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Systematic Review

Association between polycystic ovary syndrome and endometrial cancer risk: a systematic review

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ABSTRACT

Background: Objective of the study was to clarify the relationship between the risk of endometrial carcinoma (EC) and polycystic ovary syndrome (PCOS).
Methods: A thorough search across four databases identified 816 relevant publications. After removing duplicates using Rayyan Qatar Computing Research Institute (QCRI) and screening for relevance, 383 full-text articles were reviewed, with 6 studies ultimately meeting the criteria for inclusion.
Results: A total of 25,016 women with PCOS diagnoses were involved in six investigations. With a total incidence of 254 (1%), the prevalence of EC among PCOS patients varied from 0.13% to 52%. Patients with PCOS are more likely to get EC, according to many research. The underlying causes are linked to chronic endometrial estrogen stimulation, with contributing factors including hyperlipidemia, diabetes mellitus, and hypertension. These factors increase the chance of EC in women with PCOS.
Conclusions: These findings demonstrate the robust correlation between PCOS and a higher risk of EC, emphasizing the need for proactive monitoring and prevention in clinical practice. While the evidence points to significant risk factors such as metabolic disturbances, further research is needed to establish a direct causal link and address potential biases. Nevertheless, integrating cancer risk management into the care of women with PCOS is essential for reducing long-term complications.

Keywords: PCOS, Endometrial cancer, Hormonal imbalance, Reproductive health, Systematic review

INTRODUCTION

A prevalent endocrine condition that affects women of reproductive age is polycystic ovary syndrome (PCOS), characterized by a number of symptoms, such as polycystic ovaries, hyperandrogenism, and irregular menstrual periods. The incidence of PCOS is substantial, impacting about 5-10% of women in this demographic.[1] Given its widespread occurrence, understanding the associated health risks, particularly those related to reproductive health, is crucial. One of the major concerns for women with PCOS is the possible increased risk for endometrial carcinoma (EC), a malignancy originating from the lining of the uterus.[2] At its most basic, PCOS can be characterized as a hormone imbalance that may cause an assortment of symptoms.

High levels of androgens, a group of male hormones, resulting in hirsutism - excess hair growth, acne, and male-pattern baldness, are common among females with PCOS. Besides, insulin resistance often accompanies PCOS in women, which is a predisposing factor for type 2 diabetes

and metabolic syndrome. The menstrual cycle disturbances such as amenorrhea or oligomenorrhea add to the health complications of the women with PCOS.[3] In developed countries, EC is the most prevalent gynecological malignancy and is predominantly associated with hormonal disturbances. While the incidence is rising in younger age groups, the majority of cases occur in postmenopausal women. The malignancy usually manifests with postmenopausal or irregular bleeding, pelvic pain, or other menstrual abnormalities. Risk factors include obesity, diabetes, unopposed estrogen exposure, and anovulation, all of which have been documented in women with PCOS.[2]

The persistent anovulation that many women with PCOS experience is one of the main ways that the syndrome may alter their chance of developing EC. When ovulation does not occur, the endometrium is continuously exposed to estrogen without the balancing effect of progesterone, which is typically produced after ovulation. This prolonged unopposed estrogen environment can stimulate excessive proliferation of the endometrial lining, leading to hyperplasia, and eventually, increase the risk of developing EC.[4]

It has been estimated that endometrial hyperplasia occurs four to seven times more frequently in women with PCOS, and a subset of these may progress to develop EC. A meta-analysis of studies examining the cancer risk in women with PCOS reported that the incidence of EC was significantly higher than in the general population.[5]

The mechanisms linking PCOS and EC risk are multifaceted. Firstly, the insulin resistance commonly seen in most patients with PCOS often relates to higher insulin and glucose levels in the blood. High insulin levels stimulate ovarian androgen production and, simultaneously, endometrial cell proliferation. Hyperinsulinemia may result in increased bioavailability of estrogen, further compounding the risk.[2,5]

The second known risk factor for EC is obesity, a common comorbidity among women with PCOS. Adipose tissue conversion of androgens to estrogens through peripheral aromatization elevates estrogen levels, increasing exposure to the endometrium without adequate progesterone.[1]

Since there is evidence that PCOS and EC risk are connected, endometrial health should be closely monitored as part of PCOS therapeutic therapy. Regular gynecological evaluations and proactive screening methods, such as transvaginal ultrasound or endometrial biopsy, are important for at-risk patients.

