



Case Report

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Asystole Following Electroconvulsive Therapy in a Male Patient with Bipolar Disorder: A Case Report

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ABSTRACT

Electroconvulsive therapy (ECT) is known as a treatment method for psychological disorders. This method, like any other treatment, has disadvantages that could threaten the patient's life. During the process, some changes occur in the autonomic systems of the body. The aim of this study is to introduce a rare case of asystole during electroconvulsive therapy. The patient was a 48-year-old man with a history of bipolar disorder and a candidate for ECT. There were no problems in cardiac examinations. In the postictal phase of the seizure, the heart rate was not detectable. Cardiopulmonary resuscitation was started immediately with chest compressions, and airway management was performed by the anesthesiologist. The patient's cardiac rhythm converted to a sinus rhythm following five minutes of chest compressions. The ECG wave appeared, and the pulse oximeter was able to show SpO₂ and the heart rate. Meanwhile, the patient's airway was secured, and he was delivered to recovery with a blood pressure of 119.66 mm Hg and a heart rate of 105 beats/min. By retrieving the patient's consciousness and hemodynamic stability, he was transferred to the ICU for treatment and diagnostic procedures.

Introduction

Electroconvulsive therapy (ECT) is known as a treatment method for psychological disorders. A wide range of patients with mood disorders (depression and bipolar), drug-resistant schizophrenia, and critical neurological conditions benefit from this method [1]. Research shows that the mechanism of this method is related to an increase in neural growth factors and also enlargement of the hippocampus [2]. Today, ECT is known as a new treatment with gentle side effects, which is performed

under anesthesia and the administration of muscle relaxants [3].

This method, like any other treatment, has disadvantages that could threaten the patient's life. During the process, some changes occur in the autonomic systems of the body. Usually, the autonomic activity caused by electrical stimulation leads to an initial parasympathetic response, which is immediately followed by significant sympathetic activity. Due to the attenuation of vagal effect, tachycardia and a secondary increase in blood pressure are observed. In

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the textbooks of anesthesiology, transient bradycardia caused by primary vagal stimulation has also been reported [4]. Estimated mortality of ECT is close to 1.2 deaths per 100,000 cases. The most common side effect is cardiopulmonary complications, which occur in less than 1% of the cases. Serious and rare side effects include cardiac arrhythmias with or without changes in hemodynamics, respiratory distress, prolonged apnea, aspiration, prolonged muscle paralysis, and prolonged seizures. Mild side effects include muscle pain, nausea, and vomiting [5]. Complications occurring during ECT are either caused by the anesthetic drugs or the nature of ECT itself. Due to the use of anesthetic drugs, there is a possibility of hemodynamic disorder during ECT. Since most anesthetic drugs weaken the myocardium, hemodynamic disorder mostly occurs in the form of hypotension and tachycardia [6].

Case Presentation

The patient was a 48-year-old male with a history of bipolar disorder, without any history of systemic or organic diseases, who was an ECT candidate due to the lack of appropriate drug response. There was a history of smoking, with no history of drugs, stimulants, or alcohol abuse. In the examinations performed, the heart seemed healthy, and there were no problems in the electrocardiogram in terms of rhythm, number of beats, and ischemia. The patient had been fasting for more than 8 hours and had smoked before ECT.

- The patient's serum electrolyte panel was within normal limits (K: 4.1, Ca: 9.3, Mg: 1.7, Na: 137) (Table 1).

In the operating room, he was placed on the bed and monitored by measuring blood pressure, pulse oximetry, and ECG (Lead II). The patient's blood pressure was 128.73 mm Hg, heart rate was 73 beats/min, and pulse oximetry was 97%.

The ECG assessment was unremarkable for any arrhythmia. In the usual way, after performing pre-oxygenation with a flow of 10 liters/minute with a bag-valve-mask and the patient's own flow volume for 3 minutes, the propofol IV anesthetic was injected with a dose of 100 mg. Following the observation of apnea in the patient, succinylcholine 50 mg and atropine 0.7 mg were administered intravenously.

The rationale for administering atropine is based on the risk of succinylcholine-induced bradycardia. This adverse effect is particularly more common following the administration of a second dose and is also observed more frequently in pediatric patients. The incidence of bradycardia and other arrhythmias can be mitigated by pre-treatment with agents such as atropine. After making sure of the patient's conditions, standard ECT was performed with a 60 milliamper current, in the fifth session of treatment. Seizure occurred and continued for 28 seconds. In the postictal phase of the seizure, the pulse oximeter was not able to show the oxygen saturation and the heart rate. The patient's electrocardiogram (ECG) displayed asystole. The patient's radial and carotid pulses were checked immediately, but no pulse was detected. Cardiopulmonary resuscitation was started immediately with chest compressions, and airway maintenance was performed by the anesthesiologist and the anesthesiology technician. One milligram of intravenous epinephrine was injected. The patient's cardiac rhythm converted to a sinus rhythm following five minutes of chest compressions. The ECG wave appeared, and the pulse oximeter was able to show SpO₂ and the heart rate. Meanwhile, the patient's airway was secured, and he was delivered to recovery with a blood pressure of 119.66 mm Hg and a heart rate of 105 beats/min.

Following the restoration of full mentation and stable hemodynamics, the patient was admitted to the Intensive Care Unit (ICU) for continued treatment and diagnostic workup. During the patient's hospitalization in the ICU, his heart function was evaluated. Considering the ECG of 12 leads, the echocardiography, and the CTNI result being negative two times, the patient was transferred from the ICU to the psychiatric ward, and no signs of heart pathology were observed. Finally, after psychological treatments, the patient was discharged from the hospital with a good general condition.

