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Case report
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Suxamethonium apnoea in a pregnant patient undergoing emergency lower segment Caesarean section under general anaesthesia

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Abstract

We report a well-described but rare occurrence of suxamethonium apnoea, also known as Phase II block. A 16-year-old Burmese primigravida at 38 weeks and 4 days' gestation was scheduled for an emergency lower segment Caesarean section due to poor progress in labour. Her antenatal course had been uneventful, and she had no significant medical history, allergies, or history of prior surgery. General anaesthesia was chosen due to her age and clinical circumstances. Rapid sequence induction was performed using intravenous propofol and suxamethonium. Muscle relaxation was subsequently maintained with atracurium. At the end of an uneventful surgery, reversal with neostigmine and atropine was administered upon observing spontaneous breathing effort. However, 30 minutes later, the patient still exhibited poor respiratory effort with persistent hypercapnia and poor motor strength. The patient was subsequently sedated with propofol and transferred to the Intensive Care Unit for ventilatory support and neuromuscular monitoring. She was extubated and transferred to the general ward the following morning.

Keywords: apnoea, Caesarean section, neuromuscular blockade, pseudocholinesterase deficiency, suxamethonium

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Introduction

Suxamethonium is a depolarizing neuromuscular blocking agent used for rapid sequence induction of general anaesthesia. Its short duration of action of 9 to 13 minutes is due to rapid metabolism by circulating plasma pseudocholinesterase (PChE).[1] Suxamethonium apnoea is a condition in which there is delayed recovery of muscle power after administration of suxamethonium due to PChE deficiency. PChE deficiency can be congenital or acquired due to conditions such as pregnancy, renal and liver disease, hypothyroidism, or concurrent use of drugs such as anticholinesterases or monoamine oxidase inhibitors.[2] The incidence of PChE deficiency is approximately 1 in 5000.[3] Plasma PChE activity in pregnancy is reduced by approximately 30% at 20 to 24 weeks' gestation and may persist up to 1 week postpartum.[4] While this reduction is rarely clinically significant on its own, it may unmask a latent congenital PChE deficiency, resulting in a dramatically prolonged neuromuscular blockade following suxamethonium administration.

In this case report, we describe a teenage primigravida who developed prolonged neuromuscular weakness after receiving a standard dose of suxamethonium during general anaesthesia for an emergency Caesarean section. This case aims to highlight the importance of considering PChE deficiency in young, otherwise healthy patients undergoing general anaesthesia, and emphasises the value of vigilant neuromuscular monitoring to guide safe extubation and recovery, especially in obstetric emergencies.

Case presentation

A 16-year-old Burmese teenager, primigravida at 38 weeks and 4 days of gestation, was referred to our hospital for suspected foetal macrosomia. She had no significant medical history, allergies, or history of prior surgeries. She also denied any family history of adverse reactions to anaesthesia or neuromuscular disorders. Her antenatal course had been uneventful, with routine follow-ups at a private clinic. Her body mass index was 21.9 kg/m^2, with a weight of 54 kg and height of 157 cm.

The patient was subsequently admitted for induction of labour due to oligohydramnios, with an amniotic fluid index of 5 cm and an estimated foetal weight of 3.0 to 3.2 kg. Labour was induced using 2 doses of intravaginal dinoprostone 3 mg, administered 6 hours apart. However, cervical dilation progressed poorly after 10 hours, with inadequate contractions and cervical dilatation of only 5 cm. A decision was made by the obstetrician to proceed with an emergency Caesarean section due to failed induction of labour.

Informed consent was obtained from the patient and her guardian following a detailed discussion of the risks and benefits of the available anaesthetic options. Given the urgency of delivery, her young age, and concerns about cooperation and tolerability during spinal anaesthesia, general anaesthesia was selected.

Following preoxygenation with 100% oxygen, rapid sequence induction was performed using intravenous (IV) propofol 100 mg and suxamethonium 100 mg. Endotracheal intubation was achieved without complications. IV atracurium 25 mg was administered approximately 5 minutes after the suxamethonium, once initial intubation and airway security were confirmed. Anaesthesia was maintained with sevoflurane in a 50:50 oxygen-nitrous oxide mixture to achieve a minimum alveolar concentration of more than 0.8. After delivery of the neonate, the mother was administered IV fentanyl 100 mcg, paracetamol 1 g, and morphine 3 mg for analgesia. Uterotonic support was provided with IV carbetocin 100 mcg. IV dexamethasone 4 mg was also given for postoperative nausea and vomiting prophylaxis. The estimated blood loss during the surgery was 300 ml.