Lifestyle modifications, including weight management and exercise, can significantly reduce the risks associated with PCOS by improving insulin sensitivity and menstrual regularity.[6]

The administration of hormonal medications, in most cases oral contraceptives that include both estrogen and progesterone, is often indicated not only for the normalization of menstrual cycles but also for the prevention of endometrial hyperplasia and cancer. In addition, the benefits of insulin-sensitizing drugs such as metformin for women with PCOS, especially those with attendant insulin resistance, have been explored.[7]

Despite the growing literature addressing the implications of PCOS, ambiguity still surrounds its role in increasing the risk of EC. There is some evidence that a link may exist, given that women with PCOS are at a greater risk of obesity, insulin resistance, and delayed anovulation. However, inconsistencies in study methodologies, sample sizes, and population demographics make it impossible to reach a conclusive understanding of this relationship. This means, due to the lack of systematic summary of the available data, healthcare providers cannot have definite recommendations on monitoring and treating the disease in women with PCOS. Therefore, a need arises for a systematic review in order to clarify not only the association between PCOS and risk of EC but also to bring order into existing uncertainties. Such a review is thus warranted on the effects of PCOS in causing EC, with its analysis that will contribute towards making understanding rather clear about how long-term morbidity may be generated concerning women's health via the involvement of PCOS.

METHODS

The preferred reporting items for systematic reviews and meta-analyses (PRISMA) standards for systematic reviews and meta-analyses have been followed to conduct the present systematic review of articles that assessed the association between PCOS and the risk of EC.[8]

A comprehensive electronic search was carried out in several databases such as PubMed, Web of Science, Scopus, and Science Direct to identify all relevant English-language studies about the prevalence and risk factors of PCOS and its relation to EC. The search keywords were used interchangeably related to PCOS and its associated cancer risks. Two independent reviewers will analyze the search results, select studies that fulfill the eligibility criteria, extract relevant information, and evaluate the quality of the included studies using recognized evaluation instruments.

Eligibility criteria

The eligible studies should be in the English language, target women diagnosed with PCOS, and report data about incidence or risk factors of EC.

The included observational and interventional study types were cohort studies, case-control studies, and randomized controlled trials. Studies that were excluded involved nonhuman participants, articles with a lack of relevant data

regarding the risk of EC, and any studies not utilizing established criteria for the diagnosis of PCOS. Moreover, reviews, commentaries, and conference abstracts were excluded to capture empirical research that contributes valued knowledge to the topic.

Data extraction

The search results were checked against Rayyan (QCRI) to ensure that results are valid.[9] Titles and abstracts retrieved by the search were checked for relevance using the predetermined inclusion and exclusion criteria. All the included studies were critically reviewed by the study team. Any disagreements among the reviewers were settled by a consensus approach. Data from each study were systematically extracted using a structured data extraction form and included information on titles, authors, year of publication, location of study, characteristics of participants, gender distribution, and the epidemiology and risk factors associated with PCOS and EC. The risk of bias for the selected studies was also assessed using an independent rating method.

Data synthesis strategy

Summary tables were constructed using data from relevant studies to provide a qualitative assessment of the research findings and elements. After the data collection for the systematic review was completed, the best method of using the data from the selected studies was identified.

Quality review

Since bias due to missing factors is common in studies in this area, we applied the ROBINS-I tool to judge the risk of bias as it allows for comprehensive evaluation of confounding.

The ROBINS-I tool is intended to evaluate non-randomized studies and can be applied in cohort models in which individuals exposed to various staffing levels are followed for a considerable period of time. Disagreements were resolved by group discussion when each paper's risk of bias was assessed independently by two reviewers.[10]

RESULTS

The identified search strategy retrieved 816 publications (Figure 1). After duplicates were removed, n=433, 383 trials were assessed for title and abstract. Of these, 306 did not meet the eligibility criteria, leaving only 77 full-text articles that were assessed in full. Four records were identified through citation search, and only 73 were accepted into our review. A total number of 6 met the eligibility criteria with evidence synthesis for analysis, three being case-controls, two retrospective cohorts, and one was a cross-sectional study.[11-16]

Sociodemographic and clinical outcomes

A total of 25,016 women with PCOS diagnoses were involved in six investigations. The diagnosis of PCOS was variable depending on self-reported data, clinical hospital records, or validated criteria. EC in all patients depended on histological confirmation and physicians' diagnosis.

Between 0.13% and 0.13 percent of PCOS patients had EC to 52% with 254 (1%), the overall prevalence.[11,15,16] Patients with PCOS have an increased risk of acquiring EC, according to several studies.[11-16] The underlying causes are linked to chronic endometrial estrogen stimulation, with contributing factors including hyperlipidemia, diabetes mellitus, and hypertension.[11] These conditions amplify the risk of EC in PCOS-affected women.

