ARTÍCULO DE REVISIÓN

Gac Méd Caracas 2022;130(3):586-601
DOI: 10.47307/GMC.2022.130.3.5

Premenstrual Syndrome and Premenstrual Dysphoric Disorders: A Narrative Review of Etiology, Pathophysiology, and Diagnosis

Síndrome Premenstrual y Desórdenes Disfóricos Premenstruales: Una Revisión Narrativa de la Etiología, Fisiopatología y Diagnóstico

José Núñez-Troconis

SUMMARY

The objective was to review and analyze narratively the etiology, pathophysiology, and diagnosis of premenstrual syndrome and premenstrual dysphoric disorder. For that purpose, Latin-American and international references were reviewed on different web pages. Publications from 1995 to April 2022 were reviewed. Premenstrual syndrome and premenstrual dysphoric disorders are of theories diseases in medicine and are found among menstruating women of all ages. Symptoms of PMS vary from mild to severe enough to interfere with daily personal and occupational life. This review analyses the possible etiologies, the pathophysiological mechanism, and how to make its diagnosis of premenstrual syndrome and premenstrual dysphoric disorders.

Keywords: *Premenstrual syndrome, premenstrual dysphoric disorders, menstrual cycle, etiology, pathophysiology, diagnosis*

RESUMEN

El objetivo fue revisar y analizar narrativamente la etiología, la fisiopatología y diagnóstico del síndrome de tensión premenstrual y el síndrome de desórdenes disfórico premenstrual. Para este propósito, se revisaron referencias latinoamericanas e internacionales en diferentes páginas electrónicas. Se revisaron publicaciones desde 1995 a abril 2022. El síndrome de tensión premenstrual y el síndrome de desórdenes disfórico premenstrual son una de enfermedades de teorías en medicina y son encontrados en mujeres en edad reproductiva. Los síntomas del síndrome de tensión premenstrual varían desde leve a suficientemente severos capaces de interferir con las actividades diarias. Esta revisión analiza las posibles etiologías, la fisiopatología y como se realiza el diagnóstico del síndrome de tensión premenstrual y de los desórdenes disfóricos premenstrual.

Palabras clave: *Síndrome de tensión premenstrual, desórdenes disfóricos premenstrual, ciclo menstrual, etiología, fisiopatología, diagnóstico.*

DOI: https://doi.org/10.47307/GMC.2022.130.3.5

ORCID: 0000-0002-5334-7265

Profesor Titular. Departamento de Ginecología y Obstetricia, Facultad de Medicina
Universidad del Zulia, Maracaibo, Venezuela
Miembro Correspondiente Nacional de la Academia Nacional de Medicina No XXXVII
Miembro Emérito de la Academia de Medicina del Zulia

Autor Responsable: E-mail: jtnunezt@gmail.com
Tel: 0414-361-5015

Recibido: 27 de julio 2022
Aceptado: 13 de agosto 2022

INTRODUCTION

There are three disorders related to menstruation or menstrual period: premenstrual syndrome or premenstrual dysphoric disorder, premenstrual migraine, and dysmenorrhea (1). Frank (2) has been mentioned as the first who described the premenstrual syndrome (PMS) in 1931, however, the phrase "premenstrual syndrome" was used first by Greene and Dalton (3) in their report of 84 cases in 1953, at that time it was used the term "Premenstrual Tension" and they (3,4) defined it as the commonest of the minor endocrine disorders.

A large number of reproductive-age women experience at least some form of menstrual symptoms. PMS is considered a common cyclical and recurrent health problem in women in their reproductive years and has been described as a group of predictable physical, cognitive, affective, and behavioral symptoms that occur cyclically during the luteal phase of the menstrual cycle and resolve at or within a few days of the onset of menstruation (5-7). Speroff (1) states "the simplest definition of the premenstrual syndrome is a common sense one: cyclic physical and behavioral symptoms that appear in the days preceding menses, are bothersome enough to interfere with work or lifestyle, and are followed by a symptom-free interval". Other authors (8) defined PMS as a collection of emotional symptoms, with or without physical symptoms. To date, there is no universally accepted definition or diagnostic criteria for PMS (5,9).

The severity of PMS is influenced by age, race, ethnicity, and health status, particularly mental health (10), and, therefore, varies in the population, but symptoms are relatively constant in each woman over consecutive cycles, particularly for emotional symptoms. The severity of PMS symptoms varies from very mild or minor symptoms known as premenstrual moliminia to severe symptoms call premenstrual dysphoric disorder (PMDD (1,11). The World Health Organization's International Classification of Diseases used ICD-9 code 625.4 for Premenstrual Tension Syndrome and lists PMS and PMDD under this heading; there was no separate diagnostic code for PMS or PMDD (5). However, in 2019 the WHO change the classification of PMDD from ICD-10 to ICD-11 to differentiate it from the more common PMS by the severity of the symptoms and the requirement that they cause significant distress or impairment (12).

Since Mortola's works (13,14), more specific criteria for the diagnosis were established. Also, strict and rigorous scientific standards were designed to evaluate the pathophysiology and establish treatments. Different authors (5,13-15) have mentioned that PMS is not a simple condition but a set of interrelated symptom complexes with multiples and different genotypes, phenotypes or subtypes, and several different pathophysiologic events which begin with ovulation.

In 1987, the American Psychiatric Association (16) in its *Diagnostic and Statistical Manual of Mental Disorders, Third Edition Revised* (DSM-III-R) changed the diagnostic criteria of severe luteal phase symptoms and the name of PMS to luteal phase dysphoric disorder. In 1994, the association reviewed these criteria in the fourth edition (DSM-IV-R), and the name was changed again to premenstrual dysphoric disorder (PMDD) (15,17). The criteria established by APA require at least 5 symptoms, and at least one of them must be depressed mood, tension, affective lability, or irritability. Also, there must be impairment in at least one area of daily life. The premenstrual dysphoric disorder is presented in a small group of women but they are at the extreme end of PMS severity. Less rigorous definitions of PMS were subsequently published in the American College of Obstetricians and Gynecologists in 2000 (ACOG) (18); the World Health Organization International Classification of Diseases (ICD) 10th edition in 2004, and the Royal College of Obstetricians and Gynecologists criteria in 2007 (19). Although some authors (4) have mentioned that PMDD is a distinct clinical entity, however, PMDD has not been shown to be etiologically distinct from less severe adverse premenstrual symptoms (15). Premenstrual syndrome is mentioned in the DSM-IV-R discussion section for PMDD but it is not mentioned any specific diagnostic criteria. They mentioned (16,17) that the PMDD-relevant symptoms are mood problems, whereas, in PMS, physical symptoms play a more important role. So, PMDD is primarily a mood problem, and

PMS is primarily a physical one (15). However, the evidence does not support this conclusion. According to Johnson (15), premenstrual symptoms generally occur in groups and together which include physical, mood, and cognitive symptoms, and the distinguishing feature between groups of women is in the experienced severity and level of impairment of daily activities, rather than the type of symptom. The same author (15) said that luteal phase symptoms are more accurately classified as mild, moderate, and severe. PMDD is simply a severe PMS that affects dairy activity, and there is no need in clinical practice to make a distinction between severe PMS and premenstrual dysphoric disorder. So, PMS and PMDD are the same clinical entity with a broad spectrum of the syndrome (15).

