Letters

and appear to be quite similar to the etiologic reasoning presented in DSM-IV. Perhaps, they might be used as a starting point for the formal study of criteria for causal associations in the context of general medical conditions that might be incorporated into DSM-V. Our team would welcome the leadership of the developers of the DSM in this process.

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Reference


Protracted Benzodiazepine Withdrawal Syndrome Mimicking Psychotic Depression

TO THE EDITOR: The acute benzodiazepine-withdrawal syndrome is well known to clinicians, but less often appreciated is that discontinuation of benzodiazepines after long-term administration can produce disturbances in mental status lasting several months or longer. Herein described is a case that emphasizes the importance of prompt recognition and treatment of this condition.

Case Report

The patient was a 41-year-old woman without previous psychiatric problems who presented with a 6-week history of evolving symptoms of major depressive disorder with psychotic features, including persistent depressed mood, poor concentration, decreased appetite, insomnia, anhedonia, anergia, psychomotor retardation, and paranoid ideation including ideas of reference, the belief that she was being poisoned and persecuted by co-workers, and frequent complaints that the air was tainted with the odor of something burning or with the smell of rotting flesh. Medical and neurological evaluations—including electroencephalogram, magnetic resonance imaging of the brain, and an HMPAO (hexamethylpropyleneamine-oxide) brain SPECT (single photon emission computed tomography) scan—were normal. At the time of the initial evaluation, the patient’s husband mentioned that about 2 months before, she had discontinued Librax (5 mg chlordiazepoxide and 2.5 mg clidinium bromide, 1–3 capsules daily), which she had been taking for the past 25 years for irritable bowel syndrome (IBS).

Psychotic depression was diagnosed because of the characteristic presentation and negative neurologic evaluations, and because neither the patient nor husband felt that there was a causal relationship between the Librax discontinuation and her current symptoms. Initiation of risperidone (3–6 mg/d) produced improvement in all psychotic symptoms within 4 weeks and was discontinued 12 months later without reemergence of psychotic symptoms. All other “depressive” symptoms, however, persisted over the 14 months following her initial presentation despite full trials of paroxetine, fluoxetine, bupropion, venlafaxine, and lithium; the patient was considering electroconvulsive therapy as her next treatment option. Approximately 10 months into treatment, the patient briefly resumed the Librax for gastrointestinal symptoms and found that it seemed to give her “more energy” and “clearer thinking.” Believing that this medication would ultimately be harmful to her, however, she discontinued it after a few days, and her previous symptoms rapidly reemerged.

Fourteen months following her initial presentation, the patient had a recurrence of severe symptoms of IBS, for which chlordiazepoxide (10 mg tid) was prescribed. Within 1 day of starting this medication, all gastrointestinal and psychiatric symptoms had disappeared and, according to her husband, she was once again “completely normal.” This time she continued the medication, and at follow-up 4 and 12 months later the patient had a normal mental status and continued to feel and function well, vowing never to discontinue her “Librium” again.

Discussion

This case demonstrates four important points. First, withdrawal of a benzodiazepine after years of treatment can produce a protracted withdrawal syndrome that mimics other psychiatric disorders
and is unlike that observed in acute benzodiazepine-withdrawal syndromes. Second, the symptoms of protracted benzodiazepine withdrawal may not improve over the course of a year. Third, this case serves as a reminder that when psychiatric symptoms develop following discontinuation of any chronically administered medication, reinstitution of that medication should be considered as a first-line therapy whether or not a causal relationship is apparent. Finally, this case also reminds us that GABA-ergic mechanisms should not be neglected in seeking a comprehensive understanding of the neurochemistry of depression.3

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References