International Journal of Anesthesia and Clinical Medicine
2023; 11(2): 69-71
http://www.sciencepublishinggroup.com/j/ijacm
doi: 10.11648/j.ijacm.20231102.13

A Case of Pseudocholinesterase Deficiency in Patient Underwent General Anesthesia with Flexible Bronchoscopy

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To cite this article:
Dongjiao Wu, Xuejie Li. A Case of Pseudocholinesterase Deficiency in Patient Underwent General Anesthesia with Flexible Bronchoscopy. *International Journal of Anesthesia and Clinical Medicine*. Vol. 11, No. 2, 2023, pp. 69-71. doi: 10.11648/j.ijacm.20231102.13

Received: July 9, 2023; **Accepted**: July 25, 2023; **Published**: July 31, 2023

Abstract: Pseudocholinesterase deficiency is a rare clinical condition primarily associated with genetic alterations, but it can also be caused by certain diseases and medication factors. Patients with this condition experience significantly prolonged muscle paralysis when succinylcholine or mivacurium is used during general anesthesia, due to the decreased enzyme levels. The diagnosis of pseudocholinesterase deficiency is typically made after the administration of succinylcholine or mivacurium. Inquiring about the patient's family history is also crucial for proper diagnosis and intervention. Here, we report a case of a rare pseudocholinesterase deficiency patient who experienced delayed recovery following general anesthesia for flexible bronchoscopy. The patient was a healthy 67-year-old male with no history of liver or kidney dysfunction or other diseases. The plan was to perform painless flexible bronchoscopy, and after the procedure, the patient exhibited delayed recovery. Throughout the process, electrocardiographic monitoring showed normal blood pressure, heart rate, and oxygen saturation. After ruling out other factors that could cause delayed emergence, including central nervous system issues and electrolyte imbalances, it was found that succinylcholine, a depolarizing muscle relaxant, had been used. There was a high suspicion of pseudocholinesterase deficiency in the patient. Pseudocholinesterase enzyme activity testing was performed, and the patient was continued on mechanical ventilatory support. After 220 minutes from the completion of the procedure, the patient regained spontaneous breathing and full consciousness, and the endotracheal tube was removed. This article presents a case of delayed recovery in a patient with pseudocholinesterase deficiency following painless flexible bronchoscopy. It also summarizes the causes, clinical manifestations, diagnosis, and treatment of pseudocholinesterase deficiency-related delayed emergence. It is hoped that this article will contribute to timely recognition and management of such cases, thereby preventing any potential adverse outcomes for patients. Furthermore, since pseudocholinesterase deficiency is relatively rare, further research is needed to confirm the effectiveness of the preventive and therapeutic measures mentioned in this article.

Keywords: Pseudocholinesterase Deficiency, Delayed Recovery, Succinyl Choline

1. Introduction

To achieve the examination requirements and facilitate rapid recovery of the patient during painless flexible bronchoscopy, we typically administer a combination of succinylcholine. However, pseudocholinesterase deficiency is a rare condition characterized by the patient's extreme sensitivity to specific muscle relaxants, including succinylcholine and mivacurium during general anesthesia, leading to a significant prolongation of muscle paralysis [1]. Failure to promptly detect and differentiate this condition may result in serious consequences, such as respiratory failure [2, 3]. We report a case of delayed recovery in a patient with pseudocholinesterase deficiency underwent flexible bronchoscopy with general anesthesia.

2. Case Report

The patient is a 67-year-old male with a height of 172cm and weight of 62 kg. A left lung mass was discovered during a physical examination, and it was planned to perform flexible bronchoscopy under general anesthesia. The patient had no previous history of surgical anesthesia. Routine blood

tests, biochemical tests and coagulation tests did not show any significant abnormalities before following flexible bronchoscopy. The electrocardiogram indicated sinus rhythm with ST segment changes. Upon entering the examination room, the patient was conscious, and the electrocardiographic monitoring showed a blood pressure of 129/81 mmHg, a heart rate of 80 beats per minute, and an oxygen saturation (SPO_2) of 97%. General anesthesia induction was performed as follows: intravenous administration of 5 μg sufentanil, 80 mg propofol, and 100 mg succinylcholine. After the patient lost consciousness and spontaneous breathing ceased, mask-assisted ventilation was initiated. After achieving satisfactory muscle relaxation, a size 4 laryngeal mask airway (LMA) was inserted, and the flexible bronchoscopy procedure commenced. During the procedure, intermittent single boluses of 20 mg propofol and 10 mg succinylcholine were administered twice. The examination proceeded smoothly, and the vital signs remained stable throughout the 20-minute duration. Fifteen minutes after the completion of the examination, it was observed that the patient had not regained spontaneous breathing and remained unconscious. Mechanical ventilatory support was continued. Nearly 60 minutes after the examination, the patient still had not recovered spontaneous breathing and did not respond to painful stimuli. Physical examination revealed equal and round pupils, temporarily ruling out central nervous system problems. Arterial blood gas (ABG) did not show any significant abnormalities. Upon reviewing the previously administered anesthetics, it was noted that succinylcholine had been used. The possibility of pseudocholinesterase deficiency in the patient was considered, prompting an immediate pseudocholinesterase enzyme activity test. Approximately 180 minutes after the examination, the patient exhibited swallowing reflex, and spontaneous breathing gradually recovered. However, the tidal volume was only approximately 40 ml. Around 220 minutes after the examination, the patient opened his eyes upon stimulation, could raise his arm as instructed, nodded or shook his head in response to questions, and exhibited a tidal volume of around 400 ml with a respiratory rate of approximately 18 breaths per minute. After a thorough evaluation, the endotracheal tube was removed. The patient's vital signs remained stable during a 40-minute observation period, and he was transferred back to the ward. On the third day of follow-up after the examination, the patient's general condition was unremarkable. The pseudocholinesterase activity was tracked and found to be 0.85 kU/L (normal range: 4.9-11.9 kU/L).

