



A Case Report of Hemiplegia after Electroconvulsive Therapy and the Management of Subsequent Treatments

Jack Zhang, MD, PhD* and Brandon R Seifert, MD, MS

Parkside Medical Anesthesia Associates, PC, Buffalo, NY, USA

Abstract

Hemiplegia occurring during the recovery period following electroconvulsive therapy (ECT) can be a potentially serious and devastating complication. Acute cerebrovascular accident (CVA) is the first and foremost consideration given the time sensitive nature of treatment and morbidity/mortality associated with the diagnosis. There are very few case reports of temporary hemiplegia following ECT, including a lack of recommendations how to handle the decision for subsequent treatments. Here we report a case of temporary hemiplegia diagnosed as Todd's paralysis attributed to ECT and offer recommendations on how to decide whether or not to proceed with subsequent ECT treatments.

Abbreviations

ECT: Electroconvulsive therapy; CVA: Cerebrovascular accident; MDD: Major depressive disorder

Introduction

Electroconvulsive therapy (ECT) involves brief electrical stimulation of the brain to provoke a seizure while a patient is under general anesthesia. The development of hemiplegia occurring during the recovery phase of ECT is a rare and potentially devastating complication. The differential diagnosis (see Table 1) [1] includes cerebrovascular accident (CVA) as the most time sensitive and potentially catastrophic. An alternative diagnosis, such as postictal transient hemiplegia (also known as

Todd's paralysis), can only occur as a diagnosis of exclusion. The development of hemiplegia following ECT necessitates a timely and extensive evaluation with significant healthcare resource consumption.

Upon searching PubMed for transient paralysis and electroconvulsive therapy, there appears to be very little report of this phenomenon in the literature [2-8]. Most reports of transient hemiplegia are in the context of unilateral electrical brain stimulation with resulting contralateral transient hemiplegia. There is no mention of whether subsequent ECT treatments will provoke recurrence of hemiplegia or the risk factors for such. Furthermore, while one study reported subsequent successful ECT treatments following an episode of transient hemiplegia [8], there are limited studies available to help guide recommendations for subsequent ECT treatments.

The aim of this case report is to describe an episode of transient hemiplegia following ECT. An ultimate diagnosis of Todd's paralysis was made. The decision making process for whether or not to continue subsequent ECT treatments following an episode of transient hemiplegia will be discussed. HIPAA authorization has been obtained from the patient.

Case Description

A 70-year-old man with body weight of 65 kilograms

Table 1: Differential diagnosis of acute hemiplegia.

Vascular	Non-Vascular
Cerebrovascular Accident	Post ictal/Todd's Paralysis
Transient Ischemic Attack	CNS tumor/abscess
Subdural/Epidural Hematoma	Hemiplegic Migraine
Subarachnoid Hemorrhage	Conversion Disorder
Hypertensive Encephalopathy	Hysterical Hemiplegia
Cerebral Venous Thrombosis	Demyelinating Diseases (Multiple Sclerosis, Guillain Barre')
Posterior reversible encephalopathy syndrome (PRES)	Toxic/ Metabolic (hypoglycemia, drug intoxication)
Reversible cerebral vasoconstriction syndromes (RCVS)	Alternating Hemiplegia of Childhood
	Spinal cord lesions (tumor, infectious, transverse myelitis)

*Key exclusion criteria for non-vascular diagnosis is emergent intracranial imaging studies for vascular assessment (CT and/or MRI)

was scheduled to undergo a series of electroconvulsive therapy (ECT) for diagnosis of refractory major depression disorder (MDD) including suicidality. His medical history included controlled hypertension, controlled type 2 diabetes mellitus, hyperlipidemia, and chronic hepatitis C without cirrhosis. His overall general health was good. The psychiatric medications he tried without improvement in his psychiatric condition were Zoloft, Celexa, Lexapro, Vistaril and Prozac. His symptoms of depression were not well controlled and he had ongoing suicidal thoughts. He was thus undergoing ECT given lack of improvement with other therapies and ongoing suicidal ideation.

His first two ECT treatments failed to achieve an adequate duration of epileptic response at an electric current of 403 millicuries. The tonic-clonic seizures lasted 22 and 23 seconds, respectively. Subsequent treatments included the addition of 250 mg of caffeine intravenously. The epileptic response improved to 56 seconds and 63 seconds, respectively, following the addition of caffeine for his 3rd and 4th treatments. His depressive symptoms started to improve following 4 ECT treatments. His recovery from each treatment was generally uneventful. He received labetalol 5 mg intravenously following each seizure with adequate response and discharge from recovery with normal vital signs within an hour.

The case for this discussion was the patient's 5th ECT treatment. The current set by the attending psychiatrist was 454 millicuries. The caffeine dose was increased to 500 mg intravenously and 0.2 mg Glycopyrrolate was given intramuscularly 30 minutes before ECT, per routine. Following preoxygenation, the patient was induced under general anesthesia with Methohexital 80 mg intravenously followed by Succinylcholine 80 mg intravenously once unconsciousness was obtained. He was hyperventilated via bag mask ventilation and electric current was applied on bilateral frontotemporal lobes after satisfactory muscle relaxation was achieved. His epileptic activity was observed on EEG. His pre procedure blood pressure was 150/70 mmHg and heart rate was 90 bpm. Following the sympathetic response to his induced seizure, the systolic blood pressure was recorded as high as 250 mmHg with a heart rate of 130 bpm. A dose of Labetalol 10 mg was given intravenously immediately upon attaining the vital signs. The seizure duration was unfortunately less than 20 seconds, so the psychiatrist increased the current intensity to 504 millicuries. The stimulus was reapplied after waiting for approximately 30 seconds while the patient was continuously adequately ventilated with 100% oxygen. A tonic-clonic seizure was elicited with this second stimulus and lasted for approximately 80 seconds. He slowly recovered with spontaneous breathing, but he was slower to wake up and more lethargic than usual. Upon recovering from the seizure, he moved to the right lateral position with

a systolic blood pressure of 180 mmHg and heart rate of 90 bpm. He maintained an oxygen saturation of greater than 95% throughout the treatment.

