



Case Report

Suxamethonium-Induced Prolonged Apnoea: A Clinical Case Study

Md. Fakhurul Azam¹, S.M. Tauhidur Rahman², Debashis Chakraborty³, Mohammad Faysal Howlader⁴, Md. Nazmul Hasan⁵

Abstract

Suxamethonium (succinylcholine) is a depolarizing neuromuscular blocking agent widely used for rapid induction of anaesthesia due to its fast onset and short duration of action. However, in rare cases, its impaired metabolism may lead to prolonged apnoea, often due to pseudocholinesterase deficiency or other metabolic factors. We report a case of a 13-year-old male patient who developed unexpectedly prolonged apnea after receiving suxamethonium during appendicectomy operation. This case highlights the importance of recognizing pseudocholinesterase deficiency in patients with prolonged neuromuscular blockade after suxamethonium. Factors such as genetic variants, liver dysfunction, or exposure to cholinesterase inhibitors may contribute to this adverse effect. Early recognition and supportive management are crucial to preventing complications. Routine preoperative screening may not be practical, but a high index of suspicion is necessary in at-risk individuals or in cases of unexplained delayed recovery.

Keywords: Suxamethonium, Prolonged apnea, Anaesthesia complications.

Received: March 22, 2025; **Accepted:** May 25, 2025

doi <https://doi.org/10.3329/emcj.v10i2.85713>



Introduction

Suxamethonium chloride, also known as succinylcholine, is a depolarizing neuromuscular blocking agent that has a structure similar to that of two molecules of acetylcholine¹. The depolarizing muscle relaxant drug succinylcholine was introduced in the United States in 1952 and has been widely used in clinical medicine since the late 1950². The neuromuscular block begins quickly, within one minute, and lasts for a short period, approximately seven to eight minutes. Because of these characteristics, succinylcholine is used to facilitate rapid tracheal intubation during the induction of anesthesia, which is essential if aspiration of gastric contents is to be avoided. The drug is metabolized by plasma cholinesterase, which is synthesized in the liver¹.

Succinylcholine blocks acetylcholine, disrupting cholinergic receptors in both the parasympathetic and sympathetic nervous systems, which leads to skeletal muscle relaxation. This action supports quick endotracheal intubation, surgical interventions, and mechanical ventilation³. Suxamethonium may occasionally cause severe adverse effects, including hyperkalaemia, malignant hyperthermia, and prolonged apnoea³⁻⁵. One of the most clinically significant side effects of suxamethonium is prolonged apnoea⁶. Prolonged

apnoea can result from factors such as pseudocholinesterase deficiency, advanced age, compromised liver or kidney function, and interactions with certain medications⁵. Prolonged apnoea can lead to serious consequences if not promptly recognized and managed, including hypoxia, brain injury, and death⁷.

This case study describes a clinical scenario of suxamethonium-induced prolonged apnoea, emphasizing the diagnostic challenges, genetic implications, and management approaches related to this rare yet potentially life-threatening adverse reaction.

Case presentation

A 13-year-old male child hailing from Nimsar came to outpatient's department of otolaryngology at Eastern Medical College Hospital with the complains of difficulty of swallowing for 3 days and fever for last 4 days. He gave the history of the same problems 5 to 6 times in the last 3 months. On examination, the patient's tonsils were found to be red, inflamed and swollen, and his body temperature was recorded at 101°F. The patient was diagnosed as a case of acute tonsillitis. He was prescribed oral antibiotics (Capsule Cefuroxime) and antipyretics for 7 days, with follow-up scheduled after one week.

¹Assistant Professor, Department of Anaesthesiology, Eastern Medical College & Hospital, Cumilla, Bangladesh.

²Professor, Department of Anaesthesiology, Eastern Medical College & Hospital, Cumilla, Bangladesh.

³Assistant Professor, Department of Paediatrics Surgery, Eastern Medical College & Hospital, Cumilla, Bangladesh.

⁴Assistant Professor, Department of Anaesthesiology, Eastern Medical College & Hospital, Cumilla, Bangladesh.

⁵Assistant Professor, Department of Anaesthesiology, Eastern Medical College & Hospital, Cumilla, Bangladesh.

Address of Correspondence: Dr. Md. Fakhurul Azam, Assistant Professor, Department of Anaesthesiology, Eastern Medical College and Hospital, Cumilla, Bangladesh. Mobile: +8801675667993; Email: fakhurulazam.shohag@gmail.com

After 7 days he was planned for tonsillectomy under general anaesthesia. All necessary investigations were suggested to assess the patient's fitness for general anaesthesia.