At the end of surgery, which lasted 1 hour and 10 minutes, a bilateral transversus abdominis plane block was performed under ultrasound guidance with ropivacaine 0.375%, 15 mL on each side. Sevoflurane was discontinued, and non-depolarising neuromuscular blockade reversal with IV neostigmine 2.5 mg and atropine 1 mg was administered upon observing spontaneous breathing efforts. However, 30 minutes later, the patient still exhibited poor respiratory effort, with tidal volumes of 60 to 100 mL and persistent hypercapnia with $ETCO_2$ of 51 mmHg. Neurological examination revealed reactive pupils (2 mm bilaterally). Motor strength was assessed by observing spontaneous limb movement, ability to lift head, and response to verbal commands such as hand-squeezing and purposeful lower limb movements. Clinically, strength was graded as 2/5 in all four limbs using the Medical Research Council scale.

Given the unexpected, prolonged weakness and delayed emergence, neuromuscular monitoring using a peripheral nerve stimulator (PNS) was initiated in the operating room following administration of reversal agents. This reflects common practice in our setting, where PNS is generally utilised postoperatively unless intraoperative concerns arise. Train- of-four (TOF) monitoring revealed 4 twitches with a TOF ratio of 1.0; however, the absolute twitch height was notably reduced (Fig. 1a). These findings were suggestive of a prolonged depolarising neuromuscular block, despite apparent recovery from atracurium.

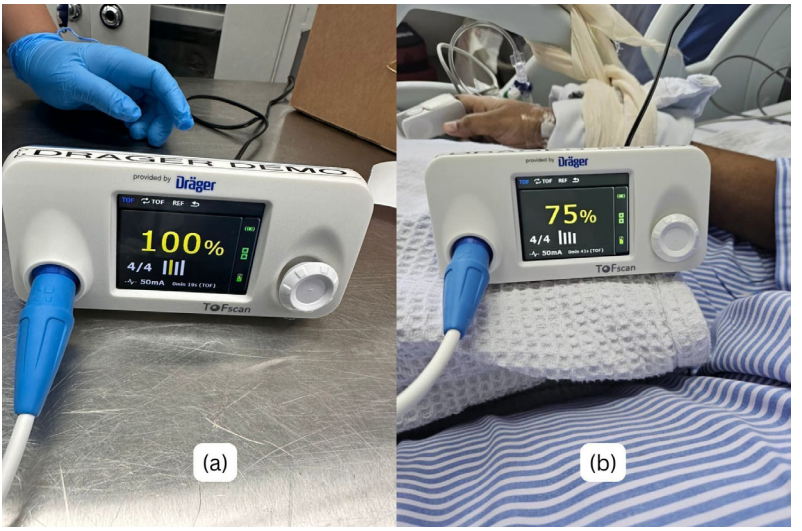

Fig. 1 (a) TOF count of 4 with a TOF ratio of 1.0 at 1-hour post-reversal of non- depolarising neuromuscular blockade. *(b)* TOF count of 4 with a TOF ratio of 0.75 at 4-hour post-suxamethonium administration.

An arterial blood gas analysis performed within an hour postoperatively revealed metabolic acidosis (pH 7.25, pO_2 351 mmHg, pCO_2 37 mmHg, HCO_3 -16 mmol/l, base excess -6 mmol/l). However, the degree of acidosis was not severe enough to explain the profound muscle weakness.

Considering the prolonged weakness and TOF findings indicative of a depolarising block, suxamethonium apnoea was strongly suspected. The patient remained in the operating theatre for approximately 1 hour under close observation, during which sedation with a propofol infusion was maintained. She was subsequently transferred to the ICU for continued mechanical ventilation, sedation, and close monitoring.

In the ICU, TOF monitoring was performed at 30-minute intervals. Sedation was gradually tapered as a Phase II block became more likely, with TOF trends showing progressive improvement. Routine postoperative investigations, including serum electrolytes—magnesium (0.91 mmol/l), potassium (4.1 mmol/l), and sodium (136 mmol/l)—were all within normal limits. A plain computerised tomography of the brain was also unremarkable, effectively excluding intracranial pathology as a cause of delayed recovery.

Approximately 4 hours after suxamethonium was administered, TOF monitoring showed a count of 4 with a ratio of 0.75 (Fig. 1b), which improved to 0.95 over the

Fig. 2. Anaesthetic alert card for patient.

following hour. With these reassuring findings and concurrent improvement in respiratory effort and tidal volumes, the patient was successfully extubated around 5 hours after the initial dose of suxamethonium. She remained stable overnight in the ICU and was transferred to the general ward the following morning after an uneventful stay.

A PChE sent on postoperative day 2 revealed a severely deficient level of < 1000 U/L. Unfortunately, she could not be further investigated as the dibucaine number test is not offered in Malaysia, and the cost of outsourcing to the United States was beyond the patient's financial means.

The patient was issued a laminated alert card detailing her diagnosis (Fig. 2), advising avoidance of suxamethonium and mivacurium in future procedures, and recommending the use of regional anaesthesia or non-depolarising agents when appropriate. She was also advised to inform first-degree relatives of the potential hereditary nature of the condition and the importance of screening prior to undergoing anaesthesia.

