



EMAS consensus statement

Epilepsy and menopausal health: A European Menopause and Andropause Society (EMAS) and Centre for Global Epilepsy clinical guide

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ABSTRACT

Introduction: Menopause presents unique clinical challenges for women with epilepsy, with hormonal changes, antiseizure medication interactions, and comorbid health risks converging to potentially influence seizure susceptibility, treatment requirements, and long-term health outcomes.

Aim: To provide a clinical guide for the parallel assessment and management of epilepsy and menopausal health.

Materials and methods: Review of the literature and consensus of expert opinion.

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Results and conclusion: Women with epilepsy may be at increased risk of early menopause and are more likely to develop cardiovascular disease, osteoporosis, and cognitive impairment than women without epilepsy. There is a paucity of data regarding the optimal management of menopausal symptomatology and treatments for osteoporosis in women with epilepsy, since the condition is often an exclusion criterion in randomized controlled trials and observational studies. A personalized approach is required. Systemic menopausal hormone therapy (MHT) can be considered if the potential benefits outweigh the risks. The use of transdermal estrogen may be preferred to minimize dose-related fluctuations in hormone levels. Potential drug interactions between antiseizure medications and systemic MHT should be taken into consideration and alterations to antiseizure medication may be necessary. Healthcare providers should be appropriately trained to assess and manage epilepsy at the menopause and beyond, with provision of multidisciplinary teams.

1. Introduction

In 2025, the global population exceeded 8 billion, including more than 1 billion women aged over 50 years, and average female life expectancy in high-income countries reached approximately 83 years [1,2]. The menopause, or the cessation of menstruation, which occurs naturally at an average age of 51 years, is therefore becoming a midlife transition. However, menopause can occur much earlier, either naturally, with no identifiable underlying cause, or as a consequence of disease, surgery, radiotherapy or chemotherapy [3]. Menopause before the age of 40 years is commonly referred to as 'premature', while that which occurs in women aged 40–44 years is termed 'early' [3]. Loss of estrogen at menopause may lead to vasomotor and genitourinary symptoms [4]. In addition, menopause, especially when premature or early, is associated with significant long-term health risks, including increased risks of cardiovascular disease (CVD), osteoporosis, dementia, cognitive decline, and overall mortality [3,5].

Epilepsy is a chronic neurological disorder characterized by recurrent seizures, which may vary from a brief lapse of awareness or muscle jerks to severe and prolonged convulsions. Epilepsy is a major public health problem worldwide and over fifty million people are thought to be affected, of whom approximately half are women [6]. Most can participate fully in life and society if their seizures are controlled. The incidence of epilepsy rises after the age of 55 years, largely due to the increasing prevalence of age-related conditions, such as cerebrovascular disease, that lower the seizure threshold [7].

Epilepsy is associated with alterations in neuroendocrine function, and menopausal changes in sex steroid hormones may have additional physiological and clinical implications for people with epilepsy.

The European Menopause and Andropause Society (EMAS) and the Centre for Global Epilepsy have prepared this clinical guide to provide advice for all healthcare and allied healthcare professionals managing menopausal symptoms and long-term health in women living with epilepsy. The available evidence is of low certainty, and several recommendations in this guide rely on expert consensus. These should therefore be interpreted as pragmatic guidance rather than definitive, evidence-driven directives.

For brevity, the term "women" is used throughout this document; however, the guidance applies to all individuals with epilepsy who experience menopause, regardless of gender identity.

2. Epilepsy

2.1. Overview and terminology

Epilepsy is defined as a disorder of the brain characterized by an enduring predisposition to generate epileptic seizures [8]. It is diagnosed when an individual experiences two unprovoked seizures occurring more than 24 h apart, or after a single unprovoked seizure that carries an elevated estimated risk of recurrence (higher than 60% recurrence risk over the next 10 years) [8]. Epilepsy can also be diagnosed when there is a single seizure in the setting of an epilepsy syndrome. Epilepsy syndromes are defined by a characteristic combination

of seizure types, electroencephalogram (EEG) pattern, and distinctive clinical features [9,10]. Causes of epilepsy include structural brain abnormalities (e.g., stroke, cortical malformations); they can also be genetic, metabolic, infectious, or immunological. In some cases, the cause remains unknown [10].

