Low prevalence of the metabolic syndrome but high occurrence of various metabolic disorders in Chinese women with polycystic ovary syndrome

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Abstract

Objective: Variations in the prevalence of metabolic syndrome (MetS) among women with polycystic ovary syndrome (PCOS) in different races were reported. We sought to report this prevalence and its components in Chinese women with PCOS and compared these characteristics with healthy controls.

Design: Anthropometric measurements and biochemical parameters were evaluated in 578 PCOS patients diagnosed by the Rotterdam criteria and 281 age- and body mass index (BMI)-matched controls. International Diabetes Federation criteria for MetS were used.

Results: The prevalence of MetS was 16.8% in this study, and 60.7% of patients displayed at least one component of MetS. Among the patients, the rates of dyslipidemia, impaired fasting glucose, and elevated blood pressure were 41.6, 19.8, and 16.1% respectively; the rates of these corresponding components in age- and BMI-matched controls were 14.6, 5.3, and 5.7% respectively. In PCOS patients, the prevalence of MetS was 0.0, 3.9, 20.2, and 51.1% for four different BMI groups respectively; the prevalence of MetS was 7.3, 14.9, 24.2, and 42.4% in the four age groups respectively. Nearly 90% of patients diagnosed with MetS belonged to overweight and obese groups. BMI and age rather than free testosterone, free androgen index, fasting insulin, or sex hormone-binding globulin were included in formulation for predicting MetS according to multivariable logistic regression.

Conclusions: Low prevalence of MetS but high occurrence of various metabolic disorders was found in women with PCOS compared with age- and BMI-matched controls in this study. BMI and age appeared to contribute more to developing MetS than other parameters associated with insulin resistance or hyperandrogenism.

Introduction

Metabolic syndrome (MetS) is characterized by metabolic abnormalities that place individuals at increased risk of cardiovascular diseases (CVD). These metabolic abnormalities include the following: diabetes and raised fasting plasma glucose; abdominal obesity; dyslipidemia; and high blood pressure (1, 2). These risk factors lead to increased morbidity and mortality due to atherosclerotic disease, kidney disease, and increased risk of stroke. Moreover, there is now strong epidemiological evidence that indicates a link between MetS and several cancers, including colon and breast cancer (3–5). Evidence has shown that insulin resistance (IR) and compensatory hyperinsulinemia play central roles in the pathophysiology of MetS.

Polycystic ovary syndrome (PCOS) is regarded as one of the most common endocrine diseases among women of childbearing age, affecting from 5 to 10% of women in this age range (6–8). Patients with PCOS often have complications associated with dyslipidemia, diabetes mellitus/impaired glucose tolerance, and hypertension, and are therefore at increased risk of having MetS (8–10). The prevalence of MetS in PCOS patients varies among different ethnicities and has been reported to be ~43.0–47.3% in America (11–13), 46.2% in India (14), 28.4% in Brazil (15), 14.5% in Korea (16), and 35.3% in Thailand (17).

Given the variations reported in the prevalence of MetS among PCOS patients of different races and the paucity of literature describing the prevalence of metabolic disorders including MetS in PCOS patients in mainland China, we sought to report these characteristics and compared them to healthy controls.
Materials and methods

Patients and control subjects

A total of 974 adult PCOS patients (20–41 years of age) were diagnosed consecutively at the Department of Gynecology and Reproductive Center of the Second Affiliated Hospital of Sun Yat-Sen University from January 2004 to October 2008. Patients’ records were reviewed and 578 patients with adequate data, specifically physical examination data, and sufficient laboratory blood tests to diagnose MetS were included in the study. Age- and body mass index (BMI)-matched controls comprised 281 women recruited randomly from an annual routine physical examination (18). All controls were clinically healthy and were euthyroid according to clinical evaluation and thyroid-stimulating hormone (TSH) measurement. The study was approved by the institutional review board of the Second Affiliated Hospital of Sun Yat-Sen University.

PCOS patients were diagnosed according to the Rotterdam criteria (19), meeting at least two out of the following three criteria: i) oligo- and/or anovulation (i.e. ≤8 menstrual periods in a year or menstrual cycles more than 35 days in length) (20), ii) clinical hyperandrogenism (i.e. acne or modified Ferriman–Gallwey scores ≥8 (21)) or biochemical hyperandrogenism (i.e. serum total testosterone (TT) > 2.6 nmol/l, free testosterone (FT) ≥ 6.0 pg/ml, and FT normal values were determined by the clinical laboratory of the gynecology department at the Second Affiliated Hospital of Sun Yat-Sen University). and iii) ultrasonographic findings of ovarian polycystic morphology (i.e. presence of ≥12 follicles in each ovary measuring 2–9 mm in diameter) and exclusion of related disorders such as hypothyroidism, hyperprolactinemia, and adrenal hyperplasia by physical examination and lab testing of TSH, prolactin (PRL), and a- hydroxyprogesterone (17a-OHP).

