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The impact of polycystic ovary syndrome on tubal ectopic pregnancy risk during first pregnancy

Polikistik over sendromunun ilk gebelikte tubal ektopik gebelik riski üzerindeki etkisi

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ABSTRACT

Aim: This study aimed to investigate the effect of Polycystic Ovary Syndrome (PCOS) on the risk of tubal ectopic pregnancy during first pregnancy and how this risk varies across different PCOS phenotypes.

Materials and Methods: This retrospective study analyzed 657 women diagnosed with ectopic pregnancy between November 2022 and November 2024 at a tertiary care hospital. Of these, 222 women had confirmed tubal ectopic pregnancies and a documented diagnosis of PCOS at the same center. The participants were divided into two groups based on the Rotterdam criteria: PCOS (n=76) and non-PCOS (n=146). PCOS phenotypes were further classified as Phenotype A (hyperandrogenism, oligo-/anovulation, and PCOM), Phenotype B (hyperandrogenism and oligo-/anovulation), Phenotype C (hyperandrogenism and PCOM), and Phenotype D (oligo-/anovulation and PCOM).

Results: Women with PCOS had a significantly higher incidence of tubal ectopic pregnancy during their first pregnancy compared to non-PCOS women (OR: 4.42, 95% CI: 2.22–8.80, $p < 0.001$). Among PCOS phenotypes, Phenotype C (hyperandrogenism and polycystic ovarian morphology) was the most common (32.9%), followed by Phenotype D (23.7%). Non-PCOS women exhibited higher rates of conventional risk factors, such as intrauterine device use, pelvic inflammatory disease (PID), and previous pelvic surgeries.

Conclusion: PCOS may be associated with an increased risk of tubal ectopic pregnancy, especially during the first pregnancy. The findings suggest that hormonal and structural disruptions in PCOS, may impair fallopian tube function and embryo transport. These results underscore the need for targeted fertility counseling and management strategies in women with PCOS to mitigate ectopic pregnancy risks.

Keywords: Ciliary motility disorders, ectopic pregnancy, fallopian tubes, polycystic ovary syndrome

ÖZ

Amaç: Bu çalışmanın amacı, Polikistik Over Sendromunun (PKOS) ilk gebelikte tubal ektopik gebelik riskine olan etkisini ve bu riskin farklı PKOS fenotiplerine göre nasıl değiştiğini incelemektir.

Gereç ve Yöntemler: Bu retrospektif çalışmada, Kasım 2022- Kasım 2024 tarihleri arasında üçüncü basamak bir hastanede ektopik gebelik tanısı alan toplam 657 kadın incelendi. Bu kadınlardan 222'sinde hem doğrulanmış tubal ektopik gebelik hem de aynı merkezde tanı almış PKOS tanısı bulunuyordu. Hastalar, Rotterdam kriterlerine göre iki gruba ayrıldı: PKOS'lu (n=76) ve PKOS olmayan (n=146). PKOS'lu kadınlar ayrıca şu fenotiplere göre sınıflandırıldı. PKOS fenotipleri, Fenotip A (hiperandrojenizm, oligo-/anovulasyon, polikistik over morfolojisi [PKOM]), Fenotip B (hiperandrojenizm, oligo-/anovulasyon), Fenotip C (hiperandrojenizm, PKOM) ve Fenotip D (oligo-/anovulasyon, PKOM) olarak sınıflandırıldı.

Bulgular: PKOS'lu kadınlarda ilk gebeliklerinde, PKOS olmayan kadınlara kıyasla anlamlı derecede daha yüksek tubal ektopik gebelik insidansı gözlendi (OR: 4.42, 95% CI: 2.22–8.80, $p < 0.001$). PCOS fenotipleri arasında Fenotip C (hiperandrojenizm ve PKOM) en yaygın olanıydı (%32.9), ardından Fenotip D (%23.7) geldi. PKOS olmayan kadınlar, daha yüksek oranlarda geleneksel risk faktörleri (rahim içi araç, pelvik inflammatuar hastalık, önceki pelvik cerrahiler) gösterdi.

Sonuç: PKOS, özellikle ilk gebelik sırasında tubal ektopik gebelik riskini artırabilir. Hormonal ve yapısal bozukluklar, fallop tüpü fonksiyonlarını ve embriyo taşınmasını bozarak bu riski artırabilir. Bulgular, PKOS'lu kadınlarda ektopik gebelik riskini azaltmaya yönelik hedefe yönelik fertilite danışmanlığı ve yönetim stratejilerinin önemini vurgulamaktadır.