Seizures can be generalized or focal. Generalized seizures arise from bilateral brain networks. They include generalized tonic-clonic seizures and absence seizures. Focal seizures originate in a specific region of the brain, and their features vary depending on the area involved. They may impair consciousness, and if the seizure activity spreads to both hemispheres, it can evolve into a bilateral tonic-clonic seizure, which resembles a generalized tonic-clonic seizure.

Epilepsy is associated with cognitive impairment. It may also adversely affect reproductive health [11], mental health [12], cardiovascular health [13], bone health [14], and quality of life [15]. It can also contribute to substantial social and occupational challenges and is associated with increased risk of mortality [16].

The interaction between sex steroid hormones and epilepsy is complex and bidirectional. Estrogen and progesterone modulate neuronal excitability and may therefore influence seizure susceptibility; conversely, epilepsy can disrupt sex steroid physiology [17]. Consequently, women with epilepsy have distinct clinical considerations across the lifespan, including during pregnancy, in the context of catamenial seizure patterns, and throughout the menopause transition.

2.2. Epilepsy management

Epilepsy is predominantly treated with antiseizure medications (ASMs), which modulate neuronal excitability. Typical first-line options include lamotrigine and levetiracetam. Around one-third of people with epilepsy are 'drug resistant', meaning that they are not free of seizures despite an adequate trial of two ASMs [18]. Further treatment options in some clinical scenarios include neuromodulation (e.g., vagal nerve stimulation) or resective/disconnective epilepsy surgery. For women with drug-resistant epilepsy, hormonal strategies are sometimes implemented, particularly for women with catamenial seizure exacerbations (see Section 7.2.3.3).

General lifestyle measures, such as maintaining regular, good-quality sleep, limiting alcohol intake, and avoiding illicit substances, are also important. Sleep deprivation, alcohol, and a range of recreational drugs are recognized precipitants of seizures due to their ability to lower the seizure threshold [19–22].

2.3. Global availability of care for epilepsy and menopause

Care for individuals with epilepsy and for those experiencing menopause remains inequitable from a global perspective. Many countries and regions lack the necessary clinicians, diagnostic tools, and medications to deliver safe epilepsy care. There can be large diagnostic gaps, treatment gaps, and education gaps. The World Health Organization (WHO) Intersectoral Global Action Plan (IGAP) on Epilepsy and Neurological Disorders is designed to improve care [23]. Epilepsy is the lead tracer condition within WHO IGAP and a groundswell of activities

across low- and middle-income countries (LMICs) are enabled through the IGAP Framework. These include upskilling primary healthcare workers in basic epilepsy care, development of new tools to help diagnose epilepsy, and engagement with policymakers to embed health-system change. The securing of levetiracetam on the WHO Model List of Essential Medicines is particularly important for women and girls, since it is among the ASMs with the lowest teratogenic risk based on current evidence and is hypothesized not to interact with menopausal hormone therapy (MHT). Whilst such recognition by the WHO is important, it is vital that levetiracetam is rapidly adopted onto national medicines lists and made widely available at an affordable price [24].

Menopause care guidelines vary worldwide. Healthcare may be delivered in primary care or secondary care settings by specialist services involving healthcare professionals [25]. Consultations may be undertaken by telemedicine as well as in person [26].

Women with epilepsy in the menopause transition require additional attention but there is a paucity of specialist services worldwide involving healthcare professionals with both menopause and epilepsy expertise.

3. Methodology

PubMed and Scopus were systematically searched using the text-word terms “epilepsy,” “menopause,” “hormone replacement therapy (HRT),” “menopausal hormone therapy (MHT),” and “antiseizure medication.” Articles published up to 22 July 2026 were eligible for inclusion. Original research articles, case reports, and review papers were considered, and only studies published in English were included.

Given the heterogeneity and limited volume of available literature, a narrative review approach was adopted. Because the evidence base is sparse and predominantly observational, the overall certainty of evidence was low. In areas where robust data were lacking, recommendations were informed by structured expert consensus, drawing on multidisciplinary clinical experience, and neuroendocrine principles to ensure clinical relevance and pragmatic applicability.

4. The menopause transition and epilepsy

Seizures and sex steroid hormones have a complex and bidirectional relationship. The sex steroid hormones present in the central nervous system are produced through both distant and local synthesis. Distant synthesis occurs through endocrine regulation and feedback mechanisms within the hypothalamic-pituitary axis, whereas local synthesis reflects de novo production within the brain [17].

