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Original article

Endometriosis and menopausal health: An EMAS clinical guide

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ABSTRACT

Introduction: Endometriosis is a common gynecological condition, and problems may persist or develop after the menopause. Endometriosis or its treatment in premenopausal women may lead to premature or early menopause. Thus, it is imperative that healthcare providers are appropriately trained in management of endometriosis at the menopause and beyond.
Aim: To provide an evidence-based clinical guide for the assessment and management of menopausal health in women with a history of endometriosis.
Materials and methods: Review of the literature and consensus of expert opinion.
Summary recommendations: Surgery is the preferred option for managing symptomatic endometriosis after the menopause, as it should reduce pain, ensure an accurate diagnosis, and decrease risk of malignancy. Women with endometriosis may experience a spontaneous early menopause or surgically induced menopause. Endometriosis is also associated with an increased risk of cardiovascular disease, ovarian, breast, and thyroid cancers, as well as osteoporosis. Menopausal hormone therapy (MHT) is indicated for managing vasomotor and genitourinary symptoms and maintaining bone health. Continuous combined MHT may be safer than other forms in both hysterectomized and non-hysterectomized women with endometriosis as the risk of recurrence and malignant transformation of residual endometriosis may be reduced. Estrogen-only MHT should be avoided, even for women who have had a hysterectomy. For women not using MHT, alternative pharmacological treatments, such as neurokinin-3 receptor antagonists, should be considered for managing vasomotor symptoms. Additionally, antiresorptive and anabolic therapies, along with calcium and vitamin D supplementation, should be provided as indicated to ensure skeletal protection. If endometriosis recurs during MHT use and the patient is symptomatic,

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several management strategies may be employed: altering the regimen, discontinuation, and use of non-hormonal strategies. Herbal preparations should be avoided as their efficacy is uncertain and some may contain estrogenic compounds.

1. Introduction

Endometriosis is defined as the presence of endometrial glands and stroma outside the uterine cavity [1]. It is a chronic inflammatory disorder characterized by being estrogen-dependent and progesterone-resistant [2–4]. Overall, it affects 5-10% of women of reproductive age and 2-5% after the menopause [2]. It is found in 35-50% of women with infertility and 15-75% of those with chronic pelvic pain [2,5]. Although endometriosis is most prevalent in premenopausal women, with 80.4% of cases under age 45, a notable proportion of patients—17.1% perimenopausal and 2.6% postmenopausal—highlights the need to consider endometriosis in women over 40 presenting with unexplained pelvic pain [6]. Hence, endometriosis should be suspected in women presenting with chronic pelvic pain, history of menstrual-related pain, dyspareunia, gastrointestinal symptoms, urinary symptoms, especially hematuria or dysuria, and a history of infertility [1].

Theories regarding the genesis of endometriosis are retrograde menstruation and implantation, coelomic metaplasia, induction of Mullerian remnants, and lymphatic or hematogenous spread of the endometrial cells [2,4,7]. Additionally, extrauterine stem or progenitor cells originating from bone marrow and endothelium may differentiate into endometriotic tissue. Hereditary or acquired genetic and epigenetic defects and environmental factors may influence and alter sex steroid and immune system functions, as well as the production and expression of certain local substances that play a crucial role in tissue remodeling in endometriosis [4,7].

Postmenopausal endometriosis is considered to have a more complex pathophysiology than premenopausal disease. It remains unclear whether postmenopausal endometriosis arises as a recurrence or persistence of a pre-existing lesion or develops de novo. This may be partly due to diagnostic delays of approximately 6 to 8 years in both premenopausal and postmenopausal women [8,9]. Activation of postmenopausal endometriotic lesions may be a consequence of exposure to exogenous (such as menopausal hormone therapy [MHT]) or endogenous estrogen [10–14]. Endogenous estrogens are generated through aromatase activation in adipose tissue or locally produced within endometriotic lesions via the activation of enzymes such as steroidogenic acute regulatory protein (StAR), aromatase, and 17-beta-hydroxysteroid dehydrogenase (17β-HSD). Local synthesis of estradiol (E2) within endometriotic lesions occurs independently of serum estradiol concentrations [15]. This may explain the activation and growth of endometriotic foci in non-users of MHT.

In most cases, but not all, endometriosis improves after the menopause. Menopause is classified as natural (after the age of 45), premature (before the age of 40) or early (between the ages of 40 and 45). In women with endometriosis, menopause may be induced through surgery or use of gonadotropin-releasing hormone analogues (GnRH-a) [16,17]. Menopause, especially when early, and premature ovarian insufficiency (POI) are associated with significant long-term health risks, including increased risks of overall mortality, cardiovascular disease (CVD), osteoporosis, dementia, and cognitive decline [18].

The loss of estrogen during menopause may lead to vasomotor and genitourinary symptoms [19]. As endometriosis is an estrogen-dependent condition, balancing the potential benefits of MHT with control of endometriosis is imperative, and non-hormonal strategies need to be considered alongside MHT [20]. The aim of this clinical guide is to provide evidence-based advice for the management of menopausal symptoms and long-term health in women with a history of endometriosis.

2. Methodology

This clinical guide is based on a literature search in PubMed, Scopus, and Google Scholar. The search terms included endometriosis, menopause, postmenopausal, menopausal transition, hormone replacement therapy (HRT) and MHT. Articles and guidelines about perimenopausal endometriosis were selected. No language restriction was applied.

3. Endometriosis and age at menopause

Endometriosis, particularly ovarian endometriomas, can lead to early menopause even in women who have not undergone surgery [21–25]. The Nurses' Health Study found an increased risk for early natural menopause in women with laparoscopically confirmed endometriosis [24]. During 1,508,462 person-years of follow-up, 6,640 women reported being diagnosed with endometriosis [24]. A 50% higher risk of early natural menopause (defined as menopause before age 45) was found (hazard ratio [HR] 1.51; 95% confidence interval [CI] 1.30, 1.74) [24]. Furthermore, a cross-sectional analysis from the Japan Nurses' Health Study found that women with endometriosis and infertility had a significantly higher risk of earlier menopause than those who were fertile (risk ratio [RR] 3.43; 95% CI 2.17, 5.44) [24].

