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A Comparative study of Pulmonary functions in Obese type II Diabetic and Obese Non diabetic women

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Abstract :

Background : The association of obesity with type2 diabetes has been recognized for decades and the major basis for this link is the ability of obesity to engender insulin resistance. Obesity, a complex metabolic condition which influences many physiological systems, including immune function that can dramatically influence the response of the lungs to inflammatory stimuli.

Aims : To evaluate and compare the spirometric findings of obese non diabetic women with those of obese type 2 diabetic women.

Materials and Methods : Pulmonary function tests were performed on 30 obese type2 diabetic and 30 obese non diabetic women using Medspiror. The values of FVC, FEV_1, FVC/FEV_1, PEFR and FEF25-75 were obtained with the help of above instrument.

Results : The results of PFT between obese diabetic and obese non diabetic subjects showed a significant decrease in the values of PEFR and MEANFEF 25-75(L/S) suggesting an obstructive pattern of lung disease in obese diabetic subjects. The findings of present study suggest that, lung is a target organ for damage in diabetes and the glycemic exposure is a strong determinant of reduced pulmonary function in type 2 diabetics. Therefore, the impact of type 2 diabetes on pulmonary function should be considered by those providing care for obese people.

Keywords : Obesity, Diabetes, PFT

Introduction :

Obesity is a complex metabolic condition that influences many physiological systems, including immune function. The prevalence of obesity is increasing at an alarming rate and this rapid increase of obesity prevalence during the last two decades has become one of the main threats to public health. WHO predicts that by 2015 at least 10% of the projected global population will be obese which might constitute a significant health and economic burden.

Obesity was first described as a low-grade inflammatory condition more than a decade ago. However, only relatively recently it has been suggested to be associated with increased macrophage infiltration of adipose tissue, as well as an increase in the number of "M1" or "classically activated" macrophages.[1]. High risk obesity is characterized by abdominal obesity with evidence of abnormal glucose and lipid metabolism and a state of heightened inflammation.

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Although there has been little focus on the impact of obesity on respiratory disease, there are clear effects on pulmonary function.[2] The total respiratory compliance in obesity may be reduced to as low as one-third of normal. These results are not only due to the effect of excess truncal fat mass but also increased pulmonary blood volume and increased closure of the dependent airways of patients who are obese. Severely obese patients may also demonstrate inefficiency of the respiratory muscles, particularly the diaphragm .

Type 2 diabetes is another epidemic disease strongly associated with obesity. The relationship between both obesity and diabetes is of such interdependence that the term 'diabesity' has been coined. Data from the Atherosclerosis risk in communities study showed a faster pulmonary function decline in type 2 diabetic patients than in other participants. This is undeniably significant, because airflow limitation is an independent predictor of death in type 2 diabetes.[3]

Obesity and insulin resistance, the cardinal features of metabolic syndrome are closely associated with a state of low-grade inflammation[4]. In adipose tissue chronic over nutrition leads to macrophage infiltration, resulting in local inflammation that potentiates insulin resistance. Moreover, systemic inflammation is also thought to play

a role in the association between reduced pulmonary function and as well as all-cause mortality[5]. Thus, obesity and diabetes associated with inflammatory processes, cause altered pulmonary function.

Though several studies have confirmed the effect of diabetes and obesity on the pulmonary functions there are only a few studies designed to investigate whether diabetes and the degree of glycemic control are independent determinants of reduced pulmonary function in obese patients. Thus the present study was designed to compare respiratory function between closely matched diabetic and non-diabetic obese participants with the most important variables of pulmonary function tests that could affect lung function.

Materials and Methods :

Thirty obese diabetic women and thirty obese non diabetic women aged between 30 & 65 yrs attending outpatient department in a tertiary care medical centre in Kuppam, Southern Andhra Pradesh, were selected for the study. The criteria for inclusion of the subjects were the following: Obese diabetic women with duration of illness between 2 &10 yrs (BMI >25)[6], Obese non diabetic women with duration of illness between 2 &10 yrs (BMI >30),non smokers, subjects with no past history of chronic respiratory illness, subjects with no symptoms of respiratory illness at the time of examination, and subjects with no chronic cardiovascular disease.

