Bronchospasm-induced Massive Lung Collapse during Thoracotomy

Hiromi KATOH, Masaki WAKAMATSU, Harutaka UEMATSU*
Tokushige TANAHASHI* and Michio YAMAMOTO*

(Key words: bronchospasm, lung collapse, thoracotomy)

Bronchospasm is one type of critical complications that can occur during anesthesia. It is rare to actually observe the lung itself under bronchospasm[1-6]. There is no previous report of unilateral bronchospasm during median sternotomy in which both normal and bronchospastic lungs can be simultaneously observed. A case was presented in which unilateral lung collapse was triggered by surgical procedures on the pleura. The collapse localized on the operative side, while the contralateral lung was normally ventilated. Each lung showed the similar reaction to the surgical retraction when the surgeon altered the operating field from one side to another. In fiberoptic bronchoscopy, marked bronchial stenosis was revealed on the collapsed side of the lungs. Administration of epinephrine was most effective for this case.

Report of a Case

An 18 year-old male was admitted complaining of pain in the right side of his chest. He had a past history of recurrent pneumothorax. X-rays and chest computed tomography revealed right pneumothorax combined with both apical small bullae. Though he had suffered from asthma attacks during his childhood, none had reoccurred during the last decade. He was scheduled for surgical bullectomy. Preoperative laboratory data were normal except for mild increases in GOT, 77IU·l^{-1}, and GPT, 71IU·l^{-1}.

Premedication consisted of atropine and hydroxyzine. When the attending anesthesiologist established i.v. access, the forearm vein became spastic for a few minutes. Anesthesia was induced with diazepam 10mg and ketamine 100mg. A single-lumen endotracheal tube was intubated with succinylcholine 60mg. Manual ventilation through the tube was easy, and the breathing sound was equal in both lungs. Anesthesia was maintained with nitrous oxide, oxygen and enflurane supplemented by pancuronium as a muscle relaxant, and the patient was mechanically ventilated.

After median sternotomy, the left chest was opened. At this time, both lungs were normally ventilated. The surgeon identified some of small bullae on the left apex, and retracted the left lung to remove them. Soon after, the anesthesiologist recognized that the entire left lung could not be reinflated. Ventilation of the right lung was normal, and no increase in the peak inspiratory airway pressure was noticed. A fiberoptic bronchoscopic examination showed a severe stenosis of the left segmental bronchi. Selective intubation into the left main bronchus was carried out under bronchoscopic observation, but the collapsed lung was not expanded manually. Intravenous administration of aminophylline 250mg and hydrocortisone 1g was not effective. Inhalation of halothane

Department of Anesthesia, Chubu Rosai Hospital, Nagoya, Japan

**Department of Anesthesiology, Gifu University School of Medicine, Gifu, Japan*

Address reprint requests to Dr. Katoh: Department of Anesthesia, Chubu Rosai Hospital, 1-10-6 Komei, Minato-ku, Nagoya, 455 Japan

J Anesth 5:320–322, 1991

1.5%, for several minutes also failed to relieve the situation. Finally, epinephrine 0.2 mg, was administered subcutaneously, resulting in gradual expansion of the left lung coupled with tachycardia and hypertension. Fiberoptic bronchoscopy revealed the dilatation of the bronchial caliber except for B3, and the left apex expanded later.

Then, a right thoracotomy was performed with continuous infusion of aminophylline. At this time, the right upper lobe rapidly collapsed immediately after surgical retraction. But no apparent narrowing of the right bronchial caliber was pointed out by bronchoscopy. Epinephrine 0.2mg, was administered subcutaneously again, and was effective for reexpansion of right upper lobe.

During these episodes, we could not detect any hypoxemia by the blood-gas analysis. The operation was completed, and the patient was discharged on the 13th postoperative day without any complications. Postoperative laboratory data indicated a mild increase in the serum IgE 500IU·ml^{-1}. Serum histamine was within normal range, 3.0 ng·ml^{-1}.