Upon arrival to the recovery room the patient became more lethargic, he was noted to have left facial droop, and the left upper and lower limbs could not move voluntarily. Cerebrovascular accident was immediately considered. The patient was transported to the nearest medical center for further evaluation. On the way he developed a convulsion in the ambulance. A paramedic gave him 5 mg of Midazolam intravenously and successfully intubated him for airway protection. In the emergency room, a neurologist performed a systematic examination, including CT imaging, and ruled out acute hemorrhagic cerebrovascular accident. The patient became more responsive and arousable within a few hours, was extubated and recovered completely with no neurologic sequelae. The attending neurologist diagnosed the patient with Todd's paralysis. The patient's psychiatrist decided to terminate future ECT treatments given improvement of his depression symptoms and concern for recurrent Todd's paralysis.

Discussion

Todd's Paralysis is a neurological disease experienced by patients with epilepsy, in which transient temporary paralysis occurs after seizures. Paralysis can be partial or complete, but usually only occurs on one side of the body. It may last for half an hour to 36 hours, with an average of 15 hours, then completely resolves. Todd's paralysis may also affect speech and vision. In 1849, Todd's original description of post-ictal paralysis was as follows: "He, who recovers from a severe fit, or from frequently repeated fits of epilepsy, are often found to labor under hemiplegia, or other modifications of palsy." He called this condition "epileptic hemiparalysis" [9].

The exact cause of Todd's paralysis is unknown, but speculation focuses around the likely decrease of electrical output of neurons in the motor cortex of the brain. Indeed, Todd's original description speculated that a certain state of "electrical exhaustion" was the cause of epileptic hemiparalysis [9]. The partial or complete paralysis that can occur as a result of epileptic hemiparalysis can be very difficult to distinguish from other causes, i.e. hemorrhagic or ischemic CVA. ECT can elicit a severe sympathetic response in patients with resulting hypertension and tachycardia, which can contribute to cerebral ischemia from profound vasoconstriction or rupture of intracranial vessels. It is paramount to immediately evaluate any patient who displays hemiparalysis for cerebrovascular accident, given the treatment and potential for improvement of symptoms is time dependent. Todd's paralysis, as was the case with our patient, completely resolves over time with supportive treatment and is a diagnosis of exclusion.

Previous reports mostly document a unilateral stimulus associated with post ECT Todd's paralysis [2-8], whereas this patient had bilateral stimulation. We speculate that the addition of a second stimulus at maximum current resulting in a second prolonged seizure was the likely culprit of Todd's paralysis in our patient. Perhaps minimizing the electrical current and utilizing a bilateral vs. unilateral stimulus is one way to reduce the risk of Todd's paralysis. However, the advantages and disadvantages of bilateral vs. unilateral ECT is not the focus of this article.

An important question to consider in a patient with a history of Todd's paralysis is whether he or she should continue with ECT treatments. In our case report, the patient discontinued ECT because of his improvement in symptoms at the time and the psychiatrist's concern about recurrence of Todd's paralysis. It is important to note that Todd's paralysis cannot be predicted. It can occur randomly and after multiple successful ECT treatments, as was the case with this patient. There is also report of multiple successful ECT treatments performed after one episode of Todd's paralysis [8]. The decision to continue with subsequent ECT treatments following an episode of Todd's paralysis should follow a multidisciplinary discussion, involving all members of the care team with the patient and supportive family members as well. The potential morbidity and resource consumption associated with post seizure hemiplegia is significant given the need for potential resuscitative measures and immediate stroke evaluation. Availability of a medical center capable of immediate stroke management and means to transfer a patient to such medical center should be considerations. Ultimately, the severity of the patient's psychiatric condition should take precedence. The timely treatment of significant suicidality or catatonic states may outweigh the potential risk of proceeding with ECT and recurrent Todd's paralysis.

In summary, we have reported a case of Todd's Paralysis following ECT. The key in management is to make a differential diagnosis including hemorrhagic or ischemic stroke, because management and patient recovery can be time dependent. Todd's paralysis following ECT is a rare event, but has a very favorable prognosis as opposed to CVA. The decision to proceed with subsequent ECT treatments following an episode of Todd's

paralysis should be patient specific and depend on the severity of their psychiatric condition and feasibility of immediate stroke evaluation.

Financial Disclosures

None.

Conflicts of Interest

None.

Author Contributions

Jack Zhang: This author performed a literature search using PubMed for obtaining information regarding hemiplegia and electroconvulsive therapy, and contributed equally in writing this manuscript.

Brandon R Seifert: This author was involved in direct perioperative care of the patient presented in this case report. This author obtained medical history and treatment records for the patient and contributed equally in writing and editing this manuscript in its entirety.

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Corresponding Author: Jack Zhang, MD, PhD, Parkside Medical Anesthesia Associates, 3871 Harlem Rd, Ste 202, Buffalo, NY 14215, USA, Tel: 716-836-7510

Editor: Renyu Liu, MD, PhD, Professor, Department of Anesthesiology and Critical Care, Perelman School of Medicine at the University of Pennsylvania, Center of Penn Global Health Scholar, Director of Stroke 120 Special Task Force, Chinese Stroke Association, 336 John Morgan building, 3620 Hamilton Walk, Philadelphia, PA 19104, USA, Phone: 2157461485, Fax: 2153495078, E-mail: RenYu.Liu@pennmedicine.upenn.edu

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