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Association Between Insulin Resistance and Inflammation in Obese Individuals

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ABSTRACT

Obesity is a very common condition we can see in most of the people living in developing countries. Most obese cases are due to their lifestyle only. Obesity will affect other metabolisms and causes metabolic disorders like diabetes, renal damage and cardiovascular problems. 30 obese patients and 30 healthy individuals from the OP of Saveetha Dental College. Serum samples were estimated the FBS by GOD-POD Method, CRP by Turbilatex Method using ERBA CHEM 5 plus analyzer. Insulin level by ELISA method using ROBONIK ELISA READER. There is a remarkable increase in FBS ($p<0.005$), Insulin ($p<0.005$), Insulin resistance ($p<0.005$) and highly remarkable increase in C reactive protein ($p<0.001$) in obese people compared to controls. Our results show that the increase in insulin resistance, insulin, sugar and inflammation states there is a potential risk of diabetes, renal damage and other metabolic abnormalities due to the obese condition.

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INTRODUCTION

The prevalence of obesity has reached epidemic ratios in today's World (Finucane, M.M *et al.,* 2011). Obesity is not only the global disease burden of cardiovascular disease and cancer but also the main risk predictor for the rapid increase in type 2 diabetes (T2D) (Chen. L. *et al.,* 2012; Calle, E.E *et al.,* 2003; Hubert, H.B *et al.,* 1983). Obesity is said to be due to a chronic energy imbalance which involves both dietary intake and physical activity patterns (Swinburn B *et al.,* 2011). Adipose tissue is an important endocrine organ which helps in producing hormones such as adiponectin and leptin. They play a role in satiety and metabolism. Meanwhile, they also have effects on the immune system of the body. In obesity, the changes are noticed in both the release of these hormones and insensitivity of various organs and tissues to their effects (Weisberg SP *et al.,* 2003; Xu H *et al.,* 2003). Obesity and it is associated with cardiovascular, metabolic and renal disorders have drastically become a considerable threat to the world population. Worldwide obesity has nearly doubled since 1980 and current estimates specify that more than 1.4 billion adults are overweight or obese. (Obesity and Overweight Fact Sheet *N°311. 2014.*)

Obesity is a hazardous factor for evolving type 2 diabetes and cardiovascular illness and has rapidly turned into an overall pandemic with few tangible and safe treatment choices. (Brian E. Sansbury *et al.,* 2014). Obesity elevates the risk for type 2 diabetes through the initiation of insulin resistance (Jianping Ye, 2013). Insulin brings down the blood glucose level by initiating glucose uptake in insulin-sensitive tissue like skeletal muscle, fat and heart. It also inhibits glucose production in liver, kidney and small intestine in control, of blood glucose. Insulin resistance tissue loss response to insulin. (Ye J, 2007; Hall JE *et al.,* 2003). Majority of the outcome of being overweight or obese comprise a higher prevalence of hypertension and a cascade of associated cardiorenal and metabolic

disorders. Studies in diverse populations throughout the world brings out the relationship between BMI and systolic and diastolic blood pressure (BP) is nearly linear (Jones DW *et al.,* 1994)

Acute phase C-reactive protein (CRP) increased in obesity and inflammation is a vital binding protein for leptin. It is thought that CRP helps in leptin resistance by preventing leptin from crossing the blood-brain barrier (BBB) (Jianping Ye, 2003) C-reactive Protein (CRP) measurements above 10 mg/L have been usually considered as acute inflammation and eliminated from epidemiologic studies of chronic inflammation. CRP elevations above 10 mg/L in obese women are likely to be from chronic rather than acute inflammation, and that CRP thresholds above 10 mg/L may be warranted to differentiate acute from chronic inflammation in obese women. (Hung Hsuchou, *et al.,* 2012, Shinya Ishii1, *et al.,* 2012). The study is concentrated on the relationship between the insulin resistance and the inflammation in obese which leads to various metabolic disorders and renal damage.

METHODS AND MATERIALS

Patients were selected from those attending the outpatient department of Saveetha Dental College, and hospitals and divided into two groups as follows

Group I – Normal healthy individuals with normal BMI (19-24.9) – 30 individuals

Group II – Obese Individuals with BMI- (30-40) – 30 individuals

Inclusion Criteria

Individuals with the age group of twenty to thirty years

Obese Individuals

Exclusion Criteria

Individuals with other systemic illness like diabetes mellitus, cardiovascular disease, Renal failure, Stroke, endocrine illness.

Individuals with an acute illness like a fever.

Immunocompromised individuals

Sample collection Procedure

Informed consent was obtained from the patient before sample collection. 5ml of venous blood was collected and distributed in plain collection tubes and centrifuged in 3000rpm for 10mins. Then serum was separated and used to estimate the FBS by GOD-POD Method, CRP by Turbilatex Method using ERBA CHEM 5 plus analyzer. Insulin level by ELISA method using ROBONIK ELISA READER.

