Published in final edited form as:

J ECT. 2012 September ; 28(3): 180–182. doi:10.1097/YCT.0b013e318255a8d4.

Tardive Seizure with Postictal Aphasia: A Case Report

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Abstract

Electroconvulsive therapy (ECT) is a highly effective treatment for certain psychiatric disorders with relatively few serious side effects or complications. Tardive seizures are one of these rare, but potentially fatal complications. Recognizing and treating tardive seizures is essential to prevent prolonged postictal confusion, progression to status epilepticus and associated soft tissue injury, anoxia, aspiration and death. Currently there is an unknown prevalence of their occurrence and an overall lack of clinical description of their phenomenology. We describe a case in which a patient develops a tardive seizure followed by a receptive and expressive aphasia, thought to be a variant of Todd's postictal paralysis. This case is further unique in that there was a lateralization of a motor seizure presumably to the hemisphere contralateral to the RUL electrode placement.

Keywords

electroconvulsive therapy; tardive seizure; adverse event

INTRODUCTION

Electroconvulsive therapy (ECT) is a highly effective treatment for certain psychiatric disorders with relatively few serious side effects or complications. Tardive seizures are one of these rare, but potentially fatal complications. They occur spontaneously after the termination of convulsion from ECT and after recovering full consciousness. They are not simply a prolonged seizure during ECT which is generally defined as lasting longer than 120 seconds on EEG¹ with a prevalence of 1-2% per new course of ECT treatment². The frequency of tardive seizures remains unknown, and there is an overall lack of clinical description of their phenomenology in the literature³, which has erroneously combined descriptions of tardive seizures with prolonged ECT seizures. Further complicating the picture is the presence of difficult to identify, nonconvulsive tardive seizures. Thus a major focus of the literature rightfully aims at reporting the sequelae of post ECT seizures, both convulsive and nonconvulsive status epilepticus. Accordingly, risk factors for tardive seizures are generalized from few retrospective case reports including all varieties of prolonged seizures. Factors include hypoxia, hyperoxygenation, more than one induced seizure and prolonged seizures during ECT; use of seizure-lowering psychotropics and antibiotics; pre-existing electrolyte imbalances; preexisting epilepsy disorder and adolescence and pregnancy⁴⁻⁶. Recognizing and treating tardive seizures is essential to

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prevent prolonged postictal confusion, progression to status epilepticus and associated soft tissue injury, anoxia, aspiration and death.

CASE REPORT

The patient is a 76-year-old married Greek-American with major depressive disorder, recurrent, severe without psychotic features without psychotic features as well as hypertension, hypercholesterolemia, benign prostatic hyperplasia and hypothyroidism who was admitted to inpatient psychiatry unit for worsening depression for 4 years with two weeks of suicidal ideation with various potentially fatal plans. The patient was determined to be a candidate for ECT, and he elected to enroll in a NIH-supported ECT clinical trial. The diagnosis of MDD was confirmed at the time of admission with a Structured Clinical Interview for Diagnosis (SCID). He had received at total of 14 ECT treatments at age 38 in 1973 with only mild, temporary, cognitive impairments. Psychiatric medications at admission included bupropion XL 300mg daily, fluoxetine 40 mg daily, alprazolam 0.25 mg twice daily, clonazepam 0.5 mg every evening, zolpidem 10 mg in the evening as needed. He had no history of epilepsy, head injury or family history of seizures. The patient was titrated off of bupropion, fluoxetine, alprazolam and zolpidem and was started on venlafaxine 37.5 mg daily. He received his first, second and third right unilateral (RUL) ECT treatments on hospitalization days 4, 7 and 9, respectively, using a Thymatron System IV (Somatics) with ultrabrief, 0.25 msec pulse width. He underwent a successful titration with the first stimulus at 5% energy which produced an electrographic seizure of 121 seconds at his first session, followed by stimulation at 30% energy (149.4 mC) at his subsequent sessions. His electrographic seizure for his second session was 92 seconds. For all treatments, he received 60mg of methohexital and 60mg of succinylcholine. Additionally he was given 800mcg of nitroglycerin during the second treatment.

During his third treatment, the stimulus dose was again 30%, resulting in a motor seizure lasting 29 seconds as confirmed by visual inspection and surface electromyogram of the cuffed right foot. The electroencephalographic (EEG) seizure was determined from a single channel of EEG (Fp1-M1), and lasted approximately 85 seconds, but the ending was indeterminate with poor post-ictal suppression by visual inspection. The EEG recording was continued for an additional 51 seconds to 136 seconds with no further EEG suppression. The computerized Thymatron analysis of post-ictal suppression yielded a response of "N/A." Due to a peak interictal blood pressure of 170 systolic he received 600mcg of nitroglycerin. The patient initially appeared to recover spontaneously from ECT with stable hemodynamics and normal respirations. Fifty-five minutes after the stimulus, the post-anesthesia care nurse noted that he was not responding appropriately to questioning. He was asked if he wanted a drink of water to which he was able to answer "yes," but provided nothing more complex in terms of verbal response. While taking a sip, the patient was noted to develop a right gaze deviation and his right arm reached upwards. Immediately following this, he developed a generalized tonic-clonic seizure. He became cyanotic and required 100% O₂ until the convulsion stopped thirty seconds later at about the same time that 2mg of intravenous midazolam was pushed. Airway patency was maintained throughout the event with a mandibular thrust, and the patient was ventilated with 100% oxygen.

