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A Comparative Study of Serum Insulin and Insulin Resistance in Obese and Obese Niddm Patients

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Abstract

Objective: Obesity is a major health problem affecting all population worldwide. It is a multifactorial disorder and its development is due to multiple interactions between genes and environment. It is an important risk factor in development of insulin resistance and metabolic syndrome that links with hypertension and non-insulin dependent diabetes mellitus (NIDDM).

Material & Methods: Present study was conducted on 35 obese patients and 35 obese NIDDM patients of either sex. Serum glucose was estimated Endpoint Enzymatic Glucose Oxidase and Peroxidase[7] (GOD–POD) Method Serum insulin was measured using ELISA techniques and Insulin resistance was calculated. Results were subjected to suitable statistical analysis using student't' test and p-value.

Result: Serum glucose, insulin level and insulin resistance was higher in obese NIDDM as compared to obese subjects. There were a positive correlation between degree of obesity and insulin resistance.

Conclusion: Hyperinsulinemia and insulin resistance are true markers for diagnosis metabolic syndrome and proper management of obesity at early stage helps in correcting a number of metabolic abnormalities and its sequel of complication.

Keyword: Obesity, Glucose, BMI (Body Mass Index), Insulin, Insulin resistance, Obese NIDDM.

Introduction

Obesity is a major health problem affecting all population worldwide. It is emerging as a serious international public health concern. It is a multifactorial disorder and its development is due to multiple interactions between genes and environment. The primary cause for being overweight and obese is unhealthy dietary habits, reduced physical activities as well as the genetic predisposition.[1] Obesity carries a definite health risk when there is 20% excess body weight over desired weight or with BMI above 85th percentile for young adults. Obesity and obesity related disorders are increasing worldwide during the last decades, among children, adolescents and adults.[2]

Obesity is possibly mother of all the important diseases of adult life. It is increases the likelihood of various co morbidities, particularly hypertension coronary heart disease, NIDDM, obstructive sleep apnoea, certain types of cancer, osteoarthritis and metabolic syndrome. [3.]Insulin is the polypeptide anabolic hormone synthesized and secretes by the pancreatic beta cells of pancreatic islets of Langerhans. The human pancreas secretes 40-50 units of insulin daily which represents about 15 - 20% of hormone stored in gland. Insulin resistance exists when a given known quantity of insulin produces less than the normal expected biological effect. Although the action of insulin are multiple, the term insulin resistance typically refers to actions of insulin on glucose homeostasis.[4]Every obese person is almost uniformly insulin resistant because there are increase concentrations of circulating insulin both in the basal state and after various stimuli of insulin secretion.[5]

Insulin resistance (IR) is an insufficient response of target tissues such as liver, skeletal muscle and adipose tissues to the physiological plasma insulin levels. The onset of insulin resistance is heralded by postprandial hyperinsulinemia, followed by fasting hyperinsulinemia and ultimately hyperglycemia.[6]It is an important risk factor in development of insulin resistance and metabolic syndrome that links with hypertension and non insulin dependent diabetes mellitus (NIDDM). It can be seen as the first wave of a defined cluster of non communicable diseases and becoming an emerging cancer. The present study was planned to evaluate serum fasting glucose, insulin level in obese and obese NIDDM subject to evaluate the role of these parameters in causation and pathogenesis of these diseases and compare the utility of estimation and significance of these biochemical markers in early diagnosis and timely management of disease to present the sequel of complications and burden of these co morbid conditions.

Aims & Objectives: To evaluate serum insulin and insulin resistance in obese and obese NIDDM patients.

Material & Methods

The present study was conducted in the Department of Biochemistry, Dr. S.N. Medical College and its associated group of hospitals, Jodhpur, Rajasthan-342001. The study design is hospital based unmatched case–control study. The study was approved by institutional research ethical committee Dr. S. N. Medical College Jodhpur, Rajasthan. The nature of study was explained and written informed consent was taken from the patients in local language. The source population is patients who visit the Medicine OPD / clinics of the Dr. S.N. Medical College and its associated group of the hospital during the study period. A total of 70 obese subjects with or without NIDDM.

Study Groups: the subjects were grouped as-

Group 1: 35 obese subjects (16 male & 19 female) and varying age from 30–70 years

Group 2: 35 obese NIDDM subjects (17 male & 18 female) and varying age from 30–70 years.