A detailed health status assessment was done for all the subjects included in the study through history taking. Body mass index (BMI) was calculated after measuring height and weight. Clinical examination was done to rule out other systemic abnormalities. The ethical clearance was obtained. The subjects were briefed about the procedure and a written consent was taken. Then, every individual was subjected to pulmonary function tests.

Pulmonary function tests were performed using Medspiror, which is a PC based spirometer with a flow transducer. Tests were performed on all the subjects in sitting position. Reference values for spirometry were based on age, sex and height provided in the software.

The whole procedure was explained and demonstrated to the subjects before testing. Later the subjects were asked to perform the forced vital capacity manoeuvre. FVC was recorded after a maximal inspiration when the subject expired forcefully in to the mouth piece. A

minimum of 3 acceptable FVC manoeuvres were performed and the best manoeuvres were selected and accepted. Acceptability criteria were: full inhalation before the start of test, satisfactory start of exhalation (maximal effort exerted with no hesitation), no cough during the 1st second of manoeuvre, no early termination of exhalation.

For FVC and FEV_1 manoeuvres , the difference of two largest values should be within 200ml. for PEFR the difference of two largest values should be within 10%. When this criteria was not met, the testing was continued for 8 trials, even after 8 trials when the criteria was not met, the testing was stopped and the 3 best acceptable were taken and interpreted. Calibration was done from time to time for accuracy. After scrutinizing the flow volume curve and the time volume curve, the parameters derived were FVC, FEV1, FEV1/FVC, PEFR and FEF-25-75%. These criteria are based on American thoracic society (ATS) and European thoracic society standards.

Results :

The pulmonary function tests were evaluated in obese type 2 diabetic and obese non diabetic women who were divided into two groups

- Group 1 included Obese type 2 Diabetic (Cases)
- Group 2 included Obese Non Diabetic subjects (controls)

The data profile of subjects and pulmonary function test values are entered (as mean and standard deviation) in table and in graph format . Student't' test was used to compare means of PFT parameters between the 2 groups. P value <0.05 was considered as significant for student't' test.

Table 1 shows the data of baseline characteristics of both groups. The mean age of obese diabetic women was 49.36, while the mean age of obese non diabetic women was 47.53. The mean height of obese diabetic women was 150.73$\pm$ 7.73(Range: 142-169cms) and the mean height of obese non diabetic women was 154.16$\pm$ 7.06 (Range: 143-171cms).The mean weight of obese diabetic women was 72.33$\pm$8.01(Range: 56-100kg) and the mean weight of obese non diabetic women was 76.03$\pm$7.66 (Range: 64-100kg). The mean value of the BMI among obese diabetic women was 32 and obese non diabetic women was 32.02.

Table 2 shows the results of PFT in obese tye2 diabetic and obese non diabetic women. The results of PFT between obese diabetic and obese non diabetic women showed a significant decrease in the values of PEFR and MEANFEF 25-75(L/S) suggesting an obstructive pattern of lung disease in obese diabetic women

Discussion :

Obesity and type-2 diabetes mellitus (T2DM) are two major features of the metabolic syndrome, the prevalence of which has reached epidemic proportions. Obesity mechanically restricts lung volume in addition to causing widespread lipid deposition in non-adipose tissue including the lung, which increases pro-oxidant and pro-inflammatory cellular stress as well as alterations in lung structure.[7]

Obesity associated with a low-grade inflammation of white adipose tissue (WAT) resulting from chronic activation of the innate immune system can subsequently lead to insulin resistance, impaired glucose tolerance and even diabetes.[8]

