

LONG-TERM HEALTH RISKS IN POLYCYSTIC OVARIAN DISORDER: FROM CARDIOVASCULAR DISEASE TO ENDOMETRIAL CANCERSamuel M¹, Jissin Mathew¹

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Received: 28-04-2025

Accepted: 24-06-2025

Published: 09-07-2025

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ABSTRACT

Polycystic Ovarian Disorder (PCOD) is a health condition caused by several factors and has an effect on systems besides the reproductive ones. The review explores how PCOD can increase a woman's chance of developing cardiovascular disease or endometrial cancer. The problem of PCOD starts with disruptions in three areas: hypothalamic-pituitary-ovarian signalling, insulin resistance, continuous low-level inflammation and excessive body fat deposited around the organs. PCOD often causes hypertension, high cholesterol, dysfunction in the blood vessels and an early form of atherosclerosis, regardless of obesity levels in women. At the same time, when a woman does not ovulate for a long time and is exposed only to oestrogen, her chances of endometrial hyperplasia and cancer increase. Because of insulin resistance and obesity, peripheral oestrogen is produced more and this leads to additional negative outcomes in cancer. The condition may lead to type 2 diabetes, metabolic syndrome, problems with ovulation and subfertility. Implementing changes in lifestyle, using medicine and regularly screening the patients can decrease the dangers of these illnesses. Here, we highlight that PCOD is not only an annoying gynaecological issue, but also a long-term health condition. This means that researchers should adopt teamwork and examine new ways to support and prevent health issues in women at each life stage.

KEYWORDS: Cardiovascular disease, Endometrial cancer, Insulin resistance, Metabolic syndrome, Endocrine disorder.

1. INTRODUCTION

Polycystic Ovarian Disease (PCOD) or Polycystic Ovary Syndrome (PCOS) is a condition of the endocrine system marked by increased signs of female hormone, complications in ovulation and many small ovarian growths (Teede et al., 2018). The global rate of PCOD has risen in women of reproductive age due to inactivity, poor diets and broader knowledge about identifying the disease (Wolf et al., 2018; March et al., 2010). Most treatment recommendations for PCOD are made for fertility, but studies are revealing that it has consequences for the heart, metabolism and certain cancers (Azziz et al., 2016).

A number of factors cause PCOD to increase the chance of conditions such as T2DM, hypertension and even endometrial cancer (Moran et al., 2010; Goodarzi et al., 2011). This means that cancer of the reproductive organs should be watched and managed closely after menopause, as it may still be a risk (Shaw et al., 2008). Many researches have pointed out that women who have PCOD are more likely to develop Cardiovascular Disease (CVD) regardless of factors such as weight gain or smoking (Wild et al., 2010). Dysfunction of blood vessels, changes in fats and lipids and inflammation in these patients lead the body to develop atherosclerosis and raise the odds of having a heart attack or stroke (Essah et al., 2008).

Besides causing heart problems, PCOD can also be related to the development of endometrial disorders. When someone has chronic anovulation, oestrogen keeps accumulating in the body, building up the cells in the lining of the uterus and increasing their chances of becoming hyperplastic or cancerous (Barry et al., 2014).

This may cause serious concern, because women with this condition may not develop symptoms or have uneven menstruation, leading to delayed diagnosis (Chittenden et al., 2009).

Moreover, both insulin resistance and gaining weight due to PCOD may make the situation worse for both hormonal and cancer health problems. To find, control and deal with problems early, healthcare must recognize and address patterns between disease processes (Randeve et al., 2012). The review explores the potential health risks associated with PCOD that appear after many years such as the occurrence of cardiovascular disease or endometrial cancer. The paper describes recent findings in PCOD to inform clinicians, researchers and public health policymakers about the current health challenges due to PCOD.

2. Pathophysiology of PCOD and Its Systemic Impact

Polycystic Ovarian Disease (PCOD) occurs due to genetic, hormonal, metabolic and inflammatory causes. The main features of PCOD include a disturbance in how the HPO axis works, increased insulin resistance and regular low-grade inflammation (Goodarzi et al., 2011). All these links support the development of elevated hormone levels, ovulation problems and issues with body chemistry.

