



## **A Review Study on PCOS and its Related Health Complications.**

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### **ABSTRACT:**

The prevention, detection, diagnosis, and treatment of illnesses specific to women are all aspects of women's health. The most common endocrinopathy in women of reproductive age is probably polycystic ovarian syndrome (PCOS), which is exceedingly common. Primary care physicians frequently are not aware of the severe morbidity associated with the syndrome, both in terms of reproductive and nonreproductive events. A woman's quality of life during her reproductive years may be greatly impacted by the condition, which also raises her risk of morbidity and mortality by the menopause. (Dahlgren 1992)

**Keywords:** *Menopause, PCOS, Reproductive health, contraceptives, Diet and Cardiovascular consequence*

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### **Introduction:**

There isn't a single definition of PCOS, largely due to how varied and heterogeneous it is. However, it is obvious to us that the condition is an endocrinopathy and that it is better to refer to it as PCOS, a syndrome, as opposed to a disease (Lobo 1995). It was generally agreed at a meeting held at the National Institutes of Health ten years ago that hyperandrogenism and chronic anovulation are the main features of the syndrome and that PCOS can be assumed to be the cause once other conditions (such as CAH and tumours) have been ruled out. This broad definition is referred to in the literature as the "NIH Consensus Statement." In fact, there was no consensus at this conference, nor was there intended to be one. In order to emphasise the fact that the diagnosis poses serious health risks for women, we will refer to PCOS in the comments using this most frequently accepted description. If the diagnosis is based on hyperandrogenism and anovulation, PCOS is very common and is thought to affect 5-7% of women of reproductive age (Nestler 1998) & (Nestler et al., 1998). The syndrome's range is still much broader, though. We have lately come to believe that there is a mild variant of PCOS that affects women who have polycystic ovaries and hyperandrogenism but whose ovulatory function is still intact (Carmina & Lobo 1999). But it's obvious that the disease is less severe and the hyperandrogenism is less apparent.

While at least 5% of people with PCOS, 16–25% of people with normal ovaries have the isolated finding of polycystic-appearing ovaries (PAO), which satisfies the traditional ultrasonographic criteria (Polson et al., 1986). It is known that PAO or PCO, which exclusively refers to ovarian morphology, can occur in CAH and hypothalamic amenorrhea, where its prevalence is over 100% (Abdel et al., 1992).

Although many doctors have made the diagnosis of PCOS based on ultrasound results, normal ovulatory women with PAO cannot be thought to have the condition. In spite of this, it is puzzling because a considerably lesser proportion of women have PCOS despite the high frequency of PAO in the general population. A theory linking PCOS with polycystic ovary syndrome (PAO/PCO) has been developed. It is well recognised that PAOs can develop in childhood before any hormonal changes associated with puberty, and they most likely result from genetic and/or environmental factors. For women with PAO to develop PCOS, we have suggested that a number of "insults" must enter the picture after puberty (Lobo 1995).

It is common for multiple factors to be at play, and the list of these "insults" is extensive (for example, insulin resistance, obesity, stress, and dopaminergic dysregulation). Various personal adaptive or compensatory mechanisms are likely working in opposition to these insults at the same time, either to lessen the manifestation of PCOS or to stop its growth completely. Therefore, despite having PAO, these adaptive characteristics may allow a woman to never acquire PCOS or to delay the onset of some form of the illness during the reproductive years. According to our theory, whereas PCOS is identified by distinctive results, a larger subset of women with PAO are also predisposed to developing the illness. The higher morbidity that is described below may therefore also affect these women. We have also demonstrated some other health complications related to PCOS in female.

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### **Reproductive Concerns**

Anovulation occurs in most PCOS-afflicted females. Infertility and issues with dysfunctional bleeding follow this. Chronically high levels of unopposed oestrogen cause endometrial hyperplasia and may even cause cancer, as will be covered later. Anaemia is frequently caused by continuous heavy bleeding.

Anovulation-related infertility may be successfully treated, although it may not be a straightforward process. The cornerstone of treatment is clomiphene citrate. Clomiphene resistance, which can affect up to 10% of women, prevents them from responding to doses as high as 150 mg per day for five days.

