Investigation of Microalbuminuria in Nondiabetic, Normotensive Obese Women

Tijen Erdem Yesim, Serdal Ugurlu, Erkan Caglar, Huriye Balcı, Ayca Ucgul, Cihat Sarkis, Ozer Acbay and Sadi Gundogdu

Abstract

Aim To investigate if obesity which is not accompanied by diabetes and/or hypertension is associated with microalbuminuria in female patients.

Materials and Methods A total of 77 obese female patients from the Outpatient Clinic of Endocrinology of Istanbul University Cerrahpasa Medical Faculty and 30 age-matched, lean, healthy women were enrolled in the study. Patients with accompanying diabetes mellitus, hypertension, obesity associated with any endocrine abnormality, hepatic or renal disease, fever, infectious disease, malignancy were excluded. Weight, height, body-mass index (BMI), waist circumference, waist/hip ratio (WHR) and systolic and diastolic blood pressures were recorded. Albumin excretion in 24-hour urine samples (UAE) were measured using SYNCHRON LX® System with MA Microalbumin kit in two separate 24-hour urine samples from every patient. Statistical analysis was performed using t-test and Pearson’s correlation in SPSS 12.0 for Windows Program.

Results The median albumin excretion in 24-hour urine sample was similar in obese and control groups (12.01 ± 10.69 mg/day vs 9.35 ± 4.09 mg/day; p= 0.211). There were no correlations between the albumin excretion in 24 hour urine samples and BMI, waist circumference, WHR, systolic or diastolic blood pressure.

Conclusion Diabetes mellitus and hypertension are known to be associated with microalbuminuria. In our study, microalbuminuria was not detected in obese women without diabetes and/or hypertension and UAE was similar in obese and lean women.

Key words: obesity, microalbuminuria, normotensive, nondiabetic women

(Introduction

Obesity is generally accepted as an important risk factor for atherosclerosis and diabetes. Particularly, abdominal obesity is a cause of insulin resistance and associated with sodium retention, glomerular hypertension and endothelial dysfunction (1, 2). Endothelial dysfunction is suggested to be related with microalbuminuria in diabetic and hypertensive patients. Also in patients with metabolic syndrome, microalbuminuria is considered as a marker of endothelial dysfunction (3). Microalbuminuria is also found to be associated with increased cardiovascular mortality in diabetic and nondiabetic obese patients. In diabetic patients, hyperglycemia may be considered as the leading cause of microalbuminuria. However, the mechanism of microalbuminuria in nondiabetic patients is not yet fully elucidated (4). In hypertensive patients, obesity is shown to increase the albumin excretion in human and animal models (5, 6). In a study from South Korea, albumin excretion was also found to be increased in the general population, when abdominal obesity is present (7). However, there are scarce data in the literature, concerning microalbuminuria in obese people without diabetes and/or hypertension. In particular, women are in greater risk for obesity compared to men in the world and in Turkey (8, 9). In TOHTA Study, the prevalence of obesity was found to be 35.4% in Turkish women older than 20 years and 1.8-fold of the prevalence in men in the same age...
A total of 77 obese women from Cerrahpaşa Medical Faculty, Outpatient Clinic of Endocrinology and 30 age-matched, lean, healthy women were enrolled in the study. Patients with diabetes mellitus, impaired fasting glucose, hypertension, obesity with any endocrine etiology, fever, infectious disease, malignancy, hepatic or renal disease and who were under any medication were excluded. Informed consent was obtained from all participants. Diabetes mellitus was defined as a fasting blood glucose of higher than 126 mg/dl in two separate analyses or a random blood glucose of higher than 200 mg/dl. Impaired fasting glucose (IFG) was defined as fasting blood glucose between 100 and 125 mg/dl. Hypertension was defined as an arterial blood pressure of higher than 140/90 mmHg. Obesity was defined as body/mass index greater than 30 kg/m² and body/mass index lower than 25 kg/m² was defined as normal. Anthropometric data were obtained from the patients as height, weight, body mass index (BMI), waist and hip circumference, waist/hip ratio (WHR). Arterial blood pressure was measured using Erka sphygmomanometer after a 20 minute-rest. Venous blood was drawn in the fasting state, using Vacutainer tubes for biochemical evaluation. Routine biochemical evaluation was performed using Olympus AU 800 autoanalyzer. Albumin excretion in 24-hour urine samples (UAE) was measured using SYNCHRON LX® System with MA Microalbumin kit in two separate 24-hour urine samples and the mean value was calculated as daily albumin excretion. BMI ≥ 30 kg/m² was defined as obesity and WHR ≥ 0.8 was defined as abdominal obesity in women. Albumin excretion of ≤ 30 mg/day was accepted as normoalbuminuria and 30-300 mg/day was defined as microalbuminuria (10).

Statistical analysis was performed using t-test in SPSS 12.0 for Windows Program. The correlation between albumin excretion in 24 hour urine samples and BMI, waist circumference, WHR and systolic and diastolic blood pressure were evaluated using Pearson’s correlation. p values less than 0.05 were accepted as significant.

