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Review

Contemporary Management of Menopausal Vasomotor Symptoms: An Evidence-Based Review of Hormonal and Non-Hormonal Interventions

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Abstract

Objective: To synthesize current evidence regarding the pathophysiology, clinical evaluation, and global management guidelines for vasomotor symptoms (VMS), providing a structured, cross-cultural approach to personalized menopause care. **Mechanism:** Central pathophysiology is driven by midlife estrogen depletion, which induces hypothalamic noradrenergic hyperactivity and neurochemical imbalance alongside hyperactive kisspeptin, neurokinin B, and dynorphin (KNDy) neurons in the arcuate nucleus. Excessive neurokinin B stimulates neurokinin 3 (NK3) receptors, resulting in a structurally narrowed thermoneutral zone and acute vasomotor instability. **Findings in brief:** Diagnosis remains primarily clinical. Menopausal Hormone Therapy (MHT) is established as the most effective treatment, particularly when initiated within the "timing window" (aged <60 years or within 10 years of onset), utilizing the lowest effective dose and transdermal administration to mitigate thromboembolic risks. For patients with contraindications, evidence-based non-hormonal options—including selective serotonin reuptake inhibitors (SSRIs), serotonin-norepinephrine reuptake inhibitors (SNRIs), gabapentinoids, newly Food and Drug Administration (FDA)-approved NK3 receptor antagonists (e.g., fezolinetant), and structured plant-based dietary restructuring—offer substantial symptom reduction. Additionally, behavioral therapies such as Cognitive Behavioral Therapy (CBT) and clinical hypnosis serve as valuable adjuncts for improving quality of life. **Conclusions:** Effective clinical management of menopausal symptoms requires moving away from standardized one-size-fits-all protocols. By integrating lifestyle modifications, targeted pharmacotherapy, and patient preferences, clinicians can optimize efficacy, and overall quality of life throughout the menopausal transition. This review underscores the importance of using the lowest effective dose and favoring transdermal administration to mitigate risks, thereby supporting a comprehensive, evidence-based approach to the menopausal transition.

Keywords: menopause; vasomotor symptoms; menopausal hormone therapy (MHT); KNDy neurons; personalized medicine

1. Introduction

Menopause, a term derived from the Greek "menos" (month) and "pausis" (cessation), represents a critical and definitive physiological milestone in a woman's life, clinically defined as the permanent cessation of menstruation following 12 consecutive months of amenorrhea. Although the mean age at natural menopause is approximately 51 years, its symptomatic precursor, perimenopause, can begin up to a decade earlier, introducing a spectrum of physiological changes driven by dynamic endocrine fluctuations [1].

Among the most characteristic of these changes are vasomotor symptoms (VMS), primarily hot flushes and night sweats, which affect an estimated 50% to 80% of women worldwide. A hot flash is not merely a sensation of warmth but a complex neurovascular event characterized by a sudden, intense wave of heat that typically originates in the chest and rapidly spreads to the neck and face. This event is accompanied by measurable physiological alterations: a sharp rise in circulating luteinizing hormone and epinephrine—a potent stimulator that increases heart rate, cardiac output, and systolic blood pressure—coinciding with a decline in norepinephrine. The episode culminates in profuse sweating, peripheral vasodilation, a marked increase in skin temperature (up to 6 °C in the fingers), and a slight paradoxical decrease in core body temperature. The experience is often preceded by an "aura" and results in profound discomfort, embarrassment, restlessness, and chronic sleep disruption, collectively establishing VMS as the most prevalent and distressing hallmark of the menopausal transition [2,3].

The therapeutic management of this transition is complicated by both clinical controversies and structural gaps in care. Central among these is uncertainty regarding the long-term safety of menopausal hormone therapy (MHT). Following the premature termination of the Women's Health Initiative (WHI) trial in 2002, concerns over increased cardiovascular and breast cancer risks precipitated a global decline in MHT prescriptions. Modern endocrinology has challenged this perspective through the "timing window hypothesis", demonstrating that starting MHT in symptomatic women under 60 or within 10 years of menopause onset confers a favorable cardiovascular profile. Nevertheless, debate persists regarding the optimal timing for discontinuation, long-term oncological risks of combined regimens, and the best approach to transitioning to non-oral formulations [4].

