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Management of Premenstrual Disorders

Committee on Clinical Practice Guidelines–Gynecology. This Clinical Practice Guideline was developed by the ACOG Committee on Clinical Practice Guidelines–Gynecology in collaboration with Kimberly A. Yonkers, MD; Luu D. Ireland MD, MPH; and Amber I. Truehart, MD.

PURPOSE: To provide recommendations for the management of premenstrual syndrome and premenstrual dysphoric disorder, collectively referred to as premenstrual disorders, based on assessment of the evidence regarding the safety and efficacy of available treatment options. An overview of the epidemiology, pathophysiology, and diagnosis of premenstrual disorders also is included to provide readers with relevant background information and context for the clinical recommendations.

TARGET POPULATION: Reproductive-aged adults and adolescents with premenstrual symptoms.

METHODS: This guideline was developed using an a priori protocol in conjunction with a writing team consisting of two specialists in obstetrics and gynecology appointed by the ACOG Committee on Clinical Practice Guidelines–Gynecology and one external subject matter expert. ACOG medical librarians completed a comprehensive literature search for primary literature within Cochrane Library, Cochrane Collaboration Registry of Controlled Trials, EMBASE, PubMed, and MEDLINE. Studies that moved forward to the full-text screening stage were assessed by two authors from the writing team based on standardized inclusion and exclusion criteria. Included studies underwent quality assessment, and a modified GRADE (Grading of Recommendations Assessment, Development and Evaluations) evidence-to-decision framework was applied to interpret and translate the evidence into recommendation statements.

RECOMMENDATIONS: This Clinical Practice Guideline includes recommendations on the following evidence-based treatment options for premenstrual disorders, with an acknowledgement that many patients may benefit from a multimodal approach that combines several interventions: pharmacologic agents (hormonal and nonhormonal), psychological counseling, complementary and alternative treatments, exercise and nutritional therapies, patient education and self-help strategies, and surgical management. Recommendations are classified by strength and evidence quality. Ungraded Good Practice Points are included to provide guidance when a formal recommendation could not be made because of inadequate or nonexistent evidence. Based on review of extrapolated data from adult populations and expert consensus, it was determined that the recommendations also apply to adolescents, with a few exceptions that are noted in the Clinical Practice Guideline.

INTRODUCTION

Premenstrual disorders comprise a spectrum of conditions that include premenstrual syndrome (PMS) and premenstrual dysphoric disorder (PMDD). Premenstrual syndrome is marked by a constellation of cyclically occurring physical or mood-related symptoms or both that occur discretely in the luteal phase and resolve during or shortly after menstruation (1). The most common symptoms include irritability, bloating, mood swings, lethargy, breast tenderness, anxiety and tension, and feelings of rejection (2, 3). Premenstrual dysphoric disorder is classified as a type of depressive disorder by the American Psychiatric Association and is characterized by the cyclic recurrence of severe, sometimes disabling changes in affect—such as mood lability, irritability, dysphoria, and anxiety—that occur in the luteal phase of the menstrual cycle and subside around, or shortly after, the onset of menses (4). In 2019, the World Health Organization added PMDD to the *International Statistical Classification of Diseases and Related Health Problems, Eleventh Revision*; although it is classified as a genitourinary disorder, it also is listed among the depressive disorders (5).

Premenstrual disorders are associated with physical and affective symptoms that interfere with daily functioning, which leads many patients to seek care from an obstetrician–gynecologist, primary care physician, pediatric and adolescent health specialist, psychiatrist, other mental health professional, or a combination of these. The purpose of this Clinical Practice Guideline is to provide evidence-based guidelines for the management of premenstrual disorders. The recommendations in this guideline apply to adults and adolescents unless otherwise indicated. Because there is limited evidence on the treatment of premenstrual disorders in adolescents, the included guidance is based largely on extrapolated data from adult populations and expert consensus.

SUMMARY OF RECOMMENDATIONS

ACOG recommends selective serotonin reuptake inhibitors (SSRIs) for the management of affective premenstrual symptoms. **(STRONG RECOMMENDATION, MODERATE-QUALITY EVIDENCE)**

ACOG recommends combined oral contraceptives (COCs) for the management of overall premenstrual symptoms. **(STRONG RECOMMENDATION, LOW-QUALITY EVIDENCE)**

ACOG recommends cognitive behavioral therapy (CBT) for the management of affective premenstrual symptoms. **(STRONG RECOMMENDATION, LOW-TO-MODERATE-QUALITY EVIDENCE)**

STRENGTH OF RECOMMENDATION

STRONG

ACOG recommends:

Benefits clearly outweigh harms and burdens. Most patients should receive the intervention.

ACOG recommends against:

Harms and burdens clearly outweigh the benefits. Most patients should not receive the intervention.

CONDITIONAL

ACOG suggests:

The balance of benefits and risks will vary depending on patient characteristics and their values and preferences. Individualized, shared decision making is recommended to help patients decide on the best course of action for them.

QUALITY OF EVIDENCE

HIGH

Randomized controlled trials, systematic reviews, and meta-analyses without serious methodologic flaws or limitations (eg, inconsistency, imprecision, confounding variables)

Very strong evidence from observational studies without serious methodologic flaws or limitations There is high confidence in the accuracy of the findings and further research is unlikely to change this.

MODERATE

Randomized controlled trials with some limitations Strong evidence from observational studies without serious methodologic flaws or limitation

LOW

Randomized controlled trials with serious flaws Some evidence from observational studies

VERY LOW

Unsystematic clinical observations
Very indirect evidence from observational studies

GOOD PRACTICE POINTS

Ungraded Good Practice Points are incorporated when clinical guidance is deemed necessary in the case of extremely limited or nonexistent evidence. They are based on expert opinion as well as review of the available evidence.

ACOG suggests gonadotropin-releasing hormone (GnRH) agonists with adjunctive combined hormonal add-back therapy for adults with severe, refractory premenstrual symptoms. **(CONDITIONAL RECOMMENDATION, MODERATE-QUALITY EVIDENCE)**

ACOG suggests routine exercise to help manage physical and affective premenstrual symptoms. **(CONDITIONAL RECOMMENDATION, LOW-QUALITY EVIDENCE)**

ACOG suggests calcium supplementation of 1,000–1,200 mg per day in adults to help manage physical and affective premenstrual symptoms. **(CONDITIONAL RECOMMENDATION, LOW-QUALITY EVIDENCE)**

ACOG suggests adequate calcium intake in adolescents to help manage physical premenstrual symptoms. **(CONDITIONAL RECOMMENDATION, LOW-QUALITY EVIDENCE)**

ACOG suggests the use of acupuncture to help manage physical and affective premenstrual symptoms. **(CONDITIONAL RECOMMENDATION, LOW-QUALITY EVIDENCE)**

ACOG suggests nonsteroidal anti-inflammatory drugs (NSAIDs) for the management of premenstrual pain symptoms. **(CONDITIONAL RECOMMENDATION, LOW-QUALITY EVIDENCE)**

ACOG suggests that clinicians provide patient education about premenstrual symptoms and self-help coping strategies as part of a holistic approach to the management of premenstrual disorders. **(CONDITIONAL RECOMMENDATION, LOW-QUALITY EVIDENCE)**

Bilateral oophorectomy with or without hysterectomy should be reserved as a treatment option for adults with severe premenstrual symptoms only when medical management has failed and patients have been counseled about the associated risks and irreversibility of the procedure. A trial period of GnRH agonist therapy (with or without estrogen add-back treatment) is advised before surgery to predict a patient's response to surgical management. **(GOOD PRACTICE POINT)**

Collaboration with or referral to a mental health professional should be considered for patients with premenstrual symptoms if the diagnosis is unclear or an underlying mood disorder is suspected. **(GOOD PRACTICE POINT)**

METHODS

ACOG Clinical Practice Guidelines provide clinical management recommendations for a condition or procedure by assessing the benefits and harms of care options through a systematic review of the evidence. This guideline was developed using an a priori protocol in conjunction with a writing team consisting of two specialists in obstetrics and gynecology appointed by the ACOG Committee on Clinical Practice Guidelines–Gynecology and one external subject matter expert. A full description of the Clinical Practice Guideline methodology is published separately (6). The following description is specific to this Clinical Practice Guideline.

Literature Search

ACOG medical librarians completed a comprehensive literature search for primary literature within Cochrane Library, Cochrane Collaboration Registry of Controlled Trials, EMBASE, PubMed, and MEDLINE. Parameters for the search included human-only studies published in English. The search was restricted to studies from 2000 to March 2021.The MeSH terms and keywords used to guide the literature search can be found in Appendix A (available online at http://links.lww.com/AOG/D430). An updated literature search was completed in September 2022 and reviewed by two members of the writing team using the same systematic process as the original literature search. One additional supplemental literature search was performed in June 2023 to ensure any newly published high-level sources were addressed in the final manuscript.

Study Selection

A title and abstract screen of all studies was completed by ACOG research staff. Studies that moved forward to the full-text screening stage were assessed by two authors from the writing team (a subject matter expert and a specialist in obstetrics and gynecology) based on standardized inclusion and exclusion criteria. To be considered for inclusion, studies had to be conducted in countries ranked very high on the United Nations Human Development Index (7) and include reproductive-aged adult or adolescent participants with premenstrual symptoms. Although systematic reviews, randomized controlled trials (RCTs), and prospective cohort studies were prioritized, case–control studies were considered for topics with limited evidence, particularly for rare outcomes. A PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) flow diagram of the included and excluded studies can be found in Appendix B, available online at http://links.lww.com/AOG/D431. All studies that underwent quality assessment had key details extracted (study design, sample size, details of interventions, outcomes) and descriptions included in the summary evidence tables (Appendix C, available online at http://links.lww.com/AOG/D432).

Recommendation Development

A modified GRADE (Grading of Recommendations Assessment, Development and Evaluations) evidence-to-decision framework was applied to interpret and translate the evidence into draft recommendation statements, which were classified by strength and evidence quality (8, 9). Ungraded Good Practice Points were incorporated to provide clinical guidance in the case of extremely limited or nonexistent evidence. They are based on expert opinion as well as review of the available evidence (10). Because of the lack of studies on the

management of premenstrual disorders in adolescents, there are few adolescent-specific recommendations. Based on review of extrapolated data from adult populations and expert consensus, it was determined that the adult recommendations also apply to adolescents, with a few exceptions that are noted in the Clinical Practice Guideline.

