E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/16.02.217

Research Article

Prevalence and Predictors of Restrictive Lung Dysfunction among Patients with Type 2 Diabetes Mellitus

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Received: 01.06.26, Revised: 04.07.26, Accepted: 03.08.26

ABSTRACT

Objective: Type 2 Diabetes Mellitus (T2DM) is a systemic disease, and the lungs are a target organ of the disease. This study was aimed to find out the prevalence of Restrictive Lung Dysfunction (RLD) in T2DM patients and to identify its independent clinical and biochemical predictors.

Material and Methods: A cross-sectional observational study of 450 adult patients of T2DM attending a tertiary care endocrinology clinic was conducted. The definition of RLD was a Forced Vital Capacity (FVC) < 80% of the predicted value and normal FEV1/FVC ratios. Multivariate logistic regression analysis was used to determine the independent risk factors for RLD.

Results: RLD was identified in 34.2% (n=154) of the participants. Multivariate analysis revealed that prolonged duration of diabetes (OR = 1.15, 95% CI: 1.08-1.22, p < 0.001), poor glycemic control (HbA1c > 8.0%) (OR = 2.45, 95% CI: 1.55-3.88, p < 0.001), presence of diabetic nephropathy (OR = 2.10, 95% CI: 1.25-3.52, p = 0.004), and increased body mass index (OR = 1.08, 95% CI: 1.02-1.14, p = 0.008) were independent predictors of RLD.

Conclusions: Restrictive lung dysfunctions is common among patients with T2DM. Significant independent risk factors include poor glycemic control, long duration of diabetes, diabetic nephropathy, and obesity. Lung function screening should be routinely performed in T2DM patients, particularly those with long duration of diabetes and microvascular complications as a part of their comprehensive management.

Keywords: Type 2 Diabetes Mellitus, Restrictive Lung Dysfunction, Pulmonary Function Test, Glycemic Control, Diabetic Complications, Spirometry.

INTRODUCTION

Type 2 Diabetes Mellitus (T2DM) is one of the most challenging public health problems of the 21st century, which is characterized by chronic hyperglycemia and severe metabolic derangements.[1] The classic microvascular and macrovascular complications of DM like diabetic retinopathy, nephropathy, neuropathy, coronary artery disease and cerebrovascular accidents (CVA) have traditionally been the primary clinical interest and research focus of T2DM.[2] But in more recent medical literature there has been a growing recognition of the role of the lung in the pathogenesis of diabetes related illness, and the term "diabetic lung disease" has come into use.[3] This paradigm shift is supported by increasing epidemiological and pathophysiological evidence that elevated levels of chronic hyperglycemia and insulin resistance can harm the pulmonary system.[4]

Restrictive Lung Dysfunction (RLD) is the most common pulmonary abnormality observed in T2DM when compared to other abnormalities.[5] Lung volumes are decreased in RLD patients, especially FVC (Forced Vital Capacity), and no significant airflow abnormalities are detected.[6] One of the major mechanism is that of non-enzymatic glycation of lung structural proteins.

In the lung parenchyma, Advanced Glycation End-products (AGEs) are produced because of chronic hyperglycaemia.[7] There is cross-linking of collagen and elastin fibers in the alveolar walls and the pulmonary interstitium due to the action of AGEs. This crosslinking leads to a decreased elastic recoil of the lungs, increased pulmonary stiffness and eventually causes a decrease in total lung capacity and FVC, which are characteristic of restrictive physiology.[8]
Furthermore, the binding of AGEs to their specific receptors (RAGE) on alveolar macrophages and the epithelial cells triggers the production of pro-inflammatory cytokines which lead to local tissue damage and fibrosis.[9] Another major pathway involved in the development of diabetic RLD is pulmonary microangiopathy which has been shown to produce structural protein glycation.[10] Systemic low-grade inflammation and oxidative stress are also features of metabolic syndrome and T2DM, and further add to endothelial damage and a pro-fibrotic environment in lung tissue.[11] The wide variation is attributed to the study populations, diagnostic criteria for a diagnosis of RLD, inclusion of confounding factors in some of the studies, and the differences in length and severity of diabetes in the different study populations examined.[12] Clinicians need to have a high index of suspicion and to routinely consider pulmonary assessment as part of the full-body evaluation of a diabetic patient, which requires a detailed understanding of the prevalence and specific clinical and biochemical risk factors for RLD.[13] Proper early diagnosis of RLD may contribute to more vigorous attempts to optimize glycemic control, weight management treatment, and even pulmonary rehabilitation or beginning specific medications to slow the progression of the disease.[14]

