



BMH Med. J. 2025;12(4):71-74. **Case Report**

A case of succinylcholine apnoea - a forgotten episode in current practice

Abdusalam PP¹, Fegis CT¹, Anjali T¹, Saji Kuriakose¹, Rajesh MC¹, Shafri Dani¹, Reshmi Aravindakshan K²

¹Department of Anaesthesia, BMH, Kozhikode, Kerala, India

²Department of ENT, BMH, Kozhikode, Kerala, India

Address for Correspondence: Dr. Rajesh MC, Senior Consultant and Academic Co-ordinator, Department of Anesthesia, Pain and Perioperative Medicine, BMH, Kozhikode- 673017, Kerala, India. E-Mail: rithraj2@yahoo.co.in

Abstract

Apnoea and prolonged paralysis after succinylcholine administration is not an uncommon occurrence in anaesthetic practice. It occurs due to inherited or acquired deficiency of butyrylcholinesterase. Suxamethonium (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.

Suxamethonium is used to achieve quality relaxation for the orotracheal intubation. It is considered as a safe drug but can rarely lead to serious adverse effects, such as hyperkalaemia, malignant hyperthermia, and prolonged apnoea. Prolonged apnoea may occur due to pseudocholinesterase deficiency, old age, impaired liver or renal function and medication interactions.

Keywords: Succinylcholine, pseudo choline esterase deficiency, apnea, anesthesia

Introduction

Prolonged paralysis and apnea after succinylcholine administration is a well-recognized event in anesthetic practice, often attributed to either an inherited or acquired deficiency of butyrylcholinesterase [1, 2]. Sensitivity to suxamethonium is a well-known risk factor, and pre-procedural assessment for sensitivities is crucial in safety protocols.

Case report

A middle aged male was admitted with complaints of hoarseness of voice. No voice fatigues. No voice breaks. No known drug allergies. He is a known case of diabetes mellitus and dyslipidemia on oral hypoglycemic agents and statins.

On examination, he was conscious, oriented, moderately built and nourished. His vitals were stable. Other systemic examinations were within normal limits. ENT examination showed good oral resonance, larynx moves up on phonation. There is hoarseness of voice. On neck bilateral thyrohyoid intervals are tender and narrow. Oral cavity and oropharynx normal. On videolaryngoscopy (VLS) - Bilobed polyp on the middle 3rd of right vocal cord, anterior phonatory gap present.

Lab investigation showed a Hb of 15.3 g/dl, platelet count of 2.51 lakh/mm³ with normal coagulation parameters. Liver and renal function tests were within normal limits. The electrocardiogram showed a normal sinus rhythm and echo showed mild mitral regurgitation, there is no regional wall motion abnormality, normal left ventricular function with ejection fraction 62%. Airway examination showed a Mallampatti grade of 3.

He was admitted under ENT department and was posted for elective micro laryngeal surgery plus biopsy under general anaesthesia. In the past history he has undergone forearm surgery under GA. He said that surgery was uneventful. On repeated questioning, bystander said that some delay was there after previous surgery for shifting back from OT. This history was given by bystander after the surgery when scoline apnoea had already been noted.

He was premedicated with midazolam 1mg, pantoprazole 40mg, dexamethasone 16mg, glycopyrrolate 0.2mg, metoclopramide 10mg intravenously. In operation theatre, all standard ASA monitors including 5 lead ECG with ST segment monitoring, pulse oximetry and noninvasive blood pressure were attached. Room air saturation was 98%. Preoxygenation with 100% oxygen was initiated with Hudson mask at 6L/minute for 3 minutes. Warm saline was started on a 18G cannula on the left forearm. Then fentanyl 100 mcg was given IV. Then he was precurarized with atracurium 5mg IV and induced with propofol 160mg IV, and succinyl choline 100 mg was given for muscle relaxation. After 1.5 minutes, he was intubated with a 6 size MLS tube and fixed at 22 cm. The cuff was inflated to pressure of 25 cm of H₂O and bolus atracurium 40 mg was given. A suction catheter of size 14 G was inserted to deflate the stomach. The patient was mechanically ventilated with pressure control volume guaranteed mode using closed circuit and end tidal carbon dioxide was monitored. The ventilatory settings were adjusted as tidal volume 625 ml, RR-15, PEEP-5cm H₂O, FIO₂-50%. Maintenance was carried out using oxygen, air, desflurane 6%, and bolus doses of atracurium ensuring adequate depth.

Under GA, patient was positioned and direct laryngoscopy was done. Endoscopic resection of right vocal cord polyp excised in toto, after infiltration with lignocaine and adrenaline. Reinke's space was reconstructed. Internal and external laryngeal block given. The surgical procedure was uneventful.

Post-extubation, the patient had a sudden breathing difficulty and he developed suspected laryngospasm. Positive pressure ventilation was continued but spontaneous breathing didn't return. I-gel was inserted, and assisted ventilation continued. Neuromuscular monitoring showed a prolonged paralysis. Spontaneous respiration returned after 4 hours and once adequate power and recovery from muscle relaxants as per neuromuscular monitoring, he was extubated. Subsequently we got the laboratory investigations which showed low plasma cholinesterase activity. He remained haemodynamically stable throughout and was discharged in good condition the next day.

Discussion

Management of Scoline Apnea

Immediate Management

1. Supportive care

The patient is kept unconscious with anaesthetic agents and placed on mechanical ventilation until muscle function returns.

2. Neuromuscular Monitoring

A nerve stimulator (Train of Four) is used to assess recovery. Full return of muscle strength is confirmed by four strong twitches without fade.

3. Allow Natural Recovery

No reversal agents are used. Recovery is monitored until the patient can breathe independently, follow commands, grip firmly, and lift their head.

4. Fresh Frozen Plasma (FFP)

In severe cases, FFP can be given to provide the missing enzyme. However, it is not routinely used due to risks associated with blood transfusion.

Special Considerations

- Pseudocholinesterase Deficiency

This inherited enzyme deficiency is the most common cause of prolonged scoline apnea.

- Genetic Testing

Patients and their families should be offered genetic testing to identify pseudocholinesterase variants.

Recovery and Follow-Up

1. Reassurance and Explanation

Once awake, the patient is reassured and the situation is explained.

2. Documentation

All events, discussions, and plans are clearly recorded.

3. Long-Term Advice

Patients with confirmed deficiency should be informed, possibly carry a medical alert card, and their family physician should be notified.

Conclusion

Serum cholinesterase testing is advised; document perioperative events, educate patients, avoid cholinesterase inhibitors, inform anaesthesiologists, and consider hormonal assays if deficiency is suspected.

References

1. Alhammadi Y, Alshamsi M, Alnuaimi A. Suxamethonium-induced prolonged apnea: insights from a clinical case. *Mathews J Anesth.* 2024;5(1):16.
2. Bulut A, Ozdemir-Karatas M, Ozdogan H, Yilmaz M, Inan LE. Development of prolonged apnoea in a suxamethonium-naïve patient during electroconvulsive therapy: a case report. *Cureus.* 2024 Oct 25;16(10):e72344. doi:10.7759/cureus.72344.

3. Ammundsen HB, Sorensen MK, Gatke MR. Succinylcholine resistance. Br J Anaesth. 2015 Dec;115(6):818-21.
4. Zavorotnyy M, Zwanzger P. Prolonged apnea during electroconvulsive therapy in monozygotic twins: case reports. Ann Gen Psychiatry. 2011;10:30.
5. Nair A, Al Qasaab R. Anesthetic practices for lower segment cesarean section in the Sultanate of Oman: a national survey. Cureus. 2024