J Anesth (1999) 13:56–59

Journal of
Anesthesia

Postoperative hypoxemia following sequential resection of bilateral pulmonary lesions by median sternotomy

Tsukasa Kondo[1], Taro Mizutani[2], and Shinji Takahashi[1]

Departments of [1]Anesthesiology and [2]Critical Care Medicine, Institute of Clinical Medicine, University of Tsukuba, Tsukuba, Ibaraki 305-8575, Japan

Key words: Anesthesia, Thoracic: One-lung ventilation, Postoperative hypoxemia, Surgery, thoracic: Sequential resection

Introduction

One-stage bilateral resection of multiple bilateral pulmonary lesions by median sternotomy, using sequential one-lung ventilation, is becoming common, as it may have several advantages. We report two cases of hypoxemia occurring in patients after sequential resection of bilateral pulmonary lesions.

Case reports

Patient 1

A 62-year-old, 68-kg man was scheduled for median sternotomy to resect multiple lung metastases of sigmoid colon cancer. Preoperative forced vital capacity (FVC) and forced expiratory volume in 1 s ($FEV_{1.0}$) were 2.841 (84% of predicted value) and 1.981 (73% of predicted value), respectively. An arterial blood gas analysis (ABG) under room air showed an arterial oxygen tension (PaO_2) of 71 mmHg and an arterial carbon dioxide tension ($PaCO_2$) of 39 mmHg.

General anesthesia was induced with thiamylal 5 mg·kg^{-1} and vecuronium 0.2 mg·kg^{-1}. A 37-Fr left double-lumen endobronchial tube (Bronchocath, Mallinckrodt Medical, Athlone, Ireland) was placed, and proper position was confirmed by auscultation and fiberoptic bronchoscopy. Anesthesia was maintained with nitrous oxide 0–50%, oxygen, and isoflurane 0.5%–2.0%, and with thoracic epidural block, using 5–6 ml of 1.5% lidocaine with epinephrine (5 μg·ml^{-1}) every 50 min. Sequential one-lung ventilation was performed during the surgery, and two-lung ventilation was performed every 1 h. Five metastatic lesions in the right lung and two in the left were removed sequentially by wedge resection. During the resection of the right lower lobe, while right one-lung ventilation was performed at a fractional inspired oxygen concentration (FIO_2) of 0.7, the oxygen saturation measured by pulse oxymetry (SpO_2) suddenly fell from 97% to 85%. ABG showed a PaO_2 of 53 mmHg and a $PaCO_2$ of 37 mmHg at the time. In response to the two-lung ventilation at the increased FIO_2 of 1.0, PaO_2 rose to 373 mmHg. During all the surgical procedures, blood pressure (BP) ranged from 100/56 to 150/75 mmHg, depending upon surgical manipulation, although heart rate (HR) was stable (66–68 beats per min; BPM). During two-lung ventilation, tidal volume (TV) was 600 ml, and respiratory rate (RR) was 10 breaths per min. Airway pressure (peak/baseline) was 15/2 cmH_2O. During one-lung ventilation, TV and RR were 450 ml and 14 breaths per min, resulting in an airway pressure of 20/4 cmH_2O. Morphine hydrochloride 2 mg, with 0.25% bupivacaine 6 ml, was given epidurally 30 min prior to the end of the operation. The duration of the surgery was approximately 4½ h. Seventy min after the end of the surgery, spontaneous TV and RR were 300 ml and 15–20 breaths per min, despite appropriate reversal of neuromuscular blockade with neostigmine 2.5 mg plus atropine sulfate 1.0 mg given intravenously. ABG showed a PaO_2 of 243 mmHg, a $PaCO_2$ of 63 mmHg, and a pH of 7.24 at an FIO_2 of 1.0, indicating impaired oxygenation. Therefore the Bronchocath was replaced by a single-lumen tracheal tube (internal diameter; ID, 8.0 mm) and the patient was transported to the intensive care unit (ICU).

