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Pulmonary Function Test in Type II Diabetics

Dr. Ratnesh Namdeo Gajbhiye & Dr. Anil Shrihari Tambe
Indira Gandhi Govt. Medical college, Nagpur, (Maharashtra- India).

Abstract

Objective: Present study was undertaken to find out the pulmonary function test in type II diabetics.

Background: Diabetes is a disease which affects multiple organ systems. Major consequence of hyperglycemia is excessive non enzymatic glycosylation of various body proteins including haemoglobin, albumin, collagen and elastin. Due to this, there occur irreversible changes in the chemical structure of tissue proteins. Basement membrane and connective tissues in skin, muscles, respiratory system, vascular bed, kidney, peripheral nervous system, etc. are the targets for glycosylation. Diabetic patients show reduced pulmonary function tests.

Method*:* 42 Type II diabetics and 40 normal subjects were selected for the study. Anthropometric parameters, blood investigations and P.F.T. were performed on all subjects.

Result and Discussion*:* Anthropometric parameters like Weight, B.M.I., and B.S.A. were found to be non significant in Type II diabetics. Fasting and Post Meal blood glucose levels as well as HbA_1c % were significantly more in Type II diabetics as compared to controls. All P.F.T. parameters excepting FEV_1 % were also significantly less in Type II diabetics. Decreased P.F.T. in Type II diabetics can be attributed to increased glycosylation of connective tissues and other proteins in the lungs, leading to a decrease in elasticity, flexibility and recoiling capacity ultimately producing stiff lung i.e. restrictive lung pathology.

Key Words: - *Diabetes Mellitus, Glycosylation, Pulmonary Function Test.*

Introduction

Diabetes Mellitus is a heterogeneous metabolic disorder characterized by common features of chronic hyperglycemia with disturbance of carbohydrate, fat and protein metabolism. Many organ systems are the targets in diabetes like cardiovascular system, eyes, kidney and nervous system. The possibility that the lung is also a target organ for diabetic complications was first suggested by Schuyler et al in 1976 [1]. Since that time there have been many studies of pulmonary function in diabetic patients with conflicting results. Many have suggested plausible patho-physiological mechanisms also. Major consequences of hyperglycemia are excessive non enzymatic glycosylation of various body proteins including hemoglobin, albumin, collagen and elastin [2,3,4,5,6]. Glycosylation leads to irreversible changes in the chemical structure of tissue proteins. These chemical changes have been implicated in producing long term complications of diabetes. Reduced elastic recoil of the lungs because of increased glycosylation of connective tissues is one of the long term effects of diabetes on the respiratory system [2,7]. There is restrictive lung pathology in diabetes [8].

Materials and Methods

The approval of Institutional ethical committee was obtained for the study. Informed written consent was obtained from all the subjects. All were males in the age group of 41 – 60 years. Study group included 42 Type II diabetics and for comparison age and height matched 40 subjects were selected from staff members as control group.

Each group was supplemented by respiratory questionnaire [9] followed by thorough physical examination and investigations. All subjects from study group and control group were free from cardiac, respiratory and other such diseases which may impair pulmonary function. All were non-smokers with no history of smoking in the past. After selection, subjects from both groups were asked to report in Dept. of Physiology, I.G.G.M.C. Nagpur in the morning hours (10 A.M. – 12.30 P.M.) for measurement of anthropometric parameters, pulmonary function testing and blood investigations.

Standing height was measured by simply making the subject stand bare foot against a wall on which measuring scale is inscribed. Weight was done by KRUPS weighing machine in light weight garments without foot wears. BSA was calculated using Dubois chart [10]. BMI was calculated using formula Weight in kg / (Height in meter) ^{2}. Fasting and post meal blood glucose level was measured by Accu Chek Active glucometer (Glucose Oxidase Biosensor assay method). HbA_1c% was measured using cation exchange resin method (monozyme's glycohemin kit).

Pulmonary function test was determined using MEDSPIROR – Recorder and Medicare system.

Necessary instructions were given to the subjects before performing P.F.T. They were asked to execute fast forcible expiration as much as possible at the end of deep full inspiration. Subjects were asked to perform the test till they become accustomed to the procedure. Then, three consecutive readings were obtained and the best was selected for the study. One single expiratory effort gives many readings, out of them FVC, FEV_1, FEV_1 %, $FEF_{25-75\%}$, $FEF_{0.2-1.2L}$, PEFR were selected for the study. After the rest of 15 minutes, the test to obtain Maximum Ventilation Volume was carried out. Subjects were asked to inhale and exhale as deep and as fast as possible for twelve seconds. The in-built calculation in the Medspiror gives MVV which was repeated for three consecutive times with a period of rest for

ten minutes between each effort and best reading was selected for the study. Statistical analysis of observations was carried out. Mean and Standard Deviation were calculated and significance of difference was tested statistically by unpaired student's t test [11].

Results

The results of the study were compared between the study group (type II diabetes mellitus) and the control group as shown in the tables.

Table 1: showing comparison of anthropometric parameters in control group and type II diabetics. (Values are expressed as Mean ± SD.)

Parameters	**Controls (n = 40)** (Mean ± SD)	**Type II DM (n = 42)** (Mean ± SD)
Age (yrs)	50.57 ± 5.81	50.73 ± 6.15
Weight (Kg)	61.32 ± 4.85	58.47 ± 8.18
Height (meter)	1.63 ± 0.05	1.67 ± 0.08
B.M.I. (Kg/m²)	23.06 ± 1.69	22.26 ± 2.35
B.S.A. (m²)	1.64 ± 0.08	1.60 ± 0.12

Table 2: showing comparison of blood parameters and P.F.T. parameters in control group and type II diabetics. (Values are expressed as Mean ± SD.)

