

# Negative pressure pulmonary edema due to upper airway obstruction after general anesthesia in a patient with Parkinson's disease: a case report

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## Abstract

A 65-year-old man with Parkinson's disease (PD) underwent spine surgery under general anesthesia, 13 hours after the last medication for PD. Postoperatively, negative pressure pulmonary edema developed following upper airway obstruction, a possible complication of PD. Oxygenation improved with high-flow nasal cannula therapy. Continued medication might have prevented such complications.

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## Abstract

A 65-year-old man with Parkinson's disease (PD) underwent spine surgery under general anesthesia, 13 hours after the last medication for PD. Postoperatively, negative pressure pulmonary edema developed following upper airway obstruction, a possible complication of PD. Oxygenation improved with high-flow nasal cannula therapy. Continued medication might have prevented such complications.

## Keywords

negative pressure pulmonary edema, Parkinson's disease, upper airway obstruction

## Background

Parkinson's disease (PD) is a common disorder that is complicated by multiple respiratory abnormalities, including upper airway obstruction (UAO).<sup>1</sup> To our knowledge, negative pressure pulmonary edema (NPPE), a noncardiogenic pulmonary edema induced by the generation of high negative intrathoracic pressure needed to overcome UAO,<sup>2</sup> has not been reported in patients with PD. We report the first case of NPPE after general anesthesia in a patient with PD.

## Case presentation

A 65-year-old man (175.0 cm, 65.0 kg) with a 23-year history of PD was scheduled to undergo C3-C7 cervical laminectomy with C3/C4 posterior fixation for cervical spondylosis. The patient was taking antiparkinson drugs, including levodopa-carbidopa taken four times a day. Symptoms of PD were well-controlled with drugs so that he was able to live independently. He was admitted to the hospital the day before surgery. The patient took the last medication the night before surgery, 13 hours before anesthesia. Considering the well-controlled PD status, oral antiparkinson drugs on the morning of surgery were neither administered nor replaced by intravenous levodopa.

General anesthesia was induced and maintained with continuous infusions of propofol and remifentanyl. Rocuronium (40 mg) was given to facilitate tracheal intubation. After the patient was placed in the prone position, neuromuscular relaxation was antagonized with sugammadex (150 mg) for monitoring of motor-evoked potentials, which remained unsuppressed throughout surgery. Arterial oxygen saturation (SpO<sub>2</sub>) was maintained between 97% and 99%, and arterial oxygen tension (PaO<sub>2</sub>) was 201 mmHg with inspired oxygen fraction (F<sub>I</sub>O<sub>2</sub>) of 0.5. Fentanyl (150 µg) and acetaminophen (1,000 mg) were intravenously administered for immediate postoperative analgesia. Durations of surgery and anesthesia were 161 and 254 minutes, respectively. Intraoperative fluid infusion, blood loss, and urine output were 1,410 mL, 60 mL, and 400 mL, respectively.

After emergence from anesthesia, a sufficient tidal volume and respiratory rate were confirmed. The patient could take a deep breath and move extremities voluntarily, as requested by anesthesiologists. Immediately after tracheal extubation, however, the patient presented with labored breathing. Inspiratory stridor was heard. Systolic blood pressure increased to 200 mmHg. SpO<sub>2</sub> dropped to 78%. PaO<sub>2</sub> was 61 mmHg during administration of oxygen at 8L/min via face mask. The patient's UAO was immediately treated with a jaw thrust maneuver and tightly fitted face mask. An immediate chest X-ray showed diffuse bilateral pulmonary consolidation without an increased cardiothoracic ratio, consistent with negative pressure pulmonary edema (NPPE) (Figure 1). Thus, 10-cm H<sub>2</sub>O positive end-expiratory pressure (PEEP) was applied to the spontaneously breathing patient using an anesthesia circuit. PaO<sub>2</sub> increased to 105 mmHg.

