ORIGINAL ARTICLE: INFERTILITY

Prevalence of polycystic ovary syndrome in Chinese obese women of reproductive age with or without metabolic syndrome

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Objective: To compare the prevalence of polycystic ovary syndrome (PCOS) and related clinical characteristics between metabolically unhealthy obese (MUO) and metabolically healthy obese (MHO) women of reproductive age.
Design: Cross-sectional clinical study.
Setting: Tertiary hospital.
Patient(s): We studied 299 MUO and 122 MHO Chinese women matched on body mass index. Metabolically healthy obese was defined as obesity with no more than one metabolic abnormality. Diagnosis of PCOS was based on the revised Rotterdam criteria.
Intervention(s): Each subject underwent physical examination, laboratory evaluation, and gynecologic ultrasound for a diagnosis of PCOS or metabolic syndrome (MetS).
Main Outcome Measure(s): Prevalence of PCOS was calculated in both groups. Insulin resistance was determined by homeostasis model assessment of insulin resistance or by the insulin sensitivity index derived from Bergman's minimal model. Fat distribution was measured with computerized tomography scan.
Result(s): Prevalence of PCOS and its components did not differ between MUO and BMI-matched MHO groups (67.89% and 66.96%, respectively). In logistic regression analysis, MetS did not predict the presence of PCOS after adjusting for confounding factors. The MHO group had lower visceral adipose tissue, relatively higher insulin sensitivity, and better β-cell function, compared with those in the MUO group; but there were no significant differences in sex hormones (except for free T and sex hormone-binding globulin) and ultrasound manifestations between MHO and MUO women.
Conclusion(s): For the first time, our findings suggest that MetS does not add additional risk for PCOS. In addition, we found that both MUO and MHO are associated with insulin resistance to some extent. (Fertil Steril® 2017;■:■–■. ©2017 by American Society for Reproductive Medicine.)
Key Words: Metabolically healthy obese, metabolic syndrome, polycystic ovary syndrome

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The prevalence of obesity is increasing worldwide. Obesity is associated with increased risk of developing comorbidities, including metabolic syndrome (MetS), type 2 diabetes mellitus (T2DM), and cardiovascular disease (CVD) (1). However, the metabolic disturbances may not be present in all obese individuals. The "metabolically healthy obese" (MHO) phenotype may exist in the absence of metabolic abnormalities, which has been recognized since the 1980s (2).

There is no consensus on how to define MHO, and its prevalence among the obese varies across studies, ranging from 10% to 40%, depending on which criteria are used (3–7). Similarly, there is conflicting evidence regarding the risk of T2DM, CVD, and mortality associated with this phenotype. Some studies suggested that MHO subjects were not at increased risk for diabetes, CVD, or mortality compared with normal-weight controls (7, 8), whereas others indicate that both MHO and metabolically unhealthy obesity (MUO) had an increased T2DM and CVD risk (9–12).

Polycystic ovary syndrome (PCOS), characterized by menstrual irregularities, hyperandrogenism, and polycystic ovaries, is one of the most common endocrine disorders among reproductive-aged women. Polycystic ovary syndrome shares many similarities with MetS in pathophysiology and clinical features, including abdominal obesity and insulin resistance. Obese women may be at higher risk for PCOS, with a prevalence of approximately 28.3% (13). However, there is currently no study regarding the prevalence of PCOS in MHO and MUO women. Because MHO phenotype seems to be associated with increased insulin sensitivity (14–20), and it is also well established that insulin resistance plays an important role in the pathophysiology of PCOS (21), we speculate whether the relatively healthy metabolic profile of MHO women has a protective effect against developing PCOS.

The aim of this study was to evaluate the prevalence of PCOS in MHO and MUO women and to examine the differences in their clinical characteristics. Through the above analysis we sought to determine whether MetS increases the risk of PCOS in Chinese obese women of reproductive age.

