

Nutritional and Metabolic Characteristics of Overweight Women with Polycystic Ovary Syndrome (PCOS): A Case-Control Study

Caractéristiques Nutritionnelles et Métaboliques des Patientes Atteintes de Syndrome des Ovaires Polykystiques (SOPK) en Surcharge Pondérale : Une étude cas-témoin

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ARSTRACT

Introduction: Polycystic Ovary Syndrome (PCOS) is a common cause of menstrual disorders, infertility, and hyperandrogenism, often associated with insulin resistance and an increased risk of metabolic disorders. This study aimed to assess the metabolic and nutritional characteristics of overweight women with PCOS.

Methods: This was a descriptive, cross-sectional, and retrospective case-control study that included 61 overweight women, divided into two groups: 31 women with PCOS and 30 controls without PCOS. Data were gathered through review of the patients' medical records.

Results: High blood pressure, type 2 diabetes, dyslipidemia, and metabolic syndrome were significantly more frequent in the PCOS group compared to the control group (p<0.001; p<0.001; p=0.002; and p=<00001, respectively). Eating disorders were more frequently observed in women with PCOS, with a significantly higher occurrence of binge eating behaviors in these patients compared to the control group (p=0.009). The intake of saturated fatty acids was higher in women with PCOS (p=0.01). Regarding micronutrients, intakes of vitamin C and vitamin B1 were significantly lower in PCOS patients (p=0.01 and p=0.04, respectively). However, significantly more prevalent nutrient deficiencies in omega-6 fatty acids (p=0.001), vitamin C (p=0.002), vitamin B1 (p=0.03), vitamin B3 (p=0.003), vitamin B6 (p=0.03), and iron (p=0.02) were noted in the PCOS group. Conclusions: Women with PCOS exhibit a higher occurrence of cardio-metabolic risk factors and multiple nutritional deficiencies. Early and personalized management is crucial to improve their long-term health outcomes and quality of life.

Keywords: Polycystic Ovary Syndrome- Overweight- Obesity- Metabolic Syndrome Nutritional Intake- Eating Behavior

RÉSUMÉ

Introduction: Le syndrome des ovaires polykystiques (SOPK) est une cause fréquente de troubles du cycle menstruel, d'infertilité féminine et d'hyperandrogénie. Il est fréquemment associé à une insulinorésistance augmentant le risque de désordres métaboliques. Notre objectif était d'étudier les caractéristiques métaboliques et nutritionnelles des femmes en surcharge pondérale présentant un SOPK.

Méthodes: Il s'agissait d'une étude descriptive, transversale et rétrospective de type cas-témoins, incluant 61 femmes en surcharge pondérale qui ont été réparties en deux groupes: un groupe comprenant 31 patientes présentant un SOPK et un groupe témoin incluant 30 femmes sans SOPK. Les données ont été collectées à partir des dossiers médicaux des patientes.

Résultats: L'hypertension artérielle, le diabète de type2, la dyslipidémie et le syndrome métabolique étaient significativement plus fréquents dans le groupe SOPK par rapport au groupe témoin (p<0,001, p<0,001, p=0,002 et p<0,001, respectivement). Les troubles du comportement alimentaire étaient plus fréquemment observés chez les patientes SOPK avec une association significative avec les compulsions alimentaires (p=0,009). Les apports en acides gras saturés étaient plus élevés chez les femmes SOPK (p=0,01). Quant aux micronutriments, les apports en vitamine C et en vitamine B1 étaient significativement plus faibles chez les patientes présentant un SOPK (p=0,01 et p=0,04, respectivement). Des insuffisances d'apport significativement plus fréquentes en acides gras oméga6 (p=0,001), en vitamine C (p=0,002), en vitamine B1 (p=0,03), en vitamine B3 (p=0,00,3), en vitamine B6 (p=0,03) et en fer (p=0,02,) ont été notées dans le groupe SOPK.

Conclusions: Les patientes atteintes de SOPK présentent une occurrence plus élevée de facteurs de risque cardio-métaboliques et de multiples carences nutritionnelles. Une prise en charge précoce et personnalisée est essentielle pour améliorer leur pronostic et leur qualité de vie.

