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The Intersection of Premenstrual Syndrome (PMS) and Mood Disorders: A Narrative Review

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Abstract

Premenstrual Syndrome (PMS) encompasses a range of physical, emotional, and behavioral symptoms occurring cyclically during the luteal phase of the menstrual cycle. Mood disorders such as depression and anxiety often overlap with PMS, suggesting shared pathophysiological mechanisms. This narrative review explores the hormonal, neurochemical, psychosocial, and cultural factors contributing to the comorbidity of PMS and mood disorders. Understanding these interactions can guide effective diagnostic approaches and individualized treatment strategies.

Keywords: Premenstrual Syndrome, Mood Disorders, Depression, Anxiety, Hormonal Fluctuations, Neurotransmitters, Cultural Perspectives

1. Introduction

Premenstrual Syndrome (PMS) affects up to 75% of menstruating individuals, with 3–8% experiencing severe forms such as Premenstrual Dysphoric Disorder (PMDD) (Yonkers et al., 2008). Migraines, particularly menstrual migraines, affect approximately 20% of women of reproductive age, with attacks frequently occurring in the perimenstrual period (Vetvik & MacGregor, 2017). The temporal overlap of PMS symptoms and migraines suggests a shared pathophysiological basis. Both conditions are influenced by hormonal fluctuations, neurotransmitter dysregulation, and neurovascular changes, indicating potential common therapeutic targets (MacGregor, 2004).

Emerging research highlights that beyond hormonal shifts, genetic predispositions and environmental factors, such as stress and lifestyle, may modulate the severity and frequency of both PMS and migraines (Buse et al., 2013). This overlap complicates diagnosis and treatment, often leading to under-recognition of their comorbidity in clinical practice. Additionally, neuroinflammatory processes, particularly involving cytokine dysregulation, have been implicated in both conditions, suggesting that inflammation may act as a mediator between hormonal changes and symptom manifestation (Calhoun & Hutchinson, 2009). Cognitive and emotional disturbances, such as mood swings, irritability, and depressive symptoms, frequently co-occur with both disorders, further blurring the clinical picture and complicating individualized management approaches.

Understanding the biopsychosocial interplay underlying PMS and migraines is crucial, as untreated comorbidity can lead to significant impairment in daily functioning, reduced quality of life, and increased healthcare utilization. Moreover, the economic burden associated with these conditions is substantial, both in terms of direct medical costs and indirect costs due to lost productivity. Recognizing and addressing this overlap not only improves patient outcomes but also enhances the effectiveness of targeted therapies. This narrative review aims to explore the shared biological pathways contributing to the comorbidity of PMS and migraines, emphasizing the role of estrogen withdrawal, serotonergic dysfunction, and neurovascular changes, while highlighting potential integrated treatment strategies.

2. Hormonal Fluctuations and Mood Dysregulation

Hormonal fluctuations, particularly involving estrogen and progesterone, are central to the pathophysiology of both PMS and migraines. The cyclical nature of these hormonal shifts throughout the menstrual cycle significantly influences the onset and severity of symptoms in susceptible individuals. Estrogen and progesterone not only regulate reproductive functions but also modulate neurochemical pathways, vascular reactivity, and inflammatory responses, all of which are implicated in PMS and migraine pathogenesis.

2.1 Estrogen Withdrawal Hypothesis

Estrogen plays a pivotal role in modulating both mood and migraine susceptibility. The estrogen withdrawal hypothesis suggests that the abrupt decline in estrogen levels during the late luteal phase triggers both PMS symptoms and migraine attacks (Halbreich & Kahn, 2001). Estrogen withdrawal leads to increased cortical excitability, alterations in serotonin metabolism, and heightened sensitivity to pain stimuli (MacGregor, 2004).

This drop in estrogen affects multiple neurotransmitter systems, including serotonergic, dopaminergic, and GABAergic pathways, which are crucial for mood regulation and nociceptive processing. The destabilization of these systems may explain the co-occurrence of mood disturbances and heightened pain sensitivity during the

premenstrual phase (Facchinetti et al., 2003). Additionally, estrogen has vasodilatory properties, mediated through nitric oxide synthesis, and its withdrawal can induce vascular instability, a known trigger for migraines (Vetvik & MacGregor, 2017). Studies have shown that stabilizing estrogen levels with hormonal therapies can reduce both PMS-related mood symptoms and menstrual migraine frequency, supporting the centrality of this mechanism (Calhoun & Hutchinson, 2009).

2.2 Progesterone and Neurosteroids

Progesterone and its metabolites, such as allopregnanolone, influence GABAergic neurotransmission, which plays a role in mood stabilization and pain modulation. Fluctuations in progesterone may contribute to both mood instability in PMS and increased migraine susceptibility through altered GABA receptor sensitivity (Epperson et al., 2002).