Second, the therapeutic landscape for non-pharmacological and lifestyle interventions is characterized by highly conflicting evidence. Although clinical trials of behavioral therapies, such as cognitive behavioral therapy (CBT) and clinical hypnosis, show robust, reproducible improvements in subjective symptom burden, sleep quality, and overall quality of life, they often fail to demonstrate objective reductions in hot flash frequency or core temperature variations. Conversely, widely popularized complementary approaches like acupuncture, black cohosh, and unstandardized herbal remedies yield findings that are frequently confounded by pronounced placebo effects, generating significant friction between patient-driven demand and evidence-based clinical guidelines [5].

Ultimately, these therapeutic challenges are intensified by notable geographical and ethnocultural differences in how symptoms are reported, diagnosed, and treated across East Asian and Western populations. Epidemiological studies show a clear divide: while up to 80% of women in North America and Europe report vasomotor symptoms, significantly lower reporting rates are consistently observed among East Asian populations [6,7]. This discrepancy is not merely biological; it results from a complex interplay of factors, including higher dietary intake of phyto-estrogens in traditional Asian diets, variations in metabolic processes, and cultural perspectives that often frame the menopausal transition around healthy aging or general vitality rather than a discrete medical condition [6,7].

Consequently, Western clinical guidelines rely heavily on early, targeted pharmacotherapy, whereas East Asian clinical practices favor herbal formulations, lifestyle integration, and dietary modifications. This review aims to synthesize current understanding of the underlying pathophysiological mechanisms, critically address these clinical controversies, and propose a balanced, cross-cultural framework for personalized menopausal care.

2. Clinical Evaluation

Women aged 40–50 years experiencing classic hot flushes generally do not require laboratory testing, as diagnosis is based primarily on clinical history. However, alternative causes such as hyperthyroidism, pheochromocytoma, medication side effects, or alcohol use should be excluded when clinically suspected. During a flush episode, thermography can detect significant peripheral vasodilation, with absolute finger and toe skin temperatures typically measuring in the range of 20–33 °C (representing a localized extremity temperature rise of up to 6 °C), alongside minor fluctuations in core body temperature [8]. Although follicle-stimulating hormone (FSH) and luteinizing hormone (LH) levels may be within normal ranges during menopausal transition, their variability during perimenopause limits their diagnostic value, especially in women over 45 years. Clinicians must remain vigilant for atypical or severe symptoms, considering a broad differential diagnosis like thyroid dysfunction, rare conditions such as pheochromocytoma or carcinoid syndrome, chronic infections, or medication effects from agents such as tamoxifen. Although thermography objectively demonstrates peripheral temperature fluctuations during a hot flash, current guidelines, including those of the North American Menopause Society, emphasize that diagnosis relies primarily on a detailed clinical history, which remains the gold standard for evaluation [8].

3. Pathophysiology of Hot Flushes

The classic thermoregulatory hypothesis proposes that menopausal hot flushes result from a severely narrowed thermoneutral zone within the hypothalamus. In asymptomatic individuals, core body temperature fluctuates within a safe range of approximately 0.4 °C without triggering physiological heat-dissipation mechanisms. However, contemporary integration of this model reveals that midlife estrogen depletion directly destabilizes central noradrenergic circuits, increasing central norepinephrine synthesis while simultaneously suppressing inhibitory serotonergic and endorphinergic pathways. This neurochemical imbalance forces the hypothalamic thermostat to overreact to minor fluctuations in core temperature, triggering peripheral vasodilation and compensatory sweating to dissipate heat prematurely [9].

Recent neuroendocrine advances have expanded this paradigm by identifying a specialized population of hyperactive KNDy neurons within the hypothalamic arcuate nucleus as the primary upstream trigger for vasomotor instability. Hypoestrogenism removes the inhibitory endocrine feedback on these neurons, causing cellular hypertrophy and excessive local release of neurokinin B. This peptide surge acts on neurokinin 3 (NK3) receptors in the adjacent medial preoptic area, directly causing the acute structural narrowing of the thermoneutral zone. Rather than treating these two concepts as competing theories, the present review proposes an integrative perspective that combines them within a Neuro-Endocrine-Metabolic Thermoregulatory Axis [10,11].

