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Case Report

General anaesthesia for laparoscopic umbilical hernia repair in a patient with pseudocholinesterase deficiency: a case report

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ABSTRACT

Pseudocholinesterase deficiency is a condition that causes prolonged muscle weakness when succinylcholine or mivacurium is used as a neuromuscular blocking agent. It can be either inherited or acquired. The practice of not using these two muscle relaxant agents may suggest that PED is not a significant problem for general anesthesia. This case report describes the successful discharge of a patient with a preexisting diagnosis of PED who underwent laparoscopic umbilical hernia repair under GA with neuromuscular monitoring without complications.

Keywords: Pseudocholinesterase deficiency, General anaesthesia, Neuromuscular block, Neuromuscular monitoring, Anaesthesia awareness

INTRODUCTION

Pseudocholinesterase is the liver's glycoprotein enzyme, also known as Butyrylcholinesterase or plasma cholinesterase. Although its exact physiological function is not fully understood, it is important in anesthesia practice due to its involvement in the metabolism of succinylcholine (Sch) and mivacurium.[1,2] While Sch is the shortest-acting muscle relaxant available, it is not widely preferred in modern anesthesia due to various risks and potential complications. On the other hand, mivacurium is a short-acting non-depolarizing muscle relaxant (NDMR) that has an onset of action for intubating dose is 2.5 to 3 minutes and a duration of the intubating dose is 15 to 20 minutes, metabolized by pseudocholinesterase and is not commonly used in current anesthesia practice.[3] Ester local anesthetics are also metabolized by pseudocholinesterase (Table 1).[1,2,4]

Table 1: List of drugs metabolized by pseudocholinesterase.

Drugs metabolized by butyrilcholineesterase
Succinylcholine
Mivacurium
Cocaine
Procaine

Butyrylcholinesterase deficiency could be inherited or acquired. Inherited forms follow an autosomal recessive inheritance pattern, and its DNA location has been identified as 3q26.1-26.20. The incidence of the homozygous form is reported as 1 in 2000-5000 individuals, while the heterozygous form is estimated to occur in 1 in 500 individuals.[1,4,5] Acquired forms of the deficiency can occur due to renal and liver failure, burns, malnutrition, malignancy, pregnancy, cardiopulmonary

bypass, and medications (steroids, anticholinergic drugs, cytotoxic drugs).[5-7] Depending on the severity of the enzyme deficiency, patients receiving Sch or mivacurium may experience residual paralysis lasting from 30 minutes to several hours. In such cases, mechanical ventilation (MV) and other supportive treatments should be implemented until muscle strength returns to normal.[4,8,9] Herein, in this case, we aim to describe the successful discharge of a patient with a preexisting diagnosis of PED who underwent laparoscopic umbilical hernia repair under GA without complications.

CASE REPORT

A 52-year-old male patient (height 168 cm, weight 74 kg) was admitted to the general surgery outpatient clinic due to abdominal swelling around the umbilicus and increasing pain for the past 3 months. An umbilical hernia was detected upon physical examination. The patient was advised to undergo surgical repair. Despite being diagnosed with hypertension, the patient did not regularly use antihypertensive medication. The medical history noted that the patient had prolonged muscle weakness following GA 26 years ago for pilonidal sinus surgery. The patient was diagnosed with PED, which led to significant anxiety. The patient was evaluated preoperatively at the anesthesia clinic, and apart from hypertension, vital signs were considered normal. In addition to hypertension and PED, it was found that the patient had a 15-pack-year history of smoking. Preoperative investigations, including radiography, showed normal findings, and the electrocardiography revealed normal sinus rhythm at 74 beats min.[1] Laboratory tests were within the normal range, including counting blood cells and renal and liver function tests. The patient's American Society of Anesthesiologists (ASA) physical classification was class II. We have obtained written and informed consent to publish this case report. Ethical approval was not obtained since this study is a retrospective case report.

The patient was admitted to the operating room after 6 hours of preoperative fasting, and standard monitoring (electrocardiography, non-invasive blood pressure, heart rate, and pulse oximetry) was performed. Fluid therapy was started with the intravenous (IV) catheter. Vital signs were stable within normal physiological ranges. GA induction was performed using 3 mg/kg propofol, 1 µg/kg fentanyl, lidocaine 1 mg/kg, and 0.6 mg/kg rocuronium. After 90 seconds of preoxygenation with a balloon mask ventilation, the patient was intubated using an 8.0 endotracheal tube. Laryngoscopy revealed Cormack-Lehane Grade IV, but no adjunctive devices were required for intubation. End-tidal carbon dioxide and lung auscultation confirmed endotracheal tube placement, and MV was initiated. Anesthetic maintenance was achieved with remifentanil infusion at 0.05-0.15 µg/kg/min and sevoflurane at 1.0 MAC. Analgesia was provided with 100 mg tramadol and 1 g paracetamol. Although the patient's blood pressure initially increased to 180/110 mmHg at the beginning of the surgery, it returned to normal ranges within the following 20 minutes and remained normotensive during the intraoperative period. We applied Train of Four (TOF) and NMT to the patient after GA induction and endotracheal intubation. The baseline TOF ratio was 0 and measurements ranged between a TOF count of 3 and a TOF ratio of 0.2. The patient underwent laparoscopic umbilical hernia repair in the supine position. After the 90-minute operation, with a TOF ratio of 0.85, 2 mg/kg sugammadex IV was administered. Spontaneous breathing was restored, and tidal volumes of 350 mL and above with a respiratory rate of 14/min, peripheral oxygen saturation of 99% on 0.5 FiO_2, and body temperature of 36.4°C were observed. The patient responded to verbal commands and was extubated. The patient, who was monitored in the recovery unit for 15 minutes and had normal vital signs and preserved muscle strength, was transferred to the general surgery ward. In the postoperative follow-up, oral intake, including solid food, was allowed starting from the 8th hour post-op. The patient, without any complications during the follow-up, was discharged on the first postoperative day.