Mots clés: Syndrome des ovaires polykystiques - Surpoids - Obésité - Syndrome métabolique - Apports nutritionnels - Comportement alimentaire

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INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is one of the most common hormonal disorders among women of reproductive age. It represents a significant public health issue, affecting 8 to 13% of women of reproductive age according to the World Health Organization (WHO) with up to 70% of PCOS cases remaining undiagnosed (1). It is linked to multiple comorbidities, including obesity, insulin resistance, high blood pressure, and dyslipidemia, all of which increase the risk of cardiovascular diseases in affected women (2).

Its etiopathogenesis is complex, involving an interaction between genetic, epigenetic, and environmental factors. Lifestyle factors, such as a Western diet high in refined sugars, saturated fats, and physical inactivity, are key contributors to obesity and can disrupt reproductive and metabolic functions (3). Insulin resistance, oxidative stress, and inflammation play crucial roles in the development of PCOS, while deficiencies in micronutrients may further exacerbate its manifestations (4).

Despite the increasing recognition of PCOS and its potential complications, limited research has focused specifically on its metabolic and nutritional associated factors in Tunisia. This study aimed to investigate the metabolic and nutritional characteristics of a group of overweight women with PCOS.

METHODS

Study design and setting

We conducted a cross-sectional, descriptive, and retrospective case-control study at the Obesity Research Unit, Zouhair Kallel Institute of Nutrition and Food Technology, Tunis, Tunisia, from January 2 to May 31, 2024.

Study population

The study included 61 overweight women, divided into two groups: 31 women with PCOS (Group 1) and 30 women without PCOS (Group 2). The two groups were matched for age and body mass index (BMI). Adult women aged 18 years or older, overweight (BMI ≥ 25 kg/ m²), and not in menopause, were included in this study. The inclusion criteria for Group 1 were the presence of PCOS, defined according to the International Evidencebased Guideline for the Assessment and Management of Polycystic Ovary Syndrome (2023), with at least two of the following criteria: menstrual cycle disorders, clinical hyperandrogenism (acne, hirsutism and/or androgenic alopecia) and/or biochemical hyperandrogenism (investigated by measuring total and free testosterone levels), and/or ultrasound features (defined as an ovarian volume greater than 10 ml and/or the presence of more than 12 follicles) (2). The diagnosis of PCOS was made after exclusion of other etiologies of hyperandrogenism and menstrual irregularities, including endocrine disorders and tumoral pathologies. The PCOS phenotype was classified into four main types (A, B, C, and D) based on the presence of specific criteria: hyperandrogenism, oligo-anovulation and polycystic ovarian morphology (5). For Group 2, inclusion criteria were the absence of clinical signs of hyperandrogenism, the absence of menstrual disturbances, and the absence of known fertility disorders. Were not included pregnant or breastfeeding women, and postmenopausal women. A consecutive sample of the first 61 patients consulting the Obesity Research Unit and meeting the inclusion criteria was enrolled in the study.

Data collection

We gathered all the required data through review of the medical records of the included patients, covering generals background information, as well as specific details regarding family history of metabolic disorders, obesity-related complications, current treatments, and weight history. Socioeconomic status was assessed based on the family's monthly income, classified as low (<1000 DT/month), middle (1000–3000 DT/month), or high (>3000 DT/month).

We assessed all patients for the presence of eating behavior disorders (bulimia, binge eating disorder, night eating syndrome, snacking, and food compulsions), based on the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) (6). The level of physical activity was assessed using the Ricci and Gagnon auto-questionnaire provided by the French National Authority for Health for adult patients (7). Dietary Intake was measured by 24-h recall method. Food intake of each patient was analyzed for energy, macronutrients, and micronutrients intake by using Nutrilog software which is based on the CIQUAL 2020 composition table validated and provided by the ANSES (8). Anthropometric measurements were collected and body composition was interpreted according to the WHO classification (9). Abdominal obesity was defined by a WC exceeding 80 cm. Biochemical analyses included glycemic and lipidic parameters. HOMA-IR was used to evaluate insulin resistance, with a value greater than 2.4 defining the presence of insulin resistance (10).