PCOS individuals are more likely to develop endometrial hyperplasia (EH), which can result in cancer, if they are obese, have longer menstrual cycles, have decreased sex hormone-binding globulin (SHBG), or have dyslipidemia.[13] EC is more prevalent in PCOS-afflicted women, especially in younger women. This highlights the unique vulnerability of younger PCOS patients to the risk of endometrial malignancies.[16]

Table 1: Outcome measures of the included studies.

Study ID	Coun-try	Study design	Sociodemog-raphic	Prevalen-ce of EC	PCOS diagnosis	EC diagnosis	Key findings
Ding et al, 2018[11]	Taiwan	Retrospective cohort	Participants: 8155, age range: 15-49	11 (0.13%)	Clinical records	Primary carcinoma with histological confirmation; doctor's diagnosis	The chance of acquiring EC was increased in those with PCOS. Chronic endometrial estrogen stimulation might represent the underlying etiology. Other risk factors for EC include hyperten-sion, diabetes mellitus, and hyperlipidemia.
Iqbal et al, 2023[12]	Pakistan	Case-control	Participants: 35, age range: 20-45	24 (34.4%)	Self-reported	Primary carcinoma with histological	Women who have PCOS are ten times more likely to develop EC since PCOS is

Continued.

Study ID	Coun-try	Study design	Sociodemog-raphic	Prevalen-ce of EC	PCOS diagnosis	EC diagnosis	Key findings
						confirmation; doctor's diagnosis	a risk factor for the disease.
Zhong et al, 2023[13]	China	Case-control	Participants: 234, mean age: 27.47	11 (4.7%)	The Rotterdam 2003 criteria	Primary carcinoma with histological confirmation; doctor's diagnosis	Patients with PCOS who are obese, have a longer menstrual cycle, have dyslipidemia, and have decreased SHBG are more prone to get EH.
Lu et al, 2023[14]	China	Retrospective cohort	Participants: 4236, mean age: 42	43 (1.02%)	The Rotterdam 2003 criteria	Primary carcinoma with histological confirmation; doctor's diagnosis	Both premalignant and malignant endometrial polyps are more common in premenopausal women with PCOS.
Abu-Zaid et al, 2024[15]	Saudi Arabia	Cross-sectional	Participants: 286, mean age: 42.1	149 (52%)	Clinical records	Primary carcinoma with histological confirmation; doctor's diagnosis	PCOS as a significant risk factor for EC, but not for ovarian or cervical cancers.
Gottsc-hau et al, 2015[16]	Denmark	Case-control	Participants: 12,070, age range: 9-49	16 (0.13%)	Clinical records	Primary carcinoma with histological confirmation; doctor's diagnosis	Women with PCOS are more likely to get EC. The increase may only impact younger women with PCOS.