A 2025 meta-analysis of 279,948 women from five international cohorts (UK, Australia, Sweden, and Japan; 1996–2022) found that women with endometriosis had a 7.5-fold increased risk of surgical menopause (HR 7.54; 95% CI 6.84, 8.32) and were 60% less likely to undergo natural menopause (HR 0.40; 95% CI 0.33, 0.49) [26]. Surgical menopause occurred 1.6 years earlier on average (β –1.59; 95% CI –1.77, –1.42), and natural menopause occurred 0.4 years earlier (β –0.37; 95% CI –0.46, –0.28). Women with endometriosis had twice the odds of premature (age <40 years) surgical menopause (OR 2.11; 95% CI 2.02, 2.20) and a 36% increased risk of spontaneous premature ovarian insufficiency (OR 1.36; 95% CI 1.17, 1.59). The study highlights a strong association between endometriosis and both earlier natural and more frequent surgical menopause.

Not only bilateral salpingo-oophorectomy but also conservative ovarian surgery for endometriomas can lead to ovarian failure. Laparoscopic cystectomy for endometriomas has been shown to decrease the antral follicle count (AFC) as well as concentrations of serum anti-Müllerian hormone (AMH) and inhibin B while increasing serum concentrations of follicle-stimulating hormone (FSH), all of which indicate POI [27,28]. A study conducted at a tertiary endometriosis center prospectively followed 302 women who had undergone surgery for endometriosis [29]. Median duration of follow-up was 8.5 years (range 2-17 years). Menopause was documented in 43 women (14.3%) at a mean age of 45.3 ± 4.3 years (range 32–52 years). Women who had undergone bilateral cystectomy reached menopause at a younger age than those with unilateral endometriomas (42.1 ± 5.1 years vs. 47.1 ± 3.5 years, p=0.003). The association between the total diameter of the preoperative ovarian endometriomas and menopausal age was significant for the group of women who had had surgery for bilateral endometriomas (r^2 = 0.754, P = 0.002). The 2022 guidelines from the European Society of Human Reproduction and Embryology (ESHRE) do not recommend cystectomy for endometriomas in women with infertility, as ovarian surgery can reduce ovarian reserve and lead to POI [30].

4. Endometriosis, quality of life, and sexual health

The World Health Organization (WHO) acknowledges the significant impact of endometriosis on multiple aspects of health-related quality of

life (HRQoL): on sexual and reproductive health, overall quality of life, and overall well-being [31]. A self-reported longitudinal survey studied 3,728 Australian women using the 36-item Short Form Survey every three years from 1996 to 2018 [32]. It found that endometriosis was significantly associated with poorer outcomes across various HRQoL dimensions, including physical functioning (OR 1.33; 95% CI 1.19, 1.50), role limitations due to physical problems (OR 1.57; 95% CI 1.41, 1.74), bodily pain (OR 1.65; 95% CI 1.48, 1.82), general health (OR 1.61; 95% CI 1.42, 1.81), vitality (OR 1.38; 95% CI 1.23, 1.55), social functioning (OR 1.38; 95% CI 1.25, 1.53), role limitations due to emotional problems (OR 1.19; 95% CI 1.06, 1.33), and mental health (OR 1.32; 95% CI 1.18, 1.48). Additionally, women with endometriosis scored significantly less for both the physical (OR 1.68; 95% CI 1.51, 1.88) and the mental health components (OR 1.28; 95% CI 1.14, 1.44).

A 2020 systematic review (of 4 studies) analyzed sexual function (via the 19-item Female Sexual Function Index [FSFI]), and dyspareunia, chronic pelvic pain, and dysmenorrhea (via a visual analogue scale [VAS]) for groups of untreated women with (n=225) and without (n=198) endometriosis [33]. Although mean total FSFI scores were not significantly different between the 2 groups, women with untreated endometriosis were more likely to have FSFI scores below 26, indicating possible sexual dysfunction (OR 2.38; 95% CI 1.12, 5.04). Additionally, all FSFI domain scores were significantly lower in the non-treated women with endometriosis: desire (mean difference [MD] -0.43; 95% CI -0.57, -0.19), arousal (MD -0.66; 95% CI -1.15, -0.17), lubrication (MD -0.41; 95% CI -0.79, -0.02), orgasm (MD -0.40; 95% CI -0.73, -0.06), satisfaction (MD -0.45; 95% CI -0.72, -0.18), and pain (MD -1.03; 95% CI -1.34, -0.72). Women with endometriosis also experienced more severe dyspareunia (MD 1.88; 95% CI 0.38, 3.37) and chronic pelvic pain (MD 2.92; 95% CI 1.26, 4.58), but no significant difference was observed for dysmenorrhea.

When menopause is induced in women with endometriosis—-whether surgically or medically—it may lead to the abrupt onset of vasomotor symptoms, sleep disturbances, mood changes, and genitourinary syndrome of menopause (GSM), all of which can further reduce quality of life [34]. This is particularly important in women who already have chronic pelvic pain or sexual dysfunction related to endometriosis.

5. Endometriosis and concomitant disease

5.1. Osteoporosis

Bone health is estrogen dependent; thus, women with a history of endometriosis are at particular risk of the long-term adverse effects of estrogen deficiency as a consequence of repeated courses of gonadotropin releasing hormone analogues (GnRH-a) or progestogens, or surgery [35,36]. Despite this, the literature is conflicting, with some studies finding that women with endometriosis have no overall increase in long-term fracture risk [37,38]. For example, the Japan Nurses' Health Study reported an increased risk of osteoporosis in women with early-onset endometriosis [39], although a cause-and-effect relationship was not established. Conversely, a case-controlled study suggested that bone mineral density (BMD) was higher following oophorectomy performed for deep endometriosis (n=31) than it was following oophorectomy conducted for other reasons (n=52); the high estrogen state of endometriosis was hypothesized to be a possible reason for this difference [40]. A prospective study of 207 women with past or present endometriosis which examined the relationship between BMD and concentrations of FSH and AMH in perimenopausal women with endometriosis found a weak negative correlation with FSH and a weak positive correlation with AMH, as would be expected in women without endometriosis. This study showed a lower-than-expected age-related AMH level in the women with endometriosis [41].

Due to the conflicting literature regarding endometriosis and osteoporosis, BMD should be assessed and monitored in accordance with current osteoporosis and POI guidelines [18,42].