Metabolic syndrome was defined based on the diagnostic criteria established by the International Diabetes Federation (11). Diabetes was diagnosed in patients who were either treated with antidiabetic medications or met the criteria outlined by the American Diabetes Association in their 2025 guidelines (12). Dyslipidemia was identified following the 2019 recommendations from the European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS) (13). High Blood Pressure was defined according to the guidelines published in 2020 by the International Society of Hypertension (14).

Statistical analysis

Data were analyzed using SPSS 26. Descriptive statistics are presented as means and standard deviations for continuous variables, frequencies and proportions for categorical variables. For comparisons, the Pearson's Chi-squared test was used for categorical variables, while

the independent samples t-test was used for continuous variables. A p-value of <0.05 was considered statistically significant.

Ethical considerations

We ensured the confidentiality of the personal and medical data of the participants in accordance with ethical guidelines and data protection regulations. This work has no conflicts of interest. Written informed consent was obtained from all participants prior to their inclusion in the study.

RESULTS

The comparison between the two groups based on the general characteristics are shown in Table 1.

Table 1. Comparison of the general characteristics between PCOS group and control group

| | Group 1 PCOS N = 31 | Group 2 Control N=30 |
|---|---------------------------|----------------------------|
| Age (years) | 31 ± 8.6 | 34.23 ± 10.6 |
| Socioeconomic level, % High Middle Low | 13 77 10 | 23 50 27 |
| Smokers, % | 0 | 11 |
| Physical activity level, % Inactive | 52 | 77 |
| Active | 48 | 23 |
| Very active | 0 | 0 |
| Obesity class, % | | |
| Class 1 | 23 | 19 |
| Class 2 | 54 | 47 |
| Class 3 | 13 | 34 |

All patients in the PCOS group exhibited at least one clinical manifestation of hyperandrogenism and 66% of them had biochemical hyperandrogenism. Amenorrhea, oligomenorrhea, and infertility were observed in 54%, 47%, and 19% of cases, respectively. All PCOS patients met the ultrasonographic criteria for PCOS.

Only phenotypes A and C of PCOS were observed in the studied population, accounting for 81% and 19% of the patients, respectively. Among the PCOS patients, 28% were on metformin, and 44% were on dydrogesterone.

The comparison between the two groups based on clinical and biological metabolic parameters is presented in Table 2.

Among diabetic patients with PCOS, 28% were treated with Metformin while 72% were on lifestyle measures. As for dyslipidemia, none of the patients were receiving treatment.

Eating disorders (ED), of all types combined, were observed in 58% of patients with PCOS and 42% of patients in the control group (p=0.15), with a significantly higher frequency of binge eating in patients with PCOS (61% vs 30%, p=0.009).

The comparison of macronutrient and micronutrient intakes between the two groups is shown in Tables 3 and 4. The average total energy intake (TEI) and fiber intake were comparable between the two patient groups (p=0.32 and p=0.55, respectively).

Table 2. Comparison of clinical and biological metabolic parameters between PCOS group and control group

| | Group 1 PCOS N = 31 | Group 2 Control N=30 | p value |
|----------------------------|---------------------------|----------------------------|---------|
| Diabetes, n (%) | 30 (97) | 4 (12) | <0.001 |
| High blood pressure, n (%) | 28 (90) | 6 (19) | <0.001 |
| Dyslipidemia, n (%) | 28 (90) | 16 (54) | 0.002 |
| Metabolic syndrome, n (%) | 30 (97) | 16 (54) | <0.001 |
| Insulin resistance, n (%) | 29 (93) | 26 (88) | 0.65 |
| FBG (mmol/l) | 5.21 ± 0.6 | 6.3 ± 2.1 | 0.06 |
| HbA1c (%) | 5.4 ± 0.5 | 5.7 ± 0.9 | 0.13 |
| HOMA-IR | 6.2 ± 4 | 5.9 ± 4.7 | 0.81 |
| CT (mmol/I) | 4.5 ± 0.8 | 5.3 ± 1.7 | 0.03 |
| TG (mmol/l) | 1.2 ± 0.7 | 2.1 ± 4 | 0.20 |
| HDLc (mmol/l) | 1.2 ± 0.24 | 1.24 ± 0.24 | 0.24 |
| LDLc (g/l) | 1.1 ± 0.3 | 1.2 ± 0.27 | 0.23 |