2.1 Neuroendocrine Disruption and HPO Axis

In PCOD, the release of GnRH becomes active and quick which causes LH to be secreted in higher amounts than FSH (Franks, 2008). When there is elevated LH, it leads the theca cells to secrete too many androgens. At the same time, whenever FSH is low, the follicles in the ovaries do not mature properly, stopping ovulation and causing the formation of more than one cyst in the ovaries (Diamanti-Kandarakis et al., 2010).

2.2 Insulin Resistance and Hyperinsulinemia

75% of lean women and almost all obese women with PCOD have insulin resistance (Dunaif, 2006). Too many insulin hormones influence theca cells, remove the suppression of sex hormone-binding globulin in the liver and make the impact of high testosterone more severe (Baillargeon & Nestler, 2006). Because of this connection, the issues with the endocrine and metabolic systems get worse.

2.3 Chronic Low-Grade Inflammation

Evidence from the past several years points to how chronic inflammation plays a part in PCOD development. In women with PCOD, it is common to find raised levels of C-reactive protein, tumour necrosis factor-alpha and interleukin-6 (González et al., 2006). Inflammatory cytokines lead to insulin resistance and increase the risk of diseases related to atherosclerosis and the heart (Spritzer, 2014).

2.4 Adiposity and Lipotoxicity

In PCOD, an abundance of visceral fat increases lipolysis, so free fatty acids are released into the blood. They are responsible for blocking insulin in the body and promote issues such as lipotoxicity in ovarian, hepatic and muscular tissues (Carmina & Lobo, 2004). The presence of obesity increases the effects of certain conditions and makes the diseases faster and more severe.

Table 1: Key Pathophysiological Features of PCOD and Their Systemic Effects

Pathophysiological Factor	Mechanism	Systemic Impact
↑ LH:FSH ratio	Excess LH stimulates androgen production	Anovulation, hyperandrogenism
Insulin resistance	Impaired glucose uptake, ↑ insulin	Type 2 diabetes, worsened hyperandrogenism
Chronic inflammation	↑ CRP, TNF- α , IL-6	Endothelial dysfunction, CVD risk
Visceral obesity	Lipotoxicity, cytokine release	Metabolic syndrome, NAFLD
SHBG suppression	↑ Free testosterone	Hirsutism, acne, infertility

3. Long-Term Cardiovascular Risks in PCOD

Now, medical professionals are seeing that besides affecting reproduction, PCOD can increase a woman's risk of heart complications. Women suffering from PCOD experience increased chances of hypertension, issues with bad cholesterol and minor cardiovascular dysfunctions, even if they are not overweight or diabetic (Wild et al., 2010; Dokras et al., 2011). Due to the development of endocrine, inflammatory and metabolic abnormalities in PCOD, women are more prone to having CVD.

3.1 Hypertension and Blood Pressure Dysregulation

Researchers have observed that women with PCOD are more likely to develop hypertension than women without the condition. Experts have found this is true among individuals of all ages and with different body types, suggesting that PCOS is directly linked to faulty vascular functioning (de Groot and others, 2011). When a person develops insulin resistance, they often keep more sodium and remain aroused, both of which contribute to raised blood pressure (Orio and colleagues, 2004).

The researchers in Kabilan et al. (2021) found that women with PCOD have an increased risk of hypertension, so it is suggested to perform regular heart screenings in these individuals.

3.2 Dyslipidaemia and Lipoprotein Abnormalities

Researchers report that atherogenic dyslipidaemia—having more triglycerides, a higher LDL-C level and lower HDL-C—strongly links to PCOD (Moran et al., 2010). Lipid abnormalities may develop in non-obese PCOD individuals and account for a big share of the risk for atherosclerosis.

When hyperandrogenism is present, there is increased oxidation of LDL in the body and more vascular inflammation (Gambineri et al., 2002). If there is an unhealthy balance of the different lipoproteins in the blood, it causes plaque in the arteries and puts some individuals at higher risk of heart disease.

