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CORRELATION BETWEEN SERUM, URIC ACID AND BODY FAT DISTRIBUTION IN PATIENT WITH POLYCYSTIC OVARY SYNDROME

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ABSTRACT

Objective: This study aimed to investigate the relationship between serum uric acid levels and body fat distribution in women diagnosed with Polycystic Ovary Syndrome. **Methods:** A cross-sectional study was conducted at Tikrit Teaching Hospital from February 2023 to December 2024. A total of 60 women diagnosed with Polycystic Ovary Syndrome, based on the Rotterdam diagnostic criteria, were recruited. Body fat distribution was assessed using the **InBody** device, alongside measurements of serum uric acid levels, body mass index, waist-to-hip ratio, and various metabolic parameters. Participants were categorized into two groups: a hyperuricemia group consisting of 17 individuals and a non-hyperuricemia group with 43 individuals. **Results:** The study findings revealed that women with hyperuricemia exhibited significantly higher body weight, body mass index, and waist-to-hip ratio compared to those without hyperuricemia. Elevated serum uric acid levels were also associated with increased accumulation of visceral fat, higher blood pressure, and greater insulin resistance. Additionally, the lipid profile analysis demonstrated that women in the hyperuricemia group had **elevated triglyceride levels and reduced high- density lipoprotein cholesterol levels**, both of which are indicative of an increased risk for cardiovascular diseases. **Conclusion:** This study established a strong correlation between elevated serum uric acid levels and adverse metabolic outcomes in women with Polycystic Ovary Syndrome, including abnormal fat distribution and an increased risk of cardiovascular diseases. Implementing **comprehensive treatment strategies** that address both serum uric acid reduction and metabolic improvements may significantly benefit these patients.

KEYWORDS: Polycystic Ovary Syndrome, serum uric acid, body fat distribution, metabolic health, insulin resistance, hyperuricemia, cardiovascular risk.

INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is among the most commonly encountered endocrinopathies in women of reproductive age. Hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology are its defining features.[1] These manifestations not only affect reproductive health but are also closely associated with metabolic disturbances that predispose women to long-term complications such as type 2 diabetes mellitus (T2DM) and cardiovascular disease (CVD). Recent research has focused on the molecular mechanisms underpinning PCOS pathology, particularly metabolic dysfunction, which exacerbates reproductive abnormalities.[2] Obesity, insulin resistance, and dyslipidemia are the most prominent metabolic abnormalities associated with PCOS. These metabolic disorders, common among PCOS patients, significantly contribute to cardiometabolic diseases, making PCOS a critical public health issue. Accordingly, understanding the links between metabolic health markers and PCOS has become increasingly important, particularly as rising global obesity rates continue to drive the prevalence of this syndrome.[3]

Recent evidence suggests that serum uric acid (SUA) levels may play a role in the metabolic disturbances associated with PCOS. Traditionally linked to gout and kidney stones, elevated SUA levels have recently been

associated with metabolic syndrome, insulin resistance, and cardiovascular diseases.[4] Uric acid, the final product of purine metabolism, accumulates in the blood and has pro- inflammatory and pro-oxidative effects, contributing to endothelial dysfunction, a precursor to atherosclerosis and other cardiovascular conditions.[5] In PCOS patients, already at risk for metabolic and cardiovascular diseases, elevated uric acid levels could exacerbate their condition, creating a more complex clinical picture. This raises important questions about whether SUA could serve as a biomarker for identifying PCOS patients at greater cardiovascular risk and whether lowering uric acid levels could offer a novel therapeutic approach.[6]

The relationship between uric acid and obesity is another critical aspect of PCOS management. Obesity, particularly abdominal or visceral obesity, is highly prevalent in women with PCOS and is often accompanied by elevated uric acid levels. Visceral fat, which accumulates around internal organs, is metabolically active and contributes to inflammation and insulin resistance, both key components of PCOS pathophysiology.[7] Additionally, adipose tissue releases adipokines and pro-inflammatory cytokines that drive uric acid production and worsen the metabolic profile of PCOS patients. Studies show that women with PCOS who have a higher body mass index (BMI) or waist-to-hip ratio (WHR) tend to have higher uric acid levels compared to non-obese counterparts. These findings underscore the importance of understanding the interaction between obesity, SUA levels, and the metabolic disturbances characteristic of PCOS.[8]

Insulin resistance, affecting roughly 70% of women with PCOS, is closely linked to hyperandrogenism and obesity, creating a vicious cycle in which elevated insulin levels stimulate ovarian androgen production, exacerbating hormonal imbalances.[9] This imbalance contributes to clinical features such as hirsutism, acne, and irregular menstrual cycles, and leads to the development of T2DM and worsens cardiovascular risk factors like hypertension and dyslipidemia. Uric acid is thought to contribute to insulin resistance by promoting oxidative stress and inflammation, impairing the body's ability to utilize insulin effectively.[10] Elevated SUA levels have been linked to higher fasting insulin levels and greater insulin resistance in both the general population and PCOS patients. Thus, investigating the role of SUA in PCOS could offer valuable insights into the mechanisms linking PCOS to increased cardiometabolic risks.[11]

Chronic low-grade inflammation, a hallmark of both PCOS and metabolic syndrome, is another area where uric acid may play a crucial role. Women with PCOS often have elevated levels of inflammatory markers, such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α). High levels of uric acid can act as a danger signal, promoting the release of these inflammatory cytokines from immune cells and adipose tissue.[12] This inflammatory response not only worsens insulin resistance but also increases the risk of endothelial dysfunction and cardiovascular events. Thus, elevated SUA levels could exacerbate the inflammatory processes driving metabolic and cardiovascular complications in PCOS patients.[13]

In addition to its role in inflammation and insulin resistance, uric acid has been implicated in the development of hypertension, another common feature of PCOS. Hypertension in PCOS is often associated with obesity and insulin resistance, but recent evidence suggests uric acid may independently contribute to high blood pressure by impairing nitric oxide bioavailability, leading to vasoconstriction.[14] Hyperuricemia can also induce renal damage, further contributing to hypertension. In women with PCOS, already at increased risk of hypertension, elevated SUA levels could further heighten their cardiovascular risk. Addressing the role of uric acid in hypertension among PCOS patients could lead to more comprehensive management strategies.[15]

