

CASE REPORT

SUCCINYLCHOLINE APNOEA IN A PATIENT POSTED FOR TONSILLECTOMY: A CASE STUDY

M. Mazharulhaq¹

HOW TO CITE THIS ARTICLE:

M. Mazharulhaq. "Succinylcholine Apnoea in a Patient Posted for Tonsillectomy: A Case Study". Journal of Evidence based Medicine and Healthcare; Volume 1, Issue 14, December 08, 2014; Page: 1788-1791.

ABSTRACT: Even after advent of newer neuromuscular blocking agents succinylcholine has been the first choice of relaxant by most of anesthesiologist. The swift onset of action and rapid recovery from the block are the prime features prompting use of succinylcholine, but in few rare and unfortunate cases have reported to have a prolonged duration of neuromuscular block which is termed as succinylcholine apnoea. Here we are reporting a case of succinylcholine apnoea and its management.

KEYWORDS: Succinylcholine, pseudocholinesterase, apnoea, anesthesia.

CASE REPORT: 17 year old girl came with history of frequent throat pain and fever, which on evaluation by otorhinologist was diagnosed to have chronic tonsillitis and was advised to undergo tonsillectomy for the same after 15 days of recovery from the sore throat.

Patient was referred for anesthetic pre- evaluation and was found to have no other significant medical or surgical histories. An adult female patient belonging to Non-vaishya who was moderately built and nourished, oriented to time place and person, patient was conscious and co-operative. Systemic examination of the patient was done and was found to be unremarkable. Patient was also subjected for routine laboratory investigations and were reported to be within normal limits. It was decided to take her up for surgery under general anesthesia, she was given preoperative instructions and was advised Tab. Ranitidine 150 mg HS and Tab Alprazolam 0.5 mg HS on night before of surgery.

On the day of surgery patient was pre medicated with Inj. glycopyrrolate 0.2 mg and Inj. Fentanyl 100 microgram. After pre-oxygenating the patient anesthesia was induced with Inj. Propofol 2.5 mg/kg and to ease the intubation Inj. Succinylcholine was used at the dose of 1mg/kg. nasotracheal intubation was accomplished with 6.0 cuffed tube and tube was fixed after ascertaining its correct placement. Anesthesia was maintained with mixture of oxygen: nitrous oxide and hand ventilation was continued to look for the neuromuscular recovery for administration of the non- depolarizing agent. Surgery was started after 10 minutes of intubation, even after the surgical incision no neuromuscular recovery was noted. Neuromuscular monitor was procured and it was found to have no muscle response for TOF on suspicion of succinylcholine apnoea patient was subjected to plasma choline esterase assay and fresh frozen plasma was asked to be arranged, in the meantime anesthesia was now maintained with mixture of oxygen, nitrous oxide and 1% Isoflurane. Surgery lasted for 90 minutes and even at the end of surgery patient was found to be apneic and no neuromuscular response on TOF stimulation.

Patient was shifted to ICU connected to ventilator with settings of tidal volume of 8 ml per kg, support respiratory rate of 10 breaths per minute in SIMV mode. FFP 10 ml per kg was given and after 30 minutes patient was found to have spontaneous respiratory attempts. Patient

CASE REPORT

gradually over next 15 minutes fulfilled the criteria for extubation, patient was extubated and was found to have uneventful recovery from there. The plasma cholinesterase report was found to be 310 U/L (normal between 3000-6000 U/L).

CASE 2: A 35 year old male patient was posted for emergency laparotomy for hollow viscera perforation. Patient gave history of pain abdomen since two days associated with fever. No other significant medical or surgical history could be elicited from the patient.

On examination of the patient, an adult male patient moderately built and nourished, dehydrated and febrile (101° F), was also found to have tachycardia with pulse rate of 120 beats per minute and was thread in nature, blood pressure was 110/74 mm of Hg. Systemic examination of patient revealed diffuse tenderness and guarding of the abdomen. Patient was restless and was disoriented. Patient's attenders gave history of his addiction to alcohol and his last intake of alcohol was 1 day ago. His last oral intake was 4 hours ago. His investigation showed hemoglobin of 12 gram percent, total count of 16,500 cell per cc, blood urea of 44 mg/dl and serum creatinine of 1.6 mg/dl. After preoperative assessment of the patient it was decided to take the patient under ASA IIIIE. Patient was taken to surgery under general anesthesia.

