Intensive Care Med (1995) 21:1048–1050

BRIEF REPORT

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Postobstructive pulmonary edema induced by endotracheal tube occlusion

Received: 14 June 1994
Accepted: 21 December 1994

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Abstract Pulmonary edema is a well-described complication of upper airway obstruction, most commonly caused in adults by postanesthetic laryngospasm. The mechanism initiating the formation of postobstructive pulmonary edema is believed to be the markedly negative intrapleural pressure generated by a forceful inspiratory effort against an obstructed extrathoracic airway. We herein describe a young, male patient who developed pulmonary edema postoperatively, upon emergence from anesthesia, after performing repeated, forceful inspiratory maneuvers directed against an endotracheal tube on which he had bitten down, thereby occluding it. To our knowledge, such an etiology of postobstructive pulmonary edema has not previously been described.

Key words Pulmonary edema · Laryngism · Anesthetic complications · Anesthesia recovery period · Intratracheal intubation · Artificial respiration

Introduction

Pulmonary edema is a well-known complication of upper airway obstruction [1]. The most commonly reported etiology of postobstructive pulmonary edema in adults is postanesthetic laryngospasm [2]. In addition, numerous other causes have been described, including epiglottitis, thyroid goiter, upper airway tumor or foreign body, angioedema, hanging, and strangulation [1]. The inciting event in postobstructive pulmonary edema is believed to be the markedly negative intrapleural pressure generated by a forceful inspiratory effort against an obstructed extrathoracic airway (modified Müller maneuver) [3]. Such high negative intrapleural pressures augment venous return, pulmonary blood volume, and pulmonary capillary hydrostatic pressure while decreasing perivascular interstitial hydrostatic pressure, resulting in fluid accumulation in the interstitium [3]. This mechanism may also explain why pulmonary edema has been observed to occur following relief of obstruction [4–6]. An abrupt decrease in airway pressure may result from bypass of the obstruction, thereby causing increased venous return and pulmonary capillary blood volume [4].

However, the pathogenesis of postobstructive pulmonary edema is probably multifactorial, and not due solely to transudation of fluid from the pulmonary vascular space to the interstitium. Kollef and Pluss [1] suggest that mechanical stress developing from respiration against an obstructed upper airway may cause direct injury to the alveolar-capillary membrane, resulting in an acquired permeability defect. Analysis of edema fluid and serum from one of the subjects they reported yielded a fluid-to-serum protein ratio supporting the presence of increased permeability. In addition, the delay (of up to 4–6 h) in the development of pulmonary edema after the onset of upper airway obstruction seen in some cases argues against the thesis that increased fluid transudation is the only causative factor. The recently introduced concept of stress failure of pulmonary capillaries [7], in which increasing pulmonary capillary pressures cause capillary endothelial and alveolar epithelial damage with resultant high-permeability pulmonary edema, may be relevant to this phenomenon.

We herein provide what is to our knowledge the first description of pulmonary edema due to acute upper airway obstruction caused by a patient's resistance to and occlusion of an oral endotracheal tube.

Case report

A 29-year-old white man underwent septoplasty and bilateral endoscopic ethmoidectomy revision for chronic sinusitis. The patient had a significant past medical history of obstructive sleep apnea syndrome, for which he had undergone uvulopalatopharyngoplasty (UPPP) several years earlier, with almost total resolution of symptoms. Physical examination revealed a large, muscular man 185 cm in height and weighing 118 kg. Preoperative laboratory studies and chest roentgenogram were normal. Anesthesia was induced with propofol (200 mg), midazolam (2 mg), and fentanyl (200 μg). Endotracheal intubation with a 7.5 gauge endotracheal tube was performed without difficulty after intravenous administration of succinylcholine (20 mg). Inhaled anesthesia consisted of nitrous oxide, oxygen, and isoflurane. General anesthesia was maintained for approximately 2.5 h. The surgery was uneventful; oxygen saturation as measured by pulse oximetry was maintained at or near 98% throughout the procedure. Upon awakening from anesthesia, the patient forcefully bit down on the endotracheal tube and made repeated strenuous inspiratory attempts before succinylcholine (20 mg i.v.) was administered. Suctioning of the endotracheal tube yielded a moderate amount of pink, frothy secretions. Oxygen saturation fell to 90%. The patient was manually ventilated without difficulty via bag and face mask until the neuromuscular blockade resolved. To avoid further problems with resistance to the endotracheal tube, the patient was extubated. Physical examination revealed good air flow and bilateral basilar inspiratory crackles. No wheezes, rhonchi or evidence of stridor were noted. A total of 1800 ml of intravenous fluid was administered during the procedure. Upon transfer to the post-anesthesia care unit, the patient was awake and tachypneic (28 breaths/min) while wearing a face mask providing FIO_2 1.0. Arterial oxygen saturation was 78%, which improved to 83% after a non-rebreather face mask was applied. Chest radiograph demonstrated bilateral lower lobe interstitial and alveolar infiltrates (Fig. 1). Furosemide (20 mg) was administered intravenously. During the next 4 h the patient's oxygen saturation gradually improved. Arterial blood gas analysis revealed a pH of 7.35, PCO_2 of 6.0 kPa (45 torr), and PO_2 of 13.7 kPa (103 torr) (FIO_2 1.0). The method of oxygen delivery was changed to face tent (FIO_2 0.7) and the patient was transferred to the intensive care unit for further observation. Over the course of several hours, he tolerated gradual decrements in supplemental oxygen. By 24 h after the surgical procedure the patient was breathing room air comfortably with normal arterial oxygen saturation. Chest roentgenogram documented complete resolution of pulmonary edema. He was discharged on the following day.

