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Case Report

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# Suxamethonium- Induced Prolonged Apnea: Insights from a Clinical Case

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## ABSTRACT

Suxamethonium, or succinylcholine, is a depolarizing neuromuscular blocker used for rapid sequence intubation in anesthesia. It acts quickly, causing temporary paralysis, but in some individuals, a deficiency or abnormality in the enzyme pseudocholinesterase (also called butyrylcholinesterase) can result in prolonged paralysis, a condition known as suxamethonium apnea.

Pseudocholinesterase deficiency can be inherited or acquired, and patients with this condition cannot efficiently metabolize suxamethonium, leading to delayed recovery from muscle paralysis. The clinical presentation typically includes prolonged apnea and muscle weakness long after the expected recovery time, often requiring mechanical ventilation until the effects of the drug wear off.

**Keywords:** Suxamethonium Apnea, Pseudocholinesterase Deficiency, Butyrylcholinesterase (BCHE), BCHE Gene Mutation, Neuromuscular Blockade, Genetic Variability, Dibucaine Number, Fresh Frozen Plasma (FFP), Neuromuscular Monitoring, Anesthesia Complications, Genetic Testing.

## GENETIC VARIABILITY

The genetic variability of pseudocholinesterase (BCHE) activity, which affects suxamethonium as well as mivacurium metabolism, is due to different mutations in the BCHE gene [1-3]. The frequencies of different variants of this gene vary across populations. Here's an estimate of the percentages of variability seen in people with respect to suxamethonium sensitivity:

1. Normal Homozygous Individuals (Normal Metabolism):

- About 96% of the population has normal pseudocholinesterase activity and experiences typical muscle relaxation and recovery after suxamethonium administration.

2. Heterozygous Individuals (Mild Deficiency):

- Around 3-4% of the population carries one normal allele and

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one abnormal allele for pseudocholinesterase. These individuals may experience slightly prolonged paralysis after suxamethonium administration, but it is usually not clinically significant [4].

3. Homozygous Atypical Individuals (Severe Deficiency):

- Approximately 1 in 2,500 to 1 in 3,000 individuals (or about 0.03-0.04% of the population) are homozygous for an abnormal pseudocholinesterase gene, leading to severe enzyme deficiency and prolonged paralysis (lasting hours) after suxamethonium administration [5].

Genetic Variants:

There are different mutations of the BCHE gene, and their distribution may vary by ethnicity:

- Atypical Variant (A allele): This variant is common in Caucasian populations, with a carrier frequency of around 4-5%

- Kalow Variant (K allele): Seen in about 1-2% of certain populations.

- Other rare variants: Some populations have other rare mutations that may affect pseudocholinesterase activity, such as the fluoride-resistant (F) 0.03-0.04 % and silent (S) variants 0.02-0.04 % in general population [6].

These percentages highlight the broad range of genetic variability in the population that can affect an individual's response to suxamethonium.

## DIAGNOSTIC INVESTIGATIONS

When suxamethonium apnea is suspected, the following laboratory and diagnostic tests can help confirm the diagnosis:

1. Pseudocholinesterase Activity Test:

- This test measures the activity level of pseudocholinesterase in the blood. A decreased level of this enzyme indicates a deficiency and confirms the diagnosis of suxamethonium apnea.

- Normal levels: 3,200–6,500 U/L.

- Decreased levels: Usually < 3,200 U/L in cases of deficiency.

2. Dibucaine Number:

- The dibucaine number indicates how well the enzyme pseudocholinesterase is functioning. Dibucaine is a local anesthetic that inhibits normal pseudocholinesterase, but it does not affect abnormal forms of the enzyme.

- A normal dibucaine number is typically between 80-85. A lower number (20-30) suggests an atypical or abnormal pseudocholinesterase variant, leading to prolonged paralysis [7].

3. Genetic Testing:

- In cases of inherited pseudocholinesterase deficiency, genetic testing can identify mutations in the BCHE gene responsible for producing pseudocholinesterase. This test is important for identifying family members at risk for the condition [8].

4. Nerve Stimulation Test:

- During recovery from anesthesia, a peripheral nerve stimulator can assess the return of neuromuscular function. In patients with suxamethonium apnea, there is minimal or absent response to nerve stimulation due to the persistent neuromuscular blockade.

## TREATMENT OF SUXAMETHONIUM APNEA

The management of suxamethonium apnea focuses on supportive care until muscle function returns. Since there is no specific antidote for suxamethonium, treatment is aimed at maintaining respiratory and cardiovascular stability during the period of paralysis. Key treatment strategies include:

1. Mechanical Ventilation:

- Patients with suxamethonium apnea may require mechanical ventilation due to prolonged paralysis and apnea. Mechanical ventilation should continue until the patient's spontaneous breathing returns [9].

2. Fresh Frozen Plasma (FFP):

- FFP contains pseudocholinesterase and can be administered to accelerate the breakdown of suxamethonium. In this case, the child received 250 ml of FFP, which led to a significant improvement in muscle strength and recovery [9].

- Dose: 10-20 ml/kg, depending on the severity of the enzyme deficiency and clinical condition.

3. Commercial Serumcholinesterase [10].

Termination with commercial serum cholinesterase involves administering exogenous (commercially produced) human serum cholinesterase as a treatment to reverse suxamethonium-induced prolonged paralysis.

