High pulse pressure and metabolic syndrome is associated with proteinuria among young adult women

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Abstract

Background

Obesity and metabolic syndrome have causative roles in the increasing prevalence of proteinuria in the general population. However, clinical significance of incidentally discovered proteinuria and its association with metabolic syndrome is unclear in young adult women. We tried to identify the prevalence and risk factors for proteinuria in this population.

Methods

A total of 10,385 women aged 20-39 years who underwent health screenings were surveyed. Each patient was tested for proteinuria with a dipstick (−,±,1+,2+, or 3+), and proteinuria was defined as 1+ or greater. Persistent proteinuria was established by confirming proteinuria in a subsequent test. Metabolic syndrome was defined in accordance with the updated National Cholesterol Education Program Adult Treatment Panel III criteria for Asia.

Results

The mean age was 30.7±5.7 years and the prevalence of persistent proteinuria was approximately 1.0%. Among those, obesity and metabolic syndrome was found in 10.4% and 5.2%, respectively, and metabolic syndrome as well as its component, hypertension, hyperglycemia, central obesity, low high-density lipoprotein and high triglyceride levels were closely related to the presence of proteinuria. In addition, wide pulse pressure ≥40 mmHg was another independent risk factor for proteinuria [odds ratio (OR) 8.61, 95% confidence interval (CI) 2.95-25.11)]. It had an additive effect to metabolic syndrome in predicting proteinuria. Even in subjects without metabolic syndrome, the influence of increased pulse pressure was consistently observed (OR 3.24, 95% CI 1.03-9.98).

Conclusions

Specific attention is necessary for proteinuria in asymptomatic young women aged 20-39 years, if they have metabolic syndrome or wide pulse pressure.

Key words: proteinuria, metabolic syndrome, pulse pressure, young women in the population
**Background**

Proteinuria is a common laboratory finding in the general population. Although it is transient and functional in most cases, it may be persistent as an independent risk factor for adverse renal and cardiovascular outcomes[1-4]. In clinical practice, nevertheless, incidentally discovered proteinuria in young adults aged 20-30 years is often overlooked as a benign proteinuria. In fact, the Third National Health and Nutrition Examination Survey (NHANES III) in the United State reported that the prevalence of overt proteinuria (urine albumin to creatinine ratio, UACR $\geq 300$ mg/L) in this age group were 0.5% in males and 0.9% in females, and the relative frequency of abnormal UACR $\geq 30$ mg/L were 6.5% and 8.1%, respectively, the lowest rates across all age groups[5]. Therefore, the cost-effectiveness and benefit of screening proteinuria in young individuals without symptoms or relevant associated diseases such as diabetes or hypertension is debatable.

Metabolic syndrome, composed of central obesity, dyslipidemia, high blood pressure (BP) and impaired fasting glucose, is known to be a risk factor for proteinuria in the general population[6, 7]. Recently, the increasing prevalence of obesity is influencing the rise of metabolic syndrome and glomerulopathy[8]. However, most of the studies about the relationship between metabolic syndrome and proteinuria were conducted in subjects over 40 years of age[9, 10], and little is known in young population.

Especially in young women aged 20-39 years, clinical proteinuria could be a critical issue, because unrecognized or ignored pre-pregnancy proteinuria could induce many complications during pregnancy, from urinary tract infection to chronic kidney disease, and it remains central to the diagnosis of preeclampsia in hypertensive pregnancy[11]. In addition, previous investigators found that even mild intermittent proteinuria in asymptomatic young individuals are frequently associated with significant underlying renal pathology, emphasizing that it may not be a benign condition[12].

Therefore, the authors wanted to evaluate the association between metabolic syndrome and proteinuria in this young female population, and tried to investigate additional risk factors for proteinuria.

**Subjects and Methods**

**Subjects and Health screenings parameters**

A total of 10,724 young women aged 20-39 years who underwent health screenings at Hallym University Sacred Heart Hospital from January 2010 to December 2011 were enrolled. Among those, medical records were available for 10,472 subjects. To exclude the possibility of false-positive results, alkaline (pH $\geq 8$, n=39) or
highly concentrated (specific gravity >1.025, n=33) specimens were excluded. Individuals with pre-diagnosed CKD (n=15) was also excluded. Thus, the data of 10,385 subjects were analyzed. This study was performed with the approval of the local Institutional Review Board.