3. Discussion

Pseudocholinesterase, also known as butyrylcholinesterase, is synthesized in the liver and predominantly found in the plasma [4]. Succinylcholine is a depolarizing neuromuscular blocking agent that acts rapidly, reaching its peak effect within 60 to 90 seconds after intravenous injection. The respiratory muscle function fully recovers in 6 to 12 minutes [5]. Upon intravenous administration, succinylcholine is immediately hydrolyzed by pseudocholinesterase in the blood and liver [6, 7]. It is first metabolized into succinylmonocholine, which is further slowly metabolized into succinic acid and choline, becoming a metabolite with no neuromuscular blocking activity. Approximately 10% to 15% of the administered dose reaches the target site, and the half-life of succinylcholine in the blood is 2 to 4 minutes [8]. Pseudocholinesterase is composed of two types of genes: a normal type and an abnormal type. The normal pseudocholinesterase accounts for approximately 94% of the population and can efficiently hydrolyze succinylcholine. On the other hand, the atypical pseudocholinesterase, composed of abnormal genes, cannot hydrolyze succinylcholine, leading to its prolonged action [9].

Deficiency of pseudocholinesterase can be caused not only by genetic factors but also by other pathological conditions or drug effects such as advanced age, late pregnancy and postpartum period, malnutrition, burns, liver disease, hemodialysis, the use of monoamine oxidase inhibitors, anticholinesterase drugs, metoclopramide, and so on. These conditions can result in reduced enzyme levels or decreased enzyme activity [10]. When the enzyme activity is reduced to 20% of normal, succinylcholine-induced respiratory arrest is prolonged from the usual 3 minutes to 9 minutes [11]. Enzyme gene variations that significantly decrease enzyme activity have a noticeable impact on the metabolic rate of succinylcholine, even rendering succinylcholine unable to be metabolized, thereby causing a significant prolongation of its neuromuscular blocking effect. It is worth noting that patients with this condition may face the risk of cardiac arrest when using cocaine. The main complication in such patients is respiratory failure due to prolonged neuromuscular blockade, and careful assessment should be performed before extubation. For patients known to have this condition, the use of succinylcholine and mivacurium should be avoided [12].

Lakshmi et al. reported a case of an 18-year-old female patient who underwent bilateral breast reduction surgery. Following administration of succinylcholine, the patient experienced delayed recovery after the procedure. She was transferred to the post-anesthesia care unit (PACU) after sedation with midazolam and propofol [13]. The endotracheal tube was removed 4 hours after surgery, and the examination of pseudocholinesterase activity revealed significantly lower levels compared to the normal range. The authors suggested that for such patients undergoing anesthesia, rocuronium could be considered as a muscle relaxant. Rocuronium is a non-depolarizing muscle relaxant that acts rapidly, and it can be effectively reversed with sugammadex, a specific muscle relaxant antagonist. Sugammadex is a γ-cyclodextrin derivative specifically designed to antagonize aminosteroid non-depolarizing muscle relaxants [14]. It has a lipophilic core and a hydrophilic outer shell, which allows it to encapsulate aminosteroid muscle relaxants such as rocuronium and vecuronium, forming stable water-soluble compounds that are excreted by the kidneys in the urine. Yildizhan et al. reported that their hospital routinely measures serum cholinesterase levels in patients scheduled for electroconvulsive therapy,

allowing for informed anesthesia decisions. They suggested that such patients may have other hidden risk factors, including malnutrition, impaired liver or kidney function, and cardiovascular events [15]. Individuals diagnosed with low cholinesterase deficiency should inform their anesthesiologist prior to surgery, and their medical records should be promptly updated to include the diagnosis of pseudocholinesterase deficiency. It is recommended that other family members of these patients undergo cholinesterase level testing before surgery, as the majority of cases are associated with genetic variations in the pseudocholinesterase gene [16, 17].

In this case, the patient is an elderly male with normal liver and kidney function prior to examination, no intake of medications that affect enzyme activity, and no history of anesthesia in other family members. The measured pseudocholinesterase activity in the patient's plasma is severely decreased. Considering the clinical presentation, we suspect pseudocholinesterase deficiency, which leads to slow metabolism of succinylcholine and a significant prolongation of the neuromuscular blocking effect, resulting in delayed patient recovery.

Therefore, when there is a significant prolongation of muscle relaxation time after the use of succinylcholine, it is important to promptly identify other factors, including excessive administration of anesthetic drugs, electrolyte disturbances such as hypokalemia, central nervous system causes, and hyperventilation. The possibility of abnormal pseudocholinesterase activity should be considered, and monitoring of muscle relaxation should be performed. Evaluation of motor function recovery and respiratory support with a ventilator should be provided until complete recovery from muscle relaxation. When feasible, testing for pseudocholinesterase activity is recommended. During this process, sedation measures may be administered. There are literature reports suggesting that the administration of fresh frozen plasma may be beneficial in replenishing functional pseudocholinesterase and thereby accelerating the metabolism of succinylcholine.

4. Conclusion

Pseudocholinesterase deficiency is a clinical condition that is often only discovered by delayed recovery after the use of succinylcholine or mivacurium during anesthesia. It is important to promptly differentiate this condition from other factors and take appropriate measures. Nerve stimulation should be performed to evaluate repeatedly the return of motor function. Respiratory support should be provided until complete recovery of muscle paralysis. In cases where necessary, fresh frozen plasma may be administered.

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