Management Of Unpredıcted Postoperatıve Negatıve Pressure Pulmonary Edema: A Report Of Two Cases

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Citation

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Abstract

Negative pressure pulmonary edema is a potentially life-threatening complication especially during emergence in patients undergoing general anesthesia for a variety of surgical procedures. Laryngospasm-mediated upper airway obstruction leads to forced inspiration generating excessive negative intrathoracic pressure and causes negative pressure pulmonary edema. However the symptoms may also develop late in the postoperative period. We describe two young, strong men who developed this clinical picture at different times following routine uncomplicated surgical procedures. This paper not only reports the importance of early recognition of this potentially serious complication for anesthesiologists but also serves as a reminder to physicians caring for postanesthesia patients in the surgical ward to prevent the delay in diagnosis and significant morbidity.

INTRODUCTION

Negative pressure pulmonary edema (NPPE) in the adult is an uncommon, medical emergency, arising as a consequence of upper airway obstruction especially during emergence from anesthesia. It is usually manifested immediately following extubation,but in some cases, onset can be delayed for several hours ([1]).

Forceful inspiration against a closed glottis generates very negative intrathoracic pressures. This results in a clinical picture of pulmonary edema and rarely hemorrhage associated with edema ([2]). Although symptoms usually resolve with restoration and maintenance of a patent airway and supplemental O_2, they may sometimes progress to adult respiratory distress syndrome and result in death ([3]). Early recognition and appropriate positive pressure breathing respiratory therapies may prevent these potentially deletorious iatrogenic complications.

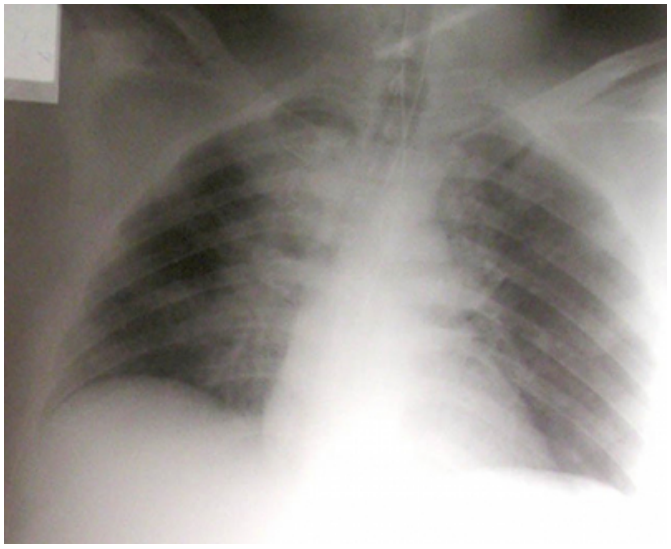
This report emphasizes the importance of rapid identification and proper management of this serious condition arising immediately or sometimes lately after general anesthesia, in achieving a successful outcome, for not only anesthesiologists but also to provide clinicians and surgeons caring for patients in the postoperative period with an understanding of NPPE.

CASE I

A 27 year old, ASA I, 118 kg, 180 cm man with a history of snoring and nasal obstruction was scheduled for elective septoplasty and nasal valve surgery under general anesthesia. His prior history was unremarkable. Physical examination revealed a young, obese man with a short neck and high palate. No premedication was given. Anesthesia was induced with fentanyl 2µg.kg^{-1} and thiopental 5mg.kg^{-1}. Intubation was achieved without difficulty with cis-atracurium 0.15mg.kg^{-1}. Anesthesia was maintained with a 1:1 mixture of N_2O and O_2 and sevoflurane 2-3 %. The surgery was uneventful and lasted 78 minutes. Over the operative period 8ml.kg^{-1}.h^{-1} of crystalloid solution was infused. Prior to extubation, neuromuscular block was reversed with neostigmine 0.04mg.kg^{-1} and atropine 0.01mg.kg^{-1}. Although he seemed to be awake, he was apneic upon extubation. Spontaneous ventilation with 100 % oxygen couldn't be assisted by bag and mask ventilation sufficiently and he developed laryngospasm with severe respiratory distress. Subsequently arterial oxygen saturation decreased to low 80's. Thus the patient was reintubated by succinylcholine 1mg.kg^{-1} and thensuctioning of the tube revealed copius amounts of pink, frothy sputum. The blood gas analysis revealed hypoxia. The chest radiograph showed diffuse alveolar and interstitial infiltration consistent with pulmonary edema. (Figure 1). He was then transferred to our intensive care unit where he was placed on mechanical ventilation and treated with positive end-expiratory pressure (PEEP) of 10 cmH_2O, high FIO_2 and 20 mg of intravenous furosemide initially. As his oxygenation and clinical picture