Insulin resistance was calculated by using HOMA-IR calculation.

<1 indicates Optimal / No insulin resistance

>1.9 SpecifiesEarly Insulin Resistance

>2.9 specifies remarkable Insulin Resistance

RESULTS

Table 1: Mean, SD and Significance value of BMI, FBS, Insulin, IR and CRP in two groups

Parameters	Controls	Obese patients	p-Value
BMI	21.62 ± 2.09	35.63 ± 4.54	<0.005*
FBS	86.8 ± 7.56	103.8 ± 18.5	<0.005*
Insulin	3.43 ± 1.48	7.7 ± 1.77	<0.005*
IR	0.74 ± 0.33	1.97 ± 0.53	<0.005*
CRP	2.93 ± 1.17	7.58 ± 1.41	<0.001**

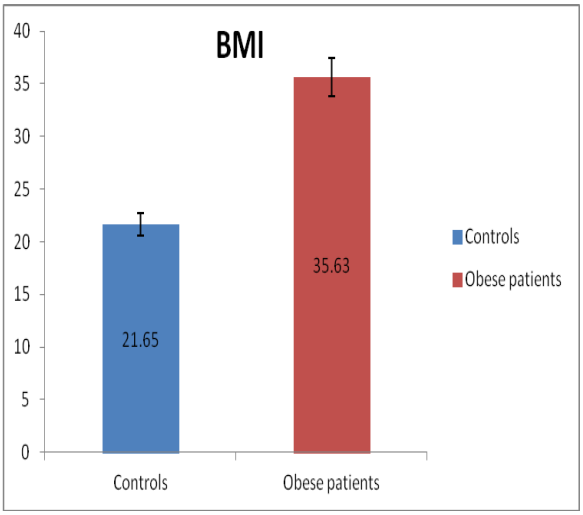

Figure:1 The BMI value of obese

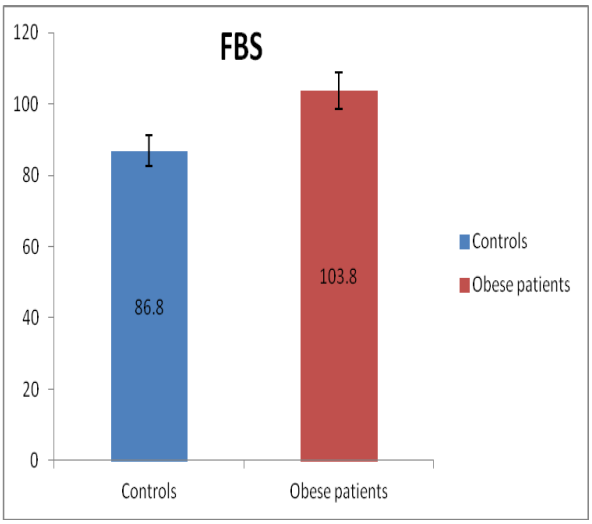

Figure 2: The FBS values of obese and non-obese individuals

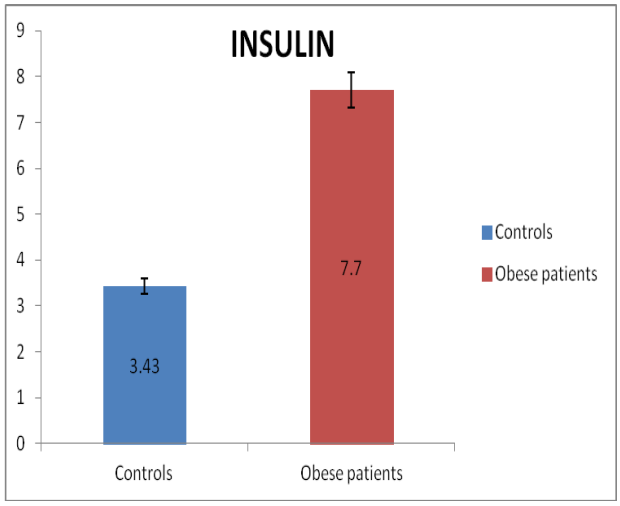

Figure 3: The graph between the insulin values of obese and non- obese individuals.

