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PREVALENCE OF URINARY TRACT INFECTIONS AMONG WOMEN WITH POLYCYSTIC OVARY SYNDROME AND ITS HORMONAL CORRELATES

Original Article

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a prevalent endocrine disorder affecting reproductive-aged women, often associated with hormonal and metabolic imbalances. While its reproductive and metabolic implications are well-documented, its potential link to urinary tract infections (UTIs) remains underexplored.

Objective: To determine the prevalence of UTIs among women with PCOS and investigate the hormonal and metabolic correlates contributing to increased susceptibility.

Methods: A cross-sectional study was conducted over four months in Sindh, involving 355 women diagnosed with PCOS based on Rotterdam criteria. Midstream urine samples were collected for culture-based confirmation of UTI. Serum levels of luteinizing hormone (LH), follicle-stimulating hormone (FSH), estradiol, testosterone, DHEA-S, sex hormone-binding globulin (SHBG), and insulin resistance (HOMA-IR) were measured. Statistical analyses included descriptive statistics, independent t-tests, Pearson correlation, and binary logistic regression.

Results: Of the 355 participants, 108 (30.4%) were UTI positive. Women with UTIs had significantly higher testosterone (68.7 ± 20.5 ng/dL), lower estradiol (45.1 ± 12.4 pg/mL), reduced SHBG (26.8 ± 9.5 nmol/L), and elevated HOMA-IR (3.4 ± 1.2), all with p-values < 0.05. Logistic regression revealed testosterone (OR=1.34), estradiol (OR=0.89), SHBG (OR=0.94), and HOMA-IR (OR=1.22) as independent predictors of UTI.

Conclusion: A substantial proportion of women with PCOS were found to have UTIs, strongly associated with androgen excess, estrogen deficiency, and insulin resistance. These findings emphasize the need for comprehensive clinical management in PCOS that includes attention to urinary health and infection risk.

Keywords: Androgens, Estradiol, Hormones, Insulin Resistance, Polycystic Ovary Syndrome, SHBG, Urinary Tract Infections.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a complex endocrine disorder that affects millions of women of reproductive age worldwide. Characterized by chronic anovulation, hyperandrogenism, and polycystic ovarian morphology, it presents with a spectrum of symptoms ranging from irregular menstrual cycles and infertility to metabolic disturbances such as insulin resistance and obesity (1). While the reproductive and metabolic consequences of PCOS have been widely studied, there is growing recognition of its potential association with other health complications that have traditionally received less attention. Among these, the susceptibility to urinary tract infections (UTIs) in women with PCOS is emerging as an area warranting closer examination. Urinary tract infections are among the most common bacterial infections affecting women, with nearly half experiencing at least one episode in their lifetime (2). The female anatomy, particularly a shorter urethra, makes women more prone to these infections, but risk is further influenced by hormonal, metabolic, and behavioral factors. In PCOS, hormonal dysregulation is central to the pathology. Elevated androgens, altered estrogen-progesterone balance, and insulin resistance not only contribute to the classical symptoms of the syndrome but may also exert effects on urogenital immunity, vaginal flora, and mucosal defense mechanisms (3). These hormonal shifts could plausibly create a biological environment more susceptible to uropathogen colonization and infection, yet this intersection remains insufficiently explored in clinical literature. Estrogen, in particular, plays a protective role in the urinary tract. It maintains the integrity of the uroepithelium, supports the proliferation of lactobacilli in the vaginal flora, and modulates local immune responses. Women with PCOS often experience lower-than-normal circulating estrogen levels during anovulatory cycles, potentially weakening this protective barrier. Moreover, hyperandrogenism—another hallmark of PCOS—may influence sebaceous gland activity and mucosal immunity, factors that may indirectly affect the urogenital environment. These hormonal patterns may disrupt the microbiological and immunological balance of the lower urinary tract, facilitating recurrent or persistent infections (4).

Insulin resistance, which affects a substantial proportion of women with PCOS, is another critical factor in the equation (5). Hyperinsulinemia may contribute to systemic inflammation, vascular changes, and compromised immunity—all of which may increase the risk and severity of infections. Furthermore, metabolic derangements can alter glucose concentrations in the urine, offering a more hospitable environment for bacterial growth (6). Combined with sedentary lifestyle patterns and increased body mass index often observed in PCOS populations, these factors create a multifactorial risk profile for UTI development that is distinct from the general female population (7). Despite the biological plausibility of a link between PCOS and increased UTI susceptibility, the empirical evidence remains limited. Existing studies addressing infections in women with PCOS have largely focused on reproductive tract infections or broader immunological markers, often overlooking the urological dimension. This gap leaves clinicians with insufficient guidance in recognizing and managing this potential complication in PCOS care. A better understanding of how hormonal imbalances characteristic of PCOS relate to urinary tract infections could offer meaningful insights for prevention, screening, and therapeutic strategies, especially in those patients presenting with recurrent symptoms (8).