The health screening examination included 1) an interview regarding current health status (diabetes, hypertension, alcohol, smoking, exercise, medication, marital status, and childbirth), 2) a physical examination - height, weight, waist circumference, body mass index (BMI) and BP, and 3) blood and urine tests. Waist circumference was measured to the nearest 0.1cm in a horizontal plane at the level of the midpoint between the iliac crest and the costal margin at the end of a normal expiration. The BMI was calculated as the individual’s weight (kg) divided by the square of the height (m) and obesity was defined as BMI ≥25 kg/m². All blood samples were obtained in the fasting state. The serum levels of hemoglobin, glucose, total cholesterol (TC), high-density lipoprotein (HDL) cholesterol, triglycerides (TG), and creatinine were measured. Low-density lipoprotein (LDL) cholesterol was calculated using the following equation; LDL = TC – HDL – TG/5.0 (mg/dL). With these parameters, metabolic syndrome was defined in accordance with the updated National Cholesterol Education Program Adult Treatment Panel (NCEP-ATP) III criteria for Asia [13, 14]. The presence of three or more of the following criteria constituted a diagnosis of metabolic syndrome: (i) waist circumference ≥80 cm; (ii) fasting triglyceride ≥150 mg/dL or medication use; (iii) HDL cholesterol < 50 mg/dL or medication use; (iv) BP ≥130/85 mmHg or antihypertensive medication use; and (v) fasting glucose ≥100 mg/dL or current medication.

**Proteinuria evaluation**

Dipstick urinalysis was performed using spontaneously voided fresh urine that was analyzed within a few minutes after collection. Urinalysis was not performed in subjects who were menstruating or exhibiting symptoms of urinary tract infection (UTI) or vaginal discharge. The results were interpreted by one physician and were scored as (−) when no staining was observed, (+) when weak staining was observed, and 1+, 2+, 3+, or 4+ when mild-to-strong staining was observed. Proteinuria was defined as 1+ or greater. However, the quantification of proteinuria was not performed in the routine screening test. For patients who had proteinuria at the initial test, follow-up test was recommended within 1 or 2 weeks. Persistent proteinuria was confirmed when repeat testing also revealed proteinuria ≥1+. For second test, proteinuria was quantified using protein-to-creatinine ratio (UPCR) and albumin-to-creatinine ratio (UACR). The clinical diagnosis of proteinuria was
made by each physician based on previous history, clinical features, and laboratory findings regardless of whether a renal biopsy was performed or not.

**Statistical analysis**

Statistical analyses were performed using SPSS version 18.0 (SPSS Inc., Chicago, IL, USA). All variables were expressed as the mean ± SD or median with ranges, unless otherwise indicated. Differences between two groups were analyzed by an independent t-test for continuous variables or the \( \chi^2 \) test for categorical data. The receiver operating characteristics (ROC) curve was constructed for evaluating the relationship between clinical factors and proteinuria and the areas under the curve (AUC) were calculated. Multiple logistic regression analysis was performed to find significant determinants for proteinuria. \( P < 0.05 \) was considered statistically significant.

**Results**

**Baseline characteristics and pathologic data**

The mean age was 28.9 ± 5.5 years, and 6791 (65.3%) subjects had a history of childbirth. In the initial urinary dipstick test, 227 (2.2%) subjects had varying degrees of proteinuria: 1+ (n=184, 1.8%), 2+ (n=40, 0.4%), and 3+ (n=3, 0.03%). Of these, repeat test was performed in 209 subjects; 97 subjects showed persistent proteinuria, but 112 had no proteinuria in second urinalysis (Figure 1). Therefore, the prevalence of persistent proteinuria in young women aged 20-39 years was approximately 1.0%. The median UPCR and UACR measured in repeat urinalysis were 0.57 and 0.45 in subjects with persistent proteinuria.

