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Serum prolidase enzyme activity in obese subjects and its relationship with oxidative stress markers

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ABSTRACT

Background: The relationship between increased serum enzyme activity of prolidase and increased rate of collagen turnover in the arterial wall has been asserted in previous studies. Collagen reflects much of the strength to the connective tissue involved in the arterial wall. Atherosclerosis is very common vessel disease and oxidative stress plays a pivotal role in the etiopathogenesis. Our objective was to examine the serum enzyme activity of prolidase and its possible relationships with oxidative stress parameters in obese subjects.

Methods: Our present study was conducted 27 obese subjects and 26 age-matched healthy control subjects. The serum enzyme activity of prolidase in all study population was evaluated spectrophotometrically. Oxidative stress levels in obese subjects were analyzed with total antioxidant capacity (TAC) and total oxidant status (TOS) as well as oxidative stress index (OSI).

Results: Obese subjects have higher serum TOS and OSI indicators as well as prolidase activity than those in control subjects (for all; $p < 0.001$). Moreover, obese subjects have lower levels of TAC than in those in healthy subjects ($p < 0.001$). In the Pearson's correlation analysis, enzyme activity of prolidase was positively related with TOS ($p < 0.001$, $r = 0.529$) and OSI ($p < 0.001$, $r = 0.519$) as well as BMI ($p < 0.001$, $r = 0.692$) and inversely related with TAC ($p < 0.05$, $r = -0.405$) in obese subjects.

Conclusions: Increased serum prolidase activity and decreased antioxidant levels are likely to be a results of increased of oxidative stress levels in obese subjects. The significantly correlation between increased oxidative stress and increased prolidase activity may play a pivotal role in etiopathogenesis of atherosclerotic cardiovascular diseases in obese subjects.

1. Introduction

Obesity, the most common nutritional disorder and a major public health problem in worldwide, can impair the quality of life that [1]. It is well known that obesity is a chronic metabolic disorder associated with atherosclerotic cardiovascular disease, insulin resistance, hyperlipidemia, diabetes mellitus and hypertension as well as the obstructive sleep apnea syndrome [2,3]. Moreover, it has been reported a relationship between obesity and increased oxidative stress levels [4]. In this context, oxidative stress subsequently leads to occur of complications related to obesity [5].

Collagen, a major extracellular matrix (ECM) component, is a main component of the media and fibrous adventitia layers of the arterial wall [6]. A mature lesion in an atherosclerotic plaque is characterized with ECM components including foam cells, smooth muscle cells, a necrotic core and a fibrous cap [7]. Collagens including type I and III are mainly consisting of primary component of fibrous cap of an atherosclerotic plaque [8]. An increase in ECM turnover plays a pivotal role by decreasing synthesis of collagen in atherosclerotic condition as well as endothelial dysfunction [8]. Collagen degradation is initiated by activation of matrix metalloproteinases [7,8].

Prolidase is an enzyme of the metalloproteinases family that destroys the imidodipeptide proline or hydroxyproline in the C terminal by releasing from oligopeptides for resynthesis of collagen and growth of cells [9]. Prolidase is one of homodimeric enzyme that has a pivotal place in the synthesis of collagen by recycling of proline [9]. Prolidase enzyme plays as limiting factor in synthesis of collagen [10]. It is detected in several types of human cells and tissues including erythrocytes, leukocytes, dermal fibroblasts and keratinocytes as well as serum or plasma [11]. In particularly, peripheral enzyme activity of prolidase has been shown to be associated with severity of various disorders [12–17]. Although some theories are proposed, the exact mechanisms involved in alterations in prolidase enzyme activity are unknown. Therefore, this issue has been the focus of our attention.

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According to our knowledge, there is no information about the serum enzyme activity of prolidase and its possible relationships with oxidative status among obese subjects in the literature. Therefore, the purpose of the present work was to analyze serum enzyme activity of prolidase with oxidative stress indicators such as total oxidant status (TOS), total antioxidant capacity (TAC) as well as oxidative stress index (OSI) and to elucidate possible relationship between serum activity of prolidase and indicators of oxidative stress in obese subjects. Thereby, we believe that present results may contribute to the understanding of the pathophysiological mechanisms regarding serum prolidase enzyme activity in obese patients.

2. Materials and methods

2.1. Subjects

We consecutively selected 27 obese subjects (body mass index (BMI) 36.31 ± 3.66 kg/m^2) and 26 healthy control subjects (BMI 21.50 ± 1.87) kg/m^2) in this study. The BMI was calculated by dividing weight by height (kg/m^2).

All of the study participants were physically examined and direct graphic of thorax, baseline ECG, function tests of liver and kidney were performed.

The obese subjects with those circumstances were excluded from study: diabetes mellitus, hyperlipidemia, chronic hypertension disease, coronary artery disease and hepatic or renal disease as well as usage of supplemental vitamins and smoking.