3.3 Endothelial Dysfunction and Inflammation

Early symptoms of cardiovascular disease can be seen in the form of endothelial dysfunction. Research with FMD has observed a decrease in the endothelium's function among individuals with PCOD (Kelly et al., 2002). The reason for this damage is chronic inflammation, higher oxidative stress and decreased availability of nitric oxide (NO).

Interleukin-6 (IL-6), C-reactive protein (CRP) and homocysteine are examples of markers found in increased amounts in people with PCOD and are believed to play a role in damaging the endothelial cells (Spritzer, 2014; González et al., 2006).

3.4 Atherosclerosis and Subclinical Cardiovascular Disease

Compared to healthy controls, women with PCOD had an increased thickness of the intima-media in their carotids (Talbot et al., 2004). This suggests that PCOD is connected to a long-term inflammation that leads to the early onset of stiff arteries.

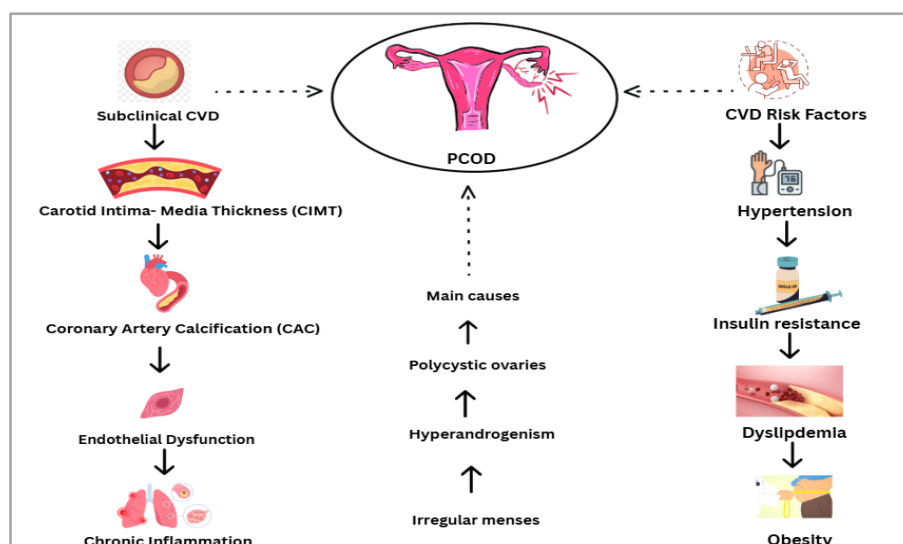


Figure 1: Cardiovascular Sequelae in PCOD

Here is Figure 1 which explains how PCOD results in damaged blood vessels and high cholesterol by impacting hormones, insulin resistance and inflammation.

4. Oncological Risks in PCOD: Endometrial Hyperplasia and Cancer

Women living with PCOD have a higher chance of developing endometrial hyperplasia and cancer. Chronic anovulation, continuous stimulation of estrogen and its effects on metabolism cause most of these oncological dangers (Barry et al., 2014; Chittenden et al., 2009). The lack of ovulation results in less progesterone being produced each cycle. This, in turn, causes uncontrolled growth of the lining of the womb so that the microenvironment permits the start of cancer.

4.1 Mechanisms Linking PCOD to Endometrial Cancer

Most experts link PCOD and endometrial malignancy to hormonal imbalances. If left unchecked by progesterone, prompted by high levels of oestrogen, endometrial cells undergo unusually rapid division which can progress to atypia (Mutter, 2000). Research has revealed that increased insulin and androgen levels increase the endometrium's growth via its IGF and androgen receptors (Fearnley et al., 2010).

Besides, when a woman has PCOD, having insulin resistance and being overweight can increase the level of estrogen in the endometrium by altering androgens into estrogen (Pasquali et al., 2006). As the changes progress, hyperplasia becomes atypical and then changes into endometrial carcinoma.

4.2 Histological Progression and Risk Levels

The endometrium in PCOD patients is sometimes proliferative, sometimes hyperplastic (without or with atypia) and in some cases shows type I adenocarcinoma (or cancer) (Kurman & Ellenson, 2011). Wang et al. (2015) discovered that 28 out of every 100 patients with polycystic ovary disorder and abnormal uterine bleeding had endometrial hyperplasia and 5 had carcinoma in situ.