He was premedicated with Inj. Glycopyrrolate 0.2 mg, Inj. Fentanyl 100 microgram, patient was pre oxygenated with 100% oxygen and induction of anesthesia was done with Inj. Thiopentone 200mg iv and for muscle relaxation Inj. Succinylcholine 75 mg was used to achieve rapid sequence intubation. Patient was maintained with gas mixture of oxygen and nitrous oxide, hand ventilation was carried out and recovery from succinylcholine was assessed before putting the patient on mechanical ventilation and non-depolarising muscle relaxant. But the patient did not show signs of recovery even after 30 minutes after intubation. Patient was found to have pulse rate of 100-110 beats per minute and blood pressure of 130/80-146/98 mm of Hg patient was also found to be in normothermia and ETCO₂ was also with in normal range. Neuromuscular monitor was now used and it was found that no response to twitch or train of four. Patient was suspected to have succinylcholine apnoea, and anesthesia was now maintained with mixture of oxygen, nitrous oxide and sevoflurane. At the end of surgery which lasted for 2 hours patient was shifted to post anesthesia care unit and mechanical ventilation was carried out with tidal volume of 500 ml, respiratory rate of 12 breaths per minute, PEEP of 5 cm of water in AC mode. Patient was not affordable for any further investigation. Patient was found to regain spontaneous respiration after 5 hours in ICU, the ventilatory mode was now changed to SIMV mode and after full recovery from the muscle relaxant he was extubated. Further stay in hospital was uneventful.

DISCUSSION: Low plasma cholinesterase was proposed mechanism for the delayed recovery from succinylcholine and was most accepted theory at the time of proposal, the theory was put forward by Thesself and Broune et al.^[1-2] But now it is becoming clear that it is not a single cause but rather a syndrome. Leyman and Kalow were pioneers in demonstrating the familial pattern of low cholinesterase activity.^[3-4] In 1957 Kalow used dibucaine number as a marker to differentiate the two allelic genes. Central depression by general anesthesia, errors in controlled ventilation and accumulation of CO₂ or acapnea, exhaustion of hering breur reflex, hypokalemia, altered response of the end plate to succinylcholine, allergy to succinylcholine and breath holding

CASE REPORT

triggered by endotracheal tube are few other reasons hypothesized for the succinylcholine apnoea.^[5]

Succinylcholine apnoea may also be reported in cases of continuous infusion or repeated doses of drug. Nevertheless geriatric patients, dehydrated and debilitated patients and those with gastrointestinal pathologies are prone for the prolonged recovery from succinylcholine.

- Succinylcholine is metabolized by pseudocholinesterase to succinylmonocholine and choline. Butyrylcholinesterase or pseudocholinesterase is synthesized in liver and has a half-life of 7-10 days. Pseudocholinesterase has large metabolizing capacity and hence significant reduction in the butyrylcholinesterase activity is required to cause clinical prolongation of action of succinylcholine. Liver disease, old age, oral contraceptive pills, burns, malnutrition, MAO inhibitors, cytotoxic drugs, neoplastic disease, bambetorol and esmolol are all noted to reduce the activity of pseudocholinesterase.^[6]
- Accumulation of succinylmonocholine, which is found to have 1/20th potency of succinylcholine when accumulates on a continuous infusion of succinylcholine is also proposed to cause prolonged apnoea. The succinylmonocholine is metabolized more slowly compared to the parent compound. It is also found that if respiration is assisted rather than controlled there is less risk of prolonged apnoea.
- Neuromuscular agents are found to have central depression on respiratory center. Central depression by succinylcholine, is also hypothesized cause for prolonged apnoea.^[7]
- Central depression is well known characteristic of most of general anesthetic agents and this is also a contributing factor for succinylcholine apnoea.
- If proper controlled ventilation in cases receiving succinylcholine is not established there can be various diselectrolytemias predominantly lowered pH which persists after the period of anesthesia and may cause prolongation of the muscle paralysis.^[8]
- Failed redistribution of the drug from the end plate in cases of increased affinity of the nicotinic receptors to the drug or dehydration also is hypothesized to prolong the action of succinylcholine unduly.^[9]
- Altered sensitivity of the end-plate to the neuromuscular blocking drugs following disease states is found to cause non-depolarising block by the depolarizing blocking agents and the block is also found to be reversed by neostigmine. Thus altered disease states can prolong the action of succinylcholine.^[10]