Discussion

Electroconvulsive therapy (ECT) is a treatment method for psychological disorders. A wide range of patients with mood disorders (depression and bipolar), drug-resistant schizophrenia, and critical neurological conditions benefit from it [7]. This

Table 1. LAB TESTS

| Range | Lab tests |
|-------|-----------|
| 4.1 | K |
| 9.3 | ca |
| 1.7 | Mg |
| 137 | Na |

method, like any other treatment, has disadvantages that could threaten the patient's life. Therefore, its possible complications and management strategies will be discussed in the following. ECT is a safe induction of serial generalized seizures, using the technique of stimulation with a brief electric current under general anesthesia and muscle paralysis, for therapeutic purposes [7]. During this process, some changes occur in the autonomic systems of the body. Usually, the autonomic activity caused by electrical stimulation leads to an initial parasympathetic response, which is immediately followed by significant sympathetic activity. Due to the attenuation of vagal effect, tachycardia and a secondary increase in blood pressure are observed. In the textbooks of anesthesiology, transient bradycardia caused by primary vagal stimulation has also been reported [4]. Estimated mortality of ECT is close to 1.2 deaths per 100,000 cases. The most common side effect is cardiopulmonary complications, which occur in less than 1% of the cases. Serious and rare side effects include cardiac arrhythmias with or without changes in hemodynamics, respiratory distress, prolonged apnea, aspiration, prolonged muscle paralysis, and prolonged seizures. Mild side effects include muscle pain, nausea, and vomiting [5]. Andreas Duma et al. conducted a meta-analysis in 2019 and concluded that major adverse cardiac events and death after electroconvulsive therapy are infrequent and occur in about 1 of 50 patients and after about 1 of 200 to 500 electroconvulsive therapy treatments [8].

Anesthesia considerations during electroconvulsive therapy: securing the airway is of high importance due to the occurrence of apnea caused by the administration of anesthetic (propofol) and a nerve-muscle junction blocker (succinylcholine). Patients should have enough fasting time. Besides, the history of heart and lung diseases must be considered. In patients with underlying factors such as diabetes and high blood pressure, complete cardiac examinations must be done. For general anesthesia, the operating room should be well-equipped, and an experienced technician and an anesthesiologist should be present [9]. A case report study was conducted by Yuji Kadoi et al. in 2020; during the ECT procedure, the heart rate gradually decreased before seizure termination, and severe bradycardia (5–6 beats/min) was identified lasting 15–20 s. Atropine administration immediately before electrical stimulus prevented any further bradycardia during the next session of ECT [10].

Since performing ECT requires establishing anesthesia and maintaining the airway, the necessary facilities must be prepared in the operating room. On the other hand, considering the patient's medical

records can prevent incidents such as arrhythmia and asystole. Therefore, precise examination of patients, pre-operative preparations, increasing the dose of atropine, reducing the dose or stopping beta-blockers, and reducing the dose of succinylcholine (if possible) could be effective in this manner.

The primary limitation of this study is its nature as a case report. As the data are derived solely from a single patient, the statistical generalizability of the findings to the broader population affected by this condition is inherently restricted. Nevertheless, the aim of this report is to provide early clinical insights into a rare novel presentation, which may serve as a hypothesis-generating foundation for future studies involving larger sample sizes.

Conclusion

Electroconvulsive therapy (ECT) remains a highly effective treatment for severe psychiatric disorders. However, as with any medical procedure involving anesthesia and electrical stimulation, it carries a risk of adverse cardiac events, including severe bradyarrhythmias and asystole, as illustrated by the present case. This underscores the paramount importance of rigorous safety protocols grounded in proactive monitoring, preventive strategies, and institutional preparedness.

First, continuous and vigilant cardiac monitoring is an absolute necessity. Monitoring for bradyarrhythmias and other dysrhythmias should be standard practice before induction (to establish a baseline), during the seizure and recovery phases, and for a sufficient period after the procedure until the patient is fully hemodynamically stable. This allows for the immediate detection of vagally mediated or other arrhythmias that may precede catastrophic events.

Second, robust pre-treatment strategies are critical for risk mitigation. These include:

Adequate pharmacologic prophylaxis: Administering an appropriate dose of atropine (typically 0.01–0.02 mg/kg) based on patient weight and comorbidities to effectively antagonize parasympathetic (vagal) tone. Fixed, subtherapeutic doses should be avoided.

Patient preparation and education: Instructing patients to avoid smoking and caffeinated beverages for several hours prior to ECT, as nicotine and caffeine can increase vagal tone and cardiovascular instability. Ensuring proper hydration and managing correctable factors like electrolyte imbalances are also essential components of pre-ECT optimization.

Finally, this case reinforces the non-negotiable

requirement for structured institutional preparedness. Every ECT unit must be equipped and staffed to function as a high-acuity procedural area. This mandates:

Immediate availability of full resuscitation equipment, including a functional defibrillator/cardioverter, advanced airway supplies, and emergency medications.

Staff competency in recognizing cardiac arrest and initiating immediate, high-quality cardiopulmonary resuscitation (CPR) without delay.

Established protocols for activating advanced cardiac life support (ACLS) and rapid transfer to an intensive care setting if required.

In summary, the safe administration of ECT hinges on a multi-layered defense strategy integrating vigilant monitoring, evidence-based pharmacological prophylaxis, patient optimization, and a culture of readiness for emergencies. Adherence to these principles is essential to minimize cardiac risk and ensure that this vital therapeutic modality can be delivered with the highest possible standard of safety.

Ethical Considerations

Ethics approval and consent to participate

(SEMUMS, Ethical code: IR.SEMUMS.REC.1403.101).

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Conflict of Interests

The authors have no conflict of interest to declare.

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