The baseline demographic and laboratory findings according to the presence of proteinuria are shown in Table 1. Persistent proteinuria was more prevalent in current or past smokers. And subjects with proteinuria had higher BMI, waist circumference, systolic BP, and wide pulse pressure. In addition, serum levels of metabolic components, fasting glucose, TG, and HDL as well as serum creatinine were significantly different between the two groups.

**Prevalence of proteinuria and risk factor analysis**

Among young women, the prevalence of obesity and metabolic syndrome was 10.4% and 5.2%, respectively. The prevalence of proteinuria was higher in subjects with BMI \( \geq 25 \) kg/m\(^2\) compared to those with BMI <25 kg/m\(^2\) (3.2% vs. 0.7%, \( p<0.001 \)) (Figure 2, upper). Proteinuria was more frequently detected in subjects with
metabolic syndrome than without (8.3% vs. 0.7%, p<0.001) and the prevalence increased significantly as the number of metabolic components increases (Figure 2, lower). In Figure 3, the unadjusted odds ratio (OR) for proteinuria according each metabolic component is shown. The significantly higher rates of proteinuria were observed across all the categories of metabolic components.

Also, increased pulse pressure was another important risk factor for proteinuria in these subjects. A 1 mmHg increase of pulse pressure was associated with 10% increased risk of having proteinuria (OR 1.10, 95% CI 1.067-1.137, p<0.001) and ROC curve analysis shows the close relationship between pulse pressure and proteinuria (Figure 4). The area under the ROC curve was 0.754, and with the cut-off values of 40 mmHg, the sensitivity and specificity was 0.91 and 0.84, respectively. When subjects were stratified into four groups according to the presence of metabolic syndrome and pulse pressure (≥40 mmHg), the risk of proteinuria was highest in those with both metabolic syndrome and high pulse pressure (relative risk 13.03) (Figure 5). With these, Table 2 shows clinical factors associated with proteinuria in young female population. Smoking, obesity, wide pulse pressure (≥40 mmHg) and metabolic syndrome were associated proteinuria in univariate analysis. In multivariate analysis, only pulse pressure [odds ratio (OR) 3.53, 95% confidence interval (CI) 1.04-11.91] and metabolic syndrome (OR 8.61, 95% CI 2.95-25.11) were significant determinants for proteinuria.

In a subgroup of subjects without metabolic syndrome, the prevalence of proteinuria was 0.7%. Smoking and obesity did not influence to the presence of proteinuria. However, the effect of wide pulse pressure was consistent in subjects without metabolic syndrome, too (OR 3.24, 95% CI 1.03-9.98).

Discussion

In the present study, we tried to evaluate the prevalence and associated factors for proteinuria in the general population, especially young women. We found that approximately 1.0% of this population has persistent proteinuria, and metabolic syndrome as well as its component, hypertension, hyperglycemia, central obesity, low HDL and high TG levels were closely related to the presence of proteinuria. In addition, wide pulse pressure ≥40 mmHg was another important risk factor for proteinuria. The effect of wide pulse pressure was independent to other clinical factors including metabolic components. Even in subjects without metabolic syndrome, the influence of wide pulse pressure for proteinuria was consistently observed. Therefore, specific attention is necessary for proteinuria in asymptomatic young women if they have metabolic syndrome or wide pulse pressure. To our knowledge, this is the first study to display the risk factors for proteinuria in the general
population of young women.

In clinical practice, incidentally discovered proteinuria in young healthy adults is often ignored or overlooked. Indeed, according to a review article published two decades ago, proteinuria screening of healthy, asymptomatic adults was not recommended because less than 1.5% of subjects with positive dipstick findings have significant disease. However, they mentioned that proteinuria screening could detect many cases of potentially significant disease such as mild glomerulonephritis or renal impairment [15]. Moreover, Muth et al reported that proteinuria, even intermittent proteinuria, might not be a benign condition. They biopsied 51 young asymptomatic patients with intermittent proteinuria and normal renal function (age, 16–34 years) and found that approximately two-thirds of patients had pathologic evidence of renal disease[12]. As these data shows, proteinuria in young adults may not be as benign a condition as previously thought. Nevertheless, recommendation of regular screening for proteinuria in all subjects of the general young population seems unreasonable as it is cost-ineffective. Therefore, early identification of risk factors for persistent proteinuria is important.