Mechanism of Action:

1) Suxamethonium Breakdown: Commercial serum cholinesterase is administered intravenously to provide functional enzyme that can break down suxamethonium in

the bloodstream.

2) Restoration of Muscle Function: By accelerating the metabolism of suxamethonium, the exogenous cholinesterase can terminate the prolonged apnea, allowing the patient's muscle function, including respiratory muscles, to recover more quickly.

3) Shortened Paralysis Duration: This approach shortens the duration of paralysis and reduces the risk of complications associated with prolonged mechanical ventilation.

Commercial serum cholinesterase is not often available, and until the effects of suxamethonium wear off, supportive care including mechanical ventilation is typically utilized. However, the injection of exogenous cholinesterase can be life-saving in emergency instances where prolonged paralysis offers additional hazards.

Supporting Care:

- If commercial cholinesterase is unavailable, patients are typically supported with mechanical ventilation until spontaneous breathing resumes, which may take several hours.

- The use of neuromuscular monitoring (train of four ratio) is crucial in identifying and managing prolonged paralysis due to pseudocholinesterase deficiency.

4. General Supportive Care:

- Continuous monitoring of vital signs, particularly respiratory function, is essential. Blood gas analysis should be performed to assess for hypoxia or hypercapnia, and ventilation should be adjusted accordingly.

- In cases where mechanical ventilation is necessary for an extended period, transfer to an intensive care unit (ICU) is advised. It is crucial to emphasize the importance of using sedation with short-acting medication such as propofol to prevent awareness, which is very common in these situations. Awareness can occur because anesthetists often focus on monitoring for any motor or respiratory activity as a sign of patient recovery.

5. Reversal of Co-administered Sedatives/Opioids:

- Opioid reversal with naloxone may be considered if there is a suspicion of opioid-induced respiratory depression, although this may not significantly affect suxamethonium-induced apnea.

6. Monitoring and Follow-up:

- Following recovery from the acute episode, patients should be advised to avoid suxamethonium in future anesthetics. It is often recommended that they wear a medical alert bracelet or carry a card indicating pseudocholinesterase deficiency. Family members should also be informed of the condition to help prevent its occurrence in them, as pseudocholinesterase deficiency can be hereditary.

## CASE REPORT

### Patient Background

A 12-year-old male child, weighing 51 kg, presented for cystoscopy and ureteric JJ stenting. He was classified as ASA III due to congenital kidney and urinary tract anomalies (horseshoe kidney) and a history of multi-organ injury following a motor vehicle accident in 2020. The child required pediatric intensive care unit (PICU) admission at that time.

### Preoperative and Intraoperative Course

The child was cleared for general anesthesia as a Class III emergency case. His preoperative blood results were acceptable range. He also has a history of multiple general anesthetic procedures in the past but has never received suxamethonium.

After preoxygenation, anesthesia was induced with propofol 120 mg and fentanyl 75 mcg. During induction, the patient developed laryngospasm, which made mask ventilation difficult and led to a drop in SpO2 to 70%. The patient received 40 mg IV suxamethonium, which resolved the bronchospasm promptly, restoring SpO2 to 99%. Ventilation was managed with a size 3 LMA during surgery, and the patient's vitals stabilized for the rest of the procedure.

### Postoperative Complication: Prolonged Apnea

Postoperatively, the child's recovery from anesthesia was notably delayed, lasting up to 2.5 hours, with no spontaneous respiratory effort observed. Naloxone was administered to reverse the effects of fentanyl, but respiratory effort remained absent. Neuromuscular monitoring revealed a profound blockade, indicating prolonged suxamethonium-induced paralysis. Arterial blood gas results were normal.

The patient remained in the operating room for nearly two and a half hours before spontaneous breathing resumed. The LMA was removed once an appropriate tidal volume was achieved through spontaneous breathing. On room air, his SpO2 levels remained between 97% and 99%. However, motor weakness persisted, with the child unable to lift his lower limbs and only making slight upper limb movements. Therefore, to rule out a stroke, a pediatric neurology

consultation was arranged in the recovery room, and a CT scan was recommended. The CT scan results were normal.

**Diagnosis and Treatment**

Given the delayed recovery, pseudocholinesterase deficiency leading to suxamethonium apnea was suspected. The child was transferred to the recovery room for close monitoring. The child was then treated with 250 ml of fresh frozen plasma (FFP), which led to a rapid improvement in muscle strength and resolution of weakness.

**Outcome**

The patient was discharged to the ward and subsequently discharged from the hospital the next day without further complications.

## CONCLUSION

This case highlights the rare but significant complication of prolonged apnea following the administration of suxamethonium, likely due to pseudocholinesterase deficiency or an atypical response to the drug. Although suxamethonium is commonly used for rapid muscle relaxation in pediatric anesthesia, its potential for causing extended paralysis underscores the importance of neuromuscular monitoring and vigilant postoperative care. In cases like this, an alternative muscle relaxant such as rocuronium may be a better option for managing bronchospasm, as it avoids the risk of prolonged paralysis and is easily available. Early recognition of suxamethonium-induced apnea and appropriate management, including mechanical ventilation, the use of commercial cholinesterase, and administration of fresh frozen plasma (FFP), is crucial. This case emphasizes the need for thorough preoperative assessment and careful selection of muscle relaxants for future procedures.

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