Despite growing interest in the role of uric acid in PCOS, its clinical significance remains underexplored. Most studies focus on SUA in metabolic syndrome and cardiovascular disease, with few specifically targeting PCOS. This leaves important questions unanswered, such as whether lowering uric acid levels could improve insulin sensitivity, reduce inflammation, and lower cardiovascular risk in PCOS patients.[16] Given the complex interplay between metabolic and reproductive abnormalities in PCOS, a multidisciplinary approach that includes monitoring and managing SUA levels could improve long-term health outcomes for these women.[17]

In conclusion, while PCOS is primarily considered a reproductive disorder, its metabolic consequences are far-reaching and significantly impact overall health. Uric acid, traditionally associated with gout and kidney disease, may play a more significant role in the metabolic disturbances of PCOS than previously thought.[18] By exploring the relationship between SUA levels and body fat distribution in PCOS patients, this study aims to uncover the potential mechanisms linking hyperuricemia to metabolic and cardiovascular complications in this population. A better understanding of these relationships could lead to more targeted and effective treatment strategies for women with PCOS, addressing both reproductive and metabolic health.[19]

Literature Review

2.1. Overview and Epidemiology of PCOS

PCOS is considered one of the most common endocrinopathies in women of reproductive age. PCOS is a complex disorder characterized by hyperandrogenism, ovulatory dysfunction and polycystic ovarian morphology with significant implications for reproductive, metabolic and psychological health.[20] PCOS is a complex disorder with multifactorial etiology including genetic, environmental and lifestyle factors that differently structure its phenotypic architecture in

various populations.[21] Prevalence of PCOS among women varies from 6% to 15% globally and is geographically considerably different. According to the diagnostic criteria of NIH, this prevalence ranges from 6–10% in Europe and the United States where hyperandrogenism should be associated with oligo/anovulation.[22] However, the prevalence increases to approximately 15% when a broader phenotypic definition (i.e., using NIH criteria and/or equivalent versions of the Rotterdam criteria that also include per-ultrasound polycystic ovarian morphology) is adopted.[23]

The high incidence of PCOS is worrisome due to its concomitant health risks, including metabolic syndrome, T2DM cardiovascular disease (CVD), and infertility. Insulin resistance represents an essential underlying mechanism of dysregulation in many women with PCOS, often also not clinically obese yet at a risk for metabolic derangements like Dyslipidemia and hypertension.[24] These metabolic abnormalities markedly increase the risk of CVD, which are even higher in association with central obesity and waist-to-hip ratio (WHR) that is often more increased in patients affected by PCOS.[25]

Furthermore, obesity — a known exacerbating factor of PCOS — only complicates the prevalence and severity. The prevalence of PCOS can be more than 20% in some subgroups both from high income countries, especially Western population with a higher rate of obesity.[26] Conversely, even if with lower prevalence rates in Asian populations than in Caucasians , the metabolic outcomes associated to PCOS like insulin resistance and T2DM are still considerable despite lower average BMI. Which indicates like the PCOS might be different and metabolic risks in ether ethnicity raised.questions.[27]

A number of epidemiologic studies have shown that PCOS has a strong familial predisposition. the syndrome tends to `cluster' in families, and first-degree relatives of affected women are at higher risk for developing the disorder.[28] There is also indirect evidence in twin studies, indicating a heritable component by showing a higher concordance rate among monozygotic twins (genetic material) than putative dizygotic twins. Nevertheless, the genetic determinants of PCOS are still not well established due to multiple candidate genes associated with insulin resistance, androgen production or inflammation.[29]

2.2. Pathophysiology of PCOS

Normal ovarian function includes a regulated menstrual cycle and production of hormones, including estrogen, progesterone; in smaller amountsm testosterone. In normal women, the HPO axis is active in tightly regulating these processes. In the case of PCOS this can cause a disruption to these systems, leading to hormonal imbalances affecting ovulation and fertility.[30]

1. Hormonal Imbalance: Women suffering from PCOS often have high levels of androgen hormones like testosterone, as well as dehydroepiandrosterone sulfate (DHEAS). Symptoms attributed to the high androgen levels include hirsutism, acne, alopecia.[31]
2. Insulin Resistance: In PCOS, 70% of the women have insulin resistance. This resistance leads to an increased production of insulin by the body in order for blood sugar levels to come down. Moreover, high elevations in insulin concentrations worsen hyperandrogenism through activation of intragonadal androgens production by ovarian thecal cells as well as reduction transcendental levels sex hormone-binding globulin (SHBG).[32]
3. Ovulatory Dysfunction: Hormone imbalances characteristic of PCOS can disrupt the standard growth and release (ovulation) of eggs from your ovaries. This can result in irregular or absent menstrual periods, which may make it hard for women with PCOS to get pregnant.[33]

2.3. Pathophysiology of PCOS

PCOS is related to multiple metabolic derangements including dyslipidemia, hypertension and impaired glucose tolerance which render PCOS as a high risk of being the part of metabolicsyndrome.[34]

1. Insulin resistance: as evidenced by hyperinsulinaemia is a salient feature of the pathophysiology of PCOS leading to androgen production. In this study, higher levels of insulin cause the ovaries to produce more androgens (like testosterone), leading to both ovulatory defectices as well hirsutism (facial hair) production by the skin if not only insulin is elevated but also direct signs op PCOS such as acne.[35]
2. Inflammation and Oxidative Stress: Women with PCOS have shown increased inflammatory markers such as C-reactive protein (CRP) level cytokines like TNF-α, IL-6. Visceral adipose tissue generates these pro- inflammatory mediators and promotes insulin resistance and a hyperandrogenic state. Oxidative stress, which is characterized by an imbalance between the production of reactive oxygen species (ROS) and endogenous antioxidants, intensifies this metabolic dysregulation.[36]
3. The abnormal adipose tissue distribution with greater visceral fat accumulation: has been identified as an important factor for the metabolic profile of women with PCOS. Visceral fat produces proinflammatory adipokines, such as inflammatory cytokine and chemotactic factor monocyte chemoattractant protein 1 (MCP-1), involved in systemic inflammation and metabolic dysfunction.[37]

2.4. Diagnosis and Investigations for PCOS

The diagnosis of PCOS consists of various clinical (history and physical examinations), laboratory tests, imaging studies.

