Original Article

ASSESSMENT OF LEPTIN AND LIPID PROFILE IN OBESE SUBJECTS

Smita Sadhwani

Assistant Professor, Department of Biochemistry, F.H. Medical College Tundla, Firozabad, Uttar Pradesh, India

Background: Obesity is a multifaceted condition and represents a pandemic that needs urgent attention. Leptin most likely indicates satiety and fullness of energy stores under physiological conditions, but obesity is characterized by hyperleptinemia and hypothalamic leptin resistance. The present study was conducted to assess the relation between leptine hormone disturbance and lipid level disorders in obese. **Materials & Methods:** It comprised of 156 subjects of both genders. All were divided into 3 groups. Group I (Control) comprised of 56 subjects with BMI (18.5-24.9 kg/m^2). Group II (Overweight) consisted of 55 with BMI (25.0–29.9 kg/m^2). Group III (Obese) consisted of 45 with BMI (≥ 30 kg/m^2). Serum Leptin was measured by ELISA. Serum GSH, serum MDA and serum lipid profile were measured by spectrophotometer. **Results:** BMI (kg/m^2) in group I was 23.21±1.2, in group II was 26.72±1.5 and in group III was 35.46±1.1. The difference was significant (P-0.01). Leptin level (ng/ml) in group I was 12.22±1.8, in group II was 27±2.5 and in group III was 33±6.3. The difference was significant (0.03). GSH (µmol/L) level in group I was 4.1±1.0, in group II was 3.25±2.1 and in group III was 1.6±1.2. The difference was significant (0.01). MDA (µmol/L) level in group I was 7.9±1.5, in group II was 12.662±1.4 and 14.12±2.4 in group III. The difference was significant (0.001). Cholesterol level was 4.2 mmol/l in group I, 5.1 mmol/l, in group II and 5.8 mmol/l in group III. Triglyceride level was 1.5 mmol/l in group I, 2.3 mmol/l in group II and 3.1 mmol/l in group III. HDL level was higher in group I (2.3 mmol/l), group II (1.6 mmol/l) and group III (1.08 mmol/l). LDL level was lower in group I (1.4 mmol/l), group II (2.7 mmol/l) and group III (3.6 mmol/l). VLDL level was 0.8 mmol/l in group I, group II (1.2 mmol/l) and group III (1.5 mmol/l). Group III had higher VLDL level. **Conclusion:** There was a significant increase in the levels of leptin. There was a significant increase in the levels of BMI, MDA, TC, triglycerides, LDLC and VLDL and decrease in the level of GSH and HDL in overweight and obese individuals.

Key words: Cholesterol, Leptin, Obesity.

Received: 22-08-2014 Revised: 04-09-2014 Accepted: 05-09-2014

Corresponding Author: Dr. Smita Sadhwani, Assistant Professor, Department of Biochemistry, F.H. Medical College Tundla, Firozabad, Uttar Pradesh, India

This article may be cited as: Sadhwani S. Assessment of leptin and lipid profile in obese subjects. J Adv Med Dent Scie Res 2014;2(3):224-227.

INTRODUCTION

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have a negative effect on health. People are generally considered obese when their body mass index (BMI), a measurement obtained by dividing a person's weight by the square of the person's height, is over 30 kg/m^2, with the range 25–30 kg/m^2 defined as overweight. Some East Asian countries use lower values. Obesity increases the likelihood of various diseases and conditions, particularly cardiovascular diseases, type 2 diabetes, obstructive sleep apnea, certain types of cancer, osteoarthritis and depression.[1]

Obesity is a multifaceted condition and represents a pandemic that needs urgent attention. Leptin most likely indicates satiety and fullness of energy stores under physiological conditions, but obesity is characterized by hyperleptinemia and hypothalamic leptin resistance. Many studies have found association between obesity leading to oxidative stress and diabetes mellitus type 2, and many others have shown

that the level of ROS increase in obesity. Glutathione is a master antioxidant cellular defense, prevent damage to the important components caused by ROS such as free radicals and peroxide.[2]

