Pulmonary Edema after Anesthesia-Related Laryngospasm

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(Key words: laryngospasm, pulmonary edema)

There have been recent reports of pulmonary edema (PE) complicating anesthesia-related laryngospasm (LS)[1-9], and most of them were anesthetized with volatile anesthetics[2-7,9]. We have also had this experience in a patient anesthetized with nitrous oxide in oxygen, supplemented with fentanyl. We describe herein the clinical events and discuss the mechanisms of PE complicating anesthesia-related LS.

Case Report

An 18-year-old Japanese boy with bilateral retrolental fibroplasia and retinal detachment was scheduled for vitreous repair of the left eye. Preoperative physical examinations revealed no heart murmurs or adventitious respiratory sounds. Chest x-ray showed no evidence of bronchopulmonary dysplasia, and the cardiothoracic ratio was 0.42. The serum protein was 7.1 g/dl, the albumin 4.1 g/dl. Other laboratory investigations, including electrocardiogram, were all normal.

He was premedicated with pentobarbital 100 mg and diazepam 10 mg, orally, and atropine 0.5 mg was administered subcutaneously 30 minutes before arrival in the operating room. The preanesthetic arterial blood pressure was 130/80 mmHg, and the heart rate 85 beats/min. Anesthesia was induced with droperidol 7.5 mg, fentanyl 0.1 mg, and thiopental 150 mg, given intravenously. After the intravenous administration of succinylcholine 60 mg, the trachea was easily intubated with a 7.5 mm cuffed Rae tube, and fentanyl 0.3 mg was given. Anesthesia was maintained with 66% nitrous oxide in oxygen. Pancuronium bromide was used for intraoperative muscle relaxation, and ventilation was controlled manually. During the two hours of surgery, the arterial blood pressure and heart rate remained stable, and he received a total of 6 mg of pancuronium and 1,000 ml of 5% dextrose in lactated Ringer's solution. At the end of the surgery, a moderate degree of spontaneous respirations was already noted, and neostigmine 2.5 mg mixed with atropine 1.0 mg was administered to antagonize the residual effect of pancuronium. He was ventilated with oxygen, and the trachea was extubated.

Immediately after extubation, intense LS developed. He could not be ventilated, despite a prompt application of positive airway pressure by bag and mask. Perioral cyanosis rapidly developed, and the heart rate transiently decreased to 60 beats/min. Succinylcholine 60 mg was administered intravenously, after which he could be easily ventilated, and the trachea was re-intubated. The heart rate increased to 70 beats/min. However, massive frothy pink fluid poured out from the endotracheal tube immediately after re-intubation. Moist rales were heard over both lung fields. An arterial blood gas analysis done 20 minutes

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J Anesth 1: 199–202, 1987

after re-intubation revealed a pH of 7.20, Pa_{O_2} 91 mmHg, Pa_{CO_2} 66 mmHg, and a base excess of −5.0 mmol/L, during which time he was ventilated manually with pure oxygen. The lungs were then mechanically ventilated with an $F_{I_{O_2}}$ of 1.0, respiratory rate 15, tidal volume 600 ml, and with an application of positive end-expiratory pressure (PEEP) of 10 cmH_2O. A balloon tipped pulmonary artery catheter inserted 45 minutes after the development of PE revealed a pulmonary artery pressure (PAP) of 36/15 mmHg and a pulmonary artery wedge pressure (PAWP) of 15 mmHg. One hour and 15 minutes after the development of PE, the PAP was 22/15 mmHg, the PAWP 8 mmHg, the central venous pressure 7 cmH_2O, and the cardiac index was 3.42 $L/min/m^2$. The serum protein was 6.5 g/dl. A chest x-ray taken at that time showed a diffuse shadow in the right middle lung field and scattered shadows in the left lung field. The cardiothoracic ratio was 0.40. One hour and 40 minutes after the development of PE, an analysis of arterial blood, with an $F_{I_{O_2}}$ of 1.0 and a PEEP of 10 cmH_2O, revealed a pH of 7.39, Pa_{O_2} 443 mmHg, Pa_{CO_2} 37 mmHg, and a base excess of −2.5 mmol/L. Thereafter , the $F_{I_{O_2}}$ and the level of PEEP were gradually decreased. Nine hours after the onset of PE, the lungs became clear on auscultation, and no fluid could be suctioned from the trachea. An arterial blood gas analysis, with an $F_{I_{O_2}}$ of 0.3 and a continuous positive airway pressure of 7 cmH_2O, revealed a pH of 7.31, Pa_{O_2} 146 mmHg, Pa_{CO_2} 42 mmHg, and a base excess of −4.4 mmol/L. He was extubated, and oxygen-enriched air was administered via a face mask.

