Pulmonary Edema Induced by Laryngospasm

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Although pulmonay edema has many causes, acute airway obstruction is rarely mentioned as one of them because of its infrequent occurrence. We describe a case of laryngospasm, which was complicated by the development of pulmonary edema. We discuss the pathophysiologic consequences of acute airway obstruction leading to this complication.

Case Report

A 63-year-old female, weight 62 kg and height 143 cm, was scheduled for a radical operation to correct an umbilical hernia. No respiratory or circulatory abnormalities were noted. The preoperative chest X-ray was clear and the electrocardiogram was normal. Serum protein was 5.9 $g \cdot dl^{-1}$ and serum albumin 3.2 $g \cdot dl^{-1}$. Other laboratory findings were normal. The patient was premedicated with atropine 0.5 mg, hydroxyzine 50 mg, and ranitidine 50 mg given intramuscularly 1 hour before the operation. Anesthe-

sia was induced with thiamylal 250 mg, vecuronium bromide 8 mg, and fentanyl 0.1 mg, given intravenously. The trachea was easily intubated with a 7.5 mm cuffed Rüsch tube. Anesthesia was maintained with 1 ~ 3% isoflurane and 66% nitrous oxide in oxygen. She received 750 ml of lactated Ringer's solution during the 43 min procedure. Analysis of arterial blood gases, with an F_{IO_2} of 0.33, revealed a pH of 7.41, Pa_{O_2} 110.3 mmHg, Pa_{CO_2} 39.1 mmHg, and base excess of 0.7 $mmol \cdot l^{-1}$. At the end of the operation, the patient's arterial pressure rose to 190/110 mmHg, and diltiazem, 4 mg, was administered. The residual neuromuscular blockade was reversed with atropine 1.0 mg, and neostigmine 2.5 mg, given intravenously. She was ventilated with oxygen, and the endotracheal tube was removed. Severe laryngospasm developed immediately after extubation. The patient could not be ventilated, and she rapidly became cyanotic. The heart rate decreased from 96 $beats \cdot min^{-1}$ to 64 $beats \cdot min^{-1}$, the blood pressure remained at 180/100 mmHg. After the administration of succinylcholine, 60 mg, an endotracheal tube was finally passed, despite the presence of severe laryngeal edema. About 7 to 8 minutes after the onset of cyanosis, the laryngospasm decreased sufficiently to allow oxygenation of the lung. Analysis of arterial blood gases following re-

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intubation revealed a pH of 7.17, Pa_{O_2} 17.2 mmHg, Pa_{CO_2} 72.3 mmHg, and base excess of -4.4 mmol$\cdot l^{-1}$. Moist rales were heard over both lung fields. Arterial blood gases 5 minutes after re-intubation revealed a pH of 7.15, Pa_{O_2} 73.2 mmHg, Pa_{CO_2} 71.5 mmHg, and base excess of -6.0 mmol$\cdot l^{-1}$, during which time the patient was ventilated manually with pure oxygen. The lungs were then mechanically ventilated with an $F_{I_{O_2}}$ of 1.0, respiratory rate 12 min^{-1}, tidal volume 550 ml, and with the application of positive end-expiratory pressure (PEEP) of 7 cmH_2O. Analysis of arterial blood gases 20 minutes after reintubation revealed a pH of 7.23, Pa_{O_2} 134.2 mmHg, Pa_{CO_2} 54.3 mmHg, and base excess of -5.5 mmol$\cdot l^{-1}$. At that time the chest X-ray demonstrated diffuse alveolar infiltration bilaterally. The diagnosis was pulmonary edema. Digoxin and furosemide were administered intravenously. A postoperative echocardiogram revealed no cardiac abnormalities, and the patient recovered gradually. On the fifth postoperative day, the chest X-ray improved markedly, and the arterial blood gases were normal on room air. She was then extubated. She made a full recovery.

Discussion

This patient developed a severe pulmonary edema following a relief of upper airway obstruction. We believe the pulmonary edema was produced by the airway obstruction since there was no history of chest infection, and excessive amounts of fluid were not administered before or during the operation. Echocardiogram suggested that this patient had developed noncardiogenic pulmonary edema. There was no hypoalbuminemia. Another possible cause of the pulmonary edema is an allergic reaction. Although the patient was given general anesthesia again one year later, and almost the same drugs

as before were administered, both the laryngospasm or the pulmonary edema did not occur. This fact helps us think that the pulmonary edema of this patient was not an allergic reaction.

While there are various causes of pulmonary edema following laryngeal obstruction, a markedly negative intrapleural pressure may be the primary pathological event. During normal breathing, the pleural pressure at inspiration is -2.5 to -10 cmH_2O. This pressure changes considerably in the presence of upper airway obstruction. During forceful inspiration against an obstructed airway, the intrapleural pressure can fall below -30 to -60 cmH_2O[1]. These extreme negative pressures can damage the capillary walls[2].

Negative pleural pressure places an afterload stress on the left ventricles. Peters et al.[3,4] studied the independent effects of applying negative pleural pressure during isolated systolic and diastolic events in dogs. The application of negative pleural pressure during systole exposed the left ventricle to an afterload stress, when afterload was expressed as the transmural aortic pressure (i.e., mean aortic pressure minus pleural pressure[5]). This caused a decrease in left ventricular stroke volume and resulted in increases in both ventricular end-systolic and end-diastolic volumes[6].

Negative pleural pressure also increases venous return to the right heart. Increased venous return to the right heart, by means of ventricular independence, decreases the left ventricular compliance and elevates the left ventricular end-diastolic pressures. This reduces the left ventricular stroke volume[7], leading to a rise in the pulmonary blood volume and microvascular pressure[3,4]. A low pulmonary vascular compliance and a mechanically induced increase in pulmonary capillary permeability develop this rise in

pulmonary microvascular pressure[8].

Hypoxia and a hyperadrenergic state each contribute to the development of pulmonary edema. Hypoxia and the resulting hyperadrenergic state promote the formation of pulmonay edema by 1) the translocation of blood from the systemic to the pulmonary circulation[9], 2) a generalized increase in pulmonary vascular resistance[10-12], and 3) an increase in pulmonary capillary permeability[13-15]. By depressing the myocardial performance, hypoxia and the resulting metabolic acidosis aggravate the tendency to develop pulmonary edema formation[16].

In conclusion, considering the potentially serious consequences of pulmonary edema, patients who recover from an acute upper airway obstruction should be closely monitored for the management of this complication.

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