Treatment of Catatonia With Electroconvulsive Therapy in a Patient With Neuropsychiatric Systemic Lupus Erythematosus, Epilepsy, and Cerebral Palsy

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Nervous system involvement impacts at least half of lupus patients and is referred to as neuropsychiatric systematic lupus erythematosus (NPSLE).[1] Common symptoms include headache, mood disorders, cognitive symptoms, and seizures.[2] Catatonia is not recognized as a NPSLE manifestation but has been reported in at least 39 cases in the literature.[1] We will discuss a case of newly diagnosed NPSLE in a patient with epilepsy and cerebral palsy who developed protracted catatonia refractory to benzodiazepines and immunosuppression with complete resolution of symptoms after 20 electroconvulsive therapy (ECT) treatments.

Case Report

An 18-year-old male with history of epilepsy and cerebral palsy presented with new-onset fatigue, headache, behavior changes, and confusion in the setting of 2 months of rash, hair loss, myalgia, synovitis, intermittent fevers, and increased frequency of seizures. The patient was diagnosed with SLE confirmed by tissue biopsy.

On admission, his home antiepileptic drugs perampanel 10 mg every night at bedtime and lacosamide 200 mg twice daily were continued, and he was given pulse dose steroids. His SLE symptoms improved; however, his mental status worsened with breakthrough seizures and hyperactive delirium. Quetiapine and haloperidol were ineffective for agitation and were discontinued due to breakthrough seizures. On hospital day 33, the patient developed symptoms of catatonia that responded to lorazepam challenge, and he was put on scheduled lorazepam up to a dose of 12 mg with limited improvement. Mycophenolate mofetil and cyclophosphamide administration resulted in only transient catatonia improvement. ECT was initiated, and his Bush-Francis Catatonia Rating Scale[3] score improved from 26 to 8 after the first 5 ECT treatments. Due to the severity of his illness, his electrode placement was changed from right unilateral to bitemporal electrode placement, and this placement was continued for all subsequent treatments. Perampanel was discontinued due to longer half-life, and lacosamide was held before ECT with seizure duration greater than 20 seconds in all treatments. His ECT timeline was 3 times weekly for 12 treatments, weekly for 3 treatments, biweekly for 3 treatments, and triweekly for 2 treatments. His SLE symptoms resolved prior to discharge after 65 days of hospitalization, and he had full catatonia resolution 201 days after onset, receiving 20 ECT treatments in total. After his 20th ECT treatment, both his family and outpatient psychiatrist deemed further ECT treatments unnecessary. At the time of publication, it has been 3 years since his final ECT treatment, and his catatonia and SLE symptoms have not returned with outpatient management.

Discussion

Our patient's systemic and neurologic symptoms suggest SLE was the trigger for his catatonia. Case reports have shown an association between high-dose steroids and catatonia[4]; however, our patient did not improve with dose reduction. Antipsychotics may have induced catatonia, with NPSLE increasing vulnerability. Our patient received 2 doses of haloperidol 2 mg and 13 doses of quetiapine 25 mg prior to developing catatonia; however, his catatonia persisted for months after antipsychotic cessation. Our patient's history of epilepsy and cerebral palsy may have contributed to increased risk of catatonia.[5,6]

No guidelines exist for managing catatonia in SLE. Despite growing evidence, catatonia is not mentioned in any criteria for NPSLE and is sparsely mentioned in the literature. In a review of 39 cases of catatonia with NPSLE, all patients except one were female, aged 12–46 years.[1] Five patients improved with immunomodulatory therapy alone. Prior analysis of these cases showed 81% received benzodiazepines and 38% received ECT, with session numbers ranging from 6 to 20.[7] Potential mechanisms for NPSLE-associated catatonia include inflammatory cytokines, microvascular occlusions, and direct antivascular antibodies.[7] There is evidence that certain antiepileptic drugs can affect the efficacy of ECT; our case suggests ECT can be safely performed with

lacosamide. Cerebral palsy has not been reported as a contraindication to ECT and did not necessitate alterations in our patient's treatment.

While catatonia may be rare in NPSLE, if not recognized and treated catatonia can be life-threatening. ECT should be prioritized in patients with catatonia in the context of NPSLE not responding to immunomodulation and benzodiazepines and can be safely used in patients with cerebral palsy and epilepsy on lacosamide.

Article Information

Published Online: July 25, 2024. https://doi.org/10.4088/PCC.24cr03707

Prim Care Companion CNS Disord 2024;26(4):24cr03707

Submitted: January 1, 2024; accepted April 4, 2024.

To Cite: Thompson TJ, Smart BD, Douglas HE, et al. Treatment of catatonia with electroconvulsive therapy in a patient with neuropsychiatric systemic lupus erythematosus, epilepsy, and cerebral palsy. *Prim Care Companion CNS Disord*. 2024;26(4):24cr03707.

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Relevant Financial Relationships: None.

Funding/Support: None.

Patient Consent: Consent was obtained from the patient's legal guardian to publish the case report, and information has been de-identified to protect anonymity.

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