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EMAS consensus statement

From PCOS to PMOS: An EMAS clinical guide to the proposed terminology and its relevance to menopause care

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ABSTRACT

Introduction: Polycystic ovary syndrome (PCOS) has recently been renamed polyendocrine metabolic ovarian syndrome (PMOS). This change is particularly relevant after menopause, when menstrual dysfunction and ovarian morphology are no longer useful for assessment, while cardiometabolic and other long-term health risks remain clinically significant.
Aim: To review the implications of the shift in terminology from PCOS to PMOS for women in the menopausal transition and after menopause and to provide guidance for assessment and management.
Materials and methods: This EMAS Clinical Guide is based on a narrative review and expert consensus. PubMed and Web of Science were searched in June 2026 for evidence on PCOS/PMOS during and after menopause, with additional references identified from key guidelines and publications. International guidelines, position statements, systematic reviews, meta-analyses, randomized controlled trials, cohort studies, and other clinically relevant publications were considered. There were notable evidence gaps in relation to postmenopausal women.
Summary recommendations: The proposed change from PCOS to PMOS reflects a broader understanding of the condition as a lifelong endocrine and metabolic disorder rather than a syndrome defined mainly by reproductive features and ovarian morphology. After menopause, given that current menstrual and ovarian morphology cannot be used to establish the diagnosis de novo, recognition of PMOS should rely primarily on a documented diagnosis before menopause or a well-characterized reproductive history consistent with the syndrome. Women with PMOS have increased risk of cardiometabolic conditions, including obesity, impaired glucose metabolism, type 2 diabetes, hypertension, dyslipidemia, and cardiovascular disease. Although evidence specifically in postmenopausal women remains limited, these risks remain clinically relevant during the menopausal transition

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and after menopause and should be considered during long-term follow-up. Clinical care should focus on cardiometabolic risk assessment, which should include evaluation of body weight, waist circumference, blood pressure, glycemic status, lipid profile, and other established cardiovascular risk factors. New, severe, or rapidly progressive hyperandrogenism after menopause should not be attributed to PMOS without further evaluation for alternative causes. Current evidence does not support additional endometrial, breast, or ovarian cancer screening or a separate osteoporosis screening strategy solely on the basis of PMOS. PMOS alone is not a contraindication to menopausal hormone therapy. Evidence specifically evaluating menopausal hormone therapy in women with PMOS is limited. Current recommendations are largely extrapolated from evidence and guidance for the general menopausal population. The decision to initiate menopausal hormone treatment and the choice of regimen and route should follow standard menopause guidance and be based on symptoms and the woman's overall individual risk.

1. Introduction

Polycystic ovary syndrome (PCOS) is the most common endocrine disorder in women of reproductive age, affecting approximately one in every eight to ten women [1]. Although the syndrome has traditionally been defined by ovulatory dysfunction, hyperandrogenism, and polycystic ovarian morphology, it is now widely recognized as a complex condition that extends far beyond reproductive health. Across the lifespan, women with PCOS have an increased risk of insulin resistance, type 2 diabetes, dyslipidemia, hypertension, metabolic dysfunction-associated steatotic liver disease (MASLD), and cardiovascular disease [1]. The 2023 International Evidence-based Guideline for the assessment and management of PCOS recognizes these metabolic features as central components of the syndrome rather than secondary consequences [1].

In 2026, the use of the term “polyendocrine metabolic ovarian syndrome” (PMOS) as an alternative to PCOS was proposed by a global consensus that was reached following a structured process involving professional societies, researchers and patient representatives [2]. The proposal was motivated by the view that the traditional term does not fully reflect the current understanding of the syndrome. In particular, “polycystic” may incorrectly suggest a primary ovarian cystic disorder and understates the endocrine and metabolic features of the condition. The proposed new terminology aims to better reflect its endocrine and metabolic nature across the lifespan rather than focusing on ovarian morphology. However, time will be needed for the term PMOS to be incorporated into diagnostic guidelines and clinical practice.

For menopause specialists, the change in terminology is important. The reproductive manifestations that traditionally define PMOS/PCOS become progressively less useful after menopause. Menstrual dysfunction is no longer informative, ovarian morphology changes with age, and circulating androgen concentrations decline over time. However, many women continue to carry the metabolic and cardiovascular consequences of the syndrome well beyond the reproductive years.

The proposed change from PCOS to PMOS therefore encourages clinicians to view the condition through a lifespan perspective. The aim of this European Menopause and Andropause Society (EMAS) Clinical Guide is to discuss the implications of the shift in terminology from PCOS to PMOS for women during the menopausal transition and after menopause, to summarize the available evidence regarding long-term health outcomes, and to provide guidance for assessment and clinical management.

2. Methodology

This Clinical Guide has been developed as a narrative review of the literature, complemented by expert discussion among the authors. PubMed was searched in June 2026 for English-language publications addressing PCOS or PMOS during the menopausal transition and after menopause. The search included combinations of terms related to PCOS/PMOS, menopause, perimenopause, postmenopause, cardiometabolic health, cardiovascular disease, cancer, bone health, hyperandrogenism, and menopausal hormone therapy. Publications from 2002 to June 2026 were considered, with additional relevant references identified from bibliographies of key articles and guidelines. International guidelines, position statements, systematic reviews, meta-analyses, randomized controlled trials, cohort studies, and other clinically relevant publications were considered. Priority was given to recent guidelines, randomized controlled trials, systematic reviews, meta-analyses, and large population-based or longitudinal studies. The draft paper was discussed and the final version approved by consensus among all authors.