All the recommendations and supporting evidence tables were reviewed, revised as appropriate, and affirmed by the Committee on Clinical Practice Guidelines–Gynecology at a meeting. The guideline manuscript was then written and subsequently reviewed and approved by the Committee on Clinical Practice Guidelines and other internal review bodies before continuing to publication.

Use of Language

The American College of Obstetricians and Gynecologists (ACOG) recognizes and supports the gender diversity of all patients who seek obstetric and gynecologic care. In original portions of this document, authors seek to use gender-inclusive language or gender-neutral language. When describing research findings, this document uses gender terminology reported by investigators. To review ACOG's policy on inclusive language, see https://www.acog.org/clinical-information/policy-and-position-statements/statements-of-policy/2022/inclusive-language.

CLINICAL OVERVIEW

Epidemiology

Up to 90% of reproductive-aged women report experiencing at least one premenstrual symptom (11), with approximately 20–30% experiencing symptoms bothersome enough to meet criteria for PMS (12, 13). Approximately 2–5% of women report severe and disabling symptoms that meet the diagnostic criteria for PMDD (14). Women who are affected by PMDD experience, on average, a total of 3,000 symptomatic days or 3.8 years of disability in their reproductive years (15). Individuals with PMDD also report lower quality of life, more interpersonal difficulties and relationship stress, and increased visits to health care practitioners compared with individuals without PMDD (16). However, many women with premenstrual disorders go untreated. In one study, three-quarters of women either did not seek help or unsuccessfully sought help from a clinician within the previous 5 years (17, 18).

The prevalence of premenstrual disorders among adolescents is challenging to measure because premenstrual symptoms can be difficult to distinguish from the emotional lability and wide variation in mood that occur as part of normal adolescent development. A cross-sectional study of 171 adolescent females found that 61.4% met diagnostic criteria for PMS (based on adapted *Diagnostic and Statistical Manual of Mental Disorders* [Fourth Edition] criteria for PMDD), with more than 50% reporting moderate or severe symptoms (19). The most common symptoms included negative affect and water retention. In a South Korean cross-sectional study of 984 adolescent females, 20% of participants reported distressing premenstrual symptoms and another 6.7% met diagnostic criteria for PMDD (20).

Limited data suggest that the prevalence of premenstrual disorders varies by race and ethnicity (21, 22). Although few studies have investigated the underlying causes of these observed differences, because there is not a biological or genetic basis for this variation given that race is a social construct, other factors must explain these disparities. For example, in a survey study of 2,718 Asian, Latina, and Black premenopausal women aged 18–40 years, those who experienced gender or racial discrimination also were more likely to report premenstrual symptoms and PMDD, which is consistent with other research that has found an association between racial discrimination and adverse mental health effects (23). It is well established that social determinants of health (ie, historical, social, political, and economic forces that help explain the relationship between environmental conditions and individual health) are responsible for a large portion of health disparities and health inequities that exist in the United States (24). Systemic racism and discrimination, in particular, are significant social determinants of mental health (25).

Black, Indigenous, and other people of color; adolescents; and transgender and gender-diverse individuals are significantly underrepresented in studies on premenstrual disorders. To help ensure equitable, patient-centered care, future research is needed to better understand the prevalence, presentation, and treatment response of premenstrual disorders in diverse populations. For example, further investigation is needed to better understand how systemic racism and bias and other social determinants of health affect the presentation of premenstrual symptoms and the care patients receive. Research also is needed to evaluate the effect of gender-affirming hormone therapy and surgical management on severe premenstrual symptoms.

Pathophysiology

Although the pathophysiology of premenstrual disorders remains unclear, it is likely multifactorial. Leading theories implicate a heightened sensitivity to normal fluctuations in estrogen and progesterone during the luteal phase of the menstrual cycle and dysfunction of the serotonin and gamma aminobutyric acid (GABA) neurotransmitter systems (26).

Given that premenstrual symptoms are absent before menarche, during pregnancy, and after menopause, fluctuations in levels of estrogen and progesterone

during the menstrual cycle are understood to play a central role. However, these cyclic changes in ovarian hormone levels do not trigger symptoms in all menstruating individuals, and symptoms do not appear to be due to an excess or depletion of ovarian hormones, because research shows that estrogen and progesterone levels are similar in individuals with and without premenstrual symptoms (27). Thus, it is believed that individuals with premenstrual disorders may have an increased sensitivity to the normal fluctuations in estrogen and progesterone levels during the menstrual cycle (28).

There are two leading theories regarding the etiopathology of premenstrual disorders. The first theory proposes that the decline in estrogen levels in the late luteal phase of the menstrual cycle may trigger or exacerbate dysregulation of the serotonin system, particularly serotonin transport (26, 28). This theory is supported by serotonin's role in the pathophysiology of other mood and anxiety disorders, evidence of the efficacy of selective serotonin reuptake inhibitors (SSRIs) as a treatment for premenstrual disorders (29), and the exacerbation of premenstrual symptoms triggered by depletion of tryptophan, the main precursor of serotonin (30).

The second theory suggests that premenstrual symptoms may be related to the effects of progesterone and its metabolite, allopregnanolone, on the GABAergic system. Allopregnanolone is a neuroactive steroid that acts as a powerful agonist of the GABA-A receptor to enhance the neurotransmitter's calming effects on mood (31, 32). Individuals with premenstrual disorders may have an increased sensitivity to the rise and fall of allopregnanolone in the luteal phase of the menstrual cycle and experience a withdrawal effect that leads to affective symptoms such as anxiety and depression (33). Allopregnanolone's role in the pathogenesis of PMDD is supported by evidence that administration of experimental agents that can stabilize allopregnanolone levels or modulate its activity are associated with an improvement in PMDD symptoms (33–36). There also is research to support that SSRIs can alter allopregnanolone levels (37, 38), which may be an additional mechanism of action that explains SSRIs' rapid therapeutic effect, even when administered at symptom onset (39).

Evaluation and Diagnosis

Premenstrual disorders are a diagnosis of exclusion, and clinicians should rule out other potential causes of premenstrual symptoms, such as other mood disorders or general medical conditions (26). The initial evaluation of patients who report premenstrual symptoms requires thorough medical history taking and evaluation of symptoms. Some experts also recommend routine screening for suicidal thoughts and behavior in individuals who report moderate-to-severe premenstrual symptoms based on evidence that suggests that premenstrual disorders are associated with an increased likelihood of suicidality and nonsuicidal self-injury (40, 41). A positive screening result should trigger immediate evaluation, ideally, by a mental health professional.

Diagnosis of premenstrual disorders is based on patients' retrospective report that symptoms have been present most menstrual cycles in the preceding year and on 2 months of prospective symptom recording. Prospective monitoring of symptoms with a symptom diary or calendar is important to help identify the pattern and severity of symptoms throughout the menstrual cycle and to help mitigate recall bias and misattribution of symptom expression only to the premenstrual phase (26, 42, 43). Because symptoms can vary from cycle to cycle, experts generally recommend that patients record symptoms for two consecutive cycles (1, 43). A commonly used, validated prospective symptom questionnaire is the Daily Record of Severity of Problems, which is aligned with *Diagnostic and Statistical Manual of Mental Disorders* diagnostic criteria for PMDD (1, 43–45). Alternatively, patients can keep a simple diary or calendar that includes the dates of their menstrual periods and a daily record of their symptoms and severity (eg, by assigning a score of 1–5) (26). If a symptom diary is inconclusive, the use of a 3-month trial of gonadotropin-releasing hormone (GnRH) agonist therapy to suppress ovarian hormone function may help to confirm the diagnosis (43).

The results of prospective symptom recording will help to discern a premenstrual disorder from an underlying medical or mood disorder. Cyclic symptoms that are limited to the premenstrual phase and the first few days of menses are associated with PMS and PMDD. Symptoms that occur intermittently across the menstrual cycle, rather than only during the perimenstrual interval, suggest the presence of an underlying medical condition or mood disorder. Chronic symptoms that worsen during the premenstrual phase suggest premenstrual exacerbation of an underlying medical or mood disorder (26). Premenstrual syndrome can be distinguished from PMDD through application of the *Diagnostic and Statistical Manual of Mental Disorders* (Fifth Edition) criteria, which require the presence of a minimum of five symptoms, including at least one of four key affective symptoms, to establish a diagnosis of PMDD (Box 1) (4). The primary distinction between PMS and PMDD is that the symptoms of PMDD are predominantly affective and severe enough to interfere with the ability to function, comparable with other mental disorders, such as a major depressive episode or generalized anxiety disorder (4). Collaboration with or referral to a mental health professional should be considered for patients with premenstrual symptoms if the diagnosis is unclear or an underlying mood disorder is suspected.

Box 1. Diagnostic Criteria for Premenstrual Dysphoric Disorder

A. In the majority of menstrual cycles, at least five symptoms must be present in the final week before the onset of menses, start to *improve* within a few days after the onset of menses, and become *minimal* or absent in the week postmenses.

B. One (or more) of the following symptoms must be present:
   1. Marked affective lability (eg, mood swings; feeling suddenly sad or tearful, or increased sensitivity to rejection).
   2. Marked irritability or anger or increased interpersonal conflicts.
   3. Marked depressed mood, feelings of hopelessness, or self-deprecating thoughts.
   4. Marked anxiety, tension, and/or feelings of being keyed up or on edge.

C. One (or more) of the following symptoms must additionally be present, to reach a total of *five* symptoms when combined with symptoms from Criterion B above.
   1. Decreased interest in usual activities (eg, work, school, friends, hobbies).
   2. Subjective difficulty in concentration.
   3. Lethargy, easy fatigability, or marked lack of energy.
   4. Marked change in appetite; overeating; or specific food cravings.
   5. Hypersomnia or insomnia.
   6. A sense of being overwhelmed or out of control.
   7. Physical symptoms such as breast tenderness or swelling, joint or muscle pain, a sensation of "bloating," or weight gain.

Note: The symptoms in Criteria A–C must have been met for most menstrual cycles that occurred in the preceding year.

D. The symptoms are associated with clinically significant distress or interference with work, school, usual social activities, or relationships with others (eg, avoidance of social activities; decreased productivity and efficiency at work, school, or home).

E. The disturbance is not merely an exacerbation of the symptoms of another disorder, such as major depressive disorder, panic disorder, persistent depressive disorder (dysthymia), or a personality disorder (although it may co-occur with any of these disorders).

F. Criteria A should be confirmed by prospective daily ratings during at least two symptomatic cycles. (**Note**: The diagnosis may be made provisionally prior to this confirmation.)

