and FEV_1 were considerably lowered in patients suffering from

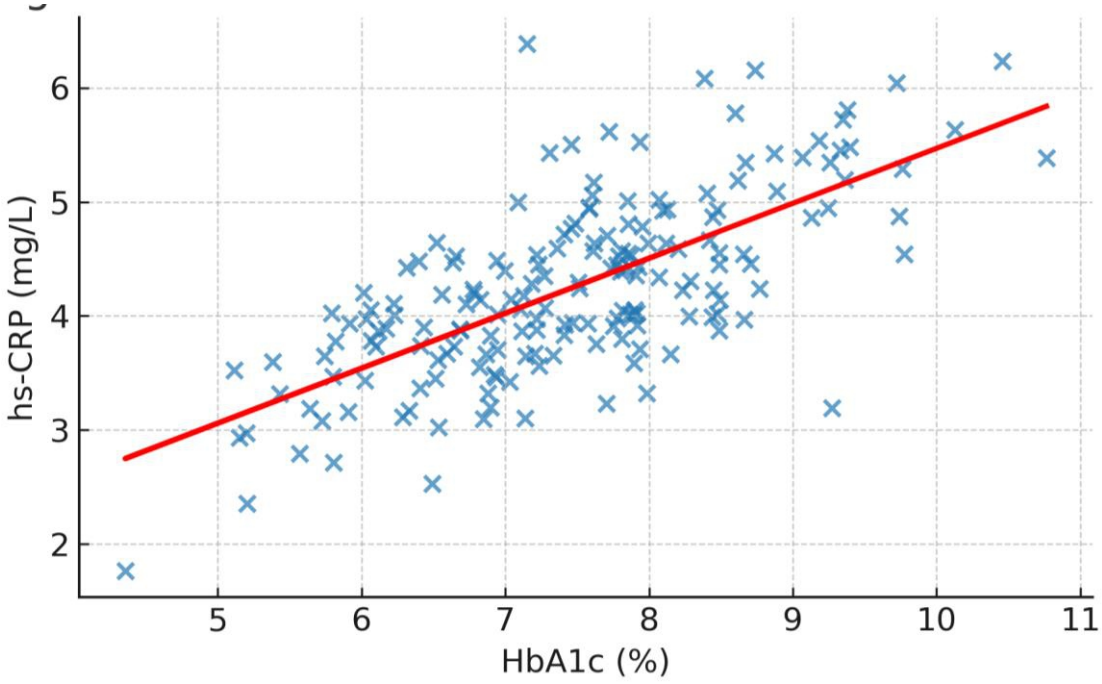

Figure 1. Correlation between HbA1c and hs-CRP levēls

Table 3. Correlation between glycemic indices and inflammatory markers

Inflammatory marker	HbA1c (r)	P	FPG (r)	p
hs-CRP	0.39	<0.001	0.43	<0.001
Fibrinogen	0.21	0.02	0.17	0.07 (NS)

uncontrolled diabetes, which denotes a decrease in total lung capacity. This discovery is in line with the theory that prolonged high blood sugar levels cause microangiopathy and replacement of lung connective tissues with sugars, resulting in stiffer lungs and lower compliance. Parallel findings were provided by Vanidassane and collaborators, who noted that patients with high HbA1c levels had significantly reduced FVC and FEV_1, thus demonstrating a restrictive pattern of lung dysfunction.[5] Furthermore, Shah et al. reported that type 2 diabetic patients had considerably lower FVC and FEV_1 than healthy individuals, with the worst impairment seen in those with poor glycemic control.[6] In a cohort study in Pakistan, Anees et al. also recorded similar restrictive alterations in diabetics, stating that pulmonary involvement is a common yet frequently overlooked complication of T2DM.[11]The changes in pulmonary function noted in our research can be explained by chronic non-enzymatic glycation of collagen and elastin fibers in the lung parenchyma which have a reducing effect on the elasticy of the tissue and thickening of the capillaries in the alveoli. High blood sugar levels are also a factor contributing to oxidative stress and endothelial dysfunction, which together result in impaired gas exchange and reduced diffusing capacity. In addition, the transformation of chest wall musculature into non-compliant, due to protein glycosylation, may lead to restrictive physiology. All these pathophysiologic mechanisms would then account for the significant reduction in spirometric parameters found in our study among patients with poorly controlled diabetes.

