Negative Pressure Pulmonary Edema: A Rare Complication Following Extubation

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Abstract

We describe a case of Negative Pressure Pulmonary Edema (NPPE) followed by laryngospasm occurred immediately after extubation. A 24-year-old man underwent a surgical correction of unilateral inguinal hernia by laparoscopy. Anesthesia was maintained with sevoflurane 2.5%. After fully awake extubation, nearly total upper airway obstruction due to severe laryngospasm was observed by a decrease in oxygen saturation and the presence of large amount frothy pink sputum, suggestive of acute pulmonary edema. A nasal airway was inserted, but face mask ventilation was difficult. Oxygenation of the airway was maintained with support of non invasive ventilation for twenty four hours, with SpO2 of 92-96 %. 48 hours later, the pulmonary edema disappeared and the patient was discharged without complications (SpO2 96% and ambient air).

Keywords: General anesthesia; Post-extubation; Laryngospasm; Negative pressure pulmonary edema

Case Presentation

A 24 year old man 72kg, with no past history of any significant illness or allergy was admitted in the day care surgery for a correction of unilateral inguinal hernia by laparoscopy under general anesthesia. Patient was perfectly well before surgery, routine laboratory findings were normal, haemodynamically stable with no respiratory complaints. He denied previous problems with general anesthesia and his baseline peripheral oxygen saturation was 99% in ambient air. The patient was accepted to operation room after he was informed and asked to sign consent form for anesthesia and surgery.

He was monitored with electrocardiogram, non-invasive blood pressure, oxygen saturation (SpO2) and a peripheral vein was catheterized for infusion and drug administration. Anesthesia was induced with intravenously propofol (150mg), fentanyl (250mcg) and atracurium (35mg). The endotracheal intubation using tube 8.0mm was easy with grade 1 of Cormack-Lehane classification. Anesthesia was maintained with sevoflurane (2.0-2.5%). Surgery lasted about one hour and during that time vital signs were normal. Patient recovered from surgery and was extubated successfully.

Immediately after extubation, the patient developed inspiratory stridor with severe laryngospasm followed by a decrease in oxygen saturation, increase in respiratory rate (around 25/minute) and increase in heart rate (about 120/min). There was large amount of frothy pink sputum. Chest auscultation revealed bilateral generalized coarse crackles. Immediate diagnosis of NPPE secondary to post extubation laryngospasm was made. A nasal airway was inserted, but face mask ventilation was difficult. The patient was moved to post anesthetic care unit. Oxygenation of the airway was maintained with support of non invasive ventilation for twenty four hours and SpO2 ranged between 92-96% in the intensive care unit.

Computed tomography of the chest at the time showed foci of...
negative pressure levels and cause pulmonary edema [1]. Site during anesthesia recovery. This may generate high intra pleural frequent when there is a major noxious stimulation in the surgical with tracheal tubes and laryngeal mask [5]. However, it may be less protective reflex is mediated by the vagus nerve [2]. Laryngospasm is chest wall muscles and tracheobronchial tree smooth muscles. This spasm is a response to mechanical or chemical stimulation intrinsic airways and lungs defensive reflex system. During laryngospasm, [3]. Laryngospasm and bronchospasm are manifestations of upper procedures [5,9]. This statistic increases to 11% when considering all patients requiring intervention for acute upper airway obstruction [5]. The same statistics are shared by different authors [10,11]. It’s more common in healthy and young males who are more predisposed to major negative pressure differences [3,10]. In adults, 50% of cases are due to laryngospasm postoperatively, but can also occur by occlusion of the endotracheal tube by biting and less frequently after foreign body aspiration, oropharyngeal surgery or residual neuromuscular blockade [5].

Post-extubation NPPE is associated with a higher incidence of cases, which are mostly due to laryngospasm. Laryngospasm is defined as glottic occlusion, secondary to laryngeal constrictor muscles contraction (interarytenoids, lateral cricoarytenoids and internal and external thyroarytenoids), in response to a stimulus [3]. Laryngospasm and bronchospasm are manifestations of upper airways and lungs defensive reflex system. During laryngospasm, spasm is a response to mechanical or chemical stimulation intrinsic or extrinsic to painful stimulation, involving all laryngeal and chest wall muscles and tracheobronchial tree smooth muscles. This protective reflex is mediated by the vagus nerve [2]. Laryngospasm is more often seen in anesthetic emergence during extubation [2], both with tracheal tubes and laryngeal mask [5]. However, it may be less frequent when there is a major noxious stimulation in the surgical site during anesthesia recovery. This may generate high intra pleural negative pressure levels and cause pulmonary edema [1].

NPPE begins with a significant obstruction of the upper airway consolidation with confluent aspect in “frosted glass” in the upper lobes of both lungs in the middle lobe and lingula, consistent with the diagnosis of pulmonary edema (Figure 1). Patient gradually started improving; after six hours was moved to intensive care unit with SpO2 up to 96% in NIMV and the crackles on his chest markedly decreased. Patient continued to improve during the next 24 hours and was completely asymptomatic at this time. A comparison between the chest X-ray after extubation and about 24 hours after the event showed marked improved; however, residual interstitial infiltrate persisted with small alveolar consolidations (Figure 2). He was discharged from the hospital within 48 hours of the event.

Discussion

The incidence of NPPE is around 0.05-0.1 % of all anesthetic procedures [5,9]. This statistic increases to 11% when considering all patients requiring intervention for acute upper airway obstruction [5]. The same statistics are shared by different authors [10,11]. It’s more common in healthy and young males who are more predisposed to major negative pressure differences [3,10]. In adults, 50% of cases are due to laryngospasm postoperatively, but can also occur by occlusion of the endotracheal tube by biting and less frequently after foreign body aspiration, oropharyngeal surgery or residual neuromuscular blockade [5].

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In conclusion, although a frequently benign condition, NPPE secondary to laryngospasm is an important cause of morbidity, hospitalization in intensive care unit and occasionally mortality in young and healthy individuals. It is a well-described clinical syndrome, but probably under-recognized, with the exact mechanism still unclear. Early recognition of the disease was a key point that...
allowed the immediate application of positive airway pressure leading to a rapid resolution of the frame, thus ensuring the favorable developments in this case. We encourage our colleagues to be vigilant in recognizing NPPE in the presence of laryngospasm.

References