G. The symptoms are not attributable to the physiologic effects of a substance (eg, a drug of abuse, a medication, other treatment) or another medical condition (eg, hyperthyroidism).

CLINICAL RECOMMENDATIONS AND EVIDENCE SUMMARY

Approach to Management

After determining the timing, type, and severity of premenstrual symptoms, clinicians and patients can discuss different approaches to management. Treatment options range from lifestyle and behavioral interventions to medical management with SSRIs that improve serotonin transmission or with hormonal agents that suppress ovulation. The following discussion presents treatment options in order of demonstrated efficacy and level of recommendation. However, this organization should not be interpreted as a treatment algorithm. Shared decision making, including a discussion of patients' treatment preferences and goals as well as the benefits and risks of available treatment options, is recommended to help patients decide on the most appropriate treatment for them.

Many patients may benefit from a multimodal approach that combines several interventions. For example, patients who desire protection against pregnancy may opt for medical treatment with COCs along with initiation of lifestyle

interventions (eg, exercise), and patients with PMDD or predominantly affective symptoms may benefit from prescription of SSRIs and referral to a mental health professional for psychological counseling (46).

Selective Serotonin Reuptake Inhibitors

ACOG recommends selective serotonin reuptake inhibitors (SSRIs) for the management of affective premenstrual symptoms. (STRONG RECOMMENDATION, MODERATE-QUALITY EVIDENCE)

The efficacy and safety of SSRIs for the treatment of premenstrual disorders is supported by evidence from multiple RCTs (29), and expert guidelines recommend SSRIs as a first-line pharmacologic treatment option for the management of affective premenstrual symptoms (43, 45, 47). Although most studies of SSRIs for the treatment of premenstrual symptoms have included participants who met diagnostic criteria for PMDD, SSRIs are also effective in the treatment of patients with moderate-to-severe PMS (2, 48, 49). Three SSRIs (sertraline, paroxetine, and fluoxetine) are currently approved by the U.S. Food and Drug Administration (FDA) to treat PMDD (50–52). Limited evidence shows that the serotonin-norepinephrine reuptake inhibitor venlafaxine also improves premenstrual symptoms (53, 54).

Efficacy

In a 2013 Cochrane review of 31 randomized placebo-controlled trials, moderate-dose SSRIs (sertraline, fluoxetine, paroxetine, escitalopram, and citalopram) were significantly more effective than placebo in improving overall symptoms, as measured by a decrease in self-rated symptom scores on a validated screening questionnaire (moderate effect size: standardized mean difference [SMD] −0.65, 95% CI −0.46 to −0.84; nine studies, N=1,276) (29). Selective serotonin reuptake inhibitors also were more effective than placebo in reducing specific symptoms: psychological (moderate effect size: SMD −0.51, 95% CI −0.37 to −0.65; five studies, N=795), functional impairment (moderate effect size: SMD −0.71, 95% CI −0.49 to −0.93; two studies, N=334), irritability (moderate effect size: SMD −0.56, 95% CI −0.40 to −0.72; five studies, N=655), and physical (small effect size: SMD −0.43; 95% CI −0.21 to −0.65; five studies, N=781) (29). The overall quality of the RCT evidence base was determined to be moderate because of concerns about poor reporting of methods, attrition and selective reporting, and moderate heterogeneity across studies (29).

Dosing Regimens

Selective serotonin reuptake inhibitors have a rapid onset of action in the treatment of premenstrual symptoms and can begin to improve symptoms within days (29). In contrast, when SSRIs are used to treat depression, daily treatment for several weeks usually is required for symptom amelioration. Because of this quick treatment response, SSRIs can be administered continuously or intermittently (ie, during the luteal phase of the menstrual cycle or from symptom onset to the start of menses). An advantage of intermittent administration is that it may reduce the risk of adverse effects and of discontinuation symptoms when the medication is stopped (39, 55). Limited available evidence suggests that continuous and intermittent dosing regimens have comparable efficacy (29, 56); however, additional long-term, head-to-head studies are needed to confirm these findings (29).

Adverse Effects

The most common adverse effects associated with a moderate dose of SSRI include nausea (odds ratio [OR] 3.43, 95% CI 2.63–4.47), asthenia or decreased energy (OR 3.28, 95% CI 2.16–4.98), somnolence and decreased concentration (OR 4.94, 95% CI 2.82–8.63), fatigue (OR 1.66, 95% CI 1.09–2.53), sexual dysfunction or decreased libido (OR 2.26, 95% CI 1.54–3.31), and sweating (OR 3.02, 95% CI 1.79–5.11) (29). Most of these symptoms attenuate with chronic treatment, although changes in sexual function and libido typically endure for as long as treatment is continued.

Treatment Duration and Discontinuation

The rate of relapse is high among individuals who discontinue SSRI treatment for premenstrual disorders (57), and most patients with premenstrual disorders likely will need treatment until menopause. Individuals on continuous treatment regimens will require medication taper if they decide to stop treatment. Assistance from a psychiatrist may be helpful to guide the taper of medication. Short-acting SSRIs (eg paroxetine, sertraline) are associated with the highest likelihood of discontinuation symptoms.

Treatment Resistance

Some individuals with premenstrual symptoms may not respond to the initial SSRI medication prescribed. Clinicians should first confirm that the patient is taking the medication as directed. Switching from an intermittent to a continuous dosing regimen may be helpful, particularly for patients who have difficulty remembering to take their medication. Another option is to increase the dose during the final week of the menstrual cycle. If the patient still does not respond or experiences bothersome adverse effects, switching to a different SSRI or referral to a psychiatrist or other mental health professional should be considered.

Considerations for Adolescents

Although fluoxetine and sertraline are FDA-approved to treat major depressive disorder and obsessive–compulsive disorder, respectively, in adolescents (50, 52), the use of SSRIs for adolescents with premenstrual disorders has not been rigorously evaluated. An important consideration for the use of SSRIs in adolescents and

young adults (through age 24 years) is the "black box" FDA warning regarding an increased risk of suicidal ideation and behavior (58). The FDA warning is based on analysis of pooled evidence from 24 clinical trials involving more than 4,400 children and adolescents that showed that short-term use (4–16 weeks) of SSRIs or other antidepressants for the treatment of major depression, obsessive–compulsive disorder, or other psychiatric illness was associated with an increased average risk of suicidal thoughts or attempts compared with placebo (4% vs 2%) (58). No suicides were reported in these trials (58).

However, SSRIs are not contraindicated in adolescents, and evidence-based expert guidelines on the management of adolescent depression and anxiety include SSRIs as a treatment option, because the overall potential benefits are believed to outweigh the potential harms with close monitoring for suicidality (59–61). Clinicians who prescribe SSRIs to adolescents or young adults should monitor patients for onset or worsening of suicidal ideation or suicidal behavior or both, particularly within the first few months of treatment initiation and when increasing or decreasing the dose of medication (58). The expression of suicidal thoughts should prompt discontinuation of the medication and referral to a mental health professional, ideally a pediatric and adolescent psychiatrist.

Hormonal Medical Management

Hormonal interventions that have been investigated for the treatment of premenstrual symptoms include combined oral contraceptives (COCs), GnRH agonists, progestin-only methods, and noncontraceptive continuous estrogen formulations. The most abundant and highest-quality evidence supports the use of COCs and GnRH agonists. Available evidence does not support the efficacy of supplemental progesterone and progestins or the 52-mg levonorgestrel-releasing intrauterine device for the treatment of premenstrual symptoms (43, 62–64). Limited low-quality evidence suggests that continuous estrogen in noncontraceptive doses (administered as a subcutaneous implant or transdermal patch) plus progesterone may improve premenstrual symptoms, but additional higher-quality studies are needed to address questions about the optimal dose and risk of adverse events (65). Although the contraceptive patch and vaginal ring have the same mechanism of action as COCs, research is needed to confirm whether these methods are associated with a reduction in premenstrual symptoms.

Combined Oral Contraceptives

ACOG recommends combined oral contraceptives (COCs) for the management of overall premenstrual symptoms. **(STRONG RECOMMENDATION, LOW-QUALITY EVIDENCE)**

Combined oral contraceptives are believed to treat premenstrual disorders by suppressing ovulation and the accompanying fluctuations in estrogen and progesterone levels that are thought to trigger symptoms. Evidence from low-quality RCTs demonstrates that COCs are associated with improvement in overall premenstrual symptom severity and functional impairment (66, 67). However, COCs may not be effective for mood symptoms, particularly premenstrual depressive symptoms (66, 67). Although higher-quality studies are needed to confirm the efficacy of COCs for the management of premenstrual symptoms, COCs are a reasonable treatment option for most patients with general premenstrual symptoms because the benefits (potential improvement of overall premenstrual symptoms and contraception) likely will outweigh the risks. However, for patients who report premenstrual depression as their main concern, shared decision making is recommended and should include discussion of other available treatment options with demonstrated efficacy for the management of premenstrual depression such as SSRIs and CBT, which can be used as adjuncts or alternatives to COC treatment.

Most research on COCs for the management of premenstrual symptoms has focused on formulations that contain the progestin drospirenone (66), and the only COC that is FDA-approved for the treatment of PMDD among individuals seeking contraception is a drospirenone-containing formulation (3 mg drospirenone, 20 micrograms ethinyl estradiol in a 24-day regimen) (68). However, other COC formulations also have been associated with a reduction in premenstrual symptoms (66).

A meta-analysis of data from nine randomized placebo-controlled trials that compared COCs that included varying progestins demonstrated a moderate reduction in overall premenstrual symptom scores on a validated screening questionnaire (SMD 0.41, 95% CI 0.17–0.67; six trials) (66). However, COCs were not more effective than placebo in decreasing depressive premenstrual symptom scores (SMD 0.22, 95% CI −0.06 to 0.47; eight trials). Treatment efficacy did not differ by progestin type (drospirenone, desogestrel, levonorgestrel, or nomegestrol) or regimen (24-day, 21-day, continuous) (66). The meta-analysis authors cautioned that the findings were limited by the low methodologic quality and small samples sizes of the included studies and the use of indirect evidence for comparison of the various COC formulations and regimens (66).

