

Treatment of Catatonia With Zolpidem

To the Editor: Zolpidem has been used successfully as a rapid test for patients suspected to be catatonic.¹ Lorazepam or electroconvulsive therapy (ECT) are considered the standard of care.² Zolpidem is well-described as a test for catatonia.³ This case report explores zolpidem as a long-term treatment option when standard therapies are not tolerated or ineffective.

"Mr. A" was 46-year-old man with schizoaffective disorder with catatonic features. He was admitted for disorganized behavior and mutism. His medications were continued, including bupropion, risperidone, and lithium, and low-dose aripiprazole was added. Ten days of treatment led to increasing depression. ECT was initiated for treatment-resistant depressive symptoms and catatonic features. Six treatments with unilateral, nondominant electrodes were administered without any notable effect. Two days later, rigidity and increased creatine phosphokinase, without fever or autonomic instability, led to diagnosis of atypical neuroleptic malignant syndrome (NMS). He was treated with dantrolene, amantadine, and bromocriptine for 1 week. Despite resolution of signs of NMS, his catatonia persisted.

Lorazepam trials improved his rigidity, but made him very agitated, requiring restraints. A zolpidem-challenge test was administered, showing partial resolution of rigidity and mutism. He was started on zolpidem 10 mg tid. On the 5th day, zolpidem was increased to 10 mg q 6 hrs., and divalproex 500 mg q 8 hrs. was added to treat the mood disturbance underlying his catatonia. Four weeks later, with complete resolution of his catatonia, he was discharged on zolpidem 10 mg tid for 3 months and divalproex 1,500 mg daily.

Growing evidence supports catatonia as associated with mood disorders and general medical conditions, rather than with schizophrenia.² Treatment of Mr. A's psychotic disorder with atypical antipsychotics did not relieve his catatonia and, even worse, precipitated NMS. Reduced dopamine in the catatonic brain is postulated to be a risk factor for development of NMS.⁴

During treatment of NMS, the patient received bromocriptine, dantrolene, and amantadine. These medications have been used experimentally for catatonia,⁵ but failed in the above patient. After NMS was treated and the patient had failed standard therapies, his catatonic features were targeted with long-term treatment with zolpidem. In one other published case (plus a number of cases presented by

Cottencin in symposia¹), zolpidem's use has been extended beyond testing purposes as treatment for catatonia.³ This case suggests that there may be some patients for whom zolpidem is a viable option for chronic management of catatonia.

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