Given this context, it becomes crucial to investigate whether the hormonal milieu typical of PCOS contributes to a higher prevalence of urinary tract infections in affected women (9). The possibility that chronic hormonal imbalances may predispose these individuals to recurrent UTIs introduces a novel dimension to PCOS management, highlighting the need for more comprehensive care strategies (10). Moreover, understanding the hormonal correlates of UTIs in PCOS could reveal specific targets for intervention, such as optimizing hormonal profiles or addressing metabolic risk factors more aggressively (11). This study seeks to examine the prevalence of urinary tract infections among women diagnosed with PCOS and to explore the hormonal correlates that may underlie increased susceptibility (12). By investigating this association through a cross-sectional lens, the research aims to illuminate potential mechanisms, identify at-risk subgroups, and contribute to a more integrative understanding of the systemic implications of PCOS. The objective is not only to quantify the burden of UTIs in this population but also to rationalize their occurrence within the context of hormonal and metabolic dysfunction—thus bridging a critical gap in current knowledge.

METHODS

This cross-sectional study was conducted over a period of four months in the province of Sindh with the objective of determining the prevalence of urinary tract infections among women diagnosed with polycystic ovary syndrome (PCOS) and exploring the hormonal correlates contributing to increased susceptibility. The study design was carefully structured to ensure both relevance to the research objective and methodological rigor, allowing the outcomes to offer meaningful insights into the intersection of hormonal dysregulation and infection risk in this population. The target population included women aged 18 to 40 years who had been clinically diagnosed with

PCOS based on the Rotterdam criteria, which require the presence of at least two of the following three features: oligo/anovulation, clinical or biochemical signs of hyperandrogenism, and polycystic ovarian morphology on ultrasound. Participants were recruited from outpatient gynecology and endocrinology clinics across various districts in Sindh through purposive sampling. To maintain consistency and reduce confounding variables, several inclusion and exclusion criteria were applied. Women with a confirmed diagnosis of PCOS and no history of current or recent antimicrobial therapy within the past four weeks were eligible. Those with anatomical abnormalities of the urinary tract, diagnosed immunodeficiency disorders, known diabetes mellitus, pregnancy, or recent urinary catheterization were excluded. These criteria ensured a homogenous cohort reflective of the typical PCOS population, without other major confounders that could independently influence UTI susceptibility.

To determine the appropriate sample size for the cross-sectional nature of the study, a prevalence estimate was required. Based on literature suggesting that the UTI prevalence in reproductive-aged women ranges from 10% to 20%, and hypothesizing an increased prevalence of approximately 30% in the PCOS population, the sample size was calculated using the single population proportion formula with a confidence level of 95% and a 5% margin of error. This yielded a required minimum sample size of 323 participants. Anticipating a 10% non-response or data incompleteness rate, the final target sample size was set at 355 women. After obtaining informed verbal consent, participants underwent structured interviews and clinical assessments. Data collection included demographic information, menstrual and gynecological history, and symptomatology suggestive of urinary tract infections, such as dysuria, frequency, urgency, suprapubic discomfort, and hematuria. Midstream clean-catch urine samples were collected for microbiological analysis. Each sample was subjected to routine urinalysis using dipstick testing and microscopic examination. For confirmation of UTI, a positive urine culture with bacterial colony counts exceeding 10^5 CFU/mL of a single pathogen was considered diagnostic.

