Delirium From Baclofen Withdrawal After Suicide Attempt

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Delirium is often observed in the setting of overdose. Although discontinuation of medications is commonly practiced in this scenario to avoid overdose toxicity, this in itself can be problematic and contribute to worsening delirium. In the case described, the abrupt cessation of baclofen resulted in psychotic symptoms and, subsequently, self-injurious behavior. The EEG obtained in this setting was clinically useful because it pointed toward drug effect and encouraged further investigation in this arena. Finally, baclofen is a γ-aminobutyric acid (GABA) agonist, and GABA-ergic neurons have been implicated in the pathophysiology of psychosis. This report raises questions as to whether the vulnerability to psychosis in delirium and the vulnerability exhibited in primary psychotic disorders overlap pathophysiologically and/or etiologically.

Case Report

Mr. A, a 39-year-old man, was admitted to our psychiatric inpatient service from the emergency department after a medication overdose in a suicide attempt. He was taking many medications, the majority of which were for spasticity and chronic pain as a result of myelopathy secondary to multilevel disc disease. Because Mr. A was uncertain about the details of his medication regimen, the exact agents used in the overdose were initially unknown. Of note, the results of urine and serum toxicologies were negative at the time of admission.

Collateral information obtained later in the hospital course revealed that Mr. A's medication regimen included 400 mg t.i.d. of oral gabapentin, 60 mg t.i.d. of oral oxycodone, 100 mg b.i.d. of oral celecoxib, 60 mg/day of oral nifedipine, 20 mg/day of oral omeprazole, 12 mg t.i.d. of oral tizanidine, and 30 mg q.i.d. of oral baclofen. With the exception of gabapentin, all of the medications were continued upon admission. The omission of gabapentin was unintentional; it was a consequence of our failure to obtain an accurate description of the medication regimen.

Mr. A's course proceeded uneventfully until he became delirious approximately 48 hours after admission. His Mini-Mental State Examination (MMSE) score decreased to 16 of 30 from 23 of 30 on admission. He was heard frequently calling out the names of individuals who were not in the room. His medications (with the exception of nifedipine) were subsequently discontinued because of concern about their possible contribution to delirium.

A delirium workup was pursued. While Mr. A was febrile to 38.5° on hospital day 2, there was no leukocytosis. Other laboratory values were notable only for an admission serum sodium concentration of 130 mEq/liter that rapidly corrected, making its contribution to delirium doubtful. A head computed tomography scan without contrast and a chest X-ray were both unremarkable.

Mr. A's course worsened further, and he required restraints and continuous observation. He engaged in self-injurious behavior, such as lacerating his neck with an aluminum can, poking his eyes with his fingers, and attempting to strangle himself with a pulse oximetry cord. His vital signs were within normal limits, with the exception of a consistently elevated pulse, which ranged from 100 to 119 bpm. Fluphenazine was initiated for the treatment of psychotic symptoms.

Because Mr. A failed to improve, an EEG was obtained to assist in diagnosis. Low-voltage fast activity was noted, pointing in the direction of drug-induced phenomena. Oxazepam was introduced under the hypothesis that Mr. A might be in benzodiazepine withdrawal. Fluphena-

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zine was discontinued. When we learned that Mr. A had been taking baclofen, a literature review was pursued. At least 10 case reports were identified implicating baclofen withdrawal in a variety of neuropsychiatric syndromes. Most applicable to this case was an article describing a withdrawal syndrome in a patient who had attempted suicide by ingesting at least 800 mg of baclofen.[1] After abruptly having his medication discontinued, the patient became febrile and experienced hallucinations in the setting of delirium (as did Mr. A); he later stabilized with reintroduction of the medication.

We thus reintroduced baclofen on hospital day 8, and Mr. A's symptoms of delirium rapidly improved; he was soon able to coherently describe auditory and visual hallucinations and the delusional thought content of the previous several days. He specifically reported his false belief that his children were deceased and described hearing voices telling him that this was the case. He also described seeing individuals that he later understood had not actually been present. Within approximately 48 hours after reintroduction of baclofen, Mr. A's MMSE score improved to 27 of 30.

Discussion

This case raises two interesting clinical points as well as a research question. First, physicians often discontinue all medications after overdose with the intention of avoiding overdose toxicity. However, as this case points out, the abrupt cessation of some medications, such as baclofen, can be problematic. Second, the utility of the EEG in the differential diagnosis of delirium is also noteworthy, as low-voltage fast activity pointed in the direction of a drug effect, encouraging further investigation of the role of the patient's medications in the delirium.

Baclofen is a GABA (principally $GABA_B$) agonist. GABA is the principal inhibitory neurotransmitter in the CNS, and GABA-ergic neurons are thought to play a role in psychosis.[2] GABA receptor subunit genes have been studied in relation to the major psychotic disorders schizophrenia and bipolar affective disorder. Whether the vulnerability to psychotic symptoms in delirium and the vulnerability seen in these disorders overlap pathophysiologically or etiologically merits further study.

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