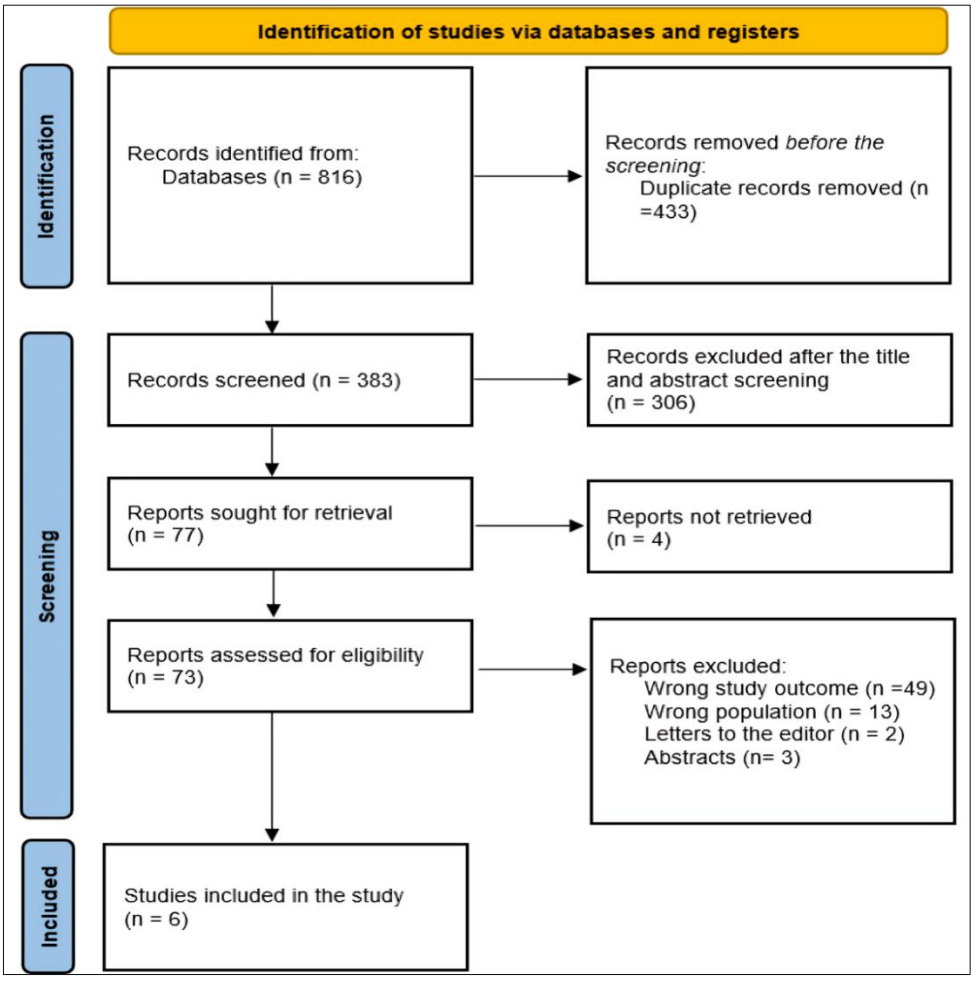

Figure 1: PRISMA flowchart.

Table 2: Risk of bias assessment using ROBINS-I.

Study ID	Bias brought induced by confusion	Bias in the way participants were chosen for	Prejudice in the way interventi-ons are category-zed	Bias resulting from departures from the planned interval	Bias resulti-ng from absent data	Bias in the way results are mea-sured	Prejudice in the chosen reported outcome	Gener-al pre-judice
Ding et al, 2018[11]	Low	Low	Mod	Low	Low	Low	Mod	Low
Iqbal et al, 2023[12]	Low	Low	Mod	Low	Low	Low	Mod	Low
Zhong et al, 2023[13]	Mod	Low	Low	Low	Low	Mod	Low	Low
Lu et al, 2023[14]	Mod	Mod	Low	Low	Low	Mod	Low	Modera-te
Abu-Zaid et al, 2024[15]	Mod	Low	Mod	Low	Low	Mod	Low	Modera-te
Gottschau et al, 2015[16]	Mod	Crit	Low	Low	Low	Mod	Low	Critical

DISCUSSION

According to this research, the prevalence of EC in PCOS patients varied from 0.13% to 52% with a total prevalence of 254 (1%).[11,15,16] EC is more likely to occur in people with PCOS, according to many research.[11-16] This elevated risk is primarily associated with prolonged estrogen stimulation of the endometrium. Additional factors, such as hyperlipidemia, diabetes mellitus, and hypertension, further increase the total risk by contributing to the increased incidence of EC in women with PCOS.[11] Similarly, Johnson et al reported that compared to women without PCOS, individuals with PCOS had a higher chance of developing EC.[17]

Shetty et al documented that in order to solidify the link between the mechanisms causing gynecological malignancies in females with PCOS, Large-scale, long-term studies are still required for further research. It is clear from analyzing the ten research that women with PCOS have a markedly higher chance of developing EC.[18]

In PCOS patients, the hormonal imbalance caused by anovulation is associated with unopposed estrogen activity.[19] Proliferation and differentiation triggered by estrogen may lead to endometrial hyperplasia and ultimately EC. Evidence of intermediate quality indicates that women with PCOS had a 2.7-fold increased lifetime risk of EC. The relationship between PCOS and EC has been the subject of several investigations over the past decade. EC is around three times more common in women with PCOS, according a meta-analysis of eight pertinent studies published between 1968 and 2008.[20] Additionally, another meta-analysis that included 72,054 non-PCOS controls and 919 PCOS-afflicted women found that women with PCOS were more likely to have EC.[21]

After controlling for BMI, the correlation between PCOS and EC vanished, which is consistent with the findings of Fearnley et al. These examples demonstrate that the association between PCOS and EC may be sufficiently explained by the fact that obesity is a factor in both illnesses.[19] Furthermore, 8155 Taiwanese women with PCOS had an aggregate 17-fold higher risk of EC than women without PCOS.[20] The incidence of EC was 1.5 instances per 100,000 person-years in the comparator group and 22.6 cases per 100,000 person-years in the PCOS group. Again, EC risk variables such as BMI, metabolic syndrome, and fertility were not adjusted or assessed as possible confounders.