Obesity is most commonly caused by a combination of excessive food intake, lack of physical activity, and genetic susceptibility. A few cases are caused primarily by genes, endocrine disorders, medications, or mental disorder. The view that obese people eat little yet gain weight due to a slow metabolism is not generally supported. On average, obese people have a greater energy expenditure than their normal counterparts due to the energy required to maintain an increased body mass.[3]

Oxidative stress is an imbalance between oxidant and antioxidant pathways that result in the accumulation of lipid oxidation products such as lipid hydro peroxides and malondialdehyde. These materials are toxic and cause increased risk of arteriosclerosis in the blood by other lipoproteins. In addition, increased oxidative stress in adults after exercise increases. It has been shown that aerobic exercise reduces oxidative stress in obese men.[4] The present study was conducted to assess the relation between leptine hormone disturbance and lipid level disorders in obese.

MATERIALS & METHODS

This study was conducted in the department of biochemistry. It comprised of 156 subjects of both genders. All were informed regarding the study and written consent was obtained. Ethical clearance was taken from institutional ethical committee.

All were divided into 3 groups. Group I (Control) comprised of 56 subjects with BMI (18.5-24.9 kg/m^2). Group II (Overweight) consisted of 55 with BMI (25.0–29.9 kg/m^2). Group III (Obese) consisted of 45 with BMI (≥ 30 kg/m^2).

About 5 ml of venous blood were withdrawn from all subjects using a disposable syringe after 12-hour fasting. The collected blood was then allowed to clot in a plain tube at room temperature, after which the serum was separated by centrifugation at 3000 rpm for 10 min, and kept frozen at -20°C to be analyzed later on. Serum Leptin was measured by ELISA. Serum GSH, serum MDA and serum lipid profile were measured by spectrophotometer. Results were tabulated and subjected to statistical analysis using chi- square test. P value <0.05 was considered significant.

RESULTS

Table I Distribution of subjects

Total- 156		
Group I (control)	Group II (overweight)	Group III (obese)
56	55	45

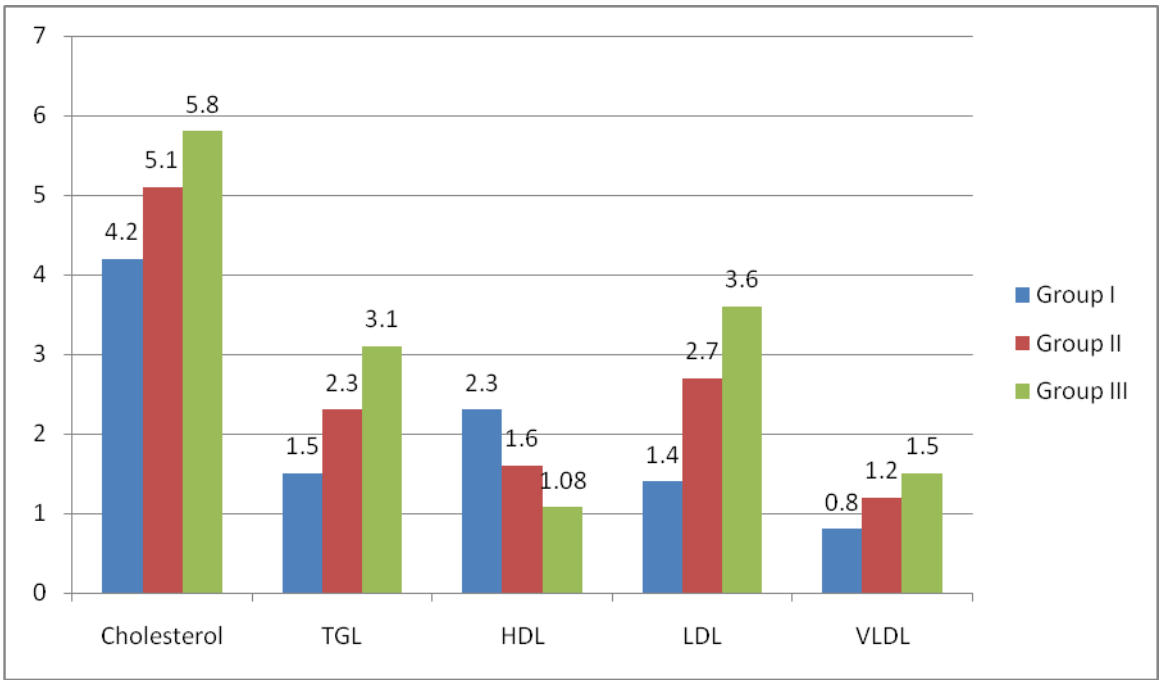
Table I shows that group I (Control) comprised of 56 subjects, group II (Overweight) consisted of 55 subjects and group III (Obese) consisted of 45 subjects. The difference was significant (P<0.05).