To advance the field of menopausal science, the present review proposes a comprehensive research framework that maps the ways in which systemic physiological inputs intersect with this central hypothalamic axis. This conceptual model views the hypothalamus not as an isolated circuit, but as a dynamic integrative hub that incorporates central neurochemical shifts, systemic endocrine depletion, and peripheral metabolic signals. Through this unified multi-system approach, researchers can evaluate how systemic metabolic markers, body fat composition, and sustained nutritional patterns directly modulate central KNDy neuron firing rates. This framework successfully bridges the longstanding conceptual divide between central neuroendocrine mechanisms and clinical lifestyle outcomes.

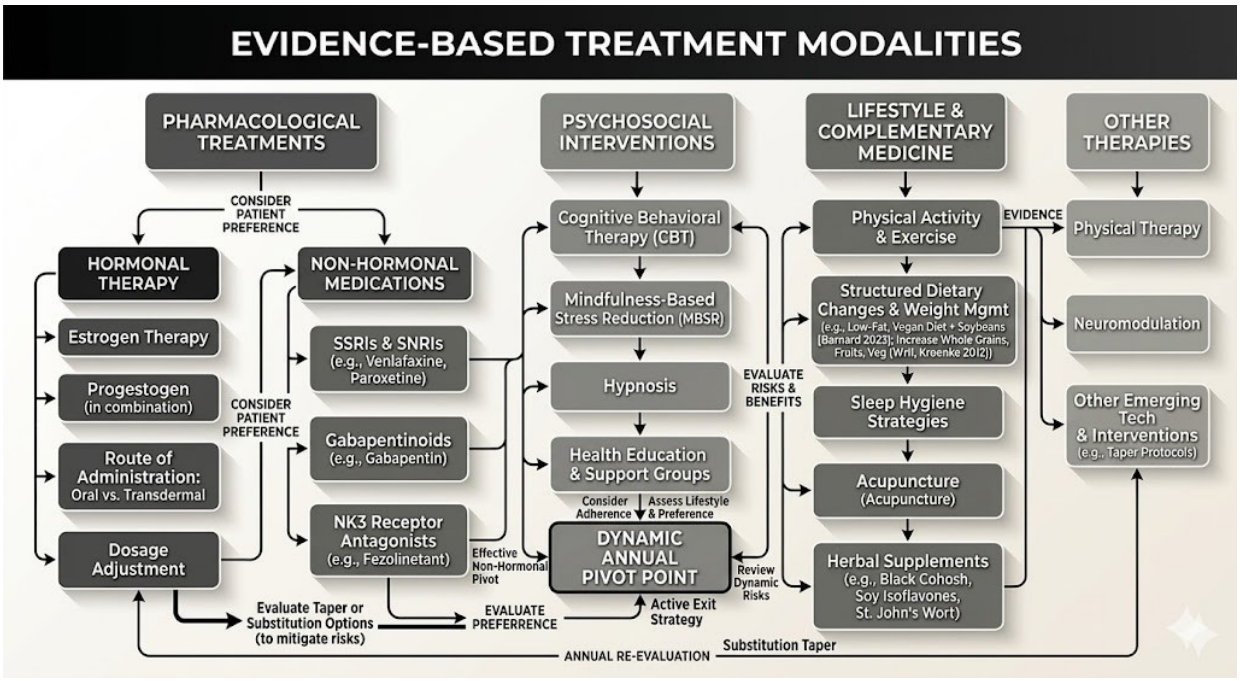

Fig. 1. Evidence-based clinical pathways for the management of menopausal VMS. Diagram generated using Gemini (Advanced version 1.5 Pro). Hormonal Pathway: Menopausal Hormone Therapy (MHT) is recommended within the timing window (aged <60 years or <10 years post-menopause) [13,14,15,16,17]; Transdermal route is preferred for elevated VTE/stroke risks [14,15,16,17]. Non-Hormonal Pharmacotherapy: Recommended when MHT is contraindicated or declined; includes NK3 receptor antagonists (fezolinetant) [12,18], SSRIs/SNRIs [12,14,15,17], and gabapentinoids [12,21]. Lifestyle & Dietary Restructuring: Includes low-fat plant-based patterns with whole soy [20] and general dietary restructuring with weight loss [19]. VMS, vasomotor symptoms; SSRIs, selective serotonin reuptake inhibitors; SNRIs, serotonin-norepinephrine reuptake inhibitors; NK3, neurokinin 3.