Anahtar kelimeler: Ektopik gebelik, fallop tüpleri, polikistik over sendromu, silyer motilite bozuklukları,tubal geçirgenlik

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INTRODUCTION

Polycystic Ovary Syndrome (PCOS) is a complex endocrine disorder that affects women of reproductive age, presenting with symptoms such as hyperandrogenism, menstrual irregularities, and polycystic ovarian morphology (PCOM) (1). PCOS is often associated with ovulatory dysfunction, insulin resistance, and chronic low-grade inflammation, all of which can have systemic effects beyond ovarian function alone (2,3). While much of the research on PCOS has focused on its impact on ovulation and fertility, there is growing interest in its potential effects on the fallopian tubes, particularly in the context of tubal patency and ciliary function (2-7).

Tubal patency, the openness and functional capacity of the fallopian tubes, is crucial for normal fertility, as it allows for the transport of sperm, eggs, and embryos through the reproductive tract (8,9). In women with PCOS, however, hormonal imbalances and inflammatory processes may impair tubal patency, increasing the risk of abnormal embryo implantation outside the uterine cavity (2,3,10). Additionally, the ciliated cells lining the fallopian tubes play an essential role in the directed movement of the embryo toward the uterus (11). Studies suggest that the hyperandrogenism and altered hormonal milieu characteristic of PCOS may reduce ciliary activity, potentially disrupting this delicate transport process (12-14).

The combined effects of altered tubal patency and reduced ciliary activity may contribute to a higher incidence of tubal ectopic pregnancies among women with PCOS (15,16). The primary objective of this study was to investigate how PCOS, particularly hormonal and structural disruptions associated with this condition, influences the risk of tubal ectopic pregnancy during the first pregnancy. We further aimed to analyze variations in risk according to different PCOS phenotypes.

MATERIALS AND METHODS

This retrospective cohort study included 657 women diagnosed with ectopic pregnancy at a tertiary hospital between November 2022 and November 2024. Approval for the study protocol was obtained from the Institutional Review Board. Participants provided informed consent during hospital admission, permitting their medical records to be utilized for future research purposes. The study strictly followed the ethical guidelines outlined in the Declaration of Helsinki.

Women with non-tubal ectopic pregnancies (e.g., cervical, interstitial, ovarian, or abdominal), incomplete medical records, or missing data regarding PCOS or ectopic pregnancy characteristics were excluded. Additional exclusions included women diagnosed with infertility who underwent assisted reproductive technology (ART) or ovulation induction treatments, those with a history of uterine or ovarian malignancy, congenital uterine anomalies (e.g., bicornuate uterus), systemic illnesses affecting reproductive outcomes (e.g., uncontrolled diabetes, severe thyroid dysfunction, or systemic lupus erythematosus), and those who declined or were unable to provide informed consent. Figure 1 shows the flow chart of the study.

A total of 222 women with confirmed tubal ectopic pregnancies were included in the study. Diagnosis was made based on a combination of clinical presentation, ultrasound findings, and laboratory markers. Ultrasound findings supporting the diagnosis included the absence of an intrauterine pregnancy along with the presence of an adnexal mass or free fluid in the pelvis. Additionally, periodic measurements of human chorionic gonadotropin (HCG) levels further confirmed the diagnosis (17,18). These criteria ensured accurate and reliable identification of tubal ectopic pregnancies within the study population.

To assess the presence of polycystic ovary syndrome (PCOS), each participant's medical history was reviewed, and previous diagnoses of PCOS at the same hospital were confirmed through medical records. The Rotterdam criteria were consistently applied to define PCOS within the cohort (19). According to these criteria, the diagnosis of PCOS requires at least two of the following: PCOM on ultrasound, clinical or biochemical signs of hyperandrogenism or hirsutism, and evidence of anovulation. Women with a confirmed diagnosis of PCOS were grouped accordingly, while those without a prior PCOS diagnosis (n=76) were placed in the non-PCOS group (n=146) (Figure 1).

PCOS phenotypes were categorized as follows: Phenotype A included clinical or biochemical hyperandrogenism, oligo-/anovulation, and PCOM; Phenotype B consisted of hyperandrogenism along with oligo-/anovulation; Phenotype C involved hyperandrogenism and PCOM; and Phenotype D was characterized by oligo-/anovulation combined with PCOM (20).