Similar findings were reported by a Cochrane review of five RCTs of drospirenone-containing COCs (67). In addition to a moderate reduction in overall symptom scores (SMD −0.41, 95% CI −0.59 to −0.24; two RCTs, N=514), drospirenone-containing COCs were associated with small-to-moderate decreases in functional impairment symptoms, such as reduced productivity (mean difference [MD] −0.31, 95% CI −0.55 to −0.08; two RCTs, N=432) and interference with social activities (MD

−0.29, 95% CI −0.54 to −0.04; two RCTs, N=432) and relationships (MD −0.30, 95% CI −0.54 to −0.06; two RCTs, N=432) (67). However, drospirenone-containing COCs were not associated with a significant improvement in premenstrual mood symptoms compared with placebo. The overall quality of the evidence was rated as low to moderate because of the large placebo response across trials and concerns about imprecision and heterogeneity (67).

When initiating COCs, comprehensive education on the benefits, potential adverse effects (such as nausea, breast pain, intermenstrual bleeding) (67), and risk of serious complications is important for ensuring that patients are well informed (69). Before prescribing COCs, clinicians should assess patients for thromboembolic risk factors and counsel patients about the increased risk of venous thromboembolism associated with the use of COCs (70, 71).

Gonadotropin-Releasing Hormone Agonists

ACOG suggests gonadotropin-releasing hormone (GnRH) agonists with adjunctive combined hormonal add-back therapy for adults with severe, refractory premenstrual symptoms. (CONDITIONAL RECOMMENDATION, MODERATE-QUALITY EVIDENCE)

Gonadotropin-releasing hormone agonists induce anovulation, which eliminates the premenstrual phase and its associated symptoms. Although available evidence supports the therapeutic efficacy of GnRH agonists for premenstrual symptoms in adults, they are not recommended as a routine treatment option because they are associated with adverse hypoestrogenic effects, particularly vasomotor symptoms and decreased bone density (43, 47). Treatment with GnRH agonists should be reserved for adult patients who have severe physical and affective premenstrual symptoms that have not responded to other treatments. Gonadotropin-releasing hormone agonists generally are not used to treat premenstrual symptoms in adolescents because of the lack of efficacy data in this population and concern about adverse long-term effects on bone health.

Adjunctive combined hormonal add-back therapy is recommended on initiation of GnRH agonist treatment to help mitigate the hypoestrogenic adverse effects (43, 47); however, some studies have found that add-back therapy may precipitate a recurrence of premenstrual symptoms (27, 72). Most studies of GnRH agonists for the management of premenstrual symptoms evaluated up to 6 months of use with or without hormonal add-back therapy (27, 72, 73). For patients who have premenstrual symptom improvement without significant adverse effects after 6 months of GnRH agonist treatment with add-back therapy, the option of continued treatment can be considered after patient-centered counseling about the potential benefits and risks.

In a meta-analysis of five placebo-controlled randomized trials, GnRH agonist analogs were associated with a decrease in overall premenstrual symptoms in reproductive-aged women (SMD −1.19, 95% CI −1.88 to −0.51; N=115) (73). Although GnRH agonists seemed to be more effective for physical symptoms than for affective symptoms, the trend was not statistically significant (73). A subanalysis of data from three of the trials showed similar treatment efficacy with and without hormonal add-back therapy (SMD 0.12, 95% CI −0.35 to 0.58; N=66) (73).

Hormonal add-back therapy was associated with a recurrence of symptoms in some studies (27, 72). However, this appears to be a short-term effect, and the use of a continuous and low dose of combined hormonal add-back therapy may mitigate premenstrual symptom recurrence (27, 72). In a randomized trial that compared three different add-back regimens among 25 women who received GnRH agonist therapy for PMDD, the combined regimen with the highest dose of estradiol (1.5 mg topical estradiol daily and 400 mg luteal phase vaginal progesterone) was associated with significantly greater symptom recurrence than the lower-dose combined regimen (0.5 mg estradiol daily with 400 mg luteal phase vaginal progesterone). Although the incidence of symptom recurrence was lowest among those who received the estrogen-only add-back regimen (1.5 mg topical estradiol daily with placebo) ($P<.001$), unopposed estrogen therapy is not recommended because of the associated risk of endometrial hyperplasia or malignancy (72). In another small study (N=12) of GnRH agonist therapy and add-back for the treatment of PMDD, premenstrual symptom recurrence was observed during the first month of combined hormonal add-back treatment (estradiol 100 mg daily by skin patch and vaginal progesterone 200 mg twice daily), but the effects were short-term and subsided in months 2 and 3, when ovarian hormone levels reached a steady state (27).

Cognitive Behavioral Therapy

ACOG recommends cognitive behavioral therapy (CBT) for the management of affective premenstrual symptoms. (STRONG RECOMMENDATION, LOW-TO- MODERATE-QUALITY EVIDENCE)

Cognitive behavioral treatments help reframe negative and irrational thought patterns and often include education on relaxation techniques, problem-solving skills, and stress management (47). Cognitive behavioral therapy is the most studied psychosocial intervention for the management of premenstrual symptoms and is included as a recommended treatment option in expert guidelines on the management of premenstrual disorders (43, 45, 47, 74). Evidence from older, low-quality RCTs and randomized trials with serious methodologic limitations (eg, small sample size, high attrition rates,

lack of a rigorous control condition, and possible reporting bias) shows that CBT is associated with small-to-moderate improvement in affective premenstrual symptoms (eg, anxiety, depression) (74–76). Similar improvement has been demonstrated in more recent, higher-quality RCTs (77, 78).

Given that CBT is not associated with medical harms or adverse effects, its potential benefits likely will outweigh the risks for most patients with affective premenstrual symptoms. An important caveat, however, is that access to CBT may be limited by several factors, including the cost of treatment, the time commitment required to participate in therapy, and the limited number of mental health professionals with the required specialized training. Online-based CBT counseling interventions may help to overcome some of these treatment barriers, but only for those who have access to reliable internet service. Patient–clinician shared decision making should include discussion of these considerations. Clinicians who plan to recommend CBT to patients should consider having a current list on hand of qualified mental health professionals for referral.

In a meta-analysis of nine low-quality RCTs on the effects of psychological interventions (CBT, patient education, symptom monitoring) on premenstrual symptoms, CBT was the only intervention associated with statistically significant reductions in symptom scores compared with control: anxiety (moderate effect size −0.58, 95% CI −1.15 to −0.01; two trials, N=51), depression (moderate effect size −0.55; 95% −1.05 to −0.05; three trials, N=67), negative behavioral changes (moderate effect size −0.70, 95% CI −1.29 to −0.10; two trials, N=47), and interference with daily living (moderate effect size −0.78, 95% CI −1.53 to −0.03; two trials, N=39) (75). Similarly, a more recent meta-analysis of 11 low-quality studies (six RCTs and five nonrandomized trials) of various psychosocial interventions (CBT, patient education, and social support group) found that cognitive–behavioral coping skills training was the only intervention associated with a statistically significant decrease in overall premenstrual symptom scores (SMD −0.53, 95% CI −0.77 to −0.28) (74).

In the only study to directly compare CBT with SSRIs for the treatment of premenstrual disorders, participants with PMDD (N=108) were randomized to receive CBT, fluoxetine, or combined treatment. Although fluoxetine was associated with a more rapid treatment response, the efficacy of CBT at 6 months was comparable with fluoxetine alone and with combined treatment with fluoxetine and CBT (76). Symptom improvement with CBT also was sustained longer. Among the 51 participants who completed 1-year follow-up, significantly fewer participants in the CBT group met PMDD diagnostic criteria (17%, 3/17) compared with the fluoxetine (59%, 10/17) and combined treatment groups (43%, 6/14) ($P<.04$) (76). However, certainty in the findings is limited by the study's small sample size, inadequate outcome assessment, and high attrition rate.

More recent low-to-moderate-quality RCTs demonstrate improvement of affective premenstrual symptoms using different delivery formats such as couples CBT and internet-based CBT (77, 78). In one small RCT (N=63), individual CBT and couples CBT performed similarly and were associated with significantly greater improvements than the wait-list control in total premenstrual symptom scores ($P=.01$), emotional reactivity/mood scores ($P=.01$), and premenstrual distress scores ($P<.001$) that were maintained at 3-month follow-up (77). Another RCT of 174 women with PMDD that compared the efficacy of an 8-week therapist-guided, internet-based CBT program with a wait-list control found that participants who received CBT treatment experienced a moderate-to-large improvement in psychological impairment ($P\leq.001$) and in functional impairment ($P\leq.001$), as well as a moderate reduction in interference with everyday life activities ($P\leq.001$) and symptom intensity ($P\leq.001$). Treatment effects were maintained at 6-month follow-up (78).

Exercise

ACOG suggests routine exercise to help manage physical and affective premenstrual symptoms. (CONDITIONAL RECOMMENDATION, LOW-QUALITY EVIDENCE)

Available evidence suggests that routine moderate exercise (eg, aerobic, yoga, Pilates) is associated with an improvement in premenstrual symptoms (79–81). Exercise may reduce premenstrual symptoms through several pathways, including effects on beta endorphin, cortisol, and ovarian hormone levels (47, 80, 82, 83).

The high heterogeneity in study design (randomized, nonrandomized, observational), intervention characteristics, and outcomes makes it difficult to draw definitive conclusions about the magnitude of benefit as well as the type and duration of exercise that are most effective. Although additional higher-quality studies are needed to confirm the benefits of exercise for premenstrual symptom management, given its general health benefits, routine exercise can be considered as part of a holistic approach to management, particularly for patients who decline or have contraindications to medical therapies. Because it is unclear whether one type of exercise is more beneficial than another for improvement of premenstrual symptoms, patients should be encouraged to engage in an exercise program of interest. The Centers for Disease Control and Prevention recommends that all adults engage in at least 150–300 minutes per week of moderate-intensity activity or 75–150 minutes per week of vigorous-intensity aerobic physical activity (or a combination of both) (84).

A 2020 systematic review of 17 randomized and nonrandomized studies that included a total of 8,817 women found that regular exercise (including aerobic exercise, strength training, yoga, and Pilates) was associated with an improvement in premenstrual symptoms, particularly anxiety, anger, general pain, constipation, and breast sensitivity (79). *Regular exercise* was generally defined across studies as 1–5 weekly sessions that ranged from 20 to 90 minutes each (although a few studies reported only the total number of hours of exercise per week). The duration of exercise in the included studies varied from 1 to 3 months (with three studies not specifying duration). Meta-analysis was not possible because of the high heterogeneity of study designs and interventions (79).

The results of another systematic review of 14 studies suggest that yoga may be beneficial for overall premenstrual symptoms, particularly affective symptoms (81). The yoga regimens in the included studies ranged from 2 to 16 weeks, with single sessions lasting 10–90 minutes each. Meta-analysis of 11 of these studies showed a significant improvement in total premenstrual symptom scores (SMD 3.15, 95% CI 0.99–5.32; two studies; N=88) and in affective symptom scores (SMD 1.46, 95% CI 0.40–2.53; five studies, N=178). Although four studies (N=152) showed a trend toward improvement in physical symptoms, it was not statistically significant (SMD 0.94, 95% CI −0.09 to 1.97) (81).

