girlfriend had visited that weekend and that they had engaged in sexual activity on several occasions. He was placed on complete rest once again and advised not to have any sexual activity. His concussive symptoms resolved by the following week. He successfully completed the graduated rehabilitative protocol without any recurrence of symptoms and was cleared for return to play. His recovery was subsequently complete and uneventful.

Case Report

A 25-year-old hockey player with a negative concussion history sustained a concussion in a game when he was elbowed in the back of the head by an opponent and then hit the boards with his head. There was no loss of consciousness or amnesia. Immediate symptoms included headache, "seeing stars," photophobia, phonophobia, and ataxia. He was evaluated by a physician and was cleared to return to play in the same game. Upon returning to the ice, his symptoms recurred quickly and he was unable to complete the shift; he was then removed from the game. Two days later, his score on the PCS was 12 (his preseason PCS baseline score was 1). Symptoms included headache, fatigue, photophobia, phonophobia, feeling slowed down, and sleeping more than usual. He was instructed to avoid physical and cognitive exertional activities for a week. Brain MRI was normal and neurological examination was negative. He was evaluated again a week later, with a PCS score of 1 (sleeping more than usual). He reported however, that four days after the injury he and his wife had initiated a sexual interaction but that during the activity he "saw stars" and was unable to continue the sexual encounter. His performance on neuropsychological testing 12 days after the concussion was consistent with his preseason baseline testing, and the athletic trainer began him on a graduated rehabilitation program. He tolerated stationary cycling without difficulty but experienced a recurrence of symptoms when he began skating. He was again withheld from all exertional activity for a week and was then once again asymptomatic (PCS score of 0). He abstained from sexual activity, completed the graduated rehabilitative program, and returned to play without any recurrence of symptoms.

Comment

Sexual behavior can be an (overlooked) exertional activity, and may need to be considered by sports medicine professionals in their operational definition of exertional physical activity. Athletes may need to be cautioned about the potentially deleterious effects of sexual activity during the rehabilitative phase of a sports-related concussion.

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References

1. Aubry M, Cantu R, Dvorak J, et al: Summary and Agreement Statement of the 1st International Symposium on Concussion in Sport, Vienna, 2001. Clin J Sport Med 2002; 12:6–11
2. McCrory P, Johnston K, Meeuwisse W, et al: Summary and Agreement Statement of the 2nd International Conference on Concussion in Sport, Prague, 2004. Br J Sports Med 2005; 39:196–204

Possible Delayed Speech Acquisition With Clozapine Therapy During Pregnancy and Lactation

SIR: Most of the study by Ernst and Goldberg[1] reported perinatal outcome with clozapine therapy with no focus on neurodevelopment. Here, I report a case of a woman with schizophrenia who continued clozapine treatment throughout her 9 months of pregnancy and during lactation.[2] This report also highlights neurodevelopmental aspects, particularly speech of the baby.

Case Report

"Mrs. B," a 30-year-old woman, had been suffering with schizophrenia for the past 10 years. Her illness was continuous with partial remission and exacerbation and did not much improve on various typical antipsychotic drugs. Therefore, a regimen of clozapine, 25 mg per day, was started and was increased to 100 mg per day over a period of 2 weeks.

After remaining stabilized on clozapine for 6 months, she became pregnant. It was not a planned pregnancy, but the couple decided to continue despite the possible risks to the fetus with antipsychotic drugs. Her routine laboratory investigations, including blood glucose, hemoglobin, and white blood cell count were within normal limits.

Mrs. B attended an antenatal clinic every 4 to 6 weeks beginning in the third month of pregnancy until her delivery. She gained weight normally throughout pregnancy, was concerned, but had positive attitude toward pregnancy. Her nutritional care was good. She continued on the same dose of clozapine and did not exhibit an exacerbation or a behavior that could harm her fetus throughout her pregnancy.