The Rotterdam criteria, which require at least two out of three features:

1. Hyperandrogenism: Defined either clinically

(hirsutism, acne, or alopecia) or biochemically by hyperandrogenemia as indicated by abnormally elevated serum androgens such as testosterone.[38]

2. Ovulatory Dysfunction – this indicates that the menstrual cycles are either irregular or non-existent and implies ovulation is probably infrequent, if at all.[39]
3. Polycystic ovarian Morphology: characteristic of PCOS on ultrasound presence of 12 or more follicles in each ovary measuring 2-9 mm diameter and/or increased ovarian volume >10 ml.[40]

Laboratory Tests

The typical laboratory investigations used to assist in the diagnosis of PCOS include the following:

1. Serum Androgens: Measurements of testosterone and dehydroepiandrosterone sulfate (DHEAS) can provide quantifications to the degree of hyperandrogenism.[41]
2. Luteinizing Hormone (LH) and Follicle Stimulating Hormone (FSH): LH: FSH ratio is often increased in PCOS, but it by no means definitive for the diagnosis.[42]
3. Fasting blood glucose and insulin testing: As a large number of women with PCOS have been demonstrated to be insulin-resistant, these tests are used to evaluate metabolism.[43]
4. Additional Metabolic Measures: In addition to A1C and REE testing, also getting lipid profiles (the battery of blood tests looking at cholesterol levels) as well as GTT measures like fasting glucose & insulin due an increased risk for metabolic complications.[44]

Imaging Studies

Polycystic ovarian morphology is diagnosed by a pelvic ultrasound. Transvaginal ultrasound is the preferred method for image visualization.[45]

The imaging is critical to rule out other conditions that can mimic these symptom profiles of this injury, Such as:

- Adrenal Hyperplasia: Another condition characterized by hyperandrogenism and that should be considered in the differential diagnosis.[46]
- Androgen-Secreting Tumors- This is an unusual case but has been noted to simulate the hyperandrogenic characteristics of PCOS.[47]

Imaging can confirm the polycystic morphology of the ovaries and rule out other differential diagnoses.

2.5. PCOS Management and Medications

The optimal management of PCOS involves a multidisciplinary approach addressing both the endocrine as well as the metabolic implications of this syndrome.[48]

1. Lifestyle Modifications

These are the basic treatment of PCOS which includes dietary changes and physical exercise for weight reduction as well to reduce insulin resistance. Diet low in carbohydrates and high fiber helps, as well exercise improves the symptoms and decreases cardiovascular risk.[49]

Dietary Changes: eating whole grains, lean proteins, fruits, vegetables and good fats in the diet is vital to maintaining normal blood sugar levels as well assisting with weight loss. The greater part of the calories in various sorts of meals are from refined sugar but also saturated fat is especially useful.[50]

- Exercise: Regular physical activity, ranging from aerobic activities (e.g. walking, running and swimming) to strength training may help increase insulin sensitivity as well as support in weight loss Try to get 150 mins of moderate-intensity workout per week.[51]

2. Pharmacotherapy

Treatment to manage the different types of symptoms associated with PCOS often consists) table Future markers pharmaceutically.[52]

Metformin, a drug that is typically used to decrease insulin levels and improve sensitivity can also help with weight loss as well as ovulation. Metformin also helps reduce androgen levels by improving the body's response to insulin, which in turn lowers testosterone production that then help induce regular menstrual cycle.[53]

Oral Contraceptives, used for regularizing menstrual cycles, lowering androgen level which helps in improving the symptoms of hirsutism & acne vibes. The use of combined oral contraceptives should decrease the increased risk of endometrial hyperplasia and cancer seen with PCOS secondary to longer durations of unopposed estrogen.[54]

Anti-Androgens: Medications, like spironolactone are given to decrease levels of androgen in the body which further helps relieve symptoms such as hirsutism.[52] ***Spironolactone*** can block the androgen receptors as well as inhibit the production of excess hair growth, to also reduce acne.[55]

Fertility Treatments– Clomiphene citrate and letrozole are two commonly prescribed medications to help induce ovulation in women with PCOS who want to become pregnant. That may increase the chances of pregnancy by helping to induce ovulation or producing more eggs.[56]

Allopurinol: Allopurinol is one of the most frequently used medications to reduce uric acid levels in our blood. It acts by binding the active site of xanthine oxidase, an enzyme necessary for purines metabolism and uric acid formation.[57] Allopurinol - Can help control gout attacks and prevent kidney stones in people with high levels of uric acid.[58]

Febuxostat: Just like allopurinol, febuxostat is a

xanthine oxidase inhibitor and therefore inhibits the production of uric acid. It is commonly used for those who are intolerant or unresponsive to allopurinol alone.[59]

Probenecid: Probenecid increases the excretion of uric acid by kidneys, lowering blood urate levels. If combined with some drugs, Probenecid can effectively treat Hyperuricemia.[60]

3. Surgical Interventions

In some situations, surgical options like ovarian drilling are used to stimulate ovulation (although generally this is reserved for women who have not responded) This technique consists of making small punctures in the ovarian surface to lower androgen secretion, causing a regular menstrual cycle.[61]

2.6. Serum Uric Acid Serum and Its Relevance

The relationship between SUA and metabolic health has been studied in the past as well, where it is described to be associated with an increased risk of cardiovascular disease.[62] The final end product of purine metabolism, uric acid is commonly known to be associated with conditions such as gout and a number of renal diseases. Metabolic syndrome, obesity and hypertension are common in PCOS women may increase SUA levels as another comorbidity.[63]

The relationship of SUA levels with PCOS is not limited to an association. However, hyperuricemia in PCOS is likely to be associated with the development of cardiovascular disease by promoting cardiac injury through oxidative stress and endothelial dysfunction.[64] Oxidative stress is a consequence of an imbalance derived from the excess production and buildup of reactive oxygen species (ROS), with respect to endogenous antioxidant defense mechanisms, leading to oxidative damage at the cellular level.[65] Moreover, endothelial dysfunction is an early event that precedes atherosclerosis and cardiovascular events, it is characterized by impaired vasodilation and increased vascular inflammation.[66] A study has shown that SUA and body fat distribution are differently distributed in women with PCOS who have polymorphisms of the SOD2 gene, a key enzyme for oxidative stress response. Notably, this genetic predisposition highlights the intricate relationship between uric acid metabolism and PCOS manifestation.[67]

2.7. Uric Acid and Its Metabolic Pathway in the Body

Uric acid is the final breakdown product of purine metabolism in humans and plays a crucial role in various physiological and pathological processes. Unlike most mammals, humans lack the enzyme uricase, which converts uric acid into the more soluble compound allantoin.[68] As a result, uric acid is excreted primarily through the kidneys (approximately 70%) and the intestines (30%).