Dysregulation of adipokine secretion, free fatty acid toxicity, and the site-specific differences in abdominal (visceral) versus subcutaneous fat support abdominal obesity as a causal factor mediating the insulin resistance, increased risk of diabetes, and cardiovascular disease in metabolic syndrome.[9]

In the present study, evidence for diabetes as a risk factor for respiratory function impairment in obese women is established. The two main abnormalities in the pulmonary function tests when comparison was made between obese diabetic and obese non diabetic women were a decrease in PEFR and mean FEF25-75 in obese diabetic suggestive of obstructive ventilator pattern. The present study is in agreement with Lecube etal whose results showed a lower forced expiratory volume at 1 s (FEV1) (mean difference −11.6% of predicted [95% CI −20.4 to −2.8]; p=0.011), FEV1/FVC ratio (mean difference −4.4% [95% CI −8.1 to −0.7]; p=0.049 in obese diabetic suggesting that the presence of diabetes and the degree of glycemic control are related to respiratory function impairment in morbidly obese women.[2]

The results of the present study is also supported by the research work of Sreeja etal, which showed a reduction in PEF and FEF25-75% in the case group.[10] and Ramirez LC whose results showed that long-term near-normoglycemia may be beneficial in preventing the deterioration of pulmonary function associated with diabetes mellitus.[11]

The findings of present study suggest that, lung is a target organ for damage in diabetes and the glycemic exposure is a strong determinant of reduced pulmonary function in type 2 diabetics. As measures of airflow limitation predict all-cause mortality in type 2 diabetes, intensive glycemic management may reduce the risk of death through improved ventilatory function independent of other beneficial effects. Therefore, the impact of type 2 diabetes on pulmonary function should be considered by those providing care for obese people. Future studies to define not only the mechanisms involved in the pulmonary dysfunction associated with type 2 diabetes, but also to determine whether blood glucose control could prevent lung injury, are needed.

Table 1: Baseline characteristics of case and control group

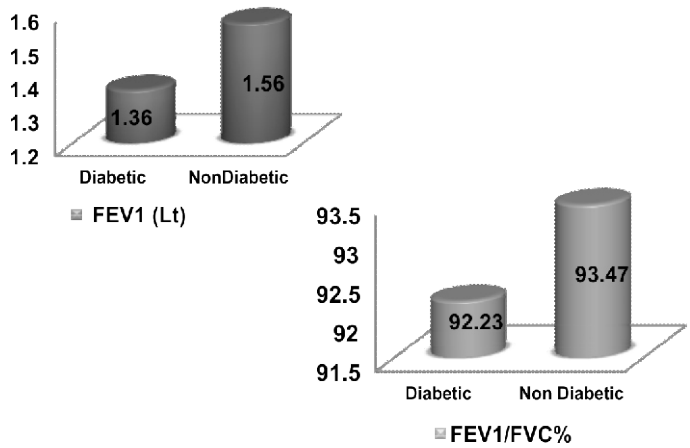
Parameters	Cases	Controls	P value
Age	49.36±7.54	47.53±9.59	0.41
Height	151.63± 7.90	154.6± 7.98	0.153
Weight	72.33± 9.31	76.50±10.07	0.09
BMI	31.57± 4.04	32.05±3.61	0.62

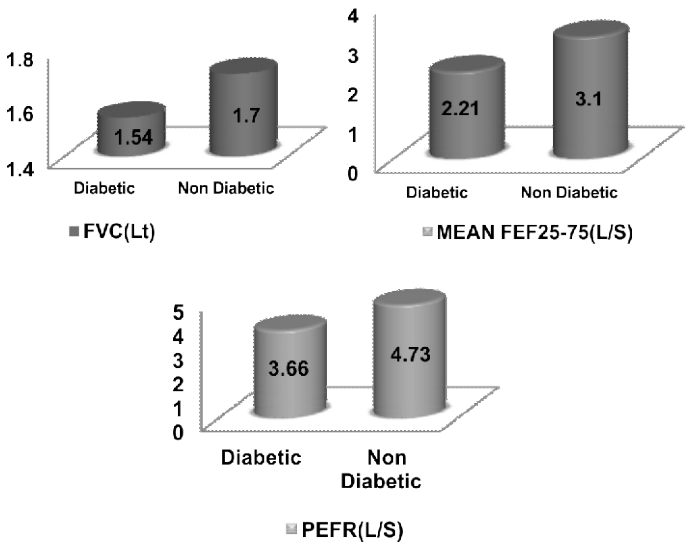
Table 2 : Comparison of pulmonary function tests between obese diabetics and obese non diabetic women

