Upper airway involvement in Parkinson's disease resulting in postoperative respiratory failure

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A 71-yr-old man with a six-year history of Parkinson's disease (PD), Type II diabetes mellitus, myocardial infarction, and remote 20 pack-year smoking history, underwent an anterior resection of the rectum for carcinoma. Sixty hours later, the patient suffered a respiratory arrest; his antiparkinsonian medications had not been resumed. Preoperative flow-volume loops showed the characteristic saw-tooth pattern of PD indicating dysfunction of the striated muscle of the upper airway. Although postoperative respiratory distress was managed as lower airway obstruction, at the time of intubation there were no signs of lower airway pathology. Upper airway dysfunction and obstruction secondary to PD is thought to have been a contributing factor to the postoperative respiratory distress and failure. This case is presented to draw attention to the risk of upper airway dysfunction in Parkinson's Disease, especially with the withdrawal of antiparkinsonian medications.

Un homme de 71 ans souffrant d'une maladie de Parkinson depuis six ans, de diabète type II, d'un infarctus du myocarde avec une histoire de tabagisme de 20 années-paquet, subit une résection antérieure du rectum pour un cancer. Soixante heures plus tard, il fait un arrêt respiratoire avant d'avoir recommencé sa médication antiparkinsonienne. Les courbes débits-volumes préopératoires montraient un tracé en dents de scie caractéristique du dysfonctionnement des muscles striés des voies aériennes supérieures consécutif au parkinsonisme. Bien que cette détresse postopératoire ait été traitée comme une obstruc-

tion des voies aériennes inférieures, on n'a pas observé de signes de cette pathologie à l'intubation. On croit que le dysfonctionnement de voies aériennes supérieures a surtout contribué à la détresse et à l'insuffisance respiratoires postopératoires. Cette observation est présentée dans le but d'attirer l'attention sur le risque de dysfonctionnement des voies respiratoires causé par la maladie de Parkinson, particulièrement après l'arrêt de la médication spécifique.

Parkinson's disease (PD) is a disorder of the extrapyramidal system resulting from the deficiency of dopamine in the basal ganglia.¹ It is characterized by bradykinesia, rigidity, rest tremour, and parasympathetic hyperactivity. Respiratory dysfunction has been noted in patients with PD and multiple causes have been proposed including impaired central control of respiration, abnormal extrapyramidal control of respiratory muscles, excessive secretions, decreased chest wall compliance, and upper airway obstruction.²⁻⁴

This report describes a patient who developed respiratory failure two days after an anterior resection of the rectum for carcinoma. Upper airway dysfunction with obstruction was considered to have had a contributory role in causing the respiratory failure.

Case report

A 71-yr-old man with a history of Parkinson's disease, Type II diabetes mellitus, myocardial infarction, and remote 20 pack-year smoking history, presented for resection of rectal carcinoma. He had undergone a remote cholecystectomy without event. His medications on admission were levodopa-carbidopa 100/25 mg tid, selegiline chlorhydrate 5 mg bid, chlorpropamide 100 mg daily, nifedipine 10 mg bid, propranolol 10 mg bid and enteric coated acetylsalicylic acid 325 mg daily. At the preoperative visit, one day before surgery, he had no complaints of dyspnoea or shortness of breath. He exercised daily on a stationary bicycle without angina. Examination of the heart and chest were unremarkable. Neurological as-

Key words

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essment found him to have stage two Parkinsonism⁵ – bilateral or midline involvement without impairment of balance. He had a soft voice with normal speech and was not hoarse. The patient had bradykinesias and cog-wheel movements at his wrists. His gait was stooped but otherwise normal. He demonstrated no tremor. His preoperative biochemistry was unremarkable except for a fasting blood glucose of $9.1 \text{ mmol} \cdot \text{L}^{-1}$. Arterial blood gas analysis while breathing room air showed pH 7.40, PCO_2 40 mmHg, PO_2 88 mmHg, and HCO_3^- $28 \text{ mmol} \cdot \text{L}^{-1}$. The ECG showed an old anterior myocardial infarction and the CXR showed a benign nodule. A multiple uptake gated acquisition scan (MUGA) performed preoperatively demonstrated septal hypokinesis and apical akinesis with moderate left ventricular dysfunction. The left ventricular ejection fraction was estimated at 50%. Pulmonary function tests revealed decreased forced expiratory volume at one second (FEV_1) 1.71 L (66% predicted) and forced vital capacity (FVC) 2.96 L (90% predicted), ($\text{FEV}_1/\text{FVC} = 72\%$) – moderate to severe flow limitation in both large and medium airways with no response to bronchodilators. The sawtooth pattern often seen in PD was noted in the flow volume loops (Figure).⁶ The patient's cardiologist and respirologist stated that the patient was in optimal condition for surgery.