2.1. Transition to PMOS terminology in clinical practice

Gradual implementation of the PMOS terminology is expected in clinical practice. During the transition period, dual labelling as “PCOS (PMOS)” may help maintain continuity in clinical documentation, communication, and research while allowing clinicians and patients to become familiar with the new terminology. Existing PCOS coding may need to be retained where appropriate until specific PMOS coding is incorporated into relevant classification systems and electronic health records. Importantly, the change in terminology should be clearly explained to women who have lived with a PCOS diagnosis for many years. The rationale for introducing the PMOS terminology and its relationship to the patients' previous diagnosis or ongoing clinical care should be clearly emphasized.

2.2. Clinical implications of the transition from PCOS to PMOS after menopause

The proposal to rename the condition PMOS is unlikely to alter the diagnostic framework or management during the reproductive years. PCOS is currently diagnosed according to the Rotterdam criteria, which require the presence of at least two of the following three features after exclusion of other etiologies: oligo- or anovulation, clinical and/or biochemical hyperandrogenism, and polycystic ovarian morphology seen on ultrasound [1,3]. These criteria were reaffirmed in the 2023 International Evidence-based Guideline for the Assessment and Management of PCOS, which also provides updated recommendations for diagnosis across different age groups and clinical settings [1,3]. With the proposed introduction of the term PMOS, the diagnostic criteria and principles of treatment remain unchanged [2]. The greatest clinical impact of this terminology change may be on the care provided for women later in life, when the reproductive features of the syndrome become less prominent but the cardiometabolic and other long-term health considerations remain clinically relevant.

After menopause, the main clinical question is no longer whether a woman fulfils the diagnostic criteria for PCOS (or, now, PMOS), as the diagnosis has usually been established earlier in life. Instead, the challenge is realizing that the condition still matters. After menopause, these women continue to present with higher rates of obesity, impaired glucose metabolism, hypertension, and other cardiometabolic risk factors than women without the syndrome [4]. Menopause does not eliminate these risks; rather, advancing age may increase their clinical importance.

The change in terminology also encourages a shift in clinical priorities. During the reproductive years, care focuses largely on menstrual function, fertility, and hyperandrogenism. After menopause, these concerns are replaced by cardiometabolic risk assessment, cardiovascular prevention, healthy aging, and decisions regarding menopausal hormone therapy. The name PMOS reflects this change more clearly than the traditional term [5].

The new terminology may also have important implications for research. Most long-term studies have followed women who were diagnosed with PCOS during their reproductive years, while evidence after menopause remains limited. The new terminology may support future studies examining aging, cardiometabolic disease, and menopause outcomes in women with PMOS. It may also encourage a more consistent approach when reporting long-term outcomes across different specialties.

2.3. Assessment of women with PMOS during the menopausal transition and after menopause

The diagnosis of PMOS/PCOS cannot be established de novo after menopause using the current Rotterdam criteria. Menstrual function is no longer interpretable in postmenopausal women, as menstruation has ceased. Age-related declines in ovarian volume and antral follicle count prevent the use of the Rotterdam ultrasound criterion, and the physiological decline in androgen concentrations reduces the discriminatory value of biochemical hyperandrogenism. No specific postmenopausal ovarian morphology has been validated as a diagnostic feature of PMOS; therefore, ovarian ultrasound should not be used to establish or reconfirm PMOS after menopause. Consequently, there are currently no validated diagnostic criteria for reliably establishing a new diagnosis or retrospectively confirming PMOS after menopause [1].

Recognition of postmenopausal women with PMOS therefore has to rely primarily on a documented previous diagnosis of the condition (when it will likely have been termed PCOS). In the absence of formal documentation, a well-characterized reproductive history may provide supportive evidence. This may include previous oligo- or anovulation, clinical or biochemical hyperandrogenism, polycystic ovarian morphology, treatment for hyperandrogenic symptoms (e.g. past antiandrogen therapy or the prescription of a combined oral contraceptive), or a history of anovulatory infertility. However, these historical features are not validated criteria for retrospective diagnosis, and a suggestive reproductive history alone may be insufficient to establish a definitive diagnosis. Alternative causes of menstrual irregularity, infertility, or hyperandrogenism should also be considered when interpreting historical information. Available medical records should therefore be reviewed whenever possible. In women without a documented diagnosis, PMOS may be considered when the overall historical evidence is sufficiently suggestive. However, the uncertainty of retrospective case ascertainment should be acknowledged when planning long-term follow-up and risk assessment. The absence of a recorded diagnosis does not necessarily exclude previous PCOS/PMOS, particularly among older women who may not have undergone assessment using contemporary diagnostic criteria.

Clinical assessment should reflect the change in health priorities after menopause. Cardiometabolic risk assessment should follow current international and national guidelines (Table **1**) and include measurement of weight, body mass index (BMI), waist circumference, blood pressure, glycemic status, and lipid profile [1,6–9]. Waist circumference should be interpreted using ethnicity-specific thresholds where available, as metabolic risk may occur at lower levels of adiposity in some populations [10]. Smoking status, physical activity, diet, and alcohol intake should also be reviewed, because they influence overall cardiometabolic risk. A history of gestational diabetes, hypertensive disorders of pregnancy, type 2 diabetes, dyslipidemia or cardiovascular disease may identify women who need closer follow-up [1,11]. Obstructive sleep apnea symptoms are recommended to be assessed at

Table 1
Suggested cardiovascular risk assessment in women with postmenopausal polyendocrine metabolic ovarian syndrome (PMOS), developed according to the 2023 International Evidence-based Guideline for PCOS and NICE guidelines [1,7].

