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ORIGINAL ARTICLE

Serum apelin levels and metabolic risk markers in obese women

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KEYWORDS

Obese;
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Abstract *Background:* Adipose tissue hormones, Adipokines, play an important role in obesity-associated complications. Apelin has recently been added to the family of adipokines. **The aim** of this study was to evaluate the relationship between serum apelin levels and metabolic abnormal parameters in Egyptian obese women.

Materials and methods: The study included 400 unrelated women; they were 200 obese women and 200 non- obese matched healthy women. All participants underwent clinical, anthropometric and biochemical examinations. Insulin resistance (IR) was determined by the homeostasis model assessment of insulin resistance (HOMA-IR). Serum apelin levels and obesity biomarkers were measured using enzyme-linked immunoassay (ELISA) kits. Fat mass was measured by Tanita Body Composition Analyzer.

Results: Obese women showed significant higher levels of serum apelin, leptin, triglycerides, LDL-C, total cholesterol, fasting insulin HOMA-IR and blood pressure levels than controls. Significant positive correlations between apelin and leptin levels with abnormal metabolic markers were noted in obese women.

Conclusion: The present study suggests the significant role that might be mediated by apelin for developing abnormal metabolic parameters among Egyptian obese women.

1. Introduction

An excess of body fat leads to obesity, which is a chronic and complex disease. The increased prevalence of obesity in adults, adolescent and children make it one of the principal public health problems [1]. Several obesity-related comorbidities are the results of an increase in the incidence of obesity and caused by adipose tissue hormones called adipokines [2]. Adipokines family includes leptin, adiponectin, and resistin and apelin has been added to that family. [3]. Adipokines participate actively in metabolic functions [4]. A relation has been established between Apelin and obesity. Glucose and lipid

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metabolism are controlled by apelin [5]. In obesity, excess apelin might be one of the last guards before the appearance of obesity complications such as type 2 diabetes or cardiovascular dysfunctions or insulin resistance (IR) [6]. Leptin is a peptide that is strongly correlated with obesity and its complications [7]. Conflicting results have been obtained from the clinical studies regarding the role of fat distribution and concentrations of serum leptin [8]. In humans, measures of obesity and percentage of body fat are powerfully related with serum leptin levels. It is well known that leptin disturbs insulin action and causes insulin resistance [9]. Therefore, the aim of the present study was to evaluate relation between levels of serum apelin levels with metabolic markers in a sample of Egyptian obese women and evaluate biochemical features of the obese women comparing with healthy normal controls.

2. Subjects and methods

All the procedures used in this study were in accordance with the guidelines of the Helsinki Declaration on Human Experimentations. The study was approved by local ethicscommittee of the National Research Centre (No.: 13176); the purpose of the protocol was explained to the women, and written informed consent was obtained from them before beginning the study. The study included 400 unrelated women; they were 200 obese women and 200 age-matched healthy women. Their age was between 21 and 36 years. Obese women were referred from different centers to the National Research Centre obesity clinic between 2013 and 2014. Insulin resistance (IR) was estimated based on calculation of the homeostasis model assessment (HOMA) index for each patient. This was done using the formula: (fasting plasma insulin in Iu/ml × fasting plasma glucose in mmol/l ÷ 22.5) [10,11].

Anthropometric parameters included body weight, height, mid upper arm circumference, and waist and hip circumferences have been measured. Skin fold thickness of biceps, triceps, subscapular, suprailiac and abdominal skin fold thickness were measured as well. All measurements were taken 3 times on the left side of the body and the mean of the 3 values was used. Body weight was measured to the nearest 0.1 kg and height was measured to the nearest 0.1 cm. Height was measured with the patients standing with their backs leaning against the stadiometer of the same scale.

