A 63-Year-Old Man with Motor and Behavioral Disturbances

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A 63-year-old man with a history of schizophrenia, seizure disorder, traumatic brain injury, and stroke presented for increased disorganization for 5 days after he suffered what appeared to be a seizure-like episode. Since that time, the patient was acting in an increasingly unusual manner: sitting and staring blankly for hours at a time, not sleeping, not eating, walking backwards, and walking into the street without any regard for his own safety.

The patient’s schizophrenia had responded well to olanzapine in the past, but he had stopped taking this medication in 2004. He had also suffered a major stroke in 2011 with residual aphasia and oropharyngeal dysphagia. He also has a seizure disorder (which pre-dates the stroke).

On presentation to the emergency department, the patient had widened eyes and was staring intensely, he moved his body up and down the gurney without purpose, answered questions inappropriately with random words such as “slurp” and “surgical,” and would repeat the same random word until interrupted. Levetiracetam 500 mg twice daily was added to his medication regimen (the patient was already taking phenytoin 100 mg three times daily) to prevent seizure recurrence. Psychiatry service added olanzapine 10 mg every 8 hours as needed for agitation while the patient waited to be admitted for medical workup. Differential diagnoses at that time included complete blood count, blood chemistry, urinalysis, urine drug toxicity screen, liver functions tests, vitamin B12, folate, HIV, RPR, and ammonia (all of which gave no explanation for the patient’s presentation). Magnetic resonance imaging showed large areas of gliosis in the left temporo-occipital lobe as well as smaller areas in the bilateral frontal regions, consistent with a history of old trauma and/or ischemia.

The patient was initially admitted to internal medicine. Tests included complete blood count, blood chemistry, urinalysis, urine drug toxicity screen, liver functions tests, vitamin B12, folate, HIV, RPR, and ammonia (all of which gave no explanation for the patient’s presentation). Magnetic resonance imaging showed large areas of gliosis in the left temporo-occipital lobe as well as smaller areas in the bilateral frontal regions, consistent with a history of old trauma and/or ischemia.

The patient was then seen by the neurology service, who also noted poverty of speech, word repetitions, and inappropriate responses to questions. Due to excessive sedation, levetiracetam was discontinued after 1 day (phenytoin was still continued), and outpatient neurology follow-up was recommended.

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For the first 3 days in the hospital while the patient stayed on the medical floor, he continued to be disorganized in his behavior but was able to follow simple commands and answer “yes/no” to questions, although his answers were often random and unreliable. He required about two doses of olanzapine 10 mg (as needed) per day for agitation.

After an unrevealing medical workup, the patient was transferred to the psychiatry floor. Olanzapine 20 mg at bedtime was prescribed.
for presumed decompensated schizophrenia; however, the patient showed no improvement on this medicine. Two weeks into treatment, the patient was noted to have decreased eating, weight loss, mutism, and peculiar movements (suggestive of carphologia), along with the appearance of frontal release signs. He would wave his arms in front of him in a purposeless manner and appeared to be picking at the air above him or at the bars on the window, or pinching his legs. Neurology was reconsulted and considered phenytoin toxicity, but the patient was not displaying the more characteristic choreiform-like movements, his phenytoin level was found to be within normal range at 16.1 mcg/mL, and his presentation was not consistent with non-convulsive status epilepticus.

Catatonia

DIAGNOSIS

Catatonia

The patient’s presentation was suspicious for catatonia. The patient scored 8 points on the first 14 screening items on the Bush-Francis catatonia rating scale (BFCRS) for excitement, staring, grimacing, stereotypy, and mannerisms. A trial of oral benzodiazepines was started (the patient adamantly shook his head “no” when injections were mentioned), and the patient was noted to have a positive clinical response. The olanzapine was tapered off quickly over a 24-hour period and a course of benzodiazepines was started: 2 mg at 6 a.m., 1 mg at 12 p.m., and 1 mg at 3 p.m. daily for 7 days. His peculiar movement disorder mostly disappeared, he became more focused, and he became more appropriately reactive. His answers to questions were now more appropriate and spontaneous, and he was able to speak two- to three-word sentences without ending in word repetitions. His appetite improved and he gained a modest amount of weight. Lorazepam was tapered off over the next week and he was discharged to a rehabilitation facility for reconditioning.