Assessment	What should be assessed?	Suggested frequency*	Comments
Anthropometry	Weight, BMI, waist circumference/ central adiposity	At diagnosis and annually	Assess overweight/ obesity and central adiposity; discuss weight management and healthy lifestyle regardless of BMI
Blood pressure	Office blood pressure measurement	At diagnosis and according to cardiovascular risk	Hypertension contributes to long-term CVD risk and should be assessed as part of cardiovascular prevention
Cardiovascular risk assessment	History of CVD, hypertension, dyslipidemia, smoking, family history; validated risk calculator when appropriate	According to national prevention guidelines	Discuss increased lifetime CVD risk; overall absolute risk may remain low before menopause
Reproductive/ obstetric history	Previous gestational diabetes, hypertensive disorders of pregnancy, pregnancy complications	Initial assessment	Identifies women at increased risk of cardiometabolicdisorders
Glycemic assessment	75 g OGTT when clinically indicated (HbA1c or fasting plasma glucose tests may be performed when OGTT cannot be performed)	At diagnosis and periodically according to risk	Assess glycemic status after diagnosis regardless of age and BMI; higher risk of impaired glucose tolerance and type 2 diabetes should be discussed
Lipid profile	Total cholesterol, LDL-C, HDL-C, triglycerides (full lipid profile)	After diagnosis, repeat according to cardiovascular risk	Full lipid profile should be considered after diagnosis regardless of age and BMI. Further monitoring should follow CVD prevention guidelines
MASLD	Metabolic risk factors, history of liver disease; consider liver enzymes and non-invasive fibrosis risk assessment where clinically indicated	At diagnosis and according to metabolic risk	Consider MASLD particularly in women with obesity, type 2 diabetes, dyslipidemia, hypertension or other features of metabolic dysfunction; further assessment should follow established clinical pathways
Lifestyle assessment	Smoking, alcohol intake, diet, physical activity, sleep	Every clinical review	Lifestyle optimization reduces cardiometabolic risk and should be advised irrespective of BMI
OSA assessment	Symptoms of OSA (snoring, witnessed apnea, excessive daytime sleepiness)	At diagnosis and when clinically indicated	OSA is common in adults with PCOS/PMOS regardless of BMI
Psychological assessment	Depression, anxiety, eating disorders,	At diagnosis and periodically	Psychological symptoms are common and should

(*continued on next page*)

Table 1 (*continued*)

Assessment	What should be assessed?	Suggested frequency*	Comments
	impact on quality of life		be identified and managed
Long-term review	Symptoms, medications, metabolic risks, CVD and diabetes risk	Annually	–

BMI, body mass index; CVD, cardiovascular disease; HDL, high-density lipoprotein; LDL, low-density lipoprotein; MASLD, metabolic dysfunction-associated steatotic liver disease; NICE, National Institute for Health and Care Excellence; OGTT, oral glucose tolerance test; OSA, obstructive sleep apnea; PCOS, polycystic ovarian syndrome; PMOS, polyendocrine metabolic syndrome.

* Assessment intervals should follow the 2023 International Evidence-based Guideline for PCOS [1] together with national cardiovascular prevention guidelines (e.g., NICE in the UK [9], ESC SCORE2 recommendations in Europe [8]), with more frequent monitoring in women at higher cardiometabolic risk.

diagnosis and when clinically indicated [1]. MASLD should be considered in women with obesity, type 2 diabetes, dyslipidemia, hypertension, or other features of metabolic dysfunction, with further assessment undertaken according to established clinical pathways.

Formal cardiovascular risk estimation should be performed using validated regional risk prediction tools, such as SCORE2 (40–69 years) or SCORE2-OP (≥70 years) in Europe [8] and QRISK3 in the UK [6] or equivalent national risk calculators. Assessment intervals should follow the 2023 International Evidence-based Guideline for PCOS together with national cardiovascular prevention guidelines, with more frequent monitoring in women at higher cardiometabolic risk [1,6,8,9]. An important limitation of current cardiovascular risk prediction tools should also be acknowledged. PMOS/PCOS is not included as an independent variable in commonly used risk calculators, including QRISK2, QRISK3 and SCORE2/SCORE2-OP. Consequently, estimated cardiovascular risk may be conservative, as any excess risk associated with PMOS is captured only indirectly, through traditional risk factors such as obesity, hypertension, dyslipidemia and diabetes. Clinicians should therefore interpret calculated risk estimates within the broader clinical context, particularly when estimated risk lies close to treatment thresholds or when other risk-enhancing factors are present.

Biochemical assessment should be guided by the woman's history and current risk factors [1]. Glycemic status and the lipid profile should be assessed according to current PCOS and cardiovascular prevention guidance [1]. The 75 g oral glucose tolerance test is the most accurate test for identifying impaired glucose tolerance and type 2 diabetes in women with PMOS. Fasting plasma glucose or HbA1c tests may be used when an oral glucose tolerance test cannot be performed, although both have lower diagnostic accuracy. Glycemic assessment should be repeated every 1–3 years according to individual risk (weight, family history, previous gestational diabetes and earlier glycemic results), with more frequent assessment in women with higher metabolic risk [1].

Clinical or biochemical hyperandrogenism may persist during the menopausal transition and after menopause. A 2023 systematic review found higher total testosterone, free androgen index and androstenedione, together with lower sex hormone binding globulin, in peri- and postmenopausal women with PCOS than in controls [12]. However, new-onset, rapidly progressive or severe hyperandrogenism should not be attributed to PMOS without further investigation. Androgen-secreting ovarian or adrenal tumors and ovarian hyperthecosis should be considered, particularly when virilization is present or symptoms progress over a short period. Markedly elevated testosterone levels, particularly >5 nmol/L, after exclusion of exogenous testosterone use and in the presence of virilization or rapidly progressive symptoms, should prompt evaluation for an androgen-secreting tumor. However, no single validated threshold has been established for this purpose in postmenopausal women [13].