Patients were defined as having MetS, based on International Diabetes Federation (IDF) criteria for MetS (22), if they had central obesity (waist circumference ≥ 80 cm) plus two or more of the following four factors: i) increased concentration of triglycerides (TG): ≥ 1.7 mmol/l, ii) reduced concentration of high-density lipoprotein cholesterol (HDL-C): < 1.29 mmol/l, iii) raised blood pressure: systolic pressure ≥ 130 mmHg or diastolic pressure ≥ 85 mmHg or treatment of previously diagnosed hypertension, and iv) increased fasting glucose (FG) level ≥ 5.6 mmol/l (impaired FG, IFG) or previously diagnosed type 2 diabetes.

Study protocol

All PCOS patients underwent anthropometric measurements, including weight, height, waist and hip circumferences, blood pressure, modified Ferriman–Gallwey scores, and acne scores. Ferriman–Gallwey scores were assessed by at least two observers. Control subjects received the same anthropometric measurements with the exception of waist and hip circumferences.

Body weight, height, and waist and hip circumferences were measured based on methods recommended by the World Health Organisation (23). BMI was calculated as weight (kg)/height (m²). Underweight was defined as a BMI < 18.5, normal weight as a BMI ≥ 18.5 and < 23.0, overweight as a BMI ≥ 23.0 and < 25.0, and obesity as a BMI ≥ 25, according to the Asia–Pacific criteria of BMI for obesity (24).

Fasting blood samples were obtained from PCOS patients between the first and fifth day of the menstrual period/withdrawal bleed in order to measure PRL, LH, FSH, estradiol (E2), and TT by the chemiluminescence immunoassays Access 2 (Beckman, Fullerton, California, USA); FT, sex hormone-binding globulin (SHBG), DHEAS, and 17-OHP were measured by Access 2 ELISAs (Beckman). TSH was measured using a chemiluminescence immunometric assay (Immulite 2000 Analyzer; CPC, Los Angeles, CA, USA). The free androgen index (FAI), a parameter of bioavailable testosterone, was calculated from TT and SHBG as follows: FAI = (TT (nmol/l) × 100/SHBG (nmol/l)) (25).

Fasting venous blood samples were also used to measure the levels of glucose, insulin, total cholesterol (CHOL), HDL-C, and TG. Plasma glucose was measured by the glucose oxidase method (Hitachi 7600) and plasma insulin was measured using a chemiluminescence immunometric assay and commercial kit (Immulite 2000 Analyzer; CPC). CHOL, HDL-C, and TG were measured using an enzymatic calorimetric method with the 7600 autoanalyzer (Hitachi 7600). Homeostasis model assessment (HOMA) was applied to estimate the degree of IR. The equation used to obtain this value is as follows: HOMA-IR = (fasting plasma glucose (mmol/l) × insulin (µU/ml))/22.5 (26).

For controls, fasting blood samples were collected and the same methodologies described above were used to measure TT, FT, SHBG, DHEAS, 17-OHP, PRL, TSH, fasting insulin (FI), FG, TG, and CHOL. HOMA-IR was also calculated.

Statistical analyses

The data were analyzed using the statistical software SPSS version 13.0 for Windows (SPSS, Inc., Chicago, IL, USA). We assessed the normality of the distribution of all continuous variables using the Kolmogorov–Smirnov test. Because the data were not normally distributed, continuous variables were presented as median values with an interquartile range (25–75th percentile), and differences in medians between groups were determined by the Mann–Whitney test. For the categorical variables, Pearson χ² test was used to analyze the differences between groups and to obtain an odds ratio (OR). Logistic regression analyses were
employed to determine risk factors for MetS and to estimate the ORs for MetS. The Wald $\chi^2$ test was used to test the logistic regression model. Statistical significance was set at $P<0.05$.