The healthy control groups were constituted with healthy age-and gender matched asymptomatic participants who had normal physical examination and clear medical history. The healthy control groups were not receiving any drugs, and were not smoking or consuming alcohol and had no known systemic disease.

After the case control study protocol was designed according to the local research committee of our Hospital, signed informed consent form of all study populations were taken from each participant in current study.

2.2. Blood sample

After an overnight fasting state, all blood samples were obtained to do this research. The blood taken from the vein was put into a tube and promptly stored at 4 °C. Then after, it was centrifuged at 3000 rpm for 10 min within 30 min after receiving from participants in order to obtain serum sample. All of serum samples were kept at − 80 °C after serum enzyme activity of prolidase and markers of oxidative stress were studied.

2.3. Measurements of serum total antioxidant capacity along with total oxidant status

The serum TAC and TOS levels were measured with an automatic system (by Erel) [18,19]. TAC was calculated by measuring the antioxidative power of the sample against the hydroxyl radical initiated reactions. Oxidants, present in the sample oxidize ferrous ion-dianisidine complex to ferric ion which is colored with xylenol orange in the acidic medium to be measured by spectrophotometer to calculate TOS. TAC is expressed as 1 mmol Trolox Equivalent/L where 1 μmol H_2O_2 Equivalent/L is used to state TOS.

2.4. Detemination of oxidative stress index

The ratio of TAC to TOS levels was accepted as the OSI. It is formulated as: OSI (Arbitrary Unit) = TOS (μmol H_2O_2 Equivalent/L)/TAS (mmol Trolox Equivalent/L) [20].

2.5. Determination of prolidase activity

Serum prolidase activity measurement was calculated according to the modified Chinard's method [21] previously described by Myara [12,22], Prolidase enzyme activity was expressed as U/L.

2.6. Other parameters

The serum lipid levels; triglycerides (TG), total cholesterol (TC) and HDL-cholesterol (HDL-C) as well as LDL-cholesterol (LDL-C) were calculated with assay kits (Abbott®, Germany) with a chemistry analyzer (Aeroset®, Abbott®, Germany). The serum levels of insulin and glucose were identified with a chemiluminescence autoanalyzer (Roche®).

2.7. Statistical analysis

The numeric values are presented as mean ± standard deviation. Categorical data were evaluated with the chi-square test. The all parametric values of each group were evaluated via the Student *t*-test. The all of correlation analyses were conducted using the Pearson's correlation coefficients. A linear regression analysis in obese subjects was performed to examine independent variables. All statistical results in the obese subjects were accepted as considered significant at $p < 0.05$. SPSS 20.0 windows computing program was used for all statistical values.

3. Results

The baseline all demographic as well as clinical characteristics of the participants in the research were described in Table 1. No considerably difference with respect to age and gender between two groups were detected (both; $p > 0.05$). Obese subjects had considerably higher BMI than those in control participants ($p < 0.001$) (Table 1).

As seen in Table 1, according to control groups, obese subjects have higher the serum levels of TG and TC as well as LDL-C (all; $p < 0.001$). Moreover, we detected decreased serum levels of HDL-C in obese subjects than those in control participants ($p < 0.001$) (Table 1).

Our findings in Table 1 indicates that both serum insulin and glucose levels were found considerably higher in obese subjects than those in control participants (both; $p < 0.001$) (Table 1).

As seen in Table 2, according to control groups, we found considerably raised the TOS levels and OSI values as well as enzyme activity of prolidase (Fig. 1) in obese subjects (all, $p < 0.001$). Moreover, obese subjects have lower levels of TAC according to control participants ($p < 0.001$) (Table 2).

According to Pearson's correlation analysis, serum enzyme activity of prolidase was positively related with TOS ($p < 0.001$, $r = 0.529$)

Table 1
The demographic and clinical data in obese subjects and healthy controls.

Parameters	Controls (n = 26)	Obesity (n = 27)	P
Age (year)	28 ± 4	30 ± 4	0.112
Sex (female/male)	14/12	15/12	0.901
Glucose (mg/dL)	88.18 ± 5.69	93.49 ± 4.85	< 0.001
Body mass index (kg/m^2)	21.50 ± 1.87	36.31 ± 3.66	< 0.001
Insulin (μU/mL)	8.76 ± 2.42	12.39 ± 5.93	< 0.001
TG (mg/dL)	134.88 ± 21.35	178.66 ± 37.40	< 0.001
TC (mg/dL)	142.34 ± 26.20	175.64 ± 31.63	< 0.001
HDL-C (mg/dL)	53.03 ± 10.66	34.37 ± 5.96	< 0.001
LDL-C (mg/dL)	62.33 ± 21.30	105.54 ± 26.95	< 0.001

TG: Triglyceride.
TC: Total Cholesterol.
HDL-C: High-density lipoprotein-cholesterol.
LDL-C: Low-density lipoprotein-cholesterol.
Values are mean ± SD.
ns = nonsignificant.