Estrogen and progesterone are intricately linked with seizure susceptibility as they modulate neuronal excitability. In simplified terms, estrogen and progesterone have proconvulsant and anti-convulsant activities respectively. Conversely, epilepsy may also affect sex steroids by directly impacting ovarian sex hormone production [27]. Seizure activity, particularly within temporal-limbic networks, has been demonstrated to alter the pulsatile secretion of gonadotrophin-releasing hormone [22]. In addition, seizure activity may alter neuronal receptor sensitivity to sex steroids [28,29]. The intrinsic link between seizure susceptibility and sex steroid hormone regulation is demonstrated by a study reporting that frequent seizures are associated with menopause occurring three to four years earlier [29]. Furthermore, women with epilepsy may have a higher risk of early menopause, with one study estimating that 14% reach menopause before age 42 [30]. This is in contrast to the reported prevalence of non-iatrogenic premature ovarian insufficiency (POI) in the general population which varies from 1% in older studies to 3.5% in recent publications [3].

During perimenopause and menopause, changing sex steroid hormone levels may affect seizure burden. Only two human studies on the effect of menopause transition on seizure frequency were identified in the search of the literature [31,32]. Both were undertaken in the 1990s. One reported an increase in seizures during perimenopause and a

Table 1
Hepatic enzyme-inducing and nonenzyme-inducing antiseizure medication (ASM).

Enzyme-inducing ASM	Non-enzyme-inducing ASM
Carbamazepine	Brivaracetam
Cenobamate	Cannabidiol
Eslicarbazepine	Clobazam
Oxcarbazepine	Clonazepam
Perampanel > 12 mg	Ethosuximide
Phenobarbitone	Gabapentin
Phenytoin	Lacosamide
Primidone	Lamotrigine
Rufinamide	Levetiracetam
Topiramate	Perampanel < 12 mg
	Pregabalin
	Retigabine
	Tiagabine
	Valproate
	Vigabatrin
	Zonisamide

decrease after menopause in women with catamenial epilepsy, but no difference in seizure control in women without catamenial epilepsy [31]. However, the other study did not find a difference in seizure frequency in menopausal women compared with premenopausal women (regardless of the presence of catamenial epilepsy) [32]. Notably in this study, around 40% reported a worsening of seizures with menopause and 20% reported that epilepsy first began around the time of menopause.

The overall effect of menopause transition on the course of epilepsy may be further influenced by individual susceptibility to hormones, epilepsy type, age-related changes in ASM pharmacokinetics, sleep disturbance, and exogenous hormone therapy (see Section 7).

5. Health implications of menopause in women with epilepsy

Women with epilepsy are at increased risk of cardiovascular disease, osteoporosis, cognitive impairment, depression and anxiety, and reduced quality of life.

5.1. Cardiovascular disease

Epilepsy and menopause represent converging risk factors for cardiovascular disease.

Menopausal decline in estrogen results in adverse alterations in lipid profiles, endothelial dysfunction, and greater central adiposity [33]. Potential consequences of this include hypertension and accelerated atherosclerosis [33]. Poor sleep, which can be a feature of menopause, is also associated with increased cardiovascular risk [34].

People with epilepsy have approximately twice the cardiovascular mortality of the general population [13]. Epilepsy is also associated with increased rates of hypertension, dyslipidemia, arrhythmias, and structural heart disease [13]. ASMs, particularly hepatic cytochrome p450 enzyme-inducing drugs (Table 1), can further exacerbate metabolic and lipid abnormalities [35,36], thereby increasing cardiovascular risk. In addition, epilepsy-related autonomic dysfunction may contribute to adverse cardiovascular outcomes [37].

5.2. Osteoporosis

Estrogen is vital for bone health. Menopause is associated with a significant decline in bone mineral density at both the lumbar spine and hip, predisposing to increased risk of fractures.

Epilepsy is also associated with an increased risk of osteoporosis, through a number of mechanisms, with fracture rates reported to be two to six times higher than in the general population [14]. Seizures themselves may contribute to falls and fractures. Epilepsy may disrupt bone

homeostasis [38] and is an independent risk factor for the development of osteoporosis [39]. Both enzyme- and nonenzyme-inducing ASMs (Table 1) are associated with risk of osteoporosis [40]. Enzyme-inducing ASMs reduce vitamin D and decrease sex hormone levels (via an increase in sex hormone binding globulin). Smoking and physical inactivity – behaviors reported to be more prevalent in people with epilepsy [41] – and excessive alcohol consumption may further contribute to osteoporosis risk.

