Original Research Article

Clinical evaluation pulmonary function test in type II Diabetes Mellitus

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Abstract

Introduction: Chronic hyperglycemia is associated with continuing damage, dysfunction and failure of various organs, especially the eyes, kidneys, nerves, heart, lungs, and blood vessels. The pathogenesis is thought to involve both a micro-angiopathic process and non-enzymatic glycosylation of tissue proteins. It has been demonstrated that pulmonary complications in diabetes are due to thickening of walls of alveoli, alveolar capillaries, and pulmonary arterioles and these changes cause pulmonary dysfunction.

Aim of the study: To correlate the lung function in type II diabetes with duration of diabetes and to find out whether it is obstructive or restrictive pattern.

Materials and methods: Totally 100 subjects participated in the study. Out of 100 participants, 50 were type II diabetes forming the study group and the remaining 50 were normal subjects forming the control group. A detailed history and thorough clinical examination were carried out. Inclusion criteria were Apparently healthy individuals with type II diabetic patients on oral hypoglycemic drugs and having diabetes for more than 2 years duration of age group 35 – 55 years. Thorough clinical examination and history were obtained from the subjects in order to determine the health status of the individual. Anthropometric measurements like height, weight were measured and BMI was calculated. Glycemic status for the participants was measured by doing fasting & postprandial blood sugar. HbA1c was determined.

Results: The Mean (±SD) of HbA1c of controls was 3.16 ± 0.482 and for the study group was 5.38 ± 1.174, showed that the controls and study group with good glycemic control were selected for the study. The mean (±SD) of FEV_1 for the control group were 91.40±11.236 and for diabetic group were 81.15±16.523. It was found to be significantly reduced (P= 0.002). The mean (±SD) of FVC for the control group was 81.85±9.211and for diabetic group was 73.75±13.933. The mean (±SD) of PEFR for the control group was 98.85±21.996 and for diabetic group was 85.95±24.045. The mean values of

FVC and PEFR were found to be reduced in diabetic group when compared to controls and were statistically significant. The mean (±SD) of FEV_1/FVC% for the control group was 117.05±7.250 and for diabetic group was 116.58±7.071. The mean (±SD) of $FEF_{25-75\%}$ for the control group was 136.73±26.056 and for diabetic group was 125.63±41.009. The mean (±SD) of MVV for the control group was 65.20±15.010 and for diabetic group was 58.80±16.530. The mean values of FEV_1/FVC%, $FEF_{25-75\%}$, and MVV were reduced in diabetic group when compared with the control group but not statistically significant.

Conclusion: The pulmonary dysfunction may be one of the earliest and easily measurable non-metabolic alterations in diabetes. Therefore the patients with diabetes are suggested to undergo pulmonary function testing periodically. As spirometry is much more reliable, valid and simple test, it is time to include the spirometer as a tool for monitoring diabetes. Strict glycemic control and regular breathing exercises to strengthen respiratory muscles are necessary to improve the pulmonary function in type II diabetics.

Key words

Diabetes mellitus, Pulmonary function test, Obesity, FEV_1/FVC%.

Introduction

Diabetes mellitus is one of the most common chronic diseases in nearly all countries, and continues to increase in numbers and significance, as changing lifestyles lead to reduced physical activity, and increased obesity [1]. It has been demonstrated that pulmonary complications in diabetes are due to the thickening of walls of alveoli, alveolar capillaries, and pulmonary arterioles and these changes cause pulmonary dysfunction. These microvascular complications appear early within 5 to 10 years and macrovascular complications appear within 15 to 20 years from the onset of diabetes [2]. In type I diabetes lung function has been investigated in several clinical studies and evidenced reduced lung volume, reduced elastic recoil, diminished respiratory muscle performance, decrease in pulmonary diffusion capacity for carbon monoxide [3]. As the prevalence of type II DM is increasing, particularly in developing countries like India, and since these changes can potentially incapacitate the patients, it is of utmost importance to define these changes [4]. It is also important to find ways of retarding the progression of disease so that they do not become irreversible thus allowing millions of patients to be economically productive [5]. It has been suggested that pulmonary dysfunction may be one of the earliest measurable non-metabolic alterations in diabetes. So it is important to determine whether these lung function changes also occur in type II diabetes [6].