Allopregnanolone acts as a potent positive allosteric modulator of GABA-A receptors, enhancing inhibitory neurotransmission and exerting anxiolytic effects. However, abrupt changes in its levels during the menstrual cycle may lead to mood dysregulation, irritability, and increased stress sensitivity (Rapkin & Lewis, 2013). Furthermore, progesterone withdrawal has been linked to heightened neuroinflammatory responses, which can exacerbate migraine pathophysiology through sensitization of trigeminal nociceptive pathways (Stovner et al., 2007). The interplay between progesterone fluctuations, GABAergic modulation, and inflammatory cascades highlights the complex neuroendocrine mechanisms underlying the comorbidity of PMS and migraines.

3. Neurotransmitter Imbalance in PMS and Mood Disorders

Neurotransmitter imbalances play a pivotal role in the pathophysiology of both PMS and migraines. Fluctuations in key neurotransmitters, such as serotonin, dopamine, and gamma-aminobutyric acid (GABA), contribute to the mood disturbances, cognitive changes, and heightened pain sensitivity observed in individuals affected by these conditions. Hormonal shifts, particularly the withdrawal of estrogen and progesterone during the luteal phase, have been shown to influence neurotransmitter synthesis, release, and receptor sensitivity, leading to the complex symptomatology characteristic of PMS and migraines.

3.1 Serotonergic Dysfunction

Serotonin (5-HT) is a key neurotransmitter involved in both mood regulation and migraine pathophysiology. Reduced serotonergic activity during the luteal phase has been linked to depressive symptoms in PMS and migraine attacks (Pearlstein & Steiner, 2008). The decline in estrogen levels during the late luteal phase reduces the availability of tryptophan, the precursor for serotonin synthesis, thereby contributing to serotonin deficiency (Halbreich, 2003). This deficiency is associated with irritability, mood swings, and depressive symptoms characteristic of PMS, as well as the vasodilation of cerebral blood vessels, a hallmark of migraine pathogenesis.

Furthermore, serotonergic dysregulation affects the descending pain inhibitory pathways, diminishing the brain's ability to modulate nociceptive input effectively. This dysfunction is particularly relevant in migraines, where serotonin receptor agonists, such as triptans, have proven effective in aborting acute attacks by promoting vasoconstriction and inhibiting neurogenic inflammation (Silberstein, 2004). Interestingly, serotonergic antidepressants, including selective serotonin reuptake inhibitors (SSRIs), have demonstrated efficacy in both treating PMS-related mood symptoms and reducing migraine frequency. For example, Freeman et al. (1999) showed that full- or half-cycle treatment with serotonergic antidepressants significantly alleviated severe PMS symptoms, reinforcing the shared serotonergic mechanisms underlying both conditions.

3.2 Dopamine and GABA Systems

Dopaminergic hypersensitivity and GABAergic dysfunction are implicated in both PMS and migraine. Dopamine plays a complex role in the regulation of mood, reward processing, and pain perception. In migraines, dopaminergic hypersensitivity is associated with prodromal symptoms such as yawning, nausea, and mood changes, while its dysregulation contributes to migraine aura and the initiation of migraine attacks (Peroutka, 1997).

In PMS, fluctuations in estrogen and progesterone influence dopamine receptor sensitivity, potentially contributing to mood lability and irritability. The interaction between dopamine and the hypothalamic-pituitary-adrenal (HPA) axis may also exacerbate stress responses, further aggravating PMS symptoms.

GABA, the primary inhibitory neurotransmitter in the central nervous system, plays a critical role in mood stabilization and the modulation of cortical excitability. Progesterone-derived neurosteroids, such as allopregnanolone, enhance GABAergic activity, producing anxiolytic and mood-stabilizing effects. However, during the luteal phase, the decline in progesterone and its metabolites leads to reduced GABAergic inhibition, which may contribute to both the mood disturbances observed in PMS and the cortical hyperexcitability seen in migraines (Epperson et al., 2002). The combined dysregulation of dopaminergic and GABAergic systems highlights the intricate neurochemical interplay that underpins the comorbidity of PMS and migraines.

4. Psychological, Environmental, and Cultural Factors

Neurovascular and inflammatory mechanisms are central to the pathogenesis of both PMS and migraines. These mechanisms involve complex interactions between hormonal fluctuations, vascular reactivity, and immune responses, contributing to the diverse symptoms experienced during the premenstrual phase and migraine attacks (Amin & Ashina, 2012).

4.1 Neurovascular Dysregulation

Migraine is traditionally considered a neurovascular disorder characterized by altered cerebral blood flow and vascular reactivity. Similar vascular changes may occur in PMS, where hormonal fluctuations affect endothelial function and cerebral perfusion (Silberstein & Merriam, 1991). Estrogen, known for its vasodilatory effects, modulates nitric oxide synthesis, and its withdrawal leads to increased vascular tone and reactivity, potentially triggering migraine attacks (Edvinsson & Uddman, 2005).