Lack of a sufficient ovarian response is the main cause of this resistance. Ovarian diathermy, pulsatile GnRH, and gonadotropins are clomiphene substitutes. While they are all effective, they all come at a higher cost and frequently have a higher likelihood of problems. All individuals with polycystic ovaries (PAO or PCOS) are more prone to the condition of hyperstimulation, which can result after gonadotropin medication. Additionally, multiple pregnancies are more common. Pulsatile GnRH therapy is not, in our opinion, as effective as the other treatments. In spite of the fact that ovarian diathermy necessitates surgery and may result in pelvic and ovarian adhesions, it has a 50% success rate in conceiving children. With the use of metformin and insulin sensitising drugs like troglitazone, promising results have recently been shown. We don't yet have data on long-term outcomes.

Pregnancy loss is arguably the most upsetting issue in terms of reproduction for PCOS patients (Sagle et al., 1988). Approximately one-third of all pregnancies in PCOS result in spontaneous abortions. The recognised early abortion rate among normal women (12–15%) is at least twice as high as this.

Uncertain causes have been proposed, but they include malformed embryos from atretic oocytes, high LH levels, insufficient progesterone secretion, and an aberrant endometrium. Retrospective investigations have shown that attempts to increase the live birth rate by reducing LH with GnRH agonist therapy were successful (Homburg et al., 1993), but a prospective research has not supported this claim (Clifford et al., 1996).

As soon as a pregnancy is confirmed, the morbidity rises, especially if the woman is obese. Preeclampsia, diabetes, early labour, and a higher stillbirth rate are all on the rise, as are perinatal mortality, which is raised by at least 1.5 times. The likelihood of having a large infant delivered is higher as a result of these complications and the increased C-section rate. It is not surprising that the prevalence of gestational diabetes rises because the majority of individuals with PCOS have insulin resistance, as shown by our own investigations in Los Angeles and New York.

This has not, however, been a general observation, which may be accounted for by the disorder's variability and endogenous compensating elements. Recent research has shown that PCOS individuals who develop gestational diabetes exhibit changes in insulin sensitivity as early as the first trimester (Paradisi et al., 1998).

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## Psychological Issues

According to numerous studies, reactive depression and other minor psychological abnormalities are more common in women with PCOS, especially those who also have hirsutism (Barth et al., 1993). Additionally, there is proof that psychological stress is getting worse and that stress-induced catabolism is getting worse. Women who are hirsute have a lower quality of life overall (Sonino et al. 1993). So one must take the psychological effects of the condition into account when looking at the morbidities connected to PCOS.

Menstrual abnormalities and hirsutism are especially upsetting in younger patients because they have a detrimental effect on their psychosocial development.

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## Obesity

44% of women with PCOS are obese overall. Depending on geography and ethnicity, this number varies substantially. Obesity, when present, exacerbates the clinical symptoms of PCOS by causing insulin resistance, which raises ovarian and adrenal androgens as well as unbound testosterone. Therefore, one of the primary objectives of any PCOS therapy is to manage obesity, even if this may be more challenging due to insulin resistance and decreased lipolysis (Ingvar et al. 1997).

The obesity associated with PCOS is of the android (central) type, which causes a higher waist-to-hip ratio and is strongly linked to diabetes mellitus and an elevated risk of cardiovascular disease. This is because to hyperandrogenism and insulin resistance. Obesity makes these effects of PCOS worse, although they appear to affect all PCOS patients, including those who are not obese.

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## Impaired glucose tolerance and diabetes

In the longitudinal study that was conducted after wedge resections (1), type 2 diabetes mellitus had manifested in 16% of PCOS-afflicted women by the time they reached menopause. Most PCOS patients have insulin resistance, especially if more sensitive probes are employed. As mentioned above, insulin resistance is more severe in obese patients. Therefore, decreased glucose tolerance and overt type 2 diabetes are risks that are shared by all PCOS-positive women. In a recent study, it was discovered that 7.5% of women of reproductive age had PCOS and 31% had poor glucose tolerance.