Hormonal profiling was conducted through venous blood sampling during the early follicular phase (days 2 to 5 of the menstrual cycle, or randomly in amenorrheic participants). Serum levels of luteinizing hormone (LH), follicle-stimulating hormone (FSH), estradiol, total testosterone, dehydroepiandrosterone sulfate (DHEA-S), and sex hormone-binding globulin (SHBG) were measured using enzyme-linked immunosorbent assay (ELISA) techniques. Insulin resistance was estimated using the homeostatic model assessment for insulin resistance (HOMA-IR), calculated from fasting insulin and glucose levels. All assays were performed in a certified central laboratory to ensure consistency and reliability of the biochemical data. Statistical analysis was carried out using SPSS version 26. Descriptive statistics were used to summarize the data, with continuous variables expressed as mean ± standard deviation and categorical variables as frequencies and percentages. The normality of data distribution was assessed using the Kolmogorov–Smirnov test. Since the dataset followed a normal distribution, parametric tests were employed for inferential analysis. The prevalence of UTIs in the PCOS cohort was expressed as a proportion with a 95% confidence interval. Independent t-tests were used to compare mean hormonal levels between UTI-positive and UTI-negative groups. Pearson's correlation coefficient was calculated to assess the relationship between hormonal parameters and UTI occurrence. Additionally, binary logistic regression was used to identify independent hormonal predictors of UTI risk, adjusting for potential confounders such as age and body mass index (BMI). A p-value of <0.05 was considered statistically significant for all analyses. This methodological framework allowed for a comprehensive evaluation of the relationship between hormonal imbalances and UTI prevalence in PCOS, using validated outcome measures and robust statistical approaches. By systematically capturing both microbiological and endocrine profiles, the study aimed to contribute empirical evidence that could help inform both clinical screening strategies and targeted preventive interventions for women living with PCOS.

RESULTS

Out of a total of 355 women diagnosed with polycystic ovary syndrome, 108 participants (30.4%) tested positive for urinary tract infection based on culture-confirmed diagnosis, while the remaining 247 (69.6%) were classified as UTI-negative. The overall mean age of participants was 27.3 ± 4.8 years, and the average body mass index was 29.6 ± 5.1 kg/m². Among the cohort, 72.7% were married, 68.1% had at least secondary education, and 40.2% were employed.

Hormonal profiling revealed significant differences between the UTI-positive and UTI-negative groups. The mean serum testosterone level in the UTI-positive group was 68.7 ± 20.5 ng/dL, which was significantly higher than the 58.3 ± 18.9 ng/dL observed in UTI-negative participants ($p = 0.004$). Estradiol levels were markedly lower in those with UTIs, with a mean of 45.1 ± 12.4 pg/mL compared to 56.8 ± 13.7 pg/mL in the control group ($p < 0.001$). Likewise, SHBG levels were significantly reduced in UTI-positive individuals (26.8 ± 9.5 nmol/L vs. 31.4 ± 10.2 nmol/L; $p = 0.006$). DHEA-S levels were moderately elevated in UTI-positive women (230 ± 64

µg/dL) compared to their counterparts (195 ± 52 µg/dL; p = 0.011). However, FSH levels showed no significant difference between groups (p = 0.317).

Insulin resistance, assessed using HOMA-IR, was significantly greater in UTI-positive participants, with a mean value of 3.4 ± 1.2, compared to 2.8 ± 1.0 in the UTI-negative group (p = 0.019), suggesting a possible metabolic contribution to infection susceptibility.

Binary logistic regression analysis identified serum testosterone, estradiol, SHBG, and HOMA-IR as independent predictors of UTI occurrence. Elevated testosterone levels were associated with a 34% increased odds of UTI (Adjusted OR: 1.34; 95% CI: 1.12–1.59; p = 0.001). In contrast, higher estradiol levels demonstrated a protective association (Adjusted OR: 0.89; 95% CI: 0.82–0.96; p = 0.004). Similarly, higher SHBG levels were inversely associated with UTI prevalence (Adjusted OR: 0.94; 95% CI: 0.90–0.98; p = 0.012). Elevated HOMA-IR was also independently associated with increased UTI risk (Adjusted OR: 1.22; 95% CI: 1.05–1.41; p = 0.008).

These findings indicate a clear hormonal and metabolic profile that differentiates women with PCOS who are at increased risk for UTIs from those who are not. Differences in androgen excess, estrogen deficiency, reduced SHBG, and heightened insulin resistance were all more pronounced among UTI-positive individuals, underscoring potential endocrine drivers of susceptibility. The statistically significant variables offer avenues for risk stratification and further investigation.

Table 1: Demographic Characteristics of Participants

Variable	Mean ± SD / %
Age (years)	27.3 ± 4.8
BMI (kg/m²)	29.6 ± 5.1
Marital Status - Married (%)	72.7%
Education - Secondary or Above (%)	68.1%
Employment - Employed (%)	40.2%

Table 2: Prevalence of Urinary Tract Infections

Group	n (%)
Total Participants	355
UTI Positive	108
UTI Negative	247

Table 3: Comparison of Hormonal Parameters by UTI Status

Hormonal Marker	UTI Positive (Mean ± SD)	UTI Negative (Mean ± SD)	p-value
LH (IU/L)	9.2 ± 3.6	7.4 ± 2.8	0.002
FSH (IU/L)	5.4 ± 1.8	5.6 ± 1.9	0.317
Estradiol (pg/mL)	45.1 ± 12.4	56.8 ± 13.7	<0.001
Testosterone (ng/dL)	68.7 ± 20.5	58.3 ± 18.9	0.004
DHEA-S (µg/dL)	230 ± 64	195 ± 52	0.011
SHBG (nmol/L)	26.8 ± 9.5	31.4 ± 10.2	0.006
HOMA-IR	3.4 ± 1.2	2.8 ± 1.0	0.019