5.2. Cardiovascular disease

Epidemiological studies have demonstrated an association between surgically confirmed endometriosis and subsequent atherosclerotic CVD (relative risk 1.62; 95% CI 1.39, 1.89). This association was more pronounced in younger women and those who had undergone hysterectomy and/or oophorectomy. The relative risks of developing hypercholesterolemia and hypertension were found to be 1.25 (95% CI 1.21, 1.30) and 1.14 (95% CI 1.09, 1.18), respectively, in women with surgically diagnosed endometriosis [43,44]. The Japan Nurses' Health Study found that women with endometriosis had 30% increased odds of hypercholesterolemia (95% CI 1.15, 1.47) [39]. A UK retrospective matched cohort study that included 56,090 women with endometriosis and 223,669 matched controls reported that the rates of incident ischemic heart disease, heart failure, and cerebrovascular disease were higher in women with endometriosis (adjusted odds ratio [aOR] 1.24; 95% CI 1.13, 1.37) [45]. In the same study, endometriosis was associated with a 1.40-fold (95% CI 1.22, 1.61) increased risk for ischemic heart disease and a 1.19-fold (95% CI 1.04, 1.36) increased risk for cerebrovascular disease. Additionally, women with endometriosis were found to have higher risks for myocardial infarction (OR 1.50; 95% CI 1.17, 1.98), angiography-confirmed angina (OR 1.91; 95% CI 1.59, 2.29), and the need for coronary artery bypass graft surgery (OR 1.35; 95% CI 1.08, 1.69), even after adjusting for demographic and lifestyle confounders. Other studies have found that women with endometriosis have lower flow-mediated dilation and a higher degree of endothelial dysfunction [46–48]. Surgical treatment of endometriosis significantly improves flow-mediated dilation two years post-operatively [49]. The regression of increased arterial stiffness and impaired flow-mediated dilation following the surgical treatment of endometriosis provides evidence of the association between atherosclerotic CVD and endometriosis [46,47]. Thus, screening and management of CVD risk in postmenopausal women with a history of endometriosis should follow guidelines [50].

5.3. Autoimmune disease

Endometriosis is associated with an increased rate of with autoimmune diseases (rheumatoid arthritis, systemic lupus erythematosus, Sjögren's syndrome, autoimmune thyroid disease, celiac disease, inflammatory bowel disease, multiple sclerosis), cardiovascular diseases, migraine, and malignancies [51]. Rheumatological conditions and fibromyalgia are known to worsen during the menopausal transition [51,52]. Thus, exacerbation of other inflammatory or autoimmune diseases coexisting with endometriosis may play a role in postmenopausal endometriosis [51,53].

5.4. Cancer

Women with endometriosis are at higher risk of both gynecological and non-gynecological malignancy.

5.4.1. Gynecological and breast cancer

A 2021 systematic review and meta-analysis found that endometriosis was associated with a higher risk of ovarian cancer, a slightly increased risk of breast cancer, and a lower risk of cervical cancer [54]. Twenty-six out of 49 population-based case-control and cohort studies showed a positive association between endometriosis and ovarian cancer risk (summary relative risk [SRR] 1.93; 95% CI 1.68, 2.22; based on 24 studies) that was strongest for clear cell (SRR 3.44; 95% CI 2.82, 4.42; based on 5 studies) and endometroid cancer (SRR 2.33; 95% CI 1.82, 2.98; based on 5 studies).

Risk of ovarian cancer is increased in women with a long-standing history (over 10 years) of ovarian endometriosis, those with recurrent endometriomas, endometriomas larger than 9 cm or exhibiting rapid growth, and in cases of newly developed endometriomas in women over 45 years of age [55–57].

The most common location of endometriosis in postmenopausal women is the ovary. Importantly, in these women, the pathognomonic "ground glass" appearance of endometriomas seen on transvaginal ultrasound is associated with a 44% risk of a malignant lesion and should prompt further investigation [8]. The absolute lifetime risk of developing ovarian cancer in women with endometriosis detailed in the 2022 ESHRE guidelines is increased from 1.3 per 100 in the general population to 2.5 [30]. A 2024 study using the Utah Population Database (1992–2019) [58] found that women with endometriosis have an increased risk of developing all types of ovarian cancer compared with those without endometriosis (overall risk 4.20; 95% CI 3.59, 4.91). The highest risk was observed in those with deep infiltrating endometriosis and/or ovarian endometriomas (adjusted hazard ratio [aHR] 9.66; 95% CI 7.77, 12.00). The strongest association was between deep infiltrating endometriosis and/or ovarian endometriomas and type I ovarian cancers, such as endometrioid, clear cell, mucinous, and low-grade serous cancers (aHR 18.96; 95% CI 13.78, 26.08). Thus, counselling regarding ovarian cancer risk and prevention should be considered for women with endometriosis.

Only a very weak association was noted between breast cancer and endometriosis (SRR 1.04; 95% CI 1.00, 1.09; based on 20 studies). Endometriosis was particularly associated with an increased risk of ER+/PR- breast cancer (aHR 1.90; 95% CI 1.44–2.50) [59]. Hormonal therapies used for endometriosis may also influence breast cancer risk [60]. Routine mammograms should be recommended to the patient, with additional imaging as warranted by personal and family history and physical exam.

5.4.2. Thyroid cancer

A 2021 systematic review found a robust association between endometriosis and thyroid cancer (SRR 1.39; 95% CI 1.24, 1.57; based on 5 studies) [54]. The 2022 ESHRE guideline highlighted the association between absolute thyroid cancer risk and endometriosis [30]. The absolute lifetime risk of developing thyroid cancer is 1.3 per 100 in the general population but 1.8 per 100 for women with endometriosis, and the latter group should be counselled about this [54]. Screening for thyroid cancer should be done according to national guidelines.

5.5. Mental health

Endometriosis has been associated with an increased risk for depression and anxiety. A 2022 systematic review and meta-analysis of 17 eligible studies found that women with endometriosis experienced significantly more symptoms of depression (standardized mean difference [SMD] 0.71; 95% CI 0.36, 1.06) and anxiety (SMD 0.60; 95% CI 0.35, 0.84) compared with healthy controls, but no differences were found in the comparison with women with chronic pelvic pain [61], which suggests that the symptoms of depression and anxiety in women with endometriosis are related to endometriosis-associated pelvic pain. Many other reviews and meta-analyses similarly suggest that endometriosis-associated pain is correlated with depression, anxiety, and decreased HRQoL [62]. However, some studies report that depression and anxiety may pre-date the diagnosis of endometriosis [63]. Additionally, a genetic predisposition between endometriosis, anxiety, depression, and eating disorders has been postulated [64].