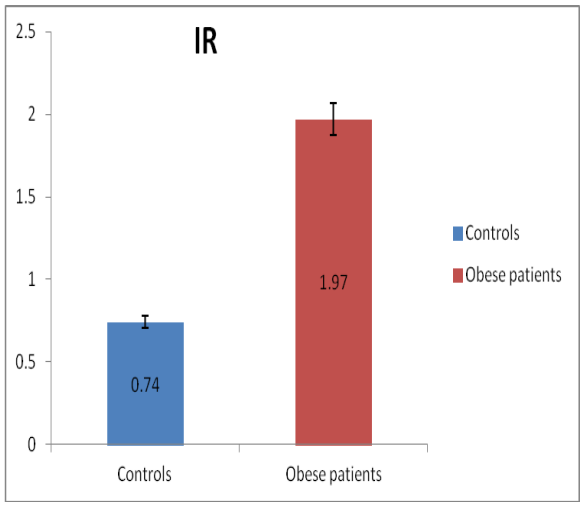

Figure 4: The graph between the IR values of obese and non- obese individuals.

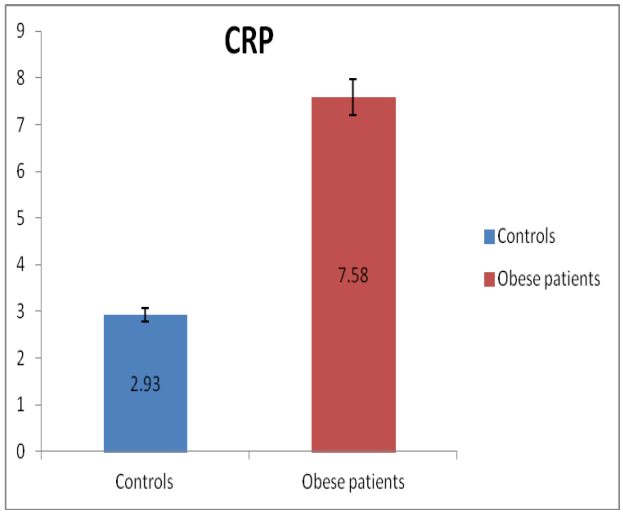

Figure 5: The graph between the CRP values of obese and non - obese individuals.

DISCUSSION

Our results in Table-1showing there is a significant increase in FBS 103.8 ± 18.5 of Obese people when compared to healthy controls 86.8 ± 7.56, the significance value is p<0.005. There is a significant increase in insulin 7.7 ± 1.77 for Obese people when compared to healthy controls 3.43 ± 1.48. The significance value is p<0.005. Relates to insulin there is a significant increase in insulin resistance 1.97 ± 0.53of Obese people when compared to healthy controls 0.74 ± 0.33, the significance value is p<0.005. In order to these, there is a very highly significant increase in inflammatory markers in obese 7.58 ± 1.41 when compared to controls 2.93 ± 1.17. The significance value is p<0.001.

Obese patients have an increase in fasting blood sugar (FBS) level when compared to non- obese patients. In the case of BMI, obese patients have an increased value when compared to non - obese patients. When we talk about insulin status, obese individuals have an increased insulin level when compared to others. Insulin resistance (IR) is a pathological condition in which cells fail to function normally to the hormone insulin. Insulin resistance precedes the development of type 2 diabetes. When the insulin resistance parameter was taken into the study, it was found that obese patients have an increased insulin resistance level when compared with non- obese patients. Hence there is a high risk for type 2 diabetes in obese individuals. C-reactive protein (CRP) is a blood test marker for inflammation in the body. CRP is synthesized in the liver and its extent is evaluated by testing the blood. CRP is considered as an acute phase reactant, which means that its measure will rise in response to inflammation. Other common acute phase reactants also include the erythrocyte sedimentation rate (ESR) and blood platelet count. When the CRP test was taken, it showed a high value in obese patients than in non- obese individuals. Hence, from this, we can conclude that obesity leads to inflammation.

The increase in renal sodium reabsorption acts as an important role in inducing the increase in Blood Pressure associated with a surplus gain of weight and obese. They are subjected to acquire higher than normal blood pressure to maintain sodium balance, showing impaired renal-pressure natriuresis (Hall JE 1997). Increased retroperitoneal and visceral fat may lead to a rise in blood pressure by compressing the kidneys physically. Excess accumulation of fat in and around the kidneys is interlinked with intrarenal pressures, impaired pressure natriuresis, and hypertension (Hall ME *et al.,* 2014). In patients with visceral obesity, intra-abdominal pressure increases in proportion to sagittal abdominal diameter, reaching extent as high as 35–40 mmHg (Sugerman H *et al.,* 1997). These show the hazardous effects of obesity on the kidney. Some studies talk about the relationship

between the excess gain of weight or obesity with reduced nighttime sleep. In contrast, other studies showed a relationship between long sleep duration, both at night and during the day, and excess body weight (Vgontzas AN *et al.,* 2008).

CONCLUSION

In this study, various parameters were taken into account with obesity**.** It was found that the BMI, FBS, insulin, insulin resistance, CRP were high in obese patients when compared to non-obese individuals. This may lead to various problems like diabetes, renal failure, metabolic abnormalities.

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