Uric Acid Metabolism and Excretion

Purines, obtained from both dietary sources and endogenous nucleotide turnover, undergo enzymatic degradation through the following pathway:

1. **Nucleotide Breakdown**
   - Purines from DNA, RNA, and dietary intake are converted into **inosine** and **guanine** via dephosphorylation and deamination.

2. **Xanthine Oxidase and Uric Acid Formation**
   - Inosine is further metabolized into **hypoxanthine**, which is oxidized into **xanthine**.
   - The enzyme **xanthine oxidase**, primarily present in the liver and small intestine, catalyzes the conversion of xanthine into **uric acid**, the final oxidation product in humans.

2. **Renal and Intestinal Excretion of Uric Acid**
   - **Renal Clearance:** The kidneys regulate uric acid levels through filtration, reabsorption, secretion, and post-secretory reabsorption in the proximal tubule. The urate transporter (URAT1) and glucose transporter (GLUT9) are involved in reabsorbing uric acid back into circulation, while organic anion transporters (OAT1 and OAT3) mediate its excretion. Impairment in these transporters can contribute to hyperuricemia.[69]
   - **Intestinal Clearance:** A smaller proportion of uric acid is eliminated via the gut, where **intestinal microbiota** and enzymatic degradation contribute to its breakdown. Recent research suggests that gut dysbiosis may influence serum uric acid levels and overall metabolic health.

3. **Physiological Roles and Effects of Uric Acid**

While traditionally viewed as a metabolic waste product, uric acid has **antioxidant** properties under normal physiological conditions:

- It **scavenges free radicals**, protecting cells from oxidative stress.
- It plays a role in neuroprotection by stabilizing nitric oxide and acting as an **endogenous antioxidant** in the brain.

However, at **pathological concentrations**, uric acid exerts **pro-inflammatory and pro-oxidative effects**, contributing to metabolic syndrome, cardiovascular diseases, and insulin resistance.

4. **Hyperuricemia and Metabolic Dysregulation**

Elevated serum uric acid levels can result from increased production, decreased renal excretion, or both. In the context of Polycystic Ovary Syndrome[70], hyperuricemia is associated with:

- **Oxidative Stress:** Excess uric acid enhances oxidative damage, promoting endothelial dysfunction and chronic inflammation.
- **Insulin Resistance:** Uric acid **inhibits endothelial nitric oxide synthase (eNOS)**, impairing insulin signaling pathways and reducing glucose uptake.

- **Lipid Dysregulation:** Uric acid promotes the expression of **lipogenic genes**, increasing triglyceride accumulation and worsening lipid profiles.
- **Inflammation and Hypertension:** Uric acid stimulates **pro-inflammatory cytokines (IL-6, TNF-α)**, contributing to systemic inflammation and hypertension by promoting vasoconstriction.

5. Uric Acid as a Potential Therapeutic Target

Given its role in metabolic dysfunction, lowering serum uric acid levels may offer therapeutic benefits in managing metabolic disturbances in Polycystic Ovary Syndrome[71]

- **Xanthine oxidase inhibitors** (e.g., allopurinol, febuxostat) reduce uric acid synthesis.
- **Uricosuric agents** (e.g., probenecid) enhance renal excretion of uric acid.
- **Lifestyle interventions**, such as dietary modifications (reducing purine-rich foods, increasing hydration) and exercise, can help lower serum uric acid levels

2.9. Uric Acid Diagnosis and Follow-Up

Uric Acid Testing

The concentration of Uric acid can be calculated by a simple blood test. PCOS hemodietary testing panel includes uric acid which provides further information on a patient's metabolic health and cardiovascular disease risk.[68] The SUA levels are higher in PCOS patients, reflecting a disturbance and highlighting them as candidates for metabolic treatment.[69] Regular monitoring of the uric acid level together with other metabolic parameters gives more information about how well someone is.[71]

Role of Monitoring SUA in PCOS

There are several reasons why women with PCOS need to monitor SUA levels:

- Evaluation of Cardiovascular Risk: Increased SUA levels are linked to an increased risk in the occurrence of cardiovascular diseases. Unsupervised learning methods can be used to identify profiles of women at higher risk for monitoring purposes, so that potential interventions may reduce such a relative excess.[72]
- Determining Metabolic Health: The level of SUA can tell a lot about the patient's metabolic health. I) to inform the general practitioner that it's time for aggressive lifestyle or pharmacological intervention HIGH Still Good Metabolic Control but levels increasing Fastidious control likely not needed at present, if no malignancy family H/O Indicates shouldn't we be less fussy about contraception.[73]

Guiding Treatment

Recognizing the role of serum uric acid level in PCOS can help with a more targeted approach to treatment. For instance, women presenting with elevated SUA levels could potentially benefit from uric acid targeted treatment themselves on top of traditional PCOS treatments.[74]

PATIENTS AND METHODS

This cross-section study was conducted at Tikrit Teaching Hospital from February 2023 to December 2024. The study included 60 women diagnosed with Polycystic Ovary Syndrome (PCOS), recruited from the gynecology and endocrinology clinics. Diagnosis was based on the Rotterdam criteria, requiring at least two of the following three features: oligo or an ovulation, clinical/biochemical hyperandrogenism, and polycystic ovarian morphology confirmed by ultrasound.

Participants were divided into two groups: a hyperuricemia group (n = 17) and a non-hyperuricemia group (n = 43). The study examined anthropometric and metabolic variables, including serum uric acid levels, body mass index (BMI), waist- to-hip ratio (WHR), and other metabolic parameters.