**Results**

**Clinical and metabolic features of patients and controls**

Of the sample with PCOS, 94.3% ($n=545$) were 35 years of age or younger. A large number of women in this sample presented oligomenorrhea or amenorrhea (84.3%), ultrasonographic appearance of polycystic ovaries (91.4%), and clinical hyperandrogenism or biochemical hyperandrogenism (78.4%). None of the 578 PCOS patients were previously diagnosed with type 2 diabetes.

The comparison of clinical and metabolic features between PCOS patients and age- and BMI-matched healthy controls was summarized (Table 1). PCOS patients demonstrated increased systolic blood pressure, diastolic blood pressure, FG, FI, HOMA-IR, TT, DHEAs, TG, and CHOL. However, there was no statistically significant difference in terms of the level of FT, SHBG, and FAI between age- and BMI-matched controls and PCOS patients.

Among the women with PCOS, those who met the criteria for MetS displayed significantly higher mean age, waist circumference, BMI, waist circumference to hip, FT, FAI, HOMA-IR, FG, FI, TG, and lower SHBG, LH, and HDL-C levels than patients who did not meet the criteria for MetS. No significant differences in the levels of FSH, E2, TT, and DHEAs were detected between PCOS patients with MetS and those without MetS (Table 2).

### Prevalence of MetS and metabolic features

The prevalence of MetS was 16.8%, based on IDF criteria, in the present cohort of women with PCOS. The rate of dyslipidemia (higher TG or CHOL), IFG, and elevated blood pressure was significantly higher in women with PCOS than that in the age- and BMI-matched control group (Fig. 1). In this sample of PCOS patients, dyslipidemia was the most common metabolic feature (41.6%), and the next most common one was overweight or obesity (38.4%). A smaller portion of patients with PCOS presented IFG (19.8%) and elevated blood pressure (16.1%). Generally, the rates of the above-mentioned metabolic disorders in PCOS patients increased over two times when compared with the age- and BMI-matched controls.

Of the 578 PCOS patients, 60.7% ($n=351$) had one component of MetS; 33.3% ($n=191$) had two components; 17.6% ($n=102$) had three components; 5.5% ($n=32$) had four components; and only 1.4% ($n=8$) had all five components.

### Prevalence of MetS in different BMI and age subgroups

We classified women with PCOS into four BMI groups and the prevalence of MetS increased with BMI (Table 3). The prevalence increased gradually from 3.9% in the normal weight group to 51.1% in the obese group ($P<0.0001$). The risk of having MetS increased nearly fivefold in the overweight group when compared with the normal weight group, threefold in the obese group when compared with the overweight group, and 2.5-fold in the obese group when compared with the normal weight group. Of the PCOS patients with MetS

#### Table 1 Comparison of clinical and biochemical parameters of polycystic ovary syndrome (PCOS) patients and the age- and body mass index-matched controls.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Age- and BMI-matched controls</th>
<th>PCOS</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$n$</td>
<td>281</td>
<td>578</td>
<td>~</td>
</tr>
<tr>
<td>Age (years)</td>
<td>28.0 (26.0–31.0)</td>
<td>27.0 (25.0–30.0)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>BMI (kg/m$^2$)</td>
<td>21.9 (20.9–23.8)</td>
<td>21.9 (19.7–24.8)</td>
<td>&gt;0.5</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>106.0 (99.0–113.0)</td>
<td>110.9 (100.0–120.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>69.0 (62.0–74.0)</td>
<td>73.0 (66.0–80.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Testosterone (nmol/l)</td>
<td>1.8 (1.5–2.2)</td>
<td>2.3 (1.6–2.9)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>FT (pmol/l)</td>
<td>146.9 (72.9–168.8)</td>
<td>156.2 (69.4–177.0)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>FAI</td>
<td>4.1 (2.3–6.0)</td>
<td>4.4 (2.4–7.5)</td>
<td>&gt;0.1</td>
</tr>
<tr>
<td>DHEAS (µmol/l)</td>
<td>4.4 (3.2–5.7)</td>
<td>5.3 (3.8–7.1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SHBG (nmol/l)</td>
<td>57.6 (29.9–85.83)</td>
<td>51.0 (31.3–82.8)</td>
<td>&lt;0.5</td>
</tr>
<tr>
<td>17-OHP (µmol/l)</td>
<td>3.5 (1.5–4.0)</td>
<td>3.6 (1.8–4.5)</td>
<td>&gt;0.5</td>
</tr>
<tr>
<td>TSH (mIU/l)</td>
<td>2.5 (1.3–4.1)</td>
<td>2.5 (1.5–3.8)</td>
<td>&gt;0.5</td>
</tr>
<tr>
<td>TG (mmol/l)</td>
<td>0.8 (0.6–1.2)</td>
<td>1.1 (0.8–1.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>CHOL (mmol/l)</td>
<td>4.8 (4.1–5.1)</td>
<td>5.0 (4.3–5.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>FG (mmol/l)</td>
<td>4.7 (4.4–5.0)</td>
<td>5.0 (4.7–5.3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>FI (µU/ml)</td>
<td>4.9 (3.3–7.0)</td>
<td>7.5 (4.6–12.0)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>1.0 (0.7–1.6)</td>
<td>1.7 (1.0–2.7)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Variables were expressed as median with (25–75th percentile). BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; Testosterone, total testosterone; FT, free testosterone; FAI, free androgen index= (T×100)/SHBG; SHBG, sex hormone-binding globulin; 17-OHP, 17-hydroxyprogesterone; TG, triglycerides; CHOL, total cholesterol; FG, fasting glucose; FI, fasting insulin; HOMA-IR, homeostasis model assessment of insulin resistance.
(n=97), 88.7% (n=86) were overweight and obese, and 11.3% (n=11) were of normal weight.

