

LESSONS LEARNED AT THE INTERFACE OF MEDICINE AND PSYCHIATRY

The Psychiatric Consultation Service at Massachusetts General Hospital (MGH) sees medical and surgical inpatients with comorbid psychiatric symptoms and conditions. Such consultations require the integration of medical and psychiatric knowledge. During their thrice-weekly rounds, Dr. Stern and other members of the Psychiatric Consultation Service discuss the diagnosis and management of conditions confronted. These discussions have given rise to rounds reports that will prove useful for clinicians practicing at the interface of medicine and psychiatry.

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Dread Complications of Catatonia: A Case Discussion and Review of the Literature

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Have you ever wondered which of your patients is at risk for catatonia, a potentially fatal syndrome with protean manifestations? Have you wondered which medical complications might arise during the course of catatonia and how you could prevent or treat such complications?

If you have, then the following vignette and case discussion should serve as a stimulus to enhance your knowledge about the complications of catatonia and about strategies that can be employed to prevent and treat them.

Case Report

Ms. A, a 63-year-old woman with a history of untreated hepatitis C virus infection, cirrhosis, aortic stenosis, type II diabetes mellitus, chronic renal disease, and hypothyroidism, was admitted to our hospital for evaluation of her medical and psychiatric state.

According to her family, Ms. A had chronic depression (lasting more than 30 years), but it had significantly worsened over the last several years in the context of marital strife; they also described periods of “excitement” (when she would not sleep for days on end and her speech was pressured) lasting less than a month every few years. Approximately 4 years before the current admission, Ms. A became paranoid about her family, and she became more isolated. During the year prior to admission, her depression worsened, and she stayed in bed almost continuously; psychotropic medications (risperidone, citalopram, and chlorprothixene) were initiated. Nonetheless, she spoke rarely, ate and drank little, became increasingly rigid, and was unable to care for herself; therefore, she was hospitalized.

On evaluation at our hospital, Ms. A had asymmetric rigidity; there was no clonus, but she had a negative Babinski sign bilaterally, tremors in her upper extremities, and cogwheeling and waxy flexibility in her left arm. She was awake and alert but did not speak. Her eyes were open, but she appeared vacant. She moved her hand to shake the interviewer’s hand, and she could roll herself over. She did not appear to be hallucinating.

Laboratory testing revealed pancytopenia and elevated transaminase, low serum albumin, increased total protein, and elevated thyroid stimulating hormone (47.9 uIU/mL) levels. Due to her rigidity, waxy flexibility, mutism, and staring, as well as a history of increased rigidity associated with use of antipsychotics, Ms. A was diagnosed with catatonia. Neuroleptics were discontinued and intravenous (IV) lorazepam was initiated.

Unfortunately, neither a specific medical nor neurologic etiology for her catatonia could be established; Ms. A’s hospital course was complicated and extended. She developed multiple drug-resistant urinary tract infections (requiring IV antibiotics), decubitus ulcers, worsening contractures, an upper gastrointestinal bleed (for which she was treated for *H. pylori*

with a proton pump inhibitor), and multiple aspiration events (that led to hypotension, fever, and care in the medical intensive care unit [ICU]). Since IV lorazepam failed to reverse her condition, electroconvulsive therapy (ECT) was initiated; Ms. A had 10 treatments without significant improvement. Following completion of the ECT trial, Ms. A was sequentially started on topiramate (which led to further sedation and was discontinued), benzodiazepines, and dopamine agonists (carbidopa-levodopa and amantadine); although these medications led to initial improvement, Ms. A relapsed.

What Is Catatonia?

Catatonia, a syndrome of either short or long duration caused by a wide range of medical, neurologic, and psychiatric illnesses, is typically manifest by affective, behavioral, cognitive, and motor symptoms.¹ Unfortunately, its etiology is often obscure and dread complications may ensue. Accurate and timely diagnosis increases the chance that the syndrome will be reversed before life-threatening complications develop.

The *Diagnostic and Statistical Manual of Mental Disorders*, Fourth Edition, Text Revision¹ (DSM-IV-TR) notes that catatonia can complicate several psychiatric conditions (e.g., major depressive disorder, mania, a mixed affective state, and schizophrenia) with psychomotor and behavioral signs and symptoms. Catatonia is also associated with medical disorders (termed *catatonic disorder* due to a general medical condition) and use of medications (e.g., dopamine-blocking agents). According to the DSM-IV-TR, criteria for catatonia are met if at least 2 of the following are present: motoric immobility as evidenced by catalepsy (including waxy flexibility) or stupor; excessive motor activity (that is apparently purposeless and not influenced by external stimuli); extreme negativism (an apparently motiveless resistance to all instructions or maintenance of a rigid posture against attempts to be moved) or mutism; peculiarities of voluntary movement as evidenced by posturing (voluntary assumption of inappropriate or bizarre postures), stereotyped movements, prominent mannerisms, or prominent grimacing; or echolalia (pathologic, parrot-like, and apparently senseless repetition of a word or phrase just spoken by another person) or echopraxia (the repetitive imitation of the movements of another person).¹ Catatonia is understood as a syndrome of complex movements and behaviors caused by neurochemical changes that are associated with multiple psychiatric, medical, and neurologic etiologies.

What Causes Catatonia?