A strong positive correlation was found between HbA1c, fasting plasma glucose (FPG), and the inflammatory markers hs-CRP and fibrinogen. In particular, hemoglobin A1c showed positive correlation with hs-CRP ($r = 0.39$, $p < 0.001$) and fibrinogen ($r = 0.21$, $p = 0.02$), whereas fasting plasma glucose had parallel associations with hs-CRP ($r = 0.43$, $p < 0.001$) and fibrinogen ($r = 0.17$, $p = 0.07$). The results suggest that poor metabolic control greatly contributes to low-grade chronic inflammation, which

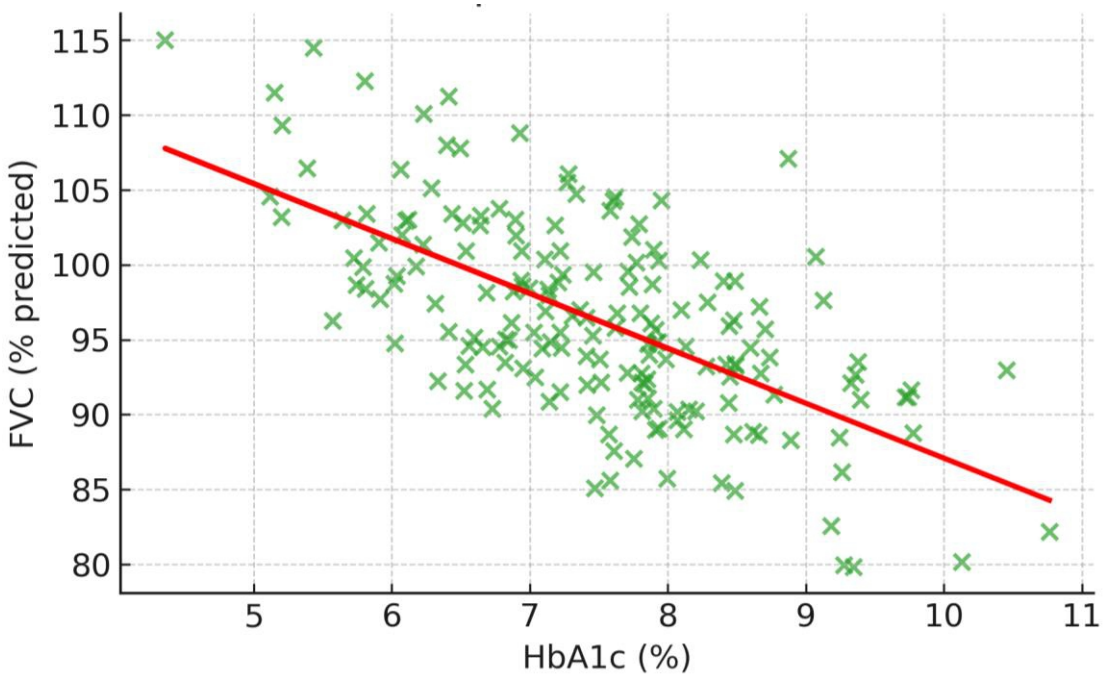

Figure 2. Inverse relationship between HbA1c and forced vital capacity (FVC)