MATERIALS AND METHODS

A cross-sectional observational study was carried out in multiple tertiary care hospitals in the departments of Endocrinology and the Pulmonology. The study period was 1 year from January through December, 2024. A large and diverse patient population with T2DM was available, as was access to standardized and good quality equipment for pulmonary function testing and to trained respiratory technologists at the setting. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for cross-sectional studies were used to develop the study protocol. A consecutive non-probability sampling technique was used to recruit the participants until the required sample size was obtained. Participants included in the study were those with a documented history of Type 2 Diabetes Mellitus for at least 1 year, had satisfied the American Diabetes Association (ADA) criteria for the diagnosis of DM (fasting plasma glucose ≥ 126 mg/dL; 2 hour plasma glucose ≥ 200 mg/dL during an oral glucose tolerance test; HbA1c ≥ 6.5 %), and were willing to provide informed consent with the ability to perform acceptable spirometry maneuvers (standard protocol). Care was taken to exclude some of the potential confounding factors that could affect pulmonary function tests independently. Patients were excluded who had: (1) A history of primary respiratory diseases such as asthma, chronic obstructive pulmonary disease (COPD), interstitial lung disease, pulmonary tuberculosis, bronchiectasis or thoracic surgery, may affect pulmonary congestion and lung volumes; (2) Patient current smoking or smoking history >10 pack-years, may cause pulmonary congestion and restrict lung volumes; (3) Known history of chronic heart failure (NYHA class III–IV), may affect pulmonary congestion and restrict lung volumes; (4) Severe valvular heart disease, ischemic heart disease with reduced ejection fraction, may affect pulmonary congestion and restrict lung volumes; (5) Severe kyphoscoliosis, morbid obesity (BMI>40 kg/m2), neuromuscular disorders (e.g., myasthenia gravis, muscular dystrophy) affecting chest wall and respiratory muscles, may lead to pulmonary congestion and restriction of lung volumes; (6) Pregnancy. The OpenEpi statistical software (Version 3) was used to calculate the sample size. A target sample size of 450 patients was chosen to account for possible data incompleteness and suboptimal spirometry maneuvers, and to enable the use of multivariate subgroup analyses with high statistical power (to which the sample size was inflated by 30%). Dietary, physical activity and alcohol consumption were also recorded. A stadiometer was used to measure the height to the nearest 0.1cm, patient was standing with the head in the Frankfort horizontal plane, barefoot. Weight was measured using a calibrated electronic scale with a 0.1 kg accuracy, after taking off shoes and wearing light clothes. Body Mass Index (BMI) was determined using the formula: kg/m^2 (kg/m^2 = weight in kg/height in m^2). Venous blood was collected from the antecubital vein after a 10-hour overnight