Following, the patient had both a receptive and expressive aphasia. He was not able to name objects, repeat words or his name, and spoke in a nonsensical manner. Neurology was consulted and noted right nasolabial fold flattening and weakness and drift of the right lower extremity. CT, CTA and CT perfusion were performed and there were no large vessel cutoffs or volume perfusion mismatches to suggest an acute stroke. Upon arrival to the Neurology floor approximately six hours later, the patient was still displaying an aphasia. He became able to read some words but then added on nonsensical words and substituted words

mid sentence maintaining a fluent speech. He was unaware of his language deficits. An MRI demonstrated no evidence of any ischemia. An EEG revealed asymmetry of background between left and right hemispheres with left demonstrating 4-5 Hz theta activity of 20 uV and the right showing 9-10 Hz of 30 uV. By day three on the neurology service, it was noted that the patient's aphasia had greatly improved. However he was very depressed and was transferred back to the inpatient psychiatry service. A second EEG 6 days later revealed symmetry between the hemispheres, interpreted as a normal awake and sleep EEG. During his readmission to the inpatient psychiatry unit, the patient was titrated off of venlafaxine secondary to concerns that it may be elevating his blood pressure and was subsequently started on pregabalin and duloxetine. He was unenrolled from the ECT clinical trial and received no further ECT. Two weeks later he was discharged home with subjective and observed improvement in depression.

DISCUSSION

Tardive seizures are a rare complication of ECT with unknown frequency, perhaps due to underreporting. They occur after the end of convulsion from ECT and after recovering full consciousness; they are not simply prolonged seizures. Thus tardive seizures may initially present in the recovery room, and anesthesia staff may be the first to respond to this emergency. To this date, there is only one report of aphasia associated with a tardive seizure but it developed in the context of nonconvulsive status epilepticus⁴. Our case is also unique in that there was a lateralization of a motor seizure presumably to the hemisphere contralateral to the RUL electrode placement, with associated postictal language deficits.

The diagnosis of a tardive seizure appears clear cut, but in light of the psychomotor and cognitive dysfunction inherent in postanesthetic and postictal state, a full recovery of consciousness prior to new seizure onset can be blurred. It remains unclear to what degree our patient recovered from ECT prior to his generalized motor seizure.

High on the differential diagnosis is a nonconvulsive status epilepticus (NCSE). While the diagnosis of NCSE requires typical EEG findings, Roshe et al (2011) note that NCSE may only be detected at times with FDG-PET and or intracranial electrodes and that aphasia may be a leading symptom. Crider and Hansen-Grant (1995) found that their patient entered post-ECT NCSE despite clear cessation of seizure on electroencephalograph. One major difference was the presence of Cheyne-Stokes respirations in their patient and normal breathing in our patient. In a letter to the editor, Smith (2000) reviews five case reports, including his own, of NCSE following ECT as a cause for failure to regain consciousness. He describes a very similar scenario in which his patient had a generalized tonic clonic seizure 15 minutes after right unilateral (RUL) ECT. Unique to his case was a focus of seizure activity captured by EEG over the right hemisphere.

We must concede that it is possible that the seizure during the ECT procedure did not fully terminate and remanifested later in a generalized tonic clonic seizure. In most if not all ECT treatments, the EEG recording was taken from an ECT machine which is limited in leads and by scalp placement. It is possible that a seizure may become focal to deep medial aspects of frontal lobes and become undetected. Thus the regeneralization of a deep seizure cannot be ruled out.

Our patient's EEG findings are of great curiosity. Having received RUL ECT, expected findings would be slowing of the right hemisphere relative to the left. However, in the EEG several hours later after the generalized tardive seizure, the left hemisphere showed relative slowing. These findings are consistent with a left hemispheric focus for the tardive seizure, with consequent postictal aphasia. Physical exam findings during the seizure were consistent

with this lateralization as there was right gaze deviation and right arm tonic prior to the generalization.

Unique to this case is the presence of a receptive and expressive aphasia associated with the tardive seizure. There may be a normal variant of postictal language delay when a seizure is localized to left temporal lobe, lasting 68 to 1276 seconds¹⁰. Ficker et al (2001) found that postictal language delay is even more prolonged when propagation spreads from nondominant temporal lobe to dominant temporal lobe. Also, the rate of spread to the dominant medial temporal lobe was inversely related to the time of language recovery, thus suggesting that rapid spread increases a refractory period. Povlsen (2003) reviews a case report of nonconvulsive status epilepticus following ECT in which after a bilateral ECT treatment, one patient develops a speechless, apathetic confusion lasting several days. Unlike our patient, EEG on day 2 revealed an ongoing focal ictal pattern of 3-5 Hz high amplitude. Interestingly it was localized to left frontal region.

More probable than NCSE, given our patient's generalized motor seizure and subsequent EEG with left hemispheric slowing, is the aphasia variant of a Todd's postictal paralysis. In a study of 229 patients with generalized tonic clonic seizures who were prospectively evaluated, 14 had transient focal deficits, with 5 of the 14 having aphasia as a deficit, lasting 8-36 hours¹². One of these patients also demonstrated a gaze deviation. However, all 5 patients were also found to have had brain lesions noted to be ischemic strokes in middle cerebral artery territory.

In our patient, it was determined that the risk of another tardive seizure from subsequent ECT treatments outweighed the potential benefit. However, ECT has been successfully repeated after tardive seizures without further complications. In these limited case reports, there appears to have been an offending agent potentially lowering the seizure threshold such as a beta lactam⁶ or theophylline⁸. After its removal, ECT treatments were not associated with further tardive seizures.

Acknowledgments

supported by NIMH 1U01MH086127-01

Appendix

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