After an overnight fast of 10–12 hours, venous blood sample was drawn from anticubital vein of each subject by using standard aseptic techniques.

- Sodium fluoride oxalate vial: The blood which was collected in this vial was used for the estimation of fasting blood glucose.
- Plain vial: In this vial, collected blood was allowed to clot. The serum was separated from the clotted specimen by centrifugation at 3000 rpm for 10

minutes and serum was used for the estimation of Serum Insulin.

Weight, height, was recorded and Body mass index (BMI) was calculated. Serum glucose was estimated Endpoint Enzymatic Glucose Oxidase and Peroxidase[7] (GOD–POD) Method and Serum insulin was measured using ELISA technique[8] using a fully automated clinical chemistry analyzer and Insulin Resistance was calculated by HOMA-IR.[9] Pre-coded data was entered on the computer using "Microsoft Office Excel Software" program (2010) for windows. Data was then transferred to the Statistical Package of Social Science Software program, version 22 (SPSS) to be statistically analyzed. Data was summarized using mean, and standard deviation for quantitative variables and frequency and percentage for qualitative ones. Comparison between groups was performed using independent sample t-test for quantitative variables. Pearson correlation coefficients were calculated to signify the association between different quantitative variables. P values <0.05 were considered statistically significant, and < 0.01 were considered highly significant.

Inclusion Criteria

1. Person with age of 30–70 years and Body Mass Index (BMI) ≥30 and those are Considered obese and subject with obese NIDDM only was selected for study.

Exclusion Criteria

1. Diabetic patients with overt complications like neuropathy, nephropathy, retinopathy, and ischemic heart disease.
2. Patients with acute complications like diabetic keto-acidosis, non-ketosis hyperosmolar coma and hypoglycemia.
3. Patients with any concurrent illness like chronic liver disease, hypothyroidism.
4. Patients on drugs like diuretics, steroids, oral contraceptives and beta blockers, magnesium supplementation etc.

Table: 1 : Mean age (in years) of the subjects studied

S. No.	Group Studied (n)	Age (Mean ± S.D.) years	Statistical significance obese v/s Obese NIDDM
1	Obese Subject (35)	48.74.±8.64 (34-69)	t = 0.58 p = 0.56 [NS]
2	Obese NIDDM Subject (35)	49.46±9.35 (33-64)	

[NON- Significant]

Table 2: Mean body mass index (BMI) (kg/m^2) of the subjects studied

S. No.	Group Studied (n)	BMI(Body Mass Index) Mean ± SD [kg/m^2]	Statistical significance obese v/s Obese NIDDM
1	Obese Subject (35)	30.55 ±2.47 (24.7-36.6)	t = .311 p = 0.94 [HS]
2	Obese NIDDM Subject (35)	31.32±2.51 (26.00-35.00)	

[HS-Highly Significant]

Table 3: Mean Fasting Plasma Glucose Levels of the Groups Studied

S. No.	Group Studied (n)	Fasting Glucose (mg/dl) Mean ± SD [Range]	Statistical significance obese v/s Obese NIDDM
1	Obese Subject (35)	92.46±5.90(78.23-105.26)	t = 16.51 p = 0.0001 [HS]
2	Obese NIDDM Subject (35)	171.07±27.53 (125.4-224.28)	

[HS-Highly Significant]

The mean Fasting Plasma Glucose was 92.46±5.90 mg/dl and 171.07±27.53 (mg/dl) in the obese subjects and obese NIDDM subjects. A statistically highly significant elevation was observed in fasting plasma glucose levels in obese NIDDM subjects as compared to obese subjects [Table 3].

Our results are in accordance with the study of Nagwa A et al (2015), Blood Glucose level was significantly higher (P < 0.01) in obese type 2 diabetic subjects than obese subjects.[10] Obesity causes glucose intolerance state that can lead to the development of impaired glucose metabolism.

Table: 4 : Mean fasting serum insulin (µIU/ml) of the groups studied.

S. No.	Group Studied (n)	Fasting Serum Insulin (µIU/ml) Mean ± SD [Range]	Statistical significance obese v/s Obese NIDDM
1	Obese Subject (35)	25.32±5.72(4.57-34.89)	t = 4.77 p = 0.0001 [HS]
2	Obese NIDDM Subject (35)	32.12±6.19 (23.6-45.13)	

[HS-Highly significant]

The mean fasting serum insulin level was 25.32±5.72 µIU/ml and 32.12±6.19 µIU/ml in the obese subjects and obese NIDDM subjects. Statistically highly significant change was observed in the serum Insulin level in obese NIDDM subjects when results were compared to obese subjects [Table 4].