improved over the next 20 hours, proper ventilator adjustments were made and his FIO_2 was decreased gradually. His extubation was succeeded 24 hours after his initial reintubation. Two days later a chest X-ray showed a complete resolution of the pulmonary infiltrate and he had an uneventful recovery and was discharged to the surgical ward.

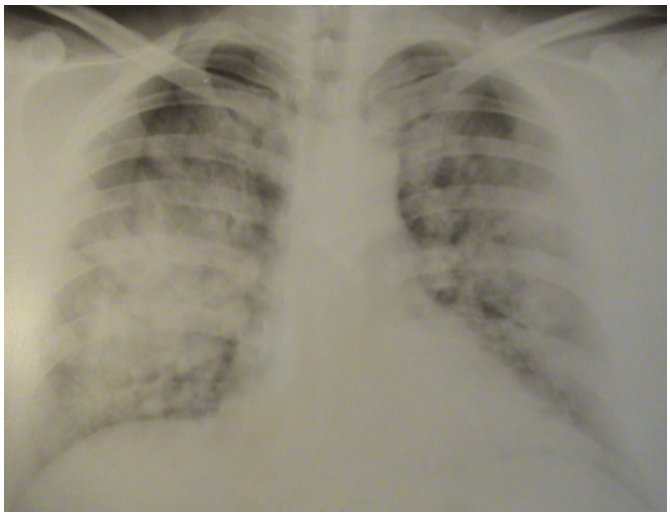
Figure 1



CASE II

A 38 year old, 96 kg, 175 cm, healthy man was scheduled for operative repair of traumatic right elbow fracture under general anesthesia since he refused to have axillary brachial plexus block. No premedication was given. After routine monitorization anesthesia was induced with fentanyl 2 $\mu g.kg^{-1}$, propofol 2 $mg.kg^{-1}$and cis-atracurium 0.15 $mg.kg^{-1}$. He was intubated on the first pass without any difficulty and mechanical ventilation was established. Anesthesia was maintained with sevoflurane at 2-3 %, and a 1:1 mixture of N_2O and O_2. No complications were observed during the 70 min operation. The patient was extubated after reversal of neuromuscular block as in the first case. Upon extubation he experienced difficult breathing. Treatment with methylprednisolone, furosemide and removal of secretions in the oral cavity and face-mask ventilation with 100 % oxygen resulted in normal breathing pattern. His oxygen saturation remained around 94-96 % during his one hour follow-up in the Postanesthesia Care Unit. He was then transferred to surgical ward without any respiratory abnormality being noticed by the anesthesiologist. However twohours later, he developed coughing and dyspnea. On auscultation, inspiratory crepitations at the bases could be revealed. A chest radiograph showed ground glass radiolucencies of both lung fields (Figure 2). The pulmonary consultant was suspicious of cardiogenicpulmonary edema but cardiac auscultation, electrocardiogram and echocardiogram showed good cardiac functions. Subsequently his oxygen saturation decreased to 85% when breathing room air thus an anesthesiologist was consulted. The clinical picture with a history of normal cardiac function, pink, frothy secretions and absence of vomiting or regurgitation made the diagnosis of NPPE more likely, so he was transferred to our intensive care unit. Clinically the patient's pulmonary edema improved by non-invasive mechanical ventilation and respiratory physiotherapy exercises within 12 hours, and he recovered without further sequelae.