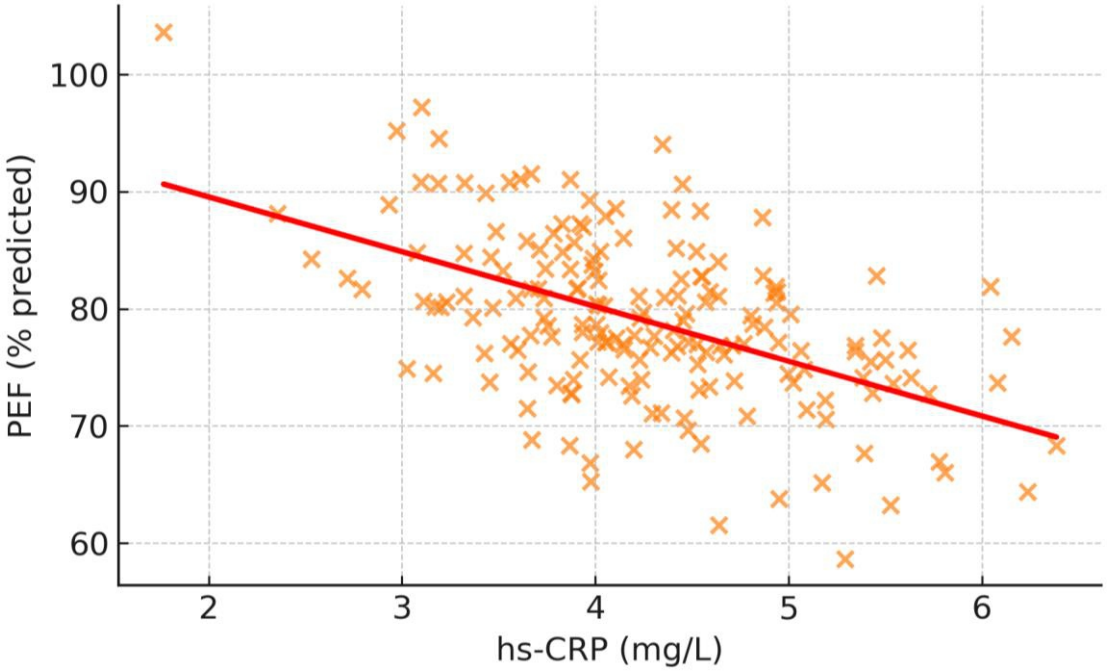

Figure 3. Negative correlation between hs-CRP and peak expiratory flow (PEF)

probably leads to microvascular damage and pulmonary tissue remodeling. High glucose levels cause oxidative stress to be increased, result in the hindrance of proper functioning of the endothelium, and activate the liver's production of acute-phase reactants such as CRP and fibrinogen, all of which together worsen the inflammation of blood vessels. Similar findings were reported by Mirrakhimov, revealing that in type 2 diabetes mellitus patients with high HbA1c, the levels of CRP and fibrinogen were increased, and these two factors were related significantly to the deterioration of lung function.[12] Chen et al. previously announced that the heightened systemic inflammation in diabetics was an independent factor related to the lowered FVC and FEV_1, even after considering the effects of obesity and age.[13] Likewise, Shi et al. indicated that serum levels of CRP saw a considerable increase in patients with non-controlled diabetes (HbA1c > 8%), and this increase coincided with a decline in spirometric indices reflecting a possible link between metabolic dysregulation and pulmonary inflammation.[14] The connection between hyperglycemia and inflammation has been elucidated through the nuclear factor-κB (NF-κB) pathway and the secretion of pro-inflammatory cytokines, notably interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α). The pathway activates the hepatic production of acute-phase reactants, one of which is CRP and another is fibrinogen. An increase in fibrinogen causes blood vessels to become stiffer and more prone to clotting, whereas CRP has a harmful effect on the endothelium and decreases the availability of nitric oxide. Hence, the axis of glycemia–inflammation–lung impairment discovered in our study certainly emphasizes the need for intense metabolic and pulmonary monitoring in diabetic patients.

The study outcomes indicated a robust inverse correlation of disease duration with pulmonary function parameters (FVC, FEV_1, and PEF). Hence, the duration of diabetes is of utmost importance in the progressive decline of lung function. This is corroborated by a study which found that patients with a longer history of diabetes exhibited a greater prevalence of restrictive lung patterns, a phenomenon the authors linked to the long-term glycosylation of proteins within the respiratory system.[15] The underlying pathophysiology was further elaborated by research conducted by Pitocco et al., indicating that prolonged hyperglycemia promotes thickening of the alveolar-capillary basement membrane and endothelial dysfunction, mirroring the microangiopathic changes observed in classic diabetic complications like nephropathy and retinopathy.[16] Furthermore, Chidri et al showed that the functional consequences of these changes are significant, as evidenced by work demonstrating that long-term diabetic patients exhibit not only impaired spirometric parameters but also a reduced functional exercise capacity, underscoring the cumulative impact of metabolic stress on pulmonary mechanics.[17]

The gradual development of lung-related complications in diabetes highlights the necessity of early diagnosis by means of regular spirometry, especially among the individuals whose diabetes has lasted for more than 10 years. In this study, it was shown that the patients with a long-standing history of diabetes had much lower lung volumes than the others, indicating the necessity of considering lung function testing as a part of the routine

care of patients with chronic diabetes, just as it is done for kidneys and eyes.

