Case Report

Rocuronium and sugammadex: An alternative to succinylcholine for electro convulsive therapy in patients with suspected neuroleptic malignant syndrome

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Abstract

We report a case of presumptive neuroleptic malignant syndrome requiring muscle relaxation for electro-convulsive therapy. short acting muscle relaxation without the use of succinylcholine was achieved using rocvronivm reversed with the novel reversal agent sugammadex. We suggest that this combination is a safe and effective alternative to succinylcholine in such cases.

Key words: Neuroleptic malignant syndrome, electro convulsive therapy, succinyl choline, rocuronium, sugammadex

Introduction

Since 1938, when the use of electro convulsive therapy (ECT) was first described in the literature, it has played a major role in psychiatric medicine.[1] Also, since its introduction in the 1950's, succinylcholine has remained the most common muscle relaxant used to modify the motor effects of ECT. A coincidental association between neuroleptic malignant syndrome (NMS) and malignant hyperthermia (MH) may render succinylcholine unsafe in some patients undergoing ECT. There is conflicting evidence for this in the literature.[2,3]

Case Report

A 69-year-old woman, of weight 50 kg, was admitted to the psychiatric service requiring anesthesia for ECT to treat severe depression. She presented with a presumptive diagnosis of NMS, Parkinson's disease, rheumatoid arthritis, celiac disease, and hypertension. Current medications included dantrolene, bromocriptine, amlodipine, thiamine, and calcium. She was found to be non-verbal, rigid, and posturing. She had an edentulous airway, with Mallampatti score of II, and had limited neck movement. Magnetic resonance imaging (MRI) showed moderate degenerative changes at C3/4 vertebrae and a normal odontoid peg. A review of other systems was unremarkable. Her heart rate was 80/min, blood pressure was 130/86 mm Hg, and temperature was 36.5°C.

All ECT procedures were performed in the operating theatre with standard monitoring. ECT was administered using Thymatron System IV (SOMATICS Lake Bluff, IL, USA), with bitemporal electrode placement. Induction of anesthesia was accomplished with propofol 80 mg and rocuronium 50 mg (1 mg/kg). The patient was hyperventilated by face mask with 100% O_2 through a vapor-free Datex-Ohmeda anesthesia machine. Three minutes after the administration of rocuronium and deep blockade confirmed with a neuromuscular monitor (Life –Tech model ms IV; Mini Stim), ECT was performed. The treatment produced a satisfactory motor and electroencephalogram (EEG) seizure. The mean EEG endpoint of seizure was 26.5 sec. Muscle relaxation with rocuronium was satisfactory in preventing violent muscle contraction. Sugammadex 800 mg (16 mg/kg) was administered after ECT approximately 5 min after the administration of rocuronium. Recovery of train of four (TOF) ratio to 0.9 was within 2 min and the time to first spontaneous breath was within 3 min from the administration of sugammadex. HR, BP, and temperature

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were measured before anesthesia induction, pre-seizure, post-seizure, and every minute for 10 min thereafter, and remained stable throughout the procedure. Rocuronium and sugammadex were employed in all subsequent ECTs and found to be an excellent and safe alternative to succinylcholine in this patient.

Discussion

Neuroleptic malignant syndrome is a relatively rare but potentially fatal complication of the use of neuroleptic drugs. [4] NMS has been associated with all dopamine blocking drugs such as antipsychotics (phenothiazines, butyrophenones, thioxanthenes, benzamides, and recent drugs such as clozapine and risperidone)[5] and antiemetics (metoclopramide, prochlorperazine, promethazine, and droperidol).[5] Abrupt withdrawal of dopaminergic drugs may also produce an NMS-like condition.

NMS pathophysiology is complex and probably involves an interplay between multiple central and systemic pathways and neurotransmitters. Dopamine blockade in the hypothalamus is believed to contribute to thermoregulatory failure, and blockade in the nigrostriatal system may contribute to muscle rigidity and hypermetabolism. The loss of dopaminergic input to the anterior cingulate–medial orbitofrontal circuit and the lateral orbitofrontal circuit likely contributes to changes in the mental status and catatonic features seen in NMS.[6] These medications in susceptible individuals block dopamine, thereby triggering NMS. NMS typically develops over a period of 24-78 hours following antipsychotic initiation, although the condition can occur at any time during treatment.

Diagnosis of NMS is based on clinical criteria. The presence of all three major or two major and four minor manifestations indicates a high probability of NMS.[7] [Table 1]

NMS is a self-limiting condition once the offending agent has been discontinued. General symptomatic treatment such as hydration, nutrition, reduction of fever, and treatment of secondary complications (hypoxia, acidosis, renal failure) are essential.[8] Specific treatments include lorazepam, dantrolene, bromocriptine, and amantadine.[8] ECT itself also appears to be rapidly effective.[9]

Muscle relaxants are often administered during ECT to prevent myalgia and more serious musculoskeletal complications.[10] Succinylcholine remains the most commonly used muscle relaxant,[11] but is not recommended in patients with a history of susceptibility to MH, NMS, catatonic schizophrenia or organophosphate poisoning.[12,13] There are clinical reports describing the use of other muscle relaxants in this high-risk group of patients.

Table 1: Clinical criteria for diagnosis of NMS[7]

Category	Manifestations
Major	Fever, rigidity, elevated creatine phosphokinase
Minor	Tachycardia, abnormal arterial pressure, tachypnea, altered consciousness, diaphoresis, leucocytosis

Mivacurium is the drug most often administered as an alternative to succinylcholine during ECT.[3] However, a dose-finding study reported that only a full intubating dose of mivacurium (0.2 mg/kg) was associated with effective muscle relaxation during ECT.[14] At least two clinical studies have demonstrated that at higher doses, it causes clinically significant histamine release and hypotension.[11,15] Atracurium has been used as an alternative, but a dose of 0.5 mg/kg required more time to achieve a satisfactory TOF ratio and recovery. [16,17] Rapacuronium at doses of 0.6-0.8 mg/kg provided effective muscle relaxation for ECT and was readily reversible with edrophonium and atropine. Frequent occurrence of bronchospasm led to its withdrawal from the market,[18,19] so it is no longer a viable alternative.

We submit that the combination of rocuronium and sugammadex offers a serious alternative to succinylcholine in patients with neuroleptic malignant syndrome for ECT.

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