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Combined propofol and thoracic epidural anesthesia for pulmonary resection in a morbidly obese patient with sleep apnea syndrome

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Introduction

Perioperative management of morbidly obese patients is a major challenge for anesthesiologists because arterial oxygenation is extremely impaired during and after surgery. Pulmonary resection may put a further burden on the already compromised lung function. In addition, sleep apnea syndrome has been shown to increase the risk of postoperative cardiorespiratory complications [1,2]. The present report describes a successful perioperative management of a morbidly obese patient undergoing pulmonary resection with a combination of propofol and thoracic epidural anesthesia.

Case report

A 46-year-old woman (height 161 cm; weight 117 kg; body mass index (BMI) 45.2 kg/m^2) was scheduled for right lower lobectomy under the diagnosis of lung cancer. Her medical history was significant for arterial hypertension and rheumatoid arthritis. Nifedipine had been prescribed for several years, and the resting arterial blood pressure on admission was 150/100 mmHg. Although loxoprofen sodium had been prescribed for arthralgia, atlanto-axial dislocation, echocardiograph (ECG) abnormalities and pulmonary lesions were not observed, all of which could have been associated with rheumatoid arthritis. Results of routine laboratory tests were within normal limits. Values of the preoperative pulmonary function test demonstrated a low vital capacity (VC 1810 ml; %VC 65.6%; forced expiratory volume in 1 s ($FEV_{1.0}$) 1700 ml; $FEV_{1.0}$% 88.1%). Preoperative arterial blood gas analysis revealed posture-dependent hypoxemia and mild alveolar hypoventilation (in the supine position P_aO_2 55 mmHg, P_aCO_2 45 mmHg; in the sitting position P_aO_2 68 mmHg, P_aCO_2 43 mmHg).

Because sleep apnea syndrome was highly suspected, we performed a nocturnal sleep study while monitoring polysomnography (Respisomnograph; Non-Invasive Monitoring Systems, Miami Beach, FL, USA), pulse oxymeter (Oxypal; Nihon Kohden, Tokyo, Japan), ECG, direct arterial pressure, central venous pressure and pulmonary arterial pressure. There were 132 apneic episodes during 6 h (11.4% of the observation period), which; according to the criteria for sleep apnea syndrome of five or more apneic episodes per hour of sleep [3], qualified the patient as having sleep apnea syndrome. Oxygen saturation level (S_pO_2) decreased from 80% to 70%–75% during sleep. Furthermore, there were several episodes of severe desaturation (S_pO_2 50%) associated with increases in heart rate, arterial blood pressure, pulmonary arterial pressure and central venous pressure, and a decrease in the ST level. Mean pulmonary arterial pressure ranged between 35 and 40 mmHg during the severe desaturation episodes.

Atropine (0.5 mg) was administered intravenously prior to the induction of general anesthesia. After 10 min preoxygenation, 100 μg fentanyl was given intravenously, followed by 150 mg propofol and 15 mg vecuronium to facilitate tracheal intubation. Immediately after placement of a 37 Fr left-sided double-lumen endobronchial tube (DLT), continuous infusion of propofol was started at a rate of 10 mg/kg per h. For the weight of the patient, we used a corrected body weight according to the equation reported by Servin et al. [4]:

corrected body weight = ideal body weight + 0.4 × (actual body weight − ideal body weight)

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The flow rate was decreased by 2 mg/kg per h every 10 min and was maintained at a rate of 6 mg/kg per h. A 10 ml aliquot of 0.25% bupivacaine was administered into the epidural space through the catheter, which was placed via the 2nd thoracic vertebral interspace on the day before the operation. This was followed by a continuous infusion of 0.25% bupivacaine (6–8 ml/h) thereafter.

Ventilation was controlled to achieve stable alveolar ventilation in the face of increased respiratory impedance. The fractional concentration of oxygen was maintained at 1.0 throughout the procedure. Tidal volume and respiratory frequency were set to 800 ml and 12 breaths/min, respectively. Monitoring included ECG, pulse oximeter, capnogram, direct arterial pressures, central venous pressure, and pulmonary arterial pressure. All were recorded on a strip chart recorder.

One-lung ventilation (OLV) was initiated after the thorax was opened and was maintained until the thorax was closed. S_pO_2 gradually declined to a minimum level, of 88%, 15 min after starting OLV. The P_pO_2 at this moment was 57 mmHg. Constant positive airway pressure (CPAP) of 5 cmH_2O was applied to the nondependent right lung, which effectively restored S_pO_2 to greater than 96% thereafter.

After completion on the uneventful operation lasting 213 min, the DLT was replaced with a single-lumen endotracheal tube (ID 8.0 mm). Then, bronchofiberscopy was performed to check the bronchial stump and to clear residual mucus and blood clots. When spontaneous breathing resumed, the propofol infusion was stopped and residual muscle relaxation was antagonized with intravenous atropine (1.5 mg) and neostigmine (2.5 mg). The patient regained responses to verbal commands approximately 5 min after stopping propofol infusion. The total dose of propofol was 2300 mg for 275 min of anesthetic management.