4. Evidence-Based Treatment Modalities for VMS

Contemporary management of menopausal symptoms necessitates a structured, individualized approach guided by evidence-based clinical decision algorithms (Fig. 1). (Ref. [12,13,14,15,16,17,18,19,20,21]). The management of menopause is evolving as clinicians navigate between conceptualizing it as a natural transition and treating it as a condition warranting intervention. After a decline in use following the 2002 WHI trial, there has been a resurgence in MHT, with a 30.5% increase in prescriptions in England between 2020/21 and 2021/22 and a 47% rise in total items dispensed by 2022/23. This surge has been driven by advocacy campaigns emphasizing symptom relief. Although menopause is a natural physiological event, its associated symptoms can severely impact quality of life, justifying therapeutic intervention. Current clinical consensus suggests that the benefits of MHT outweigh the risks in women under 60 years or within 10 years of menopause onset. VMS, including hot flushes and night sweats, can disrupt sleep, increase anxiety, and reduce productivity. Nevertheless, many women face fragmented care and limited provider training, leading to persistent confusion regarding MHT safety [21,22,23,24].

Contemporary management of menopausal symptoms requires a structured, individualized approach, moving away from standardized protocols toward personalized risk-benefit assessment. For mild-to-moderate symptoms, initial strategies often involve lifestyle modifications, including regular physical activity, mind-body practices, and dietary adjustments. When MHT is indicated, current consensus supports using the lowest effective dose, with a preference for non-oral routes of administration (e.g., transdermal patches) to mitigate thrombotic risks, particularly in susceptible individuals. For those with contraindications or a preference to avoid hormonal therapy, evidence-based non-hormonal alternatives, such as NK3 receptor antagonists or selected antidepressants, provide effective symptom relief. Although most cases can be managed within primary care, referral to a menopause specialist is advisable for complex presentations. Importantly, when appropriately prescribed, contemporary MHT is recognized as a safe and effective therapeutic option that not only alleviates VMS but may also confer long-term benefits, such as the prevention of osteoporosis. Ultimately, the development of a personalized treatment plan, in consultation with a qualified healthcare provider and based on a comprehensive evaluation of symptom severity, medical history, and individual patient preferences, is essential for achieving optimal outcomes [25,26,27].

Table 1. Comprehensive summary of global clinical practice guidelines for MHT.

Clinical parameter	Consensus recommendations and evidence-based findings
Initiation window	Generally recommended for symptomatic women <60 years of age or within 10 years of menopause onset to maximize the benefit-risk ratio [14,15,16,17,28].
Primary indications	Management of moderate-to-severe VMS [13,14,15,16,17,28] and prevention of osteoporosis [13,14,15,16,28]. ET: May reduce CHD/CVD risk [13,16,17,28].
CVD risk	EPT: Neutral effect or minimal risk increase [13,14,16,17,28]. *Note:* CVD risk factors are not contraindications when optimally managed [13,16,28].
Stroke and VTE risk	Oral MHT is associated with increased risk; transdermal estrogen is consistently identified as carrying a significantly lower or negligible risk [13,14,15,16,17,28]. ET: Little to no change in risk.
Breast cancer risk	EPT: Possible small increase; risk is duration-dependent and declines post-cessation [13,14,15,16,17,28]. *Progesterone/Dydrogesterone* may offer a lower risk profile [15,16,17,28].
Endometrial safety	Unopposed ET significantly increases endometrial cancer risk; adding progestogen in women with a uterus is a mandatory protective measure [13,15,17].
Ovarian cancer risk	Evidence remains inconsistent; risk is considered small, or data is inconclusive [15,16,17,28].
Contraindications	History of breast/estrogen-sensitive cancer, active liver disease, unexplained vaginal bleeding, and established/high-risk thromboembolic disease [14,15,17].
Duration of treatment	No universal upper limit; treatment duration should be individualized based on ongoing symptom severity and annual benefit-risk re-evaluation [13,14,15,17].
Vaginal estrogen	Recommended for genitourinary symptoms; not associated with increased CVD or breast cancer risk [15,17,28].