fasting. The glucose oxidase method was used for fasting blood glucose (FBG) assay. Glycated hemoglobin (HbA1c) was measured by high-performance liquid chromatography (HPLC). Assessment of an enzymatic colorimetric automated lipid profile (total cholesterol, triglycerides, high density lipoprotein (HDL) and low-density lipoprotein (LDL) cholesterol) was performed. Serum creatinine and estimated glomerular filtration rate (eGFR) were used to assess renal function based on the CKD-EPI equation. The spot urine albumin-to-creatinine ratio (UACR) was used to assess urinary albumin excretion. The diabetic nephropathy was defined as an eGFR < 60 mL/min/1.73 m² or a UACR ≥ 30 mg/g. Repeatability was considered as the absolute difference between the largest FVC and FEV1 being within 150 mL. Restrictive Lung Dysfunction (RLD) was defined as a reduced FVC (< 80% of predicted or < -1.645 Z score) accompanied by a normal or elevated FEV1/FVC ratio (≥ 0.70 or above the lower limit of normal) which showed that there was no significant airflow obstruction. Flow-volume loop morphology was examined closely, and tests with poor effort and/or poor repeatability were excluded, to distinguish true pulmonary restriction from pseudo-restriction (due to poor effort). The collected data were entered into a secure, password-protected electronic data base and analyzed in the IBM SPSS Statistics for Windows, Version 28.0 (IBM Corp., Armonk, NY, USA). The normality of continuous variables were assessed by Kolmogorov-Smirnov and Shapiro-Wilk test. Normally distributed continuous variables were expressed as mean ± standard deviation (SD) and compared using the independent Student's t-test or one-way analysis of variance (ANOVA). All variables showing p-value < 0.20 in bivariate analysis as well as clinically relevant covariates were included in a multivariate binary logistic regression model to determine the independent predictors of RLD. The results of the logistic regression were expressed as Adjusted Odds Ratios (aOR) with 95% Confidence Intervals (CI). The p value was considered statistically significant for all the analyses if it was < 0.05 and < 0.01 were regarded as highly statistically significant.

RESULTS

A total of 450 patients were successfully enrolled and completed the study protocol, yielding a final response rate of 92.7%.

Table 1 shows the patients with RLD were significantly older (60.8 vs. 54.1 years, p < 0.001) and had a significantly longer duration of diabetes (median 12.0 vs. 6.0 years, p < 0.001).

Table 1: Baseline Demographic and Clinical Characteristics of the Study Population Stratified by Pulmonary Function Status

Variable	Normal Lung Function (n=296)	Restrictive Lung Dysfunction (n=154)	p-value
Age (years), mean ± SD	54.1 ± 7.5	60.8 ± 8.4	< 0.001
Gender, n (%)			0.142
Male	155 (52.4%)	89 (57.8%)	
Female	141 (47.6%)	65 (42.2%)	
Duration of Diabetes (years), median (IQR)	6.0 (3.0 - 10.0)	12.0 (8.0 - 16.0)	< 0.001
BMI (kg/m²), mean ± SD	26.4 ± 3.2	29.8 ± 4.1	< 0.001
Waist Circumference (cm), mean ± SD	92.5 ± 10.2	101.4 ± 11.5	< 0.001
Hypertension, n (%)	112 (37.8%)	85 (55.2%)	< 0.001
Dyslipidemia, n (%)	145 (49.0%)	92 (59.7%)	0.028
Insulin therapy, n (%)	65 (22.0%)	78 (50.6%)	< 0.001

Table 2 shows as expected by the diagnostic criteria, the RLD group exhibited a profound reduction in Forced Vital Capacity (FVC), both in absolute liters (2.31 vs. 3.45 L, p < 0.001) and as a percentage of the predicted value (68.4% vs. 98.5%, p < 0.001).

Table 2: Pulmonary Function Parameters Comparing Patients with Normal Lung Function and Those with Restrictive Lung Dysfunction.

Pulmonary Parameter	Normal Lung Function (n=296)	Restrictive Lung Dysfunction (n=154)	p-value
FVC (Liters), mean ± SD	3.45 ± 0.52	2.31 ± 0.41	< 0.001
FVC (% Predicted), mean ± SD	98.5 ± 8.2	68.4 ± 7.5	< 0.001
FEV1 (Liters), mean ± SD	2.85 ± 0.48	2.05 ± 0.39	< 0.001
FEV1 (% Predicted), mean ± SD	96.2 ± 9.1	82.5 ± 8.8	< 0.001
FEV1/FVC Ratio, mean ± SD	0.82 ± 0.04	0.88 ± 0.05	< 0.001
FEF 25-75% (L/s), mean ± SD	2.65 ± 0.60	2.30 ± 0.55	< 0.001

Table 3; The prevalence of RLD increased significantly with worsening glycemic control, rising from 18.3% in patients with good control (HbA1c < 7.0%) to 51.0% in those with poor control (HbA1c > 8.0%) (p for trend < 0.001).

