

Electroconvulsive therapy and muscle relaxants

TO THE EDITOR: We read with great interest the letter to the editor, “the Electroconvulsive therapy in a catatonia patient: succinylcholine or no succinylcholine?” Neuromuscular blocking agents are important drugs for electroconvulsive therapy (ECT) to prevent possible musculoskeletal complications of ECT, such as hyperkalemia, myalgia, dislocation, and fracture. Succinylcholine is more effective because it typically has a fast onset and short duration of action. However succinylcholine may induce myalgia, hyperkalemia, and malignant hyperthermia [1].

Therefore, the anesthetic provider should preoperatively evaluate the patients’ physical status and laboratory tests, and select anesthetics with neuromuscular blocking agents [2,3]. Nicotinic acetylcholine receptors (nAChRs) on the neuromuscular junction are up- or down-regulated in neuromuscular diseases, burns, and upper or lower motor nerve injury. Upregulation of nAChRs increases sensitivity to depolarizing neuromuscular blocking agents (e.g., succinylcholine), while downregulation increases resistance to depolarizing neuromuscular blocking agents [4]. Supersensitivity to nAChR agonists (e.g., acetylcholine or succinylcholine) was observed throughout the muscle membrane. The increase in nAChRs after denervation is more profound and occurs more quickly than with simple immobilization [4].

In this case, succinylcholine was avoided because of the risk of life-threatening hyperkalemia resulting from the upregulation of nAChRs secondary to long-term immobilization, as the patient had been immobilized due to catatonia for the past 6 months. Glycopyrrolate (0.2 mg), propofol (80 mg), and atracurium (15 mg) were administered. I-gel insertion and propofol infusion were started for maintenance until recovery from the neuromuscular blockade in the first and second ECT sessions, respectively. In the third ECT session, succinylcholine was used instead of atracurium to monitor, diagnose, and manage the hyperkalemia. The intraoperative course remained uneventful with no electrocardiographic changes suggestive of hyperkalemia [1]. In this

patient, neuromuscular monitoring was not performed for the onset and recovery of neuromuscular function. Therefore, neuromuscular responses, such as increased sensitivity or resistance to depolarizing (succinylcholine) or nondepolarizing muscle relaxants (atracurium), were not examined in this patient. The patient was administered an antispastic drug (e.g., lorazepam, escitalopram, or syndrome) [1]. These antispastic drugs did not show any significant potentiation or inhibition of muscle relaxants, and the interval between the first and third ECT sessions was within 1 week in this patient. This period was not sufficient for the change from up- or downregulation to the normal range of nAChRs. The serum levels of potassium marginally increased from 4.5 to 4.75 mEq/L after succinylcholine administration at the third ECT session. Therefore, we suspected that the nAChR levels in this patient were within normal limits. Catatonia, a type of chronic immobilization, is a complex neuropsychiatric behavioral syndrome, which is not similar to the upper or lower motor neuron injury, burns, infection, polyneuropathy, or myopathy in critical illness [4]. We believe that the authors’ choice of atracurium was better than that of succinylcholine for the safety of this patient. If succinylcholine-induced adverse effects are prevented, other neuromuscular blocking agents and antagonists could be aminosteroid nondepolarizing neuromuscular blocking agents (e.g., rocuronium) and antagonists (e.g., sugammadex). The combination of rocuronium and sugammadex may lead to rapid onset and recovery in these patients, except in those with anaphylaxis and hypersensitivity [5].

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