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Poor Neonatal Adaptation After in Utero Exposure to Duloxetine

TO THE EDITOR: Risk-benefit analysis is central when forming a treatment plan for pregnant women with mental illness. This task is further complicated by limited data pertaining to the effects of antidepressants on the fetus, neonate, and child. To our knowledge, this is the first case report of in utero exposure to duloxetine.

"Ms. A" was a 36-year-old Caucasian female with a history of recurrent major depression, anorexia, and chronic neck pain. When she sought psychiatric consultation at 34 weeks' gestation, she was 1) in complete remission, 2) being treated by an anesthesiologist, 3) and receiving monotherapy duloxetine (90 mg/day). She was educated about neonatal behavioral syndrome, and duloxetine was subsequently decreased to 60 mg/day.

The child was delivered without complication at 38 weeks. Upon delivery, she was blue, with minimal respiratory effort and oxygen saturations in the 80s. Her Apgar scores were 7 and 9. After birth, she was transferred to the neonatal intensive care unit because she continued to require oxygen. The child was started on antibiotics while possible causes of transient tachypnea were assessed. Basic laboratory examination, blood gas, echocardiogram, and chest and abdominal x-rays were all normal on day 1. Breast feeding was discouraged because of concerns of exposure to duloxetine, and the mother was advised to switch to sertraline. Antibiotic treatment was discontinued after blood cultures remained negative.

On day 3, the child was weaned to room air but developed "jerky rhythmic movements," or "twitchiness." An electroencephalogram (EEG) showed nonspecific encephalopathic findings. The child did have episodes of shaking, and the EEG revealed no correlated changes. Phenobarbital was started, and despite a high blood level the following day, the child continued to experience occasional twitching. Head computed tomography, magnetic resonance imaging, and lumbar puncture were all normal. A repeat EEG conducted at 7 days was suggestive of subclinical seizures. A follow-up EEG at 7 weeks was normal. Phenobarbital was discontinued, and the child was diagnosed with tremors and neonatal seizures associated with neonatal behavior syndrome. At age 2, the child is healthy with consistently normal neurobehavioral development.

This case demonstrates the syndrome referred to as poor neonatal adaptation or neonatal behavioral syndrome, characterized by jitteriness, poor muscle tone, weak cry, respiratory distress, hypoglycemia, low Apgar score, and seizure (1). These symptoms start within hours, generally require only supportive care, and end within 1 to 2 weeks. The syndrome may occur in up to 30% of infants with selective serotonin reuptake inhibitor exposure (2), with a risk ratio of approximately 3.0 (3) or higher for premature infants (4). The mechanism underlying the syndrome is unclear. A dose of fluoxetine or nursing may decrease symptoms assuming they stem from withdrawal. Reports indicate a higher risk with exposure to paroxetine and venlafaxine, agents with the shortest half-lives, but data are very limited pertaining to newer antidepressants such as duloxetine, mirtazapine, and bupropion. Better characterization of poor neonatal adaptation and its etiology could reduce invasive procedures and inform the difficult decisions in treating mental illness during pregnancy.

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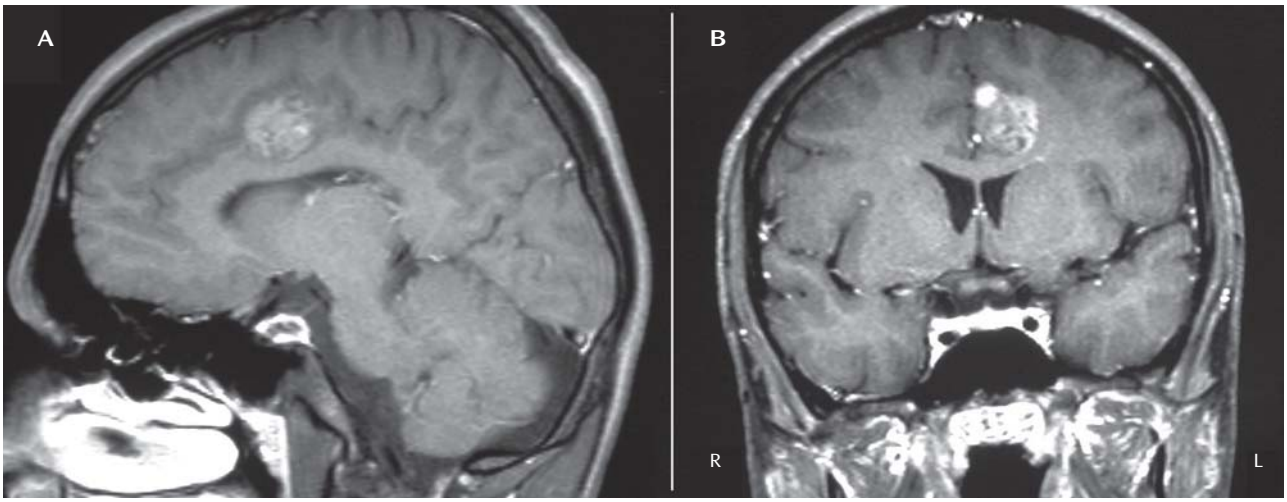
Cingulate Gyrus Tumor Presenting as Panic Attacks