Table 2: Histological Risk Progression in PCOD-Related Endometrial Pathology

Condition	Relative Risk	Clinical Concern
Chronic anovulation	2–3×	Baseline proliferative stimulus
Simple hyperplasia	3–5×	Reversible, needs surveillance
Atypical hyperplasia	10–15×	Pre-malignant, high-risk
Endometrioid adenocarcinoma	4–6× (PCOD)	Estrogen-dependent malignancy

4.3 Clinical Predictors and Symptoms

Symptoms that often show a problem in the endometrium in PCOD patients are irregular menstruation, long periods without a menstrual cycle, bleeding outside of the menstrual cycle and bleeding after menopause (Chittenden et al., 2009). On the other hand, these patients may be asymptomatic for quite a while, so screening should be done regularly in people at risk for PCOD.

Early detection can be achieved by using serum CA-125, transvaginal ultrasound (TVUS) for the endometrium and diagnostic hysteroscopy (Trimble et al., 2012). Furthermore, having comorbidities such as diabetes and obesity is seen as worsening the prognosis in endometrial cancer among PCOD patients (Lauretta et al., 2016).

5. Metabolic and Reproductive Consequences of PCOD

Along with affecting reproductive organs, Polycystic Ovarian Disease can cause changes in how the body's metabolism functions. If a woman has PCOD, she is likely to develop both metabolic syndrome and subfertility due to her insulin resistance and hormonal problems (Moran et al., 2010; Dunaif, 2006).

5.1 Insulin Resistance and Type 2 Diabetes Mellitus

Around 70% of PCOD women often experience insulin resistance before developing type 2 diabetes mellitus (Legro et al., 2005). Even when PCOD is mild, impairments in how the insulin receptor works and sends out signals suggest that the challenge exists at the start (Ehrmann et al., 2006).

If there is too much insulin in the blood, androgen levels rise in theca cells and production of SHBG in the liver is reduced. As a result, more testosterone circulates in the body which leads to hirsutism and acne (Baillargeon & Nestler, 2006). Furthermore, having too much insulin in the body can cause fatty liver disease and unhealthy cholesterol levels.

5.2 Obesity and Metabolic Syndrome

PCOD patients usually have central obesity. Obesity in women with PCOD intensifies problems with fertility and blood sugar, according to Pasquali (2006). Being abdominally obese aggravates insulin resistance and encourages the release of cytokines and adipokines which increases inflammation (Carmina & Lobo, 2004).

Table 3: Diagnostic Criteria of Metabolic Syndrome (NCEP ATP III) and Prevalence in PCOD

Component	Cut-Off Value	Prevalence in PCOD (%)
Waist Circumference	>88 cm (women)	62–80%
Triglycerides	≥ 150 mg/dL	45–65%
HDL-C	<50 mg/dL	55–70%
Fasting Blood Glucose	≥ 100 mg/dL	30–50%
Blood Pressure	$\geq 130/85$ mmHg	40–60%

(Source: Azziz et al., 2016; Wild et al., 2010)

5.3 Ovulatory Dysfunction and Infertility

Abnormal ovulation is a main issue in PCOD and is responsible for most cases of women's anovulatory infertility (Teede et al., 2018). A problem in the hypothalamic-pituitary-ovarian axis results in the inability for only one follicle to be chosen and mature. Too much LH and low FSH stop ovulation from occurring, causing troubles with menstruation as well as subfertility (Franks, 2008).

Too much exposed estrogen delays normal endometrial changes, making it more difficult for the embryo to attach in assisted reproductive technology.

6. Preventive Strategies and Risk Mitigation in PCOD

Because the effects of Polycystic Ovarian Disease (PCOD) last a lifetime, it should be treated early by controlling symptoms and reducing future risks. Connecting a healthy lifestyle, medication when needed and specialized tests is what's recommended for minimizing risks in metabolism, the heart and cancer (Teede et al., 2018).

