Electroconvulsive therapy in a catatonia patient: succinylcholine or no succinylcholine?

To the Editor: We read with great interest the review by Joung et al. [1] published in Anesthesia & Pain Medicine. We wish to describe our experience with different anesthetic drugs and techniques in a patient with catatonia undergoing multiple electroconvulsive therapy (ECT) sessions.

Catatonia is characterized by a constellation of psychomotor disturbances associated with immobility, mutism, and rigidity [2]. ECT is the preferred treatment modality [2]. As patients with catatonia are prone to muscle rigidity, hyperkalemia, and deteriorating Glasgow coma scale (GCS) score, anesthesia is challenging for ECT procedures [3].

A 48-year-old female patient weighing 35 kg presented with hallucinations, dementia, and increased muscle rigidity in all four limbs for the past 6 months and was diagnosed with catatonia (secondary to Lewy bodies in the substantia nigra). As the patient was resistant to pharmacological therapy (lorazepam, escitalopram, and syndopa), ECT was finally planned. Her GCS score was E4V1M5. As routine blood investigations were normal, our main concern was serum potassium (4.45 mEq/L), which was expected to rise further with succinylcholine, a commonly used neuromuscular blocking drug (NMBD) in ECT procedures [2].

In her first ECT session, succinylcholine was avoided because of the reported risk of life-threatening hyperkalemia as a result of upregulation of nicotinic cholinergic receptors secondary to long-term immobilization (as the patient was immobilized for the past 6 months). Glycopyrrolate (0.2 mg), propofol (80 mg), and atracurium (15 mg) were administered to the patient. I-gel insertion and propofol infusion were started for maintenance until recovery from neuromuscular blockade. After the procedure, the patient recovered to her preoperative GCS score. In the second ECT session, the same anesthesia technique was used. There was no improvement in the GCS score after two ECT sessions although an improvement in muscle rigidity was observed. In the third ECT session, it was decided to use succinylcholine instead of atracurium with complete preparation for monitoring, diagnosis, and management of hyperkalemia, in case such an event occurred. The intraoperative course remained uneventful, with no electrocardiogram (ECG) changes suggestive of hyperkalemia. Serum levels of potassium marginally increased from 4.5 mEq/L to 4.75 mEq/L.

A similar anesthetic technique was followed in the fourth and fifth ECT sessions without any significant events. Muscle rigidity resolved further, and GCS score improved. She was under observation in the hospital for another 15 days and was discharged on medication. The patient is doing well on follow-up.

Hudcova and Schumann [3] reported ventricular tachycardia and ECG changes suggestive of hyperkalemia (with a rise in serum potassium level from 4.3 mEq/L to 6.4 mEq/L) after administering succinylcholine in a patient with catatonia scheduled to undergo ECT. Cooper et al. [4] reported arrhythmias and asystoles after succinylcholine administration in a patient with catatonia undergoing ECT. Zisselman and Jaffe [5] also reported ventricular tachycardia with succinylcholine administration in these patients.

The risk of arrhythmias led us to choose a non-depolarizing NMBD over succinylcholine in the initial two settings. However, due to improvement in muscle rigidity, succinylcholine was administered in the next three sessions with all preparations in place. Based on the existing literature and our experience, the use of succinylcholine or a non-depolarizing NMBD in such patients remains controversial. Our experience suggests that succinylcholine may be safe when muscle rigidity is improved. However, with the available evidence, we can conclude that it is challenging to form definitive recommendations for managing patients with catatonia undergoing ECT.

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References