Data were collected at the time of admission and supplemented with information from electronic medical records. The variables included demographic characteristics (age, BMI, smoking status), reproductive history (parity, previous ectopic pregnancies), history of pelvic inflammatory disease (PID), history of tubal and pelvic surgeries, presence of intrauterine device (IUD) and duration of infertility if infertility treatments were present), and clinical parameters related to PCOS, such as a history of anovulation and PCOM findings.

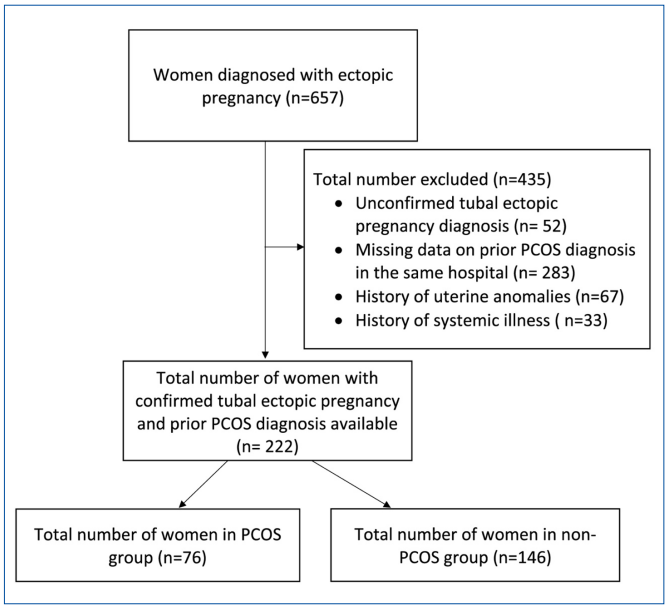


Figure 1. Flow chart of the study

Statistical Analysis

Statistical analyses were performed using SPSS software (version 26). The normality of continuous variables was assessed visually using histogram plots and by evaluating skewness and kurtosis values. Continuous variables were compared between the PCOS and non-PCOS groups using independent t-tests. Categorical variables were analyzed with Chi-square or Fisher's exact tests, depending on the distribution of the data. To examine the association between PCOS and tubal ectopic pregnancy, logistic regression analysis was conducted, adjusting for potential confounders such as age, BMI, history of PID, and history of tubal or pelvic surgeries. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated to quantify the strength of the association between PCOS and the risk of ectopic pregnancy. A p-value of < 0.05 was considered statistically significant.

Table 1. Demographic and Clinical Characteristics of Study Participants

	(N =222)
Age (y) (mean±SD)	30.37±4.57
BMI (kg/m²) (mean±SD)	25.37±4.35
Infertility time (month) (median,min-max)	6.36±13.65
Gravidity (median,min-max)	3 (1-7)
Parity (median,min-max)	1 (0-4)
Abortus (median,min-max)	1 (0-3)
	N (%)
History of Ectopic pregnancy	
no	121 (54.5)
1 time	91 (41)
2 times	10 (4.5)
History of Ectopic Pregnancy in the First Pregnancy	
No	177 (79.7)
Yes	45 (20.3)
Smoking	
No	143 (64.4)
Yes	79 (35.6)
Presence of Intrauterine Device	
No	196 (88.3)
Yes	26 (11.7)
History of PID	
No	201 (90.5)
Yes	21 (9.5)
History of Tubal Surgery	
No	214 (96.4)
Yes	8 (3.6)
History of Pelvic surgery	
No	202 (91)
Yes	20 (9)
PCOS Diagnosis	
No	146 (65.8)
Yes	76 (34.2)

BMI: Body mass index; PCOS: Polycystic ovary syndrome; PID: Pelvic inflammatory disease

RESULTS

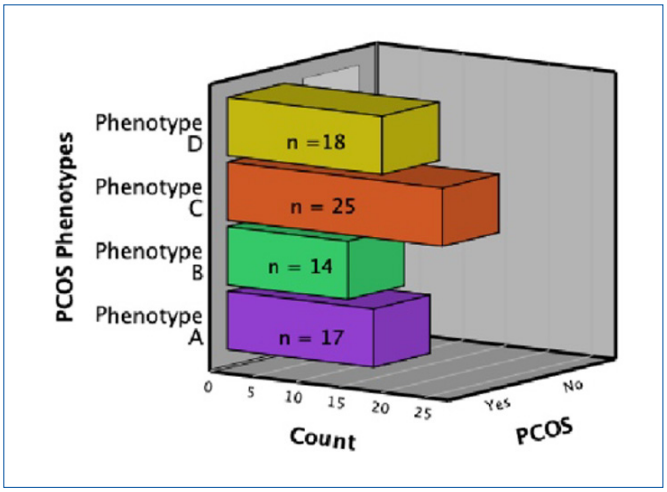
Table 1 presents the demographic and clinical characteristics of the study population, which included 222 women aged between 19 and 43 years. Among them, 76 (34.2%) were in the PCOS group, while 146 (65.8%) were in the non-PCOS group. Comparison between the two groups revealed that the non-PCOS group had a significantly higher prevalence of PID (18 vs. 3, $p = 0.043$) and prior pelvic surgeries (19 vs. 1, $p = 0.004$). Additionally, smoking prevalence was significantly higher in the non-PCOS group (41.8% vs. 23.7%, $p = 0.008$). None of the women in the study received infertility treatment. No significant difference was observed between the groups in terms of overall history of ectopic pregnancy or the number of previous ectopic pregnancies and presence of IUD. However, the incidence of ectopic pregnancy during the first pregnancy was significantly higher in the PCOS group (Table 2).

