Leptin, Adiponectin and Resistin Concentration in Obesity Class I and II at Sardjito Hospital Yogyakarta

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ABSTRACT

Aim: to determine the level of different concentration of leptin, adiponectin and resistin among obesity class I and class II population.

Methods: cross sectional study was conducted from June 2006 until January 2007 on 57 patients with obese diabetic and non diabetic Native Javanese. They were divided into obese class I (body mass index [BMI] >25 kg/m² to <30 kg/m²) and obese class II (BMI >30 kg/m²). Leptin, adiponectin and resistin level concentration were measured.

Results: leptin concentration in obese class I was 13,998 ± 13,486 ng/ml; adiponectin was 3.98 ± 1.78 µg/ml; resistin were 25.676 ± 13.434 ng/ml. Leptin concentration in obese class II was 31,074 ± 26,158 ng/ml; adiponectin 4,75 ± 1,88 µg/ml; resistin 25,46 ± 12,26 ng/ml. Leptin concentration was significantly higher in obese class II than obese class I (p=0,002) and there was a positive weak correlation between BMI and leptin level, with Spearman correlation coefficient correlation 0.363 (p=0.006)). Adiponectin and resistin concentration was not significantly higher in obese class II than obese class I (p=0,156 and p=0,956).

Conclusion: leptin concentration in obese class II was significantly higher than in obese class I but adiponectin and resistin were not different.

Key words: leptin, adiponectin, resistin, obesity class I and II.

INTRODUCTION

Adipocytes produce and secrete several proteins that act as veritable hormones, responsible for the regulation of energy intake and expenditure, known as adipocytokines. Adipocytokines include leptin, adipisin, TNF-α, adiponectin and resistin. Their dysregulation now is known to be involved in the etiology of insulin resistance. Leptin affects energy homeostasis by inhibiting food intake and stimulating energy expenditure. Both leptin synthesis and circulating levels are increased in obese patients and are correlated with the fat mass in a chronic manner, most obese patients are leptin resistant. Adiponectin is inversely correlated with leptin, its plasma levels are significantly reduced in obese subjects, in insulin-resistant subjects, and in type 2 diabetic patients and increased after weight reduction. Two independent case-control studies in healthy caucasians and in Pima Indians indicate that low plasma adiponectin levels are associated with an increased risk of type 2 diabetes. Resistin has been implicated in the pathogenesis of obesity-mediated insulin resistance and type II diabetes mellitus (T2DM). Human studies have highlighted increased resistin expression in adipose tissue, particularly abdominal depots, furthermore, positive correlation between serum resistin and body fat content has also been reported.

Studies have shown different concentration of leptin, adiponectin and resistin in obese and non obese patient. The aim of this study was to know different concentration of leptin, adiponectin and resistin in obesity class I and obesity class II.

METHODS

A cross-sectional study was conducted from June 2006 until January 2007 among 57 obese native Javanese patients suffering from type 2 diabetes and non diabetes
patients. Research subjects were chosen by consecutive sampling. Inclusion criteria of our study BMI >25 kg/m² and aged 22 until 60 years old. Exclusion criteria were used in this study: type 2 diabetes patients used insulin therapy or non diabetes used oral drugs i.e. glucocorticoid, isoproterenol, β-adrenergik receptor agonist, androgens, estrogens, PPAR-γ agonist, and smoking. All subjects were measured for height, weight and collected 10 cc for venous blood sample. They were divided into obese class I if body mass index (BMI) >25 kg/m² to <30 kg/m² and obese class II if BMI >30 kg/m². Leptin, adiponectin and resistin were measured with ELISA method, we also check lipid profile: cholesterol, HDL-cholesterol, LDL-cholesterol and triglyceride.

Student t-test was used for statistic analysis which has normal distribution. When the data had an abnormal distribution, we transformed it using Log 10 in order to have a normal distribution. For normal distribution, we used student t-test, while Mann Whitney U test was used if the data still have abnormal distribution after transformation.

RESULTS

Subjects who went into the study were 57 persons, 24 persons (42.11%) were male and 33 persons (57.89%) were female. Twenty nine persons suffered from type 2 diabetes and 28 persons non diabetes, divided into obese class I means body weight were 27.96±1.28 kg and obese class II means body weight were 33.99±3.72 kg, 41 subjects were obese class I and 16 subjects were obese class II.