MATERIAL AND METHODS

The present narrative review was conducted to investigate and analyzed recent and relevant studies about MSP and PMDD. Studies published in the English and Spanish languages were included in the review. In accordance with the PRISMA guidelines, we identified published studies through a systematic and electronical review of the literature searches of PubMed, Medline, ISI, DOAJ, Springer, and Embase. Web of Knowledge, DOAJ, and Google Scholar for original articles written in the English language and Scielo, Lantidex, Imbiomed-L, Redalyc, and Google Scholar for original articles written in the Spanish language. The searches included the keywords (Mesh): premenstrual syndrome, premenstrual tension, premenstrual dysphoric disorders, and the following search terms: ("premenstrual syndrome" AND "premenstrual dysphoric disorders OR etiology OR pathophysiology OR diagnosis").

Selection criteria included randomized clinical trials, observational trials, open-label non-randomized trials, and case reports. Publications from 1995 to April 2022 were reviewed and analyzed. Also, it was checked, reviewed, and included older references during the search when they were relevant to the review.

Premenstrual syndrome/ Premenstrual dysphoric disorder

Prevalence and Epidemiology

As we mentioned before PMS is a generic term that includes a broad group of emotional, behavioral, and physical symptoms that occur for several days to several weeks before menses and disappears following the menstrual period (9). The symptoms that occur repetitively during the luteal phase of the cycle are nearly universal among women who ovulate. Some are negative, but others are positive such as higher energy levels and increased productivity, increased sexual desire, more vivid dreaming, and increased creativity. Based on several large epidemiological studies, it appears that only about 10 % of women report no luteal phase symptoms, and approximately 50 % experience one or more symptoms that are mild, last only a few days, and are not bothersome; these women should not be diagnosed as having PMS (15).

Up to 90 percent of women report one or more physical, psychological, or behavioral symptoms during the luteal phase of their menstrual cycle without experiencing substantial disruption to their daily functioning (20) PMS is a common health problem in women of reproductive age and is defined as a collection of emotional symptoms, with or without physical symptoms, related to a woman's menstruation cycle, means that occurs during the luteal phase and disappear with menstrual flow. PMS, in which mild to moderate symptoms affect some facet or activities of the woman's life, occurs in 20 % to 32 % of premenopausal women and 30-40 % of the reproductive female population; the more severe symptoms of PMDD affect 3 to 8 percent of premenopausal women (20-23). PMS affects women's quality of life and economic and social performance. In another study, about 23 %-31 % of reproductive-aged women experience PMS to a degree that affects their daily lives (24). Also, PMS can cause mood disorders and their complications (25).

The symptoms are cyclic and recurrent. The symptoms can change in extent and strength during different cycles (26). Many women

experience PMS symptoms, particularly physical symptoms such as breast tenderness and swelling, at some time in their reproductive years, but do not perceive these symptoms as either distressing or debilitating (9). Although 50 %-90 % of reproductive-age women have at least mild premenstrual symptoms, approximately 30 %-40 % of women report PMS symptoms that are bothersome and required treatment, and 3 %-8 % of women suffer from PMDD that meets the strict DSM-IV criteria as we mentioned before (1,5,6,20,23). Angst et al. (23) reported that 8.1 % of the reproductive age women had severe psychological symptoms and 13.6 % of women mentioned had moderate symptoms.

PMS/PDMM appears to be most severe in the twenties to the mid-thirties. However, most women with premenstrual symptoms repress their symptoms without looking for medical support for diagnosis, management, and treatment (6). Freeman (9) mentions that PMS/PDMM women seek medical support approximately 10 years after they have the symptoms. This suggests that many women start to have PMS/PDMM symptomatology in their late teens or early twenties. Also, it is important to mention that the majority of women who seek care for this medical issue are over 30 years old and they describe that they have had the symptoms for several years. It is not clear whether the PMS/PMDD symptoms worsen with age or whether older women are more likely to ask for help for the problem. PMS/PMDD disappear after menopause however there is some evidence that perimenopausal symptoms may be more severe in women who have had severe PMS/PMDD symptoms (15). Deuster et al. (27) found a PMS prevalence of 8.7 % in the 18-24 years old group, 10.4 % in the 25-34 group, and 4.5 % in the 35-44 group. Eshetu et al. (28) found in university students between 17 to 26 years old a PMS prevalence of 37.9 % and half of them had a family history of PMS. However, Victor et al. (29) reported a prevalence higher, 49.9 %, in Brazilian university students between 18 to 24 years old; according to the authors 23.3 % had mild PMS, and 26.6 % had severe PMS or PMDD. Also, Rodriguez-Rezende et al. (30) mentioned a PMS and PMDD prevalence of 46.9 % and 11.1 %, respectively. Costanian et al. (31) found a prevalence of 63 % in university students in Lebanon between 17 to 29 years old, the PMS prevalence was higher in the age group between 17 to 21 years, and 42.5 % mentioned suffering from severe PMS.

Premenstrual disorders

The premenstrual symptoms are triggered by the rise and fall of ovarian sex steroids at the time of ovulation and do not remit until the onset of the next menstrual flow, often persisting until approximately day 5 of the following cycle therefore is a disorder depending on the luteal phase events following the ovulation. The phase of the menstrual cycle between ovulation and menses, the luteal phase, is physiologically determined to last between 12 and 14 days. Premenstrual symptoms, by definition, do not begin before ovulation and are most severe during the last few days of the luteal phase.

Symptoms can occur throughout this phase; there is a relatively symptom-free interval in the late follicular phase of the menstrual cycle, between the end of menses and the onset of ovulation (19,32).

As we have mentioned before, PMS presents with typical features and a wide range of both somatic and psychological symptoms. The key characteristic is their timing, which must occur during all or part of the 2-week premenstrual phase and resolve during or shortly after menstruation. The persistence of symptoms during menstruation does not exclude the diagnosis; however, there must be a clear, symptom-free interval between the end of menstruation and the approximate time of ovulation.

The length of PMS symptoms varies between 1-3 days and 2 weeks. Symptoms often worsen substantially 6 days before, and peak about 2 days before, menses start (33,34), and they could last until the last day of the menstrual period; there must be a symptom-free interval before ovulation. Typically, women have the same set of symptoms from one cycle to the next (35). Women can have true or apparent comorbidity with premenstrual symptoms: 1.- they might have another psychiatric disorder at another point in their life (lifetime comorbidity); 2.- they might have an ongoing psychiatric or general

medical condition but concurrent premenstrual symptoms are not part of the co-occurring disorder (concurrent comorbidity); 3.- they might have an ongoing psychiatric or general medical condition that becomes worse pre-menstrually, usually defined as premenstrual exacerbation (4,36). Estimates for lifetime comorbidity between PMS and other mood disorders range from 30 % to 70 % (23,37,38). This prevalence is higher than it is expected, even taking into consideration that mood disorders are common in women (at least 30 % of women have a minor or major depressive disorder at some point in their lives) (20); estimates of comorbidity, however, might be inflated because of an overlap in symptoms. Notably, the risk of developing perimenopausal depression (39) and postnatal depression (40,41) has been reported to be higher in women who have PMS, leading some to suggest that these different conditions share a vulnerability to changes in gonadal steroid concentrations.