Body mass index (BMI) was calculated as weight in kilograms divided by height in meters square (kg/m^2). Mid upper arm circumference (MUAC) was measured using a flexible tape at the midway between the olecranon and acromial process on the upper right arm with the elbow flexed 90°. Waist circumference (WC) and hip circumference (HC) were measured in cm using a plastic, non-stretchable tape. WC was measured with light clothing at a level midway between the lower rib margin and the iliac crest standing and breathing normally. Hip circumference (HC) was measured at the level at the widest circumference over the buttocks (at the greater trochanter). Waist-to-hip ratio (WHR) was calculated. Skin fold thickness was measured to the nearest mm, except for low values (usually 5 mm or less) when it was taken to the nearest 0.5 mm. These readings were made at six sites on all subjects, at the biceps, triceps, subscapular and supra-iliac areas, using Holtain caliper (Ltd, Bryberian, Crymmych, Pembrokeshire). The subscapular skin fold was measured below the lower angle of the left scapula at a diagonal in the natural cleavage of the skin. Biceps skin fold thickness was measured at the level of the midpoint between the acromion (lateral edge of the acromion process) and the radius (proximal and lateral border of the radius bone) on the midline of the anterior surface of the arm, triceps skin fold thickness (vertical fold, midway between acromion, and olecranon processes on the posterior surface of the arm), and the position of the suprailiac skinfold was the diagonal fold just above the iliac crest even with the anterior axillary line, and abdominal skin fold was at 5 cm adjacent to the umbilicus to the right side. Subsequently, sum of skin folds were calculated. Anthropometric measurements were obtained according to standardized equipment and following the recommendations of the International Biological Program [12]. Systolic and diastolic blood pressures (SBP and DBP) were measured twice in the right arm in a sitting position after a 10 min rest period; using a mercury sphygmomanometer the average of the two measurements was used for analysis. Blood pressure was measured according to a standardized operating procedure using a calibrated sphygmomanometer and brachial inflation cuff (HEM-7200 M3, Omron Healthcare, Kyoto, Japan). Fat mass was measured by Tanita Body Composition Analyzer (SC-330).

Venous blood samples were collected by direct venipuncture after an overnight fast (minimum 12 h). Fasting plasma glucose and serum lipids (total cholesterol, high-density lipoprotein cholesterol (HDL-C) triglycerides (TG)) were measured by enzymatic colorimetric methods using a Hitachi auto analyzer 704 (Roche Diagnostics. Switzerland) [13]. Low density lipoprotein cholesterol (LDL-C) was calculated according to certain equation (LDL-C = Total cholesterol – Triglycerides/5 + HDL-C) [14]. Serum insulin concentration was analyzed by chemiluminescent immunoassay (Immulite2000, Siemens, Germany [15]. Insulin resistance was determined by the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) calculated as the product of the fasting plasma insulin level (IU/mL) and the fasting plasma glucose level (mmol/L), divided by 22.5 [16]. Serum apelin and leptin levels were measured using commercially available enzyme-linked immunoassay (ELISA) kits (Phoenix Pharmaceuticals, Belmont, CA). The minimal detectable concentration was 0.17 ng/ml, the intra-assay error $<5\%$ and the inter-assay error $<14\%$. Leptin was measured using ELISA kits from Linco Research Inc., St. Charles, MO, USA and Cayman Chemicals, Road Ann Harbor, MI, USA with a sensitivity of 0.125 ng/ml, intra-assay variation of 1.4–4.9% and inter assay variation of 1.3–8.6% for the Linco kit and a detection limit of 1 ng/ml and an intra- and inter-assay variation $<9\%$ for the Cayman kit.

2.1. Statistical analysis

Statistical presentation and analysis of the results were carried out using SPSS software version 17, spss Inc., Chicago, IL, USA. Statistical tests used chi-squared test, student's '*t*' test,-analysis of variance, and tukey tests. General linear regression analysis was performed to identify associations between serum apelin levels and BMI and sum of skin folds and between leptin levels and WHR in obese women. Pearson's correlation coefficient was used to test relation between serum apelin and leptin levels with metabolic biomarkers in obese women.