CASE REPORTS

Anesthesiology
1999; 91:574-5

An Unexpected Hyperkalemic Response to Succinylcholine during Electroconvulsive Therapy for Catatonic Schizophrenia

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Case Report

A 40-yr-old, 70-kg white woman with a long history of schizophrenia was referred for electroconvulsive therapy (ECT) for catatonia. The patient had previously undergone a successful course of ECT treatment at our university for catatonia of less than 2 weeks' duration. After discharge from the hospital for this previous episode of catatonia, the patient discontinued her medication and again became symptomatic. One month before the referral to our university for a second series of ECT, the patient presented to a local hospital with fever, rigidity, and mutism. A presumptive diagnosis of neuroleptic malignant syndrome was made, and the patient was treated with amantadine, dantrolene, and bromocriptine. This diagnosis was abandoned when it was determined that the patient had not taken any neuroleptic medication before admission.

A fever work-up was performed, including gallium scan, blood and urine cultures, head and abdominal computed tomography scans, and lumbar puncture. All test results were within normal limits. Because of some nonspecific ST-T wave changes and a history of complaints of vague chest pain related by the patient's spouse, cardiac enzymes were obtained and were within normal limits.

The patient was transferred to our university psychiatric hospital and was referred to the ECT unit for treatment. The usual pre-ECT evaluation by the ECT service and anesthesiology was performed. The patient had a normal electrocardiogram (ECG), blood chemistry, and complete blood count. The records from the previous ECTs were reviewed. Because of the delay in a diagnosis of catatonia, the patient had been catatonic with rigidity and mutism for 1 month before her first treatment. She had severely restricted mobility, and her extremities were stiff but loosened with passive movement.

The patient underwent the first treatment in the series without incident. General anesthesia was induced using 0.4 mg atropine, 3 mg curare, 10 mg labetolol, 80 mg methohexital, and 120 mg succinylcholine. The patient tolerated the procedure well and was returned to the nursing unit after a routine stay in the postanesthesia care unit.

The second ECT was administered 3 days after the first. The patient was anesthetized as before, and ECT was administered. After the seizure, the patient was noted to have wide-complex arrhythmia and several seconds of asystole. Two hundred milligrams lidocaine was administered intravenously, CPR was instituted, and the patient was intubated. Oxygenation, as monitored by pulse-oximetry, remained greater than 90% throughout. An arterial blood gas measurement was obtained and was within normal limits. The serum potassium with that sample was 5.4 mEq. A sequential multichannel autoanalyzer 7 obtained after the patient was stabilized was within normal limits. The serum potassium level was 5.1 mEq. The patient was transferred to the university hospital by ambulance. She was ventilated with oxygen during the transfer. After being examined in the treatment room, she was admitted to the intensive care unit, where ventilation was weaned and the patient was extubated several hours later. Physical examination was unremarkable for cardiac disease. Laboratory tests failed to show any electrolyte abnormality. Cardiac enzymes ruled out myocardial infarction, and ECG and chest radiograph were normal. A stress dobutamine echocardiogram was normal. Electrophysiologic studies showed no evidence of an underlying predisposition to a malignant arrhythmia. The patient was transferred back to the psychiatry service for ECT.

The third ECT was administered after evaluation of electrolytes the morning of treatment. The electrolytes were normal. Potassium level before ECT was 4.2 mEq. The patient was anesthetized as before followed by ECT stimulus. After the seizure, the cardiac monitor showed marked "peaking" of the T waves that returned to normal in 7 min. A serum potassium level of 5.5 mEq was obtained. The patient recovered without incident and was returned to the nursing unit.

The fourth ECT was administered the next day. The patient was anesthetized as before and ECT was administered in the usual manner. With this treatment, potassium samples were obtained before induction of anesthesia, after induction and before stimulation, 2 min after stimulation, and 4 min after stimulation. ECG strips were obtained at the time that the blood samples were drawn. Some peaking of the T waves were noted but did not seem as pronounced as they had in the previous treatment. The potassium levels were 4.0 mEq before induction, 6.8 mEq after induction and before stimulation, 6.9 mEq 2 min after stimulation, and 5.9 mEq 4 min after stimulation. The potassium level the afternoon after ECT was 4.0 mEq.

Based on these results, a decision was made to substitute mivacurium for succinylcholine during the next treatment. Serial potassium samples were obtained as during the previous treatment, and an ECG strip was run at the time each sample was obtained. The patient tolerated the treatment without complications. No changes were

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Received from the Department of Anesthesiology, Emory University School of Medicine, Atlanta, Georgia. Submitted for publication October 12, 1998. Accepted for publication February 18, 1999. Support was provided solely from institutional and/or departmental sources.

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Key Words: Anesthesia; cardiovascular effects; ions; neuromuscular relaxant; potassium.

noted on the ECG with respect to T waves. The potassium levels were 4.0 mEq before induction, 3.8 mEq after induction and before stimulus, 3.8 mEq 2 min after stimulus, and 3.7 mEq 5 min after stimulus.

Discussion

Catatonia is a neuropsychiatric syndrome that ranges in severity from a simple to a malignant form.[1] The malignant form may be fatal in extreme instances. The clinical features of catatonia include variable combinations of motor abnormalities, including the rigidity and immobility that was observed in our patient. Psychosocial withdrawal may manifest as mutism, staring, stupor, and negativism; however, excited features of combativeness, impulsivity, and mutism may also be present.

The differential diagnosis of catatonia includes neuroleptic malignant syndrome and some encephalitic syndromes. The difficulty in making the correct diagnosis may result in the patient becoming more ill because the definitive treatment is delayed. The preferred treatment for catatonia is ECT. Although there are no prospective randomized trials of ECT in catatonia, there is evidence that the sooner treatment is begun, the better the results.[2]

Increased potassium levels after administration of succinylcholine is well known.[3] In normal patients, serum potassium increases approximately 0.5–1.0 mEq after succinlycholine administration, but catastrophic hyperkalemia has been known to occur in patients who have experienced severe burns, patients with massive tissue damage (crush injury), or patients with upper or lower motor neuron damage. This increase in potassium release is caused by the development of perijunctional or extrajunctional receptors referred to as *upregulation*. A study by Fung *et al.*[4] demonstrated that there was an increase in potassium release in beagles who had one limb immobilized by casting. This increase required 14–42 days to become apparent. In this case we believe that the increase in potassium release may be related to the long period of catatonic immobility experienced by the patient before the onset of treatment. The arrhythmia that the patient experienced was in all likelihood a result of the hyperkalemia but was not recognized because the potassium level returned to normal before the blood sample was obtained. The quick return to a normal potassium level led us to look for other explanations for the event. We also may have been given a false sense of security by having treated this patient 3 days before with succinylcholine without apparent problems. In the first series of ECT, the period between the onset of symptoms of catatonia was less than 2 weeks. This interval may have been too short for the development of upregulation.

For catatonic patients with immobility, we suggest substituting a short-acting, nondepolarizing drug such as mivacurium in place of succinylcholine, because our data suggest that it can be used without fear of inducing hyperkalemia. We also suggest that a normal potassium level before and after ECT is no guarantee that hyperkalemia will not occur during the treatment, and that the patient should be closely monitored *via* ECG for T-wave peaking throughout the treatment.

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