Table II Comparison of parameters

Parameters	Group I	Group II	Group III	P value
BMI (kg/m^2)	23.21±1.2	26.72±1.5	35.46±1.1	0.01
Leptin (ng/ml)	12.22±1.8	27±2.5	33±6.3	0.03
GSH (µmol/L)	4.1±1.0	3.25±2.1	1.6±1.2	0.01
MDA (µmol/L)	7.9±1.5	12.662±1.4	14.12±2.4	0.001

Table II shows that BMI (kg/m^2) in group I was 23.21±1.2, in group II was 26.72±1.5 and in group III was 35.46±1.1. The difference was significant (P-0.01). Leptin level (ng/ml) in group I was 12.22±1.8, in group II was 27±2.5 and in group III was 33±6.3. The difference was significant (0.03). GSH (µmol/L) level in group I was 4.1±1.0, in group II was 3.25±2.1 and in group III was 1.6±1.2. The difference was significant (0.01). MDA (µmol/L) level in group I was 7.9±1.5, in group II was 12.662±1.4 and 14.12±2.4 in group III. The difference was significant (0.001).

Graph I Cholesterol & Lipid profile in subjects



Graph I shows that cholesterol level was 4.2 mmol/l in group I, 5.1 mmol/l, in group II and 5.8 mmol/l in group III. Triglyceride level was 1.5 mmol/l in group I, 2.3 mmol/l in group II and 3.1 mmol/l in group III. HDL level was higher in group I (2.3 mmol/l), group II (1.6 mmol/l) and group III (1.08 mmol/l). LDL level was lower in group I (1.4 mmol/l), group II (2.7 mmol/l) and group III (3.6 mmol/l). VLDL level was 0.8 mmol/l in group I, group II (1.2 mmol/l) and group III (1.5 mmol/l). Group III had higher VLDL level.

DISCUSSION

Obesity is a leading preventable cause of death worldwide, with increasing rates in adults and children. In 2015, 600 million adults (12%) and 100 million children were obese. Obesity is more common in women than men. Obesity is stigmatized in much of the modern world (particularly in the Western world), though it was seen as a symbol of wealth and fertility at other times in history and still is in some parts of the world.[5] In this study we assessed leptin, cholesterol and lipid level in obese subjects.

We divided subjects into 3 groups depending upon their BMI. Group I (Control) comprised of 56 subjects, group II (Overweight) consisted of 55 subjects and group III (Obese) consisted of 45 subjects.