Calcium

ACOG suggests calcium supplementation of 1,000–1,200 mg per day in adults to help manage physical and affective premenstrual symptoms. (CONDITIONAL RECOMMENDATION, LOW-QUALITY EVIDENCE)

Calcium has been studied as a treatment option for premenstrual symptoms based on the hypothesis that symptoms may be related to dysfunctional calcium metabolism that is exacerbated by the rise in estrogen (which is known to lower calcium levels) during the luteal phase of the menstrual cycle (85). Although available evidence suggests that calcium may improve premenstrual symptoms, data are very limited and there is not expert consensus on its utility (43, 45, 47). However, given its proven beneficial effects on bone and general health (86) and low risk of harm when intake remains within recommended limits, calcium supplementation (1,000–1,200 mg/d) can be considered as part of a holistic approach to the management of premenstrual symptoms in adults, particularly for those who decline or have contraindications to medical therapies. Patients who are interested in calcium supplementation should be advised that the Institute of Medicine (now known as the National Academy of Medicine) recommends that adults aged 19–50 years consume no more than 2,500 mg/d of calcium from all sources, including food, beverages, and supplements (87).

Most of the evidence to support calcium supplementation for the management of premenstrual symptoms comes from one double-blind, placebo-controlled RCT that included 466 women (aged 18–45 years) with PMS from 12 medical centers in the northern and southern regions of the United States (88). By the third treatment cycle, calcium supplementation (1,200 mg/d) was associated with a greater reduction in symptom scores from baseline compared with placebo: negative affect (45% vs 28%, $P<.001$), water retention (36% vs 24%, $P<.001$), food cravings (54% vs 34%, $P<.05$), and pain (54% vs −15%, $P<.001$). Adverse reactions were generally minor and included headache, nausea, constipation, and gastrointestinal discomfort (88). Similarly, an earlier smaller RCT (N=33) demonstrated that 3 months of daily calcium supplementation (1,000 mg/d) was associated with a small but statistically significant decrease in premenstrual symptom scores for negative affect ($P=.045$), water retention ($P=.003$), and pain ($P=.036$) (89). However, a more recent double-blind pilot study that included 39 women aged 18–39 years with moderate-to-severe PMS who were randomized to receive fluoxetine (20 mg/d), calcium (1,200 mg/d), or placebo found that, although calcium did show a small benefit, it was not associated with a statistically significant improvement in symptom scores (90).

ACOG suggests adequate calcium intake in adolescents to help manage physical premenstrual symptoms. (CONDITIONAL RECOMMENDATION, LOW-QUALITY EVIDENCE)

Calcium supplementation has not been studied as a treatment for premenstrual symptoms in adolescents. However, very limited evidence suggests that adolescents who consume the recommended daily allowance of calcium through dietary sources may experience a reduction in physical premenstrual symptoms (19, 91). Given that calcium also has proven benefits for bone and overall health and a low risk of harm if intake is kept to daily recommended limits, it is reasonable to advise adolescent patients to consume the recommended daily allowance of calcium (1,300 mg not to exceed 2,000 mg) (87) as a nutritional option to help manage food cravings and physical premenstrual symptoms.

In a cross-sectional study that included 105 adolescents aged 10–17 years who met *Diagnostic and Statistical Manual of Mental Disorders* diagnostic criteria for PMDD, participants who consumed more than 200 mL of milk, 300 mL of yogurt, and 50 g of cheese per day reported a lower frequency of abdominal bloating ($P=.017$), cramping ($P=.017$), food cravings ($P=.021$), and increased appetite ($P=.021$) (19).

Acupuncture

ACOG suggests the use of acupuncture to help manage physical and affective premenstrual symptoms. (CONDITIONAL RECOMMENDATION, LOW-QUALITY EVIDENCE)

Acupuncture may mitigate premenstrual symptoms through several pathways, including modulation of endogenous opioid, prostaglandin, and inflammatory marker levels (92). Although available RCT evidence suggests that acupuncture is associated with improvement in physical and affective premenstrual symptoms (92, 93), certainty in its benefits is limited by the low quality and high heterogeneity of the studies. Because of these limitations, some experts advise that further study is needed before a recommendation can be made on the use of acupuncture for the management of premenstrual symptoms (43, 45). However, given acupuncture's potential benefits and low risk of harm, it is reasonable to include acupuncture as part of a holistic approach to premenstrual symptom management, particularly for patients who decline or are unable to receive medical treatment. Patient–clinician shared decision making should include consideration of treatment cost because many health insurance plans do not include coverage for acupuncture (94). For patients who must pay out of pocket, the cost of treatment may outweigh its potential benefits.

A Cochrane systematic review that reported results from one RCT (N=67) that compared two to three weekly sessions of acupuncture treatment for 2 months with sham acupuncture found that active treatment was associated with a greater reduction in premenstrual affective symptom scores (MD −9.03, 95% CI −10.71 to −7.35) and physical symptom scores (MD −9.11, 95% CI −10.82 to −7.40) and greater improvement in quality-of-life scores (MD 2.85, 95% CI 1.47–4.23) (92). No serious adverse events were reported across studies (92). Several other RCTs were included in the review, but meta-analysis was not possible because of the high heterogeneity in intervention characteristics and outcome measures across studies. Overall certainty in the evidence was very low given the high heterogeneity as well as the methodologic limitations of the included studies (small sample sizes and risk of bias related to selection and blinding).

In another systematic review and meta-analysis, participants who received acupuncture (approximately 30 sessions over three menstrual cycles) were more likely to experience premenstrual symptom improvement than those who received progestin treatment with medroxyprogesterone (4–6 mg/d) (pooled RR 1.49, 95% CI 1.27–1.74; four RCTs, N=232, $P<.001$) (93). The addition of acupuncture to progestin treatment (medroxyprogesterone, 6 mg/d) was associated with greater symptom improvement than progestin treatment alone (RR 1.52, 95% CI 1.04–2.22; one trial, N=44, $P=.03$) (93). The studies did not report on adverse events.

Vitex agnus castus

Vitex agnus castus, also known as chasteberry, has been studied as an herbal remedy for premenstrual symptoms. Although its exact mechanism of action is unclear, it is believed to relieve premenstrual symptoms through stimulation of dopamine receptors, which results in downstream modulation of prolactin and progesterone levels (95, 96). Evidence from low-to-moderate–quality RCTs demonstrates that *Vitex agnus castus* is well tolerated and associated with an improvement in premenstrual symptoms (95–97). However, a major limitation in interpreting the evidence is the high heterogeneity of formulations and dosages across studies. Given this uncertainty, ACOG agrees with other expert treatment guidelines that further study is needed before a recommendation can be made about the use of *Vitex agnus castus* for the management of premenstrual symptoms (43, 45, 47).

Nonsteroidal Anti-Inflammatory Drugs

ACOG suggests nonsteroidal anti-inflammatory drugs (NSAIDs) for the management of premenstrual pain symptoms. (CONDITIONAL RECOMMENDATION, LOW-QUALITY EVIDENCE)

Nonsteroidal anti-inflammatory drugs inhibit the production of prostaglandins, which mediate pain and inflammatory processes that are implicated in the pathophysiology of dysmenorrhea (98). Limited evidence from several low-quality randomized trials (small sample sizes, short duration, incomplete data reporting) shows that NSAIDs are associated with a reduction in physical premenstrual symptoms (eg, abdominal cramps, headaches, general body aches) as well as some mood-related premenstrual symptoms, which may be an indirect effect of pain improvement (99, 100). The most common adverse events associated with NSAID use in clinical trials include gastrointestinal effects (eg, nausea and indigestion) as well as headache, drowsiness, dizziness, and dryness (98, 101). Taking NSAIDs with food is advised to mitigate gastrointestinal effects (98). Thus, for healthy individuals with premenstrual pain symptoms, NSAIDs taken during the late luteal phase or as symptoms arise are a low-risk and readily available treatment option.

In a small RCT that compared 550 mg of naproxen sodium twice daily or placebo during the late luteal phase (ie, 7 days before expected menses until the fourth day of menses), participants in the naproxen group reported significantly lower premenstrual symptom scores for pain (naproxen 3.2±0.9 vs placebo 7.6±1.3; N=28, $P<.006$) and behavioral changes (naproxen 1.9±0.6 vs placebo 4.4±0.8, $P<.015$) (102). However, naproxen was not associated with an improvement in negative mood (naproxen 6.5±1.1 vs placebo 10.7±2.0, $P<.066$). The study reported no significant adverse effects, although two participants dropped out of the study because of nausea and abdominal pain (102).

A small (N=15) randomized double-blind crossover trial (in which participants served as their own controls) demonstrated that luteal phase dosing of the NSAID mefenamic acid (750 mg/d) was associated with statistically significant improvement in patient-reported physical symptoms such as headache, fatigue, and generalized pain ($P<.001$); mood swings ($P<.005$); and behavioral symptoms such as impaired performance ($P<.05$) (103).

Educational and Self-Help Strategies

ACOG suggests that clinicians provide patient education about premenstrual symptoms and self-help coping strategies as part of a holistic approach to the management of premenstrual disorders. (CONDITIONAL RECOMMENDATION, LOW-QUALITY EVIDENCE)

Studies on educational interventions for premenstrual-symptom reduction are limited and report mixed results (74, 104, 105). However, given that there are potential benefits with no associated harms, it is reasonable to include patient education as part of a holistic management approach. Patient education about premenstrual symptoms and how they may affect daily life can help provide validation that there is a physiologic basis for what patients are experiencing, and there may be reassurance in knowing that symptoms are predictable (91). In addition, counseling about coping strategies such as stress management and relaxation techniques, exercise, and dietary changes may help patients manage their symptoms (91) and know when to seek medical attention (106). For more information, see patient resources on premenstrual disorders from ACOG (107) and the International Association for Premenstrual Disorders (108).

Patient-education interventions were not associated with a statistically significant improvement in overall premenstrual symptom scores in a meta-analysis of two RCTs with adult participants (N=75, mean age 33–36 years) and one nonrandomized controlled trial with adolescent participants (N=94, age range 14–18 years) (SMD −0.12, 95% CI −0.42 to 0.18) (74). The educational interventions included information about premenstrual symptoms and self-care strategies such as dietary changes and exercise.