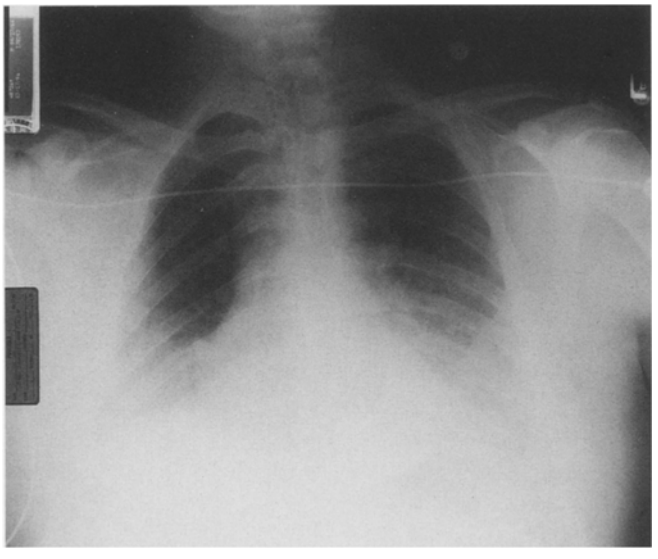

Fig. 1 Chest radiograph revealing bilateral interstitial and alveolar infiltrates in the lower lung fields

Discussion

Of the numerous causes of upper airway obstruction associated with postobstructive pulmonary edema in adults, postanesthetic laryngospasm is the most common. At least 42 cases have been reported [1, 2, 8–15]. Pulmonary edema resulting from laryngospasm induced by unsuccessful attempts at tracheal intubation has also been described [1, 16, 17]. Observations that relief of preexistent upper airway obstruction by endotracheal intubation can cause pulmonary edema are probably explained by a similar mechanism of abrupt decrease in airway pressure leading to increased venous return and pulmonary capillary blood volume, resulting in fluid transudation into the interstitial space of the lung [4].

We describe a young, muscular patient who underwent a technically uncomplicated intubation and uneventful surgery. As he emerged from general anesthesia, he forcefully bit down on the endotracheal tube and began resisting the ventilator, making numerous strenuous inspiratory attempts before a dose of succinylcholine was prepared and administered. By effectively occluding his endotracheal tube, he presumably created the physiologic equivalent of post-extubation laryngospasm, thereby producing markedly negative intrapleural pressures with his forceful inspiratory maneuvers. That this single event was solely responsible for the occurrence of pulmonary edema is supported by the presence of pink, frothy secretions emanating from the endotracheal tube and a significant decrease in arterial oxygen saturation prior to extubation. Subsequent to removal of the endotracheal tube, no evidence of laryngospasm was demonstrated. In addition, our patient had no preexistent obstruction that might have predisposed him to the development of pulmonary edema after intubation [2, 4]. His obstructive sleep apnea had been successfully treated surgically years earlier.

The sole etiology of pulmonary edema in our patient appears to be a series of intense, forceful inspiratory attempts made during a brief period against an endotracheal tube on which he had bitten down and which

he had thereby occluded. Being young, muscular and otherwise healthy, he presumably was able to generate extreme negative intrathoracic pressures. The authors of a recent review describing seven patients with postobstructive pulmonary edema [1] noted resistance to the endotracheal tube upon awakening from general anesthesia in three of the subjects. They suggested that this may be a risk factor for subsequent upper airway obstruction. Indeed, post-extubation laryngospasm was noted, in all of these patients. In a report of eight cases of post-extubation pulmonary edema [8], Cascade et al. describe one patient in whom signs and symptoms of laryngeal obstruction and pulmonary edema developed while the endotracheal tube was still in place. This patient's intubation was described as very difficult, and the authors suggest that the obstruction was due to the tip of the tube being applied against the wall of the trachea. Difficulty with intubation resulting in malpositioning of the tracheal tube has been noted by others to be a risk factor for upper airway obstruction and postanesthetic pulmonary edema [2].

The concept of stress failure of pulmonary capillaries developed recently by West et al. [7] may offer a pathophysiologic basis for the occurrence of protein-rich, and sometimes pink, frothy pulmonary secretions seen in postobstructive pulmonary edema. As we have stated above, the negative intrapleural pressure generated by forceful inspiratory efforts against an obstructed airway increases pulmonary venous return and, hence, pulmonary capillary hydrostatic pressure. This initially results in the formation of interstitial and perhaps alveolar edema. With further elevation of capillary pressures, possibly augmented by hypoxic pulmonary vasoconstriction, stress failure occurs with disruption of the pulmonary capillary wall and resultant high-permeability pulmonary edema. It has been proposed that stress failure may be involved in various conditions causing pulmonary edema and hemorrhage, including neurogenic and high-altitude pulmonary edema, and exercise-induced pulmonary hemorrhage in racehorses (and perhaps also in elite human athletes) [7].

In conclusion, the case presented suggests that resistance to an endotracheal tube may generate negative intrathoracic pressures sufficient to cause postobstructive pulmonary edema. Postoperative patients who aggressively fight the ventilator prior to extubation may warrant more vigilant observation in the post-extubation period.

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