According to our data, the prevalence of incidentally discovered proteinuria was 2.2%, and half of those had persistent proteinuria. Thereby, the prevalence of persistent proteinuria was approximately 1.0%, similar to the previously reported data[5]. Metabolic syndrome as well as its components was closely associated with persistent proteinuria. Metabolic syndrome, a clustering of various metabolic derangements, is a well established independent predictor for cardiovascular morbidity and mortality in the general population[6, 8, 9, 16, 17]. In addition, the close relationship between metabolic syndrome and the development of proteinuria was also reported. Lucove et al reported a 1.26-fold increased risk for the development of proteinuria among American Indians with metabolic syndrome[9]. Tozawa et al also reported the effect of metabolic syndrome in a Japanese population (relative risk 2.09)[3]. And, similar findings were also observed in the general Korean population, too (OR 2.30). However, those prior studies were conducted or included subjects over 40 years of age, and their relationship in young population is not known yet. According to our data, in normotensive young subjects aged 20-39 years, metabolic syndrome had 8-fold increased risk for persistent proteinuria, too. Several mechanisms including insulin resistance, chronic inflammation, and lipotoxicity have been proposed to explain the diverse renal effects of metabolic syndrome[18]. In addition, various adipocytokines have also important roles in renal damage by inducing sympathetic overactivities, systemic inflammation and oxidative stress [19]. Considering that the metabolic syndrome is a modifiable risk factor, early detection and treatment of metabolic syndrome would be a cost-effective strategy to decrease the prevalence of proteinuria as well as chronic kidney
disease (CKD) and end-stage renal disease (ESRD) in the general population.

Another important finding in our study is that subjects with persistent proteinuria had a higher systolic BP and wide pulse pressure than those without persistent proteinuria. Particularly, pulse pressure was closely associated with proteinuria and its effect was independent to other clinical factors. This finding is consistent with previous longitudinal data suggesting the pulse pressure as a risk factor for increased albuminuria in general or hypertensive atherosclerotic populations [20, 21]. The close association between increased PP and proteinuria could be explained by wide pulse pressure induced endothelial dysfunction and barotrauma to renal arteries [22-25]. However, with the limitation of the cross-sectional design, the causal relationship between proteinuria and wide pulse pressure among young population without vascular risk factors could not be exactly identified. Moreover, a recently published large-scale Japanese study reported that the close associations between high pulse pressure and proteinuria could be established only in diabetic patients, not among subjects with prediabetes or normal glucose tolerance [26]. Therefore, a further well-designed study is needed to elucidate the exact patho-physiological link between pulse pressure and proteinuria in young adult population.

As well as, the association between increased pulse pressure and metabolic syndrome is unclear, too, although several previous reports studied the relevance between pulse pressure and metabolic syndrome. Ferreira et al reported that young individuals with metabolic syndrome have increased arterial stiffness via poor cardiopulmonary fitness and high subcutaneous trunk fat[27]. The researchers suggested increased arterial stiffness as a risk factor for cardiovascular disease in subjects with metabolic syndrome. However, there is contrary opinion, too. According to the study with Mannucci et al, the close association between high pulse pressure and metabolic syndrome disappeared after adjustment for age and mean BP[28]. In our study, pulse pressure was closely associated with waist circumference, BMI and TG levels, but not with HDL and glucose levels. However, the effect of increased pulse pressure was independent to metabolic syndrome as well as its components. Therefore, more future data about the relationship are needed, too.

This study has several weaknesses. First, although the medical chart of each patient was thoroughly reviewed, it is impossible to review the precise history of past medical conditions, especially those affecting proteinuria, in all patients. As the survey was a self-reported questionnaire, patients who are reluctant to expose their medical conditions or drug history may not have provided completely accurate information. Moreover, we could not check several laboratory parameters that are known to associate with proteinuria such as uric acid or c-reactive proteins with the limitation of health screening data. Second, among those with persistent proteinuria, referral to
a nephrologist was performed only in 53.6%, therefore, we could not know the exact causes of persistent proteinuria. There is a need to emphasize the need for further evaluation and management of persistent proteinuria in this population.

