A 36-year-old Man with Agitated Behavior, Psychosis

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The patient was a single, 36-year-old white man. He was admitted to our institution due to agitated behavior and psychosis, with a diagnosis of bipolar I disorder from his most recent episode, which was mixed and severe with mood-incongruent psychotic features. He presented with depressed mood of 3 weeks’ duration, associated with early and intermittent insomnia, decreased energy, racing thoughts, and psychomotor agitation.

Furthermore, he exhibited persecutory delusions, believing that people were “out to get him,” and was thus physically aggressive to those he encountered. He denied any suicidal or homicidal ideations, and substance abuse screens returned with negative results.

The patient was diagnosed with myasthenia gravis at 19 years, but because he was “living on the streets,” he failed to receive adequate treatment. He was also diagnosed with bipolar disorder at 19 years, while incarcerated, when he exhibited isolative behavior and depressed mood. This led to his first hospitalization. As with his neurological condition, his treatment was irregular.

He was born by cesarean section with no known complications. He was removed from his mother’s custody at 7 years because of neglect and was subsequently raised in the foster care system. He has the literary equivalency of a sixth-grader, has never been substantially employed, and was homeless at 18 years. There is no family history of psychiatric illnesses other than his mother having a history of drug abuse.

Laboratory work, including CBC, CMP, LFT, BAL, and urine toxicology, were within normal limits. Computed tomography (CT) scan of the chest was negative and showed no evidence of an enlarged thymus.

On examination, the patient appeared his stated age. He was 5-feet 10-inches tall and weighed 180 lb. He had a tattoo on the left and right forearms. The patient’s attitude was guarded and evasive, with minimal eye contact. He appeared anxious. The patient reported no auditory, visual, tactile, gustatory, or olfactory hallucinations. He did not have any suicidal or homicidal ideations at time of evaluation.

The patient’s orientation, memory, and cognitive functioning could not be assessed because of the patient’s agitation. He was unable to care for himself and instigated fighting with another patient at a psychiatric inpatient setting.

During the course of his hospitalization, the patient demonstrated substantial improvement in his demeanor after a 4-week course of mood stabilizers, antipsychotics (Seroquel XR; AstraZeneca, 600 mg at bedtime), and group psychotherapy (recreational therapy and occupa-
tional therapy). On the fifth week, he again demonstrated episodes of irritability, isolativeness, and internal preoccupation — symptoms in accordance with a psychotic episode.

Of particular significance to this psychotic episode was the appearance of a drooping eyelid. As the psychosis progressed, the drooping seemed to worsen, and the onset of muscle weakness began to take hold. In the days that followed, while irritability and persecutory delusions (stating the police were after him) continued, so did his general weakness. He took to spending his afternoons in bed. A neurology consultation resulted in institution of pyridostigmine, which led to a dramatic improvement in his physical and psychiatric symptomatology.

A similar episode of myasthenia and psychotic exacerbation occurred at week 12 of his admission, and an adjustment in the myasthenia gravis treatment regimen hastened its resolution.

Documented reasons for this admission are: noncompliance with medications following his most recent psychiatric admission; lack of family support during his hospitalizations and throughout his mental illness; and being homeless.

**DISCUSSION**

Myasthenia gravis (MG) is an antibody-mediated autoimmune attack of the muscle-nicotinic acetylcholine receptors (AChRs) or of the muscle-specific tyrosine kinase (MuSK) receptors. These receptors are expressed on the postsynaptic membrane of the neuromuscular junction (NMJ). MG affects approximately two of every 100,000 people and can occur at any age. The features of the disease are fluctuating muscle weakness and skeletal muscle fatigability.

In approximately 85% of MG patients, pathological alterations of the thymus gland are found.

A literature search showed very few case reports demonstrating an association between the physiology of MG and psychiatric symptoms. In 1945, Delgado and Rosas reported one case of MG and psychosis. Eaton found six cases of psychiatric disorders among 175 myasthenia patients, including one case of manic-depressive psychosis. In 1950, Robert L. Williams and Maximilian Silbermann reported a case of a 20-year-old female patient with a diagnosis of MG for 8 years who developed symptoms of schizophrenia psychosis, which resolved with neostigmine and electric shock therapy.

In 1993, Musha et al reported psychosis in three patients with MG associated with thymoma and high serum titers of anti-acetylcholine receptor (AchR) antibody. They proposed a concept of paraneoplastic autoimmune neuropsychiatric syndrome. This concept was refuted by Keesey et al, who suggested that the antigenic differences between the AChRs and the neuronal nicotinic acetylcholine receptors (nAChR), together with the very low concentrations of muscle antibodies in the CSF, make the claim highly unlikely that CNS cholinergic systems are affected by muscle antibodies in MG patients.

The patient described here was diagnosed with MG and bipolar disorder at 19 years and was hospitalized at 36 years with neuropsychiatric syndrome (NPS) symptoms, including hallucinations and emotional disturbances with psychomotor excitement, such as agitation and anger. Literature review uncovers a pattern in which NPS symptoms usually precede by several months to years the onset of MG and, thereafter, NPS symptoms are closely related to worsening and relapse of MG.

Early case histories of MG associated with psychosis described the deteriorating mental status as occurring during periods of increasing dyspnea and cyanosis. Some authors also suggested that abnormal mental behavior could have been caused by elevated blood carbon dioxide levels associated with respiratory insufficiency in MG patients.

Mood stabilizers, such as lithium carbonate, have been reported to produce new-onset myasthenic symptoms and also exacerbation or unmasking of MG. Corticosteroids, the standard therapy for MG refractory to anticholinesterase medications, are known to cause depression, anxiety, and psychosis. Because MG and even its treatment can cause psychiatric morbidity, psychiatric symptoms must be evaluated carefully.
The interaction between MG and psychiatric disorders requires greater appreciation, especially in the primary care setting where MG is first diagnosed. MG symptoms, such as fatigue, lack of energy, and shortness of breath, often overlap with the similar psychiatric symptoms.\(^3\)

Little data are available from the literature regarding the prevalence, etiology, and psychopharmacotherapy of psychosis in patients with MG.

**CONCLUSION**

Little is known about the etiology and possible link between MG and psychosis. In literature, very few case reports were found relating MG and psychosis.

At the root of the issue is a need for more research on the relationship between MG and psychiatric illness. Our case was selected to highlight the necessity of additional information to improve the treatment of MG and comorbid psychosis.

**REFERENCES**