CLINICAL CHARACTERISTICS OF NEUROENDOCRINE DYSFUNCTIONS IN PREMENSTRUAL SYNDROME

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Annotation

This thesis evaluates the clinical manifestations of neuroendocrine dysfunctions in women suffering from Premenstrual Syndrome (PMS) and its severe variant, Premenstrual Dysphoric Disorder (PMDD). By analyzing the complex interactions between ovarian steroids and central neurotransmitter systems—specifically serotonin and gamma-aminobutyric acid (GABA)—the study elucidates why normal cyclical hormonal changes trigger profound physical and psycho-emotional symptoms in susceptible individuals. The findings underscore the necessity of recognizing PMS as a central neuroendocrine disorder, which fundamentally shifts the therapeutic approach toward neuro-pharmacological and targeted hormonal interventions.

Key words: premenstrual syndrome (PMS), premenstrual dysphoric disorder (PMDD), neuroendocrine dysfunction, allopregnanolone, serotonin, luteal phase, psychoneuroendocrinology.

Introduction

Premenstrual Syndrome (PMS) encompasses a wide array of somatic, emotional, and behavioral symptoms that predictably occur during the luteal phase of the menstrual cycle and resolve shortly after the onset of menstruation. While historically attributed to simple imbalances between estrogen and progesterone, contemporary psychoneuroendocrinology identifies PMS and PMDD as disorders of altered central nervous system sensitivity to normal gonadal steroid fluctuations. The abnormal neuroendocrine response primarily involves the dysregulation of the serotonergic system and altered central sensitivity to allopregnanolone, a neuroactive metabolite of progesterone. Understanding the specific clinical features of these neuroendocrine disruptions is crucial for accurate diagnosis, minimizing patient distress, and formulating effective, evidence-based treatment plans.

Material and methods

An observational clinical study was conducted involving 80 women of reproductive age (20–40 years) presenting with moderate to severe cyclic premenstrual complaints. Diagnosis was established using the Daily Record of Severity of Problems (DRSP) over two consecutive menstrual cycles to confirm luteal phase symptom restriction. The cohort underwent comprehensive psychiatric and endocrine evaluations. Fasting venous blood samples were drawn during the mid-follicular and late-luteal phases to measure serum concentrations of Estradiol, Progesterone, and peripheral markers of serotonergic activity. Patients with underlying thyroid disorders, major depressive disorder, or generalized anxiety disorder were excluded to prevent symptomatic overlap.

Result and discussion

The clinical evaluation demonstrated a distinct biphasic pattern of symptoms directly correlating with luteal phase neuroendocrine shifts. Psycho-emotional symptoms were predominant in 65% of the subjects, characterized by severe mood lability, irritability, heightened anxiety, and depressive episodes. These psychological markers strongly correlate with decreased central serotonergic transmission, exacerbated by the physiological drop in estrogen prior to menstruation.

Somatic symptoms, primarily pronounced mastalgia, abdominal bloating, and severe fatigue, were prominent in 35% of the cohort. Interestingly, hormonal assays revealed no statistically significant differences in absolute serum estradiol and progesterone levels between PMS patients and healthy population norms. Instead, the pathology stems from an aberrant central nervous system response to allopregnanolone. In healthy women, allopregnanolone acts as an anxiolytic via GABA-A receptors; however, in women with severe PMS, this metabolite paradoxically triggers anxiety and irritability due to altered neuro-receptor plasticity. The discussion highlights that because PMS is driven by central receptor sensitivity rather than peripheral hormone deficiency, treatments must target the central nervous system (e.g., Selective Serotonin Reuptake Inhibitors) or suppress the ovulatory trigger altogether (e.g., continuous oral contraceptives).

Conclusion and recommendation

Premenstrual Syndrome is a definitive neuroendocrine disorder characterized by an abnormal central nervous system response to regular cyclical hormonal variations. The clinical features are heavily dominated by serotonergic and GABAergic dysfunctions, leading to debilitating psycho-emotional and somatic distress. It is

recommended that clinicians abandon the outdated paradigm of treating PMS purely as a peripheral hormonal imbalance. Instead, management protocols should prioritize the use of prospective symptom charting for accurate diagnosis, followed by the first-line application of serotonergic antidepressants (SSRIs) or ovulation-suppressing hormonal therapies to stabilize the neuroendocrine axis and significantly improve the patient's quality of life.

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