Mechanical ventilation was initiated, using intermittent mandatory ventilation (IMV) mode (ten breaths per min) at an FIO_2 of 0.6 and TV of 450 ml. ABG

Address correspondence to: T. Mizutani
Received for publication on April 30, 1998; accepted on September 21, 1998

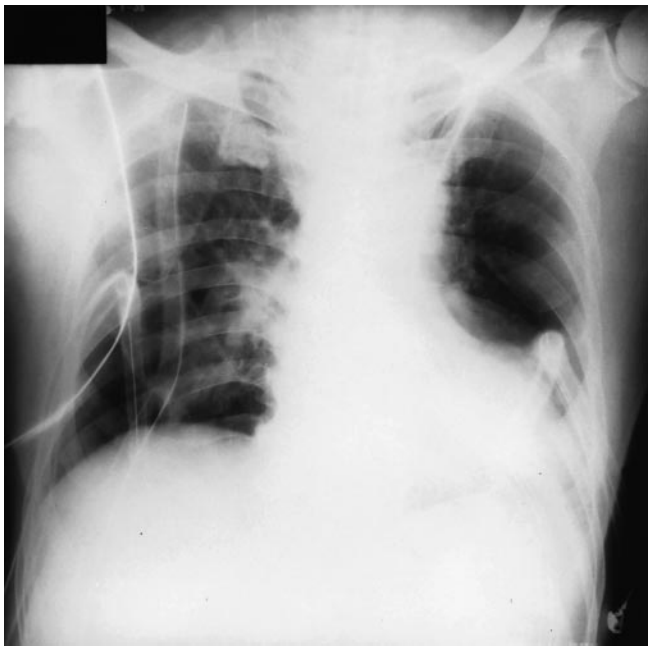

Fig. 1. Immediate postoperative chest radiograph in patient 1, showing small, ill-defined, fluffy infiltrates in the right lung and reduced aeration in the left lower lobe. No apparent atelectasis was observed (note the visible left hemidiaphragm contour)

showed a PaO_2 144 mmHg, a $PaCO_2$ 41 mmHg, and a pH of 7.33 under this condition. The immediate postoperative chest radiograph showed small, ill-defined, fluffy infiltrates in the right lung and reduced aeration in the left lower lobe. However, apparent atelectasis was not observed (Fig. 1).

Oxygenation and ventilation in the patient improved gradually over the next 15 h. Subsequently, he was successfully weaned from mechanical ventilation on postoperative day (POD) 1. An ABG just prior to extubation demonstrated a PaO_2 of 85 mmHg and a $PaCO_2$ of 38 mmHg while he was breathing spontaneously, at an FIO_2 of 0.4. The patient's subsequent recovery was uncomplicated and he was discharged on POD 33.

Patient 2

A 56-year-old, 60-kg man was scheduled for median sternotomy to resect bilateral multiple lung metastases of rectal cancer. Preoperative FVC was 4.111 (116% of predicted value) and $FEV_{1.0}$, 3.221 (85.5% of predicted value). A room air ABG showed a PaO_2 of 94 mmHg and a $PaCO_2$ of 38 mmHg. General anesthesia was induced with thiamylal 3 $mg \cdot kg^{-1}$, fentanyl 3 $\mu g \cdot kg^{-1}$, and vecuronium 0.2 $mg \cdot kg^{-1}$. A 37-Fr left Bronchocath was placed, and proper position was confirmed by auscultation and fiberoptic bronchoscopy. Anesthesia was maintained with nitrous oxide 0–50%, oxygen, and enflurane 0.2%–1.5%, and thoracic epidural block, using 3–6 ml of 1.5% lidocaine with epinephrine (5 $\mu g \cdot ml^{-1}$) every 50 min. The ventilator was set at a TV of 9.5 $ml \cdot kg^{-1}$ and an RR of 10 breaths per min. After median sternotomy, partial resection of both lungs was performed sequentially with one-lung ventilation. FIO_2 was 1.0 during one-lung ventilation. SpO_2 ranged from 96% to 99% throughout the surgery. Bilateral lung ventilation was performed three times during the surgery. The duration of the operation was approximately 5 h. BP remained at 90/56–150/78 mmHg during the surgery. Immediately after the surgery, an ABG revealed a PaO_2 of 104 mmHg and a $PaCO_2$ of 72 mmHg at an FIO_2 of 1.0 while the patient was allowed to breathe spontaneously, after reversal of neuromuscular blockade with atropine sulfate 1.0 mg and neostigmine 2.5 mg given intravenously. Spirometry revealed a spontaneous TV of 190 ml and RR of 36 breaths per min, showing that the patient had rapid shallow breathing as well as severely impaired oxygenation. Therefore the Bronchocath was removed and a single-lumen tracheal tube (ID, 7.0 mm) was placed nasally. The patient was then transported to the ICU. The ventilator was set at an FIO_2 of 0.6, TV of 450 ml, and positive end-expiratory pressure (PEEP) of 5 cmH_2O, using IMV mode at an RR of 12 breaths per min. The immediate postoperative chest radiograph showed no specific abnormal findings. After an 11-h stay in the ICU, an ABG showed a PaO_2 of 160 mmHg and $PaCO_2$ of 48 mmHg while the patient was breathing spontaneously at an FIO_2 of 0.4 and continuous positive airway pressure (CPAP) of 5 cmH_2O. The tracheal tube was removed soon after. The patient's recovery was uneventful and he was discharged on POD 15.