5.3. Dementia and cognition

There is a close, bidirectional relationship between epilepsy and cognition [42]. Many people with epilepsy report that the cognitive difficulties, particularly memory impairments, are more problematic than their seizures, noting that seizures are episodic but adverse effects on cognition are persistent. Several studies have shown that memory difficulties in those with seizures predate the initiation of ASMs, although some ASMs can have specific effects. For example, topiramate and zonisamide can affect naming and word-finding ability. Increasingly, it is becoming apparent that the intersections between epilepsy and memory are complex, encompassing sleep hygiene [43] and vascular risk factors [44].

Premature menopause and early menopause are associated with an increased risk of cognitive impairment and dementia [3]. In the menopausal transition, when memory and concentration concerns are already common, the intersections between epilepsy, sleep quality, and vascular risk factors become especially important. These overlapping contributors also offer therapeutic opportunities to support cognitive function in women with epilepsy during menopause.

5.4. Quality of life, mental health, and daily living

Both menopause and epilepsy are associated with anxiety, depression, and reduced quality of life [12,15,45]. The prevalence of depression and anxiety in women with epilepsy during the menopause, and the impact on quality of life, has not been studied.

5.5. Epilepsy and mortality

Epilepsy carries an increased risk of premature mortality, with reported standardized mortality ratios of between 2.2 and 3.6 in systematic reviews in adults [16]. SUDEP (sudden unexpected death in epilepsy) is the commonest epilepsy-related cause of death in most epilepsy populations [46], with an incidence of around 1.2 per 1000 adult patient-years [47]; effective control of generalized tonic-clonic seizures is considered central to reducing this risk [46]. Other causes include alcohol, drowning, pneumonia, suicide, cardiovascular disease, status epilepticus, and structural brain disease [47].

6. The workplace

People with epilepsy have higher rates of unemployment and typically have a lower salary than those without epilepsy. Potential reasons for this include stigma, employer discrimination, safety concerns, inadequate protective policies [48,49], and driving restrictions. Clinical factors also play a role; seizure control is identified as the strongest predictor of positive employment outcomes whilst cognitive impairment can have a negative impact on employment [50]. Cognitive impairment can be a direct consequence of epilepsy and may be a consequence of ASMs, particularly polytherapy [50] (see Section 5.3).

There is strong evidence that menopausal symptoms can diminish workplace confidence and wellbeing and may adversely affect job performance [51]. The implications of this for women with epilepsy, who already face an elevated risk of employment-related difficulties, have not been examined. However, it seems likely that challenges in sustaining employment may be compounded during the menopausal

transition for women with epilepsy.

Supportive workplace measures that benefit both employees with epilepsy and women experiencing menopause include organizational education and practical adjustments. Increasing staff understanding helps reduce stigma and fosters a more supportive workplace culture [52]. Reasonable adjustments may include flexible working hours to accommodate fatigue and optimize sleep, adjustments to break patterns, and access to well-ventilated spaces for individuals experiencing vasomotor symptoms.

7. Menopause assessment and management for women with epilepsy

7.1. Assessment

Assessment should include a comprehensive medical history covering seizure type and frequency, the presence of any catamenial seizure pattern, ASM use and its duration, menopause status, and severity of related symptoms. Both systemic and genitourinary symptoms should be evaluated [4,25,53].

Web-based tools, such as FRAXplus®, may be used for initial assessment of fracture risk [54]. Because epilepsy is not represented in this or any other tool, the risk may be underestimated. In view of this, it has been pragmatically suggested by bone health experts to select the ‘rheumatoid arthritis’ category in FRAXplus® as a surrogate risk factor [55].

Mental health assessment is a core element of a comprehensive epilepsy and menopause review, given the impact of mental health on symptom burden and overall wellbeing.

For women with epilepsy, it is particularly important to assess how menopausal symptoms are affecting sleep, as poor sleep increases seizure susceptibility [56]. Sleep quality should therefore be assessed, and strategies to improve sleep discussed.

Some, but not all, ASMs may induce the cytochrome P450 isoenzyme 3A4 (Table 1) [57]. Therefore, certain ASMs may accelerate hepatic metabolism of hormonal preparations and decrease serum concentrations of bioactive sex steroids. In addition, estrogens may reduce lamotrigine serum concentrations [58] (see Section 7.2.3.6).