However, findings from two more recent RCTs suggest that patient-education programs based on the health belief model, which aims to address perceived barriers to implementing health-promoting behaviors, may be beneficial for adolescents and young adults with premenstrual symptoms (104, 105). In an RCT that included 60 older adolescents and women (aged older than 18 years), four educational sessions (45–60 minutes each on alternating weeks) were associated with gradual, statistically significant reductions in premenstrual symptom scores at 3-month follow-up compared with the usual-care control group (107.16±28.49 vs 124.13±27.80, $P<.05$) (104). The patient-education program provided information on premenstrual symptoms and their health effects; lifestyle coping strategies involving exercise, dietary changes, and improved sleep habits; and how to overcome potential barriers to implementation of these lifestyle interventions.

Similarly, in another RCT of 163 women (mean age, 21–22 years), a patient-education intervention based on the health belief model was associated with a reduction in overall premenstrual symptom scores when delivered by itself (−23.78±12.65) and when combined with acupressure (−30.31±3.5), whereas an increase in symptom scores was observed in the usual care control group (+3.24±4.42) ($P<.001$) (105). Participants in both intervention groups also experienced statistically significant increases in psychological, social, and environmental quality-of-life scores compared with those in the control group ($P<.001$) (105).

Surgical Management

Bilateral oophorectomy with or without hysterectomy should be reserved as a treatment option for adults with severe premenstrual symptoms only when medical management has failed and patients have been counseled about the associated risks and irreversibility of the procedure. A trial period of GnRH agonist therapy (with or without estrogen add-back treatment) is advised before surgery to predict a patient's response to surgical management. (GOOD PRACTICE POINT)

Bilateral oophorectomy induces permanent anovulation, which eliminates the premenstrual phase and its concomitant symptoms. Although RCTs have not evaluated the efficacy of bilateral oophorectomy for the management of premenstrual disorders, indirect evidence from GnRH agonist treatment studies demonstrates that induced anovulation is associated with a significant reduction in premenstrual symptoms (73). Because of the associated risks and permanence of the procedure, bilateral oophorectomy should be reserved for adult patients who have severe symptoms that are refractory to medical management (43, 47), patients in whom long-term GnRH agonist therapy is required for symptom management (43), or patients with severe symptoms who have other indications for bilateral oophorectomy (43). A trial of GnRH agonist therapy (with or without estrogen add-back treatment) should be considered before surgical management to predict response to surgery (43, 47). Based on the results of studies of GnRH agonist therapy for premenstrual symptoms, a trial period of 2–3 months should be sufficient to demonstrate a treatment response (or lack thereof) (73).

Patient–clinician shared decision making should include discussion about the need for hormonal add-back therapy after surgery to mitigate hypoestrogenic

effects such as vasomotor symptoms and decreased bone density. Evidence suggests that the progestin component of combination add-back therapy may be associated with premenstrual symptom recurrence (27, 72); therefore, the option of concomitant hysterectomy (to allow for estrogen-only add-back therapy) should be discussed with patients who are considering surgical management.

REFERENCES

1. O'Brien PM, Bäckström T, Brown C, Dennerstein L, Endicott J, Epperson CN, et al. Towards a consensus on diagnostic criteria, measurement and trial design of the premenstrual disorders: the ISPMD Montreal consensus. Arch Womens Ment Health 2011;14:13–21. doi: 10.1007/s00737-010-0201-3
2. Freeman EW, Halberstadt SM, Rickels K, Legler JM, Lin H, Sammel MD. Core symptoms that discriminate premenstrual syndrome. J Women's Health 2011;20:29–35. doi: 10.1089/jwh.2010.2161
3. Halbreich U, Backstrom T, Eriksson E, O'brien S, Calil H, Ceskova E, et al. Clinical diagnostic criteria for premenstrual syndrome and guidelines for their quantification for research studies. Gynecol Endocrinol 2007;23:123–30. doi: 10.1080/09513590601167969
4. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. APA; 2013.
5. World Health Organization International statistical classification of diseases and related health problems (ICD). Accessed July 19, 2023. https://www.who.int/standards/classifications/classification-of-diseases
6. Clinical practice guideline methodology: methodology. Obstet Gynecol 2021;138:518–22. doi: 10.1097/AOG.0000000000004519
7. United Nations Development Programme. Human development index (HDI). Accessed July 19, 2023 https://hdr.undp.org/data-center/human-development-index#/indicies/HDI
8. Guyatt GH, Oxman AD, Vist GE, Kunz R, Falck-Ytter Y, Alonso-Coello P, et al. GRADE: an emerging consensus on rating quality of evidence and strength of recommendations. BMJ 2008;336:924–6. doi: 10.1136/bmj.39489.470347.ad
9. Guyatt G, Oxman AD, Akl EA, Kunz R, Vist G, Brozek J, et al. GRADE guidelines: 1. Introduction-GRADE evidence profiles and summary of findings tables. J Clin Epidemiol 2011;64:383–94. doi: 10.1016/j.jclinepi.2010.04.026
10. Guyatt GH, Schünemann HJ, Djulbegovic B, Akl EA. Guideline panels should not GRADE good practice statements. J Clin Epidemiol 2015;68:597–600. doi: 10.1016/j.jclinepi.2014.12.011
11. Dennerstein L, Lehert P, Heinemann K. Epidemiology of premenstrual symptoms and disorders. Menopause Int 2012;18:48–51. doi: 10.1258/mi.2012.012013
12. Steiner M, Macdougall M, Brown E. The premenstrual symptoms screening tool (PSST) for clinicians. Arch Women's Ment Health 2003;6:203–9. doi: 10.1007/s00737-003-0018-4
13. Borenstein JE, Dean BB, Endicott J, Wong J, Brown C, Dickerson V, et al. Health and economic impact of the premenstrual syndrome. J Reprod Med 2003;48:515–24.
14. Epperson CN, Steiner M, Hartlage SA, Eriksson E, Schmidt PJ, Jones I, et al. Premenstrual dysphoric disorder: evidence for a new category for DSM-5. Am J Psychiatry 2012;169:465–75. doi: 10.1176/appi.ajp.2012.11081302
15. Halbreich U, Borenstein J, Pearlstein T, Kahn LS. The prevalence, impairment, impact, and burden of premenstrual dysphoric disorder (PMS/PMDD). Psychoneuroendocrinology 2003;28:1–23. doi: 10.1016/s0306-4530(03)00098-2
16. Borenstein J, Chiou C, Dean B, Wong J, Wade S. Estimating direct and indirect costs of premenstrual syndrome. J Occup Environ Med 2005;47:26–33. doi: 10.1097/01.jom.0000150209.44312.d1
17. Rapkin AJ, Winer SA. Premenstrual syndrome and premenstrual dysphoric disorder: quality of life and burden of illness. Expert Rev Pharmacoeconomics Outcomes Res 2009;9:157–70. doi: 10.1586/erp.09.14
18. Hylan TR, Sundell K, Judge R. The impact of premenstrual symptomatology on functioning and treatment-seeking behavior: experience from the United States, United Kingdom, and France. J Women's Health Gender-Based Med 1999;8:1043–52. doi: 10.1089/jwh.1.1999.8.1043
19. Derman O, Kanbur NO, Tokur TE, Kutluk T. Premenstrual syndrome and associated symptoms in adolescent girls. Eur J Obstet Gynecol Reprod Biol 2004;116:201–6. doi: 10.1016/j.ejogrb.2004.04.021
20. Yang J, Joe S, Lee M, Kim S, Jung I. Survey of premenstrual symptom severity and impairment in Korean adolescents: premenstrual dysphoric disorder, subthreshold premenstrual dysphoric disorder and premenstrual syndrome. Asia-Pacific Psychiatry 2014;6:135–44. doi: 10.1111/appy.12024
21. Gold EB, Bair Y, Block G, Greendale GA, Harlow SD, Johnson S, et al. Diet and lifestyle factors associated with premenstrual symptoms in a racially diverse community sample: study of women's health across the nation (SWAN). J Women's Health 2007;16:641–56. doi: 10.1089/jwh.2006.0202
22. Pilver CE, Kasl S, Desai R, Levy BR. Health advantage for Black women: patterns in pre-menstrual dysphoric disorder. Psychol Med 2011;41:1741–50. doi: 10.1017/S0033291710002321
23. Pilver CE, Desai R, Kasl S, Levy BR. Lifetime discrimination associated with greater likelihood of premenstrual dysphoric disorder. J Women's Health 2011;20:923–31. doi: 10.1089/jwh.2010.2456
24. Importance of social determinants of health and cultural awareness in the delivery of reproductive health care. ACOG Committee Opinion No. 729. American College of Obstetricians and Gynecologists. Obstet Gynecol 2018;131:e43–8. doi: 10.1097/AOG.0000000000002459
25. Paradies Y, Ben J, Denson N, Elias A, Priest N, Pieterse A, et al. Racism as a determinant of health: a systematic review and meta-analysis. PLoS One 2015;10:e0138511. doi: 10.1371/journal.pone.0138511
26. Yonkers KA, Simoni M. Evidence-based treatments for premenstrual disorders. Am J Obstet Gynecol 2018;219:215–6. doi: 10.1016/j.ajog.2018.04.018
27. Schmidt PJ, Martinez PE, Nieman LK, Koziol DE, Thompson KD, Schenkel L, et al. Premenstrual dysphoric disorder symptoms following ovarian suppression: triggered by change in ovarian steroid levels but not continuous stable levels. Am J Psychiatry 2017;174:980–9. doi: 10.1176/appi.ajp.2017.16101113
28. Hantsoo L, Epperson CN. Premenstrual dysphoric disorder: epidemiology and treatment. Curr Psychiatry Rep 2015;17:87–3. doi: 10.1007/s11920-015-0628-3
29. Marjoribanks J, Brown J, O'Brien PM, Wyatt K. Selective serotonin reuptake inhibitors for premenstrual syndrome. The Co-

chrane Database of Systematic Reviews 2013, Issue 6. Art. No: CD001396. doi: 10.1002/14651858.CD001396.pub3

30. Menkes DB, Coates DC, Fawcett JP. Acute tryptophan depletion aggravates premenstrual syndrome. J Affective Disord 1994;32:37–44. doi: 10.1016/0165-0327(94)90059-0

31. Hantsoo L, Epperson CN. Allopregnanolone in premenstrual dysphoric disorder (PMDD): evidence for dysregulated sensitivity to GABA-A receptor modulating neuroactive steroids across the menstrual cycle. Neurobiol Stress 2020;12: 100213. doi: 10.1016/j.ynstr.2020.100213