Routine pelvic ultrasound is not required solely to reconfirm PMOS after menopause. Imaging should instead be guided by clinical findings, such as postmenopausal bleeding, pelvic symptoms, an adnexal mass or marked androgen excess. During the menopausal transition, women with PMOS should continue to be assessed for endometrial hyperplasia and endometrial cancer risk, particularly those with irregular and infrequent menstruation followed by abnormal uterine bleeding. Following appropriate evaluation to exclude endometrial pathology, endometrial protection with progestogen-containing therapy should be considered when indicated [1]. Similarly, repeated measurement of androgen concentrations is generally unnecessary in women with stable clinical features and should be guided by changes in symptoms. Hormonal testing is more relevant when hyperandrogenic features are new or worsening.

The purpose of assessment after menopause is therefore not to apply reproductive diagnostic criteria again. It is to clarify the previous history, identify cardiometabolic risk, recognize symptoms that require further investigation and develop an individualized follow-up plan.

Menopausal symptoms, bone health and fracture risk, and genitourinary syndrome of menopause should also be assessed according to standard menopause care guidelines. These aspects of care should be addressed according to their individual clinical indications and should not be altered solely by the diagnosis of PMOS.

2.3.1. *Summary recommendations*

- The diagnosis of postmenopausal PMOS relies on a documented diagnosis of the condition (likely termed PCOS) before menopause or a well-characterized reproductive history consistent with the syndrome. Current menstrual criteria and ovarian morphology cannot be used to reconfirm the diagnosis.
- Assessment should focus on cardiometabolic risk and on identifying new, severe or progressive hyperandrogenism, which may indicate an alternative diagnosis.
- Cardiovascular risk should be estimated using validated regional prediction tools. However, clinicians should recognize that PCOS/PMOS is not included as an independent variable in current risk calculators and should interpret risk estimates within the broader clinical context.
- During the menopausal transition, persistent anovulation and abnormal uterine bleeding warrant evaluation for endometrial pathology. Progestogen therapy may be considered for endometrial protection when indicated.

2.4. *Cardiometabolic health*

Cardiometabolic health is a major concern in women with PMOS throughout adult life. The risk of type 2 diabetes is clearly increased, although much of this excess risk is linked to overweight and obesity. A meta-analysis of observational studies found that women with PCOS had a 3.45-fold (95% CI 2.95–4.05) higher risk of type 2 diabetes than women without PCOS [14]. The association was stronger in women with obesity, supporting the need to consider both PMOS and adiposity when assessing glycemic risk.

Hypertension and dyslipidemia are also common in women with PMOS. A recent population-based multiregister study from Sweden found higher risks of both conditions in women with PCOS/PMOS after adjustment for birth period, country of birth, educational level, and, in separate models, BMI or obesity diagnosis [15]. The associations were strongest in women with hyperandrogenic phenotypes, suggesting that cardiometabolic risk is not uniform across the syndrome. Blood pressure should therefore be measured regularly, and a fasting lipid profile should be obtained according to current cardiovascular prevention guidance.

Evidence also suggests an increased risk of clinical cardiovascular

disease. A systematic review and meta-analysis performed for the 2023 International Guideline found higher odds of composite cardiovascular disease, ischemic heart disease, myocardial infarction, and stroke in women with PCOS than in women without the condition [4]. Another systematic review of peri- and postmenopausal women with PCOS reported increased odds of diabetes, hypertension, myocardial infarction, and stroke [12]. In that review, it was found that women with PCOS also had higher BMI, waist circumference, waist-to-hip ratio, and insulin resistance, together with lower HDL cholesterol and higher triglyceride concentrations. However, several associations became weaker or were no longer significant when analyses were restricted to groups matched for BMI. This suggests that adiposity is an important contributor to later cardiometabolic risk and should not be separated from the clinical assessment of PMOS.

PMOS should therefore be viewed as a marker for risk assessment. Individual risk is influenced by age, body composition, smoking, blood pressure, lipid levels, glycemic status, family history, and previous cardiovascular events. Menopause care offers an opportunity to review these factors together and address those that can be modified.

Lifestyle management is an important component of menopause care, particularly for women with PMOS, given the high prevalence of overweight and obesity and their contribution to cardiometabolic risk. Regular physical activity, a healthy dietary pattern, smoking cessation, and structured lifestyle interventions supporting sustainable weight loss should be discussed according to the woman's needs and preferences. For women with overweight or obesity, weight-management strategies should follow established obesity management guidelines. These may include individualized dietary and behavioral interventions, pharmacological treatment where clinically indicated, and metabolic or bariatric surgery for eligible patients [16]. Hypertension, dyslipidemia, and diabetes should be treated according to current clinical guidelines. There is no evidence that women with PMOS require different obesity treatment strategies or treatment targets solely because of the diagnosis.

2.4.1. Summary recommendations

- Women with PMOS should undergo regular assessment of weight, BMI, waist circumference, blood pressure, glycemic status, and lipid profile during midlife and after menopause. The timing of repeat testing should be based on individual risk.
- PMOS should be considered a marker of increased cardiometabolic risk, but risk assessment and treatment should remain individualized. Particular attention should be given to obesity and other established risk factors, including smoking, hypertension, dyslipidemia, diabetes, and known cardiovascular disease.
- Women with PMOS should be encouraged to adopt a healthy lifestyle, including regular physical activity, a healthy diet, smoking cessation, and sustainable weight management. PMOS alone does not require different lifestyle or weight-management targets.