Etiology and Pathophysiology

PMS and PMDD are the theories of diseases in medicine, the theories list is seen in Table 1. As we mentioned, the etiology of PMS and PMDD is unknown, but the onset of symptoms is associated with ovarian hormone levels as we mentioned before, and the etiology may be complex and multifactorial (5,26,42). Women with PMS are hypersensitive to the normal hormonal level changes that occur during the menstruation cycle and express more symptoms with normal cycling levels of estrogen and progesterone (21,42,43). However, the role of ovarian hormones is unclear, but symptoms often improve when ovulation is suppressed or inhibited, during pregnancy and after menopause (21,26,42,44,45). Different authors (21,46) mention that PMS symptoms may alter or change as women are close to menopause; however, women who experienced PMS appear to have a greater risk of menopausal symptoms associated with hormonal fluctuations during the menopausal transition period (21). The evidence strongly suggests that PMS/PMDD result from an abnormal or exaggerated effect of cyclic changes in ovarian steroid hormones on central neurotransmitter mechanisms and serotonin, in particular, plays an important role in their pathophysiology (1). Different authors (46,47) suggest that premenstrual complaints are caused by the drop in progesterone (P) concentrations in the late luteal phase and these changes are linked to changes in CNS neurotransmitters such as γ-aminobutyric acid (GABA). Other theories or hypotheses are that the symptoms are triggered by the preovulatory peak of Estradiol (E2), by the postovulatory increase in P, or both (48,49), however, these theories don't explain why the symptoms begin with the ovulation in some women and but later in the luteal phase in others, in others words, different patterns (20).

Since mood and behavioral symptoms are key features of PMS, underlying mechanisms must involve the brain. Sex steroids easily pass the blood-brain barrier, and sex steroid receptors are abundant in brain regions that regulate emotions and behavior, including the amygdala and the hypothalamus (20). One of the theories mentions or implicates as a cause of PMS/PMDD is the dysregulation or alteration of neurotransmitters like endorphins, serotonin, and GABA (5,20,32). Changes in hormone levels may influence centrally acting neurotransmitters such as serotonin (50). Yonkers et al. (20) have mentioned that serotonin terminals could exert a dampening influence on brain areas, such as the amygdala, that are under a parallel, the independent activating influence of sex steroids The importance of serotonin for the regulation of mood and aggression, and the probable role of serotonin in modulating sex-steroid-driven behavior, suggests that serotonin could be involved in the pathophysiology of PMS. There is inconsistent evidence of circulating levels of endorphins in PMS patients. Women with PMS/PMDD may have an alteration in the GABA receptor complex response (5,51); studies have demonstrated that plasma GABA levels and the sensitivity of GABA receptors are reduced during the luteal phase (5). However, the most plausible theory is the serotonergic dysregulation with reduced serotonergic function (15,44,52). There is some evidence that PMS/PMDD is related to increased sensitivity to P in women with serotonin deficiency (50,53). Even more, the role of GABA in the pathophysiology of PMS/PMDD is based on the assumption that the PMS/PMDD symptoms or complaints are due to P withdrawal, the assumption that has been questioned (20). Also, there are important interactions between

GABAergic and serotonergic neurons (54,51), this interaction could imply that there is not a conflict between GABA and serotonin in the pathophysiology of PMS/PMDD (20).

The levels of sex steroids, estrogen, progesterone, and testosterone are normal, women with PMS may be more vulnerable to normal fluctuations. Some studies strongly suggested an abnormal response to normal hormonal changes (47,48). Maybe, this vulnerability could be in part related to serotonin (6). There is no proof of a relationship between PMS and prolactin (PRL), growth hormone, melatonin, thyroid hormone, adrenal activity, luteinizing hormone (LH), follicle-stimulating hormone (FSH), antidiuretic hormone, insulin, aldosterone, renin-angiotensin, and cortisol. Studies of well-defined patient populations also have failed to demonstrate any differences in the levels of testosterone (T), FSH, LH, PRL, sex hormone-binding globulin, melatonin, cortisol, and aldosterone between women with and without symptoms of PMS, in any phase of the menstrual cycle (1), but their secretion might be aberrant in women with PMS/PMDD (56). Additional to P theory, it has been mentioned other sex steroid metabolite, allopregnanolone (AlloP) which is a metabolite of P and its levels vary similarly to progesterone with a rise in the luteal phase of the menstrual cycle (57,58). In patients with PMS, AlloP levels are low both in the follicular and luteal phases (59,60). This appears to be due to impaired synthesis of allopregnanolone by the corpus luteum and other steroidogenic organs. AlloP is a positive modulator of the GABA receptor and a potent neurotransmitter with effects on mood, behavior, and cognitive function. AlloP has a bimodal action on mood symptoms similar to benzodiazepines, barbiturates, and alcohol. In high doses, it produces anxiolytic, anti-aggressive, sedative, and antiepileptic effects, and some studies (61) have demonstrated that a higher level of AlloP in women with PMS/PMDD might have a GABA receptor dysfunction. In low doses, it causes severe emotional reactions in a group of individuals (2 %-3 %) and a moderate reaction in up to 20 % of women (62). Rapkins et al. (59) suggested that low levels of AlloP in women with PMS may lead to an enhanced GABA-mediated inhibition during states of altered central nervous system lowered metabolite levels could contribute to the genesis of various mood symptoms of the disorder, such as anxiety, tension, depression and excitability.

The thyroid function is normal in women with PMS (63); approximately 10 % of women with PMS have abnormal thyroid function, but the prevalence is not significantly different from that of subclinical hypothyroidism in the general population (1). Overall, the thyroid-stimulating hormone (TSH) response to thyroid-releasing hormone (TRH) is normal. Although abnormal responses (both exaggerated and blunted) are observed more often in women with PMS, they occur during both phase follicular and luteal (49).

Also, vitamin and mineral deficiencies including zinc, vitamin A, vitamin E, vitamin D, thiamine, magnesium, and pyridoxine (vitamin B6) have been implicated but the data show inconsistent scientific evidence (1).

Studies in twins have found evidence to suggest that genetic factors could predispose to PMS/PMDD (64,65). The correlation between menstrual symptoms in mothers and daughters and between sisters suggests a genetic influence but also might reflect a learned or conditioned response (66). Whereas many have speculated that differences in personality, stress levels, or coping mechanisms may play a role in PMS, there is little or no evidence to support the hypothesis (67). Other possible factors are high body mass index (68) and traumatic events (69). Also, deficiency of prostaglandins related to an inability to convert linoleic acid to prostaglandin precursors has been mentioned as a cause of PMS/PMDD (70) (Table 1).

Clinical symptoms

PMS/PMDD is found among menstruating women of all ages as has been mentioned. Symptoms of PMS vary from mild to severe enough to interfere with daily personal and occupational life. There are no specific physical findings or laboratory tests that can be utilized to make the diagnosis of PMS (5). PMDD is also referred to as severe PMS, which is characterized predominantly by emotional symptoms according to the Diagnostic and Statistical Manual of Mental Disorders (IV and V)

Table 1

PMS/PMDD Theories

Low progesterone levels
Low Allopregnanolone levels
High estrogen levels
Falling estrogen levels
Changes in the estrogen/progesterone ratio
Increased aldosterone activity
Increased renin-angiotensin system activity
Increased adrenal activity
Endogenous opiate withdrawal
Subclinical hypoglycemia
Central changes in catecholamines
Responsiveness to prostaglandins
Vitamin and mineral deficiencies
Excess prolactin secretion

Speroff L, Fritz MA. Clinical gynecologic endocrinology and infertility. Menstruation-related Disorders, Chapter 14. 9th edition. Philadelphia, Lippincott Williams & Wilkins; 2020.p.1150-1213.

