Case Report

Bilateral vocal cord paralysis during emergence from general anesthesia in a patient with Parkinson's disease

ABSTRACT

Parkinson's disease (PD) is a neurodegenerative disorder that affects the extrapyramidal system, and respiratory dysfunction has also been noted in patients with PD. However, acute upper airway obstruction due to bilateral vocal cord paralysis is a very rare finding in PD. Here, we describe a rare life-threatening respiratory failure caused by bilateral vocal cord paralysis in an elderly woman with PD during emergence from general anesthesia. The tracheostomy was performed on the postoperative period because the condition persisted. The general anesthesia in PD may have aggravated vocal cord impairment. We recommend when a patient with PD is scheduled for general anesthesia that the anesthesiologist performs careful preoperative examinations, strictly monitors respiratory function, and rapidly manages acute upper airway obstruction.

Key words: General anesthesia; Parkinson's disease; vocal cord paralysis

Introduction

Parkinson's disease (PD) is a neurodegenerative disorder involved in the control of motor coordination that is caused by depletion of dopamine-secreting neurons in basal ganglia.[1] The respiratory dysfunction has been noted in PD and attributed to impaired central control of respiratory muscles, upper airway obstruction, and laryngeal muscle atony. Therefore, the anesthetic consideration in patients with PD includes aspiration, atelectasis, laryngospasm, and postoperative respiratory failure. However, acute upper airway obstruction due to bilateral vocal cord paralysis during emergence from general anesthesia is a very rare finding in PD.

Here, we describe a rare and life-threatening case of acute upper airway obstruction caused by bilateral vocal cord paralysis in a patient with PD during emergence from general anesthesia.

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Case

We anesthetized a 150-cm, 50-kg, and 65-year-old female with a diagnosis of PD for percutaneous nephrolithotomy. She was diagnosed with PD in 2015 and has been under medication using a combination of carbidopa and levodopa. During preoperative evaluation, lung sounds were clear and there were no indications of a vocal cord problem. The preoperative biochemistry and chest radiologic findings were unremarkable.

In the operating room, standard anesthetic monitoring including blood pressure, electrocardiogram, pulse oximetry, end-tidal CO_2, and bispectral index (BIS) was initiated. General anesthesia was induced using propofol (1 mg/kg) and remifentanil infusion. After administering rocuronium bromide (0.8 mg/kg), tracheal intubation was performed

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without airway trauma. In laryngoscopic view, the vocal cord was intact without evidence of paralysis, and no wheezing sound was auscultated in either lung after tracheal intubation. Anesthesia was maintained with sevoflurane in air and oxygen and continuous remifentanil infusion (0.1–0.5 μg/kg/h). Ventilation was conducted with 50% oxygen at a respiratory rate (RR) of 10 breaths/min to a volume of 8 mL/kg and maintained at a BIS between 40 and 60.

At the end of the surgery, all anesthetics were stopped, and neuromuscular blockade was reversed with pyridostigmine (0.25 mg/kg) and glycopyrrolate (0.008 mg/kg). The patient opened her eyes spontaneously and breathed regularly, and thus, the tracheal tube was gently removed. At first, she breathed well and seemed comfortable, but her respiratory rate increased gradually, and stridor was heard. Despite positive pressure mask ventilation with 100% oxygen and immediate administration of 200 mg of sugammadex, oxygen saturation dropped to 90% and her RR reached 30 breaths/min with a tidal volume of only 2 mL/kg. We immediately inspected the patient's airway using bronchoscopy. The view revealed fixed bilateral vocal cord in adducted position with airway opening less than 1 mm [Figure 1]. We deemed her condition had progressed to respiratory failure and decided on intubation. The patient was transferred to the intensive care unit and placed under ventilator care. The patient started taking carbidopa/levodopa enterally by nasogastric tube on postoperative day 1 (POD 1).

The patient was able to obey verbal commands, and her blood oxygen and carbon dioxide levels had normalized on POD 2. She was weaned off the ventilator and allowed to breathe for herself in an intubated state. On POD 3, the medical team performed extubation, but her respiratory rate increased after tube removal. The patient complained of breathing difficulties and her oxygen saturation gradually dropped to 64%. Bronchoscopic view confirmed recurrence of bilateral vocal cord paralysis. The patient was reintubated immediately. The medical team decided to perform tracheostomy due to risk of persistent vocal cord dysfunction. Tracheostomy was performed on POD 6 and the respiratory problem was promptly resolved. The patient's vocal cord movement continuously showed unstable movement, and therefore finally discharged with tracheostomy.