Table 4: Logistic Regression Analysis of Predictors of UTI

Predictor	Adjusted OR (95% CI)	p-value
Testosterone	1.34 (1.12–1.59)	0.001
Estradiol	0.89 (0.82–0.96)	0.004
SHBG	0.94 (0.90–0.98)	0.012
HOMA-IR	1.22 (1.05–1.41)	0.008

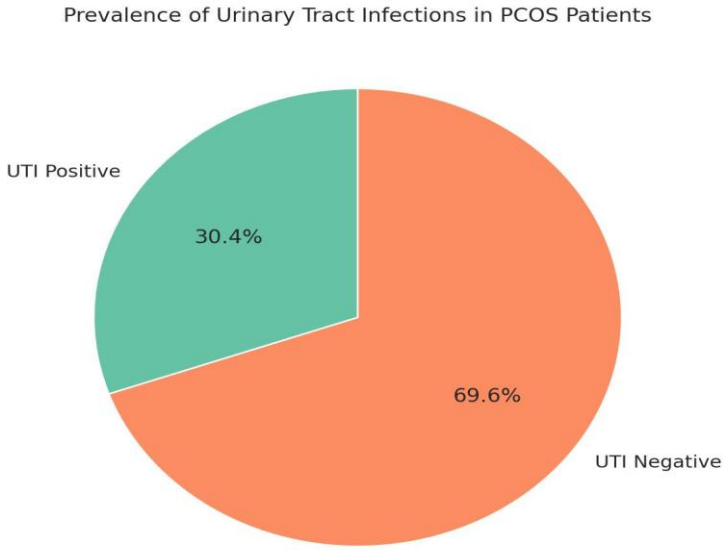

Figure 1 Prevalence of Urinary Tract Infection in PCOS Patients

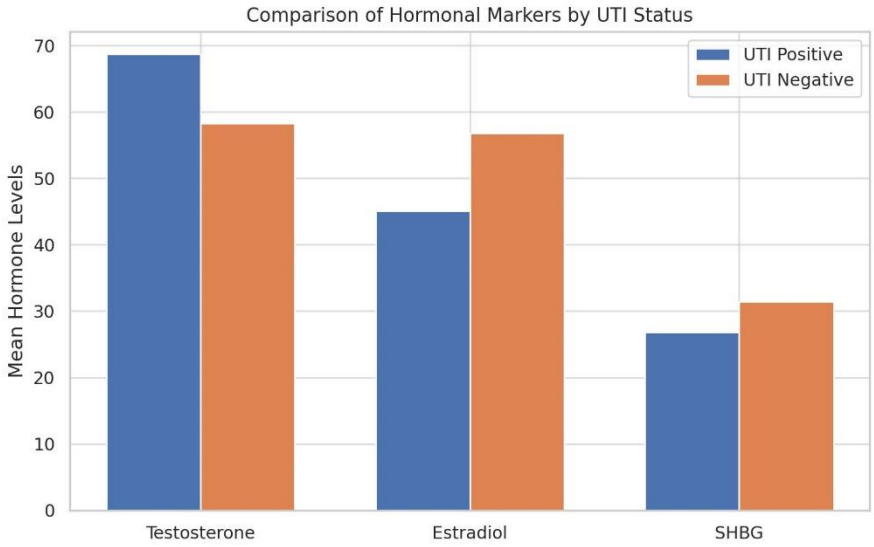

Figure 2 Comparison of Hormonal Markers by UTI Status

DISCUSSION

The findings of this cross-sectional study revealed a notable prevalence of urinary tract infections among women with polycystic ovary syndrome, with 30.4% of participants testing positive based on culture-confirmed diagnosis (13). This observed rate appears elevated when compared to the general female population, suggesting that PCOS may be a potential contributor to increased vulnerability to UTIs (14). The data also demonstrated distinct hormonal and metabolic profiles in UTI-positive individuals, particularly higher serum testosterone, lower estradiol and SHBG levels, and elevated insulin resistance (15). These associations underscore the multifaceted nature of PCOS and its influence beyond reproductive and metabolic domains. Hyperandrogenism, a defining characteristic of PCOS, emerged as a significant factor, with higher testosterone levels independently associated with increased odds of UTI occurrence. This supports the premise that excess androgens may compromise the urogenital mucosal defense or influence microbial colonization patterns in ways that facilitate infection. Lower estradiol levels among UTI-positive participants also highlighted the potential disruption of estrogen-mediated protective mechanisms within the urogenital tract, such as epithelial integrity and lactobacilli dominance. SHBG, which regulates free androgen availability, was found to be lower in the infected group, reinforcing the role of bioavailable androgens in mediating susceptibility (16).