The most common symptoms are categorized as physical, psychological, and behavioral or emotional

a.-Physical symptoms: include tiredness, edematous sensation, abdominal cramps, pain and bloating, fatigue, nausea, breast fullness, tenderness/mastalgia, dizziness, headache, weight gain, back pain, body aches, edema of the extremities, water retention, hot flushes, joint/muscle pain (9,11, 15,20,21,26,42).

b.-Psychological symptoms: also called emotional symptoms., include nervousness, anger, irritability, depressed mood, mood swings, confusion, forgetfulness, restlessness, loneliness, decreased self-esteem, sadness, depression, decreased or lack of concentration, anxiety, tension, feeling confused (9,11, 15,20,21,26,42).

c.- Behavioral symptoms: appetite swings or changes such as a craving for sweet or salty or overeating, hyperinsomnia/insomnia, dysphoria, affect lability, lack of energy, fatigue, dizziness, feeling overwhelmed or out of control, decreased interest in usual or routine activities, changes in sexual interest, and social withdrawal (9,11, 15,20,21,26,42).

PMD/PMDD symptoms present with typical features, although a wide range of both somatic and psychological symptoms has been reported (19). The key characteristic is their timing, which must occur during all or part of the 2-week premenstrual phase; the symptoms are most severe during the last few days of the luteal phase and resolve during menstruation (11,19). The persistence of symptoms during menstruation does not preclude the diagnosis; however, there must be a clear, symptom-free interval between the end of menstruation and the approximate time of ovulation; thus, for many women, there may be only 7-10 days each month without premenstrual symptoms. This cyclical chain of events must occur in most menstrual cycles (typically two out of every three. Suppression of ovulation will result in a major reduction or elimination of symptoms. It should be noted that suppression of ovulation may cause estrogen deficiency with symptoms or side effects similar to those of PMS, and could potentially confound diagnosis; however, they will be non-cyclical in nature. Also, PMS/PMDD must not be a premenstrual exacerbation of another psychiatric, physical, or medical disorder, but the severity or impact of symptoms could have a substantial negative impact on activities of daily living and quality of life and affect normal daily functioning, interfere with work, school performance or interpersonal relationships or cause significant distress (11,19). Once the PMS/PMDD symptoms begin, unless treated, the cyclic symptoms recur monthly and persist with most cycles until the waning of ovulation with the menopausal transition Symptoms recur within one to two cycles after discontinuation of treatment and spontaneous remission is unusual (11).

PMS symptoms usually start to be problematic in the adolescent years (32) and decline in the climacteric. Symptoms are most severe in the 20s to mid-30s but women are most likely to seek treatment after the age of 30 years (9). Usually, pregnancy offers a break from PMS but there is an increased risk of postpartum depression (71). Usually. PMS/PMDD patients describe having the symptoms for several years but it is not clear whether PMS/PMDD premenstrual dysphoria worsens with age or whether older women are simply more likely to ask for help for this problem. PMS/PMD resolves after menopause

as we mentioned before, although there is some evidence that perimenopausal symptoms may be more severe in women who have had severe PMS/PMDD (15).

In 1980, a multidisciplinary US National Institutes of Health consensus conference on PMS proposed criteria that were adopted by the Diagnostic and Statistical Manual III (DSM III) (16) to define the severe form of this condition. Originally entitled "late luteal phase dysphoric disorder"; in 1994 (17), it was renamed "premenstrual dysphoric disorder" (PMDD). The diagnosis of PMDD stipulates or establishes: 1.- the presence of at least five luteal-phase symptoms, at least one of which must be a mood symptom (depressed mood, anxiety or tension, affect lability, or persistent anger and irritability); 2.- two cycles of daily charting to confirm the timing of symptoms; and 3.- evidence of functional impairment. Finally, symptoms must not be the exacerbation of another psychiatric condition (17) A problem with the PMDD diagnosis is that many women with clinically significant premenstrual symptoms do not meet full diagnostic criteria; they might not have a prominent mood symptom, or the five different symptoms required as a minimum by DSM IV. The American College of Obstetrics and Gynecology (ACOG) has attempted to rectify this situation by defining moderate to severe PMS; the criteria are the presence of at least one psychological or physical symptom that causes significant impairment and is confirmed by means of prospective ratings (72). Despite differences between diagnostic systems, women with clinically significant PMS described in scientific reports usually correspond to those with a diagnosis of PMDD (20).

Pattern of symptom expression

As was mentioned before, the length of symptomatology varies between a few days and 2 weeks; symptoms often worsen substantially 6 days before, and peak about 2 days before the menstrual period begins (33,34). Among the psychological symptoms, anger and irritability are the most severe complaints and start slightly earlier than other symptoms (33). It is not rare that symptoms can persist into the next menstrual cycle but, by definition, there must be a symptom-free interval before ovulation (10,33,34). Usually and typically, women who have a set of symptoms in one cycle will have the same set of symptoms in the next one (41). Also, several patterns of true symptoms and apparent comorbidity can occur at the same time in a woman with PMS. The possible patterns: 1.- she might have another psychiatric disorder at another point in her life (lifetime comorbidity); 2.- she might have an ongoing psychiatric or general medical condition and concurrent premenstrual symptoms that are not part of the co-occurring disorder (concurrent comorbidity); 3.- she might have an ongoing psychiatric or general medical condition that becomes worse premenstrually, usually defined as premenstrual exacerbation (4,17). Estimates for lifetime comorbidity between PMS and other mood disorders range from 30 % to 70 % (4,73). According to Yonkers et al. (20), this prevalence is higher than one would expect, even taking into consideration that mood disorders are common in women (at least 30 % of women have a minor or major depressive disorder at some point in their lives) and comorbidity might be inflated because of an overlap in symptoms. Anxiety disorders also occur at a higher rate in women with PMS/PMDD (74,75). Patients with PMS/PDMM have an increased tendency to have a panic attack when they are exposed to substances as such lactate, and carbon dioxide, suggesting that panic disorder and PMS/PDMM share some pathophysiological mechanisms (74,75).

Diagnosis

Many groups have published diagnostic criteria for premenstrual diseases, including the World Health Organization, American College of Obstetricians and Gynecologists (ACOG), Royal College of Obstetricians and Gynecologists (RCOG), International Society for Premenstrual Disorders (ISPMD), and the American Psychiatric Association (APA; DSM-5) (76).

Clinical history is key to the diagnosis of PMS/PMDD. To establish the MPS/PMDD diagnosis is important to keep in mind that the symptoms, as has been mentioned, must be: 1.- characteristic of PMS/PDMM; 2.- limited to the luteal phase;

3.- producing problems to the woman, and 4.- not explained better that some other pathology or disorder (72).

The PMS/PMDD symptoms are physical, phycological, and behavioral symptoms as has been mentioned before. The ACOG in a Practice Bulletin was published in 2000 (69), based on the work of Mortola (13). PMS can be diagnosed according to the ACOG diagnosis criteria (72): 1.- the patient mentions having at least one of the affective and one physical o somatic symptoms and reports having the symptoms five days before the beginning of menses in the previous three menstrual cycles; 2.- The symptomatology must be prospectively recorded in at least 2 cycles; 3.- the symptoms must cease within 4 days of the onset of the menstrual period and not appear until after day 12 of the menstrual cycle; 4. The patient must not be taking any medication, hormone therapy, recreational drugs, or alcohol that could cause any dysfunction in social or work activities. Table 2 shows the psychological and physical symptoms according to ACOG Practice Bulletin.