6.1 Lifestyle Modifications

Overweight or obese women with PCOD are mainly managed with lifestyle changes. Being at least 5–10% lighter can greatly improve insulin sensitivity, reproductive health and hormone production (Moran et al., 2011). Routine physical activity boosts glucose handling and making dietary choices that reduce the GI index, add omega-3s and cut down on trans fats is helpful in managing metabolic syndrome (Thomson et al., 2012). Long-term adherence and mental well-being are helped by behavioural counselling since sufferers of PCOD are more likely to have anxiety and depression, says Dokras et al.

6.2 Pharmacological Interventions

Many doctors use metformin, an insulin sensitizer, to lower blood sugar, help with weight shed and bring back ovulation (Lord et al., 2003). Lowering serum androgen levels and the risk of endometrial hyperplasia are possible because stabilization of insulin helps to do this. Combined oral contraceptives are often prescribed to manage menstrual cycles and lessen symptoms of high androgens, but their use should be watched over the long term for heart problems (Legro et al., 2013).

Women who have several cardiovascular risk factors may be advised to take statins when dyslipidaemia becomes severe (Moll et al., 2011). Treating obesity and related health concerns in difficult cases has been shown to be helped by orlistat.

6.3 Screening and Monitoring

Screening tests often can reduce your chances of developing serious issues in the future. All patients with PCOD should have their glucose, lipid, blood pressure and BMI checked once a year (Teede et al., 2018). In women whose periods are not regular, once a year TVUS and endometrial biopsy are advised to find hyperplastic changes at an early stage (Chittenden et al., 2009).

Women wishing to be pregnant need to be checked for gestational diabetes and offered ovulation assisting drugs when needed (Palomba et al., 2015).

Table 4: Evidence-Based Preventive Strategies for PCOD Management

Domain	Strategy	Benefit
Diet & Exercise	Calorie deficit, aerobic training	Improves insulin sensitivity, reduces weight
Medication	Metformin, COCs, statins	Targets insulin resistance & hormonal balance
Monitoring	FBS, lipid panel, endometrial biopsy	Detects early metabolic and oncological changes
Reproductive Planning	Ovulation induction, counselling	Improves fertility and pregnancy outcomes

7. Conclusion and Future Directions

Even though Polycystic Ovarian Disease (PCOD) was once only linked to reproductive disorders, we now know it affects many body systems and lasts a lifetime. In reviewing PCOD, it became clear that neuroendocrine problems, insulin resistance, constant inflammation and being overweight all play a role in causing problems for several systems. This condition can reduce reproductive health, cause metabolic syndrome, worse cardiovascular problems and boost the risk of endometrial hyperplasia and cancer.

PCOD causes hormonal and metabolic problems that increase a woman's chances of hypertension, dyslipidaemia, endothelial dysfunction and subclinical atherosclerosis in the long term. They happen early in life, but most are not found until people experience serious symptoms. In a similar way, problems such as endometrial cancer come from chronic anovulation and extra oestrogen in the body which are often made worse by obesity and insulin resistance. All these associated dangers change PCOD from just a gynaecological problem to a serious health risk for the whole body.

PCOD also impacts a person's quality of life as it can cause subfertility, strange ovulation and problems with periods. The problems these users deal with can affect both their emotions and thoughts, underlining the value of combining approaches to treatment. Fortunately, people can reduce their risks with lifestyle interventions, drugs when needed and careful checks for symptoms. Patients are managed mainly with weight loss, exercise, balanced nutrition and medications that help insulin uptake and careful monitoring is important to prevent ongoing health problems.

Despite many studies, we are not fully aware of the genetic causes, environmental factors and future course of PCOD. Future studies need to focus on personalised medicine that changes treatment depending on hormones, metabolic markers and genetics. For best results, it is essential for endocrinologists, gynaecologists, cardiologists and oncologists to cooperate. Moreover, the results of early interventions should be closely watched over time, using big-scale studies.

In essence, PCOS means doctors have to rethink their approach, moving from quick treatments for fertility to ongoing care for a full-body health concern. Educating people, creating helpful policies and providing everyone with simple access to healthcare reduce the harm this disease causes globally.

CONFLICT OF INTERESTS

The authors declare that they have no conflict of interest.

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