32. Bäckström T, Haage D, Löfgren M, Johansson IM, Strömberg J, Nyberg S, et al. Paradoxical effects of GABA-A modulators may explain sex steroid induced negative mood symptoms in some persons. Neuroscience 2011;191:46–54. doi: 10.1016/j.neuroscience.2011.03.061

33. Bäckström T, Ekberg K, Hirschberg AL, Bixo M, Epperson CN, Briggs P, et al. A randomized, double-blind study on efficacy and safety of sepranolone in premenstrual dysphoric disorder. Psychoneuroendocrinology 2021;133:105426. doi: 10.1016/j.psyneuen.2021.105426

34. Martinez PE, Rubinow DR, Nieman LK, Koziol DE, Morrow AL, Schiller CE, et al. 5α-Reductase inhibition prevents the luteal phase increase in plasma allopregnanolone levels and mitigates symptoms in women with premenstrual dysphoric disorder. Neuropsychopharmacology 2016;41:1093–102. doi: 10.1038/npp.2015.246

35. Bixo M, Ekberg K, Poromaa IS, Hirschberg AL, Jonasson AF, Andr en L, et al. Treatment of premenstrual dysphoric disorder with the GABA(A) receptor modulating steroid antagonist Sepranolone (UC1010)-a randomized controlled trial. Psychoneuroendocrinology 2017;80:46–55. doi: 10.1016/j.psyneuen.2017.02.031

36. Bäckström T, Das R, Bixo M. Positive GABA(A) receptor modulating steroids and their antagonists: implications for clinical treatments. J Neuroendocrinology 2022;34:e13013. doi: 10.1111/jne.13013

37. Gracia CR, Freeman EW, Sammel MD, Lin H, Sheng L, Frye C. Allopregnanolone levels before and after selective serotonin reuptake inhibitor treatment of premenstrual symptoms. J Clin Psychopharmacol 2009;29:403–5. doi: 10.1097/JCP.0b013e3181ad8825

38. Griffin LD, Mellon SH. Selective serotonin reuptake inhibitors directly alter activity of neurosteroidogenic enzymes. Proc Natl Acad Sci U S A 1999;96:13512–7. doi: 10.1073/pnas.96.23.13512

39. Yonkers KA, Kornstein SG, Gueorguieva R, Merry B, Van Steenburgh K, Altemus M. Symptom-onset dosing of sertraline for the treatment of premenstrual dysphoric disorder: a randomized clinical trial. JAMA Psychiatry 2015;72:1037–44. doi: 10.1001/jamapsychiatry.2015.1472

40. Prasad D, Wollenhaupt-Aguiar B, Kidd KN, de Azevedo Cardoso T, Frey BN. Suicidal risk in women with premenstrual syndrome and premenstrual dysphoric disorder: a systematic review and meta-analysis. J Women's Health 2021;30:1693–707. doi: 10.1089/jwh.2021.0185

41. Eisenlohr-Moul T, Divine M, Schmalenberger K, Murphy L, Buchert B, Wagner-Schuman M, et al. Prevalence of lifetime self-injurious thoughts and behaviors in a global sample of 599 patients reporting prospectively confirmed diagnosis with premenstrual dysphoric disorder. BMC Psychiatry 2022;22:199. doi: 10.1186/s12888-022-03851-0

42. Rubinow DR, Roy-Byrne P. Premenstrual syndromes: overview from a methodologic perspective. Am J Psychiatry 1984;141: 163–72. doi: 10.1176/ajp.141.2.163

43. Management of premenstrual syndrome: green-top guideline No. 48. BJOG 2017;124:e73–105. doi: 10.1111/1471-0528.14260

44. Endicott J, Nee J, Harrison W. Daily record of severity of problems (DRSP): reliability and validity. Arch Womens Ment Health 2006;9:41–9. doi: 10.1007/s00737-005-0103-y

45. Hofmeister S, Bodden S. Premenstrual syndrome and premenstrual dysphoric disorder. Am Fam Physician 2016;94: 236–40.

46. Hantsoo L, Sajid H, Murphy L, Buchert B, Barone J, Raja S, et al. Patient experiences of health care providers in premenstrual dysphoric disorder: examining the role of provider specialty. J Women's Health 2022;31:100–9. doi: 10.1089/jwh.2020.8797

47. Nevatte T, O'Brien PM, Bäckström T, Brown C, Dennerstein L, Endicott J, et al. ISPMD consensus on the management of premenstrual disorders. Arch Womens Ment Health 2013;16: 279–91. doi: 10.1007/s00737-013-0346-y

48. Freeman EW, Rickels K, Sondheimer SJ, Polansky M, Xiao S. Continuous or intermittent dosing with sertraline for patients with severe premenstrual syndrome or premenstrual dysphoric disorder. Am J Psychiatry 2004;161:343–51. doi: 10.1176/appi.ajp.161.2.343

49. Kornstein SG, Pearlstein TB, Fayyad R, Farfel GM, Gillespie JA. Low-dose sertraline in the treatment of moderate-to-severe premenstrual syndrome: efficacy of 3 dosing strategies. J Clin Psychiatry 2006;67:1624–32. doi: 10.4088/jcp.v67n1020

50. National Library of Medicine. Sertraline hydrochloride tablet. DailyMed NLM 2023. Accessed July 19, 2023 https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=1f99e897-1b70-40b0-8c19-71d0bba19422

51. National Library of Medicine. Paroxetine hydrochloride tablet, film coated, extended release. DailyMed NLM; 2022. Accessed July 19, 2023 https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=087ff0d3-1761-47ea-a8c1-7c4cb679af97

52. National Library of Medicine. Fluoxetine tablet. DailyMed NLM; 2023. Accessed July 19, 2023 https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=dcebc7cf-6f95-4f7a-a22b-c7d2e0f04d74

53. Cohen LS, Soares CN, Yonkers KA, Bellew KM, Bridges IM, Steiner M. Paroxetine controlled release for premenstrual dysphoric disorder: a double-blind, placebo-controlled trial. Psychosomatic Med 2004;66:707–13. doi: 10.1097/01.psy.0000140005.94790.9c

54. Freeman EW, Rickels K, Yonkers KA, Kunz NR, McPherson M, Upton GV. Venlafaxine in the treatment of premenstrual dysphoric disorder. Obstet Gynecol 2001;98:737–44. doi: 10.1016/s0029-7844(01)01530-7

55. Yonkers KA, Pearlstein T, Fayyad R, Gillespie JA. Luteal phase treatment of premenstrual dysphoric disorder improves symptoms that continue into the postmenstrual phase. J Affective Disord 2005;85:317–21. doi: 10.1016/j.jad.2004.10.006

56. Reilly TJ, Wallman P, Clark I, Knox C, Craig MC, Taylor D. Intermittent selective serotonin reuptake inhibitors for premenstrual syndromes: a systematic review and meta-analysis of randomised trials. J Psychopharmacol 2023;37:261–7. doi: 10.1177/02698811221099645

57. Freeman EW, Rickels K, Sammel MD, Lin H, Sondheimer SJ. Time to relapse after short- or long-term treatment of severe

premenstrual syndrome with sertraline. Arch Gen Psychiatry 2009;66:537–44. doi: 10.1001/archgenpsychiatry.2008.547

58. U.S. Food and Drug Administration. Suicidality in children and adolescents being treated with antidepressant medications 2018. Accessed July 19, 2023 https://www.fda.gov/drugs/postmarket-drug-safety-information-patients-and-providers/suicidality-children-and-adolescents-being-treated-antidepressant-medications

59. Walter HJ, Abright AR, Bukstein OG, Diamond J, Keable H, Ripperger-Suhler J, et al. Clinical practice guideline for the assessment and treatment of children and adolescents with major and persistent depressive disorders. J Am Acad Child Adolesc Psychiatry 2023;62:479–502. doi: 10.1016/j.jaac.2022.10.001

60. Walter HJ, Bukstein OG, Abright AR, Keable H, Ramtekkar U, Ripperger-Suhler J, et al. Clinical practice guideline for the assessment and treatment of children and adolescents with anxiety disorders. J Am Acad Child Adolesc Psychiatry 2020;59:1107–24. doi: 10.1016/j.jaac.2020.05.005

61. Cheung AH, Zuckerbrot RA, Jensen PS, Laraque D, Stein RE, Levitt A, et al. Guidelines for adolescent depression in primary care (GLAD-PC): Part II. Treatment and ongoing management. Pediatrics 2018;141:e20174082. doi: 10.1542/peds.2017-4082

62. Ford O, Lethaby A, Roberts H, Mol BW. Progesterone for premenstrual syndrome. The Cochrane Database of Systematic Reviews 2012, Issue 3. Art. No.: CD003415. doi: 10.1002/14651858.CD003415.pub4

63. Wyatt K, Dimmock P, Jones P, Obhrai M, O'Brien S. Efficacy of progesterone and progestogens in management of premenstrual syndrome: systematic review. BMJ 2001;323:776–80. doi: 10.1136/bmj.323.7316.776

64. Leminen H, Heliövaara-Peippo S, Halmesmäki K, Teperi J, Grenman S, Kivelä A, et al. The effect of hysterectomy or levonorgestrel-releasing intrauterine system on premenstrual symptoms in women treated for menorrhagia: secondary analysis of a randomized controlled trial. Acta Obstet Gynecol Scand 2012;91:318–25. doi: 10.1111/j.1600-0412.2011.01340.x

65. Naheed B, Kuiper JH, Uthman OA, O'Mahony F, O'Brien PMS. Non-contraceptive oestrogen-containing preparations for controlling symptoms of premenstrual syndrome. The Cochrane Database of Systematic Reviews 2017, Issue 3. Art. No.: CD010503. doi: 10.1002/14651858.CD010503.pub2

66. de Wit AE, de Vries YA, de Boer MK, Scheper C, Fokkema A, Janssen CA, et al. Efficacy of combined oral contraceptives for depressive symptoms and overall symptomatology in premenstrual syndrome: pairwise and network meta-analysis of randomized trials. Am J Obstet Gynecol 2021;225:624–33. doi: 10.1016/j.ajog.2021.06.090

67. Ma S, Song SJ. Oral contraceptives containing drospirenone for premenstrual syndrome. The Cochrane Database of Systematic Reviews 2023, Issue 6. Art. No.: CD006586. doi: 10.1002/14651858.CD006586.pub5

