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Case Report

A Rare Case of Prolonged Muscle Paralysis during Electroconvulsive Therapy in a Patient with Unknown Pseudocholinesterase Deficiency

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ABSTRACT

Inherited pseudocholinesterase deficiency is a rare condition that affects the breakdown of certain drugs, such as succinylcholine, which is commonly used during electroconvulsive therapy (ECT). We observed a case of a middle-aged woman undergoing her first ECT treatment, where the use of succinylcholine caused extended muscle paralysis. The patient needed ventilation support and sedation until the effects of succinylcholine dissipated. After that, blood investigations resulted in low Pseudocholinesterase levels and low Dibucaine numbers as well. This case emphasizes the importance of identifying and monitoring pseudocholinesterase deficiency in ECT patients and the necessity of well-equipped facilities to manage any potential complications.

Keywords: Succinylcholine; Pseudocholinesterase; ECT

Introduction

Plasma cholinesterase deficiency, also called pseudocholinesterase deficiency, is a rare inherited condition that results in reduced activity of the pseudocholinesterase enzyme. This enzyme is vital in breaking down certain medications, such as succinylcholine, commonly used as a paralytic agent during electroconvulsive therapy (ECT) procedures. If someone is deficient in this enzyme, they may experience prolonged paralysis and delayed motor recovery from succinylcholine, requiring extended ventilation and sedation until the drug's effects wear off[1]. In this article, we discuss a case of suspected pseudocholinesterase deficiency in a patient undergoing ECT and the implications it has for anesthetic management.

Case Report

A 54-year-old woman with a total body weight of 56 kilograms underwent right unilateral ECT after a routine medical evaluation that revealed a history of fibromyalgia, vertigo and supraventricular tachycardia. The anesthesia plan involved using the standard institutional protocol of general anesthesia with natural airway and manual face mask ventilation. The patient fasted for more than 8 hours prior to the procedure. In the ECT suite, standard monitors, including Electrocardiogram (ECG), Blood oxygen saturation (SpO2), Non-invasive Blood Pressure, end tidal carbon dioxide (EtCO2) and Neuromuscular twitch monitor, were applied.

The patient received pre-oxygenation with 100% oxygen and received an induction dose of methohexital (60 mg) for anesthesia. Once the patient was asleep, succinylcholine (50 mg) was administered as a paralytic agent. The neuromuscular twitch monitor was used to confirm muscle paralysis and a bite block was inserted to prevent injury. The seizure-inducing stimulus was given successfully and the bite block was removed. The seizure lasted for around 40 seconds. The patient was manually ventilated with a face mask and bag on the anesthesia machine circuit, while capnography was used to maintain adequate ventilation.

However, the patient had poor motor recovery beyond the usual recovery time for succinylcholine. The Twitch monitor confirmed complete muscle paralysis and propofol infusion was started to maintain sedation. Ventilation was assisted via continued bag and mask ventilation. The patient regained complete muscle power recovery after nearly 2 hours of succinylcholine administration. Sedation was gradually stopped and the patient woke up without any memory or recall of the incident and vital parameters remained stable. The patient was investigated and results showed low blood pseudocholinesterase levels and dibucaine number as well.

For the subsequent ECT procedures, rocuronium was used as the paralytic agent and was reversed with sugammadex. The patient tolerated these procedures well with no adverse events.

Discussion

Pseudocholinesterase deficiency is a rare genetic condition that affects the metabolism of certain drugs, most notably succinylcholine, due to reduced pseudocholinesterase activity[2]. The severity of the deficiency can vary, with different genotypes leading to milder or more severe forms of the condition[3]. In the presented case, the patient's prolonged recovery from succinylcholine-induced paralysis suggests a possible homozygous genotype for pseudocholinesterase deficiency, indicating a higher degree of enzyme activity impairment. In our case, the test results confirmed this as the blood pseudocholinesterase level was 366 (normal values range 2700 -3100 U/L) and the dibucaine number was 25 (normal > 80%).

Anesthetic management during ECT procedures relies on the administration of paralytic agents to induce temporary muscle paralysis, ensuring safety during the induced seizure[4]. Succinylcholine is preferred for its short duration of action, allowing quick recovery and resumption of spontaneous breathing. However, in patients suspected of having pseudocholinesterase deficiency, alternative paralytic agents that are not metabolized by cholinesterase, such as rocuronium, should be used[1]. The use of sugammadex, a selective relaxant-binding agent, facilitates rapid reversal of the effects of rocuronium, enhancing safety in such cases[5].

This rare case emphasizes the need for heightened awareness of pseudocholinesterase deficiency in patients undergoing ECT, particularly in those with prolonged recovery from succinylcholine-induced paralysis. Accurate diagnosis is essential for appropriate anesthetic management and prevention of potential complications. Facilities performing ECT should be adequately equipped and staffed to manage prolonged ventilation and sedation, ensuring patient safety. Timely recognition and implementation of alternative strategies can minimize risks and optimize outcomes in patients with pseudocholinesterase deficiency.

Pseudocholinesterase deficiency should be considered in patients presenting with prolonged recovery from succinylcholine-induced paralysis during ECT procedures. This case highlights the importance of prompt recognition, accurate diagnosis and appropriate anesthetic management. Facilities performing ECT should be adequately prepared to manage potential complications associated with prolonged ventilation and sedation. Further studies and genetic testing are warranted to enhance our understanding of pseudocholinesterase deficiency and its implications in anesthesia practice. The use of rocuronium or other non-depolarizing muscle relaxants in ECT holds promise by avoiding this potential complication with the use of succinylcholine.

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