

Unanticipated hemothorax during general anesthesia

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Abstract We experienced a case of uncommon spontaneous hemothorax during general anesthesia. A 72-year-old woman underwent emergency repair of damaged ascending colon. Her chest roentgenogram before surgery revealed no abnormalities. After the uneventful surgery, the trachea was extubated and the patient breathed without difficulty with stable vital signs. However, the S_{PO_2} varied around 95% with a 100% oxygen mask. Chest roentgenogram obtained after extubation showed a massive amount of fluid in the pleural cavity. A chest drainage tube was inserted, and approximately 1000ml of frank blood was drawn without air leakage. Thereafter, her oxygenation remarkably improved. We could not determine the etiology of the hemothorax.

Key words Desaturation · Intrapleural problem · Hemothorax

Introduction

Intrapleural complications (i.e., atelectasis, pulmonary effusion, or pneumothorax) during anesthesia are rare, but they sometimes induce a fatal situation such as severe hypoxia or hemodynamic collapse. The presence of an intrapleural problem is suspected from patients' symptoms, physical examination, or vital signs. Although it is controversial whether routine use of a pulse oximeter improves the outcome of surgical patients, Moller et al. [1] reported that hypoxia was detected earlier in patients who were monitored by a pulse oximeter than in patients who were not. We experienced a case of symptom-free, spontaneous hemothorax in a patient undergoing surgery under general anesthesia, which was not detected by auscultation or by the patient's vital signs, but was suspected by slight oxygen desaturation after extubation.

Case report

A 72-year-old woman, with a height of 149cm and weight of 48kg, underwent emergency repair of damaged ascending colon. She had undergone laparoscopic hemicolectomy 3 weeks earlier, and had been struggling with mucous discharge from the damaged anastomosis starting 3 days after the primary operation. Her primary operation, which was performed under epidural and general anesthesia, was uneventful. Preoperative blood examination performed prior to the emergency surgery revealed the following values: WBC, $15900 \cdot \mu l^{-1}$; RBC, $284 \times 10^4 \cdot \mu l^{-1}$; hemoglobin (Hb), $8.4 \text{ g} \cdot \text{d} l^{-1}$; hematocrit (Hct), 27.5%; platelets (PLT), $51.9 \times 10^4 \cdot \mu l^{-1}$. Laboratory examination revealed the following values: total protein (TP), 5.8g·dl⁻¹; albumin (Alb), 1.8g·dl⁻¹; alkaline phosphatase (ALP), 492 IU·l⁻¹; aspartate aminotransferase (AST), 74IU·l⁻¹: alanine aminotransferase (ALT), 129IU·l⁻¹; blood urea nitrogen (BUN), 24.2 mg·dl⁻¹; creatinine (CRE), 0.3 mg·dl⁻¹. The prothrombin time (PT) and activated partial thrombin time (aPTT) were 11.7s (INR, 1.23) and 29.6s, respectively. An electrocardiogram and chest roentgenogram, obtained 5 days before the emergency operation, revealed no remarkable signs (Fig. 1). A central venous catheter had been placed uneventfully through the right subclavian vein 3 days before the primary operation (approximately 1 month before the emergency operation).

On admittance to the operating room, she had no symptoms suggesting pulmonary complications (i.e., dyspnea, shortness of breath). Her systolic blood pressure, heart rate, and oxygen saturation by pulse oximetry (S_{Po_2}) in room air were 140mmHg, $98 \cdot min^{-1}$ and 97%, respectively. Before induction of general anesthesia, an arterial catheter was placed and an arterial blood specimen was obtained. Arterial blood gas (ABG) analysis in room air showed the following values: Pa_{CO_2} , 31.4 mmHg; Pa_{O_2} , 79.4 mmHg; base excess, -0.2 mmol·l^{-1} ; Hb, 8.6 g·dl^{-1} . Anesthesia was induced

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Fig. 1. Chest roentgenogram before the emergency surgery



Fig. 2. Chest roentgenogram immediately after extubation shows massive pleural effusion