Table 3: Prevalence of Restrictive Lung Dysfunction across Different Subgroups of Glycemic Control, Disease duration, and Body Mass Index

Subgroup Category	Total Patients (n)	Patients with RLD (n)	Prevalence of RLD (%)	p-value for trend
HbA1c Level				
< 7.0% (Good control)	120	22	18.3%	< 0.001
7.0% - 8.0% (Moderate)	185	58	31.3%	< 0.001
> 8.0% (Poor control)	145	74	51.0%	< 0.001
Duration of Diabetes				
< 5 years	130	25	19.2%	< 0.001
5 - 10 years	165	52	31.5%	< 0.001
> 10 years	155	77	49.6%	< 0.001
BMI Category				
Normal (18.5 - 24.9)	110	18	16.3%	< 0.001
Overweight (25.0 - 29.9)	195	65	33.3%	< 0.001
Obese (≥ 30.0)	145	71	48.9%	< 0.001

Table 4; All evaluated continuous and categorical variables showed a statistically significant association with the presence of RLD (p < 0.05).

Table 4: Bivariate Analysis of Clinical and Biochemical Predictors Associated with Restrictive Lung Dysfunction.

Variable	Odds Ratio (OR)	95% Confidence Interval	p-value
Age (per 1 year increase)	1.12	1.08 - 1.16	< 0.001
Duration of DM (per 1 year)	1.18	1.13 - 1.23	< 0.001
BMI (per 1 kg/m² increase)	1.25	1.17 - 1.34	< 0.001
HbA1c (per 1% increase)	1.65	1.42 - 1.92	< 0.001
Fasting Blood Glucose (per 10 mg/dL)	1.08	1.03 - 1.13	0.002

Presence of Diabetic Nephropathy	3.45	2.15 - 5.54	< 0.001
Presence of Diabetic Retinopathy	2.10	1.35 - 3.26	0.001
Presence of Peripheral Neuropathy	2.85	1.80 - 4.51	< 0.001
Serum Triglycerides (per 10 mg/dL)	1.04	1.01 - 1.07	0.012
HDL Cholesterol (per 1 mg/dL)	0.96	0.93 - 0.99	0.015

Table 5 Age and the presence of peripheral neuropathy lost their statistical significance (p = 0.112 and p = 0.172, respectively), indicating their initial association was likely confounded by other variables such as disease duration and glycemic control.

Table 5: Multivariate Logistic Regression Analysis for Independent Predictors of Restrictive Lung Dysfunction.

Independent Variable	Adjusted Odds Ratio (aOR)	95% Confidence Interval	p-value
Age (per 1 year increase)	1.04	0.99 - 1.09	0.112
Duration of DM (per 1 year)	1.15	1.08 - 1.22	< 0.001
BMI (per 1 kg/m² increase)	1.08	1.02 - 1.14	0.008
HbA1c > 8.0% (vs < 7.0%)	2.45	1.55 - 3.88	< 0.001
Presence of Diabetic Nephropathy	2.10	1.25 - 3.52	0.004
Presence of Peripheral Neuropathy	1.45	0.85 - 2.48	0.172
Hypertension (Yes vs No)	1.22	0.78 - 1.90	0.381

DISCUSSION

This cross-sectional study offers strong evidence that Restrictive Lung Dysfunction (RLD) is a very common but often unnoticed complication among patients with Type 2 Diabetes Mellitus. Among 450 patients included for this study with diabetes, excluding patients suffering from primary respiratory diseases and active smoking, the overall prevalence of RLD was 34.2%. The frequency of RLD in our study (34.2%) is in line with and at the high end of the range noted in the recent global literature. In pulmonary function in T2DM, a recent meta-analysis [15] showed a pooled prevalence of restrictive patterns between 20% and 40%, depending on the criteria used to diagnose T2DM and the characteristics of the population. In a similar study, carried out in South Korea [16] reported the prevalence of RLD in diabetic patients as 28.5%, whereas a study from India [17] reported 38.1% prevalence of RLD in the diabetic population.