2.5. Endometrial, breast, and ovarian cancer

Endometrial health is an important component of long-term care in women with PMOS. Chronic anovulation increases the risk of endometrial neoplasia. Over time, this may contribute to endometrial hyperplasia and, in some women, progression to endometrial cancer. Obesity, insulin resistance, and type 2 diabetes may further increase endometrial risk. The 2023 International Evidence-based Guideline recognizes an increased risk of endometrial hyperplasia and endometrial cancer in premenopausal women with PMOS [1]. However, interpretation of this association is complicated by differences in PMOS/PCOS diagnostic criteria and by the contribution of obesity, diabetes, infertility, and prolonged anovulation [17]. It therefore remains uncertain to what extent PMOS independently contributes to endometrial cancer risk.

The cumulative duration of anovulation and unopposed estrogen exposure is an important consideration when assessing endometrial risk across the menopausal transition [18,19]. Strategies during the reproductive and perimenopausal years to reduce prolonged periods of unopposed endometrial stimulation, such as progestogen therapy, may therefore have a preventive role. The choice of regimen and route of administration should take into account bleeding patterns, contraceptive needs, comorbidities, and patient preference. The aim is to prevent prolonged unopposed estrogen exposure rather than to treat PMOS itself.

The choice of regimen should follow established guidance for endometrial protection. Current evidence does not support routine endometrial cancer screening, including pelvic ultrasound or endometrial biopsy, solely on the basis of PMOS [1]. Investigation should instead be guided by standard clinical indications. Particular attention should be given to women with additional risk factors, especially obesity, type 2 diabetes, and a history of prolonged anovulation, who may have greater cumulative endometrial risk. During the perimenopause, infrequent, irregular, and heavy bleeding as well as intermenstrual bleeding should prompt investigation for endometrial pathology and consideration of use of progestogens for endometrial protection. Abnormal bleeding should not be attributed to menopausal hormone therapy without appropriate assessment. Postmenopausal bleeding requires assessment according to current clinical guidance, regardless of whether the woman has PMOS. Pelvic pain, abnormal vaginal discharge or an endometrial abnormality identified during imaging for another indication should likewise be evaluated according to standard clinical pathways.

Evidence regarding breast cancer is less consistent. Earlier systematic reviews and studies of women during and after the menopausal transition did not demonstrate a clear increase in breast cancer risk among women with a history of PCOS [12,17]. However, a recent large nationwide cohort study reported a modest increase in breast cancer risk among women with PCOS (HR 1.21, 95% CI 1.02–1.40), with a stronger association reported in postmenopausal women (HR 1.63, 95% CI 1.23–2.15) [20]. The postmenopausal association was reported after adjustment for birth year, educational level, and, in a separate model, obesity. However, diabetes, reproductive factors, and menopausal hormone therapy were not included as confounders in the model, and residual confounding and detection bias cannot be excluded. These findings should therefore be interpreted cautiously.

Women with PMOS have a higher prevalence of several established risk factors for breast cancer, including obesity, type 2 diabetes, nulliparity, later age at first birth, reduced breastfeeding, and previous exposure to exogenous hormones. These factors may confound or mediate the observed association and are variably accounted for across studies. Consequently, the available evidence does not establish PMOS as an independent risk factor for breast cancer. Current data do not support breast cancer surveillance based solely on a history of PMOS, beyond that recommended for the general population.

Evidence regarding ovarian cancer remains limited and inconsistent. Earlier systematic review data did not demonstrate a consistent increase in ovarian cancer risk among women with PCOS [17]. Current evidence does not support additional ovarian cancer screening solely because of PMOS. Evaluation should follow standard clinical pathways when symptoms, examination findings, family history, or genetic risk raise concern.

Summary recommendations

- Women with PMOS and prolonged anovulation during the reproductive or perimenopausal years should be considered at increased risk of endometrial neoplasia, particularly in the presence of obesity, insulin resistance, or type 2 diabetes.
- Progestogens should be considered for perimenopausal women with PMOS who have abnormal uterine bleeding.
- PMOS alone does not justify routine endometrial cancer screening after menopause. Any postmenopausal bleeding should be investigated according to standard clinical guidance.

- A history of PMOS alone does not justify additional breast or ovarian cancer surveillance. Screening should follow population-based and individualized recommendations.
- Cancer risk assessment should consider established factors, including obesity, diabetes, reproductive history, family history, and previous hormone exposure, rather than the PMOS diagnosis alone.

2.6. Bone health

The relationship between PMOS and bone health remains uncertain. Several features of the syndrome may influence the skeleton in different ways. A 2023 systematic review and meta-analysis found a statistically non-significant 25% increase in fracture risk among women with PCOS compared with women without [21]. However, the available studies were heterogeneous, and fracture data in peri- and postmenopausal women were limited. These findings therefore do not establish PCOS/PMOS as an independent risk factor for fragility fracture. In contrast, more recent longitudinal evidence has suggested that women with PCOS/PMOS may have higher bone mineral density (BMD) at the lumbar spine and hip during midlife, together with lower bone turnover. These differences may become more apparent after menopause [22].

However, the clinical significance of higher BMD in PMOS remains uncertain. The observed BMD advantage may be partly explained by higher body weight [23] and androgen exposure [24], neither of which necessarily translates into lower fracture risk. Obesity and insulin resistance may influence bone quality and fracture susceptibility in ways that are not fully captured by BMD. Moreover, impaired physical function and an increased risk of falls may contribute to fracture risk.