Table 2

Symptoms ACOG Practice Bulletin 2000

Psychological/Affective	**Physical/Somatic**
Depression	Breast tenderness
Angry outbursts	Abdominal bloating
Irritability	Headache
Anxiety	Edema of extremities
Confusion	
Social withdrawal	

ACOG Committee on Practice Bulletins--Gynecology. ACOG Practice Bulletin: No 15. Premenstrual syndrome. Obstet Gynecol. 2000;95(Suppl 4):1-9. Retraction in: Obstet Gynecol. 2012;120(2 Pt 1):405.

According to the original article by Mortola (13,14), specific dysfunction in daily activities includes marital/relationship discord, parenting problems, social isolation, legal problems, suicidal ideation, school or work-related problems such as poor performance, poor attendance, tardiness, and seeking medical care for somatic complaints.

The diagnosis of PMS and PMDD depends on the presence of typical symptoms, their timing, severity, and the exclusion of other diagnoses. Both diagnoses require a prospective symptom diary documenting specific cyclic symptoms associated with the luteal and menstrual phases of the cycle and are severe enough to adversely impact the social functioning and quality of life of the affected women (77). The specific group of symptoms in a given individual is much less important than the cyclic nature of the symptom complex and its temporal relationship with menses. When symptoms are charted accurately, as much as 40 % of women presenting with presumed PMS do not exhibit the distinctly cyclic pattern required for diagnosis and have an underlying mood or anxiety disorder (78).

There are different criteria protocols to make the PMS/PMDD diagnosis; there are several criteria used for diagnosing PMS/PMMD such as the guidelines from the National Institute of Mental Health (NIMH), the University of California at San Diego (UCSD), and the APA, as they appear in the current Diagnostic and Statistical Manual of Mental Disorders (DSM-V) (1,79,80).

According to the NIMH criteria, the diagnosis of PMS also should require at least a 30 % increase in the severity of symptoms over the 5 days before menses, compared with the 5 days after onset of menses. These changes have to be registered in a daily symptom diary for at least two consecutive cycles (26).

According to UCSD criteria, at least one of the following affective and somatic symptoms during the five days before menses in each of the three previous cycles:

1.- Affective symptoms: depression, angry outbursts, irritability, anxiety, confusion, social withdrawal.

2.- Somatic symptoms: breast tenderness, abdominal bloating, headache, swelling of extremities.

Symptoms relieved from days 4 through 13 of the menstrual cycle

Based on the UCSD and NIMH criteria, it is estimated that approximately 5 % of women of reproductive age can be diagnosed with disruptive PMS (5,70,81,82).

The most commonly used criteria for the diagnosis of PMDD are those proposed by the APA, "Diagnostic and Statistical Manual of Mental Disorders-5" (DSM-V):

Criteria A: In the majority of menstrual cycles in the past year, at least five symptoms must be present in the final week before the onset of menses, and start to improve within a few days after the onset of menses, and become minimal or absent in the week post-menstrual (9,17,21):

1.- Markedly depressed mood, feelings of hopelessness, or self-deprecating thoughts.

2.- Marked anxiety, tension, feelings of being "keyed up" or "on edge".

3.- Marked affective lability (e.g., feeling suddenly sad or tearful or increased sensitivity to rejection).

4. Persistent and marked anger or irritability or increased interpersonal conflicts.

5.- Decreased interest in usual activities (e.g., work, school, friends, hobbies).

6.-Subjective sense of difficulty in concentrating.

7.- Lethargy, easy fatigability, or marked lack of energy.

8.- Marked change in appetite, overeating, or specific food cravings.

9.- Hypersomnia or insomnia.

10.-A subjective sense of being overwhelmed or out of control

11.-Other physical symptoms, such as breast tenderness or swelling, headaches, joint or muscle pain, a sensation of "bloating," weight gain.

Criteria B: one (or more) of the following symptoms must be present:

1. Marked affective lability (e.g., 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.

Criteria 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 (e.g., 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.

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

Criteria 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).

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

Criteria G: the symptoms are not attributable to the physiological effects of a substance

(e.g., a drug of abuse, a medication, other treatment) or another medical condition (e.g., hyperthyroidism)

It is important to mention that the symptoms in criteria A–C must have been met for most menstrual cycles that occurred in the preceding year. According to the APA criteria, the prevalence of PMS in women of reproductive age is 5 % (Definition of PMS).

Also, it is important to mention that the diagnosis of PMS requires both affective and

somatic symptoms whereas the diagnosis of PMDD can include but does not require somatic symptoms. Another distinction between the two disorders is that PMDD may be superimposed on another psychiatric disorder, whereas the diagnosis of PMS can only be made in their absence (1).

The diagnosis of PMS also should require at least a 30 % increase in the severity of symptoms over the 5 days before menses, compared with the 5 days after onset of menses, according to the guidelines from the NIMH (1,79) and based on USCD and NIMH criteria, it is estimated that approximately 5 % of women of reproductive age can be diagnosed with complete remission of PMS (1,73).

The diagnoses of PMS and PMDD must be differentiated from other underlying psychiatric disorders, which are common among women with similar symptoms (1,83). Also, medical conditions, such as hyperthyroidism and hypothyroidism, should be excluded.

The American College of Obstetricians and Gynecologists (ACOG) has established its own PMS diagnosis criteria (11,18,72):

A.- Criteria for PMS diagnosis

At least one or more bothersome affective and somatic symptoms, plus additional criteria

Emotional symptoms

i. depression
ii. angry outbursts
ii. irritability
iv. crying spells
v. anxiety, tension
vi. confusion
vii. social withdrawal
viii. poor concentration
ix. insomnia
x. increased nap-taking
xi. changes in sexual desire

Physical symptoms

i. thirst and appetite changes (food cravings)
ii. breast tenderness
iii. bloating and weight gain
iv. headache
v. swelling of the hands or feet
vi. aches and pains
vii. fatigue
viii. skin problems
ix. gastrointestinal symptoms
x. abdominal pain

To diagnose PMS, the woman's symptoms must

1) present in the 5 days before a period for at least three menstrual cycles in a row
2) end within 4 days after a period starts
3) interfere with some normal activities

The patient has to keep a record of symptoms. Each day for at least 2 to 3 months, the symptoms have to be written and rated.

B.- Criteria for PDMM diagnosis

At least five symptoms, including one of the first four core symptoms below with moderate-to-severe intensity, plus additional criteria

i. Depressed mood
ii. Anxiety, tension
iii. Labile mood
iv. Irritability, anger
v. Decreased interest in usual activities or social withdrawal
Vi. Difficulty concentrating/confusion
vii. Fatigue, tiredness
viii. Appetite changes (overeating/cravings)
ix. Hypersomnia/insomnia

x. Feeling out of control/overwhelmed

xi. Physical symptoms: breast tenderness, bloating, swelling of extremities, headache, joint/muscle pain

Necessary for both PMS and PMDD diagnosis

a. Criteria must be confirmed by prospective daily ratings during two consecutive menstrual cycles

b. Symptoms emerge in the second half of the menstrual cycle and subside within 4 days after the onset of menstruation

c. Alternatively, symptoms must occur within the 5 days before the onset of menses and there must be a symptom-free interval after menses until the time of ovulation

d. Interference with work/school and social activities/relationships (subjective impairment for PMDD and identifiable dysfunction for PMS)

e. Symptoms present in absence of pharmacologic or hormonal therapy, drug or alcohol intake

f. It may be superimposed on other psychiatric or medical disorders, provided it is not merely an exacerbation of that disorder

According to these criteria, the PMS prevalence is 30 % in women of reproductive age (80).

