Quetiapine for Chronic Motor Tic Disorder

TO THE EDITOR: There have been few case reports on the successful use of quetiapine for the treatment of Tourette's syndrome and other tic disorders in children and adolescents (1–3). We discuss our successful treatment of a patient with such a disorder.

Mr. A was a 26-year-old single African American man who had been diagnosed with chronic motor tic disorder at age 7. He had symptoms of social phobia, including difficulties with public speaking and daily social interactions. His tic disorder was familial. Trials of medications since grade school included haloperidol, pimozide, clonidine, sertraline, alprazolam, propranolol, diazepam, olanzapine, and risperidone. These trials included full therapeutic doses for a sufficient duration. Risperidone was the most effective medication in relieving his symptoms but caused extensive weight gain, which led to discontinuation. The weight gain stopped after the risperidone was tapered; however, the tics reappeared. Diazepam and alprazolam exacerbated the tics. Clonidine and olanzapine were not effective in controlling the tics.

Mr. A was then given quetiapine, 25 mg/day, which was titrated to 400 mg/day over several weeks. Within a month, he reported that he could socialize with more ease and was not having any tics. For the first time ever, he was able to speak publicly without any tics or problems initiating speech. Weight gain occurred, although not as much as when he was treated with risperidone.

The few case reports on this topic include patients who had not responded or had intolerable side effects to trials of haloperidol, risperidone, and clonidine. In contrast, Huang et al. (4) reported that an adult developed tics after 1 month of treatment with quetiapine. With any antipsychotic medication, tardive dyskinesia may occur and mimic a new onset of tic disorder.

There are several possible mechanisms to explain the effect of quetiapine on tics. Quetiapine is unique among the atypical antipsychotics because it has a high serotonin-to-dopamine binding ratio. Parraga and Woodward (2) suggested that quetiapine's unique binding profile to dopamine D_4 receptors and serotonin 5-HT_6 receptors, its selective inactivation of the mesolimbic cortical dopamine neurons, and/or its effect on excitatory amino acids might lead to its effectiveness in improving tics.

To our knowledge, this is the first case report of the successful use of quetiapine for the treatment of a tic disorder in an adult. Further controlled trials to determine whether quetiapine is effective in the treatment of tic disorders in adults are warranted.

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Creutzfeldt-Jakob Disease Presenting as Psychosis

TO THE EDITOR: The onset of sporadic Creutzfeldt-Jakob disease is usually characterized by dementia and/or neurological symptoms. The frequency of the prodromal psychiatric manifestations in sporadic Creutzfeldt-Jakob disease ranges between 18% and 39% (1), with mainly depressive disorders, personality changes, and emotional lability. Cases of the onset of sporadic Creutzfeldt-Jakob disease with psychotic disorders are rare (2). We report the case of a patient with sporadic Creutzfeldt-Jakob disease presenting as psychosis.

Mr. A was a 31-year-old Caucasian man who was hospitalized for delusions of being controlled and persecutory, megalomanic auditory hallucinations, somatic tactile hallucinations, and visual hallucinations. He had no history of iatrogenic exposure, psychiatric disorders, or substance abuse disorders and no family history of psychiatric illness. The onset of the disorder was insidious. According to his family, Mr. A had had psychiatric symptoms for 9 months before the hospitalization: sleep disorders, auditory hallucinations, and delusional beliefs of persecution, and subsequently, social withdrawal, affective nonresponsivity, ideas of reference, physical transformation beliefs, and aggressive behaviors. His first EEG, performed 1 month before the hospitalization, was normal.

At the onset of the hospitalization, Mr. A had memory problems characterized by an impaired ability to learn new information and daytime wandering, verbal stereotypies, inappropriately smiling, and reduced speech. Haloperidol, 15 mg/day, was used over 3 weeks with no improvement. His memory impairment gradually worsened. Neurological symptoms, such as hyperreflexia and hypertonia, appeared 16 months after his first psychiatric symptoms. He never had myoclonus. His condition deteriorated subsequently, with ataxia, mutism, and incontinence. He died 22 months after appearance of the first psychiatric symptoms.

During hospitalization, repetition of the EEG showed nonspecific cortical abnormalities, without the periodic triphasic waves typical of sporadic Creutzfeldt-Jakob disease. A computerized tomographic scan showed diffuse cortical atrophy. The results of laboratory tests and CSF analysis were normal. The neuropathological findings at autopsy confirmed the diagnosis of sporadic Creutzfeldt-Jakob disease, including frontal and temporal atrophy, extensive cortical spongiform changes, neuronal loss, and gliosis throughout the cerebral cortex.

We report an unusual initial presentation of sporadic Creutzfeldt-Jakob disease characterized by psychotic symptoms, confirming the initial report of Dunn et al. (2). However, the hypothesis of sporadic Creutzfeldt-Jakob disease and schizophrenia comorbidity cannot be ruled out. The psychotic features in sporadic Creutzfeldt-Jakob disease are usually fleeting and transient, while our patient fulfilled the