Figure 2



DISCUSSION

The incidence of NPPE has been reported to be 0.05 to 0.1 % of all anesthetic practices however it is suggested that it occurs more commonly than is generally documented ([4]). The morbidity and mortality associated with under recognised NPPE is as high as 40 %([5]). The clinical picture usually arises immediately after extubation but delayed manifestations have also been defined (1). A group of patients, especially those with a short neck, difficult intubation, endotracheal tube obstruction, and history of obstructive sleep apnea, obesity, acromegaly, and upper aerodigestive tract surgery may have increased susceptibility to this condition ([6]). The proposed pathophysiologic mechanism induced by obstructed respiration, is generation of high negative intrathoracic pressures against a closed glottis (modified Mueller maneuver) resulting in markedly negative pleural pressures (-50 to $^{-1}00$ cmH_2O). The development of hypoxiaadditionally,alters transmural

pressure which in turn favors fluid transudation into the lung, and promotes pulmonary edema ([7]). In this report NPPE has occurred in two healthy, strong, ASA I anesthesia risk, adult, males who wereregarded as obese(body mass index > 30). Thus the increased muscle mass created very high intrathoracic pressures. Besides obesity, the nasal surgery in the first case facilitated the upper airway obstruction.

The symptoms of NPPE usually develop immediately after extubation though sometimes the onset may be considerably delayed up to a few hours in the postoperative period. A possible explanation for this delayed manifestation is a positive pressure, created by forceful expiration against a closed glottis, opposing fluid transudation ([8]). As airway obstruction relieves, increased venous return causes blood shift from peripheral to central circulation and hydrostatic transudation. Thus close postoperative observation must be continued for an extended time in patients experiencing respiratory difficulty. Unfortunately postanesthesia follow up was only one hour in our second case and NPPE developed two hours after extubation.

In the first case reported, the patient couldn't suppress the glottic reflex, so a large negative intrathoracic pressure led to pulmonary edema. Optimal upper airway muscular tone, appropriately timed extubation and use of an oral airway at emergence, unfortunately what was omitted in our case, could have prevented the airway obstruction and minimize the possibility of pulmonary edema. In the current report, our patients' clinical picture, progressive arterial desaturation consistent with laryngospasm, arterial blood gas determinations, plain chest films and initial dramatic effects of oxygenation strongly suggested the diagnosis of NPPE.

NPPE is a rapidly reversible condition with relatively simple management. Specifically the use of laryngeal tracheal anesthesia, intravenous lidocaine prior to extubation, the use of LMA, and/or the use of lidocaine in the endotracheal cuff are current strategies employed to prevent laryngospasm after extubation. Besides subsequent removal of the obstructive event, providing a patent airway, and adequate oxygen saturation were the first steps in the initial management of our cases as reported elsewhere ([9]). Moreover, in the first case, intubation, mechanical ventilation, and positive end expiratory pressure (PEEP) of 5 to 20 cm of H_2O along with diuretic therapy and hemodynamic monitoring in the intensive care unit was absolutely necessary. The second patient had rapid resolution by non-invasive mechanical ventilation and respiratory physiotherapy exercises. We determined the treatment modalities according to the clinical severity of respiratory distress in each patient and emphasize the value of these interventions in order to help practitioners prevent NPPE in the perioperative period.

In conclusion NPPE, if anticipated and recognized early, will be a self-limited condition with excellent prognosis and simple management. However both surgeons and clinicians must be provided with an understanding that any patientwho is otherwise well has the potential for NPPE, which is of anesthesiologic relevance, even late in the postoperative period.

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