

Manual of **Clinical Dilemma** in **Mature Women**

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Chapter 3

How Ominous is Polycystic Ovary Syndrome at Menopause?

Kirubamani NH

It is not possible to diagnose a woman with polycystic ovary syndrome (PCOS) when she has already reached menopause because the cardinal features disappear. While the research on menopausal/PCOS mechanisms is sparse, we do know that because PCOS affects many of the body's systems, the responses of each of these systems to aging will vary and they will also vary according to the individual. PCOS is a lifelong disease. It is a challenge to distinguish menopausal changes from abnormalities connected with PCOS. The expression of PCOS may begin early and the symptoms change across the lifespan. Determining the appearance and expression of the syndrome at each stage of life will be important to expand the diagnosis and treatment of PCOS.

Polycystic ovary syndrome in postmenopausal women is not well-defined as there is no robust criteria. Regarding PCOS at menopause a few questions that cross our mind are:

- Does PCOS get worse and if so, how worse?
- Does PCOS get better after menopause?
- Could PCOS simply disappear altogether?

Criteria for the Metabolic Syndrome according to the National Cholesterol Education Program (NCEP), Adult Treatment Panel III

Three or more of the following:

- Elevated waist circumference—greater than 35 inches (88 cm)
- Elevated triglycerides—equal to or greater than 150 mg/dL
- High-density lipoprotein (HDL)—less than 50 mg/dL
- Elevated blood pressure—equal to or greater than 130/85 mm Hg
- Elevated fasting glucose—equal to or greater than 100 mg/dL.

Hyperandrogenism seen in PCOS women persists after the menopausal transition. Similarly, PCOS women continue to manifest the metabolic

alterations such as insulin resistance after menopause, which makes them more susceptible to type 2 diabetes mellitus.

Older women with PCOS still had lower levels of sex hormone binding globulin (SHBG). Postmenopausal women with PCOS are exposed to higher adrenal and ovarian androgen levels than non-PCOS women. The metabolic features are primarily related to underlying insulin resistance (IR), which is now understood to play an important role in both the pathogenesis and long-term sequelae of PCOS. The data published by Puurunen et al. revealed that enhanced ovarian androgen secretion along with unfavorable metabolic changes such as impaired glucose tolerance, chronic inflammation which were observed in a group of premenopausal women with PCOS, persist after the menopausal transition. The prevalence of hypertension was higher in postmenopausal women with PCOS compared with controls studied longitudinally, and triglyceride levels increased in both groups but remained higher in the women with PCOS. Livadas S. et al found that insulin resistance was positively associated with body mass index (BMI) and androgens level. Lean females with PCOS may present a better metabolic profile with time and PCOS may even remain as a silent disorder. It is possible that the metabolic abnormalities in women with PCOS worsen with age, because all women experience increasing insulin resistance and abdominal adiposity along with chronic inflammation and dyslipidemia with age and a specific increase in LDL across the menopausal transition. These events are partially related to persisting hyperandrogenism but are mostly correlated with excessive body weight (mainly visceral obesity). This suggests that our best long-term strategy is to ensure that women with PCOS are informed about their high-risk for metabolic and cardiovascular diseases. Women who remained anovulatory, with aging had increases in total cholesterol, low-density lipoprotein (LDL), HDL levels reduced and cardiovascular risk remained significantly higher than in the general population according to Carmina E, Campagna A, et al. Although there was an increased prevalence of type 2 diabetes in women with PCOS, compared with controls at a younger age; the prevalence of type 2 diabetes increased with age in controls, and there was no difference in the prevalence of diabetes 20 years later when women with PCOS had reached menopause. There was also no difference in fasting insulin levels, homeostatic model assessment (HOMA) of insulin resistance, and glucose levels in the two groups at an older age according to Schmidt J, Brännström M, et al. The suggestion of various studies is that "if women with PCOS do not become obese they may exhibit a better metabolic profile. The long-term risk for morbidity and mortality among postmenopausal women with a history of PCOS is uncertain and there is no established phenotype for PCOS after menopause. Long-term, multicenter cohort studies are needed where the following issues should be assessed: menopausal phenotype, metabolic syndrome, cardiovascular events, and other causes of morbidity/mortality. Small numbers of studies during menopause, point to the need for additional longitudinal studies to expand the current knowledge. The ongoing studies will provide a thorough understanding of the PCOS lifecycle, to help with diagnosis and treatment that is no longer limited to

the reproductive-age patient and should extend to menopausal women. PCOS must be managed throughout a woman's life and dietary and lifestyle changes can improve their health and lower their risk of chronic disease.

Polycystic ovary syndrome at menopause will be "ominous" when the woman increases her abdominal circumferences and BMI.

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