7.2. Management

There is paucity of data regarding the optimal management of menopausal symptomatology and treatments for osteoporosis in women with epilepsy, since epilepsy is often an exclusion criterion in randomized controlled trials (RCTs) and observational studies. A personalized multidisciplinary approach should be tailored to the woman's preferences, her epilepsy history, other concomitant medical conditions (such as breast cancer), age at menopause, and menopause-related concerns.

There is a paucity of data regarding the safety and efficacy of herbal supplements, botanicals, and alternative complementary therapies, so no recommendations can be made. In addition, some products may contain compounds which can interact with ASMs and other therapies [59].

Management of menopausal symptoms includes lifestyle measures, nonpharmacological approaches, nonhormonal treatments, and hormonal therapies. Women with epilepsy require additional, tailored consideration, as some treatments can interact with ASMs and may influence seizure control (see Section 7.1).

7.2.1. General lifestyle measures

Smoking cessation, reducing alcohol consumption, and avoiding illicit substance use may significantly help menopausal health and wellbeing [19–22]. Attention should also be given to diet, exercise, and adequate calcium and vitamin D intake [4,25,53,60–62], so that supplements can be prescribed as required [63].

7.2.2. Nonpharmacological interventions

These include menopause-specific cognitive behavioral therapy, either with or without menopausal hormone therapy (MHT) [4,25,53]. Regarding the genitourinary syndrome of the menopause (GSM), non-pharmacological interventions include lubricants, moisturizers, and hyaluronic acid [64].

7.2.3. Pharmacological interventions

7.2.3.1. Systemic and vaginal hormonal preparations. A variety of estrogen-based and non-estrogen-based hormonal medications are available [4,25,53]. For hysterectomized women, systemic MHT consists of estrogen-alone preparations; for nonhysterectomized women, progestogen is added, to reduce the increased risk of endometrial hyperplasia and carcinoma. The routes of administration can be oral, transdermal (patches, gels, and sprays), subcutaneous (implants), vaginal, and intrauterine (for levonorgestrel). Systemic and low-dose vaginal estrogens may be used together [64]. Intrauterine levonorgestrel also provides contraception. The availability of different preparations varies worldwide.

Testosterone and other hormonal therapies are discussed in Section 7.2.3.5.

The duration of systemic therapy depends on the end-points of treatment. Thus, for women with premature menopause, it is recommended that treatment is continued at least until the average age of the natural menopause (i.e. early 50s) and then reassessed [3,4]. Some women may have vasomotor symptoms for many years, into and beyond the seventh decade. With regard to osteoporosis, bone loss will resume on stopping MHT and a long-term strategy is required based on estimates of fracture risk. There are no arbitrary limits regarding the duration of use of MHT – it can be used for as long as the woman feels the benefits outweigh the risks for her, and decisions must be made on an individual basis [4,25,53].

7.2.3.2. Current evidence base for systemic MHT in epilepsy

7.2.3.2.1. Animal studies. There is mixed evidence from studies that have investigated the effects of estrogen in ovariectomized rats, as summarized in a systematic review published in 2023. Among nine studies identified, four reported an increase in seizures following estrogen administration, four reported a decrease, and in one study there was no effect [65].

7.2.3.2.2. Human studies. Only three studies and one case report have investigated the effect of systemic estrogen-based MHT on seizure control in women with epilepsy and all are over 20 years old [65]. Of the three studies, one was a randomized controlled trial [66] and the other two observational [31,32]. The case report described a woman using transdermal estradiol and an oral progestogen [67].

The placebo-controlled randomized trial recruited postmenopausal women aged 45–62 years [66]. The study stopped after 3 months due to safety concerns raised by the Women's Health Initiative study. It examined the effect of MHT consisting of 0.625 mg of conjugated equine estrogen (CEE) plus 2.5 mg of medroxyprogesterone acetate (MPA) daily ($n = 8$), or a double dose of CEE/MPA ($n = 7$). There were six women in the placebo group. MHT was associated with a dose-dependent increase in seizure frequency of the individual's 'most severe seizure type'. In the single-dose arm, 37.5% had worsening seizure frequency, compared with 71.4% in the double-dose arm ($P = 0.008$) [66]. Given the study's short duration and limited power, the dose-dependent effect observed requires confirmation in larger cohorts.

