

A Rare Case of Combined Rocuronium Induced Malignant Hyperthermia and Propofol-Induced Lactic Acidosis

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Abstract We report an unusual case of A 64-year-old female received rocuronium and propofol and subsequently developed malignant hyperthermia and particularly propofol-related infusion syndrome.

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1. Introduction

Malignant hyperthermia (MHS), is a rare inherited, autosomal-dominant disorder of skeletal muscle, which presents clinically as a hypermetabolic crisis. It is precipitated by certain anesthetic gases and neuromuscular blocking agents such as sevoflurane and succinylcholine [1,2]. The pathophysiology is not known at present, however, is believed to result from genetic skeletal muscle receptor defects that allow excessive myoplasmic calcium accumulation in the presence of certain anesthetic triggering agents [3,4].

On the other hand, propofol is commonly used in anesthesia and in the intensive care setting for sedation and refractory status epilepticus due to its rapid sedative effect. However, propofol has been associated with life-threatening adverse effects, particularly propofol-related infusion syndrome (PRIS). PRIS manifests with metabolic acidosis, pulmonary hypertension, myocardial failure, rhabdomyolysis, and death [5,6]. PRIS was initially thought to be more common among children, however, more cases have been reported in adult patients recently [7,8]. Herein, we describe a case of both propofol toxicity and vecuronium-induced malignant hyperthermia simultaneously.

2. Case

A 64-year-old female with restrictive lung disease on home oxygen, obstructive sleep apnea on CPAP, and

diastolic heart failure was admitted to the medicine floor for pneumonia secondary to COVID-19 then was transferred to the medical intensive care unit (ICU) for acute hypoxic respiratory failure requiring intubation. The patient completed a full course of remdesivir and dexamethasone treatment. She also went into septic shock requiring vasopressors. Of note, she was previously on cisatracurium and midazolam for paralysis and sedation, respectively. Propofol was added on day 24 and cisatracurium was switched to rocuronium on day 26.

The patient had normal acid-base status until the 29th day of her admission when she developed severe acidemia with a pH of 7.08 on ABG which was consistent with predominant metabolic acidosis with concomitant respiratory acidosis. She also had hyperkalemia of 7.1 mMol/L for which the patient received calcium gluconate, sodium bicarbonate, and regular insulin. Repeat potassium was 6.3 mMol/L, which was confirmed with a blood gas sample, and the same medical treatment was given. At the same time, she developed a febrile episode with a temperature of 38.5 C along with a slight worsening of renal function. A brisk increase in lactic acid was noted on her ABG from 1.54 mMol/L to 12.8 mMol/L within 24 hours despite being on norepinephrine and vasopressin drips with broad-spectrum antibiotics. Propofol was highly suspected as the culprit for lactic acidosis as the patient has been on high doses continuously for over 4 days. The decision was made to discontinue propofol, which resulted in a dramatic decrease of lactate level within 24 hours with subsequent normalization in her acid-base status. Also, there was suspicion of rocuronium-induced malignant hyperthermia with the elevated CPK, febrile episode, severe hyperkalemia

and worsening hyperphosphatemia, most of which resolved after discontinuation of the paralytic agent.

3. Discussion

Propofol is commonly used for sedation in the ICU because of its short duration of action and rapid clearance. However, administering higher doses of propofol can have damaging effects on a patient's condition such as propofol infusion syndrome (PRIS). PRIS, first described in children in 1990, describes a life-threatening syndrome characterized by refractory lactic acidemia, bradyarrhythmia, lipidemia, hypotension, and oliguria which usually resolves after the discontinuation of a propofol infusion [9,10]. However, the major limiting factor in almost many PRIS cases is that etiologies such as untreated sepsis could not be ruled out. The variability in the presenting features of PRIS can often make it difficult to diagnose [11]. An analysis of the published case reports suggests that a high total dose of propofol is an important factor in the development of PRIS, either through high infusion rates, prolonged duration, or both [12].

Furthermore, malignant hyperthermia is a rare disease with an estimated incidence of up to 1 in 3000 patients undergoing anesthesia procedures [13,14]. MHS is more prevalent among men, however, the mortality rate is higher in women. The overall mortality rate is greater in adults compared to children with a mortality rate of around 17% and less than 1% respectively [14]. Most patients who developed MHS have associated critical conditions, therefore, detecting the definitive cause for this adverse drug reaction might be difficult. In the reported case, other possible causes that might mimic MHS were not identified. Moreover, few reported rare cases revealed rhabdomyolysis induced by prone positioning, nevertheless, the reported case had ARDS but did not require prone positioning [15]. In addition, CPK was slightly elevated in the reported case, however, the suspicion of rhabdomyolysis was still low.

To our knowledge, there are few published cases regarding MHS adverse reaction and no clear guidelines for managing high-risk patients. Nevertheless, the early discontinuation of neuromuscular blockade and early administration of antidotes including dantrolene and bromocriptine in suspected cases of malignant hyperthermia is important.

4. Conclusion

A 64-year-old female received rocuronium and propofol and subsequently developed malignant hyperthermia and PRIS, both of which were resolved after both medications were discontinued.

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