At 9 months and 2 days of gestation, she delivered a baby girl weighing 2.95 kg without any perinatal complication. Until the baby was 1 year old, she was breast-fed

by her mother, who continued on the same dose of clozapine. The baby had normal developmental milestones, except for speech. She began to use consonants m, k, p, and b at the age of 1 year and started using combined syllables, like ba-ba and da-da, at the age of one-and-a-half years. At the age of 2 years she spoke only six to eight words. She would speak only 12 to 15 words until the age of 3 years and she also exhibited stuttering. Even intervention by a speech therapist did not render better results. At the age of 4, she acquired speaking skills in small sentences by joining two or three words, and she would repeat small sentences that she heard. By the end of 5 years, she gained normal fluent speech. Relevant investigations including audiometric analysis were within normal limits. ENT examination ruled out local pathology. There was no history of impaired mother-child relationship, familial phonological disorder, or bilingual environment.

Comment

Most probably, this baby fulfills the criteria of phonological disorder or may have exhibited delayed onset of speech. At least 3% of preschool children present with this disorder of unknown origin, which is often referred as developmental or functional.[3] In my case, the patient was treated only with clozapine throughout her pregnancy. However, it is difficult to draw conclusions that speech difficulty in this child is a possible consequence of pre- and postnatal exposure to clozapine or a consequence of maternal mental illness because schizophrenia in the mother implies an increased risk for poor perinatal outcome.[4] Further studies should address the magnitude of neurodevelopmental difficulties with clozapine.

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References

1. Ernst CL, Goldberg JF: The reproductive safety profile of mood stabilizers, atypical antipsychotics, and broad-spectrum psychotropics. J Clin Psychiatry 2002; 63(suppl 4):42–55
2. Gentile S: Clinical utilization of atypical antipsychotics in pregnancy and lactation. Ann Pharmacother 2004; 38:1265–1271
3. American Psychiatric Association: Disorders usually first diagnosed in infancy, childhood and adolescence, in DSM-IV-TR. Washington, DC, APA, 2002, pp 39–134
4. Nilsson E, Lichtenstein P, Cnattingius S, et al: Women with schizophrenia: pregnancy outcome and infant death among their offspring. Schizophr Res 2002; 58:221–229

Is There a Role for Valproic Acid in the Treatment of Catatonia?

SIR: Catatonia is a complex neuropsychiatric disorder, with various medical and psychiatric etiologies, that presents with autism, rigidity, motor immobility (motor signs of catatonia), as well as uncontrollable anxieties, emotional lability, and compulsive emotions (affective signs of catatonia).[1] Catatonia has been treated successfully with GABAergic agents, such as lorazepam and ECT. Research shows that several treatments with ECT in rats increase gamma-aminobutyric acid (GABA) levels in the CNS.[2] We present a case where valproic acid was used successfully in the treatment of catatonia.

Case Report

"Ms. G" was a 46-year-old woman with a history of alcohol dependence who possibly suffered from a mood disorder for years based on her biopsychosocial history; however, she was never treated for her mood disorder. She presented with motor as well as affective signs of catatonia on multiple admissions to various hospitals within a span of 4 months. She was treated with benzodiazepines with improvement in catatonic motor signs. Ms. G was placed on various antipsychotics for her catatonic affective symptoms, which failed to improve, with her motor signs invariably returning. She was transferred to our institution for further evaluation and after several weeks with no improvement was placed on a regimen of valproic acid, 1000 mg, and within 3 to 4 days her catatonic symptoms dissipated. The patient was discharged on this regimen with no subsequent catatonic episodes.

Comment

Case reports using valproic acid in the treatment of catatonia successfully have documented neuroimaging studies showing a GABA-A receptor density reduction in the brains of catatonic patients.[2] These data further support a GABAergic deficit in catatonic patients. The relationship between catatonia and mood disorders is well established, where approximately a quarter to half the patients with catatonia also meet criteria for a mood disorder.[3] Plasma GABA levels have also been noted to be low in approximately 40% of patients with mood disorders, which further supports the hypothesis of a GABAergic deficit in catatonia and bipolar disorder.[4] Extrapolating from the above, we suggest that GABA modulation may be a significant factor involved in illness ranging from mood disorders to catatonia. The number of patients with catatonic schizophrenia has declined, yet the number of