The patient was transferred to the intensive care unit (ICU), where the endotracheal tube was removed after confirming sufficient lung expansion with a chest X-ray, and the absence of hypoxia or hypercapnia with an arterial blood gas analysis. Values of P_pO_2 while breathing 50% oxygen via a Venturi mask on the day of operation ranged between 91 and 107 mmHg and the corresponding values of P_aCO_2 amounted to 45–47 mmHg. For postoperative analgesia, we continued epidural administration of 0.25% bupivacaine at a flow rate of 6–8 ml/h with fentanyl (10 μg/h) until the 7th postoperative day. The patient was maintained in a semirecumbent position (45°) during the initial 3 days after the operation. As a result, S_pO_2 was maintained above 90% for most of the time, even during sleep, although the incidence of apnea was 140–160 per night, 12–14% of the observation period. There were three to four desaturation episodes per night; however, the lowest value of S_pO_2 was 75%. The mean value of pulmonary arterial pressure ranged between 15 and 23 mmHg postoperatively, which was approximately 5 mmHg higher than the preoperative data. However, no further increase in pressure was observed during the episodes of apnea. For the prevention of deep vein thrombosis, a pneumatic compression–decompression device was applied to the patient's lower extremities upon entering the operating theater, followed by intravenous administration of heparin (10 000 U/day) for 3 days in the ICU. The patient stood up at the bedside on the 1st postoperative day and walked with assistance the next day. Her postoperative course was uneventful and she was discharged on the 20th postoperative day.

Discussion

Obesity is thought to be one of the risk factors predicting complications after pulmonary resection. Epstein et al. have reported that patients with postoperative cardiopulmonary complications had a significantly higher pulmonary risk index, consisting of obesity (BMI larger than 27 kg/m^2), smoking, productive cough, wheezing or rhonchi, $FEV_{1.0}$% lower than 70% and P_aCO_2 higher than 45 mmHg [5]. Because the present case had no risk factors other than an abnormal BMI of 45.2 kg/m^2, she was thought not to be at a high risk for developing postoperative pulmonary complications.

Sleep apnea syndrome has long been recognized as a typical clinical feature of morbid obesity [6,7]. Both anatomic and neuromuscular factors are responsible for upper airway obstruction during sleep. Anatomic factors include narrowing of air spaces in the pharynx because of redundant fatty tissues [6]. Neuromuscular factors comprise altered sensitivity to hypoxia and hypercapnia of the central integrating mechanism, including respiratory center output [8,9], and the balance between opposing forces against the negative intraluminar pressure created during inspiration [10]. These two factors interact with each other, together with an alteration in the mechanisms of the respiratory system, and result in impaired oxygenation [10,11]. All these mechanisms may significantly affect perioperative management of morbidly obese patients with sleep apnea syndrome. Application of nasal CPAP has been widely accepted as a therapeutic option to prevent upper airway obstruction during sleep [11,12]. However, it is often difficult to precisely titrate oxygen concentration and to humidify inspired gas [13]. Considering gravitational effects on upper airway patency [14] and the relationship between functional residual capacity and closing capacity [15], we maintained the patient in a semirecumbent position (45°) soon after she woke. As a result, the baseline S_pO_2 during sleep was kept above

90%. It is also of note that, in contrast with the previous observation that the incidence of apnea substantially increases postoperatively [16], we found a 10%–20% increase in apneic episodes, at least during the first 3 days. In addition, the incidence of desaturation episodes was comparable with the preoperative period (three to four times per night), with the lowest S_pO_2 value being higher than the preoperative data (75% *vs* 50%).

Early ambulation and avoidance of potent central depressive agents are of paramount importance in morbidly obese patients. For this purpose, a combination of propofol and continuous epidural anesthesia was extremely useful. The patient became responsive to verbal command 5 min after stopping propofol infusion and could sit on the bed 1 h after admission to the ICU. Continuous epidural analgesia with 0.25% bupivacaine enabled a reduction in the requirement for propofol and vecuronium intraoperatively, of which the total doses administered were 2300 and 28 mg, respectively. Postoperatively, epidural analgesia was effective in relieving pain associated with intensive physiotherapy and vigorous efforts to cough. Because orthostatic hypotension was not seen in the ICU, the cardiovascular effects of thoracic epidural analgesia were negligible, as described by Gelman et al. [17].

Concerning the use of propofol as a maintenance anesthetic, hypoxic pulmonary vasoconstriction (HPV) responses should be mentioned because we chose OLV during the procedure. It has been reported that none of the injectable anesthetics, including propofol, has any effect on HPV responses, while there is a possibility that volatile anesthetics impair the response [18]. Abe et al. have also reported that, during OLV, P_aO_2 values with propofol were greater than those with isoflurane and sevoflurane, and shunt fraction values with propofol were lower than those with both volatile anesthetics [19]. In our case, maintenance anesthesia with propofol was chosen for the purpose of better oxygenation during OLV and early ambulation, and was thought to contribute to the uneventful perioperative course.

Postoperative pulmonary embolism occurs in obese patients twice as often as in nonobese patients [20]. In addition to prophylactic heparin therapy and early ambulation, we applied a pneumatic compression–decompression device on the patient's lower extremities for the prevention of deep vein thrombosis and the resultant pulmonary embolism [21].

In conclusion, we experienced a case of morbid obesity with sleep apnea syndrome undergoing pulmonary resection. A combination of propofol and thoracic epidural anesthesia provided excellent perioperative respiratory conditions. Caring for the patient in a semirecumbent position with the use of continuous epidural analgesia was extremely effective in the maintenance of oxygenation and upper airway patency during sleep.

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