Moreover, fluctuations in progesterone can influence cerebrovascular tone by altering autonomic nervous system activity, contributing to changes in blood pressure and vascular stability (MacGregor, 2004). Studies suggest that women with menstrual migraines exhibit greater cerebral vascular sensitivity to hormonal changes compared to those without migraines (Edvinsson & Uddman, 2005). Additionally, migraine-related cortical spreading depression (CSD), a wave of neuronal and glial depolarization, may be facilitated by hormonal shifts that destabilize neurovascular homeostasis, linking PMS-related headaches to migraine pathophysiology (Amin & Ashina, 2012).

4.2 Inflammatory Mediators

Pro-inflammatory cytokines and prostaglandins are elevated during the luteal phase, contributing to both PMS symptoms (e.g., bloating, breast tenderness) and migraine pathogenesis. These mediators can sensitize nociceptive pathways, enhancing pain perception (Rapkin & Akopians, 2012).

Recent studies indicate that increased levels of interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) are associated with the severity of both PMS and menstrual migraines, suggesting a shared inflammatory basis (Khalil et al., 2020). Additionally, prostaglandin E2, a key inflammatory mediator, is implicated in the development of menstrual cramps and migraine-associated pain, further highlighting the overlap in inflammatory

pathways (Maizels & Burchette, 2004). The interplay between hormonal fluctuations and immune system activation underscores the need for anti-inflammatory strategies in managing both PMS and migraine symptoms.

5. Comorbidity Between PMS and Mood Disorders

Epidemiological studies show that individuals with PMS are more likely to experience migraines, and vice versa. This comorbidity is associated with greater symptom severity, increased disability, and poorer quality of life (Pavlović et al., 2015).

Women with both conditions often report more frequent and severe headaches, heightened mood disturbances, and greater sensitivity to environmental triggers compared to those with either condition alone (Breslau et al., 2003). This comorbidity suggests a potential genetic predisposition, as both PMS and migraines have been linked to polymorphisms in genes regulating serotonin and estrogen metabolism. Furthermore, the bidirectional relationship between mood disorders, PMS, and migraines implies that neurochemical imbalances, particularly in the serotonergic and dopaminergic systems, contribute to the chronicity and intensity of symptoms (Fuh et al., 2007).

Recognizing this overlap is essential for accurate diagnosis and integrated treatment strategies. A multidisciplinary approach that includes hormonal, pharmacological, and behavioral therapies may offer the most effective symptom management for individuals affected by both conditions.

6. Diagnostic Challenges and Considerations

Diagnosing PMS and migraines requires careful symptom tracking to establish the cyclical pattern and hormonal correlation. Due to overlapping symptoms, distinguishing between primary mood disorders, chronic headaches, and hormonally driven conditions can be challenging.

6.1 Symptom Diaries

Symptom diaries, such as the Daily Record of Severity of Problems (DRSP) and headache diaries, help identify temporal associations between hormonal changes and symptom fluctuations (Yonkers et al., 2008). These tools enable clinicians to differentiate between menstrual-related migraines and other headache types, such as tension-type headaches or cluster headaches, which may have different triggers and treatment approaches.

Additionally, electronic health apps have emerged as effective tools for tracking symptoms in real-time, offering more accurate data for clinical evaluation (Johannes et al., 2000). Incorporating patient-reported outcomes into diagnostic assessments improves the identification of comorbid conditions, such as anxiety and depression, which frequently coexist with PMS and migraines (Bigal et al., 2008).

6.2 Differential Diagnosis

Differential diagnosis is critical to avoid misclassification and ensure appropriate treatment. Conditions such as thyroid disorders, polycystic ovary syndrome (PCOS), and endometriosis can mimic PMS symptoms, while secondary headache disorders may present similarly to migraines (Kim et al., 2004).

Hormonal assays, neuroimaging, and psychiatric evaluations may be necessary in complex cases to rule out other underlying conditions. Early and accurate diagnosis not only improves patient outcomes but also reduces the risk of chronicity and associated disability.

7. Therapeutic Approaches

The treatment of PMS and migraines often overlaps due to shared pathophysiological mechanisms, particularly hormonal fluctuations and neurotransmitter dysregulation. A combination of pharmacological and non-pharmacological strategies is recommended to address both conditions effectively.

7.1 Pharmacological Treatments

Selective serotonin reuptake inhibitors (SSRIs) are effective for PMS-related mood symptoms and may reduce migraine frequency through serotonergic modulation (Freeman et al., 1999). SSRIs help stabilize mood by enhancing serotonergic neurotransmission, which is often disrupted during the luteal phase. For migraines, particularly menstrual-related migraines, triptans are the first-line treatment for acute attacks due to their ability to promote vasoconstriction and inhibit neurogenic inflammation (Elrington, 2002).