PCOS: Polycystic Ovary Syndrome; FBG: fasting blood glucose; HbA1c: A1c hemoglobin; TC: total cholesterol level; TG: triglyceride level; HDLc: HDL cholesterol level; LDLc: low-density lipoprotein cholesterol level

Table 3. Macronutrient intake in PCOS group and control group

| | Group 1 PCOS N = 31 | Group 2 Control N=30 | p value |
|------------------------------------|---------------------------|----------------------------|---------|
| Proteins (g/kg of ABW/d) | 1.6 ± 0.4 | 1.3 ± 0.4 | 0.07 |
| Protein deficiency intake, n (%) | 3 (10) | 1 (4) | 0.37 |
| Carbohydrates (g/kg of ABW/d) | 5.9 ± 2.2 | 5.3 ± 2.2 | 0.28 |
| Excess carbohydrates intake, n (%) | 21 (68) | 14 (46) | 0.08 |
| Simple sugar intake (% TEI) | 0.6 ± 0.9 | 2.45 ± 0.9 | <0.01 |
| Excess simple sugar intake, n (%) | 13 (42) | 17 (58) | 0.17 |
| Fat intake (g/kg of ABW/d) | 2 ± 0.9 | 1.8 ± 0.8 | 0.30 |
| Excess fat intake, n (%) | 27 (87) | 26 (88) | 0.60 |
| MUFA intake (% TEI) | 15.3 ± 6.4 | 15.6 ± 5.8 | 0.84 |
| PUFA intake (% TEI) | 10.4 ± 3.7 | 9.4 ± 4.4 | 0.37 |
| SFA intake (% TEI) | 10.2 ± 2.8 | 8.2 ± 2.9 | 0.01 |
| Excess SFA intake, n (%) | 11 (36) | 5 (16) | 0.09 |
| W6 deficiency intake, n (%) | 18 (58) | 2 (5) | 0.001 |
| W3 deficiency intake, n (%) | 29 (93) | 29 (95) | 0.6 |
| Cholesterol intake (mg/j) | 282 ± 247 | 265 ± 176 | 0.77 |
| Excess cholesterol intake, n (%) | 10 (32) | 12 (39) | 0.79 |

PCOS: Polycystic Ovary Syndrome; ABW: adjusted body weight; TEI: total energy intake; SFA: saturated fatty acid; MUFA: monounsaturated fatty acid; PUFA: polyunsaturated fatty acid; W6: linoleic fatty acid; W3: linolenic fatty acid