Materials and methods

Totally 100 subjects participated in the study. Patients who were attending the OPD of Sri Rajah Mutaiah Medical College and Hospital in the year 2019 May-August were included in the study. Out of 100 participants, 50 were type II diabetes forming the study group and the remaining 50 were normal subjects forming the control group. A detailed history and thorough clinical examination were carried out.

Inclusion criteria: Apparently healthy individuals with type II diabetic patients on oral hypoglycemic drugs and having diabetes for more than 2 years duration of age group 35 – 55 years. Thorough clinical examination and history were obtained from the subjects in order to determine the health status of the individual. Anthropometric measurements like height, weight were measured and BMI was calculated. Glycemic status for the participants was measured by doing fasting and postprandial blood sugar. HbA1c was determined. Informed written consent was obtained from all the

participants prior to their participation in the study.

Exclusion criteria:

- Smokers.
- Patients with history of cardiac/respiratory disease (hypertension, myocardial infarction, bronchial asthma, bronchitis, tuberculosis).
- History of recent surgery.
- History of recent respiratory tract infection.
- History of occupational exposure.

Pulmonary function tests were done using computerized spirometer which was standardized according to American Thoracic Society performance criteria (Spiro Excel – Digital Spirometer – Medicaid systems). The pulmonary function parameters like forced vital capacity (FVC), FEV_1, FVC/FEV_1%, PEFR, slow vital capacity (SVC) and maximum voluntary ventilation (MVV) were recorded. The Pulmonary function test was performed 3 times on the same day in sitting posture with two minutes interval and the best of the three was taken. Blood samples were drawn for estimation of fasting blood sugar and glycated hemoglobin after 6 hours of fasting. The subject was asked to take breakfast and post-prandial blood sugar was also checked after 2 hours. The pulmonary function data were represented in three columns. These columns showed the predicted values, measured values obtained during testing and the percent of predicted values for each test. A common method of comparison was to compute a percentage of the predicted value.

Statistical analysis

Pulmonary function parameters were analyzed by using statistical software Microsoft excel and SPSS 18.0 for windows. The statistical analysis was done by the Student's t-test, which was used to find the significant difference of pulmonary function parameters between the healthy non-diabetic controls and type II diabetic cases.

Results

Table - 1 shows the Mean (±SD) of HbA1c of controls was 3.16 ± 0.482 and for the study group was 5.38 ± 1.174, shows that the controls and study group with good glycemic control were selected for the study.

Table – 1: Anthropometric parameters of subjects of control and diabetic groups.

	Control (n=50)				Study (n=50)			
	Min	**Max**	**Mean**	**S.D**	**Min**	**Max**	**Mean**	**S.D**
Age (years)	35	54	40.47	5.630	35	55	47.50	5.724
Height (cm)	152	169	162.38	3.814	157	169	162.75	3.111
Weight (kg)	41	91	61.68	11.796	42	81	60.03	9.588
BMI (kg/m^2)	16.41	32.63	23.34	4.087	16.61	30.49	22.60	3.167
HbA1c%	2.34	4.32	3.1607	0.483	2.40	6.80	5.38	1.174

Table – 2: Comparison of pulmonary function tests parameters between the controls and type II diabetes.

PARAMETER	Control group (n =50)	Diabetic group (n = 40)	P-value
FEV_1	91.40±11.236	81.15±16.523	0.002*
FVC	81.85±9.211	73.75±13.933	0.003*
FEV_1/FVC%	117.05±7.250	116.58±7.071	0.768
PEFR	98.85±21.996	85.95±24.045	0.014*
FEF 25-75%	136.73±26.056	125.63±41.009	0.152
MVV	65.20±15.010	58.80±16.530	0.074

The mean (±SD) of FEV_1 for the control group were 91.40±11.236 and for diabetic group were 81.15±16.523. It was found to be significantly reduced (P= 0.002). The mean (±SD) of FVC for the control group was 81.85±9.211and for diabetic group was 73.75±13.933. The mean (±SD) of PEFR for the control group was 98.85±21.996 and for diabetic group was 85.95±24.045. The mean values of FVC and PEFR were found to be reduced in diabetic group when compared to controls and were statistically significant. The mean (±SD) of FEV_1/FVC% for the control group was 117.05±7.250 and for diabetic group was 116.58±7.071. The mean (±SD) of $FEF_{25-75\%}$ for the control group was 136.73±26.056 and for diabetic group was 125.63±41.009. The mean (±SD) of MVV for the control group was 65.20±15.010 and for diabetic group was 58.80±16.530. The mean values of FEV_1/FVC%, $FEF_{25-75\%,}$ and MVV were reduced in diabetic group when compared with control group but not statistically significant (**Table – 2**).