DISCUSSION

Pseudocholinesterase deficiency (PED) is a rare genetic or acquired disorder that affects the ability to metabolize Sch, mivacurium, and ester-linked local anesthetics, which are choline esters. Most cases of PED have an absence of signs or symptoms.[1,2,4] It is first suspected after GA in which Sch or mivacurium is administered, such as a prolonged recovery from paralysis. Rocuronium, vecuronium, atracurium, and cisatracurium are NDMRs commonly used in anesthesia and intensive care practice. They exert their pharmacological effects by binding to nicotinic acetylcholine (ACh) receptors at the neuromuscular junction. They competitively inhibit ACh, leading to muscle relaxation. Acetylcholinesterase (AChE) inhibitors or sugammadex are employed to reverse the pharmacological actions of these agents. AChE inhibitors indirectly increase ACh levels at the neuromuscular junction, preventing the competitive inhibition of NDMRs. On the other hand, Sugammadex acts by encapsulating steroid-based NDMRs, preventing their active presence in the circulation and at the neuromuscular junction. Pseudocholinesterase is not involved in the metabolism of these agents.[5-8] In older periods of anesthesia practice, such as when Sch and mivacurium were commonly used, PED could be a potential drug side effect causing prolonged muscle weakness. More recently, publications have demonstrated prolonged muscle weakness and the need for MV in patients with PED due to Sch.[4,6-8]

Robles et al used Sch in induction in a 64-year-old patient diagnosed with achalasia and underwent endoscopic pneumatic dilation under GA due to dysphagia.[9] They reported no personal or family history of adverse anesthetic reactions in the patient's preoperative evaluation. The preoperative assessment revealed no significant personal or family history of adverse anesthetic reactions. They reported that they followed the patient

intubated with MV after the procedure, who could not clinically recover from the neuromuscular blockade and did not respond to TOF stimulation. After 18 hours, the patient started to breathe spontaneously, and after clinical improvement, extubated and this situation, they reported that they attributed it to PED. Zhou et al in the preoperative evaluation, a 32-year-old ASA I female patient who did not have a negative personal or family history of anesthetic drugs performed GA for laparoscopic hysteroscopy exploration and Sch in induction and then used cisatracurium and sevoflurane for intubation anesthesia maintenance and continued for 10 minutes after the operation reported that they remained unresponsive to external stimuli.[10] They reported that they applied neostigmine and atropine to the patient to reverse the neuromuscular blockade after the surgery, and 6 hours after the operation, the patient recovered from the paralysis, was extubated, and fully recovered. They reported that they attributed this situation to PEDs. Three days after surgery, they were reported that the patient was discharged from the hospital without any problem. In Robles A and Zhou W cases, they reported that they could not use NMT during the procedure, but they used TOF after the paralysis continued at the end of the operation.[9,10] We applied TOF and *NMT* monitoring to the patient.

In the case presentation by Wecksell and Koutsospyros, they described a patient who received Sch during anesthesia induction but did not exhibit evoked potentials during NMT and had no twitch response in TOF monitoring, leading to the intraoperative diagnosis of PED.[11] Such a situation, where NMT can impact the course of the operation, can increase the risk of complications and morbidity, particularly in surgeries involving vertebral stabilization and neck dissection.[8,11] We administered rocuronium as a muscle relaxant in our patient in GA induction, and after badgering with sugammadex according to TOF and NMT, the patient who responded to verbal stimuli was extubated without any problem. It has been stated that approximately 50% of patients with PED can emerge from anesthesia with residual neuromuscular block, which can be noticed by awareness of anesthesia.[6-8] Thomsen et al. recommend NMT even when using short-acting pseudocholinesterase-dependent neuromuscular blocking agents.[8] Our institution would not have changed the patient's anesthesia management and drug selection without a previous diagnosis. PEDs may require caution, particularly regarding the use of topical cocaine in nasal surgeries.[6-9]

CONCLUSION

Sch and mivacurium are not preferred neuromuscular blocking agents in current anesthesia practice. The absence of these drugs from the anesthesia regimen eliminates the clinical significance of pseudocholinesterase deficiency. The presence of PED is no longer a condition necessitating alterations or modifications in the drugs commonly used in contemporary general anesthesia and procedures requiring muscle relaxation.

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