The World Health Organization (WHO) (12,84) classified the PMS as an International Statistical Classification of Diseases and Related Health Problems 10 (ICD-10) and the organization established:

a) 1/9 mood symptoms or one physical symptom required

b) Can include the exacerbation of another psychiatric disorder?

c) Does not require significant functional dysfunction

d) 80 % Prevalence in women of reproductive age

As it was mentioned before in 2019, the WHO change the classification of PMDD from ICD-10 to ICD-11 to differentiate it from the more common PMS by the severity of the symptoms and the requirement that they cause significant distress or impairment (12,85). The symptoms are severe enough to cause significant distress or significant impairment in personal, family, social, educational, occupational, or other important areas of functioning, and do not represent the exacerbation of another mental disorder (12).

The Daily Record of Severity of Problems (DRSP) allows for an accurate diagnosis of PMS/PMDD. This chart is used to document the frequency and intensity of emotional and physical symptoms associated with the menstrual cycle. Patients are asked to prospectively describe their symptoms without any treatment for at least 2 months consecutively (42,86) and the clinician can make the diagnosis and take decisions based on the timing of symptoms, which commonly start any time before the beginning of menstruation, as well as the cyclic variability of symptoms and disappearance of symptoms within 1 or 2 days after menstruation onset The symptom diary continues to be recorded after starting treatment to allow for detailed observation of treatment-related alterations in symptoms (42).

Differential Diagnosis

As it has been mentioned before, numerous symptoms have been attributed to PMS and include a broad spectrum that includes physical, behavioral, and emotional symptoms which are commonly reported by women seeking PMS treatment A woman who seeks medical attention usually has multiple symptoms but is likely to report the mood and behavioral symptoms as the most distressing. Severe PMS clearly involves emotional symptoms such as irritability, mood swings, tension, and depression. Irritability may be the cardinal symptom of the syndrome (9,87). Although many women mention that premenstrual symptoms increase, the majority of women do not report these symptoms as distressing. Moreover, women typically report the greatest premenstrual increases for symptoms of physical discomfort rather than mood symptoms that characterize severe PMS (9,87).

As it is known PMS is characterized by a cyclic pattern of symptoms that occur premenstrually

and abate with menses, PMS can be confused with other disorders that manifest premenstrual magnification of symptoms such as (5,9,18,42):

A.- Psychiatric disorders

1) Major depression
2) Intermittent depressive disorder
3) Seasonal affective disorder
4) Dysthymia
5) Anxiety
6) Panic disorder
8) Somatoform disorder
9) Personality disorder
10) Drug abuse
11) Alcoholism

B.- Medical disorders

1) Endometriosis
2) Thyroid disorders
3) Seizure disorders
4) Dysmenorrhea
5) Auto-inmune disorders
6) Allergies
7) Diabetes mellitus
8) Anemia
9) Chronic fatigue syndrome
10) Irritable bowel syndrome
11) Perimenopause

Medical disorders such as migraines, asthma, epilepsy, irritable bowel syndrome, diabetes, allergies, and autoimmune disorder may worsen during the premenstrual period; it is assumed that fluctuations in gonadal hormones in accordance with the menstrual cycle can affect the symptoms of these medical disorders (88).

PMS diagnosis is based on the clinical reports of symptoms which are provided by a dairy recorded but certain laboratory tests are recommended to practice and can provide additional information for differential diagnoses such as a complete blood cell count and chemistry panel and thyroid function tests (42).

CONCLUSION

The global prevalence of PMS is high and about half of women of reproductive age experience these symptoms (6). PMS/PDMM represents one of the female medical conditions that remain a medical pathology of theories. As it has been mentioned before PMS/PDMM has physical, psychological, and behavioral symptoms that vary from mild to severe enough to interfere with daily personal and occupational life, in other words, the lifestyle of women of reproductive age. There are no specific physical findings or laboratory tests that can be utilized to make the diagnosis of PMS. The Daily Record of Severity of Problems (DRSP) allows for an accurate diagnosis of PMS/PMDD. The diagnosis of PMS and PMDD depends on the presence of typical symptoms, their timing, severity, and the exclusion of other diagnoses.

Conflict of Interest: the author declares that there is none

Funding Sources: none exist

REFERENCES

1. Speroff L, Fritz MA. Clinical gynecologic endocrinology and infertility. Menstruation-related Disorders. Chapter 14. 9th edition. Philadelphia, Lippincott Williams & Wilkins; 2020:1150-1213.
2. Frankl RT. The hormonal causes of premenstrual tension Arch Neurol Psychiat. 1931;26:1053-1057.
3. Grenee R, Dalton K. The premenstrual syndrome. Br Med J. 1953;1(4818):1007-1014.
4. Steiner M, Born L. Diagnosis and treatment of premenstrual dysphoric disorder: An update. Int Clin Psychopharmacol. 2000;15(Suppl 3):S5-S17.
5. Braverman P. Premenstrual syndrome and premenstrual dysphoric disorder. J Pediatr Adolesc Gynecol. 2007;20:3e12.
6. Direkvand-Moghadam A, Sayehmiri K, Delpisheh A, Kaikhavandi S. Epidemiology of Premenstrual

Syndrome (PMS)-A Systematic Review and Meta-Analysis Study. J Clin Diagn Res. 2014;8(2):106-109. Erratum in: J Clin Diagn Res. 2015;9(7):ZZ05.