The role of endometriosis-related infertility in the development of depression remains unclear; the correlated factors include age, quality of life, quality of sleep, fatigue, sexual function, gastrointestinal symptoms, comorbidity, self-esteem, emotional self-efficacy, coping style, social adjustment, pain imagery, and pain sensitization [65]. Perimenopause, but not postmenopause, is also independently associated with an increase in mood disorders, especially depression [66].

6. Management of menopause in women with endometriosis

6.1. Non-pharmacological interventions

6.1.1. Cognitive behavioral therapy

Cognitive behavioral therapy (CBT) is an alternative used by women who experience symptoms associated with the menopause who do not wish to take hormone therapy, or those for whom it may be contraindicated [67].

It shows promise in treating the physical and psychological symptoms of women with endometriosis and chronic pelvic pain, particularly the emotional aspects [68]. However, more structured studies with consistent, clear, and replicable methods are needed to establish a standardized psychological intervention protocol for women living with endometriosis.

6.1.2. Lubricants and moisturizers

Lubricants and moisturizers can be used alone or while taking systemic or topical estrogens. Lubricants are used during sexual activity to reduce vaginal dryness and discomfort. They may be based on water, oil, hyaluronic acid, or silicone. Lubricants provide temporary relief and may need repeated application. On the other hand, moisturizers can be used regularly, as they usually contain a polycarbophil-based polymer, which retains water and has a long-lasting relieving effect on the vagina [69]. A randomized trial comparing a vaginal estrogen cream and a hyaluronic acid suppository found that they were equally effective for the treatment of GSM [70].

6.1.3. Laser therapy

Laser therapy has gained interest as a treatment for GSM and vulvovaginal atrophy (VVA) [71,72]. The CO2 fractional and the non-ablative erbium YAG (Er:YAG) laser are the two main lasers used. Additionally, the diode vaginal laser has been introduced as a treatment option for GSM and has been found to be both effective and well tolerated [73]. However, randomized trial data are limited, and the studies are small and short term. Mension et al. in a randomized controlled trial (RCT) with a six-month follow-up found that vaginal laser treatment was safe but they observed no statistically significant differences in efficacy compared with the sham treatment group [74]. A 2023 review by a working group of the European Urogynecological Association (EUGA) concluded that both Er:YAG and CO2 vaginal lasers are safe energy-based therapeutic options for management of GSM and VVA in postmenopausal women and breast cancer survivors [72].

In 2018, the US Food and Drug Administration (FDA) warned against the use of energy-based devices for any vaginal "rejuvenation" or cosmetic vaginal procedure, or procedures intended to treat vaginal conditions and symptoms related to menopause, urinary incontinence, or sexual function [75]. The concern was that their safety and effectiveness had not been established for these indications. Subsequently, the International Urogynecological Association (IUGA) recommended that information from large, long-term, sham-controlled randomized studies is required to inform evidence-based practice regarding efficacy and safety [76]. Nevertheless, the Er:YAG laser was approved by Health Canada in August 2019 for use in GSM [77].

6.2. Pharmacological interventions

6.2.1. Hormonal preparations

6.2.1.1. Estrogens. Systemic MHT is considered the most effective therapy for vasomotor symptoms and urogenital atrophy, with potential beneficial effects on other menopause-related complaints, quality of life, and bone health [78]. In postmenopausal women with a history of endometriosis, MHT is effective for both natural and surgical menopause (Fig. 1). As the risk of recurrence and malignant transformation seems to

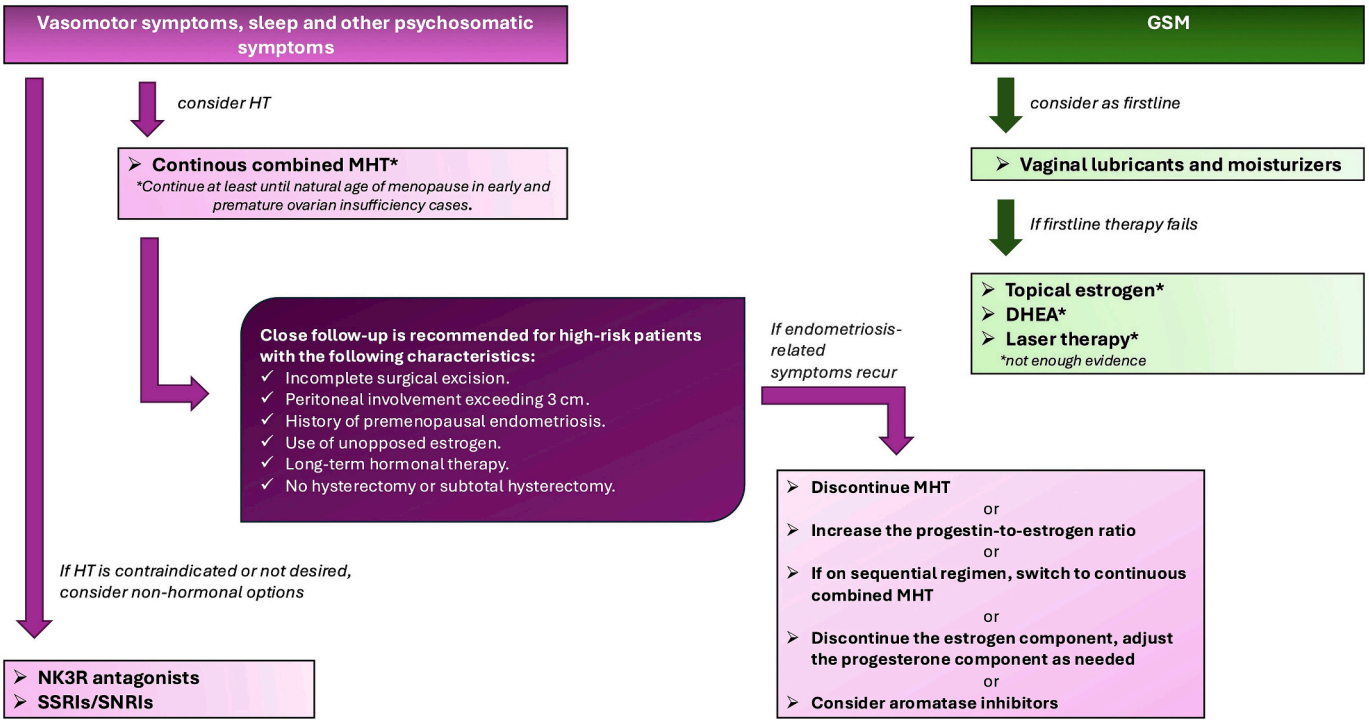

Fig. 1. Management of menopausal symptoms in women with endometriosis history
(**DHEA:** Dehydroepiandrosterone, **GSM:** Genitourinary syndrome of menopause, **HT:** hormone therapy, **MHT:** menopausal hormone therapy, **NK3R:** Neurokinin 3 receptor antagonists, **SSRI:** Selective-serotonin reuptake inhibitors, **SNRI:** Serotonin-norepinephrine reuptake inhibitors).

be associated with use of unopposed systemic estrogen, continuous progestogen addition is advised despite the small increase in breast cancer risk [79,80]. In any case, progestogen selection should be individualized according to CVD risk, presence of diabetes mellitus, and risk of breast cancer. Micronized progesterone and dydrogesterone have been associated with reduced CVD and breast cancer risk compared with other progestogens [81,82]. Recurrence of endometriosis and malignant transformation are discussed below.