EH, which can result in cancer, is more likely to develop in PCOS individuals who are obese, have longer menstrual cycles, have decreased SHBG, or have dyslipidemia.[13] EC is more common in women with PCOS, particularly in younger women. This highlights how younger PCOS patients are especially vulnerable to the rise in EC.[16] These findings are controversial. Obesity, nulliparity, age over 50, infertility, hypertension, diabetes, chronic anovulation, and unopposed estrogen supplementation all dramatically raise EC in women with PCOS.[23]

It is still unknown to what extent BMI increases the risk of EC in women with PCOS. It is also necessary to take into consideration that additional confounding variables, such as diabetes, parity, and hormone use, might alter the results of the current review.

These studies put into perspective a proactive management by the clinicians in women with PCOS for EC. In fact, it supports the encouragement of frequent screenings and diligent follow-ups that should be afforded to all patients with PCOS, with special emphasis on those with extra risk

factors such as obesity, hypertension, hyperlipidemia, and diabetes mellitus. These metabolic and cardiovascular conditions, so common in PCOS, appear to enhance the risk of EC. The same hormonal therapies, including progestogens, and drugs usually prescribed for managing insulin resistance in PCOS, such as metformin, may also be protective against endometrial hyperplasia and subsequent cancer development. It is equally important to educate patients about the importance of controlling these risk factors through lifestyle changes like exercise and food. Such preventive strategies can reduce the long-term cancer risks faced by this patient population.

Strengths and limitations

The robustness of these findings is supported by several key strengths. First, the inclusion of large sample sizes in many of the studies strengthens the statistical power and reliability of the associations drawn between PCOS and EC risk. These large cohorts also enable more precise estimates of risk across a wide range of age groups and demographic backgrounds. The diversity in the study designs used, both retrospective cohort and case-control methods, helps in arriving at a comprehensive understanding of how PCOS influences EC risk across different populations. These studies have been conducted on subjects from various parts of the world, including Asia and Europe, giving a wider geographical perspective and ensuring that these results are not limited to only one ethnic or geographical group. Also, with several risk factors being identified such as obesity, dyslipidemia, and longer menstrual cycle, the clinicians get an all-round view of the possible facilitators of cancer in PCOS patients.

Several limitations must be acknowledged, however. Another limitation is that these studies fail to identify a direct cause-effect relationship of PCOS on EC. Associations demonstrated may be strong, yet the causality remains indefinite, especially considering that EC in women with PCOS might also be influenced by some confounding variables like lifestyle and genetic predispositions. Longitudinal tracking could help elucidate the exact nature of this association. Moreover, several studies depended on self-reported data for the diagnosis of PCOS, which introduces the possibility of reporting bias or diagnostic inaccuracy. Furthermore, despite the geographical dispersion of the studies, some populations are still overrepresented, especially in Asian cohorts, which may limit the generalizability of findings to other ethnicities or less-well-studied geographic locations.

CONCLUSION

In the light of these facts, these results emphasize the need for raising awareness and aggressively managing the risk of EC in women suffering from PCOS. These strong associations between PCOS, metabolic disturbances, and EC risk suggest that clinicians should broadly monitor and prevent the condition. Although the findings provide important insights, further research will be needed to establish causality and address potential biases in the current literature. Future studies are needed focusing on diverse populations and long-term outcomes to better understand the mechanisms through which PCOS is associated with EC. Available information, however, gives sufficient reason to include EC risk management into the therapeutic treatment of women afflicted with PCOS.