Parameters	Diabetic (Mean ± SD)	Non Diabetic (Mean±SD)	p Value
FEV1(Lt)	1.36± 0.58	1.56 ± 0.38	0.11
FVC(Lt)	1.54± 0.73	1.70± 0.49	0.31
FEV1/FVC%	92.23± 14.10	93.47± 11.11	0.70
PEFR(L/S)	3.66± 1.88	4.73 ± 1.78	0.02
MEANFEF 25-75(L/S)	2.21± 1.2	3.10 ± 1.21	0.005

FVC - Forced vital capacity, FEV1- Forced expiratory volume in 1 second, FEF 25-75 % - Forced expiratory flow 25-75 %, PEFR– Peak expiratory flow

Graph 1:Graphical representation of the results of PFT in obese diabetic and obese non diabetic subjects:





References :

1. Heilbronn LK, Campbell LV. Adipose tissue macrophages, low grade inflammation and insulin resistance in human obesity. Curr Pharm Des. 2008;14(12):1225-30.
2. Lecube & G. Sampol & X. Muñoz & C. Hernández ;Type 2 diabetes impairs pulmonary function in morbidly obese women: a case–control study. Diabetologia (2010) 53:1210–6
3. Davis WA, Knuiman M, Kendall P, Grange V, Davis TM ; Glycemic exposure is associated with reduced pulmonary function in type 2 diabetes. The Fremantle Diabetes Study. Diabetes Care. 2004; 27:752–7.
4. Odegaard JI, Ricardo-Gonzalez RR, Goforth MH, Morel CR; Macrophage-specific PPAR gamma controls alternative activation and improves insulin resistance. Nature. 2007; 447(7148):1116-20.
5. Ochs-Balcom HM, Grant BJB, Muti P, Sempos CT, Freudenheim JL, et. al. Pulmonary Function and Abdominal Adiposity in the General Population. CHEST. 2006; 129:853–62.
6. Maria chiu, Peter C Austin,Douglas G. Manuel, Baiju R Shah,:Deriving Ethinic specific BMI cut off points for assessing diabetes risk, Care. Diabetes 2011; 35(8):1741-8.
7. Cuneyt Yilmaz1, Priya Ravikumar1, Dennis J. Bellotto1, Roger H. Unger2, and Connie C. W. Hsia; Fatty diabetic lung: functional impairment in a model of metabolic syndrome. J App phys 2010; 109 (6) :1913-9
8. Bastard JP, Maachi M, Lagathu C, Kim MJ, Caron M, Vidal H ;Recent advances in the relationship between obesity, inflammation, and insulin resistance. Eur Cytokine Netw. 2006;17(1):4-12.
9. Phillips LK, Prins JB; The link between abdominal obesity and the metabolic syndrome; Curr Hypertens Rep. 2008;10(2):156-64.
10. Sreeja CK, Samuel E, Kesavachandran C, Shashidhar S;Pulmonary function in patients with diabetes mellitus. Ind J Phys Pharm 2003, 47(1):87-93
11. Ramirez LC,Dal Nogare A, Hsia C,Arauz C,Butt I;Relatioinship Between Diabetes Control and Pulmonary Function In Insulin Dependent Diabetes Mellitus. Am J Med.1991;91(4):371-6.

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