Systemic MHT should be offered up to the age of natural menopause in women with premature or early menopause and thereafter further use discussed. Duration of use should be individualized based on a careful assessment of risks and benefits.

Topical estrogens can be used for GSM [83]. They can be used alone or combined with systemic MHT. Topical estrogens have minimal systemic absorption. They do not significantly increase the risk of endometrial hyperplasia or cancer [84,85]. Although direct evidence in endometriosis is lacking, topical vaginal estrogens are unlikely to pose a significant risk, based on safety data from other estrogen-sensitive conditions such as breast cancer [86].

The potential for exogenous estrogen stimulation to reactivate endometriotic foci or promote malignant transformation is still under investigation [79,87–89]. A venous plexus between the vaginal veins and the uterine and ovarian plexuses could direct compounds from the upper vagina to uterine tissue. To avoid a potential uterine first-pass effect, it is recommended that vaginal estrogens are inserted in the lower third of the vagina [90,91].

6.2.1.2. Tibolone. Tibolone is a synthetic steroid that is inert, but whose metabolites have estrogenic, progestogenic, and androgenic actions. It is classified as MHT. It is administered as an oral 2.5 mg tablet and has been used in women with endometriosis [92]. A National Danish Registry study found that tibolone is associated with a higher risk of endometrial cancer compared with that among women who have never used MHT [93]. Tibolone is not available worldwide and is not marketed in the USA.

6.2.1.3. Dehydroepiandrosterone (DHEA) and DHEA sulfate (DHEA-S). DHEA and DHEA-S are adrenal androgens which are converted into testosterone, dihydrotestosterone (DHT), and estrogens in peripheral tissues and upon oral administration. Vaginal DHEA is licensed in both the USA and Europe for the treatment of vulvar and vaginal atrophy in postmenopausal women with moderate to severe symptoms [94]. No studies have been undertaken in women with a history of endometriosis.

6.2.2. Non-hormonal preparations

Non-hormonal preparations are used for vasomotor symptoms and to maintain bone health.

6.2.2.1. Vasomotor symptoms. The non-hormonal neurokinin (NK)-3 receptor antagonist fezolinetant was approved by the US FDA and in Europe in 2023 and 2024 respectively for the treatment of moderate to severe vasomotor symptoms due to the menopause [95,96].

Five RCTs involving 3,302 patients showed that fezolinetant significantly reduced the daily frequency of moderate to severe vasomotor symptoms (weighted mean difference [WMD] − 2.36; 95% CI −2.92, −1.81) and significantly improved quality of life (WMD −0.42; 95% CI −0.58, −0.26) and sleep disturbance (WMD −1.10; 95% CI −1.96, −0.24) at 12-week follow-up. Additionally, the 30 and 45 mg daily doses of fezolinetant were efficacious and well tolerated [97].

Similarly, elinzanetant, a dual antagonist of NK-1 and NK-3 receptors, was found to reduce the frequency of vasomotor symptoms in a RCT at doses of 120 mg and 160 mg [98,99]. At the time of writing, elinzanetant has been approved by the Medicines and Healthcare products Regulatory Agency (MHRA) in the UK, but not by the European Medicines Agency (EMA) or the FDA [100]. Studies evaluating the effect of NK-3 receptor antagonists on the endometrium have demonstrated their safety [101]. The FDA issued a warning for fezolinetant, noting that it can cause serious liver injury, albeit rarely though it is reversible upon discontinuation of the medication [102]. As a result, liver function testing is required before prescribing fezolinetant, with follow-up testing recommended monthly for the first two months and subsequently at

months 3, 6, and 9.

Non-estrogen-based treatments for hot flushes include clonidine, selective serotonin reuptake inhibitors (SSRIs), serotonin and noradrenaline reuptake inhibitors (SNRIs), and gabapentin (Fig. 1) [103,104]. The efficacy and safety of herbal and botanical preparations are uncertain, and some may contain estrogenic compounds [104–106]. **Oxybutynin,** an anticholinergic agent traditionally used for overactive bladder, has shown effectiveness in reducing the frequency and severity of hot flushes in menopausal women [107].

6.2.2.2. Osteoporosis. Antiresorptive agents, such as bisphosphonates (alendronate, risedronate, and zoledronic acid), are first-line treatments for preserving bone mass in postmenopausal osteoporosis. However, limited data are available for younger women, and concerns remain regarding the safety of long-term use [108,109]. Calcium and vitamin D dietary intake may be insufficient, and supplementation is therefore recommended [42]. Anabolic agents, such as teriparatide, are options for postmenopausal women with severe osteoporosis who are intolerant of other treatments or who experience new fractures despite antiresorptive therapy [110].

7. Management of sexual dysfunction in postmenopausal women with endometriosis

Sexual dysfunction is frequently reported in postmenopausal women with endometriosis, commonly involving problems with desire, arousal, satisfaction, and pain during intercourse [111]. This dysfunction may result from both persistent endometriotic lesions and hormonal changes associated with menopause. Dyspareunia in postmenopausal endometriosis can be superficial—often related to VVA—or deep, typically linked to deep infiltrating endometriosis (DIE) affecting the uterosacral ligaments, rectovaginal septum, or posterior fornix. Therefore, a comprehensive evaluation in menopausal women is crucial to distinguish between hormonally driven atrophic changes and residual or reactivated endometriotic disease as underlying causes of intercourse-related pain.