7. Gao M, Gao D, Sun H, Cheng X, An L, Qiao M. Trends in Research Related to Premenstrual Syndrome and Premenstrual Dysphoric Disorder From 1945 to 2018: A Bibliometric Analysis. Front Public Health. 2021;9:596128.
8. Ussher JM, Perz J. PMS as a process of negotiation: women's experience and management of premenstrual distress. Psychol Health. 2013;28(8):909-927.
9. Freeman EW. Premenstrual syndrome and premenstrual dysphoric disorder: Definitions and diagnosis. Psychoneuroendocrinology. 2003;28(Suppl 3):S25-S37.
10. Sternfeld B, Swindle R, Chawla A, Long S, Kennedy S. Severity of premenstrual symptoms in a health maintenance organization population. Obstet Gynecol. 2002;99(6):1014-1024.
11. Rapkin AJ, Winer SA. Premenstrual syndrome and premenstrual dysphoric disorder: Quality of life and burden of illness. Expert Rev Pharmacoecon Outcomes Res. 2009;9(2):157-170.
12. Reed GM, First MB, Kogan CS, Hyman SE, Gureje O, Gaebel W, et al. Innovations and changes in the ICD-11 classification of mental, behavioural and neurodevelopmental disorders. World Psychiatry. 2019;18(1):3-19.
13. Mortola JF, Girton L, Yen SS. Depressive episodes in premenstrual syndrome. Am J Obstet Gynecol. 1989;161(6 Pt 1):1682-1687.
14. Mortola JF, Girton L, Beck L, Yen SS. Diagnosis of premenstrual syndrome by a simple, prospective, and reliable instrument: The calendar of premenstrual experiences. Obstet Gynecol. 1990;76(2):302-307.
15. Johnson SR. Premenstrual syndrome, premenstrual dysphoric disorder, and beyond: A clinical primer for practitioners. Obstet Gynecol. 2004;104(4):845-859.
16. American Psychiatric Association. Diagnostic and statistical manual of mental disorders-DSM-III (3rd ed). Washington DC: American Psychiatric Association, 1980.
17. American Psychiatric Association. Diagnostic and statistical manual of mental disorders-DSM-IV (4th edition). Washington DC: American Psychiatric Association; 1994.
18. Premenstrual Syndrome (PMS). ACOG. (Visited and reviewed on April 14, 2022). Available at: https://www.acog.org/womens-health/faqs/premenstrual-syndrome.
19. 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(1):13-21.
20. Yonkers KA, O'Brien PM, Eriksson E. Premenstrual syndrome. Lancet. 2008;371(9619):1200-1210.
21. Biggs WS, Demuth RH. Premenstrual syndrome and premenstrual dysphoric disorder. Am Fam Physician. 2011;84(8):918-924.
22. Baker LJ, O'Brien PM. Premenstrual syndrome (PMS): A peri-menopausal perspective. Maturitas. 2012;72(2):121-125.
23. Angst J, Sellaro R, Merikangas KR, Endicott J. The epidemiology of perimenstrual psychological symptoms. Acta Psychiatr Scand. 2001;104(2):110-116.
24. 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 Womens Health Gend Based Med. 1999;8(8):1043-1052.
25. Bowen R, Bowen A, Baetz M, Wagner J, Pierson R. Mood instability in women with premenstrual syndrome. J Obstet Gynaecol Can. 2011;33(9):927-934.
26. Dickerson LM, Mazyck PJ, Hunter MH. Premenstrual syndrome. Am Fam Physician. 2003;67(8):1743-1752.
27. Deuster PA, Adera T, South-Paul J. Biological, social, and behavioral factors associated with premenstrual syndrome. Arch Fam Med. 1999;8(2):122-128.
28. Eshetu N, Abebe H, Fikadu E, Getaye E, Jemal S, Geze S, et al. Premenstrual syndrome, coping mechanisms and associated factors among Wolkite university female regular students, Ethiopia, 2021. BMC Women's Health. 2022;22:88.
29. Victor FF, Souza AI, Barreiros CDT, Barros JLN, Silva FACD, Ferreira ALCG. Quality of Life among University Students with Premenstrual Syndrome. Rev Bras Ginecol Obstet. 2019;41(5):312-317.
30. Rezende APR, Alvarenga FR, Ramos M, Franken DL, Dias da Costa JS, Pattussi MP, et al. Prevalence of Premenstrual Syndrome and Associated Factors Among Academics of a University in Midwest Brazil. Rev Bras Ginecol Obstet. 2022;44(2):133-141.
31. Costanian C, Akiki Z, Rabah Z, Daou S, Assaad S. Factors Associated with Premenstrual Syndrome and its Different Symptom Domains among University Students in Lebanon. Int J Womens Health Wellness. 2018;4:068.
32. Rapkin AJ, Mikacich JA. Premenstrual syndrome in adolescents: diagnosis and treatment. Pediatr Endocrinol Rev. 2006;3(Suppl 1):S132-S137.
33. Pearlstein T, Yonkers K, Fayyad R, Gillespie J. Pretreatment pattern of symptom expression in

premenstrual dysphoric disorder. J Affect Disord 2005;85:275-282.

34. Meaden PM, Hartlage SA, Cook-Karr J. Timing and severity of symptoms associated with the menstrual cycle in a community-based sample in the Midwestern United States. Psychiatry Res. 2005;134(1):27-36.
35. Bloch M, Schmidt PJ, Rubinow DR. Premenstrual syndrome: evidence for symptom stability across cycles. Am J Psychiatry. 1997;154(12):1741-1746.
36. Bell CC. DSM-IV: Diagnostic and Statistical Manual of Mental Disorders. JAMA. 1994;272(10):828-829.
37. Pearlstein TB, Frank E, Rivera-Tovar A, Thoft JS, Jacobs E, Mieczkowski TA. Prevalence of axis I and axis II disorders in women with late luteal phase dysphoric disorder. J Affect Disord. 1990;20(2):129-134.
38. Mackenzie TB, Wilcox K, Baron H. Lifetime prevalence of psychiatric disorders in women with perimenstrual difficulties. J Affect Disord. 1986;10(1):15-19.
39. Richards M, Rubinow DR, Daly RC, Schmidt PJ. Premenstrual symptoms and perimenopausal depression. Am J Psychiatry. 2006;163(1):133-137.
40. Studd J. Estrogens and Depression in Women. In: Crosignani PG, Paoletti R, Sarrel PM, Wenger NK, Meschia M, Soma M, editors. Women's Health in Menopause. Medical Science Symposia Series, 1994, vol 7. Springer, Dordrecht.
41. Bloch M, Schmidt PJ, Danaceau M, Murphy J, Nieman L, Rubinow DR. Effects of gonadal steroids in women with a history of postpartum depression. Am J Psychiatry. 2000;157(6):924-930.
42. Ryu A, Kim TH. Premenstrual syndrome: A mini-review. Maturitas. 2015;82(4):436-440.
43. Schmidt PJ, Nieman LK, Danaceau MA, Adams LF, Rubinow DR. Differential behavioral effects of gonadal steroids in women with and in those without premenstrual syndrome. N Engl J Med. 1998;338(4):209-216.
44. Freeman EW, Sondheimer SJ, Rickels K. Gonadotropin-releasing hormone agonist in the treatment of premenstrual symptoms with and without ongoing dysphoria: A controlled study. Psychopharmacol Bull. 1997;33(2):303-309.
45. Freeman EW, Sammel MD, Rinaudo PJ, Sheng L. Premenstrual syndrome as a predictor of menopausal symptoms. Obstet Gynecol. 2004;103(5 Pt 1):960-966.
46. Smith SS, Ruderman Y, Frye C, Homanics G, Yuan M. Steroid withdrawal in the mouse results in anxiogenic effects of 3alpha, 5beta-THP: A possible model of premenstrual dysphoric disorder. Psychopharmacology (Berl). 2006;186(3):323-333.
47. Sundström I, Smith S, Gulinello M. GABA receptors, progesterone and premenstrual dysphoric disorder. Arch Womens Ment Health. 2003;6(1):23-41
48. Schmidt PJ, Nieman LK, Grover GN, Muller KL, Merriam GR, Rubinow DR. Lack of effect of induced menses on symptoms in women with premenstrual syndrome. N Engl J Med. 1991;324(17):1174-1179.
49. Schmidt PJ, Grover GN, Roy-Byrne PP, Rubinow DR. Thyroid function in women with premenstrual syndrome. J Clin Endocrinol Metab. 1993;76(3):671-674.
50. Wyatt K, Dimmock PW, O'Brien PM. Premenstrual syndrome. In: Barton S, editor. Clinical evidence. 4th issue. London: BMJ Publishing Group, 2000.p.1121-1133.
51. Steiner M, Pearlstein T. Premenstrual dysphoria and the serotonin system: pathophysiology and treatment. J Clin Psychiatry. 2000;61(Suppl 12):S17-S21.
52. Halbreich U. The diagnosis of premenstrual syndromes and premenstrual dysphoric disorder--clinical procedures and research perspectives. Gynecol Endocrinol. 2004;19(6):320-334.
53. Kessel B. Premenstrual syndrome. Advances in diagnosis and treatment. Obstet Gynecol Clin North Am. 2000;27(3):625-639.
54. Bhagwagar Z, Wylezinska M, Taylor M, Jezzard P, Matthews PM, Cowen PJ. Increased brain GABA concentrations following acute administration of a selective serotonin reuptake inhibitor. Am J Psychiatry. 2004;161(2):368-370.
55. Sundström I, Bäckström T. Citalopram increases pregnanolone sensitivity in patients with premenstrual syndrome: An open trial. Psychoneuroendocrinology. 1998;23(1):73-88.
56. Parry BL, Newton RP. Chronobiological basis of female-specific mood disorders. Neuropsychopharmacology. 2001;25(5 Suppl):S102-S108.
57. Indusekhar R, Usman SB, O'Brien S. Psychological aspects of premenstrual syndrome. Best Pract Res Clin Obstet Gynaecol. 2007;21(2):207-220.
58. Genazzani AR, Petraglia F, Bernardi F, Casarosa E, Salvestroni C, Tonetti A, et al. Circulating levels of allopregnanolone in humans: Gender, age, and endocrine influences. J Clin Endocrinol Metab. 1998;83(6):2099-2103.
59. Rapkin AJ, Morgan M, Goldman L, Brann DW, Simone D, Mahesh VB. Progesterone metabolite allopregnanolone in women with premenstrual syndrome. Obstet Gynecol. 1997;90(5):709-714.
60. Bicíková M, Dibbelt L, Hill M, Hampl R, Stárka L. Allopregnanolone in women with premenstrual syndrome. Horm Metab Res. 1998;30(4):227-230.

