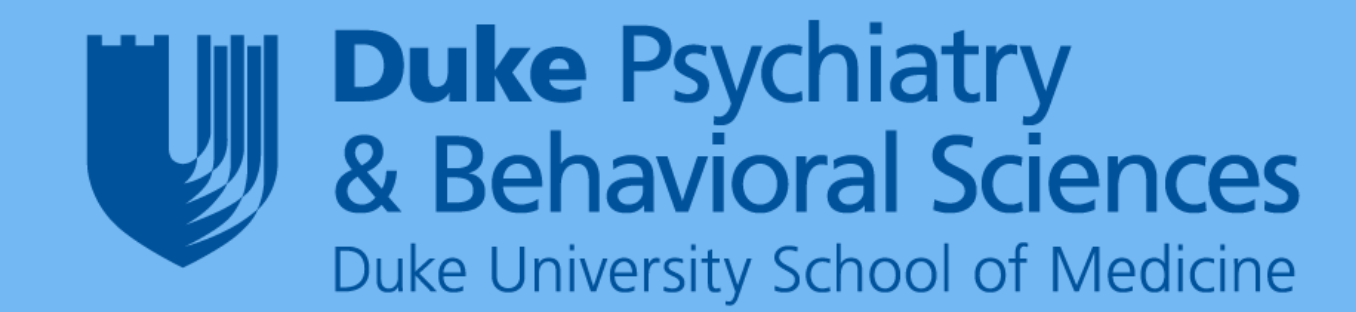


A Wolf in Cat's Clothing: Amantadine in the treatment of SLE Catatonia

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Background

- Catatonia is a syndrome with a broad differential including psychiatric, substance-induced, infectious or inflammatory causes
- Treating catatonia involves treating the syndrome itself, as well as identifying and addressing its underlying etiology
- We present a case of catatonia with atypical features and treatment course

Case

- Ms. A is a Haitian woman in her 40s with poorly controlled hypertension, preeclampsia and two psychiatric hospitalizations for postpartum mood episodes
- She presented with subacute progressive anxiety, paranoia, poor oral intake, withdrawal after being brought to the hospital for "getting stuck" at home
- In the ED, she displayed blunted affect, minimal engagement and eye contact, restless fidgeting, soft mumbled speech, and tangential, vague, perseverative thoughts
- Physical exam was notable for significantly elevated blood pressure and BFCRS of 13/39 (for mutism, staring, catalepsy, stereotypy, negativism, mitgehen and preservation)
- Oral lorazepam challenge led to symptom improvement but not resolution
- She was admitted to inpatient psychiatry

Work Up

- Labs were notable for pancytopenia, hypokalemia and mildly elevated liver enzymes
- Infectious workup and UDS were negative
- Given the unclear past psychiatric history and atypical clinical features, rheumatologic workup was sent and notable for elevated ESR, +ANA, hypocomplementemia, +dsDNA/Smith concerning for systemic lupus erythematosus (SLE)
- Rheumatology confirmed the diagnosis of SLE meeting criteria for an active flare
- Catatonia symptoms progressed despite scheduled lorazepam, with withdrawal and medication refusal resulting in hypertensive urgency.

Management

- On inpatient psychiatry, serial interviews in the patient's native language and collateral did not yield additional diagnostic clarity
- Screening tools for depression, mania, PTSD, psychosis were administered and inconclusive
- Mrs. A was transferred to medicine for treatment of presumed neuropsychiatric lupus (NPSLE) causing catatonia
- MRI brain, total spine and LP were, somewhat surprisingly, unremarkable
- After definitive treatment for NPSLE with IV steroids, hydroxychloroquine and cyclophosphamide she remained agitated, disorganized and increasingly paranoia
- On return to psychiatry, lorazepam was restarted with short-term but incomplete improvement in symptoms.
- Neuromodulation was considered for refractory catatonia, amantadine was added first with **surprisingly significant improvement in BFCRS, mental status and adherence to treatment**

- 13/39 • BFCRS on presentation
- 13-16/39 • After treatment of NPSLE
- 6/39 • Ativan + Amantadine 100mg BID
- 2/39 • Ativan + Amantadine 200mg BID
- 9/39 • Reduced Ativan + Amantadine

Diagram 1: Changes in BFCRS with interventions

Discussion

- NPSLE has protean clinical manifestations with no definitive diagnostic criteria, making timely diagnosis challenging
- History of psychosis can anchor clinicians to a psychiatric cause of catatonia, which may delay treatment
- Our case represents possible simultaneous contributions of psychiatric and medical causes to catatonia
- Literature supports a connection between SLE and catatonia, with most cases requiring treatment with benzodiazepines, immunosuppression, and often electroconvulsive therapy (ECT)^{1, 2}
- Adjunctive amantadine therapy prior to ECT or when ECT is not immediately available has been recommended but may be underutilized, especially when a medical cause of catatonia is suspected
- Our case supports the possible benefits of aggressively treating both the syndrome itself and its possible underlying causes simultaneously

Classifications and Prevalences of NPSLE

Central NPSLE	
Diffuse	
Cognitive impairment	(up to 80.0%)
Mood disorder	(up to 80.0%)
Anxiety disorder	(up to 40.0%)
Psychosis	(up to 11.0%)
Acute confusion state	(up to 7.0%)
Focal	
Headache	(up to 28.3%)
Cerebrovascular disease	(up to 15.0%)
Seizure disorders	(up to 20.0%)
Myelopathy	(up to 3.9%)
Demyelinating syndrome	(up to 2.7%)
Chorea	(0.9%)
Aseptic meningitis	(up to 2.7%)
Peripheral NPSLE	
Guillain-Barré syndrome	(up to 1.2%)
Autonomic disorder	(up to 1.3%)
Mononeuropathy	(up to 6.9%)
Plexopathy (...)	
Polyneuropathy	(up to 5.4%)
Myasthenia gravis	(0.2%)
Cranial neuropathy	(1.0%)