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REFERENCES

1. Collaborative Group on Epidemiological Studies of Ovarian Cancer; Beral V, Doll R, Hermon C, Peto R, Reeves G. Ovarian cancer and oral contraceptives: collaborative reanalysis of data from 45 epidemiological studies including 23,257 women with ovarian cancer and 87,303 controls. Lancet. 2008;371(9609):303-14.
2. Dumesic DA, Lobo RA. Cancer risk and PCOS. Steroids. 2013;78:782-5.
3. Daniilidis A, Dinas K. Long term health consequences of polycystic ovarian syndrome: a review analysis. Hippokratia. 2009;13:90-2.
4. Pan ML, Chen LR, Tsao HM, Chen KH. Relationship between Polycystic Ovarian Syndrome and Subsequent Gestational Diabetes Mellitus: A Nationwide Population-Based Study. PLoS One. 2015;10(10):e0140544.
5. Gottschau M, Kjaer SK, Jensen A, Munk C, Mellemkjaer L. Risk of cancer among women with polycystic ovary syndrome: a Danish cohort study. Gynecol Oncol. 2015;136(1):99-103.
6. Azziz R, Carmina E, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Futterweit W, et al; Androgen Excess Society. Positions statement: criteria for defining polycystic ovary syndrome as a predominantly hyperandrogenic syndrome: an Androgen Excess Society guideline. J Clin Endocrinol Metab. 2006;91(11):4237-45.
7. Ding DC, Chen W, Wang JH, Lin SZ. Association between polycystic ovarian syndrome and endometrial, ovarian, and breast cancer: A population-based cohort study in Taiwan. Medicine (Baltimore). 2018;97(39):e12608.
8. Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. Int J Surg. 2021;88:105906.
9. Ouzzani M, Hammady H, Fedorowicz Z, Elmagarmid A. Rayyan—a web and mobile app for systematic reviews. Systemat Rev. 2016;5:1-10.
10. Sterne JA, Hernán MA, Reeves BC, Savović J, Berkman ND, Viswanathan M, et al. ROBINS-I: a tool for assessing risk of bias in non-randomised studies of interventions. BMJ. 2016;12:355.
11. Ding DC, Chen W, Wang JH, Lin SZ. Association between polycystic ovarian syndrome and

endometrial, ovarian, and breast cancer: A population-based cohort study in Taiwan. Medicine. 2018;97(39):e12608.
12. Iqbal K, Khanum Z, Parveen A. Association of polycystic ovarian syndrome with endometrial carcinoma among premenopausal females. Risk. 2023;42(57.1):100.
13. Zhong X, Li Y, Liang W, Hu Q, Zeng A, Ding M, et al. Clinical and metabolic characteristics of endometrial lesions in polycystic ovary syndrome at reproductive age. BMC Women's Health. 2023;23(1):236.
14. Lu L, Luo J, Deng J, Huang C, Li C. Polycystic ovary syndrome is associated with a higher risk of premalignant and malignant endometrial polyps in premenopausal women: a retrospective study in a tertiary teaching hospital. BMC Women's Health. 2023;23(1):127.
15. Abu-Zaid A, Baradwan S, Alyafi M, Al Baalharith M, Alsehaimi SO, Alsabban M, et al. Association between polycystic ovary syndrome and the risk of malignant gynecologic cancers (ovarian, endometrial, and cervical): A population-based study from the USA National Inpatient Sample 2016–2019. Eur J Obstet Gynecol Reprod Biol. 2024;299:283-8.
16. Gottschau M, Kjaer SK, Jensen A, Munk C, Mellemkjaer L. Risk of cancer among women with polycystic ovary syndrome: a Danish cohort study. Gynecol Oncol. 2015;136(1):99-103.
17. Johnson JE, Daley D, Tarta C, Stanciu PI. Risk of endometrial cancer in patients with polycystic ovarian syndrome: A meta analysis. Oncol Lett. 2023;25(4):1-9.
18. Shetty C, Rizvi SM, Sharaf J, Williams KA, Tariq M, Acharekar MV, et al. Risk of gynecological cancers in women with polycystic ovary syndrome and the pathophysiology of association. Cureus. 2023;15(4).
19. Ali, A.T. Reproductive factors and the risk of endometrial cancer. Int J Gynecol Cancer. 2014;24:384-93.
20. Ding DC, Chen W, Wang JH, Lin SZ. Association between polycystic ovarian syndrome and endometrial, ovarian, and breast cancer: A population-based cohort study in taiwan. Medicine (Baltimore). 2018;97:e12608.
21. Harris HR, Terry KL. Polycystic ovary syndrome and risk of endometrial, ovarian, and breast cancer: A systematic review. Fertil Res Pract. 2016;2:14.
22. Iversen L, Sivasubramaniam S, Lee AJ, Fielding S, Hannaford PC. Lifetime cancer risk and combined oral contraceptives: The royal college of general practitioners' oral contraception study. Am J Obstet Gynecol. 2017;216:580.e1-e9.
23. Papaioannou S, Tzafettas J. Anovulation with or without PCO, hyperandrogenaemia and hyperinsulinaemia as promoters of endometrial and breast cancer. Best Pract Res Clin Obstet Gynaecol. 2010;24:19-27.

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