61. Girdler SS, Straneva PA, Light KC, Pedersen CA, Morrow AL. Allopregnanolone levels and reactivity to mental stress in premenstrual dysphoric disorder. Biol Psychiatry. 2001;49(9):788-797.

62. Miczek KA, Fish EW, De Bold JF. Neurosteroids, GABA-A receptors, and escalated aggressive behavior. Horm Behav. 2003;44(3):242-257.

63. Korzekwa MI, Lamont JA, Steiner M. Late luteal phase dysphoric disorder and the thyroid axis revisited. J Clin Endocrinol Metab. 1996;81(6):2280-2284.

64. Kendler KS, Karkowski LM, Corey LA, Neale MC. Longitudinal population-based twin study of retrospectively reported premenstrual symptoms and lifetime major depression. Am J Psychiatry. 1998;155(9):1234-1240.

65. Treloar SA, Heath AC, Martin NG. Genetic and environmental influences on premenstrual symptoms in an Australian twin sample. Psychol Med. 2002;32(1):25-38.

66. Wilson CA, Turner CW, Keye WR Jr. Firstborn adolescent daughters and mothers with and without premenstrual syndrome: A comparison. J Adolesc Health. 1991;12(2):130-137.

67. Fontana AM, Palfai TG. Psychosocial factors in premenstrual dysphoria: stressors, appraisal, and coping processes. J Psychosom Res. 1994;38(6):557-567.

68. Masho SW, Adera T, South-Paul J. Obesity as a risk factor for premenstrual syndrome. J Psychosom Obstet Gynaecol. 2005;26(1):33-39.

69. Perkonigg A, Yonkers KA, Pfister H, Lieb R, Wittchen HU. Risk factors for premenstrual dysphoric disorder in a community sample of young women: the role of traumatic events and posttraumatic stress disorder. J Clin Psychiatry. 2004;65(10):1314-1322.

70. Daugherty JE. Treatment strategies for premenstrual syndrome. Am Fam Physician. 1998;58(1):183-92, 197-8.

71. Halbreich U. The etiology, biology, and evolving pathology of premenstrual syndromes. Psychoneuroendocrinology. 2003;28(Suppl 3):S55-S99.

72. ACOG Committee on Practice Bulletins--Gynecology. ACOG Practice Bulletin: No 15: Premenstrual syndrome. Obstet Gynecol. 2000;95(Suppl 4):S1-S9. Retraction in: Obstet Gynecol. 2012;120(2 Pt 1):405.

73. Wittchen H -U, Becker E, Lieb R, Krause P. Prevalence, incidence and stability of premenstrual dysphoric disorder in the community. Psychol Med. 2002;32(1):119-132.

74. Landén M, Eriksson E. How does premenstrual dysphoric disorder relate to depression and anxiety disorders? Depress Anxiety. 2003;17(3):122-129.

75. Yonkers KA. Anxiety symptoms and anxiety disorders: How are they related to premenstrual disorders? J Clin Psychiatry. 1997;58(Suppl 3):S62-S67; discussion 68-69.

76. Dilbaz B, Aksan A. Premenstrual syndrome, a common but underrated entity: Review of the clinical literature. J Turk Ger Gynecol Assoc. 2021;22(2):139-148.

77. Mortola JF. Issues in the diagnosis and research of premenstrual syndrome. Clin Obstet Gynecol. 1992;35(3):587-598.

78. Bailey JW, Cohen LS. Prevalence of mood and anxiety disorders in women who seek treatment for premenstrual syndrome. J Womens Health Gend Based Med. 1999;8(9):1181-1184.

79. Jequier AM. Premenstrual Syndrome: Current Findings and Future Directions Edited by Howard J. Osofsky and Susan J. Blumenthal Cambridge: Cambridge University Press. 1986. Pp 95. British J Psychiatry. 1987;151(3):426-427.

80. Vahia VN. Diagnostic and statistical manual of mental disorders 5: A quick glance. Indian J Psychiatry. 2013;55(3):220-223.

81. Ramcharan S, Love EJ, Fick GH, Goldfien A. The epidemiology of premenstrual symptoms in a population-based sample of 2 650 urban women: Attributable risk and risk factors. J Clin Epidemiol. 1992;45(4):377-392.

82. Merikangas KR, Foeldenyi M, Angst J. The Zurich Study. XIX. Patterns of menstrual disturbances in the community: results of the Zurich Cohort Study. Eur Arch Psychiatry Clin Neurosci. 1993;243(1):23-32.

83. Osofsky HJ, Blumenthal SJ. Premenstrual syndrome: Current findings and future directions. Washington, DC, American Psychiatric Press; 1985.

84. Definition of MPS/PDMM. National Association for Premenstrual Syndromes. (Internet) (Visited and reviewed on April 14, 2022). Available at: https://www.pms.org.uk/about-pms/definitions-pms-pmdd/.

85. Figueira ML, Dias VV. Postpartum depression and premenstrual dysphoric disorder: Options for ICD-11. World Psychiatry. 2012;11(Suppl 1):S73-S76.

86. Endicott J, Nee J, Harrison W. Daily Record of Severity of Problems (DRSP): reliability and validity. Arch Womens Ment Health. 2006;9(1):41-49.

87. Freeman EW. Luteal phase administration of agents for the treatment of premenstrual dysphoric disorder. CNS Drugs. 2004;18(7):453-468.

88. Halbreich U, O'Brien PM, Eriksson E, Bäckström T, Yonkers KA, Freeman EW. Are there differential symptom profiles that improve in response to different pharmacological treatments of premenstrual syndrome/premenstrual dysphoric disorder? CNS Drugs. 2006;20(7):523-547.