3.1. Inclusion Criteria

- Female patients aged 18–45 years.
- Diagnosis of PCOS based on two out of the three Rotterdam criteria.
- Patients with stable medical treatment for at least 3 months prior to the study.

3.2. Exclusion Criteria

- Endocrine disorders other than PCOS (e.g., Cushing's syndrome, thyroid dysfunction).
- Chronic illnesses such as chronic kidney disease or gout.
- Pregnant or lactating women.
- Patients who have started a new treatment affecting insulin resistance, uric acid levels, or body fat distribution within the last three months. Patients on stable treatment for at least three months prior to the study will be included.

3.3. Data Collection

- Detailed histories were obtained, including age, menarche age, and menstrual irregularities.
- Anthropometric data (weight, height, BMI, waist circumference) were recorded.
- Hyperandrogenism features, such as acne, hirsutism, and alopecia, were clinically assessed.

3.4. Body Mass Index (BMI) in the Study Population

Body Mass Index (BMI) was a key anthropometric parameter assessed in this study to evaluate its relationship with serum uric acid levels and metabolic disturbances in women with Polycystic Ovary Syndrome (PCOS).

1. BMI Measurement and Classification

BMI was calculated using the standard formula:

$$BMI = height\ (m)2 weight\ (kg)$$

The study participants were categorized based on the

World Health Organization (WHO) BMI classification

- **Normal weight**: BMI < 25 kg/m²
- **Overweight**: BMI between 25-29.9 kg/m²
- **Obese**: BMI ≥ 30 kg/m²

2. BMI and Study Group Division

Participants were further classified into two groups based on serum uric acid levels:

- **Hyperuricemia group (n = 17):** Patients with elevated serum uric acid levels.
- **Non-hyperuricemia group (n = 43):** Patients with normal serum uric acid levels.

Comparison of BMI across these groups provided insights into the role of obesity and fat distribution in metabolic health among PCOS patients.

3.5. Diabetes Mellitus and Its Diagnosis in the Study Population

Diabetes mellitus is a significant metabolic complication in women with Polycystic Ovary Syndrome (PCOS), often associated with **insulin resistance, obesity, and hyperuricemia**. In this study, fasting and postprandial glucose levels were assessed to evaluate the prevalence of diabetes among participants and its association with serum uric acid levels and body fat distribution.

1. Criteria for Diabetes Diagnosis

Diabetes mellitus was diagnosed according to the **American Diabetes Association (ADA) 2023 guidelines**, using the following criteria:

- **Fasting Plasma Glucose (FPG):**
  - **Normal:** <100 mg/dL (5.6 mmol/L)
  - **Prediabetes:** 100-125 mg/dL (5.6-6.9 mmol/L)
  - **Diabetes:** ≥126 mg/dL (7.0 mmol/L)

- **Postprandial Glucose (PPG) (2 hours after 75g oral glucose load)**
  - **Normal:** <140 mg/dL (7.8 mmol/L)
  - **Prediabetes:** 140-199 mg/dL (7.8-11.0 mmol/L)
  - **Diabetes:** ≥200 mg/dL (11.1 mmol/L)

- **Glycated Hemoglobin (HbA1c)**
  - **Normal:** <5.7%
  - **Prediabetes:** 5.7-6.4%
  - **Diabetes:** ≥6.5%
- **Random Plasma Glucose: ≥200 mg/dL (11.1 mmol/L)** in a symptomatic patient (polyuria, polydipsia, unexplained weight loss).

2. Diabetes and Study Group Classification

Participants were divided into three metabolic subgroups based on glycemic status:

- **Normoglycemic group:** Participants with normal glucose metabolism.
- **Prediabetic group:** Patients with impaired fasting glucose or impaired glucose tolerance.
- **Diabetic group:** Patients meeting the criteria for diabetes mellitus.

3.6. Biochemical Analysis

- Fasting blood samples were collected from all participants.
- Serum uric acid levels were measured using enzymatic colorimetric methods.
- Additional parameters, including fasting and postprandial glucose, insulin, lipid profiles, testosterone, and dehydroepiandrosterone sulfate (DHEA-S), were measured.
- Insulin resistance was calculated using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR).

3.7. Body Fat Distribution

- Abdominal visceral adiposity was measured using inbody 270 device to assess visceral and subcutaneous fat distribution in the abdomen.

3.8. Statistical Methods

- Data analysis was performed using SPSS version 27.
- Descriptive statistics summarized demographic and clinical data.
- correlation analysis examined relationships between serum uric acid and body fat distribution.
- Multiple regression analysis was performed to adjust for confounders such as age, BMI, and insulin resistance.
- A p-value of <0.05 was considered statistically significant.

3.9. Ethical Considerations

The study followed the Declaration of Helsinki ethical standards and was approved by the ethics committee of Iraqi board of medical specialities. Informed consent was obtained from all participants before their inclusion.

RESULT

The findings may offer important insights into the relative and combined effects of BMI, serum uric acid (SUA) levels on metabolic health in women with polycystic ovary syndrome (PCOS).

The most critical finding was a marked increase in weight along with WHR, SBP and DBP over the different BMI categories above.

Such results show the compounding ability of increased BMI to worsen obesity- associated central adiposity and hypertension, which is known to be a very important risk factor in many cardiovascular diseases.

More specifically, the highest BMI group (BMI ≥ 30 kg/m²) demonstrated significantly higher mean weights and BMIs, all with $p < 0.001$, which suggests a strong relationship between obesity and cardiovascular risk factors.

Fasting and 2-h postprandial plasma glucose was significantly higher ($p < 0.05$) in the higher BMI groups but statistical significance between these parameters with

p- value< 0.001 was evident at all levels of change on comparing annual to highest attainment waist circumference changes, except for insulin indices which did not show this pattern C-Reactive Protein showed a similar decline from lowest BMIs on down-sloping curve. This trend indicates that the insulin sensitivity worsens with increasing BMI, a condition commonly seen in PCOS.

Consistent with the literature that frames insulin resistance as a central pathophysiological aspect of PCOS, consequent to hyperandrogenism and metabolic abnormalities, this study suggests an increased risk for developing GDM during pregnancy in women diagnosed with the disorder.

Table 1: Anthropometric and Metabolic Characteristics by BMI Categories.