Laboratory investigations: In CBC report there was mild leukocytosis. All other parameters were found to be within normal limits. RBS, Serum creatinine, serum bilirubin, SGPT, BT and CT were within normal range. The chest X-ray and routine examination of urine was also normal. Blood grouping was also performed.

Diagnosis and Management: On operative morning anaesthesia was induced with Thiopental sodium and suxamethonium to facilitate endotracheal intubation. The intubation was uneventful, and surgery proceeded without complications. But there was no improvement in patients after 30 minutes. Neuromuscular stimulation was done, and he was diagnosed as suxamethonium apnoeas. The patient remained hemodynamic stability throughout. Given the unexpected duration of paralysis, pseudocholinesterase deficiency was suspected. Plasma cholinesterase testing could not be performed at this hospital due to limited resources. One unit of fresh blood was immediately arranged and infused without delay. Spontaneous movement and respiratory effort were observed approximately 3 hours after surgery. The patient's condition remained stable, and no additional complications were observed.

Discussion

Pseudocholinesterase (PChE) deficiency is an enzymatic disorder resulting from mutations in the PCHE gene, which is located on chromosome 3. As a result, the body is unable to efficiently metabolize certain drugs, such as succinylcholine. This deficiency is inherited in an autosomal recessive manner, requiring mutations in both copies of the gene in each cell for the condition to manifest. This deficiency occurs 1 one in every 3200-5000 people. Patients with pseudocholinesterase deficiency show varying responses based on genetic status. Heterozygous individuals typically experience mild prolongation of muscle paralysis (30–60 minutes), while homozygous patients may have significantly longer blockade lasting 4 to 8 hours⁸. Deficiency can be acquired due to conditions like liver or kidney disease, malnutrition, cancer, collagen disorders, hypothyroidism, pregnancy, or certain medications, or it can be congenital and inherited in an autosomal recessive pattern⁹.

But our patient did not have any of the above as proved from preoperative investigations. Hence, our case may have some genetic consideration. Case reports published in 2018 have shown that patients with suxamethonium-induced prolonged apnoea

may need post-procedural ventilation for as long as 18 hours. In that report, the patient's age (64 years) and a history of previous prolonged apnoea were likely contributing factors to the extended recovery time¹⁰. In contrast, our younger patient may have had higher pseudocholinesterase activity, leading to a quicker recovery. The management of prolonged paralysis resulting from PChE deficiency is mainly supportive and conservative. Patients are maintained with mechanical ventilation under sedation while the effect of the paralytic is cleared¹¹. The use of pharmacological reversal agents, such as anticholinesterases and neostigmine, are avoided as they could lead to worsening of the condition⁶.

Theoretically, if urgent reversal of paralysis becomes necessary, for example to assess neurologic function, fresh frozen plasma (FFP) or packed red blood cells can be utilized to provide exogenous PChE¹². Fresh whole Blood is rich in cholinesterase enzyme as the half-life of cholinesterase is short & hence stored blood may not have much cholinesterase to donate. But, since now a days, blood bank seldom keeps whole blood, fresh frozen plasma is a good alternative. It is roughly estimated that one unit of FFP raises levels of plasma cholinesterase by 600-800 IU/L¹³.

When suxamethonium-induced prolonged apnoea is observed in clinical practice, it is important to inform the patient's family and recommend appropriate testing to assess any potential genetic predisposition. This discussion can be challenging because patients and their relatives often show no symptoms and may find the condition difficult to understand. A safer alternative is rocuronium, which has become a more viable option. As a nondepolarizing neuromuscular blocker, its effectiveness is not affected by pseudocholinesterase deficiency^{14,15}.

Conclusion

Suxamethonium-induced prolonged apnoea is a rare but serious complication that requires prompt recognition and appropriate management. Genetic factors such as pseudocholinesterase deficiency play a significant role in the delayed recovery observed in affected patients. Awareness of this condition, timely diagnosis, and clear communication with the patient's family about the possibility of genetic testing are crucial for effective management and prevention of future occurrences.

Conflict of interest

The authors declared that they have no conflicts of interest.

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Citation of this article

Azam MF, Rahman SMT, Chakraborty D, Howlader MF, Hasan MN. Suxamethonium-Induced Prolonged Apnoea: A Clinical Case Study. *Eastern Med Coll J.* 2025; 10 (2): 143-5.
 doi: <https://doi.org/10.3329/emcj.v10i2.85713>