Refractory Catatonia Due to N-methyl-D-Aspartate Receptor Encephalitis Responsive to Electroconvulsive Therapy

The Clinical Use of the Clock Drawing Test

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Anti–N-methyl-D-aspartate receptor (NMDAR) encephalitis is an autoimmune disorder with a prominent neuropsychiatric presentation including catatonia. Electroconvulsive therapy (ECT) has been reported to improve psychotic and catatonic symptoms in NMDAR encephalitis.[1] We present images of the clock-drawing test (CDT), which demonstrate the clinical neurologic dysfunction[2] and document the response of catatonic symptoms to ECT in a case of NMDAR encephalitis.

A highly educated woman in her late 20s with no previous neuropsychiatric history began experiencing insomnia, emotional lability, delusions, and hallucinations over a 2-month period. She was admitted to a psychiatric hospital and treated with antipsychotics and mood stabilizers for suspected bipolar disorder with minimal relief of her symptoms. Her symptoms progressed to abnormal movements, memory deficits, and catatonia. A lumbar puncture revealed antibodies to NMDAR, and she was transferred to our facility. Neuroimaging was normal. Pelvic imaging for ovarian teratoma and whole body positron emission tomography showed no underlying malignancy. Treatment with intravenous immunoglobulin and corticosteroids provided minimal improvement. Her catatonia was treated with lorazepam up to 36 mg/day with only partial response. She subsequently received ECT for refractory catatonia.

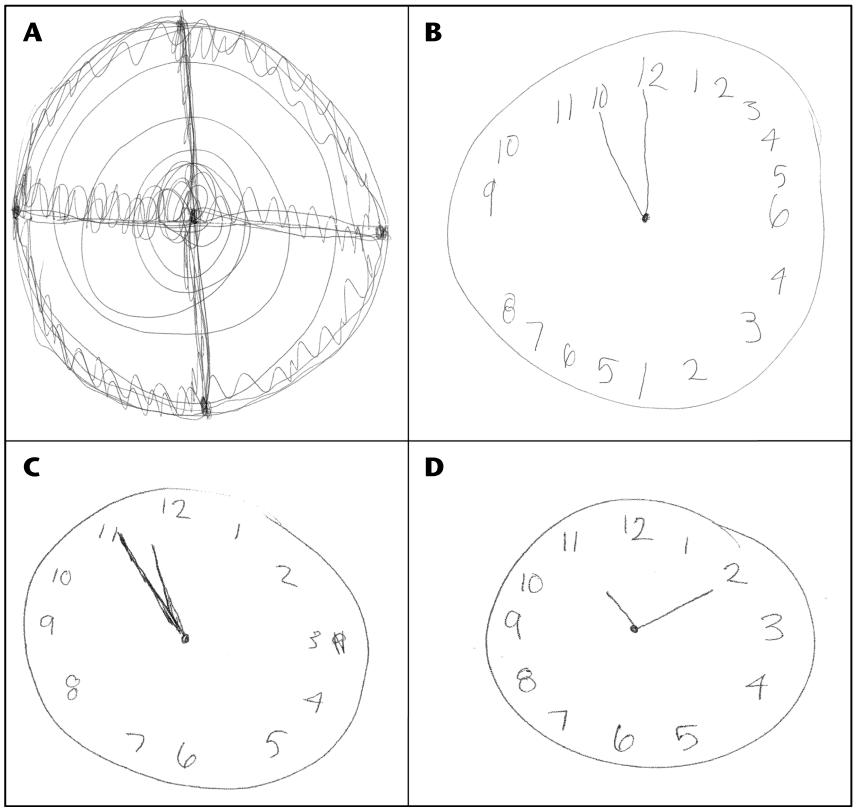

FIGURE 1. Clock-drawing tests performed pre-ECT (A), after second ECT (B), after sixth and final ECT (C), and 6 months follow-up (D).

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Figure 1A shows an initial CDT demonstrating profound perseveration obtained during the patient's severe catatonic symptoms and before initiating ECT. Bitemporal ECT was initiated (pulse width, 0.5 milliseconds; energy, 75 mC; frequency, 20 Hz) using a Thymatron System IV Integrated ECT Instrument (Somatics, LLC, Lake Bluff, Ill). Her catatonic symptoms improved immediately, Bush-Francis Catatonia Rating Scale[3] was 24 before her first ECT and 11 immediately after recovery. After the second ECT, her improvement on the CDT is notable (Fig. 1B). During the fourth ECT, energy was increased to 150 mC due to shortening seizure duration. After the sixth treatment, her catatonic symptoms had largely resolved with substantial improvement on the CDT (Fig. 1C), ECT was well tolerated and without complications.

Persecutory delusions persisted, and she was started on quetiapine for psychotic symptoms in addition to lorazepam for prophylaxis against the return of catatonia and mycophenolate mofetil for immunosuppression. She required no further ECT and was discharged to rehabilitation. At 6 months postdischarge, CDT was normal (Fig. 1D). Quetiapine was successfully discontinued, and lorazepam slowly tapered without complications. Neuropsychological testing found no residual deficits, and she successfully returned to work having made a full recovery.

The CDTs were shown to the patient and her family and were positively received as helpful documentation of the severity of her state and her recovery. She provided written consent to publish her case. She remains amnestic for the period of her severe catatonia and ECT. Nonneuropsychiatric physicians and others not familiar with catatonia were struck by the recovery seen in these clocks. The CDT was able to demonstrate the severity of her catatonic disorder and help break down stigma around this poorly understood condition. We agree with calls to reclassify catatonia as a systemic medical condition[4] and feel additional face-valid data will help this cause.

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