VMS, vasomotor symptoms; CVD, cardiovascular disease; ET, estrogen therapy; EPT, estrogen-progestogen therapy; CHD, coronary heart disease; VTE, venous thromboembolism; MHT, menopausal hormone therapy.

Hormonal therapy (HT) remains the most effective treatment for moderate-to-severe VMS, such as hot flushes and night sweats, which can significantly impair quality of life [12,13]. The clinical decision to initiate HT is based on a favorable benefit-risk profile, particularly for symptomatic women under the age of 60 years or within 10 years of menopause onset, a recommendation strongly supported by high-level evidence and endorsed across international consensus [14,15,16,17]. Crucially, this decision should not be guided by age alone but must incorporate individual patient preference and a thorough, personalized assessment of benefits versus risks, a principle consistently upheld by all major international guidelines.

The regimen and administration of HT must be tailored to the individual. In women with an intact uterus, estrogen must be combined with a progestogen to prevent endometrial hyperplasia and carcinoma, whereas women who have undergone hysterectomy may be treated with estrogen-only therapy [13]. The general principle is to use the lowest effective dose, with transdermal estrogen specifically recommended for women with risk factors for venous thromboembolism (VTE) or stroke, as evidence indicates that it does not increase the risk of thromboembolic events compared to oral administration. This aligns with the broader guideline consensus that transdermal estrogen confers a lower risk of cardiovascular disease (CVD), stroke, and VTE compared with oral formulations [14,15,16,17]. Contraindications to HT are strictly defined to ensure patient safety. Absolute contraindications include a history of or current estrogen-sensitive malignancy (e.g., breast cancer), active or prior thromboembolic disease, active hepatic disease, and unexplained vaginal bleeding. Furthermore, established CVD (e.g., recent stroke or myocardial infarction) continues to be an additional noted contraindication [14,15,17]. It should be emphasized that HT is not indicated for the primary or secondary prevention of CVD. For women in these high-risk categories, non-hormonal alternatives like selective serotonin reuptake inhibitors (SSRIs), serotonin-norepinephrine reuptake inhibitors (SNRIs), or gabapentin are recommended.

There is no fixed upper age limit for MHT use. International guidelines recommend individualizing treatment duration and conducting annual benefit-risk assessments. These reviews should actively incorporate lifestyle factors and emerging non-hormonal options, rather than merely monitoring for adverse events and defaulting to continued hormone use. As a patient's cardiovascular, metabolic, and oncological risk profile evolves, alternatives like dietary changes, weight management, or NK3 receptor antagonists can significantly alter the risk-benefit balance. For instance, adopting a low-fat, plant-based diet can reduce VMS and lower cardiovascular and breast cancer risks. Clinicians should use annual evaluations to assess whether non-hormonal or behavioral therapies can safely replace or reduce HT, especially as patients transition out of early postmenopause. This approach ensures dynamic, personalized management rather than static treatment continuation. The major consensus recommendations from global clinical practice guidelines for MHT are summarized in Table 1 (Ref. [13,14,15,16,17,28]).

The pharmacological approach to non-hormonal VMS management (Table 2, Ref.

Table 2. Pharmacological profiles and international guideline recommendations for non-hormonal management of VMS.