The patient was transferred to the intensive care unit. High-flow nasal cannula therapy (HFNC) was initiated with F<sub>I</sub>O<sub>2</sub> of 0.6 and a flow rate of 50 L/min. NPPE improved by the morning of postoperative day (POD) 1; PaO<sub>2</sub> increased to 204 mmHg with F<sub>I</sub>O<sub>2</sub> 0.6. Administration of antiparkinson drugs was resumed. On POD 2, the patient was weaned from HFNC, and returned to the ward without any oxygen supply.

## Discussion

Although UAO can develop as a manifestation of PD,<sup>1</sup> it seemed necessary to rule out other causes of UAO in this patient. First, anesthesia- or opioid-related respiratory depression or UAO was unlikely because the patient showed adequate respiration, alertness, and responsiveness before extubation. Second, UAO due to alterations in the upper airway anatomy caused by cervical spine surgery<sup>3</sup> was unlikely because of no finding of upper airway stenosis on cervical spine X-rays taken before extubation. Therefore, it was likely that UAO developed as a manifestation of PD.<sup>1</sup>

The patient had taken the last antiparkinson drugs 13 hours before anesthesia. The withdrawal of drugs might be long enough to diminish their effects, thereby causing UAO. Reportedly, duration of action of

levodopa-carbidopa to reduce motor disability lasts as short as 3 to 6 hours.<sup>4, 5</sup> After a 12-hour withdrawal of antiparkinson drugs, a significant change in the spirometry indicative of UAO occurs in a substantial number of PD patients,<sup>6, 7</sup> which is reversed by levodopa.<sup>6</sup> Especially, PD patients exhibiting laryngopharyngeal motor dysfunction, indicated mainly by hypophonia, after a 12-hour drug withdrawal have a threefold greater chance of presenting with obstructive sleep apnea (OSA) unrelated to obesity, neck circumference, or the Mallampati score, compared with those without such dysfunction.<sup>7</sup> Episodes of severe UAO that were successfully reversed by antiparkinson drugs, including levodopa, have been reported.<sup>8-10</sup> These data suggest that in our patient, daily antiparkinson drugs should have been continued perioperatively or replaced by intravenous levodopa to minimize the risk of developing UAO and subsequent NPPE.

In our patient, UAO and NPPE improved with HFNC without a resumption of antiparkinson drugs. HFNC is able to deliver a consistent oxygen supply to the alveoli by constantly applying positive pressure, enabling the patient to maintain a high level of oxygen supply.<sup>11</sup> Further, generation of PEEP helps in reduction of anatomical dead space, carbon dioxide washout, recruitment of collapsed alveoli, and ultimate improvement in oxygenation.<sup>11</sup> Through such effects, HFNC improves pulmonary edema.<sup>11</sup> In addition, HFNC improves UAO in patients with OSA.<sup>12</sup> Therefore, our experience also suggests that HFNC is effective in improving UAO and subsequent NPPE in patients with PD.

## Conclusion

We experienced upper airway obstruction and negative pressure pulmonary edema that developed immediately after anesthesia and extubation in a patient with Parkinson's disease. Our experience suggests that in patients with Parkinson's disease, antiparkinson drugs should be continued perioperatively to minimize risks of such complications, and high-flow nasal cannula therapy improves upper airway obstruction and negative pressure pulmonary edema.

## List of abbreviations

FiO<sub>2</sub>, inspired oxygen fraction; HFNC, high-flow nasal cannula therapy; NPPE, negative pressure pulmonary edema; OSA, obstructive sleep apnea; PaO<sub>2</sub>, arterial oxygen tension; PD, Parkinson's disease; PEEP, positive end-expiratory pressure; SpO<sub>2</sub>, arterial oxygen saturation; UAO, upper airway obstruction

## Consent for publication

Written informed consent was obtained from the patients for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

## Competing interests

The authors declare that they have no competing interests.

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None

## Authors' contributions

AH was a major contributor to manuscript writing. AH and NN were responsible for patient care and data collection. CM and MH contributed to data interpretation.

All authors read and approved the final manuscript.

## Figure legend

Figure 1.

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