Catatonia is a critical syndrome characterized by psychomotor symptoms including stupor, mutism, waxy flexibility, and posturing. Historically associated with psychiatric illness, catatonia is now recognized as a systemic medical syndrome, precipitated by medical, neurologic, and psychiatric disorders (1).

When symptoms of catatonia, psychosis, and delirium overlap, prioritizing which symptoms to target can present a diagnostic and treatment challenge. This case report highlights some of the overlapping features of these clinical syndromes and provides strategies to manage them in a medically ill patient, particularly when electroconvulsive treatment (ECT) is not readily available.

Case Report

A 71-year-old African American male with psychiatric history of schizophrenia disorder, bipolar type and medical history notable for prior neuroleptic malignant syndrome (NMS) from haloperidol, stage 4 sacral decubitus ulcer, chronic indwelling Foley catheter due to urinary tract infections (UTIs) was admitted to inpatient medicine from a nursing home due to acute alteration of mental status (AMS) and agitation. Patient was diagnosed and treated for a new UTI, acute renal failure (creatinine 2.28 mg/dL, baseline 0.8 mg/dL), and Clostridium difficile infection. Hospital course was complicated by agitated delirium responding to parenteral haloperidol. Home medication of divalproex sprinkles (will refer to as valproic acid [VPA] for simplicity) 125 mg by mouth daily was continued. Over eight days, intravenous (IV) fluids and antibiotics resolved acute medical issues, and patient was discharged back to nursing home.

Three days following discharge, patient was re-admitted to medicine for continued AMS and agitation with recurrence of acute kidney injury (creatinine 2.66 mg/dL). On day 2 of this admission, the psychiatry consultation liaison service was consulted for management of delirium related agitation and found the patient to be catatonic with prominent mannerisms (e.g., saluting repeatedly). Please see Figure 1 for depiction of hospital course.

Medical etiology of catatonia

In our patient, VPA and diazepam likely increased the seizure threshold (7). Benzodiazepines with longer duration of action, such as diazepam, are known anticonvulsants. With VPA use, a higher ECT stimulus may be necessary.

Our patient had decreased renal function intermittently, but decreased renal function does not appear to affect the elimination of diazepam (6).

Use of benzodiazepines to treat catatonia

In our patient, olanzapine (and later ziprasidone) were chosen due to their properties of these agents.

Evidence of VPA in treatment of catatonia

Evidence is mixed about use of second generation antipsychotics (SGAs) for treating catatonia

Simplicity, VPA’s safety and potential efficacy in catatonic patients.

In our patient VPA may have initially aided in catatonia improvement; however, the perceived benefit was not long lasting, especially later in hospitalization.

Managing breakthrough catatonia with diazepam

While our patient’s catatonia initially improved with lorazepam, he experienced episodes of breakthrough catatonia, prompting trial of the longer-acting diazepam. The transition from lorazepam to diazepam decreased the frequency of breakthrough catatonia, but precipitated a new hypoaffective delirium.

With VPA use, a higher ECT stimulus may be necessary.

This case highlights several difficulties that may be encountered while managing a medically ill patient with catatonia, delirium and predominant psychotic symptoms. Each syndrome requires different therapeutic approaches, with the treatment of one syndrome potentially worsening another. Care must be taken to address the most imminent harms associated with avoiding exacerbation of comorbid conditions (9). Ultimately, risks and benefits of each treatment should be carefully weighed when making these clinical decisions.

References