Conclusions

Approximately 1.0% of young women aged 20-39 years exhibited persistent proteinuria. Metabolic syndrome as well as its component, hypertension, hyperglycemia, central obesity, low HDL and high TG levels were closely related to the presence of proteinuria. Also, increased pulse pressure could be another important independent risk factor for proteinuria. It had an additive effect to metabolic syndrome in predicting proteinuria. Specific attention is necessary for proteinuria in asymptomatic young women if they have metabolic syndrome or wide pulse pressure.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

1) Jwa-Kyung Kim: Data analysis and writing up
2) Young-Su Ju: Data recruitment and analysis
3) Sung Jin Moon: Writing up and modification
4) Young Rim Song: Data analysis and statistical advisory
5) Hyung Jik Kim: Study design determination
6) Sung Gyun Kim: Research initiative and study design determination

Acknowledgements

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References


Figure legends

**Figure 1** Flow diagram of proteinuria evaluation. The prevalence of persistent proteinuria was approximately 1% in young women of general population.

**Figure 2** The prevalence of proteinuria in subjects with obesity and metabolic syndrome. Obese patients showed a high prevalence of proteinuria, and the prevalence increased significantly as the number of metabolic components increases.

**Figure 3** Unadjusted odds ratio for proteinuria according to each metabolic component.

**Figure 4** ROC Curve. Sensitivity and specificity of pulse pressure for proteinuria. Cut-off pulse pressure of 40 mmHg maximizes the prediction of proteinuria (sensitivity of 91.2% and specificity of 84.4%). Area under the curve=0.754

**Figure 5.** Subjects with both metabolic syndrome and elevated pulse pressure showed the highest relative risk for proteinuria in the general young population.
Table 1. Baseline characteristics of the study subjects

<table>
<thead>
<tr>
<th></th>
<th>Total*</th>
<th>Persistent proteinuria</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Negative</td>
</tr>
<tr>
<td>Number, n (%)</td>
<td>10,367</td>
<td>10,270 (99.0%)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>28.9 ± 5.5</td>
<td>28.8 ± 5.5</td>
</tr>
<tr>
<td>Smoker, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ex-smoker</td>
<td>95 (0.9)</td>
<td>87 (0.8)</td>
</tr>
<tr>
<td>Current</td>
<td>102 (1.0)</td>
<td>93 (0.9)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>161.5 ± 4.9</td>
<td>161.4 ± 4.9</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>54.5 ± 8.5</td>
<td>54.0 ± 7.9</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>20.9 ± 3.2</td>
<td>20.7 ± 2.9</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>69.6 ± 7.3</td>
<td>69.3 ± 6.6</td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>107.0 ± 11.4</td>
<td>105.0 ± 10.9</td>
</tr>
<tr>
<td>Diastolic BP (mmHg)</td>
<td>66.7 ± 8.9</td>
<td>66.7 ± 9.0</td>
</tr>
<tr>
<td>Pulse pressure (mmHg)</td>
<td>43.3 ± 9.3</td>
<td>38.4 ± 8.7</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>12.7 ± 0.9</td>
<td>12.7 ± 0.9</td>
</tr>
<tr>
<td>Fasting glucose (mg/dL)</td>
<td>86.5 ± 13.3</td>
<td>85.7 ± 10.8</td>
</tr>
<tr>
<td>Serum creatinine (mg/dL)</td>
<td>0.66 ± 0.10</td>
<td>0.64 ± 0.09</td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>178.8 ± 29.4</td>
<td>177.8 ± 29.0</td>
</tr>
<tr>
<td>HDL cholesterol (mg/dL)</td>
<td>67.6 ± 18.3</td>
<td>68.2 ± 18.5</td>
</tr>
<tr>
<td>LDL cholesterol (mg/dL)</td>
<td>95.7 ± 27.3</td>
<td>94.7 ± 26.8</td>
</tr>
<tr>
<td>Triglyceride (mg/dL)</td>
<td>78.0 ± 36.8</td>
<td>75.5 ± 35.0</td>
</tr>
<tr>
<td>UACR (mg/g)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>UPCR (mg/g)</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Blood on dipstick test, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>8558 (82.6%)</td>
<td>8495 (82.7%)</td>
</tr>
<tr>
<td>Trace</td>
<td>1540 (14.8%)</td>
<td>1534 (14.9%)</td>
</tr>
<tr>
<td>Positive</td>
<td>269 (2.5%)</td>
<td>241 (2.3%)</td>
</tr>
</tbody>
</table>

