

Different diagnosis of postpartum psychosis

An interesting article that is important in the differential diagnosis of postpartum psychosis was published in the June, 2011, issue of American Journal of Psychiatry. (Am J Psychiatry 168:576-580). Fassier et al, from The Hospices Civils de Lyon in France, described a woman who had been treated for two previous episodes of postpartum psychosis. She developed an episode after the birth of her third child and was hospitalized. The woman had an inherited metabolic disorder--a late-onset urea cycle disorder. Late-onset implies that the enzymatic deficiency in the urea cycle, the metabolic process by which the body gets rid of nitrogen, is partial and therefore may not be diagnosed until adulthood. There are six enzymes that take part in the metabolic process. A deficiency of any one results in excess body nitrogen, which accumulate as ammonia. The patient described in the case report was initially diagnosed through high plasma levels of ammonia.

The authors recommended obtaining ammonia levels on women who present with postpartum psychosis, severe mood disorder or delirium, to rule out this metabolic disorder. Identification is important to decrease the risk of permanent neurological sequelae or death. The various urea cycle disorders range in prevalence from 1/14000 to 1/350,000 persons, and all can be identified with ammonia levels. Women are vulnerable in the postpartum period because of the catabolic demands of pregnancy and childbirth, which trigger high ammonia levels due to an overburdened capacity to clear urea from the body. This patient had metabolic studies to confirm that she had carbamyl phosphate synthetase 1 deficiency, a defect with a 1/62,000 frequency of occurrence.

The clinical presentation was difficult to differentiate from postpartum psychosis, which is characterized by confusion and many symptoms that are usually considered indicative of an organic etiology. However, clues to the metabolic abnormality in this woman included fever, headaches and a "habitual reluctance to consume meat." The latter is a clue because the metabolism of protein is the defect in urea cycle disorders, and it is intriguing to consider that the patient was aware of some physiological consequences of high protein intake. The authors noted that the patient was "more confused and less delusional" than might be expected for a postpartum psychosis, but the level of confusion and psychosis are difficult to operationalize, and confusion is a hallmark of postpartum psychosis. She had urinary incontinence, which is unusual in women with postpartum psychosis. The patient did not respond as robustly as she had previously to antipsychotic medication in the postpartum period, which may also characterize increasing morbidity from multiple psychotic episodes. Finally, it is interesting is that valproate, which might be used for treatment of a manic psychotic episode, increases the urea cycle overload.

The patient had been a journalist but had neuropsychological deficits after the third episode with diminished overall function. She had remarked that her previous episodes of psychosis had caused her to be less able to care for her children, which might indicate her awareness of residual mild deficits from the two previous episodes. Treatment included a low-protein diet, sodium benzoate and sodium phenylbutyrate and citrulline, an amino acid intermediate in the urea cycle.

