Assessment of insulin resistance among obese people at Shendi locality, Sudan

ABSTRACT

Insulin resistance (IR) is an essential factor in the etiology of type2 diabetes and is likewise related to a big range of different pathophysiologic sequelae along with hypertension, hyperlipidemia. we carried out this cross-sectional examination to the evaluation of insulin resistance in obese people. A total of 60 obese people 20% males and 80% female were divided into 4 groups overweight (20.0%), obesity class1(36.6%), obesity class2(16.6%), and obesity class3(26.6%). Blood samples were obtained from each participant and analyzed for fasting blood glucose (FBG) as well as insulin levels. Data were analyzed by using SPSS version 20. The results showed a positive correlation between insulin resistance and insulin levels (mean 1.1+0.91, 7.1+10.1, r=0.490, *P. value*= 0.0001 respectively). There was no correlation between insulin resistance and FBG (mean 1.1+0.91, 75.7+10.9, r=0.52, *P. value* = 0.691 respectively). The analysis also showed a significant positive correlation between insulin resistance and Waist-to-hip Ratio (WHR) (mean 1.1+0.91,0.92+.14, r=0.287, *P. value* =0.026 respectively). Additionally, there was no correlation between insulin resistance and BMI (mean 1.1+.91,36.6+9.92, r= 0.122, *P. value* =0.351 respectively). There was no correlation between insulin resistance and age (mean 1.1+.91, 30 +8.68, r=-0.154, *P. value* = 0.30 respectively). The analysis also showed a significant between insulin resistance and gender, males (mean 0.85+1.26) and females (mean 1.1+.91,0.64+0.97) which *P. value*=0.031. We conclude that insulin resistance in obesity has a positive correlation with fasting insulin levels and waist-to-hip ratio. Insulin resistance was more prevalent in males more than in females.

Keywords: Insulin resistance, Obesity, C-peptide, β-cell, Body Mass Index.

Introduction

Obesity is a clinical circumstance wherein extra-frame fats have gathered to the extent that they can harm fitness, main to a decrease in life expectancy and/or multiplied fitness issues. people are taken into consideration as obese while their Body Mass Index, a degree that compares their weight and square height, exceeds 30 kg / m2 **[1].** Obesity is mostly caused by extra power consumption relative to power expenditure, the etiology of weight problems is highly complicated and entails complex and

involves genetic, physiological, environmental, psychological, and even financial elements that interact to various tiers to contribute to the improvement of obesity **[2].** Insulin resistance(IR) refers to the condition in which peripheral tissues are relatively insensitive to the effects of hormones. IR plays an important role in improving and progressing Cardiometabolic risk factors (CMRF), which is part of the metabolic syndrome of obesity (e.g., type 2 diabetes, dyslipidemia, hypertension, fatty liver, and cardiovascular disease) **[3].** As a regular disease in obesity and type 2 diabetes, insulin resistance is characterized as a condition wherein cells fail to reply to insulin, mainly to the improvement of hyperglycemia **[4].** Insulin resistance visible in obesity is an idea to particularly consist of muscle and liver, with increased adipocyte-derived free fatty acids promoting triglyceride accumulation in those tissues **[5].** Insulin resistance develops pathologically through many interactions between genotypic lifestyles, especially sedentary lifestyles, and binge eating **[6].** Physiologically, many circulatory elements regulate insulin sensitivity in target tissues, including adipokines, plasma lipids, circulatory hormones, and, their signaling pathways **[7].** By modulating the insulin sensitivity of the target tissue, there is an axis of the animal tissue indicator system in which the brain and gut monitor the insulin response **[8].** Adipokines are internal secretions secreted by adipocytes, whether they significantly stimulate or inhibit insulin sensitivity. In peripheral tissues, insulin action is stimulated by leptin and adiponectin, while TNFα, IL-6, and retinol-binding protein 4 inhibit insulin sensitivity **[9].** Responses and nutrition are regulated by adipocyte-derived factors, but lifestyle, obesity, and genes can upset this balance **[10].** Levels of inhibitory adipokines and circulating fatty acids have been shown to increase with obesity **[7].** Homeostatic model assessment and insulin resistance become first defined in 1985 **[11].** This method may be a technique for assessing B cell function and IR from basal glucose and insulin or C-peptide concentrations. The model has been widely utilized since it had been initially printed, and we present here and summary of the model and its appropriate use and limitations in clinical science **[11].** Homeostatic model assessment (HOMA) is used to obtain estimates of insulin sensitivity and B cell function from fasting plasma insulin and glucose concentrations **[11].** The basal glucose-insulin ratio reflects the balance between hepatic glucose output and hormone secretion maintained by the electrical circuit between the liver and B cells **[12].** HOMA is a glucose-insulin dynamics relationship model that predicts rapid steady-state glucose and insulin concentrations for various possible combinations of insulin resistance and β-cell function. HOMA uses a series of simple formulas to describe this glucose-insulin homeostasis. The approximation of resistance to hypoglycemic agents has been simplified. Use a quick blood test. This is the result of using the insulin-