The PCOS patients were divided into four age groups and the prevalence of MetS for four age groups was presented in Table 3. The risk of having MetS in the older group (≥35 years) was nine times higher than that in the younger group (20–24 years; \( P<0.0001 \)). When these PCOS patients were divided into three age groups (<30 years, 30–39 years, ≥40 years), the prevalence of MetS was 12.7% (54/425), 26.8% (40/149), and 75.0% (3/4) \( (P<0.0001) \) respectively. This indicated that the prevalence of MetS increased with age.

### Predicting factors for MetS by multivariables regression analysis

Because of the statistically significant differences in variables of age, BMI, SHBG, FT, FAI, LH, and FI between PCOS patients with MetS and without MetS \( (P<0.0001) \), multivariables logistic regression analyses were performed to estimate the OR of the MetS using these variables as covariables, and the presence of MetS as the dependent variable. Two multiple logistic regression models were run using the same covariates, with the inclusion or absence of age (Table 4). All covariables were considered to be continuous variables. In the first model, only BMI \( (P<0.0001) \) was included in the equation and the OR was 1.451 (95% confidence interval, CI 1.298–1.622). After adjusting for age in model 2, the OR for BMI barely changed (1.413; 95% CI, 1.259–1.586); furthermore, only age and BMI were included in the second model, and the OR for age was 1.134 (95% CI 1.044–1.231; \( P=0.003 \)).

### Discussion

In this study, the prevalence of MetS, as diagnosed according to IDF criteria, was 16.8% in the studied Chinese PCOS patients. Metabolic disorders were common in these Chinese women as more than half of...
them (60.7%) exhibited at least one component of MetS. Dyslipidemia and obesity were the two most common metabolic features. The rates of IFG and raised blood pressure were relatively low, but they were over three times as high as observed in age- and BMI-matched controls. This suggested that the risk of having metabolic disorders was significantly higher in PCOS patients compared with age- and BMI-matched controls.

Apart from the impact of different criteria for MetS used (27, 28), the prevalence of MetS detected in our population of PCOS patients differed from the results that have been documented for other ethnicities and nationalities, and this to some extent might be due to different genetics and lifestyles or dietary structures (29, 30). The prevalence of MetS was 43.0–47.3% in USA (8, 9, 11) by NCEP-ATP III criteria. Even based on the same IDF criteria, the prevalence of MetS still differed between ethnicities, with 33.8% in Germany (31), 40.0% in Australia (27), 46.2% in India (14), and 35.3% in Thailand (17). In the case of Thailand, the prevalence of MetS was 22.5%, 27.8%, and 53.5% in three age groups (<25, 25–30, and <30 years) respectively, which was higher than those observed in similar age groups in our study. In Taiwan, the prevalence of MetS in PCOS patients based on IDF criteria was 16.0% (16/107) (32), similar to our results. Another factor, which contributed to the lower prevalence of MetS in our population of PCOS patients, was BMI. The BMI values in the Chinese PCOS patients were usually lower and central obesity less popular than other ethnicities (30, 33–35), whereas central obesity is one of the main contributors to developing the MetS. In our study, mean BMI was also lower than those in America, Germany, Australia, India, and Thailand. In addition, the size of studied sample may have had an effect on the difference of the prevalence of MetS. The prevalence of MetS increased with BMI in the present study. After adjustment by age, the increasing trend in this prevalence remained. Among the underweight group, none suffered from MetS. Nearly 90% of PCOS patients with MetS belonged to overweight or obesity.

