

## Prolonged Apnea Case Report

Hayriye Alp

Necmettin Erbakan University, GETAT CENTER, Konya, Turkey.

**Corresponding Author:** Hayriye Alp, Necmettin Erbakan University, GETAT CENTER, Konya, Turkey.

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### Abstract

Succinylcholine affects nicotinic receptors and depolarizes the neuromuscular junction, such as atilkolin. Succinylcholine mimics acetylcholine by tightly binding to alpha subunits of acetylcholine receptors after junction. In the obstetrics department of our hospital, a 35-year-old female patient was hospitalized for her 5th pregnancy, who had 1 abortus 3 normal births before. The patient was intubated with a number 7.5 cuffed intubation tube after mask mask ventilation, number 4 after 200mg propofol, 100mg lystenon2%(Suksametonyum chlorure) in induction. The maintenance was achieved with 1% mac sevoflurane. Although the anesthetic agents were closed after the operation, pseudocoline esterase deficiency was considered due to the patient not being able to wake up, not reaching sufficient tidal volume, and having snake-like movements. A total of 3 tdp (fresh frozen plasma) was given. When the patient reached sufficient tidal volume and regained consciousness, he was extubated after 4 hours and followed up in the waking room.

**Keywords:** prolonged apnea, pseudocoline esterase

### Introduction

Succinylcholine affects nicotinic receptors and depolarizes the neuromuscular junction, such as atilkolin. Succinylcholine mimics acetylcholine by tightly binding to alpha subunits of acetylcholine receptors after junction. As a result, depolarization occurs, ion channels remain open and muscle cells cannot respond to new stimuli; muscle paralysis occurs. The order of paralysis may differ slightly. However, the respiratory muscles are paralyzed last [1, 2]. The most important drug in depolarizing, muscle relaxants is succinylcholine. Succinylcholine contains two acetylcholine molecules in structure. It hydrolyzes automatically at room temperature and alkaline environment. Therefore, it should be stored in the refrigerator.

Succinylcholine is used during anesthesia induction, in patients requiring rapid intubation, in procedures requiring a short period of relaxation, such as endoscopy and electroconvulsive therapy, since its effect begins quickly and lasts a short time [3]. It is also the most commonly used relaxing medicine in obstetrics. Today, new emerging short-acting non-depolarizing muscle relaxants are more preferred because they can cause conditions such as dual block and fasciculation pain.

### Case

In the obstetrics department of our hospital, a 35-year-old female patient was hospitalized for her 5th pregnancy, who had 1 abortus 3 normal births before. She was not pregnant for 38 weeks, and anesthesia was informed because of fetal distress. In his tests, glucose was 68mg/dl creatine 0.4mg/dl bk 12,000ul, plt 128ul, fibrinogen 453mg/dl. Other routines were normal. **Legal informed consent was obtained from the patient.** The patient was intubated in induction with a number 7.5 cuffed intubation tube after 200mg propofol((2,6-diisopropilfenole). 100mg lystenon 2% (suksametanyum chlorure) and number 4 mask ventilation. Maintenance was provided with 1% sevoflurane. The operation took 1

hour. The baby cried, his spontaneous enough tone was good. Although the anesthetic agents were closed after the operation, pseudocoline esterase deficiency was considered due to the patient not being able to wake up, not reaching sufficient tidal volume, and having snake-like movements. Since blood pressure increased up to 180/90mmHg due to strain, a total of 15 cc was made from 1/10 perlinganit(gliserol trinitrate). Sedation was entered with propofol(2,6-diisopropilfenole). Fresh frozen plasma (tdp) compatible with the patient's blood group was requested from the blood bank. As the patient's urine output decreased, lasix (furosemide) was performed. A total of 3 tdp (fresh frozen plasma) was given. When the patient reached sufficient tidal volume and regained consciousness, he was extubated after 4 hours and followed up in the waking room. After awakening, vital signs were stable. The need for analgesia was met with 1amp oxamene (tenoxicam). On the postoperative day 1, the white sphere was 17,000ul hemoglobin 10g / dl platelet 104ul. Metiler ampule(metilergonovine maleate) received 3 \* 1 methimazole(thyamosole 10mg), 3 \* 1 genta(gentamisine) 160mg 1 \* 1(im).

It was learned that the patient had not received general anesthesia before the visit. The patient was warned that his enzyme level was low and his file was included in the case of late awakening. Postoperative pseudocoline esterase level was 2 (lower limit 3.3). If the patient received general anesthesia, a short-acting muscle relaxant was not warned and the relatives were informed.

### Discussion

The greatest advantage of succinylcholine to other relaxants is that it is fast and short-term due to its rapid hydrolysis by plasma cholinesterase (3). IV dose is 1 mg / kg and its effect; It starts at 10-30 seconds and lasts for 2-5 minutes. It is administered in small repeated doses (10 mg) for long-term muscle relaxation. It can be given at a dose of 1.5–2 (maximum

4) mg / kg, especially in young children, where it is difficult to find a vein by means of M. Its effect starts at 1–1.5 min and 10–15 min. takes. Since succinylcholine does not dissolve in fat, its distribution is outside the cell. Extracellular volume per kilogram is higher in young children, infants and newborns than adults. For this reason, children are given higher doses of succinylcholine per kilogram than adults.

The effect of succinylcholine starts quickly (30-60 seconds) and lasts short (5-10 minutes). When it enters the circulation, succinylcholine is hydrolyzed by pseudocholinesterase into the nerve-muscle junction. Its half-life is 2.6 minutes. The amount of succinylcholine (sch) reaching the motor last plate is 1/10 of that given. The effect of plasma cholinesterase is no longer involved here. This succinylcholine amount, which escapes plasma hydrolysis at the beginning of the application, determines the block time [1, 2, 3 and 4]. As the serum level decreases, succinylcholine molecules move away from the nerve muscle junction and the choline and succinic acid are destroyed in the plasma. When used in the form of a long-term and high-dose, infusion, and when the dose exceeds 1g, the block type may change and its effect may be prolonged. (Prolonged apnea due to low enzyme level can be treated with blood transfusion).

Plasma cholinesterase and butyryl-cholinesterase, also called PChE (Pseudocholinesterase) It is synthesized in the liver. It is found in plasma, liver, brain, pancreas and kidneys. When the enzyme is insufficient or atypical in quantity, the Sch effect is prolonged [10]. Atypical enzyme is found in some people as a result of a disorder in the genes responsible for making this enzyme. Atypical pseudocholinesterase has low affinity to Sch and hydrolysis of the drug occurs very slowly [5, 6, 7, 8 and 9]. The inhibition percentage of pseudocholinesterase activity is determined by the number of dibucain (DN)

Enzyme deficiency: In radiotherapy states, chemotherapy states, organic phosphorus poisoning, hyperpyrexia, heart failure, liver failure, hunger, uremia, hypoproteinemia, pregnancy, puerperium, obesity, myxedema or cyclophosphamide, echotifat eye drops, procainamidine, quinidine, There may be enzyme deficiency in ketamine and pancuronium propanidid users [4,10].

## Suggestions

Genotype studies together with preoperative BChE enzyme levels will help increase anesthesia safety, especially in pregnant women who will undergo elective cesarean operation and have a history of postoperative apnea due to muscle relaxants.

## Conflict of Interest

There is no conflict of interest in this study.

Since the case was presented retrospectively, an ethical committee was not taken.

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