Cholesterol concentration in obese class I was 223.56±41.52 mg/dl; LDL-cholesterol 142.90±32.78 mg/dl; HDL-cholesterol 53.02±9.89 mg/dl; triglyceride 174.83±92.01 mg/dl. Cholesterol concentration in obese class II 210.50±33.38 mg/dl; LDL-cholesterol 127.31±29.33 mg/dl; HDL-cholesterol 51.25±7.18 mg/dl, triglyceride 213.25±139.15 mg/dl. Statistically there were no significant lipid profiles on obese class I and obese class II.

Leptin concentration in obese class I was 13,998±13,486 ng/ml; adiponectin 3.98±1.78 µg/ml; resistin 25.676±13.434 ng/ml. Leptin concentration in obese class II was 31.074±26.158 ng/ml; adiponectin 4,75±1,88 µg/ml resistin 25.46±12.26 ng/ml. Leptin concentration was significantly higher in obese class II than obese class I (p=0.003). Adiponectin and resistin concentration was not different in obese class II than obese class I (p=0.516 and p =0.956) (Table 2).

Non parametric test with Spearman correlation showed a positive weak correlation between BMI and leptin level (coefficient correlation 0.363; p=0.006).

DISCUSSION

The results of our study indicate that leptin, adiponectin and resistin in obese patient as classifying obese class I and obese class II were different only on leptin concentration.

Adiponectin is a novel peptide expressed specifically, abundantly in adipose tissue and the levels are lower in obese (defined by BMI) than in non obese subjects. It is unknown whether adiponectin is related to direct measures of abdominal obesity (e.g. visceral fat and subcutaneous abdominal fat) which is known to be associated with insulin resistance. Several studies have examined plasma adiponectin levels in humans and have found decreased levels in obese and diabetic subjects and significant inverse associations with some measure of insulin resistance. In this study we examined concentration of adiponectin in non diabetic subjects and type 2 diabetic subjects with varying degrees of obesity and were classified obese class I and obese class II. We found that plasma adiponectin concentration was not significantly different with degrees of obesity.

Resistin is a member of the newly discovered family of cysteine-rich secretory proteins called “resistin-like...
molecules”. Studies in rodents suggest that resistin is up-regulated in obesity and participating in the pathogenesis of insulin resistance. However, studies in humans have been controversial. The majority of the studies regarding resistin regulation have performed analyzing the mRNA adipose tissue expression in different models of rodents with insulin resistance or in adipose tissue from obese and type 2 diabetic patients. In this study, we examined resistin concentration in non diabetic subjects and type 2 diabetic subject with varying degrees of obesity and were classified obese class I and obese class II. We found that the plasma resistin concentration was not significantly different compared with the obesity.

Leptin the product of the “ob” gene may be one of such factor. This adipocyte-derived hormone exerts pleiotropic effects including profound effects on satiety, energy expenditure and neuroendocrine function. The proposed role for rising leptin as a strong (adipostatic) signal to prevent obesity is easily subverted by leptin resistance. Leptin synthesis and circulating levels are increased in obese patients and are correlated with the fat mass in a chronic manner, most obese patients are leptin resistant.3,10,12,13

Obesity is associated with leptin resistance as evidenced by hyperleptinemia and studies showed that hyperleptinemias correlated with hypertension and cardiovascular risk in obesity.14-17 In this study, we examined leptin concentration in non diabetic subjects and type 2 diabetic subjects with varying degrees of obesity and were classified obese class I and obese class II. We found that plasma leptin concentration had significance different with degrees of obesity and there was positive weak correlation between BMI and leptin level with Spearman correlation (coefficient correlation 0.363 (p=0.006)).

Leptin resistance arises from impaired leptin transport across the blood-brain barrier (BBB) defects in leptin receptor signaling and blockades in downstream neuronal circuits. The mediator of this resistance is unknown. Several mechanisms contributing to leptin resistance have been identified so far. First, a saturable transporter allowing leptin to pass the blood–brain barrier was discovered. This mechanism impedes the hormone’s transfer to the hypothalamus once certain plasma concentrations are exceeded. Secondly, negative regulators of leptin signalling such as the tyrosine phosphatases protein tyrosine phosphatase 1B and SH-containing protein tyrosine phosphatase as well as the suppressors of cytokine signalling (SOCS) have been shown to contribute to leptin resistance.18-22

Other studies showed that the leptin receptor gene (LEPR) polymorphism Q223R is associated with circulating leptin levels and one of the most common in the general population, is thought also to be associated with an impaired signaling capacity of the leptin. The genotype and allele frequencies of the LEPR p.Q223R variant were significantly different between the normal weight and obese groups.23-26

CONCLUSION

Leptin concentration in obese class II was significantly higher than in obese class I, but adiponectin and resistin were not different.

REFERENCES