Drug class/Agent	Mechanism of action	Common side effects	Duration/Clinical notes	Guideline recommendations and consensus
SSRIs/SNRIs (e.g., Paroxetine, Venlafaxine)	Modulate serotonergic and noradrenergic hypothalamic circuits [9].	Nausea, dry mouth, sexual dysfunction, insomnia [18].	Significant VMS reduction within 4 weeks; efficacy persists over time [18].	Recommended as first-line non-HT [12,14,15,17]. Contraindication: Avoid paroxetine/fluoxetine with tamoxifen [12,13,14,15].
Gabapentinoids (e.g., Gabapentin)	Modulates voltage-gated calcium channels; reduces neuronal excitability [21].	Dizziness, drowsiness, fatigue, peripheral edema [21].	Particularly effective for nocturnal VMS and sleep disturbances [29].	Recommended when MHT is contraindicated or for nighttime symptoms [12,14,15,17]. Pregabalin is generally not recommended [12,13].
Clonidine	α2-adrenergic agonist; reduces central sympathetic outflow.	Dry mouth, hypotension, constipation, drowsiness.	Lower efficacy (20–40% reduction) compared to other options.	Considered a second-line or trial option for refractory cases [14,15,17]. Not recommended as first-line by NAMS/AWMF [12,16].
NK3 receptor antagonists (e.g., Fezolinetant)	Blocks neurokinin B in KNDy neurons to stabilize thermoregulation.	Headache, insomnia, transient liver enzyme elevation [18].	Rapid onset within 4 weeks; sustained efficacy up to 52 weeks [18].	FDA-approved (2023) for moderate-to-severe VMS. Recommended by NAMS as a highly effective non-hormonal choice [12].
Oxybutynin [29]	Anticholinergic agent; affects thermoregulatory sudomotor responses.	Dry mouth, blurred vision, constipation, urinary retention.	Long-term use in older adults may be associated with cognitive decline.	Recommended by NAMS for VMS (off-label) [12]. Not extensively discussed in other global guidelines.
Phyto-estrogens /Isoflavones [29]	Plant-derived compounds that bind weakly to estrogen receptors.	Generally well-tolerated; potential GI upset.	Evidence for efficacy is inconsistent across studies.	Inconsistent evidence; some benefit for VMS burden, but concerns remain regarding preparation quality/safety [13,15,16,17].

VMS, vasomotor symptoms; SSRIs, selective serotonin reuptake inhibitors; SNRIs, serotonin-norepinephrine reuptake inhibitors; NAMS, The North American Menopause Society; AWMF, Arbeitsgemeinschaft der Wissenschaftlichen Medizinischen Fachgesellschaften; NK3, neurokinin 3; HT, hormonal therapy.

[9,12,13,14,15,16,17,18,21,29]) primarily targets thermoregulatory and neurochemical pathways within the central nervous system. SSRIs and SNRIs, such as venlafaxine and paroxetine, modulate serotonergic and noradrenergic circuits in the hypothalamus, leading to a significant reduction in hot flash frequency of approximately 40–65%. This established efficacy is reflected in the guidelines, recommending SSRIs and SNRIs as a primary non-hormonal option when MHT is contraindicated or not preferred [12,14,15,17]. However, a critical caveat related to their metabolic pathway is consistently highlighted: paroxetine and fluoxetine are contraindicated in breast cancer patients receiving tamoxifen due to a detrimental pharmacokinetic interaction that may reduce tamoxifen's efficacy [12,13,14,15]. A more conservative position is adopted by some guidelines, which do not recommend them as first-line therapy for VMS due to a perceived moderate risk of harm or insufficient evidence for their use in treating menopausal low mood without a diagnosed depressive disorder [13,16]. Gabapentinoids, such as gabapentin, act by modulating voltage-gated calcium channels to reduce neuronal excitability within the thermoregulatory center. This mechanism translates to a robust 45–60% reduction in VMS, with particular efficacy for nocturnal symptoms. The clinical guidance on its use is mixed but generally supportive; multiple organizations endorse gabapentin for VMS when MHT is not suitable, though some formally note its off-label status for this indication [12,14,15,17]. By contrast, other guidelines do not recommend it as a first-line option due to a moderate risk profile [16]. Pregabalin is rarely addressed in current guidelines and is generally not recommended due to limited evidence and a higher burden of adverse effects [12]. Clonidine, an α2-adrenergic agonist, reduces

Table 3. Clinical evidence and guideline consensus for complementary, behavioral, and lifestyle interventions in menopause care.