A chest x-ray taken the next morning revealed no infiltrative shadow in either lung field. An arterial blood gas analysis on room air disclosed a pH of 7.42 Pa_{O_2} 88 mmHg, Pa_{CO_2} 44 mmHg, and a base excess of +3.1 mmol/L. No other untoward events occurred. An echocardiography performed postoperatively revealed no abnormality in the heart.

Discussion

Since the 1980s, there have been at least 12 cases of PE after anesthesia-related LS. This case illustrates the potential of development of PE after LS even in patients anesthetized with narcotics that are believed to blunt the larynx to mechanical stimuli.

The mechanisms related to PE are considered to be an increase in hydrostatic pressure secondary to increase in the left atrial pressure (cardiogenic) or enhanced permeability of the pulmonary capillaries[10]. Some[6,7] suggested that the mechanism of PE after anesthesia-related LS might be cardiogenic in origin, because an increase in the negative intrapleural pressure, which inevitably accompanies LS, can bring about a decrease in the fraction of blood ejected from the left venticle of the heart[11]. This, in turn, might elevate the left atrial pressure up to the level causing PE. However, a definite decrease in cardiac output is not observed even in cardiac patients having nearly normal left ventricular function with increase in the negative intrapleural pressure up to −60 cmH_2O, though a decrease in the fraction of blood ejected from the left ventricle has been documented[12]. It might be possible that such a transient and slight decrease in the fraction of blood ejected from the left ventricle leads to a florid PE in those with markedly impaired left ventricular function, however, none of the patients thus far reported, including ours, had any clinically apparent cardiac disease[1−9]. The cardiovascular hemodynamics in our patient showed a PAP of 36/15 mmHg and a PAWP of 15 mmHg 45 minutes after the onset of PE. These increaes might suggest the presence of left ventricular dysfunction, however, these magnitudes of increases in the PAP and the PAWP are usual to occur at the time of laryngoscopy and intubation and never develop PE in subjects without apparent cardiac disease[13]. These lines of evidence make it difficult to attribute PE after anesthesia-related LS to, if any, a

direct increase in left atrial pressure.

LS is liable to occur when extubation is not carefully done shortly after discontinuation of volatile anesthetics, however, PE does not usually develop. On the contrary, LS that was associated with the development of PE occurred at following periods: immediately after extubation performed following administration of a full dose of antagonizing agent to reverse the residual neuromuscular paralysis when a non-depolarizing neuromuscular blocking agent was concurrently used with volatile anesthetics[2–4,6]; immediately after extubation done at an almost complete recovery phase from anesthesia when it was maintained solely with a volatile anesthetic[7]; at emergence from anesthesia (and immediately before the completion of surgery) when anesthesia was maintained with a volatile anesthetic by mask[2]; shortly after the induction with a volatile anesthetic[9]; and at the time of multiple-failed attempts to intubate the trachea after administration of succinylcholine[1]. Our case also developed LS immediately after extubation following administration of an apparently full dose of neostigmine. Therefore, LS developed at the time where an almost full muscular strength is assumed to have been regained or remained in all these patients. These circumstantial evidences strongly suggest that full muscular strength plays a crucial role in the development of PE after anesthesia-related LS.

With full muscular strength, an extremely negative intra-alveolar pressure, though transient, appears to be generated during the first inspiratory effort against a closed glottis. This extreme negative intra-alveolar pressure may be transmitted to the pulmonary interstitium and, in turn, lead to an extremely negative pressure around the pulmonary capillaries, by which the integrity of the pulmonary capillary vessels might be disrupted. Hence, there would be extrusion from the capillaries into the interstitium and, in turn, eventually into the alveolus. Thus, the PE after anesthesia-related LS seems to be a barotrauma to the lungs caused by an extremely negative intra-alveolar pressure.

There was one patient in whom signs and symptoms of PE developed 105 minutes after the onset of LS, during which time she had not received oxygen[9]. Although there is the traditional idea that alveolar hypoxia, per se, can produce PE[14], this proposal is not supported by careful animal experiments[15,16]. However, the persistence of hypoxia can elevate the PAP via hypoxic pulmonary vasoconstriction[16] and release of catecholamines[17]. An elevation of the PAP can produce extrusion of fluid from the pulmonary capillaries into the interstitium and into the alveolus[16] and aggravates the signs and symptoms of PE[18], once the integrity of the pulmonary capillary vessels is lost. Therefore, if a patient develops LS during or after anesthesia, oxygen-enriched air should be given to avoid the development of hypoxia.

Some advocate that when LS cannot be relieved promptly by positive pressure with bag and mask must be relieved with succinylcholine to prevent the development of PE[2,3,7]. However, as PE after LS may be caused by the extremely negative intra-alveolar pressure generated at the time of the onset of LS, even such an early administration of succinylcholine may not prevent the development of PE, unless it is given before the first inspiratory effort of LS.

(Received Oct. 22, 1987, accepted for publication May 29, 1987)

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