68. National Library of Medicine. Drospirenone and ethinyl estradiol kit. DailyMed. NLM; 2023. Accessed July 19, 2023. https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=0f8f8a21-cee8-462f-98b3-6c06f2f33e0d

69. Dehlendorf C, Kimport K, Levy K, Steinauer J. A qualitative analysis of approaches to contraceptive counseling. Perspect Sex Reprod Health 2014;46:233–40. doi: 10.1363/46e2114

70. Curtis KM, Jatlaoui TC, Tepper NK, Zapata LB, Horton LG, Jamieson DJ, et al. U.S. selected practice recommendations for contraceptive use, 2016. MMWR Recomm Rep 2016;65:1–66. doi: 10.15585/mmwr.rr6504a1

71. Curtis KM, Tepper NK, Jatlaoui TC, Berry-Bibee E, Horton LG, Zapata LB, et al. U.S. medical eligibility criteria for contraceptive use, 2016. MMWR Recomm Rep 2016;65:1–103. doi: 10.15585/mmwr.rr6503a1

72. Segebladh B, Borgström A, Nyberg S, Bixo M, Sundström-Poromaa I. Evaluation of different add-back estradiol and progesterone treatments to gonadotropin-releasing hormone agonist treatment in patients with premenstrual dysphoric disorder. Am J Obstet Gynecol 2009;201:139.e1–8. doi: 10.1016/j.ajog.2009.03.016

73. Wyatt KM, Dimmock PW, Ismail KM, Jones PW, O'Brien PS. The effectiveness of GnRHa with and without 'add-back' therapy in treating premenstrual syndrome: a meta analysis. BJOG: Int J Obstet Gynaecol 2004;111:585–93. doi: 10.1111/j.1471-0528.2004.00135.x

74. Han J, Cha Y, Kim S. Effect of psychosocial interventions on the severity of premenstrual syndrome: a meta-analysis. J Psychosomatic Obstet Gynecol 2019;40:176–84. doi: 10.1080/0167482X.2018.1480606

75. Busse JW, Montori VM, Krasnik C, Patelis-Siotis I, Guyatt GH. Psychological intervention for premenstrual syndrome: a meta-analysis of randomized controlled trials. Psychother Psychosom 2009;78:6–15. doi: 10.1159/000162296

76. Hunter MS, Ussher JM, Browne SJ, Cariss M, Jelley R, Katz M. A randomized comparison of psychological (cognitive behavior therapy), medical (fluoxetine) and combined treatment for women with premenstrual dysphoric disorder. J Psychosomatic Obstet Gynecol 2002;23:193–9. doi: 10.3109/01674820209074672

77. Ussher JM, Perz J. Evaluation of the relative efficacy of a couple cognitive-behaviour therapy (CBT) for premenstrual disorders (PMDs), in comparison to one-to-one CBT and a wait list control: a randomized controlled trial. PLoS One 2017;12:e0175068. doi: 10.1371/journal.pone.0175068

78. Weise C, Kaiser G, Janda C, Kues JN, Andersson G, Strahler J, et al. Internet-based cognitive-behavioural intervention for women with premenstrual dysphoric disorder: a randomized controlled trial. Psychother Psychosom 2019;88:16–29. doi: 10.1159/000496237

79. Yesildere Saglam H, Orsal O. Effect of exercise on premenstrual symptoms: a systematic review. Complement Therapies Med 2020;48:102272. doi: 10.1016/j.ctim.2019.102272

80. El-Lithy A, El-Mazny A, Sabbour A, El-Deeb A. Effect of aerobic exercise on premenstrual symptoms, haematological and hormonal parameters in young women. J Obstet Gynaecol 2015;35:389–92. doi: 10.3109/01443615.2014.960823

81. Pal A, Nath B, Paul S, Meena S. Evaluation of the effectiveness of yoga in management of premenstrual syndrome: a systematic review and meta-analysis. J Psychosomatic Obstet Gynecol 2022;43:517–25. doi: 10.1080/0167482X.2022.2086457

82. Daley A. Exercise and premenstrual symptomatology: a comprehensive review. J Women's Health 2009;18:895–9. doi: 10.1089/jwh.2008.1098

83. Kossman DA, Williams NI, Domchek SM, Kurzer MS, Stopfer JE, Schmitz KH. Exercise lowers estrogen and progesterone levels in premenopausal women at high risk of breast cancer. J Appl Physiol 2011;111:1687–93. doi: 10.1152/japplphysiol.00319.2011

84. U.S. Department of Health and Human Services. Physical activity guidelines for Americans. 2nd ed. U.S. Department of Health and Human Services; 2018.

85. Thys-Jacobs S. Micronutrients and the premenstrual syndrome: the case for calcium. J Am Coll Nutr 2000;19:220–7. doi: 10.1080/07315724.2000.10718920

86. Osteoporosis prevention, screening, and diagnosis. Clinical Practice Guideline No. 1. American College of Obstetricians and Gynecologists. Obstet Gynecol 2021;138:494–506. doi: 10.1097/AOG.0000000000004514

87. Ross AC, Taylor CL, Yaktine AL, Del Valle HB. Dietary reference intakes for calcium and vitamin D. National Academies Press; 2011. doi: 10.17226/13050

88. Thys-Jacobs S, Starkey P, Bernstein D, Tian J. Calcium carbonate and the premenstrual syndrome: effects on premenstrual and menstrual symptoms. Am J Obstet Gynecol 1998; 179:444–52. doi: 10.1016/s0002-9378(98)70377-1

89. Thys-Jacobs S, Ceccarelli S, Bierman A, Weisman H, Cohen MA, Alvir J. Calcium supplementation in premenstrual syndrome: a randomized crossover trial. J Gen Intern Med 1989;4:183–9. doi: 10.1007/BF02599520

90. Yonkers KA, Pearlstein TB, Gotman N. A pilot study to compare fluoxetine, calcium, and placebo in the treatment of premenstrual syndrome. J Clin Psychopharmacol 2013;33:614–20. doi: 10.1097/JCP.0b013e31829c7697

91. Rapkin AJ, Mikacich JA. Premenstrual dysphoric disorder and severe premenstrual syndrome in adolescents. Pediatr Drugs 2013;15:191–202. doi: 10.1007/s40272-013-0018-4

92. Armour M, Ee CC, Hao J, Wilson TM, Yao SS, Smith CA. Acupuncture and acupressure for premenstrual syndrome. The Cochrane Database of Systematic Reviews 2018, Issue 8. Art. No.: CD005290. doi: 10.1002/14651858.CD005290.pub2

93. Kim S, Park H, Lee H, Lee H. Acupuncture for premenstrual syndrome: a systematic review and meta-analysis of randomised controlled trials. BJOG: Int J Obstet Gynaecol 2011;118: 899–915. doi: 10.1111/j.1471-0528.2011.02994.x

94. Candon M, Nielsen A, Dusek JA. Trends in insurance coverage for acupuncture, 2010-2019. JAMA Netw Open 2022;5: e2142509. doi: 10.1001/jamanetworkopen.2021.42509

95. Csupor D, Lantos T, Hegyi P, Benkő R, Viola R, Gyöngyi Z, et al. Vitex agnus-castus in premenstrual syndrome: a meta-analysis of double-blind randomised controlled trials. Complement Therapies Med 2019;47:102190. doi: 10.1016/j.ctim.2019.08.024

96. Verkaik S, Kamperman AM, van Westrhenen R, Schulte PF. The treatment of premenstrual syndrome with preparations of Vitex agnus castus: a systematic review and meta-analysis. Am J Obstet Gynecol 2017;217:150–66. doi: 10.1016/j.ajog.2017.02.028

97. van Die MD, Burger HG, Teede HJ, Bone KM. Vitex agnus-castus extracts for female reproductive disorders: a systematic review of clinical trials. Planta Med 2012;79:562–75. doi: 10.1055/s-0032-1327831

98. Marjoribanks J, Ayeleke RO, Farquhar C, Proctor M. Nonsteroidal anti-inflammatory drugs for dysmenorrhoea. The Cochrane Database of Systematic Reviews 2015. Issue 7. Art. No.: CD001751. doi: 10.1002/14651858.CD001751.pub3

99. Jarvis CI, Lynch AM, Morin AK. Management strategies for premenstrual syndrome/premenstrual dysphoric disorder. Ann Pharmacother 2008;42:967–78. doi: 10.1345/aph.1k673

100. Kwan I, Onwude JL. Premenstrual syndrome. BMJ Clin Evid 2009;2009:806.

101. National Library of Medicine. Naproxen sodium tablet. DailyMed. NLM; 2010. Accessed July 19, 2023 https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=50954ade-19ad-40dd-8e06-0b33394895f7

102. Facchinetti F, Fioroni L, Sances G, Romano G, Nappi G, Genazzani AR. Naproxen sodium in the treatment of premenstrual symptoms. Gynecol Obstet Invest 1989;28:205–8. doi: 10.1159/000293579

103. Mira M, McNeil D, Fraser IS, Vizzard J, Abraham S. Mefenamic acid in the treatment of premenstrual syndrome. Obstet Gynecol 1986;68:395–8. doi: 10.1097/00006250-198609000-00022

104. Ayaz-Alkaya S, Yaman-Sözbir Ş, Terzi H. The effect of Health Belief Model-based health education programme on coping with premenstrual syndrome: a randomised controlled trial. Int J Nurs Pract 2020;26:e12816. doi: 10.1111/ijn.12816

105. Simsek Kücükkelepce D, Timur Tashan S. The effects of health belief model-based education and acupressure for coping with premenstrual syndrome on premenstrual symptoms and quality of life: a randomized-controlled trial. Perspect Psychiatr Care 2021;57:189–97. doi: 10.1111/ppc.12546

106. Houston AM, Abraham A, Huang Z, D'Angelo LJ. Knowledge, attitudes, and consequences of menstrual health in urban adolescent females. J Pediatr Adolesc Gynecol 2006;19: 271–5. doi: 10.1016/j.jpag.2006.05.002

107. American College of Obstetricians and Gynecologists. Premenstrual syndrome (PMS) FAQs. Accessed July 19, 2023 https://www.acog.org/womens-health/faqs/premenstrual-syndrome

108. International Association for Premenstrual Disorders. Patient and partner resources. Accessed September 19, 2023. https://iapmd.org/toolkit

APPENDICES

Supplemental Digital Content

A. Literature search strategy: http://links.lww.com/AOG/D430

B. PRISMA diagram: http://links.lww.com/AOG/D431

C. Evidence tables: http://links.lww.com/AOG/D432

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