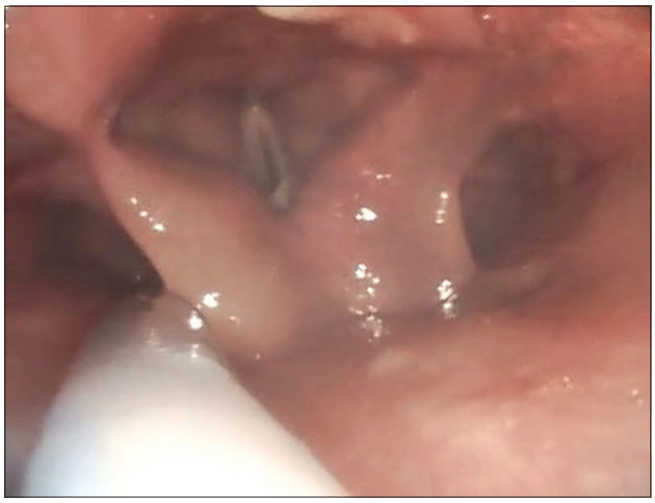

Figure 1: Vocal cord finding during emergence from general anesthesia. Fixed bilateral vocal cord in adducted position with airway opening less than 1 mm

Discussion

The anesthetic implications of PD include hypotension or hypertension, muscle rigidity, and respiratory failure.[2] The etiologies of upper airway obstruction in PD are suspected laryngeal spasm, dystonia of laryngeal muscles, and very rarely bilateral vocal cord paralysis.[3,4] In our patient, life-threatening respiratory failure occurred because of upper airway obstruction caused by bilateral vocal cord paralysis, which developed during emergence from general anesthesia.

After extubation during emergence, the patient's RR increased, and stridor was auscultated. We suspected incomplete reversal of neuromuscular block and laryngeal spasm. In spite of sugammadex administration and continuous positive pressure ventilation, her oxygen saturation dropped continuously. In bronchoscopic view, we found that the bilateral vocal cord was adducted in paramedian position with paralysis. We concluded that the vocal cord paralysis was responsible for the upper airway obstruction during emergence rather than laryngeal spasm. It is difficult to differentiate between laryngospasm and vocal cord paralysis in an emergent situation, but the most important management course is to secure the airway in both situations quickly and safely. During postoperative periods, when the medical team tried to extubate, the symptoms of upper airway obstruction reoccurred, and recurrence of bilateral vocal cord paralysis was confirmed by bronchoscopic view. Finally, after tracheostomy, the patient breathed normally through the tracheostomy site. The reoccurrence of vocal cord paralysis suggested that the patient had irreversible alteration of laryngeal muscles movements.

A well-controlled extrapyramidal system is needed for automatic cyclic laryngeal muscle activity to maintain the stability and patency of the upper airway. The muscles include the posterior cricoarytenoid muscle and interarytenoid and thyroarytenoid muscles as adductors. The pathophysiology of vocal cord paralysis in PD has been proposed that it is due to persistent tonic activity of intrinsic laryngeal muscles, which are extrapyramidal involuntary movements.[2,5,6] Several

authors have suggested that the mechanism responsible for vocal cord paralysis involves dystonia and spasm of these adductor laryngeal muscles.[3,7,8] Furthermore, the extrapyramidal signs and symptoms of the vocal cord in PD are related to degeneration of nucleus ambiguous. The nucleus ambiguous controls efferent motor fibers of the vagus nerve terminating in intrinsic laryngeal muscles. Degeneration of the nervous system with innervation to intrinsic laryngeal muscles is considered as the cause for dysfunctional abduction of vocal cord.[9,10]

The extrapyramidal symptom in the respiratory function also shows fluctuations in severity. In stressful situation such as general anesthesia and surgery, in which various stress hormones are increased, fluctuation gets worse and induces severe airway compromise. Inhalational anesthetics have complex effects on brain dopamine concentrations during general anesthesia. Opioids inhibit dopamine release in the central nervous system and increase the likelihood of muscle rigidity. Therefore, anesthesiologists should consider whether patients with PD are vulnerable to the side effects of anesthetics and opioids. The responses to a neuromuscular blocking agent are generally normal, but hyperkalemia may rarely follow succinylcholine administration. The continuation of antiparkinson drugs is essential during the perioperative periods, as they reduce abnormal control and function of the upper airway.

In summary, we are of the opinion that upper airway obstruction is highly likely due to vocal cord impairment in PD, and that these complications be acutely exacerbated by perioperative stress related to surgery and general anesthesia. Therefore, we recommend that evaluation of respiratory function should be performed carefully prior to surgery and antiparkinson drugs should be continued. Finally, we recommend anesthesiologists to prepare for life-threatening upper airway obstruction during the perioperative period in patients with PD.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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