In the PCOS group, the most commonly reported feature was polycystic ovarian morphology, observed in 78.9% of women, followed by clinical or biochemical hyperandrogenism in 75% and oligo-/anovulation in 64.5%. Based on these features, the women were classified into four phenotypes: 17 women were classified

Table 2. Comparison of Demographic and Clinical Characteristics Between Non-PCOS and PCOS Groups

	non-PCOS group (n= 146)	PCOS group (n= 76)	p
Age (y) (mean±SD)	30.47±4.36	30.18±4.97	0.657
BMI (kg/m²) (mean±SD)	25.82±4.37	24.5±4.21	**0.032**
Infertility time (month) (median,min-max)	4.79±10.12	9.39±18.36	**0.045**
Gravidity (median,min-max)	3 (1-7)	3 (1-5)	0.324
Parity (median,min-max)	1 (0-4)	1 (0-2)	0.621
Abortus (median,min-max)	1 (0-3)	1 (0-2)	0.231
	N (%)		
History of Ectopic pregnancy			0.234
no	85 (58.2)	36 (47.4)	
1 time	56 (38.4)	35 (46.1)	
2 times	5 (3.4)	5 (6.6)	
History of Ectopic Pregnancy in the First Pregnancy			**<0.001**
No	129 (88.4)	48 (63.2)	
Yes	17 (11.6)	28 (36.8)	
Smoking			**0.008**
No	85 (58.2)	58 (76.3)	
Yes	61 (41.8)	18 (23.7)	
Presence of Intrauterine Device			0.086
No	134 (91.8)	74 (97.4)	
Yes	12 (8.2)	2 (2.6)	
History of PID			**0.043**
No	128 (87.7)	73 (96.1)	
Yes	18 (12.3)	3 (3.9)	
History of Tubal Surgery			0.843
No	141 (96.6)	73 (96.1)	
Yes	5 (3.4)	3 (3.9)	
History of Pelvic surgery			**0.004**
No	127 (87)	75 (98.7)	
Yes	19 (13)	1 (1.3)	

BMI: Body mass index; PCOS: Polycystic ovary syndrome; PID: Pelvic inflammatory disease



as Phenotype A, 14 as Phenotype B, 25 as Phenotype C, and 18 as Phenotype D (Figure 2). When comparing the phenotypes, no significant differences were found in terms of age, BMI, duration of infertility, history of PID or the presence of IUD. The prevalence of ectopic pregnancy during the first pregnancy varied across the phenotypes: 41.2% in Phenotype A (n=7), 57.1% in Phenotype B (n=8), 20% in Phenotype C (n=5), and 33.3% in Phenotype D (n=6).

Figure 2. Distribution of Women Across PCOS Phenotypes (Phenotype A includes clinical or biochemical hyperandrogenism, oligo-/anovulation, and polycystic ovarian morphology (PCOM); Phenotype B comprises hyperandrogenism along with oligo-/anovulation; Phenotype C includes hyperandrogenism and PCOM; and Phenotype D is characterized by oligo-/anovulation combined with PCOM)

Table 3. Logistic Regression Analysis of PCOS and PCOS Phenotypes for the Association with Ectopic Pregnancy During the First Pregnancy

Variable		ß	Lower 95% CI	Upper 95% CI	p
PCOS diagnosis	No	ref	-	-	-
	Yes	4.42	2.22	8.8	**< 0.001**
Phenotype A		4.75	1.61	13.98	**0.005**
Phenotype B		9.05	2.83	28.96	**< 0.001**
Phenotype C		1.69	0.57	5.05	0.342
Phenotype D		3.39	1.13	10.11	**0.028**

PCOS: Polycystic ovary syndrome

The diagnosis of PCOS was found to be 4.42 times more likely to be associated with ectopic pregnancy during the first pregnancy (OR: 4.42, 95% CI: 2.22–8.80, $p < 0.001$). When examining the PCOS phenotypes, Phenotypes B, A, and D were found to be associated with an increased likelihood of ectopic pregnancy during the first pregnancy (Table 3). However, none of the other variables, including age, BMI, smoking status, history of PID, history of tubal or pelvic surgery, or the presence of IUD, were found to be significantly associated with ectopic pregnancy during the first pregnancy.

