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ORIGINAL ARTICLE

Assessment of insulin resistance among type II diabetics and normal subjects

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ABSTRACT:

Background: Insulin resistance is accepted to be a major risk factor in the etiology of type 2 diabetes mellitus, hypertension, dyslipidemia, atherosclerotic vascular disease, and may be a risk factor for coronary heart disease and stroke. The present study compared IR levels newly diagnosed T2DM and normal subjects. **Materials & Methods:** 90 subjects were divided into 3 groups. Group I was subjects with NGT, group II was hyperinsulinemia with normal blood glucose tolerance (HINS) and group III was newly diagnosed T2DM. Oral glucose tolerance test (OGTT) was administered to all. Assessment of weight, BMI, SBP, DBP, TG, LDL, HDL, TC and HOMA- IR was performed in all groups. **Results:** Group I had 15 males and 15 females, group II had 13 males and 17 females and group III had 12 males and 18 females. The mean SBP (mm Hg) was 126.0, 122.6 and 128.8, DBP (mm Hg) was 76.2, 82.4 and 78.4, BMI (Kg/m2) was 24.3, 25.3 and 27.4, waist (cm) was 76.2, 88.4 and 92.2, TG (mmol/L) was 1.26, 2.08 and 1.78, LDL (mmol/L) was 2.56, 2.96 and 3.14, HDL (mmol/L) was 1.82, 1.54 and 1.08, TC (mmol/L) was 4.92, 5.28 and 4.76, Glu 0 (mmol/L) was 4.14, 4.76 and 5.26, Glu 120 (mmol/L) was 5.46, 6.23 and 13.8 and HOMA- IR was 0.96, 2.58 and 3.04 in group I, II and III respectively. The difference was significant (P< 0.05). **Conclusion:** Insulin resistance existing in newly diagnosed T2DM, subjects with normal glucose tolerance and subjects with hyperinsulinemia with normal blood glucose tolerance.

Key words: Insulin resistance, Blood glucose tolerance, Diabetes

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INTRODUCTION

Insulin resistance is accepted to be a major risk factor in the etiology of type 2 diabetes mellitus, hypertension, dyslipidemia, atherosclerotic vascular disease, and may be a risk factor for coronary heart disease and stroke as well.[1] Several risk factors (e.g. obesity, physical inactivity, body fat distribution, age and hyperinsulinemia) may be considered markers of insulin resistance. Insulin resistance is a predictor for the development of Type 2 diabetes mellitus even in individuals with normal glucose tolerance. Therefore, it is important to recognize insulin resistance in the pre-disease stage when therapeutic intervention is likely to be more successful than in manifest disease.[2] Data suggested that IR already exists before blood glucose abnormalities in diabetic patients and that hyperinsulinemia occurs before IGT shows several pathophysiological abnormalities. Therefore, several scholars have suggested that the T2DM process should be divided into the following three phases: hyperinsulinemia stage, prediabetes stage and diabetes stage. In other words, hyperinsulinemia and IGT are both reserve forces of T2DM. Hyperinsulinemia and IR are harmful even in subjects with NGT.[3] For example, several researchers have indicated that a fasting plasma insulin level (FINS) of 39 mU/mL or greater was associated with a 31% increased risk of cardiovascular events in individuals without diabetes.[4] In the transition from normal to impaired and diabetic glucose tolerance, IR is the initiating agent. When the pancreatic beta cells produce enough insulin for compensation, blood glucose is maintained in the normal range; however, when the beta cells do not produce enough insulin to compensate for IR, the blood glucose level is inevitably elevated.[5] The present study compared IR levels newly diagnosed T2DM and normal subjects.

MATERIALS & METHODS

The present study comprised of 90 subjects of both genders. All were informed regarding the study and their written consent was obtained.

Demographic data such as name, age, gender etc. was recorded. All subjects underwent a thorough physical examination. Patients were divided into 3 groups of 30 each. Group I was subjects with normal glucose tolerance (NGT), group II was hyperinsulinemia with normal blood glucose tolerance (HINS) and group III was newly diagnosed T2DM. Blood samples were obtained to determine plasma glucose and insulin concentrations before (0 min) and after (120 min) consuming a 75 g glucose drink. All underwent assessment of oral glucose tolerance test (OGTT). Assessment of weight, BMI, SBP, DBP, TG, LDL, HDL, TC and HOMA- IR was performed in all groups. Results were statistically analyzed. P<0.05 as considered significant.