These numbers were 10.3% and 1.5% in nonobese PCOS, which is about three times the usual population rate (Legro et al., 1999). These findings held true for women of various races.

Diabetes's morbidity is well established. Therefore, diabetes screenings and regular monitoring are necessary for even young women with PCOS. This assumes much greater significance in women who want to become pregnant and need to be a key component of preconception counselling.

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**Cardiovascular consequence:** dyslipidemia, hypertension, coronary disease

PCOS patients may have a range of aberrant lipid and lipoprotein profiles, as has long been known (Wild & Bartholomew 1988). Patients typically have excessive cholesterol, triglycerides, and LDL cholesterol as well as low levels of high density lipoprotein and Apo A1. These results, however, are extremely variable and rely on the ethnicity, diet, and obesity status of the group under study. While hyperandrogenism is likely to contribute in some way to these anomalies, hyperinsulinemia (insulin resistance) seems to be the main factor, especially given the increase in triglycerides.

It is well recognised that these anomalies are very prognostic of cardiovascular disease. Particularly in older women with PCOS and those who are fat, hypertension is very common. Again, there is a strong correlation between this anomaly and insulin resistance.

According to the risk profile, PCOS-positive women had a 7-fold higher risk of myocardial infarction (Dahlgren et al., 1992). Women with PCOS are more likely to have coronary disease (Talbot et al., 1995) & (Birdsall et al., 1997) & (Conway et al., 1992) majority of the metabolic and other abnormalities mentioned above are probably responsible for some of this risk. The prevention of cardiovascular disease in women with PCOS should be a top public health priority due to the high incidence of PCOS in the general population and the fact that cardiovascular disease is the leading cause of mortality in older women.

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**Cancer risk**

Endometrial cancer risk is higher in women with PCOS. The most important risk factor is probably long-term, unopposed oestrogen exposure. Obesity, hypertension, and diabetes, which are known risk factors for endometrial cancer, could complicate this. All PCOS-positive women should be screened, even if they are thought to be too young to experience endometrial hyperplasia or cancer.

Women with PCOS also have an elevated risk of ovarian cancer by 2- to 3-fold (Schildkraut et al., 1996). It's interesting to note that this risk is higher in non-obese individuals and is highest in females who have not used oral contraceptives. Use of oral contraceptives should be strongly evaluated as a prophylactic therapy due to the established protective effect of these drugs on ovarian and endometrial cancer risk.

It is uncertain whether breast cancer risk is higher for women with PCOS, in part because additional confounding variables including obesity and nulliparity exist. Given the possibility of a link between PCOS and breast cancer, it is crucial to monitor all women with PCOS for breast disease throughout follow-up care.

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**Women with PAO**

As was previously mentioned, 16–25% of women who ovulate normally have polycystic appearing ovaries (PAO) without any signs of the full-blown condition. However, up to 30% of women with PAO may also have mild abnormalities similar to PCOS (Carmina et al., 1997). Among these traits are androgenic ovarian responses to gonadotropin stimulation, as well as metabolic modifications such decreased high density lipoprotein-C levels and signs of insulin resistance. These findings show that significant yet undetected abnormalities may exist in otherwise healthy women who have a PCOS feature (specifically PAO), even though the data produced by our team still require further evaluation.

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**Conclusions**

According to this article, PCOS is a common illness that affects women and is linked to severe reproductive and nonreproductive morbidity. For the health of women, perceptions about this and preventative treatments are crucial. Diet, exercise, and oral contraceptives are logical preventative treatments for PCOS. The mainstay of care for women with PCOS should be screening for hypertension, abnormal lipid profiles, insulin resistance, and reproductive problems, including malignancy.