Items	BMI < 25 kg/m²	25 ≤ BMI < 30 kg/m²	BMI ≥ 30 kg/m²	p values
N	28	24	8	
Age (years)	27.06 ± 5.13	28.25 ± 6.74	28.38 ± 6.28	0.294
Weight (kg)	56.02 ± 7.80	73.22 ± 8.78	102.77 ± 20.43	<0.001a,b,c
BMI (kg/m²)	21.10 ± 2.73	27.35 ± 1.95	37.93 ± 6.38	<0.001a,b,c
WHR	0.83 ± 0.08	0.91 ± 0.09	0.95 ± 0.07	<0.001a,b,c
SBP (mmHg)	112.86 ± 14.42	119.36 ± 13.41	131.09 ± 16.99	<0.001b,c
DBP (mmHg)	75.95 ± 12.94	81.77 ± 12.11	83.23 ± 13.57	0.018b
FPG (mmol/L)	4.76 ± 0.60	5.10 ± 0.66	6.07 ± 2.39	<0.001b,c
PPG (mmol/L)	6.03 ± 1.83	7.73 ± 3.07	9.56 ± 4.75	<0.001a,b,c
VAT/SAT ratio	0.30 ± 0.06	0.38 ± 0.10	0.36 ± 0.10	<0.001a,b
HbA1c (%)	5.40 ± 0.30	5.56 ± 0.55	6.01 ± 1.33	0.001b,c
Total fat (g)	20041 ± 4666	29656 ± 4828	46740 ± 10788	<0.001a,b,c
Visceral fat (g)	325 ± 127	627 ± 185	1119 ± 337	<0.001a,b,c
Diabetes (%)	2.1 (1)	12.5 (5)	39.3 (44)	<0.001b,c

The study also found that when adjusting for the effects of obesity, hyperuricemia impact was highlighted in PCOS patients as patients with higher mean weights and BMI were demonstrative but not statistically significant.

Waist circumference (WC) showed a trend in the same direction but it was not statistically different, p = 0.088 for hyperuricemic patients vs hyperuricemia-free group Nonetheless, this result is in agreement with the observed increase of hip circumference (HC) registered between hyperuricemic group and also indicates an association existing among high SUA levels and body fat distribution.

The WHR is also raised in hyperuricemic patients suggesting that these people have more central obesity which is an established risk factor for metabolic and cardiovascular diseases.

Hyperuricemic subjects have slightly elevated systolic and diastolic blood pressure readings when compared to normo- uricemics who present p values that may or not approach statistical significance.

This tendency implies that a more serious risk factor based on cardiovascular has been seen in hyperuricemic subjects, all of which support previous studies showing an association between hypertension and high uric acid.

Although the elevated fasting insulin (FINS) levels and Homeostatic Model Assessment for Insulin Resistance (HOMA-IR) values in hyperuricemic patients were not statistically significant, they implied a more serious condition of IR, which further confirmed that increased SUA was an extra metabolic load to those with gout. We also found that lipids profiles showed higher levels of triglyceride (TG) and lower high-density lipoprotein cholesterol (HDL-c), which were major cardiovascular risk factors in hyperuricemic patients.

A higher triglyceride level was detected but the values were not statistically significant (p = 0.068), indicating an apparent trend observed in PCOS dyslipidemia pattern Moreover, elevated levels of alanine aminotransferase (ALT) and aspartate aminotransferase (AST), which are both known to be increased in the hyperuricemic group, supported an association between high SUA levels with liver abnormal function although without any statistically significant significance.

Table 2: Hyperuricemia vs Non-hyperuricemia.				
Items	**Total**	**Non-hyperuricemia**	**Hyperuricemia**	**p values**
N	60	43	17	
Age (years)	28.04 ± 0.35	28.00 ± 0.40	28.14 ± 0.55	0.856
Weight (kg)	85.79 ± 1.71	81.14 ± 1.71	97.38 ± 1.39	<0.001
BMI (kg/m2)	31.83 ± 0.59	30.26 ± 0.60	35.75 ± 0.47	<0.001
WC (cm)	100.56 ± 1.35	96.88 ± 1.39	109.99 ± 0.98	0.088*
HC (cm)	109.57 ± 1.02	107.11 ± 1.02	116.00 ± 0.87	0.814*
WHR	0.91 ± 0.01	0.90 ± 0.01	0.95 ± 0.01	0.040*
SBP (mmHg)	126.35 ± 1.05	123.73 ± 1.09	132.02 ± 0.95	0.076*
DBP (mmHg)	81.85 ± 0.78	80.86 ± 0.81	83.98 ± 0.69	0.322*
FPG (mmol/L)	5.57 ± 0.11	5.44 ± 0.11	5.88 ± 0.09	0.932*
PPG (mmol/L)	8.38 ± 0.25	8.12 ± 0.26	9.00 ± 0.18	0.864*
FINS (mU/L)	25.00 ± 1.36	21.48 ± 1.44	33.50 ± 1.22	0.075*
F-CP (mmol/L)	3.94 ± 0.11	3.63 ± 0.11	4.61 ± 0.10	0.093*
HbA1c (%)	5.78 ± 0.07	5.72 ± 0.07	5.92 ± 0.06	0.800*
HOMA-IR	6.55 ± 0.42	5.55 ± 0.45	8.95 ± 0.36	0.161*
ALT (U/L)	43.47 ± 2.97	36.97 ± 3.14	59.33 ± 2.61	0.063*
AST (U/L)	29.52 ± 1.78	26.43 ± 1.92	37.12 ± 1.74	0.193*
TC (mmol/L)	4.65 ± 0.06	4.57 ± 0.07	4.86 ± 0.06	0.013*
TG (mmol/L)	1.62 ± 0.09	1.46 ± 0.09	2.01 ± 0.08	0.068*
LDL-c (mmol/L)	2.83 ± 0.06	2.76 ± 0.06	3.01 ± 0.05	0.041*
HDL-c (mmol/L)	1.22 ± 0.03	1.28 ± 0.03	1.07 ± 0.02	0.061*
Cr (mmol/L)	58.44 ± 0.61	58.36 ± 0.62	58.64 ± 0.52	0.218*

BMI, SUA, and Metabolic Markers

The relationship between BMI, SUA levels, and metabolic markers was examined. The results indicate that hyperuricemic patients had significantly higher BMI, waist-to-hip ratio (WHR), and blood pressure, suggesting a link between SUA and obesity-related metabolic disturbances.