DISCUSSION

The findings indicate that women with PCOS may be at an elevated risk for ectopic pregnancy, especially during their first pregnancy. This elevated risk highlights a potentially significant, but understudied, area in reproductive medicine—how PCOS-related hormonal and structural alterations may influence tubal function and, consequently, pregnancy location.

PCOM was the most common feature in the PCOS group (78.9%), followed by clinical or biochemical hyperandrogenism (75%) and oligo-/anovulation (64.5%). These findings are consistent with previous research showing that PCOM is a key feature of PCOS and is linked to hormonal imbalances and ovulatory dysfunction (21). Ozel et al. found a significant association between PCOM and ectopic pregnancy, suggesting PCOM could serve as an early indicator of increased risk for ectopic pregnancies (7).

Different PCOS phenotypes may uniquely influence tubal health. In our study, women with Phenotypes C and D exhibited a lower prevalence of ectopic pregnancy during their first pregnancy. This finding aligns with a study by Ghobrial et al. who showed that women with these phenotypes had a lower likelihood of tubal occlusion (5). These phenotypes may be associated with a reduced risk of tubal damage and ectopic pregnancy.

Research on the impact of testosterone on fallopian tubes suggests that elevated androgen levels—common in PCOS—can impair ciliary function and disrupt embryo transport, thereby increasing the risk of ectopic pregnancy (22). In another study examining testosterone exposure, changes such as ciliary clumping and partial luminal blockage were observed, which might increase the risk of tubal occlusion and ectopic pregnancy (23). These findings support the hypothesis that hyperandrogenism impacts both ovarian morphology and tubal function. Interestingly, Mayrhofer et al. did not observe significant differences in tubal occlusion between women with PCOS and controls, suggesting that PCOS might not always impair tubal patency, especially in medication-resistant anovulation cases (6).

Although our study found that non-PCOS women had higher rates of PID, prior surgeries, smoking, and IUD use—known contributors to tubal dysfunction—our results suggest that the hormonal milieu in PCOS may be a more significant factor in increasing ectopic pregnancy risk during the first pregnancy. This underscores the need for more focused investigation of PCOS-related tubal changes beyond traditional risk factors.

One of the key strengths of this study is the single-center diagnosis of PCOS, which enhances the reliability and consistency of the data. By diagnosing all participants within the same clinical setting, we were able to ensure a standardized approach, reducing variability in the diagnostic process and strengthening the validity of the findings. Despite adjusting for potential confounders, there may be other unmeasured factors that could still influence the results. Given the retrospective nature of this study, we acknowledge that causality cannot be conclusively determined. Further prospective studies are recommended to validate our findings and clarify the

mechanisms underlying the relationship between PCOS and tubal ectopic pregnancy. Additionally, multi-center studies are needed to validate these results in broader populations.

CONCLUSIONS

Our findings indicate that women with PCOS may be at an increased risk for ectopic pregnancy, particularly during their first pregnancy, likely due to altered tubal patency and reduced ciliary function. These results emphasize the need for further prospective studies to explore the specific mechanisms through which hormonal imbalances in PCOS affect fallopian tube function. Targeted interventions may be beneficial in reducing the risk of ectopic pregnancy in this population. Additionally, our findings highlight the importance of targeted fertility counseling and management strategies for women diagnosed with PCOS. Regular hormonal evaluations, fertility counseling, and close monitoring during early pregnancy could be beneficial in mitigating the risk of tubal ectopic pregnancies.

Author Contributions

Conceptualization: B.K, Data Curation: S.M., S.K.E, Formal Analysis: B.K., C.K, Investigation: B.K., S.M., S.K.E, Methodology: B.K., C.K., S.M, Project Administration: B.K,, S.K.E, Supervision: C.K, Writing – Original Draft: B.K, Writing – Review & Editing: B.K., C.K.

Conflict of Interest

Authors declare no conflict of interest.

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