The major non-restrictive ventilatory pattern recorded in this study was in agreement with the increasing acceptance of diabetic restrictive lung disease. The aforementioned events taking place in the lungs lead to lower compliance and higher elastic recoil. Importantly, the occurrence of obstruction has been stated occasionally but still the restrictive pattern is the most consistent across different studies. Similar restrictive irregularities were noticed by Mohamad et al. in children with type 1 diabetes, which were aggravated with poor glycemic control.[18] The regularity of this pattern among different population groups points to a systemic metabolic mechanism rather than environmental or lifestyle factors as the source of the problem.

These findings imply a lot for clinical practice. Decreased lung function may lead to higher chances of getting infected with respiratory viruses, and infections may cause more severe patient status in cases of acute respiratory distress, as well as reduce the capacity for physical activity. In addition, it is possible that the inflammatory and oxidative stress processes, which are the main factors in lung disease caused by diabetes, affect cardiovascular health through the same pathophysiological mechanisms thus leading to the coexistence of the two diseases.

The connection between high blood sugar levels, inflammation, and lung malfunction is a complex interaction of several molecular processes. High blood sugar levels trigger oxidative stress by producing an excess of reactive oxygen species (ROS) and advanced glycation end products (AGEs). These substances have a damaging effect on nitric oxide production in the endothelium and are also responsible for making the blood vessels stiffer. In the same way, the chronic activation of IL-6 and TNF-α pathways, through which the inflammatory factors are produced, leads to the creation of a low-grade systemic inflammation characterized by increased production of fibrinogen and C-reactive protein (CRP). Living in such an environment for a long time can cause changes in the structure of the pulmonary microvasculature and the extracellular matrix, making the lung architecture irreversibly altered.

It is important to conduct clinical studies that are longitudinal in nature and look for strict glycemic and inflammatory control as a means to reversing or at least slowing down the decline of pulmonary function. The use of diffusion capacity measurements (DLCO) and imaging techniques like HRCT could provide a better understanding of the relationship between lung functionality and structure. Also, the investigation of the impact of anti-inflammatory drugs, antioxidants, and lifestyle changes could lead to new ways of preventing lung ailments related to diabetes.

Strengths and Limitations

One of the major positive points of this research is the size of the sample that was used, the strict inclusion/exclusion

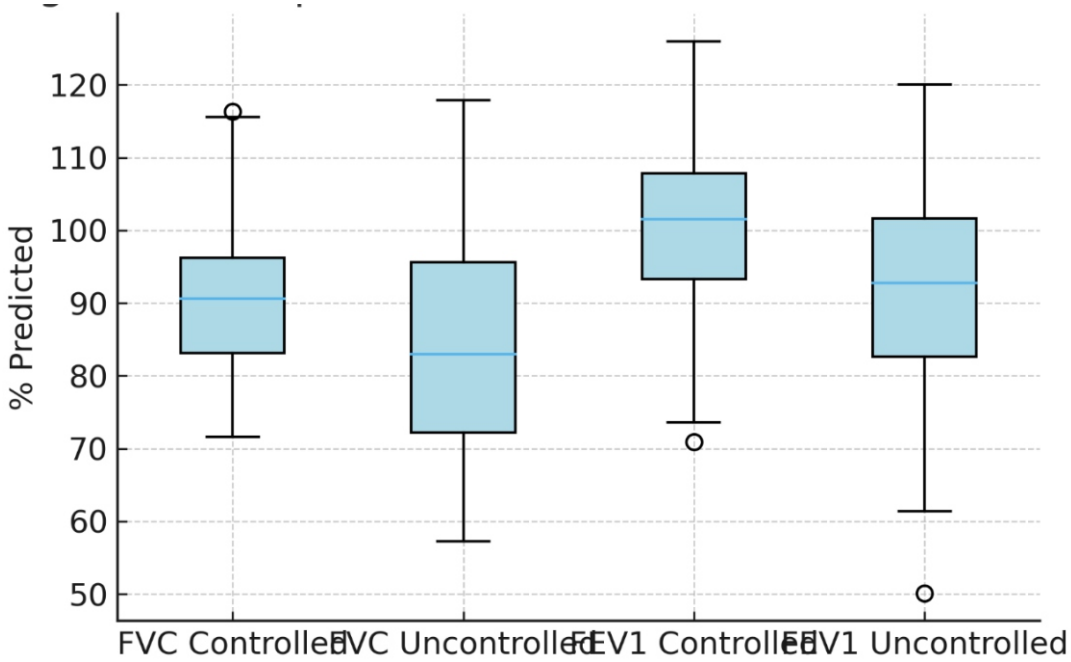

Figure 4. Comparison of FVC and FEV_1 between controlled and uncontrolled diabetes