Taken together, menstrual dysfunction, hyperandrogenism, insulin resistance, and higher body weight may influence bone metabolism in different ways, but their combined effects remain incompletely understood. Differences in age, body weight, PMOS phenotype, and menopausal status may partly explain the inconsistent findings across studies.

Current evidence does not support a separate osteoporosis screening strategy solely because of PMOS. Instead, assessment should follow standard menopause and osteoporosis guidance. Measurement of BMD should be performed when indicated by the woman's overall fracture risk rather than by a diagnosis of PMOS alone.

Lifestyle measures that support skeletal health should be encouraged. These include regular weight-bearing and muscle-strengthening exercise, adequate dietary calcium and protein intake, smoking cessation, and avoidance of excessive alcohol consumption. Vitamin D status should be assessed and managed according to standard clinical indications [25]. Osteoporosis, when present, should be treated according to current clinical guidelines rather than with a PMOS-specific approach.

2.6.1. Summary recommendations

- PMOS alone should not be considered an indication for routine measurement of bone mineral density or a separate osteoporosis screening program.
- Bone health assessment should follow standard risk-based recommendations and should consider age, menopausal status, previous fracture, body weight, family history, smoking, alcohol use, glucocorticoid exposure, and other established osteoporosis risk factors.
- Lifestyle measures and treatment of osteoporosis should follow current clinical guidance. Available evidence is insufficient to recommend PMOS-specific fracture prevention strategies or treatment targets.

3. Menopausal hormone therapy

The availability of different hormonal and non-hormonal preparations for managing menopause varies worldwide. This Clinical Guide will not consider herbal supplements, botanicals, and alternative complementary therapies as there is a paucity of data regarding safety and efficacy. In addition, some products may contain compounds which can interact with menopausal hormone therapy (MHT) and other therapies [1,3].

Evidence specifically examining MHT use by women with PCOS/PMOS is limited. Major randomized trials of MHT, including the Women's Health Initiative (WHI), the Kronos Early Estrogen Prevention Study (KEEPS), and the Early versus Late Intervention Trial with Estradiol (ELITE), were conducted according to general menopause eligibility criteria and did not evaluate women with PMOS as a separate prespecified subgroup [26–29]. Therefore, WHI, KEEPS, and ELITE do not provide direct evidence for a PMOS-specific MHT regimen or risk profile. Similarly, other studies have not established a hormone therapy regimen specifically for this population.

Current guidance from EMAS and the International Menopause Society (IMS) does not identify PMOS as a contraindication to MHT [30,31]. Systemic hormone therapy may be offered to women with bothersome vasomotor symptoms or other appropriate indications after individualized assessment of age, time since menopause, symptom burden, cardiovascular risk, venous thromboembolism risk, breast cancer risk, and patient preference. Decisions regarding MHT should follow standard indications and contraindications while taking into account the cardiometabolic and endometrial features that may accompany PMOS.

Cardiometabolic assessment is particularly relevant in women with PMOS because obesity, hypertension, dyslipidemia, and impaired glucose metabolism are common. These conditions do not automatically exclude MHT, but they may influence the choice of formulation and route of administration. Blood pressure, body weight, glycemic status, lipid profile, smoking status, and personal and family history of cardiovascular disease and venous thromboembolism should be reviewed before treatment.

Women with PMOS may have an increased baseline risk of venous thromboembolism. A 2020 systematic review and meta-analysis of observational studies reported a higher risk of venous thromboembolism among women with PCOS, including in analyses adjusted for obesity and hormonal treatment [32]. However, the available evidence is derived predominantly from reproductive-age populations, and data specifically evaluating venous thromboembolism risk after menopause are limited. Therefore, whether any excess risk persists after menopause independently of obesity and other conventional risk factors remains uncertain. This potential baseline risk should be distinguished from the additional thromboembolic risk associated with MHT.

Transdermal estradiol may be preferred over oral estrogen in women with obesity, hypertriglyceridemia, diabetes, hypertension, or an increased risk of venous thromboembolism because it avoids first-pass hepatic metabolism and has less effect on coagulation and triglycerides [31]. However, the route of administration should be selected according to the woman's overall thromboembolic and cardiometabolic risk profile, treatment goals, availability, and preference rather than the diagnosis of PMOS alone. Evidence comparing oral and transdermal estrogen specifically in women with PMOS is lacking.

Women with an intact uterus require adequate endometrial protection with a progestogen. The progestogen regimen should follow standard MHT guidance. A history of PMOS does not require a higher progestogen dose or additional routine endometrial surveillance in asymptomatic women. The choice of progestogen may be relevant in women with cardiometabolic risk factors, as individual progestogens differ in their androgenic and metabolic effects. Micronized progesterone or dydrogesterone may be considered according to the woman's cardiometabolic profile, bleeding pattern, tolerability, availability, and need for endometrial protection.

A 52 mg levonorgestrel intrauterine device provides both contraception and endometrial protection as part of MHT. Studies support endometrial protection for up to 2 years [33]. The licensed duration for endometrial protection with concomitant estrogen therapy varies between countries, extending up to 4 years in some countries [34]. Local

prescribing and licensing recommendations should therefore be followed. In some clinical settings, off-label use may be considered beyond the licensed duration, possibly up to 5 years [35].

MHT should not be prescribed for the primary prevention of cardiovascular disease or solely to improve the metabolic features of PMOS. Although hormone therapy may influence glucose metabolism, lipid concentrations, and body fat distribution, these effects do not replace lifestyle management or appropriate treatment of hypertension, dyslipidemia, and diabetes. Cardiometabolic conditions should continue to be managed according to current clinical guidelines.