*Total: All screening subjects (n=10,385) – loss of follow-up (n=18)

Abbreviations: BP, blood pressure; HDL, high-density lipoprotein; LDL, low-density lipoprotein; UACR, urine albumin-to-creatinine ratio; UPCR, urine protein-to-creatinine ratio
Table 2. Univariate and multivariate analysis for prediction of proteinuria

<table>
<thead>
<tr>
<th></th>
<th>Univariate analysis</th>
<th>Multivariate analysis*</th>
<th>Multivariate analysis†</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR (95% CI)</td>
<td>P</td>
<td>OR (95% CI)</td>
</tr>
<tr>
<td><strong>Total patients</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>1.33 (1.06-6.83)</td>
<td>0.002</td>
<td>1.15 (0.80-2.63)</td>
</tr>
<tr>
<td>BMI ≥ 25</td>
<td>4.34 (2.05-9.18)</td>
<td>&lt;0.001</td>
<td>1.61 (0.62-4.20)</td>
</tr>
<tr>
<td>Pulse pressure ≥ 40mmHg</td>
<td>5.01 (1.53-16.58)</td>
<td>0.008</td>
<td>4.21 (1.14-11.81)</td>
</tr>
<tr>
<td>Metabolic syndrome</td>
<td>10.65 (5.10-27.96)</td>
<td>&lt;0.001</td>
<td>9.22 (2.98-22.60)</td>
</tr>
<tr>
<td><strong>Without metabolic syndrome</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>1.26 (0.58-3.66)</td>
<td>0.216</td>
<td>1.06 (0.62-2.61)</td>
</tr>
<tr>
<td>BMI ≥ 25</td>
<td>1.50 (0.43-5.23)</td>
<td>0.525</td>
<td>1.31 (0.37-4.62)</td>
</tr>
<tr>
<td>Pulse pressure ≥ 40mmHg</td>
<td>3.68 (1.10-12.30)</td>
<td>0.032</td>
<td>3.26 (1.04-10.99)</td>
</tr>
</tbody>
</table>

* adjusted smoking, BMI, pulse pressure and metabolic syndrome
† adjusted smoking, BMI, pulse pressure, metabolic syndrome and creatinine
Asymptomatic women undergoing health screenings over 2 year (N=10,385)

- (n=9,260, 89.2%)
  - +/- (n=898, 8.6%)
  - + (n=184, 1.8%)
  - ++ (n=40, 0.4%)
  - +++ (n=3, 0.03%)

227 (2.2%) → No Repeat Urinalysis (n=18, 7.9%)

Repeat Urinalysis (n=209, 92.1%)

→ Transient (n=112, 53.5%)
  → Persistent (n=97, 46.5%)

→ No Nephrology Visit (n=45, 46.4%)

Nephrology Visit (n=52, 53.6%)
  : <Clinical diagnosis>
  - Glomerulonephritis (n=25, 48.1%)
  - Hypertensive (n=10, 19.2%)
  - Diabetes (n=8, 15.3%)
  - Asymptomatic bacteriuria (n=9, 17.3%)
Figure 3

Prevalence of proteinuria (%)

Metabolic components

<table>
<thead>
<tr>
<th>Metabolic Component</th>
<th>Unadjusted OR (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>High BP</td>
<td>5.93 (2.73-12.89)</td>
<td>0.003</td>
</tr>
<tr>
<td>High Glucose</td>
<td>6.22 (3.12-10.98)</td>
<td>0.001</td>
</tr>
<tr>
<td>High TG</td>
<td>7.78 (4.04-15.01)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Abdominal obesity</td>
<td>7.52 (4.14-13.56)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Low HDL</td>
<td>4.99 (2.73-9.12)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>