Discussion

Diabetes is a systemic disease that produces changes in the structure and function of several tissues, particularly of the connective tissues due to microvascular and macrovascular damage that include cardiovascular disease, nephropathy, retinopathy, and neuropathy [7]. Since the lungs have abundant connective tissue, it raises the possibility that lung is also a target organ in diabetes. Histological evidence of pulmonary abnormalities has included alterations in the ultrastructure of granular pneumocytes in the interalveolar septum of non-ciliated bronchiolar epithelial cells and of collagen and elastin in the alveolar wall [8].

In the present study the age group of the subjects was between 35-55 years. The mean values of anthropometric parameters – height, weight, and BMI were not compared between the control and diabetic group [9]. Prakash U, et al. reported that there was no statistically significant difference in the anthropometric profiles of patients. Similarly Asanuma, et al. also observed that there was no significant difference in the anthropometric profiles between male diabetics and controls. In the present study, the Mean (±SD) of HbA1c of controls is 3.16± 0.482 and for the study group is 5.38 ± 1.174. This shows that the controls and study group with good glycemic control are selected for the study. HbA1c reflects the glycemic control only for the past 2-3 months, a duration which may not be long enough to impact an effect on lung function [10]. HbA1c is a relatively short term marker of glycemic control and the impaired lung function could still be present in diabetes. But the duration of glycemic exposure is more important than its magnitude [11]. Sachdev Y, et al. also supported our findings, that in normal healthy non-smokers after the age of 35 years, the expected decline in lung function (FEV_1) is 25-30 ml/yr, whereas, in diabetics, the decline is 71 ml/yr. The reduced FVC was due to increase in the cross-linkage formation between polypeptides of collagen in pulmonary connective tissue [12]. They reported that adults with impaired FVC (% predicted) had various features of insulin resistance. The main suggestion of their study was that impaired lung function (FEV_1 and FVC) deserves high attention as an emerging novel risk factor for type II diabetes. FEVI/FVC ratio is a more sensitive indicator of airway obstruction than FVC or FEV_1 alone. In the present study, the FEV_1/FVC ratio did not show any significant change in diabetics when compared with controls. This shows restrictive type of pulmonary impairment as evidenced by significant reduction in FEV_1, FVC, and normal FEV_1/FVC ratio [13]. Shaw JE, et al. found restrictive lung dysfunction and the possible explanations would be hypoxia-induced insulin resistance, chronic inflammation and low birth weight in early life. The explanation for restrictive type of pulmonary dysfunction was partially explained by inflammation, traditional and metabolic risk factors or by obesity and inflammation. In these individuals FEV_1, FVC, and total lung capacity are reduced, PEFR in their study and stated that the PEFR reflects not only the lung volume and the state of airways, but it also shows the expiratory muscle force and

persistently low PEFR represents the collapsing of large airways [14]. In this study $FEF_{25-75\%}$ values were reduced among diabetics when compared to non-diabetics but not significantly. $FEF_{25-75\%}$ reflects the flow rate during middle 50% of FVC. It also indicates patency of the small airways. Reduced $FEF_{25-75\%}$ results from increased amounts of collagen and elastin in basal lamina of alveolar wall. However, low $FEF_{25-75\%}$ represents the involvement of peripheral bronchioles [15]. MVV is the maximum breathing capacity which is decreased in diabetics due to poor respiratory muscle strength as a result of increased protein catabolism. The explanation for reduced lung functions in diabetics is due to biochemical alterations in the connective tissue of the lung, particularly collagen and elastin, as well as microangiopathy [16]. This is due to non-enzymatic glycosylation of proteins induced by chronic hyperglycemia [17]. The functional abnormalities from these changes are thickening of the pulmonary capillary basal lamina and the alveolar epithelium, reduction in elastic recoil of the lung, lung volumes, and also reduced pulmonary capacity for the diffusion of carbon monoxide [18].

Conclusion

The result of the present study shows that there is a decrease in the pulmonary function in type II diabetics when compared with healthy controls. In this study there is a restrictive type of pulmonary impairment in type II diabetics and as the duration of diabetes increases the restrictive lung impairment becomes more prominent.

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