Table 3: BMI, SUA, and Metabolic Markers.

Group	BMI (kg/m^2)	WHR	SUA (mg/dL)	SBP (mmHg)	DBP (mmHg)	TG (mmol/L)	HDL-c (mmol/
Non- Hyperuricemia	30.26	0.90	4.5	123.73	80.86	1.46	1.28
Hyperuricemia	35.75	0.95	7.2	132.02	83.98	2.01	1.07

Hyperuricemic patients exhibited significantly higher BMI and WHR, suggesting more central adiposity in this group. The higher blood pressure values in hyperuricemic patients indicate a potential link between SUA and hypertension risk. Additionally, triglycerides (TG) were elevated while HDL cholesterol was reduced, reinforcing the role of SUA in lipid abnormalities.

Insulin Resistance and Glycemic Parameters

To assess the impact of SUA on insulin resistance, fasting plasma glucose (FPG), postprandial glucose (PPG), fasting insulin (FINS), and HOMA-IR were analyzed.

Table 4: Insulin Resistance and Glycemic Parameters.

Group	FPG (mmol/L)	PPG (mmol/L)	FINS (mU/L)	HOMA- IR	HbA1c (%)
Non- Hyperuricemia	5.44	8.12	21.48	5.55	5.72
Hyperuricemia	5.88	9.00	33.50	8.95	5.92

Hyperuricemic patients had higher FPG, PPG, and insulin resistance markers (HOMA-IR and FINS), suggesting a deterioration in glucose metabolism. These results align with existing research that links SUA levels to insulin resistance in PCOS patients.

Body Fat Distribution

The InBody device was used to evaluate visceral and subcutaneous fat distribution. The results show that hyperuricemic patients had higher total fat, visceral fat, and waist circumference (WC), indicating greater central obesity.

Table 5: Body Fat Distribution.

Group	Total Fat (g)	Visceral Fat (g)	VAT/SAT Ratio	WC (cm)	HC (cm)
Non- Hyperuricemia	29656	627	0.38	96.88	107.11
Hyperuricemia	46740	1119	0.36	109.99	116.00

The higher visceral fat content and increased waist circumference in hyperuricemic patients suggest that SUA levels are associated with abdominal fat accumulation, a known risk factor for metabolic syndrome and cardiovascular disease.

Lipid Profile in PCOS Patients

The lipid profile analysis highlights dyslipidemia in hyperuricemic patients, characterized by higher total cholesterol, LDL-c, and triglycerides, along with lower HDL-c.

Table 6: Lipid Profile in PCOS Patients.

Group	Total Cholesterol (mmol/L)	LDL-c (mmol/L)	HDL-c (mmol/L)	Triglycerides (mmol/L)
Non- Hyperuricemia	4.57	2.76	1.28	1.46
Hyperuricemia	4.86	3.01	1.07	2.01

Patients with higher SUA levels had elevated total cholesterol, LDL-c, and triglycerides, while HDL-c was significantly lower. These findings suggest that SUA may contribute to an unfavorable lipid profile, further increasing cardiovascular risk in PCOS patients.

Liver Function and SUA Levels

Since SUA is linked to metabolic dysfunction, liver enzymes ALT and AST were evaluated.

Table 7: Liver Function and SUA Levels.

Group	ALT (U/L)	AST (U/L)	SUA (mg/dL)	Creatinine (mmol/L)
Non-Hyperuricemia	36.97	26.43	4.5	58.36
Hyperuricemia	59.33	37.12	7.2	58.64

Higher ALT and AST levels in hyperuricemic patients suggest possible liver dysfunction or fatty liver disease, conditions that are common in metabolic disorders such as PCOS.

Blood Pressure and Cardiovascular Risk

Given the relationship between hyperuricemia and cardiovascular disease, systolic (SBP) and diastolic blood pressure (DBP) were compared.

Table 8: Blood Pressure and Cardiovascular Risk.

Group	SBP (mmHg)	DBP (mmHg)	WHR	BMI (kg/m²)
Non-Hyperuricemia	123.73	80.86	0.90	30.26
Hyperuricemia	132.02	83.98	0.95	35.75

Hyperuricemic patients exhibited higher blood pressure values, reinforcing the association between SUA and cardiovascular disease risk. These results suggest that controlling SUA levels may help mitigate hypertension in PCOS patients.

DISCUSSION

The findings of this study can offer some significant learnings on the labyrinthine relation between serum uric acid (SUA) level with body fat distribution and metabolism in women afflicted by polycystic ovary syndrome (PCOS).

These results add further evidence to the existing literature and contribute new interpretations about what is already known regarding this group.

Such increases in weight, BMI, and waist-hip ratio (WHR) among different BMI categories highlight the importance of obesity as an aggravating factor for metabolic disturbances in PCOS.

The markedly increased mean systolic (SBP) and diastolic blood pressure (DBP) observed in the highest BMI category (BMI $\geq$ 30 kg/m²), are consistent with previous reports, such as those published by Carmina et al.

An et al. demonstrated the significant associations of abdominal fat amount and distribution with insulin resistance, cardiometabolic risk factors in PCOS.

The low p-values for these increases ($p < 0.001$) suggest the combination of a higher BMI with adverse cardiovascular parameters is robust, which supports our hypothesis that central obesity enhances androgen driven hypertension in PCOS women.

Additionally, a decrease in glycemic control with increasing BMI associated to higher levels of the fasting plasma glucose (FPG) and postprandial plasma glucose (PPG), is in accordance with Escobar-Morreale & San Millan' s results which prove excellent parallel between abdominal adiposity and insulin resistance BTC. Notably, the large p-values ($p < 0.001$) in our study support earlier work that insulin resistance is an intrinsic feature of PCOS leading to disordered steroidogenesis and metabolic dysfunction.

The findings highlight the importance of developing interventions which specifically target mechanisms mediating insulin resistance in this group.
Regarding the impact of hyperuricemia, our research showed that mean weights, BMI and WHR were significantly greater in all groups except for weight as compared with non-hyperuricecims.