7.1. Testosterone

Testosterone is used for the treatment of the hypoactive sexual desire disorder (HSDD). There is currently insufficient evidence to support its use for other conditions, or disease prevention [112]. A study examining the effect of oral testosterone on endometrial proliferation in healthy postmenopausal women found no evidence of endometrial stimulation with short-term use [113]. In a randomized placebo-controlled study of postmenopausal women receiving 12 weeks of vaginal testosterone or estrogen demonstrated endometrial safety of the hormonal preparations comparable to that of placebo [114]. However, no RCTs have been conducted to evaluate the safety of testosterone use concerning endometriosis recurrence. A single case report describes the de novo occurrence of endometriosis in a 64-year-old woman following the use of estradiol and testosterone for menopausal symptoms (duration unspecified), though no malignant transformation was reported [115].

It is important to note that in most countries testosterone therapy is prescribed off-label, with women either using modified doses of testosterone formulations approved for men or relying on compounded therapies [112].

7.2. Flibanserin

Flibanserin, a drug that selectively targets central serotonin postsynaptic receptors with agonistic effects on 5-hydroxytryptamine-1A (5-HT1A) and antagonistic effects on 5-HT2A receptors [116], has shown significant efficacy in improving sexual desire and satisfaction, and reducing distress in both pre- and postmenopausal women with HSDD, as demonstrated by a 2024 meta-analysis of 8 RCTs with a total of 7,906 participants [117]. While the improvements were consistent across various validated measures of sexual function and satisfaction, its use was associated with a higher incidence of mild adverse effects such as dizziness, fatigue, and nausea, although serious adverse events were comparable to those with placebo, suggesting a generally favorable safety profile. Flibanserin is approved for the treatment of HSDD in premenopausal women in the United States and in both premenopausal and postmenopausal women in Canada; however, it is not currently approved for use in Europe [118,119].

7.3. Bremelanotide

Bremelanotide, a melanocortin type 4 receptor dopaminergic agonist [120], was found to significantly improve both **sexual desire and arousal** in women with HSDD related to desire, arousal, or orgasm, but not related to pain conditions [121,122]. It also contributed to an overall improvement in total FSFI scores and reduction in sexual distress [121,122]. It is approved only in the United States [120].

7.4. Ospemifene

Ospemifene is a third-generation selective estrogen receptor modulator (SERM) which is used for the treatment of GSM-related symptoms, in particular vaginal dryness and dyspareunia. Endometrial safety has been shown in studies lasting up to 52 weeks [123]. In a review of 25 studies, ospemifene treatment demonstrated a high clinical symptom response, a favorable safety profile with minimal adverse events, a neutral impact on the endometrium, breast, bone, and thrombosis, and potential improvement in cardiovascular risk factors [124].

8. Management of endometriosis

Management of endometriosis includes general lifestyle advice, including referrals for counseling as needed, non-pharmacological and pharmacological interventions, and surgery. A personalized approach incorporating shared decision-making should be tailored to women's needs, the symptoms experienced, and imaging results, and—although less likely in the perimenopausal and postmenopausal age group—fertility concerns. The availability of individual medicinal products and/or devices varies worldwide, so local availability should be considered in decision-making. Additionally, this guide will not consider herbal supplements, botanicals, and alternative complementary therapies as there is a paucity of data regarding safety and efficacy. The potential for drug interactions or estrogenic activity of these compounds further discourages their use [105].

Whilst treating endometriosis, general care and screening such as for cancer (cervical, breast and colorectal), cardiovascular disease, and osteoporosis should be undertaken in accordance with national programs. In addition, monitoring should consider risk of recurrence or malignant transformation of endometriosis.

Postmenopausal women with symptomatic endometriosis should undergo surgical management to remove all visible endometriotic tissue due to the increased risks of recurrence and malignancy [8,30,125]. Medical therapy is reserved for cases where pain recurs post-surgery or surgery is contraindicated.

8.1. General and lifestyle advice

8.1.1. Pain management

Endometriosis may present with menstrual or non-menstrual pelvic pain or dyspareunia. In peri- and postmenopausal women, symptoms may include gastrointestinal or urinary symptoms. Management of endometriosis-associated pain should be individualized and aimed at the specific pain symptoms. Intermittent pain treatment may involve the use of analgesics, such as paracetamol or acetaminophen, and non-steroidal

anti-inflammatory drugs. Women with more chronic pain, especially if central sensitization is suspected, may benefit from tricyclic antidepressants, SSRIs and antiseizure therapies (e.g., gabapentin, pregabalin) [30]. In the pre- and perimenopausal population, hormonal therapies such as birth-control pills, oral progestins—such as norethisterone acetate (NETA), medroxyprogesterone acetate (MPA), dydrogesterone, and dienogest—levonorgestrel intrauterine systems (LNG-IUS) or GnRH agonists or antagonists may be considered [126,127]. In the postmenopausal population on MHT a progestogen should be added, even if the woman has undergone hysterectomy. In postmenopausal women with pain attributed to endometriosis who are not on MHT, aromatase inhibitors may enhance pain management and improve outcomes [30]. Women with dyspareunia who are being treated with estrogen-lowering medications, or in the peri- and postmenopausal period, should be evaluated and treated as needed for coexisting GSM with locally applied estrogen, DHEA, and/or lubricants and moisturizers. Referral for pelvic floor physical therapy or counseling can also be considered.

A stepwise approach or trialing different methods individually before using combinations should be considered. Opioids should be avoided as ongoing treatment and should be reserved for postoperative pain relief in women undergoing surgical treatment for disease. Pain management specialists may be consulted as needed.

8.1.2. Dietary changes and the microbiome

Dietary interventions show promise for the treatment, management, and prevention of endometriosis, particularly in pain management. The role of intestinal microbiota in the occurrence of endometriosis is still under debate. Many animal models have been used to explore the relationship between diet, nutrients, and endometriosis. These studies have highlighted the role of intestinal microbiota in regulating the immune system [128,129]. Significant changes in the intestinal microbiota have been observed following the induction of endometriosis in mice, regardless of diet [130]. Differences in the oral, gut, and vaginal microbiota have been documented in women with and without endometriosis, suggesting a potential role for microbial-based treatments [131].

The Mediterranean diet has shown promise in relieving pain symptoms, which may be due to its anti-inflammatory effects [30,132,133]. Specifically, fish rich in omega-3 fatty acids and extra virgin olive oil may be beneficial because of their antioxidant, polyphenol, and anti-inflammatory content [134].

