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Anesthetic management of electroconvulsive therapy in a patient with a known history of neuroleptic malignant syndrome

Naoko Kato, Yusuke Asakura, Misako Mizutani, Nobuhisa Kandatsu, Yoshihiro Fujiwara, and Toru Komatsu
Department of Anesthesiology, Aichi Medical University, Nagakute, Aichi 480-1195, Japan

To the editor: Since 1938, when the use of electroconvulsive therapy (ECT) was first described in the literature, ECT has played a pivotal role in the treatment of severe mania and depression, schizophrenia, and conditions such as suicidal drive and vegetative dysregulation [1]. Since the 1950s, when succinylcholine was introduced to modify convulsions to prevent severe muscle contractions during ECT, and, therefore, to prevent serious musculoskeletal complications, succinylcholine has remained the most commonly used muscle relaxant for reducing the intense muscle contractions associated with ECT-induced seizure activity [2]. However, the use of succinylcholine may have potentially deteriorative effects on the cardiovascular system and plasma potassium levels, as well as showing adverse effects in patients with a known history of susceptibility to malignant hyperthermia (MH), and patients with a history of neuroleptic malignant syndrome (NMS) [2,3]. An alternative for succinylcholine has been sought, and the short acting non-depolarizing muscle relaxant mivacurium has been shown to be capable of attenuating muscle contractions during ECT, although with limited efficacy when compared to succinylcholine [4].

NMS is a relatively rare, but potentially fatal, complication of the use of neuroleptic drugs, with an incidence of approximately 0.07% to 2.2% among patients receiving neuroleptic agents, and with mortality rates of 15%–25%, or even higher [5,6]. The syndrome consists of the sudden onset of fever, muscle rigidity, and autonomic dysfunction, and an elevated creatine kinase (CK) level, reflecting rhabdomyolysis [5,6]. Due to the similarity between the clinical symptoms manifested by NMS and those manifested by MH, several previous reports have provoked some controversy regarding the safety of the use of succinylcholine in patients with a known history of NMS [7,8].

A 72-year-old woman was admitted to our hospital suffering from severe depression. Following treatment with haloperidol, maprotiline, and clomipramine, she developed nausea and vomiting, pyrexia of 37.2°C, labile blood pressure, urinary retention, and elevated CK, reflecting rhabdomyolysis (CK, 1145 $U \cdot l^{-1}$; normal <132 $U \cdot l^{-1}$). NMS was suspected because of her symptoms, and the medications were immediately discontinued. With the discontinuation of the medications and with supportive therapy such as hydration, symptomatic control of temperature, and acid-base and fluid balance, the clinical symptoms resolved almost completely within 10 days. With the resolution of NMS, anesthesia for ECT was requested.

The first and all the subsequent ECT procedures were performed in the post-anesthesia recovery room with equipment and drugs to treat MH immediately available. During the procedure, EEG was continuously monitored (Thymatron System IV; SOMATICS, Lake Bluff, IL, USA). Induction of anesthesia was accomplished with propofol (target concentration at 2–2.5 $\mu g \cdot ml^{-1}$ by target-controlled infusion [TCI]) and succinylcholine (60 mg), intravenously. Because an excess of propofol has anti-epileptic properties, the optimal concentration for ECT had been titrated by monitoring EEG in a couple of patients undergoing ECT. The patient was ventilated by face mask with oxygen. Then the ECT stimulus was applied, and it produced an ensuing seizure. The durations of the repetitive spikes in the EEG were continuously measured, and successful ECT was defined as that with repetitive epileptic spikes on the EEG lasting at least more than 15 s. Spontaneous respiration resumed within 5 min. The body temperature was measured on several occasions following ECT, at intervals of 3–4 h. We did not observe any changes in body temperature or serum potassium level, and consequently, MH did not result. She received ECT on four subsequent occasions without any complications, and was discharged 3 weeks after the last ECT.

Although NMS and MH may result from totally different pathophysiologies [5,6,9], the possibility that individuals susceptible to developing NMS may be vulnerable to developing MH remains to be determined, because MH and NMS have some clinical features in common. However, thus far, limited information is available regarding the safety of the use of succinylcholine in a patient with a known history of NMS [10]. We have reported here a patient with a known history of NMS who underwent ECT under anesthesia with propofol and succinylcholine without any complications, and MH did not result. The observations described here could ultimately help to clarify the as yet undetermined pathophysiology of NMS.

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Address correspondence to: Y. Asakura
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