Etiologies of catatonia include psychiatric (most often affective disorders and schizophrenia), neurologic (including structural brain disease, seizure disorders, and

postencephalitic states), and medical (e.g., systemic lupus erythematosus, toxic-metabolic states [e.g., acute intermittent porphyria]), or the syndrome may result from use of medications (e.g., neuroleptics, steroids, disulfiram, or hallucinogens).² While it is not known how each of these conditions leads to catatonic states, researchers believe it is a result of abnormalities in central nervous system neurotransmitters (including dopamine, γ -aminobutyric acid, and glutamate).³

Before effective treatment strategies were developed, mortality rates approached 50% in cases of “lethal catatonia”³ as a result of medical complications associated with the syndrome. The current response rate of acute catatonia to first-line treatments (i.e., benzodiazepines and ECT) varies from 70% to 85%,⁴ and cases of treatment-refractory chronic catatonia are rare.

Although effective treatments for acute catatonia exist, if a patient does not respond to either use of IV lorazepam or ECT, or to discontinuation of any aggravating medications, the clinician must continue searching for a treatable etiology of catatonia. Diagnosis is often a challenge due to the medical comorbidities that cloud the clinical picture. The differential diagnosis of catatonia is broad: vascular abnormalities include midbrain lesions; basal ganglia strokes; spinal arteriovenous malformations; infectious diseases including tetanus, rabies, and encephalitis; strychnine poisoning; metabolic disorders including hypocalcemia; inherited disorders including malignant hyperthermia; autoimmune disorders such as stiff-person syndrome; neurologic diseases such as Parkinson’s disease/parkinsonism, akinetic mutism, and neurodegenerative disorders; syndromes associated with medication use including serotonin syndrome and neuroleptic malignant syndrome; mass lesions; delirium; and coma.⁵

What Complications of Catatonia Can Arise?

Due to the prolonged nature of Ms. A’s catatonic illness, a host of dread medical complications arose. Already malnourished (as evidenced by her low albumin level) on admission to the hospital, she frequently received IV fluids and oral nutritional supplements. She then developed multiple drug-resistant urinary tract infections (secondary to urinary incontinence) and experienced multiple aspiration events (which caused severe hypotension and fever) that required ICU treatment. Despite aggressive nursing and physical therapy care, Ms. A also suffered from decubitus ulcers and severe contractures. Such complications are common among those with catatonia.

Regardless of its underlying etiology, catatonia can lead to medical complications involving every organ system; therefore, clinicians must be aware of, prepare for, and treat these conditions when detected.³ Treatable complications include dehydration and malnutrition (i.e., the

patient must be adequately hydrated and provided with nutritional support [with either total parenteral nutrition or enteric feeding]), incontinence (that requires hygiene precautions to reduce the likelihood of serious infectious complications and skin breakdown), and prolonged staring and reduced blinking (for which use of eye drops can prevent serious ocular complications [e.g., ocular surface irritation, ocular pain, and decreased vision] from a reduction in tear formation).³ Immobility places the patient at risk for deep venous thrombosis (DVT) and pulmonary emboli (PE) (which can be prevented by use of prophylactic anticoagulation), decubitus ulcers, contractures, and infections (which can be minimized by diligent nursing techniques and physical therapy).^{2,3} A wide array of additional complications (e.g., acute renal failure, adult respiratory distress syndrome, burns, cachexia, cardiac arrest, dehydration, disseminated intravascular coagulation, gait abnormalities, gastrointestinal bleeding, hepatocellular damage, hypoglycemia, laryngospasm, myocardial infarction, pneumonia, pulmonary emboli, respiratory arrest, rhabdomyolysis, seizures, sepsis, thrombophlebitis, urinary retention, and death) have also been associated with catatonia.^{3,6}

How Can Catatonia Be Treated?

The treatment of catatonic patients is complicated and often involves a multidisciplinary approach, with input from nursing staff, physical and occupational therapists, nutritionists, and other specialties that focus on preventative strategies with the physician. Nonetheless, despite adequate preventative measures, medical complications commonly occur; the treatment team must be prepared to treat complications aggressively.³

Even after rigorous investigation, the underlying etiology for the catatonia may go undiscovered. However, whenever possible, the specific etiology should be targeted. Clinicians must be open minded regarding the differential diagnosis and anticipate complications; staff should not just react when complications arise.³ The medical workup may need to be broadened to include a lumbar puncture, an electroencephalogram, and magnetic resonance imaging and positron emission tomography scans. Creation of a multidisciplinary treatment team (e.g., with a nurse, physical therapist, occupational therapist, nutritionist, speech-language pathologist, case manager, and physician) will help anticipate the needs of the patient and his or her family to prevent further medical

complications. Novel psychiatric treatment strategies must also be employed.³

Supportive care is essential; it includes hydration, nutrition, mobilization, anticoagulation (to prevent thrombophlebitis), and precautions against aspiration. Agents thought to exacerbate the syndrome (such as antipsychotics or other dopamine blockers, e.g., metoclopramide) should be discontinued, and recently withdrawn dopamine agonists should be restarted.

Several case reports show a response to topiramate (which acts as a glutamate antagonist) after failure with benzodiazepines and ECT.⁷ Other case reports have used carbamazepine and IV valproic acid.⁸ Empirical treatment for an underlying medical or neurologic disorder should also be employed, as was the case with our patient, Ms. A.

Conclusion

Despite advances in the treatment of catatonia, approximately 30% of patients fail to respond to conventional strategies. Since medical conditions cause catatonia and may herald a serious illness, the mortality rate with catatonia is high. It is the physician's responsibility to provide prophylactic strategies against DVT/PE, aspirations, infections, and contractions, while utilizing the whole treatment team, and to treat complications when they arise. Open communication with the patient's family also helps to establish realistic goals for care.

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