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Postpartum Psychiatric Episodes can be the Markers of Familial Bipolar Disorder

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DESCRIPTION

Postpartum psychosis is one of the few psychiatric disorders for which a clear etiology can be defined. An important predictor is elementary. Thus, if a woman's first delivery was not exacerbated by postpartum psychosis, her chances of subsequent deliveries are greatly reduced. Furthermore, researchers have not consistently identified obstetric risk factors for the development of postpartum psychosis. Conducting neurobiological studies in patients with postpartum psychosis is challenging due to the low incidence and acute severity of disability. The developmental mechanisms are likely related to specific physiological changes at birth in genetically vulnerable females.

During pregnancy, estrogen and progesterone levels among other hormones increase exponentially. Around 35 weeks of gestation, Corticotropin-Releasing Hormone (CRH)-binding protein decreases, leading to an increase in her CRH and ACTH before delivery. After giving birth, estrogen and progesterone levels drop sharply, and these hormones return to normal within three weeks. This multiple concentration change in reproductive hormone levels associated with labour is involved in the pathogenesis of postpartum psychosis. Since estrogen mediates dopaminergic tone in the hypothalamus, postnatal development of mood disorders has been reported to result in increased sensitivity of dopamine receptors. However, others have not replicated this finding. If a sharp drop in estrogen levels is the trigger for postnatal psychosis, administration soon after birth can prevent psychosis.

Suggestive small case series and randomized studies demonstrating no effect of transdermal estradiol regimens on recurrence in women at postpartum risk are included for mood disorders. Although genetic studies of women with postpartum psychosis in the first year of life are lacking, susceptibility to postpartum relapse in women with bipolar disorder has been studied. Postpartum psychiatric episodes may be markers of familial bipolar disorder. As an important step toward identifying the causative etiology, Jones and Craddock identified specific genetic variants in the serotonin transporter gene and genome-wide mutations on the chromosomes of patients with a history of bipolar disorder identified the accumulation of important chain signals in postpartum psychosis. Studies of estrogen receptor or glucocorticoid receptor polymorphisms associated with postpartum psychosis have not shown an association. The Perinatal Psychiatric Genetics Consortium was established to encourage research potential. This is a large collaborative research effort to elucidate the pathophysiology of postpartum psychiatric disorders, including postpartum psychosis. Animal studies suggest that postpartum glucocorticoid secretion is regulated in part by the dopaminergic system. The release of oxytocin during breastfeeding is associated with feelings of well-being and relaxation, as well as a reduced response to stressors.

The treatment of lactating women with acute episodes of psychosis is a difficult clinical situation that must be individually tailored. The well-known benefits of breastfeeding must be weighed against the mother's mental health and are a priority in this context. Lack of sleep due to frequent waking for breastfeeding may contribute to the onset and persistence of mania and restoring regular circadian patterns is essential for recovery. Overall postpartum recurrence rates are similar in both groups, but there are significant differences in approaches to prevention.

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CONFLICT OF INTEREST

The author's declared that they have no conflict of interest.

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