Parameters	**Controls** (Mean ± SD)	**Type II DM** (Mean ± SD)
Fasting (mg %)	93.87 ± 7.17	157.30 ±53.42**
Post Meal (mg %)	128.45 ± 7.01	250.59 ±90.71**
HbA_1c %	4.40 ± 1.04	8.30 ±1.78**
FVC (Litre)	2.90 ± 0.22	2.43 ± 0.50**
FEV_1 (Litre)	2.49 ± 0.22	2.31 ± 0.40**
FEV_1 %	85.74 ± 2.74	84.80 ± 3.96*
FEF 25-75 % (L/sec)	3.21 ± 0.40	2.69 ± 0.55**
FEF 0.2-1.2 L (L/sec)	5.96 ± 0.65	4.78 ± 1.16**
PEFR (L/sec)	7.21 ± 0.96	5.90 ± 1.10**
MVV (L/min)	110.87 ± 11.92	90.33 ± 20.00**

*p > 0.05 non significant
**p < 0.001 highly significant

Discussion

Anthropometric parameters were found to be non significant in type II diabetics as compared to controls.

Fasting and post meal blood glucose levels as well as HbA_1c % were found to be significantly more in Type II diabetics [12], pointing to the fact that there is poor glycemic control. This may be because of irregular drug intake, inappropriate drugs, sub-dosing, overeating, lack of diabetic life style discipline, etc. practiced by the patients.

HbA_1c is an indicator of diabetic control. Higher the level of HbA_1c , poor is the diabetic control i.e. higher level of circulating glucose. If circulating glucose is constantly lingering at higher level for 3 months (as measured by HbA_1c %), it can lead to more non-enzymatic glycosylation of tissue proteins. If respiratory system is a target of this affection, this will reflect in P.F.T. parameters analysed.

FVC and FEV_1 are significantly less in Type II diabetics. But FEV_1% is not significantly different in both cases and controls. This signifies that restrictive lung pathology occurs in diabetics. Similar findings were observed by other authors [7,13,14,15,16,17,18].

$FEF_{25-75\%}$ is an indicator of force of expiration of gases during middle 50% of forced expiration. In type II diabetics $FEF_{25-75\%}$ is significantly less compared to controls. Forced expiration is considered to be supported by muscular and recoil forces of respiratory system. The flow can be decreased even due to obstruction, but this is excluded as FEV_1% is normal. Thus decrement in recoiling forces of the lung because of increased glycosylation of respiratory apparatus is responsible for significant decrease in $FEF_{25-75\%}$. Similar finding was observed in other study [19].

$FEF_{0.2-1.2\ L}$ is the initial portion of forced expiratory manoeuvre. First 200 ml of the gas is from the dead space. Remaining 1 litre is exhaled from lung broncho-alveolar tree. This includes some gas from functional residual capacity,

as normal tidal volume is 500 ml. This extraction of gas is due to compression forces that are built up by expiratory muscles. But due to glycosylation of connective tissues of the respiratory apparatus, compression forces that are built up by expiratory muscles might be reduced, resulting in significant decreased $FEF_{0.2\text{-}1.2\ L}$ in type II diabetics.

PEFR is gas exhaled in $1/10^{th}$ of a second during forced expiratory manoeuvre. At this time, recoil forces of the lung and contractile forces of musculature are functionally maximal and supporting the expiration to the maximal. But due to glycosylation of connective tissues of the respiratory apparatus, the muscular and recoil forces of the lung and the respiratory system for expiratory purpose might be decreased, leading to significantly decreased PEFR in type II diabetics [16,17]

MVV is the manoeuvre where maximum ventilator efforts are made. MVV in type II diabetics is significantly less indicating that muscular forces are weakened causing decrease in lung compliance. This again is due to glycosylation of connective tissues of respiratory apparatus.

So in the present study, HbA_1c% was found to be significantly more in type II diabetics that is indicative of improperly monitored blood sugar in last 3 months. This may be due to improper and irregular drug intake, drug resistance and many other factors which seem to be responsible for failure in lowering of blood sugar levels and has lead to increased glycosylation of the various body tissues in general.

The message through the present work is that there is deterioration of lung function, in the form of restrictive lung pathology [8], the obstructive element can be produced depending upon the exposure to infections, irritations, environmental pollutions etc. MVV supports the above finding that it has decreased due to restriction. The restrictive pathology is due to overall effect of glycosylation on the collagen and elastic framework of the respiratory apparatus [2]. These tissues being present everywhere such as in skin, muscles, fascia, joints, lung parenchyma, pleura, there is overall damage to the whole respiratory apparatus [5,6]. These micro damages produce less effective "negative-pressure-pump" and a less compliant lung.

But normally the diabetic patient may not appreciate the respiratory muscle weakness as during normal tidal respiration, diaphragm is the only muscle used and tidal work load on non-diaphragmatic respiratory muscles (i.e. intercostal muscles) being comparatively less, weakness of respiratory muscles is not much appreciated by the patient in routine life. This does not mean that respiratory muscles are not weakened. Here there is ample space to predict that in diabetes the force building ability of respiratory musculature decreases but magnitude of restriction will depend upon individual susceptibility and susceptibility of the lungs. Keeping this individual variation in mind it becomes necessary to test HbA_1c % and P.F.T. at regular intervals to find out early deterioration of lungs in diabetic patients.

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