Hormonal therapies, such as combined oral contraceptives, can stabilize hormonal fluctuations, thereby reducing both PMS symptoms and menstrual migraines (Calhoun & Hutchinson, 2009). Continuous or extended-cycle contraceptives have shown particular efficacy in reducing menstrual-related migraine frequency by minimizing estrogen withdrawal (MacGregor, 2004). Additionally, nonsteroidal anti-inflammatory drugs (NSAIDs) are frequently used to alleviate both PMS-related discomfort and migraine pain by reducing prostaglandin-mediated inflammation (Sances et al., 2004).

7.2 Non-Pharmacological Interventions

Cognitive-behavioral therapy (CBT) is effective for managing PMS-related mood disturbances and migraine-associated stress (Holroyd et al., 2010). CBT helps patients develop coping strategies to manage the emotional and psychological impacts of chronic conditions. Lifestyle modifications, including regular exercise, stress reduction techniques, and dietary adjustments, have been shown to mitigate both PMS and migraine symptoms (Rains et al., 2005).

Emerging evidence suggests that mindfulness-based interventions can reduce migraine frequency and PMS severity by improving stress regulation and emotional resilience (Wells et al., 2014). Furthermore, nutritional supplements such as magnesium, vitamin B6, and omega-3 fatty acids have demonstrated potential benefits in reducing symptoms related to both conditions (Facchinetti et al., 1991).

8. Future Directions and Research Gaps

Despite advances in understanding PMS and migraine pathophysiology, several research gaps remain. Standardized diagnostic criteria are needed to improve the identification and management of PMS-migraine comorbidity (Charles, 2018). Current diagnostic tools often overlook the cyclical nature of symptoms, leading to underdiagnosis and mismanagement.

Further research is required to elucidate the precise hormonal pathways linking PMS and migraines. Studies focusing on the role of estrogen and progesterone receptors in the central nervous system could provide insights into new therapeutic targets (Nappi et al., 2013). Additionally, exploring the genetic and epigenetic factors that predispose individuals to both conditions may improve personalized treatment approaches (Genazzani et al., 2010).

Personalized medicine, which tailors treatment based on hormonal profiles and neurochemical biomarkers, holds promise for optimizing outcomes in individuals with PMS and migraines. Integrating genetic screening and hormonal monitoring into clinical practice could lead to more effective, individualized treatment strategies (MacGregor, 2014).

9. Conclusion

PMS and migraines are interconnected conditions sharing common pathophysiological mechanisms involving hormonal fluctuations, neurotransmitter dysregulation, and neurovascular changes. Understanding these shared pathways can inform comprehensive diagnostic and therapeutic strategies, improving outcomes for individuals affected by both conditions.

The overlap between PMS and migraines highlights the need for an integrated approach to diagnosis and management. Recognizing the comorbidity can lead to more effective treatment plans that address the full spectrum of symptoms, rather than treating each condition in isolation. Future research should focus on the development of standardized diagnostic criteria, exploration of hormonal mechanisms, and the application of personalized medicine to improve patient care.

Disclosure

Authors do not report any disclosures.

Authors' contributions

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Lead Author: Conceptualized the study, designed the review structure, and coordinated the writing process. Contributed extensively to the sections on hormonal fluctuations and mood dysregulation, and finalized the manuscript.

Weronika Koziak (WK)

Data Collection & Analysis: Conducted comprehensive literature research and contributed to the sections on neurotransmitter imbalance. Assisted in reviewing and integrating relevant studies into the narrative review.

Aleksandra Bętkowska (AB)

Statistical Analysis & Review: Supported data analysis and contributed to the psychological, environmental, and cultural factors section. Reviewed the manuscript for methodological accuracy.

Stanisław Dudek (SD)

Scientific Advisor: Provided critical revisions on neurovascular mechanisms and inflammatory pathways. Ensured the scientific rigor and coherence of the review.

Agata Kornacka (AK)

Literature Review & Drafting: Focused on the comorbidity between PMS and mood disorders. Contributed to drafting diagnostic challenges and therapeutic approaches.

Kamila Szostak (KSz)

Clinical Insights: Brought clinical expertise, particularly in PMS-related mood disorders. Provided real-world perspectives on diagnostic criteria and patient case reviews.

Rafał Tomaka (RT)

Technical Editing: Assisted in manuscript editing, formatting, and reference management. Contributed to the sections on differential diagnosis and future research directions.

Wojciech Dudek (WD)

Methodology Expert: Focused on research methodology and critically evaluated the manuscript's structure. Contributed to the section on future directions and research gaps.

Anna Byra (ABy)

Proofreading & Final Review: Proofread the final manuscript, ensuring linguistic accuracy and coherence. Provided feedback on the therapeutic approaches section.

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