Table 4. Micronutrient intake in PCOS group and control group

| | Group 1 PCOS N = 31 | Group 2 Control N=30 | p value |
|--|---------------------------|----------------------------|---------|
| Vitamin A intake (μg/d) | 228 ± 241 | 209 ± 246 | 0.77 |
| Vitamin A deficiency intake, n (%) | 28 (90) | 29 (95) | 0.46 |
| Vitamin D intake (μg/d) | 8.9 ± 1.9 | 8.5 ± 1.25 | 0.57 |
| Vitamin D deficiency intake, n (%) | 31 (100) | 29 (96) | 0.27 |
| Vitamin E (mg/d) | 22.8 ± 15.2 | 27.2 ± 16.6 | 0.32 |
| Vitamin C intake (mg/d) | 91 ± 69 | 142 ± 85 | 0.01 |
| Vitamin C deficiency intake, n (%) | 20 (64) | 7 (23) | 0.002 |
| Vitamin B1 intake (mg/d) | 1.03 ± 0.32 | 1.24 ± 0.42 | 0.04 |
| Vitamin B1 deficiency intake, n (%) | 9 (29) | 0 | 0.03 |
| Vitamin B2 intake (mg/d) | 1.47 ± 0.5 | 1.28 ± 0.53 | 0.18 |
| Vitamin B2 deficiency intake, n (%) | 17 (55) | 13 (42) | 0.32 |
| Vitamin B3 intake (mg/d) | 17.3 ± 6.4 | 17.1 ± 7.6 | 0.94 |
| Vitamin B3 deficiency intake, n (%) | 9 (29) | 0 | 0.003 |
| Vitamin B6 intake (mg/d) | 1.7 ± 0.54 | 1.8 ± 0.71 | 0.56 |
| Vitamin B6 deficiency intake, n (%) | 11 (35) | 4 (12) | 0.03 |
| Vitamin B9 intake (mg/d) | 320 ± 126 | 351 ± 99 | 0.31 |
| Vitamin B9 deficiency intake, n (%) | 12 (39) | 13 (42) | 0.78 |
| Magnesium intake (mg/d) | 341 ± 115 | 333 ± 94 | 0.78 |
| Magnesium deficiency intake, n (%) | 7 (23) | 6 (19) | 0.46 |
| Calcium intake (mg/d) | 650 ± 230 | 633 ± 241 | 0.78 |
| Calcium deficiency intake, n (%) | 27 (87) | 20 (65) | 0.05 |
| Potassium intake (mg/d) | 3165 ± 841 | 2803 ± 754 | 0.09 |
| Potassium deficiency intake, n (%) | 13 (42) | 16 (53) | 0.47 |
| Iron intake (mg/d) | 11.6 ± 4.34 | 11.3 ± 3.6 | 0.74 |
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Discussion

While numerous studies have investigated the factors associated with PCOS worldwide, research on this condition remains notably scarce in Tunisia.

Our study revealed that all patients in the PCOS group exhibited high blood pressure, type 2 diabetes, dyslipidemia, and metabolic syndrome. These metabolic abnormalities were significantly more prevalent in the PCOS group compared to the control group. In contrast, insulin resistance was observed in the majority of patients, regardless of PCOS status. These findings were in agreement with several studies (2,15,16). Insulin resistance has been identified as the pathophysiological mechanism contributing to the increased risk of these metabolic abnormalities in women with PCOS, with a higher prevalence and earlier onset compared to the general population (17). In a study conducted by Ezeh et al. more than 80% of women with PCOS presented

insulin resistance with compensatory hyperinsulinemia, and approximately 31% to 35% of cases developed prediabetes, while 7.5% to 10% developed type 2 diabetes (18). However, some authors suggest that dyslipidemia is the most common metabolic disorder in PCOS, affecting 70% of patients. Most studies report a decrease in HDL cholesterol and an increase in triglyceride levels, a lipid profile known to be associated with insulin resistance (19). Insulin resistance and hyperinsulinemia, further exacerbated by obesity, intensify metabolic abnormalities and elevate the risk of long-term cardiometabolic complications, including dyslipidemia, metabolic syndrome, and type 2 diabetes.

In addition to the metabolic abnormalities associated with insulin resistance, cardiovascular risk seems to vary by phenotype, with higher prevalence of risk factors in patients with phenotypes A and B compared to other PCOS phenotypes (20). Phenotypes A and B are characterized by a combination of hyperandrogenism and ovulatory dysfunction, which together exacerbate the risk of developing cardiovascular diseases. Thus, patients with the classical PCOS phenotype A, predominant in our population, are exposed to multiple cardiovascular risk factors over their lifetime, increasing the likelihood of cardiovascular events.

In our study, ED were more frequently observed in women with PCOS, with a significantly higher occurrence of binge eating behaviors in these patients compared to the control group. In literature, the prevalence of eating disorders in women with PCOS ranged from 2.8% to 23.3%, depending on the diagnostic tools used (21). Cesta and al. in their study showed in their study that the risk of ED in women with PCOS was 1.43 times higher (CI: 1.31-1.56) compared to women without PCOS (22). This higher prevalence could be linked to several factors. Previous research has shown that women with PCOS are at higher risk for anxiety and depressive disorders, both of which are independently associated with ED (22,23). Moreover, clinical features of PCOS, such as hirsutism, menstrual irregularities, infertility, and obesity, are linked to low self-esteem and body dissatisfaction, factors that are well-known risk factors for ED.