On the day of surgery he received his usual medications including those for PD. The addition to the usual monitors – ECG, BP cuff, pulse oximeter and end-tidal CO_2 monitor – arterial and pulmonary artery catheters were placed. After a lumbar epidural catheter was inserted at L_{3-4} , general anaesthesia was induced with thiopentone $5 \text{ mg} \cdot \text{kg}^{-1}$, fentanyl $3 \mu\text{g} \cdot \text{kg}^{-1}$ and the trachea was intubated after with succinylcholine $1.5 \text{ mg} \cdot \text{kg}^{-1}$. Anaesthesia was maintained with isoflurane, pancuronium (7 mg total) and epidural bupivacaine 0.25% with epinephrine (20 ml total). Epidural morphine 4 mg was added for postoperative pain control. The four-hour procedure was uneventful with haemodynamic stability. Intraoperative ABG analysis with FiO_2 of 0.3 showed pH 7.47, PCO_2 35 mmHg, PO_2 127 mmHg, and HCO_3^- $28 \text{ mmol} \cdot \text{L}^{-1}$. The estimated third space and blood loss of 600 ml were replaced with 4 L Ringer's lactate. Urine output was 400 ml.

At the end of the procedure, muscle relaxation was reversed with neostigmine 3 mg and glycopyrrolate 0.6 mg. The patient was given naloxone 0.1 mg for apnoea and was transferred to the recovery room with the trachea intubated, breathing spontaneously with a t-piece circuit until more fully awake. On arrival in the recovery room, his vital signs were BP 120/70 mmHg, HR 48 bpm, RR 16 breaths $\cdot \text{min}^{-1}$ and SpO_2 98% (FiO_2 0.4). The trachea was extubated four hours after surgery, when the patient was awake and pain-free. Postoperative CXR

FIGURE Flow-volume loop showing sawtooth pattern.

showed clear lung fields, with ABG, with an FiO_2 of 0.4 showing pH 7.41, PCO_2 50 mmHg, PO_2 83 mmHg, and HCO_3^- $30 \text{ mmol} \cdot \text{L}^{-1}$. The postoperative 12 lead ECG was unchanged. His SpO_2 remained at 94–98% with RR 12–24 over the next 36 hrs. No respiratory difficulties were noted and the chest was clear to auscultation. The patient was described as alert and comfortable with no mention of rigidity. Postoperative day one was unremarkable except for decreased urine output and an expiratory wheeze. Adequate urine output was obtained with fluid boluses as pulmonary wedge pressure was low normal (10–12 mmHg). Treatment was started with salbutamol aerosol 0.5 ml/2.5 ml normal saline q4h, with which he improved. Chest physiotherapy was intensified. Intravenous morphine PCA was started on postoperative day one (1.0 mg bolus doses with seven minute lock-out – total dose 21 mg over the next 22 hr). The patient used it well and was comfortable. That evening, he was found to have brief periods of desaturation, SpO_2 89%, associated with laboured breathing. Clinical assessment was of atelectasis with increased airway secretions and salbutamol aerosol treatments were ordered every two

hours. Forty-five hours later ABG analysis with FiO_2 of 0.40, was pH 7.39, PCO_2 51 mmHg, PO_2 135 mmHg, and HCO_3^- 30 mmol $\cdot$ L $^{-1}$, he was noted to be restless with mild tachypnea. Early in the morning, on the second postoperative day the patient began to experience increased dyspnoea, agitation, and diaphoresis. There was no mention of stiffness or tremour. The trachea was intubated. Before intubation ABG analysis showed pH 7.10, PCO_2 109 mmHg, PO_2 33 mmHg, and HCO_3^- 33 mmol $\cdot$ L $^{-1}$ and immediately after intubation, FiO_2 of 1.0, pH was 7.26, PCO_2 63 mmHg, PO_2 56 mmHg and HCO_3^- 28 mmol $\cdot$ L $^{-1}$. Although the respiratory failure was believed to be due to lower airway obstruction (secretions, atelectasis, asthma) immediately after the intubation, the chest was clear without wheezing. There was no increased airway pressure or decreased compliance. Scant secretions were noted when the trachea was suctioned. Although mild hypotension followed morphine for sedation (BP 75/50 mmHg) and the patient required blood pressure support with dopamine 5 mg $\cdot$ kg $^{-1}$ $\cdot$ min $^{-1}$ for one hour, there was no evidence of cardiac ischaemia or arrhythmias preceding or following the event. Pulmonary wedge pressures remained at 12–14 mmHg and his cardiac output was 6.07 L $\cdot$ min $^{-1}$ immediately after his respiratory arrest. The lack of bronchospasm, secretions or pulmonary oedema and rapid improvement after intubation suggested that there was involvement of the upper airway. He was rapidly weaned to spontaneous ventilation with pressure support. The episode was investigated with CXR, $\dot{V}/\dot{Q}$ scan, ECG, electrolytes and cardiac enzymes, and echocardiography. There was no evidence of pulmonary oedema, infection, or pulmonary embolism. Cardiac enzymes did increase to CPK 495 U $\cdot$ L $^{-1}$ with CKMB 55 U $\cdot$ L $^{-1}$, and MB fraction of 0.11. The 2-D echocardiogram performed that day showed good overall systolic function with mild diastolic dysfunction and no change from the preoperative study. Parkinsonian medications which had been discontinued for 60 hr were restarted. The trachea was extubated two days later and the patient was transferred to a convalescent hospital after 42 days following a course complicated by atrial fibrillation and congestive heart failure.