### Table 3

<table>
<thead>
<tr>
<th>Variables</th>
<th>PCOS (n)</th>
<th>PCOS with MetS</th>
<th>Prevalence of MetS (%)</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 1 (≥20 and &lt;25)</td>
<td>123</td>
<td>9</td>
<td>7.3</td>
<td>9.3 (3.5–24.6)(^a)</td>
</tr>
<tr>
<td>Group 2 (≥25 and &lt;30)</td>
<td>302</td>
<td>45</td>
<td>14.9</td>
<td>2.2 (1.4–7.4)(^b)</td>
</tr>
<tr>
<td>Group 3 (≥30 and &lt;35)</td>
<td>120</td>
<td>29</td>
<td>24.2</td>
<td>1.8 (1.1–3.1)(^c)</td>
</tr>
<tr>
<td>Group 4 (≥35)</td>
<td>33</td>
<td>14</td>
<td>42.4</td>
<td>2.3 (1.0–5.2)(^d)</td>
</tr>
<tr>
<td>BMI (kg/m(^2))</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Underweight</td>
<td>74</td>
<td>0</td>
<td>0</td>
<td>–</td>
</tr>
<tr>
<td>Normal weight</td>
<td>282</td>
<td>11</td>
<td>3.9</td>
<td>6.3 (2.8–13.8)(^e)</td>
</tr>
<tr>
<td>Overweight</td>
<td>89</td>
<td>18</td>
<td>20.2</td>
<td>4.1 (2.2–7.7)(^f)</td>
</tr>
<tr>
<td>Obesity</td>
<td>133</td>
<td>68</td>
<td>51.1</td>
<td>25.8 (12.9–51.5)(^g)</td>
</tr>
</tbody>
</table>

BMI, body mass index; Underweight, BMI <18.5; Normal weight, BMI ≥18.5 and <23; Overweight, BMI ≥23 and <25; Obesity, BMI ≥25.
\(^a\)OR (95% CI), group 4 versus group 1 (P <0.0001).
\(^b\)OR (95% CI), group 2 versus group 1 (P <0.05).
\(^c\)OR (95% CI), group 3 versus group 2 (P <0.05).
\(^d\)OR (95% CI), group 4 versus group 3 (P <0.05).
\(^e\)OR (95% CI), overweight versus normal weight group (P <0.0001).
\(^f\)OR (95% CI), obesity versus overweight group (P <0.0001).
\(^g\)OR (95% CI), obesity versus normal weight group (P <0.0001).

### Table 4

<table>
<thead>
<tr>
<th>Model</th>
<th>Covariables</th>
<th>Coefficient (B)</th>
<th>OR (Exp(B)) (95% CI)</th>
<th>Wald ((\chi^2))</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>BMI</td>
<td>0.372</td>
<td>1.451 (1.298–1.622)</td>
<td>42.802</td>
<td>0.000</td>
</tr>
<tr>
<td></td>
<td>LH</td>
<td>−0.020</td>
<td>0.980 (0.921–1.042)</td>
<td>0.419</td>
<td>0.518</td>
</tr>
<tr>
<td></td>
<td>FT</td>
<td>0.011</td>
<td>1.011 (0.947–1.080)</td>
<td>0.106</td>
<td>0.744</td>
</tr>
<tr>
<td></td>
<td>FAI</td>
<td>0.039</td>
<td>1.040 (0.983–1.101)</td>
<td>1.842</td>
<td>0.175</td>
</tr>
<tr>
<td></td>
<td>SHBG</td>
<td>−0.001</td>
<td>0.999 (0.988–1.010)</td>
<td>0.039</td>
<td>0.884</td>
</tr>
<tr>
<td></td>
<td>FI</td>
<td>0.039</td>
<td>1.040 (0.995–1.086)</td>
<td>3.054</td>
<td>0.081</td>
</tr>
<tr>
<td>2</td>
<td>BMI</td>
<td>0.346</td>
<td>1.413 (1.259–1.586)</td>
<td>34.408</td>
<td>0.000</td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>0.125</td>
<td>1.134 (1.044–1.231)</td>
<td>8.868</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td>LH</td>
<td>−0.012</td>
<td>0.988 (0.927–1.052)</td>
<td>0.146</td>
<td>0.702</td>
</tr>
<tr>
<td></td>
<td>FT</td>
<td>0.032</td>
<td>1.033 (0.965–1.105)</td>
<td>0.874</td>
<td>0.350</td>
</tr>
<tr>
<td></td>
<td>FAI</td>
<td>0.045</td>
<td>1.046 (0.986–1.111)</td>
<td>2.206</td>
<td>0.137</td>
</tr>
<tr>
<td></td>
<td>SHBG</td>
<td>−0.001</td>
<td>0.999 (0.989–1.010)</td>
<td>0.010</td>
<td>0.922</td>
</tr>
<tr>
<td></td>
<td>FI</td>
<td>0.043</td>
<td>1.044 (0.996–1.093)</td>
<td>3.234</td>
<td>0.072</td>
</tr>
</tbody>
</table>