The observational studies consisted of a questionnaire and an interview study. In the questionnaire study, of the 16 menopausal women who took MHT, 10 (63%) reported an increase in seizures, compared with three (12%) of the 26 women who did not ($p = 0.001$) [31]. There was no association between MHT use and increased seizures in perimenopausal women ($n = 8$). In an interview study, of the 31 women

with epilepsy who had experience with MHT, 22 (71%) noted no change in their seizures while receiving treatment, five (16%) reported some improvement in their seizures, and four (13%) described worsening [32]. The authors noted that the types and doses of MHT prescribed varied considerably, and many of the women could not identify exactly what had been prescribed, thus limiting analysis. Both observational studies are limited by recall bias and small sample size.

The case report describes the effect of weekly transdermal estradiol (0.1 mg/day) with subsequent addition of medroxyprogesterone acetate (2.5 mg daily) over a period of 6 months in a 51-year-old postmenopausal woman with epilepsy who presented with frequent vasomotor flushing [67]. MHT was associated with a decreased incidence of seizures, cessation of vasomotor flushing, improved sleep, and a positive impact on lipid profile.

It should be noted that MHT formulations and delivery methods have evolved considerably over time. These studies undertaken over 20 years ago evaluated preparations that are no longer routinely used, limiting the applicability of those past findings to modern therapies.

7.2.3.3. Summary and clinical considerations regarding systemic MHT in epilepsy. The current evidence base is insufficient to determine the effect of systemic MHT on seizure frequency in women with epilepsy. In women who require management of menopausal symptoms, MHT may be considered following a comprehensive, individualized evaluation of potential risks and benefits. This assessment should incorporate the possible therapeutic advantages of MHT for sleep disturbance, mood symptoms, and osteoporosis prevention, all of which are particularly relevant for women with epilepsy [4,25,53,68]. Where a shared, informed decision is reached to initiate MHT, the use of transdermal estrogen is preferred to minimize dose-related fluctuations in hormone levels. Close monitoring for any subsequent changes in seizure frequency is advised throughout treatment.

Randomized controlled trials are needed to study progesterone and progestogens and their route of administration in combined MHT regimens. Transdermal and intrauterine progestogens may be preferred, to avoid hormone fluctuations. The latter also provide contraception. Micronized progesterone may be preferred over synthetic progestogens in women with epilepsy because it is metabolized to allopregnanolone, enhancing GABA-A mediated neuronal inhibition. Clinical evidence supporting this approach is limited, but natural progesterone has shown benefit in selected women with catamenial epilepsy characterized by progesterone withdrawal-related seizure clustering, mainly in a secondary analysis of a randomized controlled trial and in uncontrolled studies [27].

Consideration must also be given to potential drug interactions between systemic MHT and ASM (see Section 7.2.3.6).

7.2.3.4. Low-dose vaginal MHT. Women who take low-dose vaginal estrogen alone do not need to take a progestogen [69]. The dose of estrogen in licensed vaginal preparations is very low, leading to minimal systemic absorption, with circulating estrogen levels remaining in the postmenopausal range. Although no studies have evaluated their use specifically in women with epilepsy, the low systemic exposure suggests there is no clear reason they cannot be used in the usual way.

7.2.3.5. Other hormonal preparations. These include oral tibolone for vasomotor symptoms and sexual dysfunction; testosterone for sexual problems; and vaginal dehydroepiandrosterone and oral ospemifene for genitourinary symptoms [4,25,53,64]. In 2019, the Global Consensus Position Statement on the Use of Testosterone Therapy for Women concluded that restoration of testosterone to premenopausal levels improves sexual function in naturally or surgically postmenopausal women [70]. The 2024 National Institute for Health and Care Excellence (NICE) guideline on the diagnosis and management of menopause recommends considering testosterone supplementation for women with hypoactive

sexual desire disorder/dysfunction (HSDD), if MHT alone has been ineffective [25]. In the absence of female preparations, women are prescribed male preparations, but with dose modification. There is no literature on the potential impact of these other preparations on seizures in women with epilepsy. Data from animal studies and limited evidence in men suggest that testosterone may have either proconvulsant or anticonvulsant effects, depending on the balance of its metabolic pathways within the brain. Similarly to the MHT literature in epilepsy, these studies are over 20 years old [71–74].