Discussion

Inherited PChE deficiency is caused by abnormalities in the BCHE gene, which encodes the PChE enzyme located on chromosome 3. The most common variants include the atypical, K-variant, fluoride-resistant, and silent types. In addition to inherited forms, several patient- and drug-related factors can contribute to an acquired PChE deficiency, as summarised in Table 1.[5]

Table 1. Factors causing acquired PChE deficiency

Disease states	Drugs
Hypothyroidism Kidney disease Liver disease Major burns Malignancy Malnutrition Pregnancy Prolonged infections	Cyclophosphamide glucocorticoids Metoclopramide Monoamine oxidase inhibitors Neostigmine Oral contraceptives Pancuronium terbutaline

The duration of action of suxamethonium depends on the individual's PChE activity and genotype. In homozygous atypical individuals (E1aE1a), a single dose may result in paralysis lasting 2 hours or more. In those with the silent type (E1s), recovery may be delayed for up to 3 hours. Heterozygous individuals typically recover within approximately 30 minutes. In contrast, acquired PChE deficiency usually prolongs suxamethonium action only by a few minutes.[6]

Suxamethonium typically induces a Phase I block, characterised by rapid onset, short duration, and no fade on TOF stimulation. However, with prolonged exposure or in patients with PChE deficiency, a Phase II block may develop. This mimics a non-depolarising neuromuscular blockade and is associated with fade on TOF stimulation, reduced twitch height, post-tetanic facilitation, and potential resistance to neostigmine reversal.[2,3]

In our patient, initial TOF monitoring after administration of reversal agents showed a count of 4 with a TOF ratio of 1.0, but with significantly diminished twitch height. Subsequent readings demonstrated a transient drop in TOF ratio to 0.75 before gradually increasing to 0.95 over the next hour. This evolving pattern, combined with the persistently reduced twitch response, was suggestive of a transition from Phase I to Phase II block—a phenomenon described in individuals with significant PChE deficiency, even after a single dose of suxamethonium.[7]

During pregnancy, PChE activity can decrease by up to 30%, a physiological change that may unmask an underlying congenital deficiency.[4] In this case, the patient's young age, lack of prior anaesthetic exposure, and prolonged weakness following a standard dose of suxamethonium raise suspicion of a homozygous silent genotype (E1sE1s). Although confirmatory dibucaine number testing was unavailable, the markedly low PChE level (< 1000 U/L) and absence of organophosphate exposure support a diagnosis of inherited PChE deficiency. The prolonged recovery time of approximately 6 hours further aligns with the clinical phenotype of

suxamethonium apnoea due to a homozygous silent genotype.

Routine intraoperative neuromuscular monitoring is strongly recommended by international anaesthesia societies, including the Association of Anaesthetists of Great Britain and Ireland and American Society of Anesthesiologists, especially when neuromuscular blocking agents are used. In our setting, PNS monitoring was only initiated after delayed emergence was noted, which reflects common practice in many centres. Earlier intraoperative use of TOF monitoring might facilitate earlier recognition of abnormal neuromuscular responses, particularly in high-risk groups such as obstetric patients, those with no prior anaesthetic history, or individuals with genetic predispositions. However, it is important to note that TOF monitoring alone is not fully reliable—TOF ratios of 1.0 can coexist with clinical weakness and reduced twitch height in cases of Phase I block. Thus, clinical assessment remains critical in guiding management.

The postoperative management strategy in this case was supportive: sedation and mechanical ventilation until spontaneous recovery of neuromuscular function occurred. This remains the safest approach in managing suxamethonium apnoea. Although exogenous sources of PChE, such as fresh frozen plasma transfusion, are available, the risks associated with transfusion were deemed to outweigh the potential benefits in this case. Notably, stored plasma retains 87% of initial PChE activity after 21 days at 4°C, without loss of enzymatic function.[8]

Experimental treatments, including plant-derived recombinant PChE[9] and large-scale purification of PChE from serum,[10] are currently under development. However, due to limited availability and insufficient supporting evidence, these are not yet recommended for routine use in the management of suxamethonium apnoea.

Conclusion

Suxamethonium apnoea is a rare but potentially life-threatening condition, especially if unrecognised. It highlights the importance of safe emergence and extubation practices to prevent harmful adverse events. A key aspect of safe practice is ensuring spontaneous recovery from suxamethonium before administering non-depolarising muscle relaxants, which helps mitigate the risk of further complications in cases of delayed neuromuscular recovery. The hallmark of treatment is supportive, with precautionary measures such as a warning card in addition to patient education, family screening, and genetic counselling to help prevent future episodes from reoccurring.

Declarations

Informed consent for publication

Informed consent was obtained from the patient for the inclusion of the clinical data and images contained in this case report. The informed consent for publication form has been completed, signed, and submitted along with the manuscript.

Competing interests

None to declare.

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