RESULTS

Table I Distribution of subjects

Groups	Group I	Group II	Group III
Status	NGT	HINS	T2DM
M:F	15:15	13:17	12:18

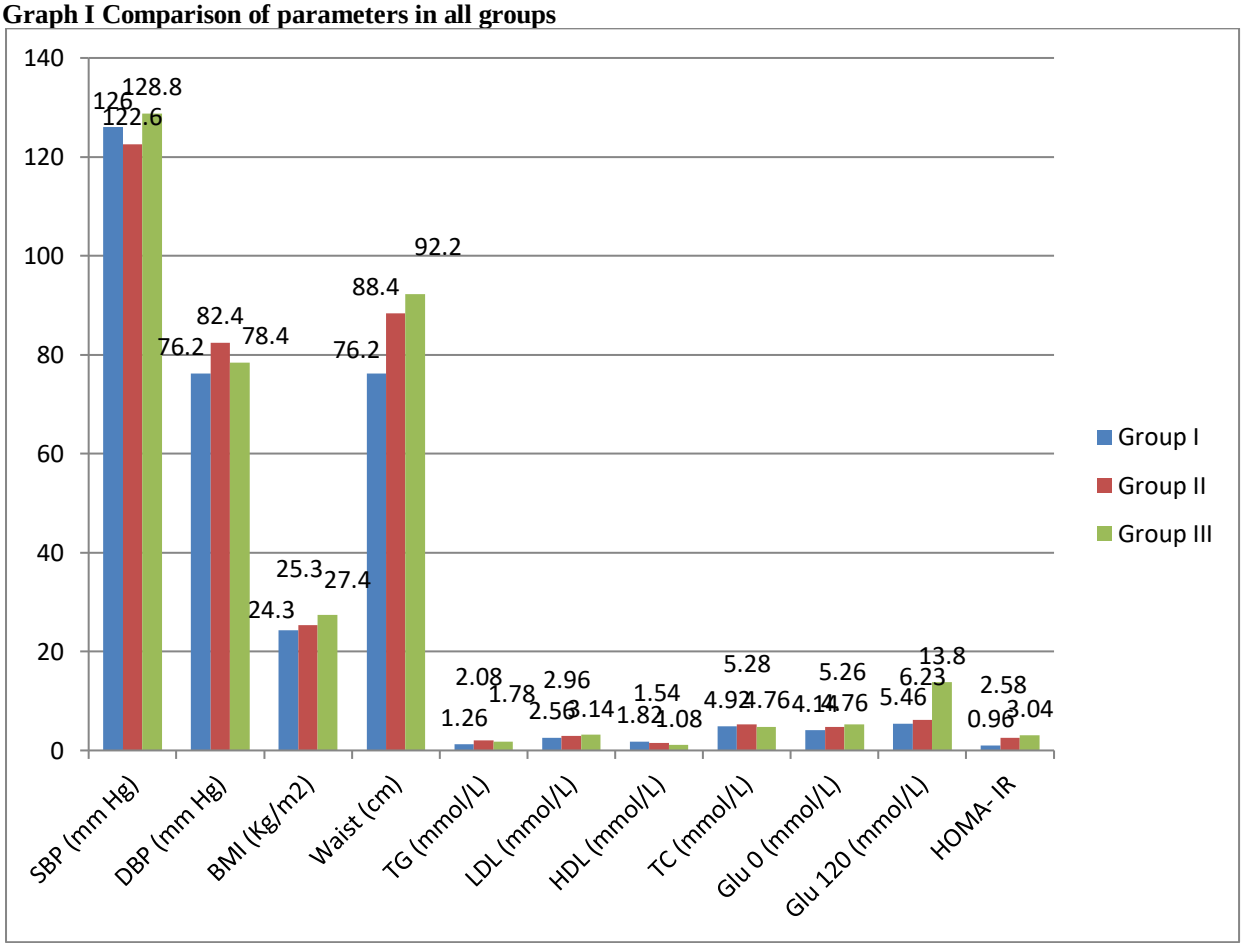
Table I shows that group I had 15 males and 15 females, group II had 13 males and 17 females and group III had 12 males and 18 females.