We evaluated BMI level in al groups. The BMI is an attempt to quantify the amount of tissue mass (muscle, fat, and bone) in an individual, and then categorize that person as underweight, normal weight, overweight, or obese based on that value. However, there is some debate about where on the BMI scale the dividing lines between categories should be placed.[6] Commonly accepted BMI ranges are underweight: under 18.5 kg/m^2, normal weight: 18.5 to 25, overweight: 25 to 30, obese: over 30. We observed that in obese subjects BMI was significantly higher as compared to other groups. This is similar to Sedlak.[7]

Leptin level (ng/ml) in group III was 33±6.3 which is higher than other groups. This is in agreement with Check et al. There are many possible pathophysiological mechanisms involved in the development and maintenance of obesity. This field of research had been almost unapproached until the leptin gene was discovered in 1994 by J. M. Friedman's laboratory. These investigators postulated that leptin was a satiety factor. In the ob/ob mouse, mutations in the leptin gene resulted in the obese phenotype opening the possibility of leptin therapy for human

obesity. However, soon thereafter J. F. Caro's laboratory could not detect any mutations in the leptin gene in humans with obesity.[8] On the contrary Leptin expression was increased proposing the possibility of Leptin-resistance in human obesity. Since this discovery, many other hormonal mechanisms have been elucidated that participate in the regulation of appetite and food intake, storage patterns of adipose tissue, and development of insulin resistance. Since leptin's discovery, ghrelin, insulin, orexin, PYY 3-36, cholecystokinin, adiponectin, as well as many other mediators have been studied. The adipokines are mediators produced by adipose tissue; their action is thought to modify many obesity-related diseases.[9]

We assessed GSH (µmol/L) level and found that it was higher in group I as compared to other groups. MDA (µmol/L) level in group II was higher than other groups. We found that cholesterol level was higher in group III than other groups. Similarly triglyceride level was higher in group III. HDL level was higher in group I (2.3 mmol/l), group II (1.6 mmol/l) and group III (1.08 mmol/l). Group III had higher LDL and VLDL level. This is in agreement with Allian.[10]

CONCLUSION

We observed that there was a significant increase in the levels of leptin. There was a significant increase in the levels of BMI, MDA, TC, triglycerides, LDLC and VLDL and decrease in the level of GSH and HDL in overweight and obese individuals.

REFERENCES

1. Chandra A, Biersmith M, Tolouian R Obesity and kidney protection. J Nephropathol. 2011; 3: 91-97.
2. Martin SS, Qasim A, Reilly MP Leptin resistance: a possible interface of inflammation and metabolism in obesity-related cardiovascular disease. J Am Coll Cardiol. 2008; 52: 1201–1210.
3. Schinzari F, Tesauro M, Rovella V, Di Daniele N, Mores N, et al. Leptin stimulates both endothelin-1 and nitric oxide activity in lean subjects but not in patients with obesity-related metabolic syndrome. J Clin Endocrinol Metab. 2011; 98: 1235–1241.
4. Galletti F, Delia L, De Palma D, Russo O, Barba G, et al. Hyperleptinemia is associated with hypertension systemic inflammation and insulin resistance in overweight but not in normal weight men. Nutrition Metabolism and Cardiovascular Diseases. 2010; 22: 300–306.
5. Kawakita S, Kitahata H, Oshita S. Glutathione level estimation in obese individuals. World J Gastroentirol. 2009; 15: 4137-4142.
6. Pastore A, Ciampalini P, Tozzi G, Pecorelli L, Passarelli C, et al. All glutathione forms are depleted in blood of obese and type 1 diabetic children All Diabetes. 2010; 13: 272-277.
7. Sedlak J, Lindsay RH. Estimation of total protein-bound and nonprotein sulfhydryl groups in tissue with Ellman's reagent. Anal Biochem. 1968; 25: 192-205.
8. Vincent HK, Bourguignon C, Vincent K. Resistance training lowers exercise-induced oxidative stress and homocysteine levels in overweight and obese older adults. Obesity. 2006; 14: 1921-1930.
9. Guidet B, shah SV. Enhanced in vivo H2O2 generation by rat kidney in glycerol-induced renal failure. Am J. Physiol. 2004; 257: 440-445.
10. Allian CC. Estimation of serum cholesterol. Clin Chemistry. 1974; 25: 470-475.

Source of support: Nil

Conflict of interest: None declared