Results

Our study group consisted of 77 nondiabetic, normotensive obese women and 30 age-matched, lean women served as the control group. The characteristics of obese and lean groups are shown in Table 1. Both groups were age-matched (34.15 ± 8.97 years in obese group and 36.73 ± 8.82 years in lean group). BMI, waist circumference and WHR were significantly higher in obese group than in lean group (37± 7.12 kg/m² vs 21.6 ± 2.16 kg/m²; p<0.001; 113.5 ± 15.96 cm vs 73.83 ± 6.59; p<0.001; 0.87 ± 0.08 vs 0.76 ± 0.05; p< 0.001 respectively). The systolic and diastolic arterial blood pressure was similar in both groups (107.5± 10.33 mmHg vs 103.33 ± 7.28 mm Hg; p= 0.173 and 70.08 ± 6.84 mmHg vs 67.67 ± 7.28 mmHg; p= 0.316 respectively). The mean albumin excretion in 24 hour urine sample was similar in obese and lean groups (12.01 ± 10.69 mg/day vs 9.35 ± 4.09 mg/day; p= 0.211).

There was no correlation between UAE and age in obese and lean groups (r=0.071; p=0.73 and r= -0.039; p= 0.337 respectively). Also BMI, waist circumference, WHR, systolic and diastolic blood pressure were not correlated with UAE in both groups (r= -0.109 p=0.579 and r= -0.180 p= 0.342; r= 0.346 p=0.327 and r= -0.145 p=0.444; r= 0.102 p= 0.727 and r= -0.076 p= 0.692; r= -0.151 p= 0.551 and r= -0.112 p= 0.557; r= 0.313 p= 0.298 and r= -0.088 p= 0.644 respectively). These findings demonstrated no statistically significant correlation between UAE and the anthropometric data in our study group.

Discussion

Microalbuminuria is suggested to be associated with obesity in the presence of endothelial dysfunction and insulin resistance (7). Also in the normotensive, normoalbuminuric population, daily albumin excretion in urine is found to be related with salt sensitivity. In this study, no relationship between abdominal obesity and microalbuminuria was detected (11). This finding is consistent with our results. However, Kim et al (7) found an independent relationship between waist/hip ratio and the prevalence of microalbuminuria, which we could not detect in the presence of abdominal obesity.

Valensi et al (12) reported that daily albumin excretion in urine was significantly higher in obese people than in lean people. In their study, the prevalence of microalbuminuria was found to be increased in nondiabetic obese people. In

### Table 1. Anthropometric and Biochemical Measures of Patients’ and Control Groups

<table>
<thead>
<tr>
<th></th>
<th>Obese group</th>
<th>Lean group</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean ± SD years (range)</td>
<td>34.15 ± 8.97</td>
<td>36.73± 8.82</td>
<td>NS</td>
</tr>
<tr>
<td>Body-mass index, kg/m²</td>
<td>37± 7.12</td>
<td>21.6± 2.16</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Waist circumference cm</td>
<td>113.5 ± 15.96</td>
<td>73.83± 6.59</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Waist/hip ratio</td>
<td>0.87 ± 0.08</td>
<td>0.76± 0.05</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Systolic BP mmHg</td>
<td>107.5± 10.33</td>
<td>103.33± 7.28</td>
<td>NS</td>
</tr>
<tr>
<td>Diastolic BP mmHg</td>
<td>70.08± 6.84</td>
<td>67.67± 7.28</td>
<td>NS</td>
</tr>
<tr>
<td>Fasting blood glucose, mg/dl</td>
<td>86.08± 8.87</td>
<td>82.86± 8.84</td>
<td>NS</td>
</tr>
<tr>
<td>Cholesterol total, mg/dl</td>
<td>201.73± 40.10</td>
<td>192.39±48.24</td>
<td>NS</td>
</tr>
<tr>
<td>HDL</td>
<td>50.23±13.27</td>
<td>46.35±11.64</td>
<td>NS</td>
</tr>
<tr>
<td>LDL</td>
<td>131.13±34.66</td>
<td>123.81±37.18</td>
<td>NS</td>
</tr>
<tr>
<td>Triglycerides, mg/dl</td>
<td>121.63±87.01</td>
<td>116.85±79.29</td>
<td>NS</td>
</tr>
<tr>
<td>UAE mg/ day</td>
<td>12.01± 10.69</td>
<td>9.35± 4.09</td>
<td>NS</td>
</tr>
<tr>
<td>NS, Nonsignificant</td>
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</table>
In the present study, we could not find a significant difference in UAE between obese and lean groups.

The relationship between microalbuminuria and cardiovascular risk is not fully elucidated. The mechanism of microalbuminuria is suggested to be an increased permeability for albumin in renal and systemic circulation. Furthermore, microalbuminuria could be assessed as a marker of generalized endothelial dysfunction and vasculopathy. Endothelial dysfunction is suggested to contribute to the formation of atherosclerotic lesions by enhancing the penetration of atherogenic lipoprotein particles into the arterial wall. However, there are many factors involved in the pathogenesis of atherosclerosis, such as hyperglycemia, insulin resistance, procoagulant state, and adhesion molecules (13). Whereas these factors could be accepted as contributors of microalbuminuria in diabetic patients, in nondiabetic obese people, insulin resistance is suggested to be the prominent factor associated with microalbuminuria. Kim et al (7) demonstrated that the microalbuminuria triggered by insulin resistance was independent of hypertension and type 2 diabetes. In their study, abdominal obesity and insulin resistance were reported to accompany microalbuminuria, which was inconsistent with our findings.

In conclusion, the relationship between obesity, which is not complicated by hypertension and/or diabetes, and microalbuminuria is not yet fully understood. The results of the studies concerning microalbuminuria in obese people without diabetes and/or hypertension are not consistent. Larger studies are necessary to elucidate the association of microalbuminuria and obesity.

References