Although sufficient evidence is lacking, a 2021 Australian survey found that dietary modifications are commonly used by women with endometriosis, with the greatest benefits observed for gastrointestinal symptoms [135]. Reducing or eliminating gluten, reducing or eliminating dairy products, and the low-fermentable oligosaccharides, disaccharides, monosaccharides, and polyols (FODMAP) diet were the most commonly reported dietary strategies [135]. However, a 2025 large international survey investigating dietary and supplement-based self-management strategies among individuals with endometriosis reported that the most popular dietary modifications included reducing alcohol (53.2% reported pain improvement), gluten (45.4%), dairy (45.2%), and caffeine (43.4%) [136]. In contrast, the low-FODMAP diet was less commonly used (32.1% reported benefit), and among those who used magnesium supplements, 32.3% experienced pain relief. These findings suggest that no single approach was universally effective, highlighting the need for well-designed RCTs to accurately assess the short- and long-term effectiveness and safety of various dietary strategies for managing endometriosis-related pain [132,136,137].

8.1.3. Exercise and smoking

Regular exercise is recommended to prevent midlife weight gain, preserve muscle mass, and as a general cancer-prevention strategy at the menopause [138,139]. However, there are insufficient data to draw firm conclusions about the effectiveness of exercise in relieving chronic pelvic pain or endometriosis-related pain [140]. A 2025 systematic review and meta-analysis (6 studies, 251 participants) demonstrated physical activity significantly improved quality of life in women with chronic pelvic pain secondary to endometriosis, particularly in the domains of pain ($P < 0.0001$), control and powerlessness ($P < 0.00001$), and emotional well-being ($P = 0.006$), but the conclusions were limited by both the quality and duration of the studies [141]. Given its overall health benefits and low risk of side-effects, exercise could be considered as part of a combined therapeutic approach in menopausal women with a history of endometriosis [30,142]. Smoking cessation is recommended at all points in a woman's life, and, as such, is also advised for menopausal women [138]. However, smoking has not been found to be associated with an increased risk of endometriosis [143].

8.2. Surgery

When treating postmenopausal women with a history of endometriosis it is important to consider the potential increased risk of underlying malignancy and the uncertainty of the diagnosis (Fig. 2). Recent comparative studies have suggested the superiority of hysterectomy and bilateral salpingo-oophorectomy for alleviating endometriosis-associated pain in premenopausal women [144]. Moreover, limited and low-quality evidence from five cohort studies indicates that surgical treatment may improve pain in postmenopausal women [30]. A significant proportion of postmenopausal women with endometriosis, particularly those with endometriomas, appear to have concurrent malignancy [30,54,79,88]. Therefore, in 2022 ESHRE recommended surgical excision of endometriosis from the ovaries and other locations to reduce the risk of ovarian cancer [30]. Furthermore, removing the affected ovary may offer a greater reduction in cancer risk compared with excising the disease or preserving the ovary. In a postmenopausal woman, bilateral salpingo-oophorectomy should be considered even if only one ovary is affected [145].

There are no robust data regarding the complications of surgery in postmenopausal women, but surgery for endometriosis is generally considered to be safe [30]. Potential complications such as chronic pain and ileus should alert clinicians to the risk of adhesion formation after endometriosis surgery, a serious issue affecting up to 90% of patients [146]. The benefits of surgical treatment, particularly in alleviating pain symptoms and reducing the risk of malignancy, appear to outweigh the potential complications of surgery.

Clinicians may consider surgery to enable histological confirmation of the diagnosis. Diagnostic workup and treatment should follow national oncology guidelines if a pelvic mass is detected. There remains no consensus on monitoring for the detection of malignancy, especially in asymptomatic patients.

8.3. Medical treatments

A medical approach to alleviate endometriosis-associated symptoms should be reserved for cases where surgery is contraindicated or when symptoms persist after surgery and malignancy has been excluded (Fig. 2) [8,30,147]. However, comprehensive data on the use of progestogens in postmenopausal endometriosis are still lacking, highlighting the need for further studies [30,148]. In premenopausal women, combined oral contraceptives (COCs), progestogens, the LNG-IUS, and GnRH antagonists (which may be combined with estradiol and norethisterone) may be used to alleviate pain. However, there are insufficient data for their use after the menopause [35].

8.3.1. Progestogens

Progestogen administration, either orally or via an intrauterine system, has been suggested as a viable alternative for patients unable to undergo surgery. However, efficacy data are lacking, and further studies are required to evaluate use in postmenopausal endometriosis [20,149]. Currently, there is no robust clinical evidence that micronized progesterone is as effective as synthetic progestins such as NETA or MPA in

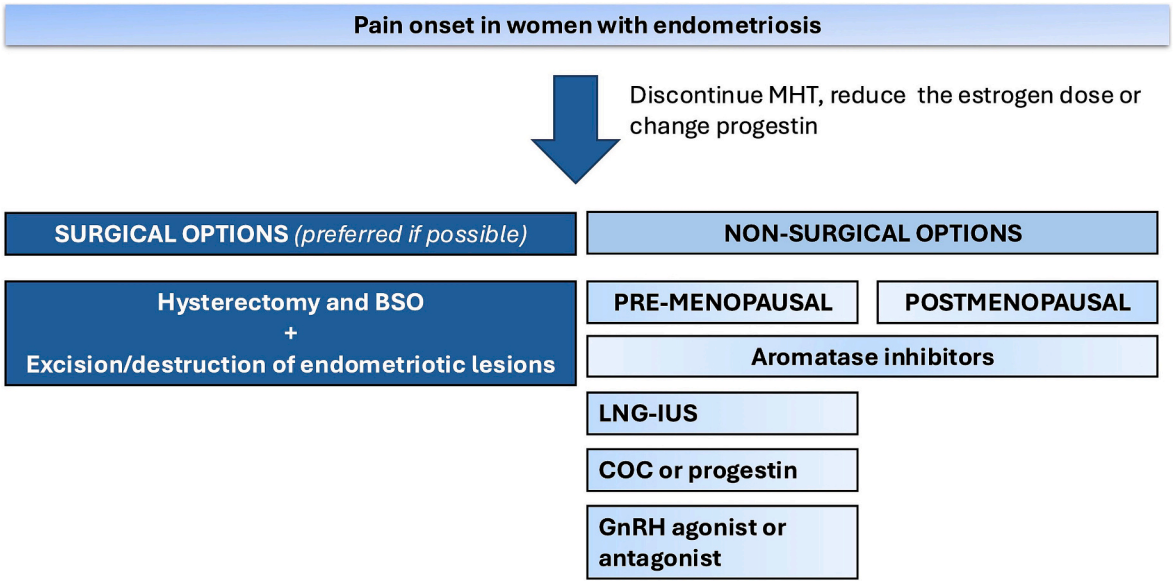

Fig. 2. Management of pain symptoms in postmenopausal women with endometriosis history
(**BSO:** bilateral salpingo-oopherectomy, **COC:** Combined Oral Contraceptive, **GnRH:** Gonadotropin-Releasing Hormone Agonists, **LNG-IUS:** Levonorgestrel-Releasing Intrauterine System, **MHT:** menopausal hormone therapy).

suppressing residual endometriotic lesions or preventing malignant transformation. While direct evidence is limited, expert consensus supports increasing the dose or changing the type of progestogen—particularly synthetic progestins—if symptoms of endometriosis recur under MHT. No studies currently support dose escalation of micronized progesterone in this setting.