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**References**

- Abdel Gadir A, Khatim MS, Muwati RS, et al. 1992 Implications of ultrasonically diagnosed polycystic ovaries. 1. Correlations with basal hormonal profiles. *Hum Reprod.* 4:453–457.
- Barth JH, Catalan J, Cherry CA, Day A. 1993 Psychological morbidity in women referred for treatment of hirsutism. *J Psychol Res.* 37:615–619.
- Birdsall MA, Farquahar CM, White HD. 1997 Association between polycystic ovaries and extent of coronary artery disease in young women having cardiac catheterization. *Ann Intern Med.* 126:32–35.
- Carmina E, Lobo RA. 1999 Do hyperandrogenic women with normal menses have PCOS? *Fertil Steril.* 71:319–322.
- Carmina E, Wong L, Chang L, et al. 1997 Endocrine abnormalities in ovulatory women with polycystic ovaries on ultrasound. *Hum Reprod.* 12:905–909.
- Clifford K, Rai R, Watson H, et al. 1996 Does suppressing luteinizing hormone secretion reduce the miscarriage rate? Results of a randomized controlled trial. *Br Med J.* 312:1508–1511.

- Conway GS, Agrawal R, Betteridge DJ, et al. 1992 Risk factors for coronary artery disease in lean and obese women with polycystic ovary syndrome. *Clin Endocrinol (Oxf)*. 37:119–125.
- Dahlgren E, Janson PO, Johansson S, et al. 1992 Polycystic ovary syndrome and risk for myocardial infarction. Evaluated from a risk factor model based on a prospective population study of women. *Acta Obstet Gynecol Scand*. 71:599–603.
- Dahlgren E. 1992 Women with polycystic ovary syndrome wedge resected in 1956 to 1965: a long-term follow up. *Fertil Steril*. 57:505–513.
- Homburg R, Berkowitz D, Levy T, et al. 1993 In vitro fertilization and embryo transfer for the treatment of infertility associated with polycystic ovary syndrome. *Fertil Steril*. 60:858–863.
- Ingvar EK, Arner P, Bergqvist A, et al. 1997 Impaired adipocyte lipolysis in nonobese women with the polycystic ovary syndrome: a possible link to insulin resistance. *J Clin Endocrinol Metab*. 82:1147–1153.
- Legro RS, Kusanman AR, Dodson VC, et al. 1999 Prevalence and predictions of the risk for type 2 diabetes mellitus and impaired glucose tolerance in polycystic ovary syndrome: a prospective, controlled study in 254 affected women. *J Clin Endocrinol Metab*. 84:165–174.
- Lobo RA. 1995 A disorder without identity “HCA,” “PCO,” “PCOD,” “PCOS,” “SLS.” What are we to call it? *Fertil Steril*. 63:1158–1160.
- Lobo RA. 1995 A unifying concept for polycystic ovary syndrome. In: Chang RJ, ed. *Polycystic ovary syndrome*. New York: Serono Symposia USA, Springer-Verlag; 334–352.
- Nestler JE. 1998 ES, Key TJ, Kahsar-Miller M, et al. 1998 Prevalence of the polycystic ovary syndrome in unselected black and white women of the southeastern United States: a prospective study. *J Clin Endocrinol Metab*. 83:3078–3082.
- Nestler JE. 1998 Polycystic ovary syndrome: a disorder for the generalist. *Fertil Steril*. 70:811–812.
- Paradisi G, Fulghesu AM, Ferrazzani S, et al. 1998 Endocrino-metabolic features in women with polycystic ovary syndrome during pregnancy. *Hum Reprod*. 13:542–546.
- Polson DW, Wadsworth J, Adams J, et al. 1986 Polycystic ovaries: a common finding in normal women. *Lancet*. 1:870–872.
- Sagle M, Bishop K, Ridley N. 1988 Recurrent early miscarriage and polycystic ovaries. *Br Med J*. 297:1027–1028.
- Schildkraut JM, Schwingl PJ, Bastos E, et al. 1996 Epithelial ovarian cancer risk among women with polycystic ovary syndrome. *Obstet Gynecol*. 88:554–559.
- Sonino N, Fava GA, Mani E, et al. 1993 Quality of life of hirsute women. *Postgrad Med J*. 69:186–189.
- Talbott E, Guzick D, Clerici A, et al. 1995 Coronary heart disease risk factors in women with polycystic ovary syndrome. *Arterioscler Thromb Vasc Biol*. 15:821–826.
- Wild RA, Bartholomew MJ. 1988 The influence of body weight on lipoprotein lipids in patients with polycystic ovary syndrome. *Am J Obstet Gynecol*. 159:423–427.