

Original Research

“STUDY ON CORRELATION OF INSULIN RESISTANCE AND LDL IN PATIENTS WITH OBESITY”

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Abstract

Introduction: Obesity has turned into a worldwide epidemic. In the last decades, the number of obese patients has increased considerably. It is especially alarming that in recent years the increase was most pronounced in children and that it occurs both in developed, but perhaps even more, in developing countries. The aim of the study is to estimate the levels of LDL and calculate the insulin resistance using HOMA-IR formula in Obese Subjects.

Materials and methods: We enrolled a total of 100 obese subjects with BMI >30 kg/m² and 100 healthy controls with BMI 18-24 kg/m². A fasting blood sample was collected for the estimation of lipid profile (total cholesterol, TAG, LDL and HDL) and fasting insulin levels. HOMA-IR was calculated using the formula fasting insulin x fasting glucose/405.

Results and Discussion: In the present study it is quite evident that the HOMA IR values are significantly increased in obese subjects compared to healthy controls. The mean and SD values of total cholesterol, LDL, HDL and triglycerides are significantly increased in obese subjects compared to healthy controls.

In obese subjects 58% had elevated LDL-C, 55% had elevated total cholesterol, 62% had decreased HDL-C and 53% had elevated TAG levels. We found positive correlation existed between HOMA-IR and LDL levels in obese subjects. Elevated levels of LDL, smoking, elevated blood pressure and type 1 and type 2 diabetes, are well known risk factors for CVD, however, insulin resistance, hyperglycaemia and inflammation can also lead to and predict adverse cardiovascular events. Furthermore, insulin resistance is related to disorders such as hypertriglyceridemia as well as low levels HDL.

Conclusion: In the present study it is quite evident that the HOMA IR values are significantly increased in obese subjects compared to healthy controls. The mean and SD values of total cholesterol, LDL, HDL and triglycerides are significantly increased in obese subjects compared to healthy controls. In obese subjects 58% had elevated LDL-C, 55% had elevated total cholesterol, 62% had decreased HDL-C and 53% had elevated TAG levels. We found positive correlation existed between HOMA-IR and LDL levels in obese subjects.

Key-words: fasting insulin, insulin resistance, low density lipoproteins, cholesterol and body mass index.

INTRODUCTION

Obesity has turned into a worldwide epidemic. In the last decades, the number of obese patients has increased considerably. It is especially alarming that in recent years the increase was most pronounced in children and that it occurs both in developed, but perhaps even more, in developing countries [1]. Visceral obesity leads to insulin resistance in part mediated by adipokines and free fatty acids (FFA). Adipokines such as resistin and retinol-binding protein 4 decrease insulin sensitivity, whereas leptin and adiponectin have the opposite effect. In addition, cytokines like TNF- α and IL-6, which originate from macrophages in adipose tissue, are involved [2]. Obesity, especially central obesity, is probably the main cause of the metabolic syndrome (MetS), which includes insulin resistance, type 2 diabetes mellitus, hypertension, the obstructive sleep apnea syndrome, non-alcoholic fatty liver disease (NAFLD) and dyslipidemia, all risk factors for cardiovascular disease [3,4]. Although doubts have arisen about the significance of the term metabolic syndrome in relation to cardiovascular complications, it has been suggested that identifying the condition will stimulate the physician to search also for the other risk factors clustering in the MetS [5]. The typical dyslipidemia of obesity consists of increased triglycerides (TG) and FFA, decreased HDL-C with HDL dysfunction and normal or slightly increased LDL-C with increased small dense LDL. The concentrations of plasma apolipoprotein (apo) B are also often increased, partly due to the hepatic overproduction of apo B containing lipoproteins [6,7]. Insulin resistance is known to be major risk factor in the etiology of type 2 diabetes mellitus, hypertension and dyslipidemia and may be a risk factor for coronary heart disease (CHD). The homeostasis model assessment of insulin resistance (HOMA-IR), one of the indirect indices for the measurement of insulin resistance correlates well with euglycemic clamp measurement in men and women, younger and older adults, obese and non-obese individuals. HOMA-IR is currently being proposed by investigators as a useful index of insulin sensitivity, particularly in epidemiological studies. Hence we have taken up this study to determine the levels of LDL and calculate insulin resistance in obesity subjects.

AIM AND OBJECTIVES:

The aim of the study is to estimate the levels of LDL and calculate the insulin resistance using HOMA-IR formula in Obese Subjects.

MATERIALS AND METHODS:

Source of data and place of study: The present study was conducted in the Dept. of General Medicine our hospital. This was a cross-sectional study.

Sample size: we included a total 200 subjects (100 obesity subjects and 100 healthy controls) during the study period.

Inclusion criteria: we included 200 patients in the age group between 20-50 years presenting to General Medicine Department OPD of our hospital with obesity.