DISCUSSION

Catatonia is a psychomotor syndrome first defined by German physician Ludwig Karl Kahlbaum in his monograph *Die Katatonie oder das Spannungsirresein* in 1873. It was defined as specific motor abnormalities in a progressive disease course with stages that include depression, mania, stupor, confusion, and eventually dementia. It has been reported that the prevalence of catatonia can range from 7.6% to 38% in psychiatric patients (10% in acute psychiatric inpatients) and more commonly occurs in the setting of mood disorders, especially mania.

There are many hypotheses for the pathophysiology of catatonia, including disturbances in motor circuitry and neurotransmitters, but it is still relatively unclear. Studies have suggested involvement of the basal ganglia, thalamus, connections with the cortex, and gamma-aminobutyric acid (GABA) and D2 neurotransmitter disturbances.

Catatonia can be associated with metabolic, toxic, neurologic, or psychiatric conditions, and its treatment is unlike other psychiatric disorders in that it responds well to benzodiazepines and usually worsened by antipsychotics. It is also often subtyped into a “retarded-stuporous” form and an “excited-delirious” form. There is also a malignant subtype characterized by acute excitement, delirium, hyperthermia, autonomic instability, and catalepsy. The *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, text revision (DSM-IV-TR) provides limited diagnostic classifications of catatonia, including as due to a general medical condition, as a specifier in mood episodes, and as a subtype of schizophrenia, with the latter two requiring the presence of at least two symptoms to meet criteria. Some authors advocate for catatonia as a condition of its own, with its own set of specific diagnostic criteria.

There are a number of rating scales that describe criteria to define catatonia. Since its development in 1996, BFCRS has been the most widely used in catatonia research. It includes 23 items, the first 14 of which can be used as a shorter screening tool. The presence of two or more items is enough to identify catatonia. Most physicians seem to accept that a minimum of two of the more than 40 described catatonic conditions are present.
motor signs are needed to make the diagnosis.\(^3\)

Conditions that can present with similar symptoms to catatonia include stroke, stiff-person syndrome, malignant hyperthermia, locked-in syndrome, akinetic mutism, non-convulsive status epilepticus, and Parkinson’s disease. Of interest, neuroleptic malignant syndrome (NMS) and serotonin syndrome (SS) are felt to be variants of malignant catatonia. The term NMS was applied when the condition was associated with antipsychotic drug use, and except for additional gastrointestinal issues of SS, the symptoms of SS and malignant catatonia are virtually identical.\(^2\)

Although there are no current, large-scale studies for evaluating therapies in the treatment of catatonia, clinical experience and the literature consistently have shown benefit and decreased mortality with the use of electroconvulsive therapy (ECT) and/or benzodiazepines in the treatment of catatonia.\(^6\) Benzodiazepines are considered first-line treatment except in cases of malignant catatonia, which have shown better response to ECT.\(^6\) Acute treatment of catatonia has a good prognosis, and symptoms will usually improve within 24 hours of lorazepam administration.\(^6\) However, if unrecognized, untreated, or incorrectly treated, (eg, by the continuation of antipsychotics), catatonia can worsen, develop into the malignant form, and/or cause permanent disability or death.\(^2\) The long-term prognosis is determined by its underlying etiology and symptomatology; malignant symptoms (eg, autonomic instability, hyperthermia, delirium, etc) are a harbinger of worse overall prognosis.

**CONCLUSION**

Catatonia is a relatively common syndrome of motor abnormalities that can often be overlooked or mistaken for another condition. Identifying catatonia may present a challenge, but if unrecognized it increases the risk for significant morbidity and mortality. Treatment with benzodiazepines and/or ECT has been found to be effective. Physicians should be aware of this syndrome’s manifestation, differential diagnoses, and treatments. Screening for catatonia should be undertaken for patients with increased risk factors such as those with organic brain injury or major systemic illness (consult and liaison cases), underlying severe affective or psychotic illness (psychiatric inpatients), or in patients with history of catatonia.\(^7\)

**REFERENCES**