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Original Article

Correlation between Quantitative Insulin Sensitivity Check Index, Homeostatic Model Assessment and Body Mass Index

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Abstract

Introduction: Normal insulin sensitivity varies widely and is influenced by age, ethnicity and obesity. The hyperinsulinemic-euglycemic clamp technique is the most scientifically sound technique for measuring insulin sensitivity and it is against this standard that all other tests are usually compared. Insulin resistance is part of a metabolic syndrome that affects multiple systems and whose key pathogenic element is hyperinsulinemia. Insulin resistance is compounded by a common occurrence of obesity although insulin resistance is also present in non-obese women.

Purpose: Weight gain is the more important as it is associated with increased risk of coronary artery disease, endometrial cancer, poly cystic ovarian disease, hypertension, and diabetes mellitus.

Materials and Methods: A total of 50 healthy women in reproductive age group of 18-34 years selected as subjects and divided into two groups based on body mass index (BMI) normal range additional cut-off points as 18.5-22.9 (Group I) and 23-24.99 (Group II). Quantitative insulin sensitivity check index and homeostatic model assessment were calculated based on fasting blood sugar and fasting insulin of the subjects in both groups.

Results: There is a significant increase with $P < 0.0001$ in fasting insulin levels with a decrease in insulin sensitivity and increase in insulin resistance as the BMI increases.

Conclusion: As BMI increases insulin sensitivity decreases and insulin resistance increases.

Key words: Body mass index, Fasting insulin, Homeostatic model assessment, Insulin resistance, Insulin sensitivity, Quantitative insulin sensitivity check index

INTRODUCTION

The WHO consensus group concluded that insulin sensitivity of the lowest 25% of a general population can be considered as insulin resistant. As the clamp techniques are expensive, time-consuming and not very practical in office setting, the fasting state homeostatic assessment methods have been developed, which help to evaluate insulin resistance or insulin sensitivity in a clinical setting. Increased waist to hip ratio compounded by increased body mass index (BMI) is called android obesity. It is associated with insulin resistance as it is more sensitive to catecholamines and less sensitive to insulin and is associated with glucose intolerance, diabetes, increased androgen production rate, decreased testosterone-estradiol binding globulin, increased free levels of testosterone and estradiol, dyslipidemia hypertension and cardiovascular risk factors and breast cancer.[1-3]

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IR may be due to genetic or acquired causes like defect in receptors, number of receptors or postreceptor mechanism at various stages-abnormalities of insulin signaling pathway, increased activity of protein tyrosine phosphatase which can attenuate insulin signaling by internalization and

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dephosphorylation of the receptor, mutations affecting insulin receptor numbers, splicing, trafficking, binding, and phosphorylation.[4-6]

In skeletal muscle, GFAT carries out rate limiting step of hexose monophosphate pathway, i.e., conversion of fructose 6-p and glutamine to glucosamine 6-phosphate and glutamate. By a pathway that is unclear, glucosamine overproduction results in a disruption of the ability of insulin to cause translocation of glucose transporter type 4 to the cell surface resulting in hyperglycemia which in turn causes hyperinsulinemia.[7-11]

Hyperinsulinemia causes insulin resistance by downregulating insulin receptors by receptor internalization and desensitizing post-receptor pathways. Weight gain increases free fatty acids (FFA) and triglyceride levels. Reduction in insulin-stimulated insulin receptor substrate-1 associated PI3-kinase may play a role in pathogenesis of insulin resistance.[12]

MATERIALS AND METHODS

We had selected 50 healthy women in reproductive age group of 18-34 years as subjects from out-patient clinics in Warangal. The subjects were divided into two groups based on their BMI by taking into consideration of BMI normal range additional cut-off points as18.5-22.9 (Group I) and 23-24.99 (Group II).

Each subject was informed in detail of its objectives, the aims of the thesis protocol and the methods to be used. Their consent was obtained in the history. Any past or present intake of steroids is ruled out.

About 5 ml of blood sample was collected from a cubital vein in the morning after overnight fasting between 8 am and 9 am for the estimation of fasting blood sugar and fasting insulin. All the information obtained was recorded in case sheet pro forma and later analysis was done.

Flow-injection (FI) was estimated by chemiluminiscence method which is a type of immunoassay that uses certain compounds that emit light as they return from the activated to the resting state. In all the cases, the biochemical reaction leads to the formation of the electronically excited state which on decaying to the ground state emits photons of energy which are converted to light energy. The emitted light energy is quantified in three ways namely-peak light intensity, decay part integration and total light production.

BMI was calculated after measuring height in cm and weight in kg.

$$\text{BMI} = \frac{\text{wt (kg)}}{\text{Ht}^2\text{ (m)}}$$

Among the many mathematical homeostatic models available two indices - homeostatic model assessment (HOMA) and quantitative insulin sensitivity check index (QUICKI) were selected, calculated, and compared between the two groups.