TREATMENT: Treatment depends on the suspected underlying mechanism and also on the period of development of apnoea. If the apnoea develops on table during surgery stop the succinylcholine if used in continuous drip or repeated dose pattern, correct other factors like too deep plane of anesthesia, over dosed narcotics and breath holding spells.

Patient should be ventilated till the spontaneous respiration is regained, cholinesterase can be provided to patient in form of fresh frozen plasma or cholinesterase concentrate.

Complications such as hyperkalemia, raised intraocular pressure, raised intracranial pressure, masseter spasm, myalgia, sinus bradycardia, malignant hyperthermia have to be kept in mind and treated appropriately.

CASE REPORT

CONCLUSION: Here we have mentioned the caste of the patient because pseudocholinesterase deficiency is found to run in few castes. Prolonged apnoea in this case is because of deficiency of pseudocholinesterase, intraoperative neuromuscular monitoring and pseudocholinesterase assay helped us to clinch the diagnosis. In absence of the predisposing factors for the reduced pseudocholinesterase the patient was ventilated and cholinesterase supplemented in form of fresh frozen plasma.

REFERENCES:

1. Thesieff, S.: Personal communication to J. Kennedy Harper. Brit. M. I., 1: 1352. 1952.
2. Bourne, G. Collier H. O. J., and Somers, G. F.: Succinylcholine (Succinylcholine), Muscle Relaxant o. Short Duration. Lancet, 1: 1225-1229, 1952.
3. Layman H and Rayann F, 1956, the familial incidence of low pseudocholinesterase level; Lancet; 2: 124.
4. Kalow W 1956, the familial incidence of low pseudocholinesterase level; Lancet; 2: 576.
5. Francis F. Foldes, M.D., L. Rendell-Baker, Causes and Prevention of Prolonged Apnea with Succinylcholine, Anesthesia and analgesia, November/December 1956 - Volume 35 - Issue 6, 609-33.
6. Mohamed Naguib, Cynthia A.Lien, Claude Meistelman, Pharmacology of Neuromuscular Blocking Drugs, Miller's anesthesia 8th edition, 1: 958-94.
7. Ellis, C. H., Morgan, W. V. and Beer, E. J. de: Central Depressant Actions of Certain Myoneural Blocking Agents. J. Pharmacol. & Exper. Therap., 106: 353-363, 1952.
8. Davis, D. A, Ellis, F. I: Reese, N. O., and Grosskreutz, D. C.: Anaesthesiology, 16: 333-342, 1955.
9. Marsh D. F.: The Distribution, Metabolism and Excretion of d-Tuhocurarinc Chloride and Related Compounds in Man and Other Animals. J. Pharwz; 6. Exo. Thrap., 105: 299-316, 1952.
10. Churchill Davidson H. C., and Richardson, A. T.: The Action of Decamethonium Iodid (Clo) in Myasthenia Gravi;. I. Neurol. Neurosurg. and Psychiat., 15: 129-133, 195%.

AUTHORS:

1. M. Mazharulhaq

PARTICULARS OF CONTRIBUTORS:

1. Associate Professor, Department of Anaesthesia, Bidar Institute of Medical Sciences, Bidar, Karnataka.

NAME ADDRESS EMAIL ID OF THE CORRESPONDING AUTHOR:

Dr. M. Mazharulhaq,
8-8-13, KHB Colony,
Bidar-585401.
E-mail: dr.mmh_anae@yahoo.in

Date of Submission: 25/10/2014.
Date of Peer Review: 26/10/2014.
Date of Acceptance: 27/11/2014.
Date of Publishing: 05/12/2014.