Treatment should be reviewed periodically to assess symptom control, bleeding, adverse effects, and changes in cardiovascular, metabolic or cancer risk. There is no fixed duration of MHT determined by PMOS. Continuation should be based on ongoing indications, shared decision making, and repeated assessment of benefits and risks.

There is currently no evidence that a history of PMOS alters the indications for testosterone therapy in postmenopausal women.

3.1.1. Summary recommendations

- PMOS alone should not be considered a contraindication to MHT. Treatment should follow standard indications and contraindications and should be individualized according to age, time since menopause, symptom burden, cardiovascular risk, venous thromboembolism risk, breast cancer risk, and patient preference.
- Cardiometabolic risk factors should be assessed before initiation of systemic MHT. The route and formulation should be selected according to the woman's overall risk profile rather than the diagnosis of PMOS alone.
- In women with obesity or elevated venous thromboembolism or cardiometabolic risk, transdermal estradiol may be preferred. However, current recommendations are largely extrapolated from evidence and guidance for the general menopausal population.
- MHT should not be prescribed solely for the primary prevention of cardiovascular disease or to treat the metabolic features of PMOS.

Table 2
Summary recommendations.

Assessment of women with PMOS during the menopausal transition and after menopause
• The diagnosis of postmenopausal PMOS relies on a documented diagnosis of PMOS/PCOS before menopause or a well-characterized reproductive history consistent with the syndrome. Current menstrual criteria and ovarian morphology cannot be used to reconfirm the diagnosis. • Assessment should focus on cardiometabolic risk and on identifying new, severe or progressive hyperandrogenism, which may indicate an alternative diagnosis. • Cardiovascular risk should be estimated using validated regional prediction tools. However, clinicians should recognize that PCOS/PMOS is not included as an independent variable in current risk calculators and should interpret risk estimates within the broader clinical context. • During the menopausal transition, persistent anovulation and abnormal uterine bleeding warrant evaluation for endometrial pathology. Progestogen therapy may be considered for endometrial protection when indicated.
Cardiometabolic health
• Women with PMOS should undergo regular assessment of weight, BMI, waist circumference, blood pressure, glycemic status, and lipid profile during midlife and after menopause. The timing of repeat testing should be based on individual risk. • PMOS should be considered a marker of increased cardiometabolic risk, but risk assessment and treatment should remain individualized. Particular attention should be given to obesity and other established risk factors, including smoking, hypertension, dyslipidemia, diabetes, and known cardiovascular disease. • Women with PMOS should be encouraged to adopt a healthy lifestyle, including regular physical activity, a healthy diet, smoking cessation, and sustainable weight management. PMOS alone does not require different lifestyle or weight-management targets.
Endometrial, breast, and ovarian cancer
• Women with PMOS and prolonged anovulation during the reproductive or perimenopausal years should be considered at increased risk of endometrial neoplasia, particularly in the presence of obesity, insulin resistance, or type 2 diabetes. • Progestogens should be considered for perimenopausal women with PMOS who have abnormal uterine bleeding. • PMOS alone does not justify routine endometrial cancer screening after menopause. Any postmenopausal bleeding should be investigated according to standard clinical guidance. • A history of PMOS alone does not justify additional breast or ovarian cancer surveillance. Screening should follow population-based and individualized recommendations. • Cancer risk assessment should consider established factors, including obesity, diabetes, reproductive history, family history, and previous hormone exposure, rather than the PMOS diagnosis alone.
Bone health
• PMOS alone should not be considered an indication for routine measurement of bone mineral density or a separate osteoporosis screening program. • Bone health assessment should follow standard risk-based recommendations and should consider age, menopausal status, previous fracture, body weight, family history, smoking, alcohol use, glucocorticoid exposure, and other established osteoporosis risk factors. • Lifestyle measures and treatment of osteoporosis should follow current clinical guidance. Available evidence is insufficient to recommend PMOS-specific fracture prevention strategies or treatment targets.
Menopausal hormone therapy
• PMOS alone should not be considered a contraindication to MHT. Treatment should follow standard indications and contraindications and should be individualized according to age, time since menopause, symptom burden, cardiovascular risk, venous thromboembolism risk, breast cancer risk, and patient preference. • Cardiometabolic risk factors should be assessed before initiation of systemic MHT. The route and formulation should be selected according to the woman's overall risk profile rather than the diagnosis of PMOS alone. • In women with obesity or elevated venous thromboembolism or cardiometabolic risk, transdermal estradiol may be preferred. However, current recommendations are largely extrapolated from evidence and guidance for the general menopausal population. • MHT should not be prescribed solely for the primary prevention of cardiovascular disease or to treat the metabolic features of PMOS.

3.2. Research agenda for PMOS

Research should focus on women during the menopausal transition and after menopause, as this group remains underrepresented in current studies. Studies are needed to validate approaches for identifying women with a history of PMOS/PCOS after menopause and to determine the reliability of retrospective diagnosis when documentation from the time of diagnosis is unavailable. Longitudinal studies should evaluate the independent associations of PMOS with type 2 diabetes, cardiovascular disease, endometrial and breast cancer, bone health, and fracture risk, while appropriately accounting for obesity, aging, menopausal status, and other established risk factors. Research should also examine whether different PMOS phenotypes are associated with different long-term outcomes. Direct studies of MHT are needed to assess symptom control, metabolic effects, cardiovascular and thromboembolic safety, and cancer outcomes in women with PMOS. Finally, studies should evaluate the acceptability and understanding of the proposed terminology among healthcare professionals and women. They should also assess its clinical consequences, including whether it improves recognition of the condition and continuity of long-term care.