glucose product divided by a constant **[13].** HOMA-IR breakpoints for prominent metabolic syndrome can vary by population and body mass index (BMI). We aim to investigate the HOMA insulin resistance breakpoints that best distinguish between insulin-resistant and metabolic syndrome individuals in each BMI class in many examples of nondiabetic adults **[14].** HOMA may give different cell functions or IR estimates than the minimal model. It should be noted that HOMA can live on basal insulin sensitivity and B cell performance, and unlike clamps, it is not always intended to provide data on stimulation status **[15].** HOMA-IR may help in the early assessment of insulin resistance in all probabilistic populations and may provide long-term usefulness for prophylactic and diagnostic therapeutic interventions **[16].** Therefore, this examines pursuits to assess insulin resistance amongst overweight human beings in Shendi state from august to November 2021.

Materials and Methods

The present study was a descriptive cross-sectional study, conducted, in Shendi city in River Nile State in Northern Sudan. Sixty (60) obese people were enrolled in the study, twelve (12) of them were over-weight, twenty-two (22) were in obesity class 1, ten (10) were in obesity class 2, and, sixteen (16) were obesity class 3.

Their ages are 18 to 60 years old.

Fasting blood samples were collected using a dry plastic syringe, a tourniquet was used, five (5) ml of blood was collected, and equal volumes of blood were dispensed into fluoride oxalate and heparin containers. Fluoride-oxalate blood is separated as fast as feasible by centrifuging at 4000 rpm at room temperature to obtain plasma. Blood in a heparin container was centrifuged at 4000 rpm at room temperature to obtain heparinized plasma and stored at -20°C. Plasma glucose and Insulin level was measured by using Cobas 411, Cobas 311 according to the manufacturer instructions.

Data Analysis

All collected data were analyzed using SPSS for Windows, version 16. Paired Student t-test was used for calculating the degree of variation, with P *.value* (≤ 0.05) considered as significant. Analysis of variance (ANOVA) was used for continuous data and the statistical results were presented as means ± SD.

Results

60 obese people 20% males and 80% female were divided into 4 groups overweight (20%), obesity

class1(36.6%), obesity class2(16.6%), and obesity class3(26.6%). Statistical analysis showed a positive correlation between insulin resistance and insulin levels (mean 1.1+.91, 7.1+10.1, r=0.490, *P. value*= 0.0001 respectively). There was no correlation between insulin resistance and FBG (mean 1.1+.91, 75.7+10.9, r=0.52, *P. value* = 0.691 respectively).The analysis also showed a significant positive correlation between insulin resistance and WHR (mean 1.1+0.91,0.92+.14, r=0.287, *P. value* =0.026 respectively). Additionally, there was no correlation between insulin resistance and BMI (mean 1.1+.91,36.6+9.92, r= 0.122, *P. value* =0.351 respectively).There was no correlation between insulin resistance and age (mean 1.1+.91, 30 +8.68, r=-0.154, *P. value* = 0.30 respectively) (Table 1). The analysis also showed a significant between insulin resistance and gender, males (mean 0.85+1.26)) and females (mean 1.1+.91,0.64+0.97) which *P. value*=0.031 (Table 2). And there is no correlation between insulin resistance and residences, rural (mean1.16+1.05) urban (mean 0.70+1.15) which *P. value*=0.670 (Table 2). The analysis also showed no significant between insulin resistance and family history of o, with FH (mean1.15+1.08) and without an FH (mean 1.06+0.71) (Table 2). No correlation between insulin resistance and social status and educational level (Table 3).