7.2.3.6. Impact of systemic menopausal hormone therapy on antiseizure medications. There is a bidirectional relationship between ASM and sex steroid hormones. The evidence base for this is largely from studies conducted in pregnancy. However, the evidence about the potential interaction between ASM and MHT is sparse. ASMs that induce hepatic enzymes (Table 1) can increase sex steroid hormone metabolism. Levels of enzyme-inducing ASM and lamotrigine (one of the most commonly prescribed ASMs) are known to be decreased by circulating estrogen. However, there are limited studies examining in vivo effects of MHT on lamotrigine levels or other ASMs. One study has reported a decreased concentration to dose ratio in MHT users [58]. The randomized controlled trial described above reported that two women taking lamotrigine had a decrease in lamotrigine levels of 25–30% while taking 0.625 mg of conjugated equine estrogens plus 2.5 mg of medroxyprogesterone acetate [66]. In one of the women, the decrease in lamotrigine level was associated with seizure increase, but in the other there was no change in seizures.

For women with epilepsy who opt to use MHT, we propose that pretreatment trough lamotrigine serum levels are obtained followed by trough levels around four weeks after treatment initiation to guide potential lamotrigine dose increases. Dose changes should be individualized and undertaken under guidance of the treating neurologist. If lamotrigine has been increased to compensate for reduced levels during MHT use, it is important to review and potentially reduce the dose once MHT is discontinued, to minimize the risk of adverse effects.

7.2.4. Nonhormonal preparations

Nonhormonal treatments can be used for menopausal symptoms and osteoporosis by women who have concerns about or contraindications (e.g. a history of breast cancer) to hormone-based therapy. It must be underlined that epilepsy is often an exclusion criterion in clinical trials.

7.2.4.1. Nonhormonal preparations for menopausal symptoms. Nonhormonal agents for vasomotor symptoms include selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs), although they are less effective than systemic MHT [4,25,53]. In a Cochrane review, SSRIs did not increase seizure frequency [75]. Clonidine, gabapentin, and pregabalin may be also considered for the treatment of vasomotor symptoms, although they are of moderate efficacy [4,25,53]. Gabapentin and pregabalin also have antiseizure effects and as such may be good options for women with focal epilepsy, although these medications can worsen seizures in generalized epilepsy [76].

Neurokinin receptor antagonists such as fezolinetant and elinzanetant constitute a novel group of non-estrogen-based medications for the management of vasomotor symptoms [4,77]. Neurokinin B normally stimulates the hypothalamic thermoregulatory center. Neurokinin receptor antagonists are effective only for vasomotor symptoms, and liver function testing is recommended [78,79]. Fezolinetant has not been studied in women with a history of seizures or other convulsive disorders. There were no cases of seizures or convulsive disorders during clinical studies [78]. A seizure was reported in one patient with a history of seizures in the clinical trials of elinzanetant [79]. Postmarketing cases of seizures have been reported in patients treated with elinzanetant [79]. There are potential interactions with enzyme-inducing ASMs,

particularly because elinzanetant is metabolized via CYP3A4 and is affected by CYP3A4/P-gp inducers. Until epilepsy-specific clinical data become available, these agents should be used with caution in women with epilepsy.

7.2.4.2. Nonhormonal preparations for osteoporosis. Regarding postmenopausal osteoporosis, the current armamentarium includes both anti-osteoclastic (i.e., selective estrogen receptor modulators, such as raloxifene and bazedoxifene, bisphosphonates and denosumab) and osteoanabolic agents (i.e., parathyroid hormone (PTH) and PTH-related peptide analogues, such as teriparatide and abaloparatide, respectively) [80]. Romosozumab is a high-potency agent which exerts a dual anti-osteoclastic and osteoanabolic effect [80]. The choice of an anti-osteoporotic agent depends on the patient's baseline 10-year risk of fracture, providing priority to bone-formation agents in patients at very high risk, according to international guidelines [80,81].

There are no data regarding the use of anti-osteoporotic medications in postmenopausal women with epilepsy. Evidence is limited to one randomized trial undertaken in 80 male veterans with ASM-treated epilepsy. It showed significant improvement of bone mineral density at the lumbar spine with risedronate plus calcium/vitamin D, compared with the placebo, after two years of treatment. The use of risedronate also prevented the incidence of new vertebral and nonvertebral fractures [82]. In addition, an observational study showed a lower 3-year risk of mortality among adults with epilepsy who used anti-osteoporosis medications (83% bisphosphonates) [83].