Intervention	Mechanism of action	Efficacy/VMS reduction	Side effects/Safety	Guideline recommendations and consensus
Weight loss and dietary modification	Adiposity intensifies hypothalamic instability [18]. Healthy dietary patterns reduce systemic inflammation and stabilize thermoregulation.	Weight loss significantly reduces VMS. Dietary restructuring (low-fat, high fruit/vegetables/whole grains) increases the likelihood of complete VMS clearance by 14%–23% [20]. Combining effect of ≥10% weight loss with dietary changes nearly doubles the odds of complete symptom clearance [19].	Improved cardiovascular fitness and mood.	Universally recommended for overall health. Strongly supported by WHI data demonstrating that healthy dietary shifts combined with weight loss offer robust non-hormonal VMS resolution [20].
CBT [30]	Reinterprets symptom experience; reduces perceived intensity/anxiety.	Improves quality of life and sleep; frequency reduction is modest or subjective.	None.	Recommended as an adjunct or primary choice when pharmacotherapy is declined [12,13,14,17].
Acupuncture [31]	Subjective well-being; potential physiological modulation.	20–30% reduction; often similar to placebo/sham in controlled trials.	Minor bruising or localized pain.	Inconsistent evidence; considered for VMS by IMS/AWMF [15,16]; insufficient evidence by NICE/NAMS [12,13].
Clinical Hypnosis [12,17]	Alters the cortical perception of thermal stimuli.	Up to a 50% reduction in frequency has been shown in controlled trials.	None.	Recommended by NAMS [12] and Endocrine Society [17] (with trained practitioners).
Mindfulness & Yoga [12,15]	Reduces stress-related sympathetic activity.	Modest effect on frequency; improves sleep and psychological well-being.	None.	Recommended as complementary approaches for quality of life rather than frequency reduction [12,15].
Black cohosh (*C. racemosa*) [32]	Estrogenic and progestogenic effects (proposed).	25–30% reduction; efficacy is variable across guidelines.	Hepatotoxicity risk, GIT complaints, dizziness.	Use with caution due to safety/quality concerns [13,15]; Not recommended by NAMS [12].
Soy, phytoestrogens, & plant-based diets	Weak binding to estrogen receptors; structurally similar to estradiol [19]. Shifting to a low-fat, plant-based environment alters the metabolic processing of isoflavones.	Variable efficacy alone (10–30%) [19]. However, an *ad libitum* low-fat, vegan diet with 1/2 cup of daily cooked soybeans reduces moderate-to-severe hot flushes by 88%, achieving total elimination in 50% of individuals within 12 weeks [20].	Gastrointestinal (GIT) upset (minor with whole foods), generally exceptionally well-tolerated. High adherence due to no calorie limits.	Guidelines show inconsistent evidence for isolated supplements [13,15,16]. However, contemporary data support a structured low-fat, plant-based dietary pattern with whole soy as a highly effective intervention regardless of equol status [19].
St. John's wort [33]	Inhibits reuptake of serotonin, norepinephrine, and dopamine	Modest improvement in symptoms/mood.	Allergic reactions, GIT complaints, neuropathy.	Not recommended or insufficient evidence across most major guidelines [12,17].
Herbal extracts (Wild Yam, Dong Quai, Ginseng) [33]	Proposed estrogenic or undetermined effects.	Generally found to be not superior to placebo in clinical trials.	Ginseng: Vaginal bleeding, Stevens-Johnson syndrome. Dong Quai: Bleeding, photosensitivity.	Not recommended for VMS due to lack of evidence and potential safety risks [12,14,15,17].

VMS, vasomotor symptoms; CBT, cognitive behavioral therapy; GIT, gastrointestinal; WHI, Women's Health Initiative; IMS, International Menopause Society; AWMF, Arbeitsgemeinschaft der Wissenschaftlichen Medizinischen Fachgesellschaften; NICE, National Institute for Health and Care Excellence; NAMS, The North American Menopause Society.

central sympathetic activity, resulting in a more modest 20–40% reduction in VMS. Although some societies list it as a recommended option [14,15], others suggest it only as a trial for women unable to tolerate alternative therapies [17]. Several major guidelines explicitly do not recommend it as a first-line treatment, citing its adverse effects and the availability of more effective alternatives [12,16].