This study found no significant differences in macronutrient, cholesterol, or fiber intake between women with PCOS and those without. However, excessive intake of SFA was more frequently observed in patients with PCOS. In contrast, deficiencies in omega-6 polyunsaturated fatty acids were significantly more common in the PCOS group compared to the control group (58% vs 5%, p=0.001). Some studies suggest that a pro-inflammatory state induced by lipids may contribute to insulin resistance, hyperandrogenism, and dyslipidemia in PCOS, with these abnormalities more closely linked to saturated fatty acid intake (24). This suggests that optimizing macronutrient distribution, regardless of its impact on weight, could help reduce low-grade inflammation. In opposition to the findings of other studies, the intake of added sugars was significantly lower in the PCOS group of our study (25). This discrepancy may be due to the fact that most of our PCOS patients had dyslipidemia and were likely receiving nutritional education, which included recommendations to reduce added sugar consumption.

As for vitamin intake, our results revealed that the average intake of vitamin C and vitamin B1 was significantly lower in the PCOS group compared to the control group (p=0.01 and p=0.04, respectively). A significantly higher frequency of deficiencies in vitamin C, vitamin B1, vitamin B3, and vitamin B6 intakes was observed in these patients. The literature data regarding vitamin intake in women with PCOS remains limited. According to some studies, deficiencies in riboflavin, vitamin B6, and vitamin B12 may influence fertility, likely by affecting reproductive hormones (26). Additionally, Gaskings and al. found that increased dietary folate intake reduces the risk of sporadic anovulation, likely by lowering homocysteine levels (27). This implies that folate deficiencies could worsen ovulatory disorders in women with PCOS.

In our study, we observed a higher frequency of deficiencies in calcium intake (p=0.05) and iron intake (p=0.02). Various trace elements, such as iron, zinc, copper, selenium, manganese, iodine, and chromium are essential for oogenesis, oocyte maturation, ovulation, and oocyte function (28). Deficiencies in these elements can significantly disrupt oogenesis, reduce oocyte quality, and contribute to infertility. Sun et al. studied the relationship between trace element levels in follicular fluid and PCOS, finding a global alteration in trace element profiles among PCOS patients. Copper, manganese, and calcium were most affected, with elevated copper levels being particularly significant, potentially influencing follicle development through an impact on steroidogenesis (29). Other studies have shown that manganese deficiencies impair follicular maturation, while chronic iodine deficiency affects ovarian reserve and ovulation frequency (30). However, excessive intake of zinc and iron has been linked to increased oxidative stress, reduced ovarian volume, lower serum sex hormone levels, and impaired follicular development (28).

Larger-scale studies are needed to assess micronutrient intake in women with PCOS, including the impact of both deficiencies and excesses on metabolic and hormonal abnormalities, as well as the effects of nutritional supplementation on these issues.

Our study adds to the national data on polycystic ovary syndrome. However, it has some limitations. The relatively small number of patients included and the cross-sectional design of the study limit our ability to establish causal relationships and draw definitive conclusions.

Conclusions

PCOS is a prevalent endocrine and metabolic disorder associated with various comorbidities, including insulin resistance, high blood pressure, dyslipidemia, and metabolic syndrome. Our study highlights the frequent occurrence of these metabolic abnormalities in women with PCOS. Moreover, eating disorders, particularly binge eating behaviors, were more frequently observed in the PCOS group. Significant deficiencies in key micronutrients such as vitamin C, vitamin B1, vitamin B3, vitamin B6 and iron were also noted. The findings also suggest that

certain dietary patterns, including excessive intake of saturated fatty acids, may contribute to the inflammatory and metabolic abnormalities observed in these patients. It is crucial to educate women with PCOS about these metabolic complications and to promote the adoption of a healthy lifestyle, including a balanced diet, regular physical activity, and stress management. Given the complexities of PCOS and its impact on both metabolic and reproductive health, further large-scale studies are needed to better understand the nutritional needs of these women and the potential benefits of targeted interventions.

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