Hyperinsulinemia a surrogate marker of insulin resistance is a classical symptom of metabolic syndrome in obesity. Further, its elevation indicates development of type 2 diabetes and metabolic syndrome.

Table 5: Mean Insulin Resistance (Homa – IR) Index Of The Groups Studied.

S. No.	Group Studied (n)	Homa - IR Index Mean ±SD [Range]	Statistical significance obese v/s Obese NIDDM
1	Obese Subject (35)	5.74±1.27 (1.00-7.4)	t = 14.13 p = 0.0001 [HS]
2	Obese NIDDM Subject (35)	13.37±2.93 (8.2-19.8)	

[HS: Highly significant]

The mean insulin resistance was 5.74±1.27 and 13.37±2.93 in the obese subjects and obese NIDDM subjects. Statistically highly significant increase was observed in the serum Insulin resistance of obese

NIDDM subjects (t= 14.13, p=0.0001) as compared to obese subjects [Table 3].

Table 6: Correlation coefficient (r) of various parameters studied.

S.NO.	Parameters	Group Studied	r value
1	BMI v/s Insulin Resistance	Obese subjects	0.9106
		Obese NIDDM subjects	0.1001
2	Plasma glucose v/s Insulin Resistance	Obese subjects	0.9514
		Obese NIDDM subjects	0.6896

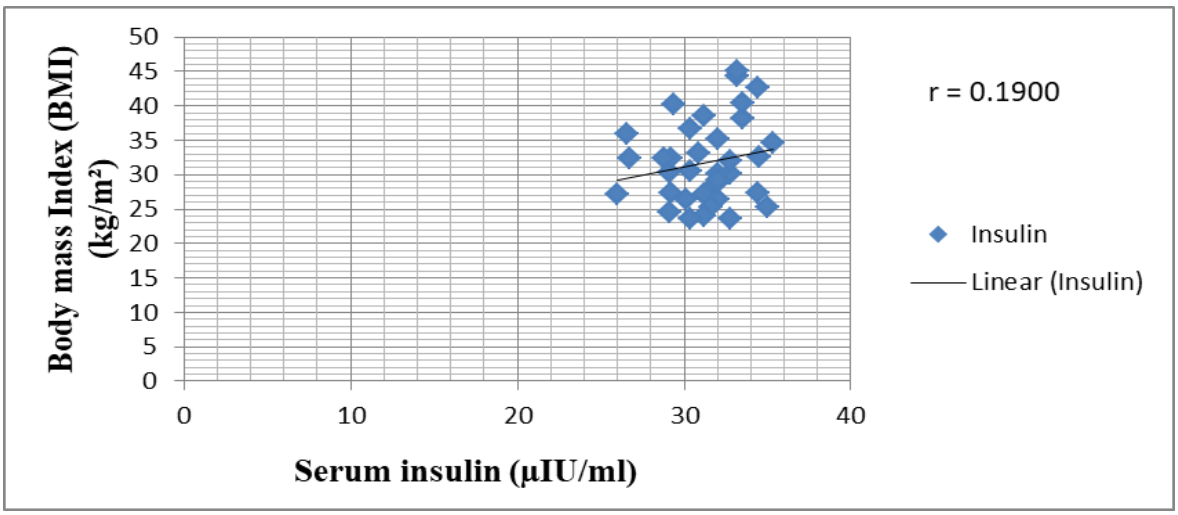

Fig. 1: Correlation between serum insulin and body mass index in obese NIDDM subjects.

