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Review Article

POLYENDOCRINE METABOLIC OVARIAN SYNDROME (PMOS): A NARRATIVE REVIEW OF CONTEMPORARY CONCEPTS AND AYURVEDIC PERSPECTIVES

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ABSTRACT

Polyendocrine metabolic ovarian syndrome (PMOS) previously named Polycystic Ovary Syndrome (PCOS) is a heterogeneous endocrine–metabolic disorder affecting women of reproductive age. It is characterized by hyperandrogenism, ovulatory dysfunction, and/or polycystic ovarian morphology, and is associated with infertility, obesity, insulin resistance, dyslipidemia, psychological morbidity, and long-term reproductive and cardiometabolic complications. Objective of this article is to review contemporary biomedical understanding of PMOS and explore its possible interpretation within the Ayurvedic framework. PCOS/ PMOS is best understood as a multisystem disorder involving reproductive, metabolic, neuroendocrine, inflammatory, dermatological, and psychological pathways. Key pathophysiological mechanisms include insulin resistance, hyperandrogenism, altered gonadotropin secretion, adipose-tissue dysfunction, chronic low-grade inflammation, and oxidative stress. Management is individualized and includes lifestyle modification, pharmacological therapy for menstrual and hyperandrogenic symptoms, metabolic-risk reduction, and fertility-directed treatment. In Ayurveda, PCOS may be interpreted through overlapping features of Nashtartava, *Artavakshaya, Vandhyatva, Sthoulya*, and *Prameha*, with involvement of *Kapha–Vata dosha*, impairment of *Agni*, *Artavavaha Srotas*, and *Rasa, Rakta, Mamsa, Medo, Artava Dhatus*. PMOS requires long-term, individualized, and multidisciplinary care. Ayurvedic concepts provide a useful interpretive framework for understanding diet, lifestyle, metabolic disturbance, and reproductive dysfunction and advice managements along with lifestyle and diet. However, direct equivalence between PMOS and any single classical diagnosis should be avoided. Standardized Ayurvedic interventions supported by rigorous clinical research are needed to define their role in integrative PMOS management.

INTRODUCTION

Polyendocrine metabolic ovarian syndrome (PMOS) is one of the most common endocrine disorders in women of reproductive age, with a reported global prevalence of 5–20% depending on diagnostic criteria and population studied.[1] the previous term PCOS was misleading and inaccurate because it implied the presence of pathological ovarian cyst, obscured its diverse endocrine metabolic symptoms and reinforced stigma.[2]

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PMOS is a heterogeneous endocrine and metabolic disorder characterized by variable combinations of clinical or biochemical hyperandrogenism, insulin resistance, ovulatory dysfunction, and polycystic ovarian morphology. It represents one of the leading causes of anovulatory infertility. Beyond its reproductive manifestations, PMOS is associated with a broad spectrum of metabolic and psychological comorbidities, including obesity, dyslipidemia, impaired glucose tolerance, type 2 diabetes mellitus, obstructive sleep apnea, depression, anxiety, and reduced quality of life. Furthermore, women with PMOS may be at increased risk of long-term complications, including gestational diabetes mellitus, hypertensive disorders of pregnancy, and

endometrial hyperplasia, with a possible association with an elevated risk of cardiovascular disease.[2,3]

PCOS historically considered primarily an ovarian disorder, is now recognized as a complex, multisystem condition involving genetic susceptibility, neuroendocrine dysregulation, adipose-tissue dysfunction, chronic low-grade inflammation, and oxidative stress. Effective management therefore requires an individualized, multidisciplinary approach addressing reproductive, metabolic, dermatological, and psychological dimensions.

In Ayurveda, no single disease entity corresponds exactly to PMOS. However, its clinical features appear close to descriptions of *Nashtartava*, *Artavakshaya*, *Vandhyatva*, *Sthoulya*, and *Prameha*. This narrative review synthesizes contemporary biomedical understanding of PMOS and explores its possible Ayurvedic interpretation, with a view toward integrative management.

MATERIALS AND METHODS

A narrative literature review was conducted using electronic databases including PubMed, Scopus, and Google Scholar, along with classical Ayurvedic texts and commentaries, published guidelines, systematic reviews, narrative reviews, clinical trials, observational studies, and relevant classical Ayurvedic sources were considered. Contemporary biomedical evidence and classical Ayurvedic descriptions were analyzed separately and subsequently integrated through a conceptual interpretive framework. As this was a narrative review, no formal meta-analysis was performed.