8.3.2. Aromatase inhibitors

Aromatase inhibitors (AIs) could be effective in reducing endometriosis-associated pain in premenopausal women with severe endometriosis [150]. Evidence is limited to case reports in postmenopausal women [151]. For postmenopausal women with endometriosis-associated pain, clinicians may consider AIs, especially if surgery is not feasible. AIs, including anastrozole, letrozole or exemestane, are administered orally at 1-5 mg/day [152]. However, their use may increase the severity of hot flushes, vaginal dryness, and arthralgia, and may induce bone loss [151,152].

8.3.3. Tamoxifen

Tamoxifen, as a SERM, should be avoided because of the increased risk of endometrial malignancy [153]. Moreover, it has been reported that tamoxifen administration for breast cancer may lead to the progression and recurrence of endometriosis [154].

8.3.4. Emerging medical treatments: GnRH antagonists

Recent advancements in endometriosis management include the development of oral GnRH antagonists such as relugolix and linzagolix, which have demonstrated efficacy in reducing endometriosis-associated pain in premenopausal women [155,156]. Although these treatments represent a significant advance in non-surgical options, their use in postmenopausal women has not been studied and is currently not recommended, given the natural decline in ovarian hormone production after menopause.

9. Reactivation of endometriosis and malignant transformation

A major concern about the use of systemic MHT is the risk of reactivation or malignant transformation. Recurrence has mainly been reported in users of unopposed estrogen and less so with women taking combined MHT [79]. Furthermore, the manifestations of recurrence can be non-specific and variable. They include abdominal pain, vaginal bleeding, hematuria, and pelvic masses [79].

In 2009, a Cochrane review of studies of MHT in patients with endometriosis after surgical menopause identified two relevant RCTs, but of low-quality [92,157,158]. Both studies evaluated estrogen supplementation (transdermal 17β-estradiol or estradiol patches) combined with sequential progesterone supplementation (in the form of medroxyprogesterone acetate or micronized progesterone) [92,158]. In one study, the control group was treated with continuous tibolone (2.5 mg daily), while the other study did not administer a placebo or other interventions to the control group [92,158]. Statistically significant differences in pain or confirmed endometriosis recurrence were not demonstrated, most likely due to the small number of events in both studies [157]. Specifically, in the first trial pain recurrence occurred in 4 out of 10 patients in the intervention group and in 1 out of 11 in the tibolone group [92]; and in the second trial pain recurrence was documented in 4 (and confirmed in 2) out of 115 women in the MHT-treated group and in no cases in the control group [158]. It has been suggested that close monitoring is required because of the risk of recurrence in non-hysterectomized women, those who have undergone subtotal hysterectomy, long-term users of MHT and unopposed estrogen, as well as women with peritoneal involvement exceeding 3 cm or incomplete surgery [20,79,87,152,157,158].

A longitudinal study of 330 patients undergoing bilateral hysterectomy with bilateral salpingo-oophorectomy provided further evidence of endometriosis recurrence, recorded as pelvic pain, vaginal nodules, or bleeding [159]. The authors demonstrated recurrence rates ranging from 1.5% to 5.5% after a median follow-up of 6.0 years [159]. Similar to previous RCTs, no significant differences between MHT users and non-users were observed, even though recurrence occurred in the MHT user group (10 cases among 287 MHT users: 7 with estrogen-only therapy, 3 with combined estrogen–progestogen therapy, and none in the tibolone group) versus no cases among non-users (43 participants)] [159]. Discontinuation of hormonal supplementation or modification of the regimen (primarily adding progestogens to unopposed estrogen therapy) resulted in complete symptom resolution [159]. Therefore, management of recurrence in MHT users needs to be individualized and can include cessation of treatment, modulating the regimen (reduction of estrogen or increase in progestogen dose), or consideration of AIs.

A systematic review on malignant transformation of postmenopausal

endometriosis examined patient characteristics, MHT use, and outcomes over a period of 52 years (1969-2021) [88]. The review concluded that previous endometriosis, major definitive surgery before menopause, and long-term estrogen-only MHT were risk factors for malignant transformation of postmenopausal endometriosis. A case series found extragonadal disease was commonly associated with unopposed estrogen therapy [160].

There is no consensus on the frequency or type and duration of follow-up and monitoring [30]. The timing and diagnostic workup should be individualized based on previous and current treatments, disease severity, and symptoms. For women with previously identified endometriomas on imaging who have not undergone surgical excision, at least one pelvic ultrasound annually should be performed to monitor for increases in lesion size or other concerning changes [161]. Women with DIE may have a higher malignancy risk. An initial follow-up with magnetic resonance imaging (MRI) at 6 months is recommended, with intervals adjusted based on findings. Minimal changes may warrant imaging in 2–3 years, while significant changes require closer monitoring with repeat MRI after 6–12 months [161].

10. Conclusion and summary recommendations

Although endometriosis is a common disease, there is a paucity of data for the treatment of women after the menopause, which can be natural, induced, or premature. A major concern is disease reactivation or malignant transformation.

- A personalized approach should be tailored to women's needs and incorporate shared decision-making.
- Management can include general lifestyle advice, non-pharmacological and pharmacological interventions, and surgery.
- Estrogen-based hormone therapy is required in women with premature or early menopause until the average age of the natural menopause and should be considered thereafter in women with severe climacteric symptoms.
- Continuous combined estrogen–progestogen therapies are preferred in both hysterectomized and non-hysterectomized women as the risk of recurrence and malignant transformation of residual endometriosis may be reduced.
- Non-hormonal pharmacological treatments should be considered for climacteric symptoms or skeletal protection in women not taking MHT.

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