Although clinical guidelines have often emphasized isolated nutritional supplements, growing evidence suggests that comprehensive dietary changes are crucial in reducing VMS. The WHI Dietary Modification trial by Kroenke et al. [19], enrolling over 17,000 postmenopausal women, demonstrated that altering food intake can significantly improve symptom outcomes. Regarding non-pharmacological approaches, adopting a healthy dietary pattern significantly improved outcomes, increasing the likelihood of complete VMS relief by 23% after adjustment for baseline confounders [20]. Moreover, combining this dietary approach with weight loss of 10% or more nearly doubled the chances of complete VMS resolution, highlighting dietary restructuring as an effective, non-pharmacological strategy for long-term symptom management.

Considering established healthy eating patterns, recent trials have identified specific macronutrient compositions and plant-based dietary regimens with strong therapeutic potential. Barnard et al. [20] demonstrated that a low-fat, vegan diet supplemented with half a cup of cooked soybeans daily led to an 88% reduction in moderate-to-severe hot flushes over 12 weeks. Half of the participants experienced complete relief from these episodes and reported

significant improvements in physical, psychosocial, and sexual quality of life. Because this *ad libitum* regimen avoids strict portion or calorie restrictions, adherence was high. The observed success is attributed to the interaction between a low-fat, plant-based dietary environment and soy isoflavones, which appear to modulate thermoregulation independently of seasonal changes or baseline equol-production capacity. Other complementary mind-body approachesoffer distinct clinical utility. Clinical hypnosis is recognized across major international guidelines as an effective mind-body intervention capable of reducing hot flash frequency and symptom severity when delivered by trained practitioners [12,17]. Conversely, guideline evaluations of mindfulness-based practices and yoga indicate limited direct effects on vasomotor symptom frequency; however, these approaches may provide valuable complementary benefits by improving sleep quality, reducing stress, and enhancing overall quality of life during the menopausal transition [12,15]. The clinical evidence and guideline recommendations for complementary, behavioral, and lifestyle interventions are summarized in Table 3 (Ref. [12,13,14,15,16,17,18,19,20,30,31,32,33]).

To integrate these varied guidelines, the present review proposes a comprehensive therapeutic approach that moves away from static pharmacological selection toward a flexible, multi-level treatment strategy. Instead of treating MHT, non-hormonal options, and plant-based dietary changes as separate, competing methods, this model positions them as complementary interventions addressing distinct components of the neuro-endocrine system. By integrating systemic estrogen replacement, central NK3 receptor blockade, and peripheral metabolic adjustments into a cohesive clinical framework, healthcare providers can move beyond rigid, protocol-driven approaches. This comprehensive approach effectively translates standardized, evidence-based guidelines into highly personalized, patient-centered treatment plans.

5. Conclusions

Management of VMS requires a personalized, evidence-based approach. Although HT remains the most effective treatment for women within the appropriate age and timing window, non-hormonal pharmacotherapy and behavioral interventions like CBT offer valuable alternatives for high-risk patients. International guidelines emphasize individualizing care based on each patient's specific medical history and preferences to optimize safety and quality of life throughout the menopausal transition. Furthermore, clinicians should prioritize using the lowest effective dose and prefer transdermal administration routes whenever possible to mitigate cardiovascular and thromboembolic risks during hormone therapy.

Author Contributions

EA conceptualized the review, performed the literature search and data extraction, analyzed the clinical guidelines, and wrote the manuscript. The author has participated sufficiently in the work and agreed to be accountable for all aspects of the work. The author read and approved the final manuscript.

Ethics Approval and Consent to Participate

Not applicable.

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Conflicts of Interest

The author declares no conflicts of interest.

Declaration of AI and AI-Assisted Technologies in the Writing Process

During the preparation of this work, the author utilized Gemini (Advanced version 1.5 Pro, Google) for the generation and visual optimization of the included figures, as well as to check spelling and grammar in conjunction with Grammarly. The core conception, clinical content, and structural design of the figures and manuscript were entirely undertaken by the author. The author fully reviewed, edited, and approved all generated content and takes full responsibility for the integrity and accuracy of the final publication's content.

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