Epidemiology and Clinical Definition

PCOS/PMOS affects approximately 5–20% of women of reproductive age worldwide, with variation largely attributable to differences in diagnostic criteria and population characteristics. Metabolic disturbances such as insulin resistance and obesity are reported in 60–80% of women with PMOS, contributing to both reproductive and long-term health risks.[4]

Clinically, PCOS/PMOS presents with menstrual irregularities (oligomenorrhea, amenorrhea, or abnormal uterine bleeding), signs of hyperandrogenism (hirsutism, acne, androgenic alopecia), and/or polycystic ovarian morphology on ultrasound. Psychological distress, including depression and anxiety, is common and often related to visible symptoms, infertility concerns, and fear of long-term complications.

Evolution of Diagnostic Criteria

The condition now recognized as PMOS was first described by Stein and Leventhal in 1935 in women presenting with hirsutism, amenorrhea, obesity, and enlarged polycystic ovaries. In 2003, the Rotterdam consensus expanded the definition, allowing diagnosis when at least two of the following three features are present: hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology.

Current Diagnostic Approach

The 2023 International Evidence-based Guideline retains a Rotterdam-based approach for adults. PMOS is diagnosed when at least two of the following are present after exclusion of relevant differential diagnoses: clinical or biochemical hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology.

When irregular menstrual cycles and hyperandrogenism coexist in an adult, neither ultrasound nor AMH testing is required to establish the diagnosis. When only one of these features is present, either pelvic ultrasound or AMH may contribute to the diagnostic algorithm; AMH should not be used as a stand-alone test or combined indiscriminately with ultrasound to increase diagnostic certainty.

In adolescents, both hyperandrogenism and ovulatory dysfunction are required for diagnosis after exclusion of mimicking disorders. Ultrasound and AMH are not recommended for diagnosis in adolescence because multifollicular ovaries and changing AMH concentrations have poor specificity during this developmental stage.

The clinical assessment should exclude thyroid disease, hyperprolactinaemia, non-classic congenital adrenal hyperplasia, Cushing syndrome, and androgen-secreting neoplasms where clinically indicated. A high LH:FSH ratio is neither necessary nor sufficiently accurate for PCOS diagnosis.

Pathophysiology

Genetic Factors

PCOS/PMOS is a multifactorial, polygenic condition with strong heritability, similar to type 2 diabetes and other complex metabolic disorders. Genome-wide association studies have identified multiple susceptibility loci involved in hormonal signaling, metabolic pathways, and follicular development. These findings support shared pathophysiological mechanisms between PMOS, obesity, insulin resistance, and cardiovascular disease. Genome-wide association studies (GWAS) have implicated different genes and proteins in the development of PCOS/PMOS. The majority of these genes encode proteins involved in insulin signaling, sex hormone function, and regulation of metabolism, processes associated with PCOS/PMOS.[5]

Insulin Resistance and Hyperinsulinemia

Insulin resistance is a central feature of PCOS/PMOS, present in a majority of affected women, including a substantial proportion of lean individuals. Compensatory hyperinsulinemia stimulates ovarian

theca-cell androgen production, reduces hepatic sex hormone–binding globulin (SHBG) synthesis, and exacerbates hyperandrogenism. Insulin also interacts with luteinizing hormone (LH) and insulin-like growth factor-1 (IGF-1) pathways, further promoting androgen excess and follicular arrest.

Hyperinsulinemia and insulin resistance contribute to oxidative stress, advanced glycation end-product accumulation, adipokine imbalance (reduced adiponectin, increased leptin), and mitochondrial dysfunction in granulosa cells, impairing oocyte quality and ovulation.[6]

Hyperandrogenism

Hyperandrogenism is a defining feature of PCOS, arising from ovarian theca cells and, to a lesser extent, adrenal sources. Dysregulation of enzymes such as P450c17 in theca cells leads to excess androgen synthesis, driven by LH, insulin, and IGF-1. Elevated androgens contribute to hirsutism, acne, alopecia, anovulation, and metabolic disturbances.[7]