by thiopental, 200 mg IV, and 10 mg vecuronium facilitated tracheal intubation. Anesthesia was maintained by isoflurane, 0.7%–1.5%, and oxygen, 35%, in nitrous oxide mixture. Incremental administration of vecuronium induced muscle relaxation. The patients was mechanically ventilated with a tidal volume of 450 ml at a rate of 10 breaths min⁻¹, without elevation in airway pressure. Her vital signs were stable during the surgery. ABG analysis was performed several times, and the Pa_{Oa} at the beginning of the surgery was 149mmHg and that at the end of surgery was 114mmHg. The surgery was uneventful, except for blood loss of as much as 1500 ml. During anesthesia, which was extended to 7 h, four units of packed cells and six units of fresh frozen plasma were transfused. After reversal of the muscle relaxant, spontaneous ventilation began and the tidal volume increased to approximately 350ml. She emerged from anesthesia and the trachea was extubated after auscultation. She was breathing without difficulty and her vital signs were stable. However, the Spo, varied around 95% even though the patient was being administered 100% oxygen in an anesthesia mask (Inflatable Face Mask; King Systems, Noblesville, IN, USA). To observe the respiratory condition, we gave oxygen at 61·min⁻¹ via a conventional facemask instead of the anesthesia mask. The values for S_{PO_2} ranged between 93% and 94%. ABG analysis revealed the following: Pa_{CO₂}, 46 mmHg; Pa₀, 73mmHg; and Hb, 8.9g·dl⁻¹. We administered 0.1 mg buprenorphine intravenously because we thought that the desaturation was related to unsatisfactorily managed pain.

We suspected that she had an intrapleural problem, and a chest roentgenogram was taken. The chest roentgenogram taken after extubation demonstrated a massive amount of fluid in the left pleural cavity (Fig. 2). A chest drainage tube was inserted, and approximately 1000 ml of frank blood was drawn, but no air leakage was found. A blood specimen was obtained from the drainage tube, and the hemoglobin concentration was $6.7 \text{ g} \cdot \text{d} \text{l}^{-1}$. Her vital signs before and after drainage did not differ. After drainage, the ABG values remarkably improved. She went back to the surgical ward and the chest tube was removed 2 days after the surgery. In the surgical ward, no additional remarkable bleeding, or air leakage, was noted.

Discussion

Unstable oxygen saturation after extubation in a patient who is fully awake, who does not have difficulty in breathing, and with adequate tidal volume suggests the presence of atelectasis, pulmonary effusion, or other intrapleural problems. Spontaneous hemothorax is usually associated with chest trauma, and it induces abrupt symptoms, such as dyspnea, shortness of breath, tachycardia, and hypotension. Bleeding from any pleural structure causes hemothorax. One common cause of hemothorax is the rupture of an intercostal vein that has become enlarged and serpentine with age [2]. In rare cases, an aberrant artery is the bleeding source and may cause massive hemothorax [3]. Hemopneumothorax, which is seen in 0.5% of pneumothorax cases, may be induced by the tearing of an adhesion between an aberrant artery and the apex of the lung [4]. Delayed appearance or delayed diagnosis, due to any reason, leads to a fatal situation [5]. Because pleural effusion, including even a small amount of blood, is sometimes misdiagnosed as hemothorax, the Hb concentration or Hct in the fluid drained through the drainage tube is measured [2]. The majority of patients with hemothorax are conservatively treated by draining through a chest tube. In 10% of cases, thoracotomy is performed to control bleeding or air leakage.

Several cases of hemothorax that developed following a perioperative, surgical, or anesthesia procedure have been reported. Hemothorax occurred after intercostal or epidural catheterization due to accidental perforation of the intercostal vein or artery [6,7]. Cases of hemothorax after the insertion of a central venous or pulmonary artery catheter have also been reported [8,9]. Vascular erosion and right-sided pleural effusion that were diagnosed 0 to 10 days after the insertion of a central venous catheter were reported [10]. An anatomical study, using magnetic resonance imaging, suggested that the tip of the central venous catheter may impinge on the right atrium and perforate the atrium [10]. In patients with iatrogenic hemothorax, the development of hemothorax occurred over a longer period of time, taking several hours to several days [6,7,9,11].

In our patient, the cause and development of the hemothorax could not be determined. The preoperative roentgenogram obtained prior to the emergency surgery, and the postoperative course, except for mucous discharge after the primary operation, suggested that the hemothorax was not related to the anesthesia or to the surgical procedure in the primary surgery (i.e., central venous catheter placement, epidural catheterization). Because our patient had stable vital signs, no symptoms, and normal ABG values, the massive hemothorax may not have been present before the induction of general anesthesia. The patient did not have a coagulation abnormality, as the hematologic examination was normal. However, the Pa_{O2} value slowly decreased over time. During anesthesia, bleeding may have been occurring in the pleural cavity. Unfortunately, bleeding in the surgical field and the requirement for blood transfusion interrupted us from evaluating the hemodynamic consequences. In our patient, hemothorax could not be detected during and after anesthesia by auscultation, and the patient did not have any symptoms. The only sign of hemothorax was oxygen desaturation, despite the fact that the patient was administered supplemental oxygen. An oxygen desaturation value of approximately 95% immediately after major surgery is sometimes considered as borderline [12]. However, slight oxygen desaturation after emergence from anesthesia while the patient is given supplemental oxygen may suggest the presence of an intrapleural problem such as hemothorax.

In conclusion, we experienced an unanticipated and symptom-free hemothorax after surgery, which was suggested only by slight desaturation after extubation.

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