Discussion

One-stage bilateral resection of metastatic pulmonary lesions by median sternotomy, using sequential one-lung ventilation, is often performed, because it enables faster recovery of pulmonary function than a lateral thoracotomy, thereby reducing the patient's hospital stay [1]. However, our two patients illustrate some of the problems that can occur immediately after sequential one-lung ventilation. Antognini and Hanowell [2] reported two cases of severe hypoxia occurring during median sternotomy for sequential resection of pulmonary metastasis. In their two patients, postoperative recovery was uncomplicated. In our two patients, hypoxemic episodes occurred mainly after the surgery, although patient 1 had a temporary hypoxemic episode during the surgery.

We speculate that these postoperative hypoxemic episodes resulted chiefly from the extensive surgical manipulation of both lungs. Local pulmonary injury may release vasoactive substances, such as prostaglandins, that interfere with hypoxic pulmonary vasoconstriction (HPV) [3–5]. Disturbed HPV increases intrapulmonary shunt, causing of hypoxemia of varying degrees of [6]. Further, interstitial pulmonary edema, due to surgical manipulation, may also contribute to the postoperative hypoxemia. Thus, extensive surgical manipulation of both lungs exerted sequentially in close succession could result in severe postoperative hypoxemia. In an awake sheep model, it was shown that unilateral lung ischemia-reperfusion injury caused hypoxemia after reperfusion [7]. Although the surgical procedures performed in our patients did not invariably constitute complete ischemia of the unilateral lung, it is probable that some ischemic damage was incurred by the lung because of the surgical manipulation. In our two patients, the hypoxemic events occurred chiefly after the initiation of two-lung ventilation following the discontinuation of one-lung ventilation after surgery. Therefore, ischemia-reperfusion injury of the lung may also have been involved in the mechanism of postoperative hypoxemia in these patients.

Other less likely causes could have contributed to these postoperative hypoxemic events. Epidurally administered opiates such as morphine and fentanyl can suppress respiratory drive, leading to hypoventilation with or without hypoxemia [8]. However, in our two patients, the dose of morphine was relatively small and the primary problem was not hypoventilation but impaired oxygenation. Therefore, it is inconceivable that epidurally administered morphine was the primary cause of hypoxemia in these patients. Pulmonary embolism, which often leads to severe hypoxemia, was also unlikely. Pulmonary embolism frequently manifests in association with circulatory shock, electrocardiogram (ECG) abnormality, and abnormal blood chemistry [9]. Neither of our patients demonstrated these signs, and the hypoxemia was alleviated without specific treatments for thromboembolism. Re-expansion pulmonary edema is caused by large negative intrathoracic pressure after pulmonary re-expansion [10], increased permeability of pulmonary vasculature [11], obstruction of the bronchus [12], disappearance of the surfactant [13], and a variety of other reasons. However, there were no signs of pulmonary edema, such as cough, bubbly sputum, or tachycardia in our patients.