Hypothalamo–Pituitary–Ovarian Axis Dysfunction

Altered gonadotropin-releasing hormone (GnRH) pulse frequency in PCOS preferentially stimulates LH secretion over follicle-stimulating hormone (FSH), resulting in an elevated LH:FSH ratio in many patients. Increased LH promotes theca-cell hypertrophy and androgen production, while relatively low FSH impairs follicular maturation and aromatization of androgens to estrogens, contributing to follicular arrest and anovulation.[8]

Obesity and Adipose-Tissue Dysfunction

Approximately 50–70% of women with PCOS are overweight or obese, with visceral adiposity playing a key role in metabolic dysfunction. Adipose tissue functions as an active endocrine and immune organ, releasing adipokines, cytokines (e.g., IL-6, TNF-α), and free fatty acids that worsen insulin resistance, inflammation, and androgen excess.[9]

Oxidative Stress and Inflammation

PMOS is associated with increased oxidative stress and chronic low-grade inflammation, characterized by elevated reactive oxygen species, reduced antioxidant defenses, and activation of inflammatory signaling pathways such as NF-κB and JNK. These processes damage granulosa cells, impair mitochondrial function, reduce estrogen production, and contribute to anovulation and follicular atresia.

Chronic inflammation may arise from the relatively lower levels of progesterone due to anovulation, as progesterone exhibits anti-inflammatory properties.[10]

Clinical Manifestations and Complications

Menstrual Irregularities and Ovulatory Dysfunction

Ovulatory dysfunction in PCOS/PMOS commonly presents as oligomenorrhea, amenorrhea, or abnormal uterine bleeding, and is a major cause of infertility. Even women reporting regular cycles may experience anovulation; therefore, confirmation of ovulation via mid-luteal serum progesterone may be necessary in selected cases.

Hyperandrogenism

Clinical hyperandrogenism manifests as hirsutism (present in 65–75% of women with PMOS), acne, and androgenic alopecia. Biochemical hyperandrogenism is assessed using total and free testosterone, free androgen index, and other androgen measurements after exclusion of other causes.[11]

Metabolic Complications

Women with PCOS/PMOS have an increased prevalence of insulin resistance, impaired glucose tolerance, type 2 diabetes, dyslipidemia (elevated triglycerides, reduced HDL cholesterol), and metabolic syndrome. Screening recommendations include measurement of body mass index, waist circumference, blood pressure, fasting lipid profile, and oral glucose tolerance testing at diagnosis and at regular intervals thereafter.

Reproductive and Pregnancy Outcomes

PMOS is associated with reduced clinical pregnancy rates, increased miscarriage risk, and higher rates of gestational diabetes, hypertensive disorders, preterm birth, and cesarean delivery. Preconception weight optimization and lifestyle modification improve reproductive outcomes, particularly in overweight and obese women.

Psychological Morbidity

Depression, anxiety, reduced quality of life, negative body image, and psychosexual dysfunction are more common in women with PCOS, independent of body mass index. Routine screening and appropriate psychological support are recommended as part of comprehensive care.

Other Comorbidities

PCOS/PMOS is associated with increased risk of obstructive sleep apnea, metabolic dysfunction–associated steatotic liver disease (MASLD), and possibly cardiovascular disease over the long term. Endometrial hyperplasia and cancer risk are increased due to chronic unopposed estrogen exposure from anovulation.[12]

Contemporary Management

Lifestyle Modification

Lifestyle intervention, including dietary modification, increased physical activity, and

behavioral support, is the cornerstone of PMOS management for all women, regardless of fertility goals. Weight loss of 5–10% in overweight and obese women can improve menstrual regularity, ovulation, metabolic parameters, and hyperandrogenic symptoms. No single diet has been proven superior; however, diets emphasizing unsaturated fats, low–glycemic index carbohydrates, fiber, antioxidants, and adequate protein are generally beneficial.[13,14]

Menstrual, Androgen, and Metabolic Care

Combined oral contraceptive pills are commonly first-line pharmacological therapy for irregular cycles and hyperandrogenic symptoms when pregnancy is not desired. They suppress ovarian androgen production, increase SHBG, and provide endometrial protection.

Metformin improves insulin sensitivity, may restore menstrual cyclicity, and is particularly useful in women with glucose intolerance or those unable to tolerate COCs. It is not currently recommended solely for miscarriage prevention.