4. Conclusion and summary recommendations (Table 2).

The transition from PCOS to PMOS reflects a broader understanding of the condition as a lifelong endocrine and metabolic disorder rather than a syndrome defined mainly by reproductive features and ovarian morphology. This change is particularly relevant after menopause, when menstrual criteria and ovarian morphology are no longer useful, but cardiometabolic, endometrial, and other long-term health considerations remain important.

Based on the current evidence, the key recommendations for the management of women with PMOS/PCOS during the menopausal transition and after menopause are summarized below, with a graphical overview provided in Fig. 1:

- PMOS should be considered a lifelong endocrine and metabolic condition.
- Recognition after menopause should be based mainly on a previous diagnosis of PMOS/PCOS or a well-documented reproductive history.
- Current menstrual criteria and ovarian morphology should not be used to reconfirm the diagnosis after menopause.
- Women with a history of PMOS/PCOS may have increased long-term cardiometabolic risk, including obesity, impaired glucose metabolism, type 2 diabetes, hypertension, dyslipidemia, and cardiovascular disease. Much of this excess risk is closely related to adiposity and other established cardiometabolic risk factors, and evidence specifically in postmenopausal women remains limited. These risks should nevertheless be considered when planning long-term follow-up after menopause.
- Cardiometabolic risk assessment should include weight and waist circumference, blood pressure, glycemic status, lipid profile, and other established risk factors.
- New, severe or rapidly progressive hyperandrogenism should be investigated for other causes.
- PMOS alone does not require additional endometrial, breast or ovarian cancer screening.
- PMOS alone does not require a separate osteoporosis screening program.
- PMOS is not a contraindication to MHT.
- MHT should follow standard guidance and indication and should be individualized according to symptoms and overall clinical risk.
- Evidence specifically evaluating MHT in women with PMOS is limited, and current recommendations are largely extrapolated from evidence and guidance for the general menopausal population.

Contributors

Fatih Aktoz, C. Tamer Erel, and Ipek Betul Ozcivit Erkan prepared

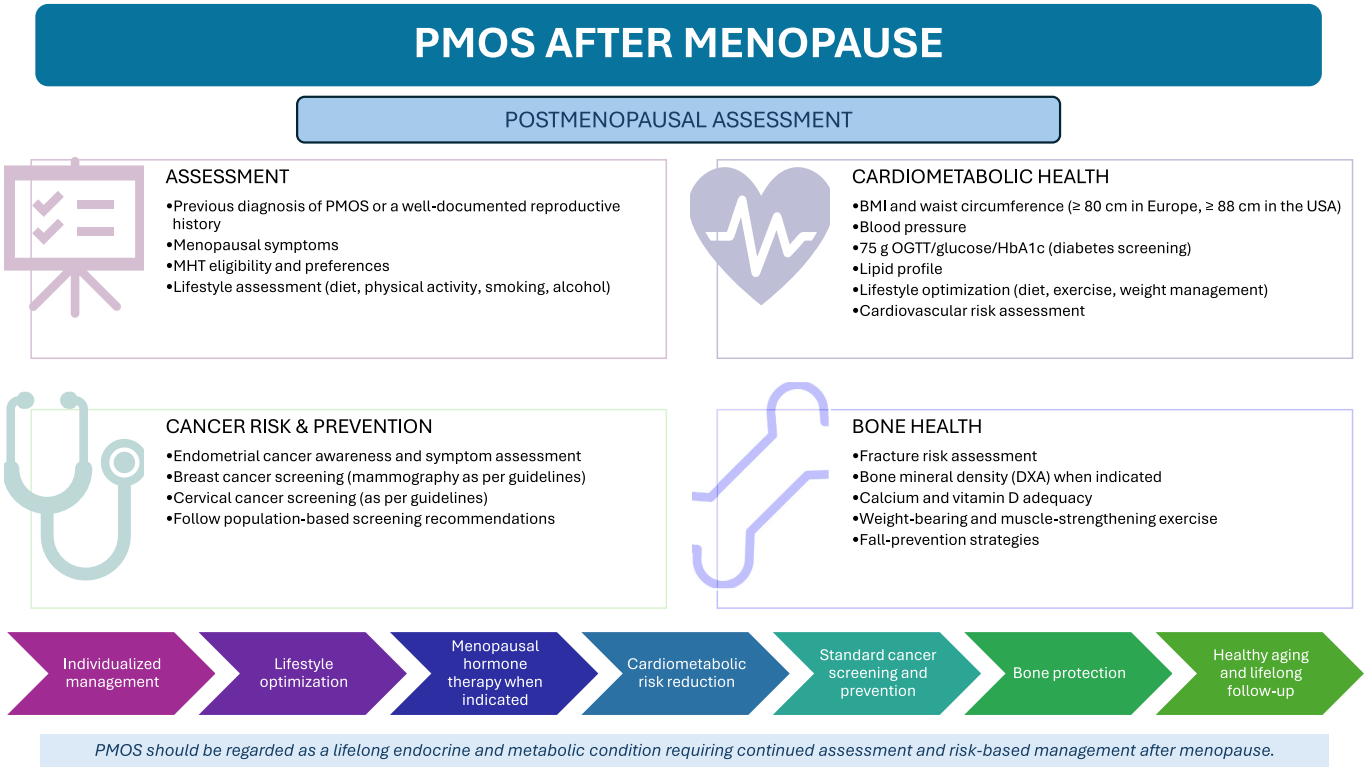

Fig. 1. Comprehensive postmenopausal assessment and management of PMOS after menopause (BMI, body mass index; DXA, dual-energy X-ray absorptiometry; MHT, menopausal hormone therapy; OGTT, oral glucose tolerance test; PMOS, polyendocrine metabolic ovarian syndrome).

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