TO THE EDITOR: The diagnosis of panic disorder is usually straightforward, but tumors or epilepsy of the temporal lobe may rarely present as panic attacks (1). We report the case of a teenager who presented with short-lasting episodes resembling panic attacks secondary to a dorsal anterior cingulate ganglioglioma.

A 15-year-old boy presented with a 3-month history of recurrent, unexpected panic attacks occurring four to five times daily. His clinical history was unremarkable, and no stressful events were reported. During his panic attacks, he experienced intense anxiety, palpitations, trembling, shortness of breath, feelings of choking, dizziness, lightheadedness, and hot flashes. The episodes were unprovoked and usually lasted 1 to 2 minutes. On two occasions, he reported loss of muscle tone in the lower limbs. He developed concern (after >1 month) about having fur-

FIGURE 1. Magnetic Resonance Imaging of Patient[a]



[a] Sagittal (A) and coronal (B) gadolinium-enhanced T1-weighted sections of the left dorsal anterior cingulate tumor (Talairach coordinate: –12.5, 9.6, 44.1; Montreal Neurological Institute: –12.6, 8.3, 4.8). R=right side; L=left side.

ther attacks and their implications. No drug abuse, medication intake, criteria for other anxiety disorders, or agoraphobia was present. A diagnosis of panic disorder was presumed, and the patient was treated with bromazepam without benefit. Neurological consultation was then requested. General neurological examination was normal. Blood and urine tests, including thyroid hormones, were unremarkable. Magnetic resonance imaging (MRI) revealed a tumor on the left dorsal anterior cingulate (Figure 1). An electroencephalogram (EEG) showed epileptiform frontal discharges (left >right) during a panic attack. The patient started carbamazepine with complete disappearance of panic attacks. He underwent neurosurgical radical resection of grade II ganglioglioma that did not invade the corpus callosum or surrounding structures. After resection, carbamazepine was discontinued without reappearance of panic attacks.

This case underscores the importance of ruling out general medical conditions for panic disorder diagnosis. The differential diagnosis between panic disorder and partial epileptic seizures on the basis of symptoms is sometimes challenging (1). In light of this case report, it would be prudent for patients with similar short duration (<2 minutes) of the episodes or loss of muscle tone to have an EEG and MRI before a diagnosis of panic disorder is determined. Partial temporal lobe epilepsy and panic disorder have overlapping symptoms; however, this is the first report of left frontal seizures from a left dorsal anterior cingulate brain ganglioglioma (<0.1% among brain tumors) that presented as panic disorder. The EEG findings and the good response to carbamazepine and tumor removal further support the hypothesis of partial seizures (1).

The most prevailing neuroanatomical model suggests that panic disorder patients may have an especially sensitive limbic "fear network" involving the amygdala and related brain areas, including the cingulate (2, 3). This model is consistent with converging experimental evidence that indicates a role of the left dorsal anterior cingulate in the pathogenesis of panic disorder (4).

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Correction

In the article "Reduced Amygdala Response to Fearful Expressions in Children and Adolescents With Callous-Unemotional Traits and Disruptive Behavior Disorders," by Abigail A. Marsh et al. (published online February 15, 2008; doi: 10.1176/appi.ajp.2007.07071145), in Table 2, in the heading for the F values, the degrees of freedom should have been listed as "2, 33."