8. Contraception

The likelihood of unintended pregnancy is increased by the misconception that perimenopausal women are infertile. Women in heterosexual relationships should be advised by healthcare professionals to use contraception if they do not wish to become pregnant. The Faculty of Sexual and Reproductive Healthcare (FSRH) guidelines support the use of long-term and emergency contraception in women over 40 [84]. In general, all women can cease contraception at age 55 as spontaneous conception after this age is exceptionally rare, even in women still experiencing menstrual bleeding [84].

Women with epilepsy may experience particular challenges in contraceptive use, and potential pharmacological interactions between hormonal contraceptives and ASMs must be considered [85]. Enzyme-inducing ASMs (Table 1) accelerate hepatic metabolism of contraceptive hormones, which can reduce their effectiveness. Lamotrigine appears to modestly reduce circulating levels of contraceptive progestogens; however, the impact of this on the effectiveness of combined hormonal contraceptives and progestogen-only contraceptives is not fully understood. It remains possible that lamotrigine could reduce the effectiveness of hormonal contraception. Conversely, estrogen in combined hormonal contraceptives induces glucuronidation of lamotrigine, significantly lowering serum lamotrigine levels, which may reduce seizure control.

9. Epilepsy in assisted living or other long-term care environments

Long-term care and support may be required as women age due to disability or functional limitations. Epilepsy is more common in nursing home settings, where the point prevalence has been reported to reach 7.7%, compared with 5.4% in general older populations [86]. New-onset epilepsy in older adults typically arises in the context of comorbidities such as stroke or dementia. Seizures in older adults may be subtle or atypical in presentation, making under-recognition a potential issue, especially when communication is limited or there are behavioral changes [7].

Management of epilepsy in this group is complicated by

MANAGEMENT OF MENOPAUSE IN EPILEPSY

Key considerations

POTENTIAL SEIZURE VULNERABILITY

Menopause transition may increase seizure risk due to:

- Sex hormone changes
- Interactions between MHT and antiseizure medication (ASM)
- Sleep disturbance

SYMPTOM MANAGEMENT

Lifestyle: aim for good quality sleep and stress reduction

Non-hormonal options should be considered

MHT: not contraindicated in epilepsy and can be considered when the individualised benefits outweigh risks. Transdermal routes are preferred.

DRUG INTERACTIONS

Lamotrigine + MHT: May need higher dose of lamotrigine. Consider monitoring lamotrigine levels.

Enzyme inducing ASM + HRT: May lower oestrogen.

BONE HEALTH

Epilepsy and ASM increases osteoporosis risk.

Assess bone health, provide lifestyle advice, advise vitamin D and arrange DEXA scan when indicated.

MENTAL HEALTH & COGNITION

Women with epilepsy already experience higher rates of cognitive and mood symptoms.

Menopause can add a further burden, with overlapping effects on memory, concentration, and mental health.

CONTRACEPTION

Contraception remains essential in perimenopause.

Women with epilepsy need tailored options because ASMs can alter contraceptive effectiveness and hormone levels.

Fig. 1. Management of epilepsy and menopausal health: Key considerations.

polypharmacy and age-related alterations in pharmacokinetics. This necessitates careful selection of well-tolerated ASMs and ongoing monitoring for long-term adverse effects [87].

Education of nursing home staff using interactive videos and teaching modules has been suggested to improve seizure identification [87]. Complementing this with individualized seizure care plans may also reduce unnecessary emergency visits in this patient group [87].

10. Summary recommendations

Menopause presents complex clinical considerations for women with epilepsy and requires individualized, shared decision-making [88] informed by local pathways and guidance. The following recommendations should be interpreted within this clinical framework (Fig. 1).

- Women with epilepsy should be provided with information about the potential health impacts of the menopause.
- Assessment and management need to be individualized.
- Lifestyle advice should be provided to optimize menopausal symptoms and long-term health.
- The benefits of any pharmacological interventions must be balanced against potential risks for that individual.
- When systemic MHT is prescribed, transdermal estrogen is preferred, to minimize dose-related fluctuations in hormone levels.
- Consideration should be given to the potential drug interactions between ASMs and MHT, and alterations to lamotrigine doses may be necessary.
- There is a need for multidisciplinary management worldwide, with specialist training of healthcare professionals.

CRediT authorship contribution statement

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