QUICKI can be applied to normoglycemic and hyperglycemic patients and is believed to be superior to HOMA as a way of determining insulin sensitivity although the two values correlate well. As the HOMA value decreases QUICKI value increases.

HOMA is frequently used to assess insulin resistance.

$$\text{QUICKI} = \frac{1}{[\log\text{FBS (mg/dl)}] + [\log\text{FI (}\mu\text{u/ml)}]}$$

$$\text{HOMA} = \frac{\text{FI (}\mu\text{U/ml)} \times \text{FBS (mmol/l)}}{22.5}$$

For conversion of blood sugar from mg/dl to mmol/l following formula was used:

- S.I. (mmol/l) = CF × C (mg/dl)
- When CF = 0.05551

Low HOMA values indicate high insulin sensitivity, whereas high HOMA values indicate low insulin sensitivity (insulin resistance).

RESULTS

The mean BMI in Groups I and II is 21.314 and 23.954, respectively, with $P < 0.0001$. There is a significant increase in BMI in Group II when compared to Group I.

The mean fasting insulin in Groups I and II is 4.724 and 9.772, respectively. There is a significant increase in FI in Group II when compared to Group I with $P < 0.0001$.

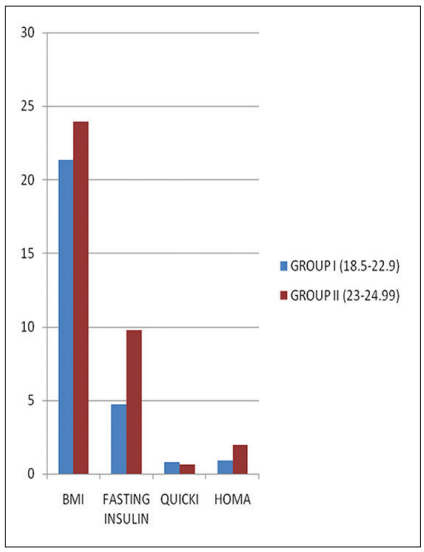
The mean value for QUICKI in Groups I and II is 0.882 and 0.64 with $P < 0.0001$. There is a significant increase in Group II when compared to Group I.

The mean value for HOMA in Groups I and II is and 1.954 with $P < 0.0001$. There is a significant decrease in Group II when compared to Group I (Table 1).

Table 1: Comparison of insulin sensitivity and resistance

Parameter	(*n*=25) (mean±SD)	
	Group I	Group II
BMI (kg/m^2)	21.348±0.732	23.954±1.954
Fasting insulin (μU/l)	4.724±1.743	9.772±5.116
QUICKI	0.812±0.140	0.64±0.0746
HOMA	0.882±0.343	1.954±1.049

HOMA: Homeostatic model assessment, QUICKI: Quantitative insulin sensitivity check index, BMI: Body mass index, SD: Standard deviation



DISCUSSION

Weight gain is an independent factor for insulin resistance, and there is more marked dysregulation of insulin levels and impairment of insulin sensitivity in women with more BMI which may lead to hyperinsulinemia.

The insulin sensitivity and resistance in Group II in our study probably suggest post receptor defect or genetic factor causing more severe insulin resistance in addition to obesity. The raised insulin levels are able to dispose the blood sugar and maintain normal levels. These patients become overt diabetics only when the pancreas is exhausted or there is a coexisting defect in β cells of islets along with insulin resistance.[13-16]

A normal phase of insulin resistance appears due to obesity leading to increased serum fasting insulin levels to maintain normal glucose disposal. Due to interplay of genetic factors, obesity and postreceptor insulin defects the phase of insulin resistance becomes aggravated and persistent leading to endocrine and metabolic derangement.

"Hyperinsulinemia" by itself causes insulin resistance by downregulating insulin receptor internalization and desensitizing postreceptor pathways. Insulin being antilipolytic and lipogenetic factor causes more abdominal fat deposition, aggravating obesity.[17,18]

The clinical presentation of patients with insulin resistance depends on the ability of the pancreas to compensate for the target tissue resistance to insulin. During the first stages, when compensation is effective the only metabolic abnormality is hyperinsulinemia. Later on, when β cells fail to meet the challenge, the decreased insulin levels lead to impaired glucose tolerance test and frank diabetes.[19,20]

CONCLUSION

BMI is an independent risk factor for insulin sensitivity and resistance that worsens the condition when imposed on other factors. Weight gain also induces atherosclerosis due to increased circulating FFA and triglycerides imposing more strain on the heart to maintain circulation.

1. Evaluation of insulin sensitivity, resistance rather than a single insulin value is a more sensitive indicator. The concept of insulin sensitivity and resistance is relatively easy to understand but determining precisely is more complicated. The relationship between glucose and insulin is quite complex and involves the interaction of many metabolic and regulatory factors. Weight control, diet modification like high fiber diet and low-fat diet with an increase in monounsaturated fats, lifestyle changes, exercise and screening with oral glucose tolerance test are to be considered for early recognition and intervention.

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