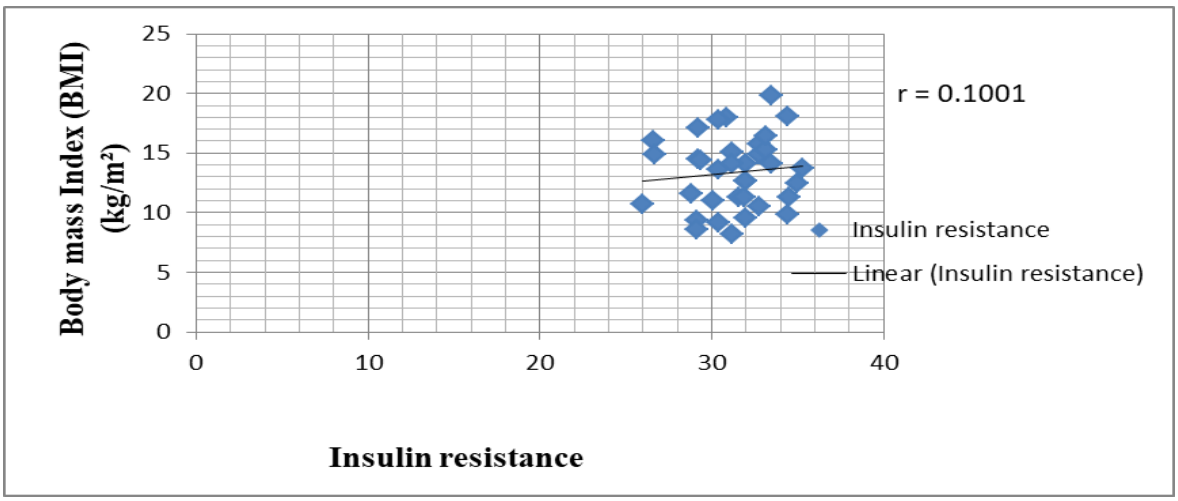

Fig. 2: Correlation between insulin resistance and body mass index in obese NIDDM subjects.

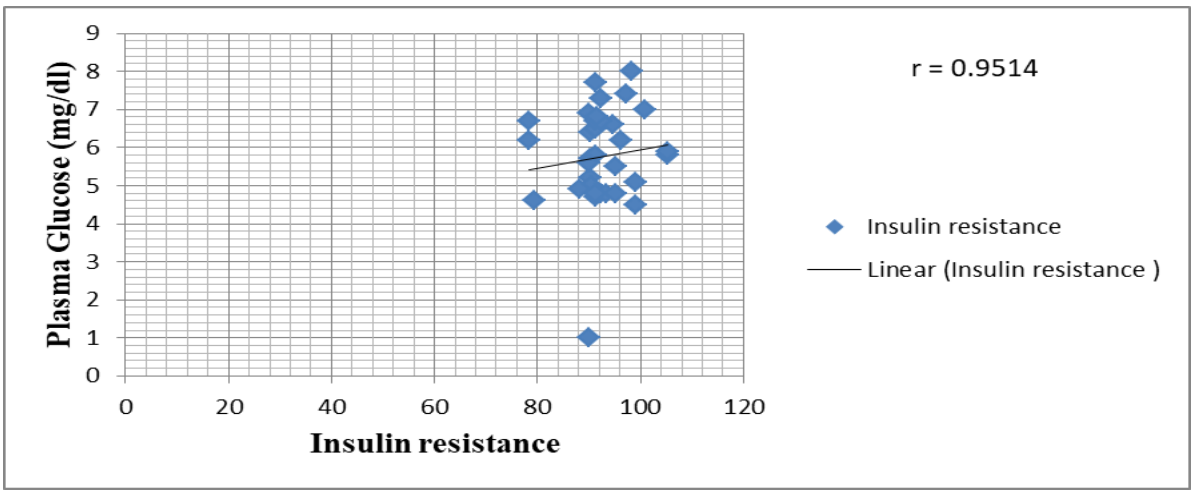

Fig. 3: Correlation between serum glucose and insulin resistance in obese subjects.