MATERIALS AND METHODS

Patients

This study was approved by the institutional review board at Shanghai Jiao-Tong University School of Medicine. From August 2008 to December 2015, 411 female obese patients (aged 18–40 years, body mass index [BMI] $\geq$30 kg/m^2) were enrolled from Shanghai Ruijin Hospital, Shanghai Jiaotong University School of Medicine. They were of Chinese ethnicity, living in the Shanghai region, and gave informed consent.

Diagnosis of MetS was based on the definitions proposed by the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) (22). The waist circumference (WC) criterion was not used because of its collinearity with BMI (10). Participants who met fewer than two of the following four criteria were considered metabolically healthy: [1] a systolic blood pressure $\geq$130 mm Hg and/or a diastolic blood pressure $\geq$85 mm Hg, or on antihypertensive treatment; [2] triglycerides $\geq$1.7 mmol/L; [3] fasting plasma glucose (FPG) $\geq$5.6 mmol/L; and [4] high-density lipoprotein cholesterol $<$1.3 mmol/L (in women).

Diagnosis of PCOS was based on the revised criteria of Rotterdam (23), which require the presence of at least two of the following three features: [1] chronic anovulation (CA): menstrual cycles at $\geq$35-day intervals or $<$10 bleeds per year, and polymenorrhea as $\leq$25 days; [2] biochemical hyperandrogenism (HA): total T or free T (FT) higher than reference intervals established in our laboratory or clinical manifestations of hyperandrogenism; [3] polycystic ovaries on ultrasound (PCO): $\geq$12 follicles (2–9 mm in diameter) in at least one ovary and/or ovarian volume $>$10 cm^3. All subjects with PCOS can be categorized into four phenotypes: [1] phenotype A, HA and CA and PCO; [2] phenotype B, HA with CA only; [3] phenotype C, HA with polycystic ovary morphology only; and [4] phenotype D, CA with PCO only (24).

Exclusion criteria were pregnancy and other related disorders (congenital adrenal hyperplasia, thyroid dysfunction, Cushing syndrome, or hyperprolactinemia). All patients were free of medications known to affect gonadal or adrenal function, energy metabolism, or lipid metabolism within the 3 months preceding enrollment in the study, and with no history of smoking or excessive drinking, or no history of ovarian surgery.

After meeting the inclusion/exclusion criteria, 112 obese women were classified into the MHO group. On the basis of the average BMI of the MHO subjects, 299 MUO women were enrolled in our study, so that the average BMI gap between the two groups did not exceed 1 kg/m^2.

Anthropometric Measurements

A thorough medical history (including menstrual history) was recorded for each patient. Height and weight (light clothes and without shoes), waist and hip circumference, and seated blood pressure were determined by an experienced physician. Body fat percentage was measured by bioelectrical impedance analysis using Inbody720 Body Composition Analysis.

Biochemical Measurements

All blood samples were taken in the morning after an overnight fast of 10–12 hours. Liver enzymes (serum alanine aminotransferase [ALT], aspartate aminotransferase [AST], alkaline phosphatase, γ-glutamyl transferase); lipid profile, including triglycerides, total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and free fatty acids; and high-sensitivity C-reaction protein (hsCRP) were measured using an autoanalyzer (Beckman CX-7 Biochemical Autoanalyzer). Testosterone was analyzed using chemiluminescence immunoassay (Abbott). All measures were performed in Shanghai Institute of Endocrine and Metabolic Diseases. Westguard rules were followed for quality control procedures. The intra- and interassay coefficients of variation were 1.5%–4.9% and 3.1%–8.0%, respectively. Free T was analyzed using radioimmunoassay (Beckman Coulter). Other sex hormones, like DHEAS, sex hormone-binding globulin (SHBG), and PRL, were analyzed using chemiluminescence immunoassay (Abbott); 17α-hydroxyprogesterone and androstenedione were analyzed using radioimmunoassay (Beckman Coulter). Thyroid hormones and morning cortisol were measured. Immediately after baseline blood sampling an oral glucose tolerance test was performed; 75 g of glucose was administered orally, and serum glucose levels and plasma insulin levels were detected after 30, 60, 120, and 180 minutes. Serum insulin was measured by a radioimmunoassay (Sangon). Insulin resistance was

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