BMI, body mass index; FT, free testosterone; FAI, free androgen index; SHBG, sex hormone-binding globulin; FI, fasting insulin. All covariables were treated as continuous variable. In model 1, only BMI was included; in model 2, both BMI and age were included.
obese groups. In multivariables logistic regressions, BMI was an independent risk factor for MetS; however, FT, FAI, SHBG, and FI were not included in the formulation by logistic regression analyses, which suggested that BMI is a key factor for developing MetS. Thus, controlling BMI appears to have the most potential in reducing the occurrence of the MetS in women with PCOS.

Age is regarded as an important risk factor for MetS in both general populations and PCOS patients (36–39). In our study, the increasing trend was also found in the prevalence of MetS in two different ways for age-grouping that was consistent with previous reports concerning the relationship between age and MetS in PCOS patients (11, 13). Between the two independent risk factors, BMI appeared to contribute more to MetS than age according to the logistic regressions in the present study. This may result from the fact that obesity plays a key role in IR, while IR is one of the most important mechanisms of metabolic syndrome.

Although the prevalence of MetS in the present study was lower than that observed in western and some Asian countries, it was still significantly higher than that in healthy counterparts. Since waist circumference values and HDL-C were not available for the controls, the prevalence of MetS could not be calculated according to IDF criteria. We had originally intended to compare our results with an age-matched study on the general population of mainland China based on IDF Criteria; however, no such study was found. As an alternative, we compared our results to a study in Hong Kong (40), in which the prevalence of MetS was ~2 and 8% in age groups of <30 years and 30–39 years respectively. The prevalence of MetS in PCOS patients aged <30 years in our study was almost five times higher than that of their age-matched healthy counterparts; in PCOS patients aged 30–39 years, the prevalence of MetS was over two times higher. This suggests that PCOS patients in China are also at a higher risk of having MetS than those without PCOS, a finding similar to that observed in other countries (12, 13, 16, 31).

The purpose of defining the MetS is to identify those at high risk of CVD and type 2 diabetes. However, documents revealed that the MetS was not superior to the measurement of glucose (fasting or 2-h post load) in identifying those who developed diabetes. Instead, a person with individual components of the MetS can still be associated with high risk of diabetes and CVD in the AusDiab study (41). In our study, we found a low prevalence of the MetS but a high occurrence of individual metabolic disorders in Chinese PCOS patients. Based on the conclusion reached in the AusDiab study, such patients without MetS but with individual components of MetS are at high risk of diabetes and CVDs. Therefore, it is meaningful in clinical practice to detect the various metabolic disorders in PCOS women.

In conclusion, we found a low occurrence of MetS, but high prevalence of the metabolic disorders in the Chinese PCOS patients. The prevalence of MetS increases with BMI and age, the two independent risk factors for predicting MetS, which appeared to contribute more to developing MetS than FT, FAI, FI, or SHBG in this cohort of PCOS patients. So, controlling BMI is not only beneficial to PCOS per se but also important for reducing the long-term detriment of MetS. Since PCOS patients without MetS but with individual components of MetS are also at risk of suffering from diabetes and CVD, it is worthwhile to evaluate the various components of MetS in PCOS patients, particularly in those who are overweight or older than 35 years.

Despite these relevant findings, it is important to point out the limitations of our study. Waist circumference measurements and HDL-C level were unavailable in the controls; therefore, the prevalence of MetS in women with PCOS could not be compared with that in controls from the same region. We look forward to evaluating this issue in a future paper. In addition, selection biases may have existed, since only those who had complete medical records were selected for inclusion in the study.

Declaration of interest

Authors have no possible conflict of interest to disclose.

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Author contribution statement

Each of us acknowledges that he or she participated sufficiently in the work.

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