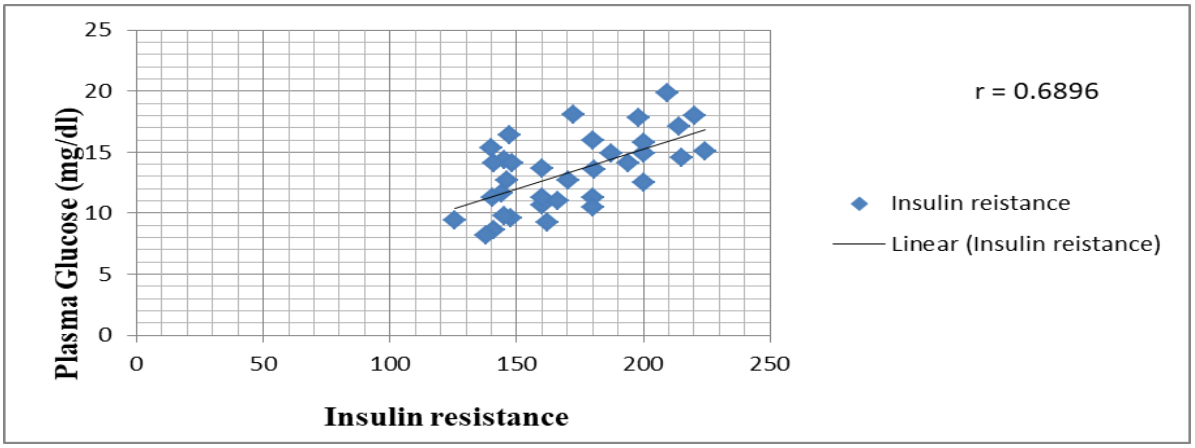

Fig. 4: Correlation between serum glucose and insulin resistance in obese NIDDM subjects.

Result

Serum glucose, insulin level and insulin resistance was higher in obese NIDDM as compared to obese subjects. There was a positive correlation between degree of obesity and insulin resistance. In present study, The mean age was 48.74 ±8.64 years and 49.46±9.86 years in the obese and obese NIDDM subjects, which varies from 34-69 and 33-64 years. A statistically non-significant change was observed in the age of obese NIDDM male and obese NIDDM female subjects. A statistically non-significant difference was observed in the age of obese NIDDM subjects (t=0.58, p=0.56) when results were compared with obese subjects [Table: 1]. Majority of the study population were females in both group. The Mean Body Mass Index (BMI) of obese and obese NIDDM subjects 30.55 ± 2.47 kg/m^2 and 31.32 ± 2.51 kg/m^2 which varies from 24.7-36.6 and 26.00-35.00 kg/m^2 respectively. A statistically non-significant was observed in the Body Mass Index (BMI) of obese NIDDM subjects (t=1.31, p= 0.94) when results were compared with obese subjects. Majority (97-100%) of the study population in both groups suffered from moderate obesity. A non-significant change in Body Mass Index (BMI) (p<0.94, t = 1.31) was observed in obese NIDDM subjects as compared to obese subjects [Table: 2]. The mean Fasting Plasma Glucose was 92.46±5.90 mg/dl and 171.07±27.53 (mg/dl) which varies from 78.23-105.26

and 125.4-224.28 in the obese subjects and obese NIDDM subjects. A statistically highly significant ($p<0.0001$, $t = 16.51$)elevation was observed in fasting plasma glucose levels in obese NIDDM subjects as compared to obese subjects [Table: 3]. The mean fasting serum insulin level was 25.32±5.72 µIU/ml and 32.12±6.19 µIU/ml which varies from 4.57-34.89 and 23.6-45.13 in the obese subjects and obese NIDDM subjects. Statistically highly significant ($t = 4.77$,$p = 0.0001$) Change was observed in the serum Insulin level in obese NIDDM subjects when results were compared to obese subjects [Table: 4]. The mean insulin resistance was 5.74±1.27 and 13.37±2.93 which varies from 1.00-7.4 and 8.2-19.8 in the obese subjects and obese NIDDM subjects. Statistically highly significant ($t = 14.13$, $p = 0.0001$) increase was observed in the serum Insulin resistance of obese NIDDM subjects as compared to obese subjects [Table: 5].

Conclusion

Hyperinsulinemia and insulin resistance are true markers for the diagnosis of metabolic syndrome and proper management of obesity at early stage helps in correcting a number of metabolic abnormalities and its sequele of complication. Overweight and obesity are posing a great threat to the health of people. Extra cautiousness with respect to diet, physical activity and metabolic derangements should be taken into consideration to reduce the incidence of this wide spread new world syndrome. Obesity management should be made a public health priority and we should focus on preventing the obesity linked diseases. India at one time had gained recognition as a traditional malnutrition country but, Indians now report more and more frequently with overweight obesity & their consequences. Obesity is a major driver in the widely prevalent metabolic syndrome and Type 2 diabetes mellitus and it is alarming to see obesity as the first wave of a defined cluster of non communicable diseases called the new world syndrome.

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