In small mammals, such as rats, 100% oxygen used continuously for several days causes severe pulmonary toxicity [14], possibly from the intrapulmonary production of superoxide and H_2O_2 [14–16]. In primates, although hyperoxia does not affect normal lungs, which have enough enzymes to keep oxygen radicals from accumulating, hyperoxia can damage the endothelial cells of previously damaged lungs. In humans, although an alveolar oxygen tension of less than 350 mmHg ($FIO_2 < 0.6$) does not significantly damage parenchyma, depending on the product of FIO_2 and the duration of inspiration, an alveolar oxygen tension of more than 250 mmHg ($FIO_2 > 0.5$), can further disrupt previously damaged parenchyma [17]. In our patients, the duration of $FIO_2 > 0.5$ was relatively long. Thus, high FIO_2 may have contributed to the lung injury resulting in postoperative hypoxemia, although it may not have been the primary causative factor.

Accordingly, to minimize the possibility of postoperative hypoxemia occurring after sequential resection of bilateral pulmonary lesions using sequential one-lung ventilation, various preventive measures may be undertaken. First, to reduce lung injury, the extent and duration of the surgical manipulation should be minimized. Second, to prevent postoperative atelectasis and to shorten the ischemic period of the lung, two-lung ventilation should be interposed as often as possible. Third, to avoid possible oxygen toxicity, FIO_2 should be kept as low as possible unless hypoxemic events ensue.

In conclusion, anesthesiologists should be aware that postoperative hypoxemia may occur after sequential resection of bilateral pulmonary lesions by median sternotomy using sequential one-lung ventilation.

References

1. Johnston MR (1983) Median sternotomy for resection of pulmonary metastases. J Thorac Cardiovasc Surg 85:516–522
2. Antognini JF, Hanowell LH (1991) Intraoperative hypoxemia complicating sequential resection of bilateral pulmonary metastases. Anesthesiology 74:1137–1139
3. Benumof JL (1995) Special physiology of the lateral decubitus position, the open chest and one-lung ventilation. In: Benumof JL (ed) Anesthesia for thoracic surgery 2nd edn. WB Saunders, Philadelphia, pp 123–151
4. Piper P, Vane J (1971) The release of prostaglandins from lung and other tissues. Ann N Y Acad Sci 180: 363–385
5. Benumof JL (1986) Isoflurane anesthesia and arterial oxygenation during one-lung ventilation (editorial). Anesthesiology 64:419–422
6. Benumof JL (1995) Conventional and differential lung management of one-lung ventilation. In: Benumof JL (ed) Anesthesia for thoracic surgery, 2nd edn. WB Saunders, Philadelphia, pp 406–431
7. Wickersham NE, Johnson JJ, Meyrick BO, Gilroy RJ, Loyd JE (1991) Lung ischemia-reperfusion injury in awake sheep: Protection with verapamil. J Appl Physiol 71:1554–1562
8. Bailey, Stanley (1994) Intravenous opioid anesthetics. In: Miller RD (ed) Anesthesia. Churchill Livingstone, New York, pp 314–318
9. Szucs MM Jr, Brooks HL, Grossman W, Banas JS, Meister G, Dexter L, Dalen JE (1971) Diagnostic sensitivity of laboratory findings in acute pulmonary embolism. Ann Intern Med 74:161–166
10. Ziskind MM, Weil H, George RA (1981) Acute pulmonary edema following the treatment of spontaneous pneumothorax

with excessive negative intrapleural pressure. Am Rev Respir Dis 124:422–428
11. Sprung CL, Loewenherz JW, Hauser MJ (1981) Evidence for increased permeability in re-expansion pulmonary edema. Am J Med 71:497–500
12. Brennan NJ, FitzGerald MX (1979) Anatomically localized reexpansion pulmonary edema following pneumothorax drainage. Respiration 38:233–237
13. Sautter RD, Dreher WH, MacIndoe JH, Myers WO, Magnin GE (1971) Fatal pulmonary edema and pneumonitis after re-expansion of chronic pneumothorax. Chest 60:399–401
14. Freeman B, Crapo J (1981) Hyperoxia increases O_2 radical production in rat lungs and lung mitochondria. J Biol Chem 256:10986–10992.
15. Gerschman R, Gilbert BL, Nye SW (1954) Oxygen poisoning and X-radiation: A mechanism in common. Science 119:623–626
16. Stevens J, Autor A (1980) Proposed mechanism for neonatal rat tolerance to normobaric hyperoxia. Fed Proc 39:3138–3143
17. Shapiro BA, Cane RD (1981) Metabolic malfunction of the lung Noncardiogenic edema and adult respiratory distress syndrome. Surg Annu 13:271–298