Exclusion criteria: we excluded the patients with type 2 diabetes, anti-diabetic medications, hypothyroidism and PCOS.

Data Collection and Methodology: We enrolled a total of 100 obese subjects with BMI >30 kg/m² and 100 healthy controls with BMI 18-24 kg/m². A fasting blood sample was collected for the

estimation of lipid profile (total cholesterol, TAG, LDL and HDL) and fasting insulin levels. HOMA-IR was calculated using the formula fasting insulin x fasting glucose/405.

RESULTS:

Table 1: Shows demographic data and clinical data of the study subjects (n=200)

	Obese subjects	Controls
Age	38.6±9.48	39.6±10.2
Males/females	126/74	122/78
BMI (kg/m ²)	21.6±2.4	33.6±8.48
HOMA-IR	5.98±1.82	2.22±0.23
Total Cholesterol	232.2±12.23	186.5±15.23
LDL Cholesterol	138.4±8.8	98.6±14.32
HDL Cholesterol	48.4±9.6	49.34±10.23
Triglycerides	162.6±10.2	138.4±8.66

Table 2: Frequency of lipid derangements in obese subjects (n=100)

	Number	Percentage
LDL Cholesterol		
>=100 mg/dL	58	58%
<100 mg/dL	42	42%
Total Cholesterol		
>200 mg/dL	55	55%
<200 mg/dL	45	45%
HDL Cholesterol		
<50 mg/dL	62	62%
>50 mg/dL	38	38%
Triglycerides		
>=150 mg/dL	53	53%
<150 mg/dL	47	47%

DISCUSSION:

In the present study it is quite evident that the HOMA IR values are significantly increased in obese subjects compared to healthy controls. The mean and SD values of total cholesterol, LDL, HDL and triglycerides are significantly increased in obese subjects compared to healthy controls. In obese subjects 58% had elevated LDL-C, 55% had elevated total cholesterol, 62% had decreased HDL-C and 53% had elevated TAG levels. We found positive correlation existed between HOMA-IR and LDL levels in obese subjects.

Insulin resistance is defined as an experimental or clinical condition in which insulin exerts a biological effect lower than expected. This phenomenon is due to marked defects in the insulin-stimulated glucose uptake, particularly, in glycogen synthesis and, to a lesser extent, glucose oxidation. The effects of insulin resistance in different tissues depend on the physiological as well as metabolic function of the tissues. Due to their high metabolic demand insulin resistance has significant effects on skeletal muscle, adipocytes and liver tissue, which are the main targets of intracellular glucose transport as well as glucose and lipid metabolism [8]. Skeletal muscle and adipocytes accounts for about 60–70% and 10% of insulin-stimulated glucose uptake respectively via the GLUT 4 receptors. Insulin resistance cause impaired glycogen synthesis and protein catabolism in skeletal muscles and inhibit lipoprotein lipase activity in adipocytes leading to an increased release of free fatty acids and inflammatory cytokines such as IL-6, TNF α , and leptin. Additionally, the liver accounts for 30% of insulin-stimulated glucose disposal and insulin resistance leads to impaired glucose output and fatty acid metabolism leading to increased triglyceride content and VLDL secretion from liver [9]. Insulin resistance causes endothelial cell

dysfunction by decreasing the production of nitric oxide from endothelial cells and increasing the release of pro-coagulant factors leading to platelet aggregation. In an insulin resistant state, the PI3K pathway is affected whereas the MAP kinase pathway is intact, which causes mitogenic effect of insulin in endothelial cells leading to atherosclerosis [10].

Elevated levels of LDL, smoking, elevated blood pressure and type 1 and type 2 diabetes, are well known risk factors for CVD, however, insulin resistance, hyperglycaemia and inflammation can also lead to and predict adverse cardiovascular events. Furthermore, insulin resistance is related to disorders such as hypertriglyceridemia as well as low levels HDL. Additionally, insulin resistance has been found in approximately 30% of subjects with a diagnosis of hypertension. In 1996, investigators in the Insulin Resistance Atherosclerosis Study (IRAS), showed a direct relation between insulin resistance and atherosclerosis and a follow-up prospective study in a cohort of 2938 patients reported insulin resistance as an important risk factor for CVD. A 2012 meta-analysis of 65 studies, which included 516,325 participants, revealed that insulin resistance, evaluated by HOMA index, was a good predictor for CVD [11-13].

CONCLUSION:

In the present study it is quite evident that the HOMA IR values are significantly increased in obese subjects compared to healthy controls. The mean and SD values of total cholesterol, LDL, HDL and triglycerides are significantly increased in obese subjects compared to healthy controls. In obese subjects 58% had elevated LDL-C, 55% had elevated total cholesterol, 62% had decreased HDL-C and 53% had elevated TAG levels. We found positive correlation existed between HOMA-IR and LDL levels in obese subjects.

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