given at the same frequency, although steady-state serum level equivalency has yet to be studied in dosing less frequent than QID. IV valproate sodium is supplied 100 mg per ml in 5 ml single-use vials, and Abbott Laboratories recommends administration over a 60 minute period, not to exceed 20 mg/min, although infusion rates of 3–6 mg/kg/min have been shown to be well-tolerated.[7] Our patient was given infusions of 250 mg over 60 minute TID and tolerated this well.

In this case, intravenous valproate was effective as a mood stablizer and was well-tolerated. We would like to encourage our colleagues to consider intravenous administration of valproate sodium as maintenance therapy for patients with bipolar disorder who are temporarily unable to take oral medication, especially those patients with relative contraindications to, or problematic side effects from, IV benzodiazepines or antipsychotics. Further study is needed to evaluate IV valproate's long-term efficacy in preventing manic or depressive relapse.

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Moria and Witzelsücht from Frontotemporal Dementia

SIR: Frontal lobe disorders can produce moria, or foolish or silly euphoria, and Witzelsücht, or a tendency to tell inappropriate jokes.[1,2] Although previously characterized in a number of pathologies, little is known about the manifestations of these behaviors in frontotemporal dementia (FTD).

Case Report

A 57-year-old right-handed female had a 2-year personality change described as increased gregariousness, excitement, and a tendency to indiscriminately approach strangers without apprehension. She had become the life of the party and would laugh, joke, and sing all the time. The patient had decreased self-care and hygiene and wore the same clothes every day. In addition, she had developed a compulsive tendency, particularly with hoarding of money, and an addiction to ice cream with marked weight gain.

Her past medical history was negative, but her family history was positive for dementia and amyotrophic lateral sclerosis (ALS). Her paternal grandmother expressed dementia in her 50's, and her paternal uncle died at 58 with ALS.

On examination, her general demeanor was slightly euphoric and unconcerned. She was very talkative, animated, and disinhibited. Her most salient behaviors were almost continuous silly laughter and excitement (moria of Jastrowitz) and frequent childish jokes and puns (Witzelsücht). The patient would also make frequent personal comments about the examiner or touch the examiner.

The rest of her examination revealed cognitive changes primarily in language. She was fluent except for word-finding pauses, and she had good auditory comprehension and repetition but had difficulty with naming and made literal paraphasic errors. The patient's performance on a 10-item verbal memory test was zero spontaneously but 8 on recognition. The patient was able to do praxis tasks and simple drawings, and the rest of her neurological examination was entirely normal.

Most of her laboratory results, including syphilis serology, were unremarkable. Magnetic resonance imaging showed prominent atrophy in the anterior temporal lobes. Single-photon emission tomography scan showed circumscribed areas of hypoperfusion in both temporal lobes, much larger on the right. The pa-

tient was treated with citalopram with some behavioral improvement, and the family was informed of specialized genetic testing and genetic counseling.

Comment

Frontotemporal dementia (FTD) is a neuropsychiatric syndrome with progressive degeneration of the frontal lobes, anterior temporal lobes, or both.[3,4] The core clinical criteria for diagnosing FTD include progressive declines in social conduct, personal regulation, insight, and emotional reactivity.[3] This patient met criteria for FTD, and her family history suggested a familial form of the FTD-ALS spectrum.

Frontal temporal dementia is a variable and often asymmetric disorder with a range of neuropsychiatric symptoms.[4] Previously described from vascular lesions and tumors involving the orbitofrontal region and from neurosyphilis, moria and Witzelsücht can also be the most prominent symptoms of FTD. Moria includes childish euphoria and cheerful excitement,[1] and Witzelsücht includes excessive and inappropriate facetiousness, jokes, and pranks.[2] These behaviors may be specifically related to disturbances in the right anterior temporal orbitofrontal region,[5] as evidenced in this patient. Furthermore, moria and Witzelsücht may respond to serotonin selective reuptake inhibitor and other psychoactive medications.

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Frovatriptan-Induced Hypomania

SIR There has been an ongoing debate in the literature about whether antidepressants can induce hypomania in patients with underlying bipolar disorder.[1] There is, however, less knowledge about other medications inducing such changes. The following case is about how a medication belonging to the class of drugs, called the triptans, induced a hypomanic episode in a bipolar patient.

Case Report

Mr. A is a 51-year-old single, white male who was seen at an outpatient private psychiatry clinic. He was diagnosed with bipolar II disorder and maintained on lamotrigine 150 mg/day. The patient was last seen 3 weeks previously with a Young Mania Rating Scale (YMRS)[2] of 7, with no hypomanic or mixed episodes in the previous 3 months. In that interim the patient was only taking lamotrigine and sumatriptan 50 mg as needed for migraine headaches as prescribed by his neurologist. Mr. A had few mood switches under the current medication regimen. Various prophylactic treatments for these migraine headaches had been tried in the past with either poor results or intolerable side effects.

Mr. A was then started on frovatriptan 2.5 mg per day for migraine headache prophylaxis. Two days later the patient was seen in the psychiatry clinic. At that time Mr. A was very irritable, anxious, sleeping poorly, rapid speech, and frequently tangential with intermittent looseness of associations: with an YMRS score of 29. Mr. A denied any increase in stress in home, work, or social environments. With the patient's permission, Mr. A's radical change was discussed with his neurologist and it was agreed to try the following: immediately discontinue the frovatriptan and take olanzapine 2.5 mg as needed for sleep.

The patient was seen 6 days later in the outpatient clinic. At this time he was less anxious, calmer, exhibited regular speech with goal direction, and was less agitated: his YMRS score was now 9. The patient reported not having taken any olanzapine. Another week later Mr. A was even calmer with a YMRS score of 6.

A review of the available literature by the author was unable to elicit any other cases of a triptan causing irritability and/or hypomania. Mr. A's reaction could be attributed to either the other triptans having a half-life ranging from 2–6 hours, while frovatriptan's half-life is 25 hours causing it to more readily accumulate in the body—as it did after 3 days[3] or could be caused by frovatriptan's four times greater affinity for $5HT_{1B}$.[4] If more reports of this adverse effect surface, it may become necessary to add frovatriptan to the antidepressants as possible causes of inducing mania/hypomania.

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