Discussion

Parkinson's disease is an important cause of morbidity in the elderly and the implications of PD to the anaesthetist have been reviewed.⁷ However, involvement of the upper airway and its perioperative importance has not been addressed.

Respiratory abnormalities have been noted in patients with PD since the first description in 1817, especially obstructive airway defects. Respiratory complications, in particular aspiration pneumonia, are the most common

causes of death.⁴ Although some authors have found evidence of obstructive respiratory defects which increase with the degree of neural impairment,² others have found the incidence to be comparable with elderly patients without PD.³ Vincken *et al.*⁴ implicated the upper airway as the primary site of involvement. They examined 27 patients with extrapyramidal disorders with flow-volume loops and fibreoptic visualization. Twenty-four had abnormal loops characterized by regular or irregular oscillations. This "saw-tooth" pattern has been noted in PD by others^{6,8} and is also seen in patients with sleep apnoea. Direct fibreoptic visualization of the upper airway showed these oscillations to be due to involuntary movements of the glottis and supraglottic structures causing intermittent obstruction. Ten patients were disabled by this obstruction and four had stridor or dyspnoea. The effect of antiparkinsonian medication on the function of the upper airway has been studied.⁹ Ten patients with Parkinson's disease performed pulmonary function tests both on and off medication. They confirmed that patients with PD often have impaired maximum flows and pressures especially during rapid respiratory manoeuvres. Medication improved both inspiratory and expiratory effort. Vincken *et al.*¹⁰ report a patient with PD with reversible upper airway obstruction after treatment with levodopa. Flow oscillations persisted despite improvement of respiratory symptoms and flow rates. They suggested that patients with this oscillatory pattern may present with recurrent episodes of respiratory distress.

The patient we describe was asymptomatic before surgery, yet showed moderate to severe limitation in both inspiratory and expiratory volumes on the flow-volume loops. He denied dyspnoea or upper airway symptoms yet showed distinctive oscillations. This pattern may be seen in patients with PD with and without medication. He had not received any antiparkinsonian medication for 60 hr; the serum half-life of levodopa being one to three hours.¹¹ We believe that the acute withdrawal of his medication caused further dysfunction of the upper airway musculature which led to dyspnoea and subsequent respiratory failure. Other contributory factors were his diminished respiratory reserve before surgery and postoperative fluid shifts which required increased work of breathing. We consider that cardiac ischaemia, congestive heart failure, epidural opioids and PCA morphine were unlikely to have been factors. The ability to return to spontaneous respiration was against the diagnosis of chest wall muscle fatigue. The rapid resolution of symptoms by intubation without evidence of lower airway obstruction also suggests upper airway dysfunction.

We present this case to bring attention to the risk of upper airway dysfunction and obstruction in PD especially with the withdrawal of antiparkinsonian medica-

tion. A detailed preoperative assessment of pulmonary symptoms such as shortness of breath, dyspnoea, asthma or stridor is helpful but patients may be asymptomatic. There is little correlation between severity of the disease and degree of upper airway dysfunction; this patient has mild disease. Flow-volume loops with saw-tooth oscillations indicate upper airway involvement. There has been no study of these patients and postoperative outcome. The effects of intubation or specific anaesthetic agents are unknown. Patients should continue taking their antiparkinsonian medications on the morning of surgery and resume them as quickly as possible postoperatively. Upper airway dysfunction presenting as respiratory distress should be considered in patients with PD not responding to the usual management.

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