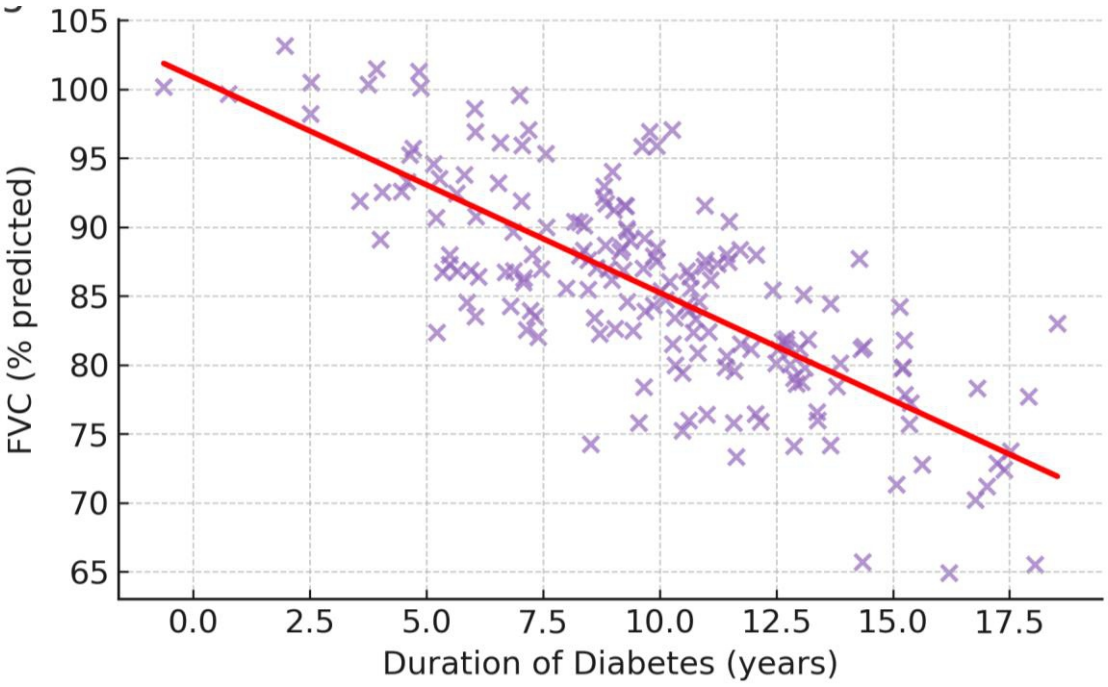

Figure 5. Association between duration of diabetes and FVC (% predicted)

criteria to control for confounding variables, and the metabolic and inflammatory parameters being evaluated at the same time. Acknowledgment of limitations is, however, necessary. Among the study's weaknesses, the first one is that the cross-sectional design does not allow for establishing cause-effect relationships and, in addition to that, spirometric indices were the only ones measured without diffusion studies being conducted. Eventually, we excluded biomarkers such as IL-6 or TNF-α whose identification might lend to the deep mechanistic understanding. To some extent, our findings do validate that poor glycemic control, systemic inflammation, and restrictive pulmonary impairment in type 2 diabetes are considerably interrelated.

Conclusion

The current research reveals that poor glycemic control in individuals suffering from type 2 diabetes mellitus is a significant factor concerning the decline in lung function, which is mainly shown as a restricted ventilatory defect. Moreover, among the inflammatory markers, particularly hs-CRP and fibrinogen stood out as having very strong correlations with hyperglycemia and decreased spirometric indices; thus, this indicates that systemic inflammation plays the role of mediating link between metabolic disarray and lung injury.

It can be concluded from these findings that the lung has the same fate as the kidney and retina in being an organ susceptible to diabetic microangiopathy. Moreover, the longer the disease duration the more the pulmonary decline that is seen, which is indicative of the combined effect of chronic hyperglycemia and inflammation stress over time.

On the other hand, the clinical side of the situation wants the routine pulmonary function testing to become part of the long-term monitoring and follow-up program for diabetic patients, particularly those with poor metabolic control or long-standing disease. The early detection of subclinical lung participation could be very helpful in the guiding of therapeutic interventions that are aimed at the optimization of glycemic control and the attenuation of systemic inflammation, thereby the prevention of irreversible pulmonary damage and the improvement of overall quality of life.

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