Anti-androgens such as spironolactone may be used in combination with COCs for hirsutism and acne when COCs alone are insufficient.

Fertility Care

Letrozole is generally preferred as first-line pharmacological therapy for anovulatory infertility in PCOS, with clomiphene citrate as an alternative. Gonadotropins and laparoscopic ovarian drilling are second-line options in selected cases.

Surgical and Procedural Interventions

Laparoscopic ovarian drilling may be considered in women with clomiphene-resistant anovulation who are not candidates for or do not respond to gonadotropins. Bariatric surgery may be appropriate for morbidly obese women with PMOS when conservative measures fail, with careful preconception counseling.

Ayurvedic Interpretation of PMOS

PMOS is not described as a distinct disease entity in classical Ayurvedic texts. Its Ayurvedic interpretation is therefore based on correlation of contemporary clinical features with descriptions of *Nashtartava*, *Artavakshaya*, *Sthoulya, Prameha*, and *Yonivyapad* such as *Vandhyatva*.

According to Acharya Sushruta, *Nastartava* is a condition in which vitiated *Vata* and *Kapha Dosha* causes *Avarana* to *Artavavaha Srotas* and results in *Artavanasa*, this can be considered as secondary amenorrhea or anovulation associated with PCOS.[15]

Artavakshaya is also mentioned by Susruta Aacharya with *Lakshanas* '*Yathochitha Kala Adarshana* (irregular menstruation), *Alpata* (scanty menstruation) and *Yonivedana* (pain in genital region), so this condition can be considered as abnormal uterine bleeding such as oligomenorrhea and hypomenorrhea.[16]

Acharya Charaka mentioned *Sthoulya Lakshanas* like *Chala Sphik Udara Stana* (flabbiness of buttocks, abdomen and breasts) due to increased *Meda* and *Mamsa Dhatu*, these having close resemblance with visceral obesity in women with PCOS.[17]

***Nidana* (Etiological Factors)**

Mithyachara include *Kapha*-aggravating diet and lifestyle, excessive intake of heavy, sweet, cold, and oily foods, *Atimatra asana* (overeating), *Virudhahara*, daytime sleep, physical inactivity, which impair *Jatharagni*. Genetic predisposition may be understood as *Beeja Dushti* and *Sahaja Vyadhi*, while psychological stress (*Chinta, Bhaya, Krodha, Shoka*) may aggravate *Vata* and disturb the HPO axis and contribute to menstrual dysfunction.

Impaired *Jatharagni*, *Rasadhatvagni*, and *Medodhatvagni* lead to *Ama* formation, *Srotorodha*, and disturbed *Dhatu* metabolism, particularly affecting *Rasa*, *Rakta*, *Mamsa*, *Meda*, *Asthi* and *Artava*. *Ama* and *Srotorodha* may be conceptually paralleled with metabolic disturbance, inflammation, and oxidative stress, though they are not identical.

***Dhatu* and *Srotas* Involvement**

Rasa: Srotorodha, Kapha Vriddhi, fatigue, heaviness.

Rakta: *Asrigdara*, skin discoloration

Mamsa: Abdominal heaviness

Meda: Sthoulya, Prameha-like features

Asthi: Kesa–Loma–Smasru abnormalities (hirsutism, alopecia)

Shukra and *Artava: Vandhyatva, Artavanasha*

Clinical Correlations

Nashtartava: Amenorrhea due to *Vata–Kapha* obstruction of *Artavavaha Srotas*.

Artavakshaya: Oligomenorrhea, scanty menstruation, pelvic pain.

Vandhyatva: Infertility due to *Artava* impairment.

Sthoulya: Central obesity, *Medodhatu Vriddhi*.

Prameha: Insulin resistance and dysglycemia-like features.

Yuvanapidika, *Atiloma*, *Neelika*: Acne, hirsutism, hyperpigmentation/acanthosis nigricans.

Rogamarga* and *Sadhyasadyata

PCOS involves *Bahya* (skin, hair), *Madhyama* (HPO axis, endocrine), and *Abhyantara* (metabolic, gastrointestinal) *rogamarga*, and may be classified as *Krichrasadhya* or *Yapya*, requiring long-term management.