Discussion

Obesity has emerged as an internationally toxic disease and will increase the threat of many diseases, especially insulin resistance, type 2 diabetes, and cardiovascular disease **[17].** This study found a positive correlation between insulin resistance and insulin. This discovery was confirmed by research by Morales and his team in Venezuela. A significant correlation was found between insulin and HOMA-IR concentrations **[18].** This is expected because the HOMA-IR value is derived from insulin concentration. In this study, there was a significant increase in mean levels of insulin resistance in men compared to women. This finding is consistent with Badri's cross-sectional study in the United States. They observed that men struggled to improve insulin sensitivity compared to women with comparable weight loss and baseline HOMA-IR **[19].** Probably due to the gender difference in visceral fat. This may also mean that insulin-resistant men require more intervention than women to stop the progression of metabolic syndrome or polygenic disease. We also found that there was no correlation between insulin resistance and FBG. This finding contradicted them in developing Asian countries: a moderate positive correlation between fasting insulin and HOMA-IR (P <0.05) **[20].** This variation in results means that blood glucose levels may appear normal even in the early stages of insulin resistance. Therefore, the blood glucose test is not always a reliable test for insulin resistance. There was no correlation between the insulin resistance index (HOMA-IR) and age and BMI, which was inconsistent

with the studies conducted by Pang and his team on the Chinese population. They found that FBG, insulin, and HOMA-IR scores decreased with age (P<0.001) and increased with BMI **[21].** This change leads to changes in physical activity and nutritional status or determines changes in body composition. It was also observed that there was a significant correlation between insulin resistance and WHR. This is similar to the results of the study in China. WHR was significantly associated with IR (P <0.001) **[22].** It was found that there was no statistical significance between insulin resistance and family history. This finding is consistent with a study conducted in Messina, Italy. Other prevalence of FH in arterial hypertension, type 2 diabetes, and coronary heart disease **[23].** This difference may be due to genetic variation between populations in the two countries. There is no statistical significance between insulin resistance and educational levels (*P. value* = 0.706). This finding contradicts the cross-sectional study conducted in Mexico **[24].** between insulin and residence, with a *P. value* of 0.670. This finding was inconsistent with a cross-sectional study of significantly higher fasting blood glucose, fasting insulin, and HOMA-IR levels in urban subjects. Had more than local subject (P <0.001) **[25].** This discrepancy is a possible duo of environmental and lifestyle qualities. In addition, there was no statistical significance between insulin resistance and social status (*P. value* = 0.672). This is inconsistent with the cross-sectional study conducted by Bermudez and his colleagues in 2016. They said the marriage situation needed to be linked to IR, which was more common among married people. Most of them are men, inactive, with an increase in BMI and WC, explaining that married men are at increased risk of overweight and obesity associated with eating more often and abundantly. Married persons with a *P. value* = .001 are associated with significant IR **[26].** This variation can be due to differences in living standards, education, or family relationships.

Conclusion

The insulin resistance in obesity is raised by an increased level of fasting insulin and the waist-hip ratio, men's more insulin resistance than women, no correlation between insulin resistance and BMI, fasting blood glucose, age, education level, place of residence, and family history of obesity.

Ethical Approval and Consent

Ethical approval for the study was obtained from the Board of the Faculty of Graduates Studies at Shendi University. The written informed consent form was obtained from each guardian of the participant as well as from the subject himself before recruitment into the study. All protocols in this study were done according to the Declaration of Helsinki (1964).

Declarations

Availability of data and materials

The datasets used and/or analyzed during the current study are available upon reasonable request from the corresponding author.

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Table 1: The correlation between FBG, Insulin, IR, age, BMI, and WHR:

Parameter		INSULIN	FBG	BMI	WHR	Age
IR	P. V	0.0001	0.691	0.351	0.026	0.30

	R	0.490	0.052	0.122	0.287	0.281
INSULIN	P. V R	------	0.151 0.188	0.654 0.059	0.370 0.118	0.465 0.96
FBG	P. V R	0.151 0.188	-------	0.515 0.086	0.688 0.53	0.958 0.007

Table 2: The mean and St.d of Insulin resistance according to gender, Residence, and family history among case group:

Variables		No	Mean	St.d	Mean difference	Sig (2-tailed)
Gender	Female	48	0.970	0.644	0.640	**0.031**
	Male	12	1.260	0.852	0.640	
Residence	Urban	35	1.150	0.700	0.970	**0.670**
	Rural	25	1.052	1.160	0.970	
Family history	With FH	30	1.153	1.081	0.900	**0.706**
	Without FH	30	1.063	0.718	0.900	

Table 3: Comparison between types of social status and levels of education with insulin resistance among case group:

Variables IR		Sum of squares	Mean Squares	Sig
Social status	Between groups Within groups	0.594 48.412	0.297 0.849	0.706
levels of education	Between groups Within groups	1.702 47.304	0.567 0.849	0.672