Table 3: Classification and Prevalence of NPSLE from Sarwar et al³

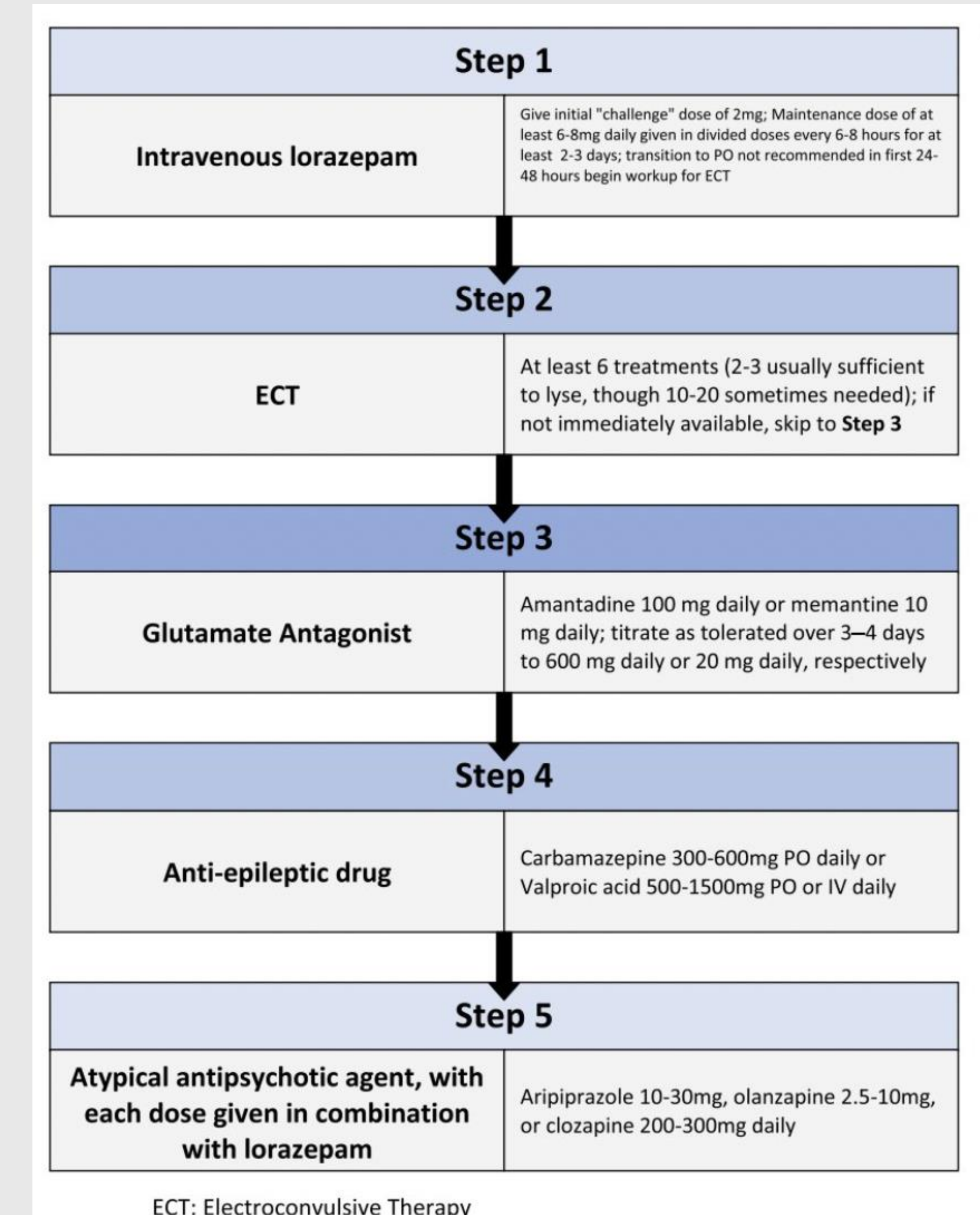


Diagram 2: Treatment algorithm for catatonia from Beach et al.⁴

Cultural Context

Our case of managing catatonia of unclear etiology in a Haitian woman who's first language is Haitian creole highlights the challenges of timely, accurate psychiatric assessment in the light of language barriers and the limitations of interpreter services in psychiatry. Moreover, in presenting this case we describe our efforts to ascertain more cultural context to clarify elements of the patient's thought process, including involving a religious leader and several family members to provide such context for diagnosis. We advocate for diversity in psychiatric recruitment to promote psychiatrists with lived knowledge of our patients' varied experiences, cultures and languages.

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Initial Work Up		Additional Work Up	
WBC	2.8	ANA	1:2560, speckled
Hgb	10.7	Anti-ds DNA	324 (<100 nl)
Plt	152	RF	15 (<13 nl)
MCV	78	ANCA	negative
AST/ALT	34/59	Anti-Sm	461 (<100 nl)
UA	2+ blood	Anti-RNP	401 (<100 nl)
ESR/CRP	33 / 0.42	Anti-Ro	20
		Anti-La	25
		C3	72
		C4	11

Table 1 and Table 2: Lab values from initial work up and additional work up