Table II Comparison of parameters in all groups

Parameters	Group I	Group II	Group III	P value
SBP (mm Hg)	126.0	122.6	128.8	0.84
DBP (mm Hg)	76.2	82.4	78.4	0.26
BMI (Kg/m^2)	24.3	25.3	27.4	0.17
Waist (cm)	76.2	88.4	92.2	0.94
TG (mmol/L)	1.26	2.08	1.78	0.03
LDL (mmol/L)	2.56	2.96	3.14	0.19
HDL (mmol/L)	1.82	1.54	1.08	0.12
TC (mmol/L)	4.92	5.28	4.76	0.36
Glu 0 (mmol/L)	4.14	4.76	5.26	0.05
Glu 120 (mmol/L)	5.46	6.23	13.8	0.01
HOMA- IR	0.96	2.58	3.04	0.02

Table II, graph I shows that mean SBP (mm Hg) was 126.0, 122.6 and 128.8, DBP (mm Hg) was 76.2, 82.4 and 78.4, BMI (Kg/m2) was 24.3, 25.3 and 27.4, waist (cm) was 76.2, 88.4 and 92.2, TG (mmol/L) was 1.26, 2.08 and 1.78, LDL (mmol/L) was 2.56, 2.96 and 3.14, HDL (mmol/L) was 1.82, 1.54 and 1.08, TC (mmol/L) was 4.92, 5.28 and 4.76, Glu 0 (mmol/L) was 4.14, 4.76 and 5.26, Glu 120 (mmol/L) was 5.46, 6.23 and 13.8 and HOMA- IR was 0.96, 2.58 and 3.04 in group I, II and III respectively. The difference was significant (P< 0.05).

Graph I Comparison of parameters in all groups



DISCUSSION

Hyperinsulinemic euglycemic clamp (HEC) is known to be the "gold standard" for the measurement of insulin sensitivity.[6] However, the realization that it is time and money consuming led to the development of a simplified approach in quantification of insulin sensitivity.[1] Various indices of insulin sensitivity/resistance using the data from oral glucose tolerance test (OGTT) were proposed in last 20 years.[7] Various indices of insulin sensitivity based on the interrelations between the concentration of insulin, glucose and other parameters obtained either in the fasting state or during OGTT and correlated the indices with the data obtained during a HEC have been suggested.[8] There are two groups of insulin sensitivity indices such as indices calculated by using fasting plasma concentrations of insulin, glucose and triglycerides and indices calculated by using plasma concentrations of insulin and glucose obtained during 120 min of a standard (75 g glucose) OGTT.[9,10] The present study compared IR levels newly diagnosed T2DM and normal subjects.

We observed that group I had 15 males and 15 females, group II had 13 males and 17 females and group III had 12 males and 18 females. Morino et al[11] reported a series of studies that provide evidence of a genetic mechanism linking expression of lipoprotein lipase (LPL) to peroxisome proliferator–activated receptor (PPAR)-δ expression and mitochondrial function. This is likely to contribute to the muscle insulin resistance that predisposes to type 2 diabetes.

We found that mean SBP (mm Hg) was 126.0, 122.6 and 128.8, DBP (mm Hg) was 76.2, 82.4 and 78.4, BMI (Kg/m2) was 24.3, 25.3 and 27.4, waist (cm) was 76.2, 88.4 and 92.2, TG (mmol/L) was 1.26, 2.08 and 1.78, LDL (mmol/L) was 2.56, 2.96 and 3.14, HDL (mmol/L) was 1.82, 1.54 and 1.08, TC (mmol/L) was 4.92, 5.28 and 4.76, Glu 0 (mmol/L) was 4.14, 4.76 and 5.26, Glu 120 (mmol/L) was 5.46, 6.23 and 13.8 and HOMA- IR was 0.96, 2.58 and 3.04 in group I, II and III respectively. Pyoral et al[12] showed that the most significant predictor of progression to dysglycemia was hyperinsulinemia. Hyperinsulinemia is harmful in subjects with normal or abnormal glucose tolerance. The Helsinki policemen study showed that high plasma insulin, fasting or after oral glucose load, was associated with increased risk of major CHD events independently of other conventional cardiovascular risk factors.

Kelly et al[13] suggested that increased serum insulin levels be used as a clinical marker in a primary care setting for early diagnosis and preventative care, which may be beneficial for patients at high risk of diabetes.

CONCLUSION

Authors found that insulin resistance existing in newly diagnosed T2DM, subjects with normal glucose tolerance and subjects with hyperinsulinemia with normal blood glucose tolerance.

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