Ayurvedic Management Principles

Nidana Parivarjana

Avoidance of causative factors through gradual life style correction is fundamental. It involves

avoiding unhealthy dietary practices, such as excessive consumption of heavy, oily, sweet, and processed foods, along with lifestyle factors such as physical inactivity, excessive daytime sleep, irregular sleep patterns, and psychological stress. Recommendations include: Favoring light, food having *Kapha medohara* properties, regular inclusion of ash gourd, bottle gourd, fenugreek, mint, ginger, garlic, lemon, *Amalaki* and *Agneya dravya* such as Horse gram, sesame, buttermilk, fermented liquids is beneficial. Avoiding daytime sleep, late-night eating, excessive snacking, ultra-processed foods, transfats, and incompatible food combinations. *Dinacharya*, or the Ayurvedic daily regimen, may support PCOS management by promoting circadian rhythm regulation, helping maintain cortisol balance, and improving digestive function and *Agni*.

Shamana Chikitsa

Therapeutic strategies may include: *Kapha–Vatahara* and *Medohara* regimens to reduce excess *Kapha* and *Meda*. *Agnideepana* and *Amapachana* to improve *Jatharagni* and *Dhatvagni*, *Srotoshodhana* to clear channel obstruction. *Swayoni Vardhana Dravya* (e.g., sesame, horse gram) to support *Artava* formation, used judiciously.

Shodhana Chikitsa

Shodhana removes *Ama, Srotorodha* and eliminate accumulated *Dosha*. *Sodhana* help to regain *Pusthi* and *Vrishata.*[17] *Vamana* helps in *Soumya dhatu nirharana* and *Agneya dhatu vridhi*. *Vamana* helps to reduce *Kapha avarana*. *Virechana*, in particular, may help reduce *Kapha* obstruction, normalize *Vata*, and support metabolic and reproductive function. *Beeja karmukata* can be achieved through *Virechana*. *Virechana* detoxifies liver and gut thereby improving hormonal metabolism. *Vasthi* or medicated enema acts as *Apanavatanulomana* and restore normal ovulatory cycles. *Apana Vata* is the root cause of all *Yoniroga* and governs the reproductive physiology. *Rasayana* is also important because of improving overall *Dhatuposhana*, boost immunity and optimize metabolism. *Pathya ahara vihara*- Recommended Foods: "*Matsya*, *Kulatha*, sour items, sesame, black gram, wine, cow urine, buttermilk, curd, and fermented liquids are beneficial

DISCUSSION

PMOS is a complex, multisystem disorder requiring integrated biomedical and lifestyle-based management. Contemporary evidence highlights insulin resistance, hyperandrogenism, neuroendocrine dysfunction, adipose-tissue abnormalities, inflammation, and oxidative stress as central mechanisms. Management is individualized and includes lifestyle modification, pharmacological therapy for menstrual and hyperandrogenic symptoms, metabolic-risk reduction, and fertility-directed treatment.

Ayurvedic concepts of *Dosha, Agni, Ama, Dhatu*, and *Srotas* provide a coherent interpretive framework for understanding diet, lifestyle, metabolic disturbance, and reproductive dysfunction in PMOS. Correlations with *Nashtartava, Artavakshaya, Vandhyatva, Sthoulya,* and *Prameha* help integration of classical principles with modern clinical features. However, these should be viewed as conceptual models rather than definitive diagnostic equivalents.

Integrative management may combine evidence-based biomedical care with Ayurvedic strategies including *Nidana Parivarjana, Agni* correction, *Dosha* balancing, and individualized *Shamana* or *Shodhana* interventions. Psychological support, stress management, and attention to quality of life are essential components of comprehensive care.

CONCLUSION

PMOS is a heterogeneous, multisystem endocrine–metabolic disorder requiring long-term, individualized, and multidisciplinary care. Contemporary biomedical understanding emphasizes insulin resistance, hyperandrogenism, neuroendocrine dysfunction, adipose-tissue abnormalities, inflammation, and oxidative stress. Ayurvedic concepts provide a useful interpretive framework for understanding diet, lifestyle, metabolic disturbance, and reproductive dysfunction in PMOS. However, direct equivalence between PMOS and any single classical diagnosis should be avoided. Standardized Ayurvedic interventions supported by robust clinical research are needed to define their role within integrative PMOS management.

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