The relatively high prevalence observed in our study could be explained by the fact that our patient population included long-standing diabetes and the fact that obesity is more prevalent in our regional population. This is consistent with the biochemical hypothesis of "diabetic lung" in which the non-enzymatic glycation of structural proteins occurs. Advanced Glycation End-products (AGEs) are formed in the pulmonary interstitium and alveolar wall during chronic hyperglycemia.[18] Recent in vivo studies (2021) revealed that AGEs cause irreversible cross-linking of collagen and elastin fibres, stiffening the lung tissue and resulting in a decrease in its compliance, physiologically represented by a restrictive defect.[19] In addition, AGEs also bind to the RAGE receptors on the cells of the lungs, leading to the release of pro-inflammatory cytokines and profibrotic growth factors, such as TGF-β, which will lead to interstitial fibrosis, and thus to further reduction of lung volumes.[20] In our cohort, diabetes duration was also a strong independent risk factor for RLD (aOR = 1.15 per year of diabetes). This dose-response relationship emphasizes the time-dependent accumulation of parenchymal metabolic and microvascular injury. Hyperglycemia, insulin resistance and systemic inflammation result in progressive pulmonary microangiopathy.[21] One novel and significant finding of our study is that of independent association between diabetic nephropathy and RLD (aOR = 2.10). After adjustment for BMI and hypertension were considered in our study; however, nephropathy remained an independent predictor. This

indicates that the microvascular damage systemic inflammation and endothelial dysfunction is shared between the lungs, and kidneys.[22] The cephalad displacement of the diaphragm is caused by the mass-loading effect of central adiposity, which in turn decreases functional residual capacity (FRC) and expiratory reserve volume (ERV) in particular at the supine position.[22]

It is important to note that our multivariate model was adjusted for BMI, but the metabolic factors (HbA1c, duration, nephropathy) were still significant. This suggests that the "diabetic restriction" is also different from the "obesity-related restriction", since the metabolic and microvascular effects of diabetes have an additive negative impact on the lung volumes beyond the impact of mechanical loading.[23] The clinical implications of these findings are very significant. Current guidelines do not recommend for regular spirometry testing in diabetic patients without symptoms.[24] There is increasing acceptance, however, that cardiac risk should be monitored in high-risk diabetic populations [23] because of the high prevalence of RLD and its relationship with increased cardiovascular and all-cause mortality. Based on our data, patients with longstanding diabetes, poor glycemic control, obesity, especially those with established nephropathy, have the highest risk for RLD. These sub-groups might be targeted for baseline and periodic pulmonary function testing. If RLD is diagnosed early, more aggressive management of glycemic control, weight loss measures and early introduction of pulmonary rehabilitation may help slow the course of lung dysfunction and enhance outcomes for patients.[24] Additionally, the diagnosis of RLD is critical when assessing diabetic patients for surgery or when there is unexplained dyspnea, and this is frequently attributed to only cardiovascular disease or deconditioning.

CONCLUSION

Restrictive Lung Dysfunction is a very common complication of Type 2 Diabetes Mellitus and was seen in more than one-third of the patients. The duration of diabetes, glycemic control, BMI, and diabetic nephropathy are important and independent risk factors for this pulmonary impairment. Due to the high prevalence and the possible morbidity and mortality, physicians should have a high clinical suspicion to consider RLD in diabetic patients, especially those with a long diabetic history, poor glucose control, and microvascular complications of diabetes. PFT is strongly recommended to be integrated into the comprehensive care plan of high-risk T2DM patients, to enable early intervention and to maximize patient outcomes.

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