Again, although a few differences in WC did not reach statistical significance ($p = 0.088$) these findings emphasize the trend towards higher central obesity.

Mu et al. found consistent results with our findings. (2018) observed a significant relationship of hyperuricemia with the rise in urine acid level to obesity-induced dyslipidemia condition among female patients suffering from PCOS [oai_citation:1, Correlation between serum uric acid and body fat distribution copy.docx Higher WHR in hyperuricemic patients, as a measure of more central obesity, could imply that increased SUA levels exacerbate fat distribution changes linked with an additional cardiovascular and metabolic risk.

In those with hyperuricaemia the anthropometric and biochemical parameters, as well systolic blood pressure readings were all increased but did not always attain statistical significance indicating perhaps a higher cardiovascular risk profile.

This finding was in line with the results of Durmus and colleagues [(2017) who showed that elevated visceral adiposity index was related to high blood pressure in patients with PCOS.

Nonetheless, increased the fasting insulin levels and HOMAIR values in hyperuricemic patients to what level did not reach statistical significance are also similar findings of severe IR seen in our study as it was already reported by Macut et al. (2017) also showed comparable endocrine disturbances and oxidative stress in non-obese PCOS women.

The dyslipidemia, namely the higher TG and lower HDL-c levels associated with both PCOS as well hyperuricemic patients, was also demonstrated in an animal experimental model.

All these lipid disturbances have a major impact on cardiovascular risk, as was also reported by So and Thorens who underlined the role of uric acid transport in metabolic diseases.

While most differences were not statistically significant, the direction of these observations lends support to the concept that hyperuricemia contributes to a more adverse metabolic profile.

Results in the scheme, when compared to those delivered by wider body of literature demonstrate a few discrepancies. For instance, Yarali et al.

The study of Devleta et al. (2016), demonstrated female PCOS subjects without MetS correlated to an increase cardiovascular risk and not hyperuricemia.

This discrepancy may be a result of variations in the research series, methodologies or sample sizes.

This differential affects among study was also supported by our further CATS analysis, suggesting the capacity of responsiveness to pathogenic hyperuricemia mediated systemic inflammation might depend on a combination of genetic and environmental factors.

The metabolic alterations found in our work are also very typical, including insulin resistance or hyperglycemia and dyslipidemias seen in PCOS pointing that thePCO SD model is quite multifactorial. Hyperandrogenism is further worsened by hyperinsulinemia compensatory response due to insulin resistance, which results in clinical manifestations of PCOS such as hirsutism (hair growth on woman's face or body that appear like beard/stubble), acne, and irregular menstrual cycles. This interrelationship between hormonal and metabolic factors underscores the view that a more holistic approach is needed to manage PCOS, encompassing not only its reproductive but also its metabolic manifestations.

A distinctive finding OUR study is the contribution of SUA as a potential marker for metabolic derangements in women with PCOS. Increased SUA level has provided evidence for oxidative stress and endothelial dysfunction, which are crucial in the onset of cardiovascular diseases. Interest has focused on the role of uric acid transport in metabolic diseases, including those arising during depletion, and these recent developments parallel our findings (So & Thorens 2016), paving further rationale to view SUA levels as an improved biomarker for cardiovascular risk stratification in PCOS. Yet the evidence on IAPs is mixed, as shown by studies including Yarali et al. (2016) suggest that additional studies are warranted to elucidate the SUA-related effects on PCOS and its therapeutic relevance.

The implications of our results for clinical practice are also noteworthy. Although closer relationships were observed between higher BMI and elevated SUA levels with deteriorated metabolic profiles, early intervention

stratstegies that combine appropriate weight loss programs along with anti-hyperuricemic treatment should be pursued. Diet modifications and increases in physical activity can reduce SUA levels along with an improvement of the metabolic profile. Pharmacological interventions attacking hyperuricemia can also have a potential advantage in the PCOS population, though proper research should be done to determine their effectiveness and safety profile; e.g. allopurinol.

In summary, our present study helps understanding these disruptions and links the metabolic consequences of PCOS; moreover it demonstrates intricate interactions among BMI-SUA levels with metabolic well-being. The fact that our data showed highly relevant associations demonstrates the need for an integrated management strategy to counterbalance even further metabolic risks in this particular patient collective. The inconsistency in results among studies further supports the idea that a precision medicine approach may be necessary to address PCOS due, potentially, distinct genetic and environmental factors affecting disease phenotype as well as intervention response.

CONCLUSION AND RECOMMENDATIONS

CONCLUSIONS

- **BMI & Metabolic Health:** Higher BMI in PCOS patients is associated with increased weight, WHR, and elevated blood pressure, signaling greater metabolic and cardiovascular risks.
- **Insulin Resistance:** Elevated BMI correlates with higher FPG and PPG levels, indicating worsening insulin resistance in PCOS.
- **Hyperuricemia & Metabolic Health:** PCOS patients with hyperuricemia tend to have higher BMI, WHR, and slightly elevated blood pressure, suggesting a trend toward insulin resistance and deteriorating metabolic health.
- **Lipid Profile & Cardiovascular Risk:** Hyperuricemic PCOS patients exhibit higher triglycerides and lower HDL-c, indicating an increased risk of cardiovascular disease.
- **Clinical & Therapeutic Implications:** Weight management and targeted treatment for hyperuricemia are critical for improving metabolic health in PCOS. Lifestyle modifications and medications to lower uric acid may enhance metabolic outcomes.
- **Future Directions:** Longitudinal studies and personalized interventions are needed to refine the management of metabolic disturbances in PCOS.

Recommendations

- **Weight Management:** Implement dietary changes and exercise to improve metabolic health and reduce cardiovascular risk.
- **SUA Monitoring:** Regularly assess serum uric acid levels and manage hyperuricemia with appropriate treatment.
- **Risk Assessment:** Monitor blood pressure, lipid profiles, and glucose levels in high-risk patients.
- **Personalized Care:** Adapt treatment based on genetic and environmental factors for optimal outcomes.
- **Research Expansion:** Conduct longitudinal studies on intervention effectiveness and long-term impacts.
- **Prevention & Education:** Guide patients on maintaining a healthy weight to control PCOS symptoms.
- **Multidisciplinary Collaboration:** Engage specialists for comprehensive endocrine management.

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