Metabolic dysfunction, represented by elevated HOMA-IR values, further differentiated the UTI-positive subgroup (17). Insulin resistance may impair innate immune responses or alter urinary glucose excretion, thereby creating a favorable environment for uropathogenic bacteria. The interplay between hormonal imbalance and metabolic derangement reflects a synergistic contribution to UTI risk in women with PCOS (18). These findings suggest that addressing hormonal and metabolic dysregulation may serve as an indirect but effective strategy for reducing infection risk (19). The current findings align partially with prior literature that has explored genitourinary complications in women with PCOS, though few studies have focused specifically on UTIs. Most existing research has emphasized reproductive or metabolic outcomes, often overlooking infections as a comorbidity. This study provides empirical support for the hypothesis that endocrine and metabolic dysfunction in PCOS is not confined to reproductive consequences but may extend to increased infection burden. In doing so, it expands the clinical narrative around PCOS and encourages a more holistic approach to patient care (20).

A key strength of this study lies in its structured assessment of both endocrine and microbiological parameters within a single population. The use of validated hormonal assays, standardized UTI diagnostics, and adjusted regression analysis enhanced the reliability of the findings. Furthermore, the relatively large sample size improved the statistical power and generalizability within similar demographic settings (21). The cross-sectional design, while limiting causal inference, was appropriate for establishing prevalence and associations. Nonetheless, several limitations should be acknowledged. The absence of a control group without PCOS prevented direct comparison with the general population, which could have provided more definitive insights into the excess burden attributable to PCOS (22). The single geographic setting may also restrict external validity to broader populations with different ethnic, environmental, or lifestyle characteristics. Although hormonal assays were conducted in a standardized laboratory, variations in cycle phase among oligo- or amenorrheic participants may have introduced some degree of hormonal fluctuation that could affect interpretation. Additionally, the reliance on self-reported symptoms and behavioral factors may carry recall or reporting bias, particularly in relation to hygiene practices or sexual activity, which are known contributors to UTI risk (23). Despite these constraints, the findings offer meaningful contributions to clinical understanding. They reinforce the importance of comprehensive screening in women with PCOS, not only for reproductive and metabolic concerns but also for urological health. These insights also support the incorporation of infection risk assessment in PCOS management algorithms, particularly in patients with recurrent or unexplained urinary symptoms. Future research should consider prospective cohort designs to establish temporal and causal relationships between hormonal changes and UTI incidence. Interventional studies evaluating whether hormonal or insulin-sensitizing treatments reduce infection rates would also be valuable. Moreover, microbiome studies may help elucidate the specific bacterial dynamics influenced by hormonal shifts, providing a more nuanced understanding of pathogenesis. In conclusion, this study demonstrated that women with PCOS exhibit a higher prevalence of urinary tract infections and that this risk is significantly associated with distinct hormonal and metabolic profiles. The evidence points toward an endocrine-immune interface that may predispose this population to infections, underlining the need for broader clinical awareness and integrative management strategies (24).

CONCLUSION

This study identified a significantly higher prevalence of urinary tract infections among women with polycystic ovary syndrome, closely linked to elevated androgen levels, reduced estrogen and SHBG, and increased insulin resistance. These findings highlight the need for broader clinical vigilance toward urological health in PCOS management and suggest that hormonal and metabolic regulation may play a key role in reducing infection susceptibility in this population.

AUTHOR CONTRIBUTION

Author	Contribution
Shabahat Arain*	Substantial Contribution to study design, analysis, acquisition of Data Manuscript Writing Has given Final Approval of the version to be published
Rimal Rashid	Substantial Contribution to study design, acquisition and interpretation of Data Critical Review and Manuscript Writing Has given Final Approval of the version to be published
Naheed Shah	Substantial Contribution to acquisition and interpretation of Data Has given Final Approval of the version to be published
Haniyah Gul	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Samreen Iqbal	Contributed to Data Collection and Analysis Has given Final Approval of the version to be published
Umair Latif	Substantial Contribution to study design and Data Analysis Has given Final Approval of the version to be published
Summyya Rasheed	Contributed to study concept and Data collection Has given Final Approval of the version to be published

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