Discussion

In this particular case, the problem of the lungs were triggered by the surgical manipulations and resolved by epinephrine. A fiberoptic bronchoscopic examination revealed the narrowing of the bronchial caliber during the left lung collapse. On the contrary, the collapsed right upper lobe showed no apparent bronchial stenosis. These facts strongly suggest that the collapse was caused by the constriction of small airway which was invisible by fiberoptic bronchoscopy. Moreover, the etiology of the bronchial stenosis in this case should be different from humorally activated pathway which has been similarly discussed as a cause of bronchospasm or asthma, since the collapse localized unilaterally[7].

In this case, epinephrine was finally effective for bronchospasm. Epinepherine has a powerful beta$_2$ action which stimulates adenyl cyclase to catalyze ATP to c-AMP, and impairs release of bronchoconstrictor substances. In fact, the second episode was rapidly resolved by epinephrine administration alone. Several authors discribe the effectiveness of lidcaine administration intratracheally or directly to pulmonary hilum when neurogenic bronchospasm occurred[5,8]. Although we did not carry out this mean, it should be a first choice for an attempt because lidocaine accompanies no cardiovascular actions or arrhythmias.

Several authors previously documented the appearance of the lung, in either form of hyperinflation or collapse, when bronchospasm occurred during thoracotomy[1-6]. In these reports, cases of collapsed lungs were complicated in the lateral position, and there was no information available regarding the contralateral lung. We could observe both lungs simultaneously because of median sternotomy, and recognized unilateral lung collapse restrictively. The mechanism of the lung collapse during bronchospasm, in general, is still controversial: 1) rapid absorption of trapped air, 2) a decrease in the concentration of surfactant material and 3) release of prostaglandins were proposed[1-6,8,9]. These explanations, however, cannot be completely applied to the rapid onset of the lung collapse and complete relief by epinephrine in this case. Furthermore, all previously reported cases of collapsed lungs were complicated following the surgical retraction[1-6]. Therefore, the physical stimulation of surgical manuevers should be considered as one of triggering factors. Some of the chemical stimulation may cause the lung collapse via a neural route[10]. It is unknown why the neurogenic bronchospasm results in lung collapse. If one way valve mechanism causes hyperinflation of the lung, the direction of one way valve may be altered in some stenotic bronchi, leading to the lung collapse. Our observation of the peak inspiratory airway pressure suggests increase in the lung compliance on intact, or non-operative side, although none of previous reports described such phenomenon. This may be partially due to the high compliance of the lung itself in a young patient. Further observations are

expected to investigate the altering of lung compliance on the intact side when unilateral bronchospasm occurred.

A case of intraoperative lung collapse was reported. The collapse localized on the operative side. Epinephrine was most effective for the relief of bronchial stenosis. It is suggested that even minor physical stimulation on the pleura may trigger intraoperative bronchospasm. Furthermore, there is a suspicion of an increase in the lung compliance on intact side when unilateral bronchospasm occurred.

(Received Sep. 12, 1990, accepted for publication Jan. 25, 1991)

References

1. Smolnikoff VP: Total bronchospasm and lung massage. Anaesthesia 15:40–44, 1960
2. Kyle WD: Acute pulmonary collapse – a case report. Can Anaesth Soc J 10:550–554, 1963
3. Imrie DD, McClelland RMA, Shardow WB: Massive pulmonary collapse during thoracotomy. Br J Anaesth 38:973–976, 1966
4. Coroneos NJ, Lawrence J, Torda TAG: Acute massive pulmonary collapse. Anaesthesia 25:259–264, 1970
5. Benett DJ, Torda TA, Horton DA, Wright SW: Severe bronchospasm complicating thoracotomy. Arch Surg 101:555–557, 1970
6. Shiroka A, Rah KH, Keehan RL: Bronchospasm during cardiopulmonary bypass. Anesth Analg 61:538–540, 1982
7. Hirshman CA: Airway reactivity in humans-anesthetic indications. Anesthesiology 58:170–177, 1983
8. Ree SC, Lin YS, Lin KJ, Wang MH: Reflex bronchospasm-induced acute massive pulmonary collapse. Anesth Prog 35:244–246, 1988
9. Gruenwald P, Johnson RP, Hustead RF, Grements JA: Correlation of mechanical properties of infant lungs with surfactant activity of extracts. Proc Soc Bio Med 109:369–371, 1962
10. Armour CL, Black JL, Johnson PRA: A role for inflammatory mediators in airway hyperresponsiveness. Alan R, Liss Inc, 1988, pp. 99–108