Florid Negative Pressure Pulmonary Edema

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Citation

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Abstract

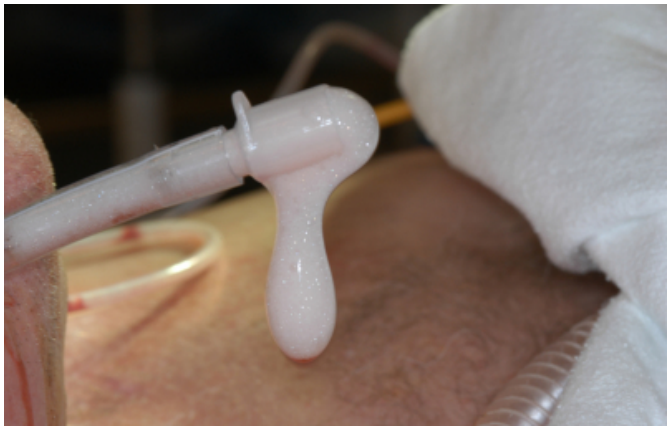
Negative pressure pulmonary edema (NPPE) is described in two distinct clinical scenarios. Type I NPPE develops immediately after onset of acute airway obstruction. Type II NPPE develops after the relief of chronic upper airway obstruction. We report the case of a 44 year old man who developed florid NPPE post extubation following local flap closure of an orocutaneous fistula. Pathophysiology, treatment and outcome of NPPE are discussed.

CASE REPORT

A 44 year old man with a history of T4N1 squamous cell carcinoma of left tonsil underwent wide excision left tonsil, left neck dissection and pectoralis major myocutaneous regional tissue transfer after pre-operative combined chemotherapy and irradiation therapy at an outside institution. He presented to our hospital with an orocutanous fistula and was scheduled to undergo closure of the fistula with local rotation flaps. His past medical history was significant for hypertension and hepatitis. He was allergic to morphine and codeine. His review of systems was negative. Physical examination revealed severe irradiation changes of neck. He had moderate to severe trismus. There was a 3 x 1.5 cm fistulous tract with an exposed reconstruction plate. His ASA class was 3. His preoperative vitals were within normal limits. His weighed 100 kilograms. He was intubated orally with a size 6.0 endotracheal tube without difficulty. He underwent uneventful closure of the fistula with local flap. The total time of anesthesia was 3 hours and 20 minutes. At the conclusion of the case the patient was extubated in the operating room. Within 5 minutes post extubation, the patient experienced laryngospasm with high pitched inspiratory stridor for approximately 3 minutes. He was rapidly reintubated with the return of frothy sputum (see photograph). He was taken to the intensive care unit, extubated after 15 minutes and maintained oxygen saturations greater than 95% on 35% face mask. He was transferred from the ICU to the floor and was discharged to home on post-operative day number four.

Figure 1

Photograph: Frothy secretions of negative pressure pulmonary edema (NPPE).



DISCUSSION

Oswalt et al[1] are credited for the first description of negative-pressure pulmonary edema (NPPE). NPPE is a non-cardiogenic pathologic process in which a transudation of fluid into the pulmonary interstitium develops in response to the generation of markedly negative intrathoracic pressures. NPPE is described in two distinct clinical scenarios.[2] Type I NPPE develops immediately after onset of acute airway obstruction such as laryngospasm or epiglottitis. Laryngospasm is widely regarded as the most common precipitant of Type I NPPE in adults. Type II PPE develops after the relief of chronic upper airway obstruction, such as adenotonsillar hypertrophy or laryngeal neoplasm.[3] The incidence of this phenomenon is reported to be 11% of patients requiring intervention for acute airway obstruction with a male predominance of 2:1.[3]

The pathophysiology of NPPE has been defined. High negative intrathoracic pressures initiate a cascade of events in the development of NPPE. Forceful inspiration against a closed glottis, such as in laryngospasm, can result in markedly negative intrathoracic pressures, up to -140 cm H20 from baseline average of -4 cm $H20_4$. With such negative intrathoracic pressure, venous return is increased to the right heart subsequently raising pulmonary hydrostatic pressure. The increased pulmonary hydrostatic pressure leads to the transudation of fluid from pulmonary capillaries into the pulmonary interstitial space, resulting in pulmonary edema. Hypoxic vasoconstriction of both pulmonary and systemic arterioloes raises systemic blood pressure and increases the afterload of the left and right ventricles. Catecholamine release occurs simultaneously which increases total peripheral resistance. Increased fluid volume is trapped within the thoracic cavity with unbalanced transmembrane pressures in the alveoli. Expiration against a closed system generates high intraluminal airway pressures. This serves as auto-PEEP (positive end-expiratory pressure) that prevents fluid from leaving the pulmonary capillaries and from entering the alveoli. When laryngospasm is relieved, either spontaneously or by reintubation, the resultant drop in airway pressure causes a transudation of fluid into the alveoli and severe acute pulmonary edema. Some authors have proposed that NPPE develops from damage to capillary membranes leading to increased pulmonary capillary permeability. Other authors implicate hypoxia and increased adrenergic activity generated after acute airway obstruction as the mechanisms responsible for the development of NPPE.

Rapid diagnosis of NPPE is made by the surgeon or anesthesiologist with a high index of suspicion. Typically, patients with Type I NPPE have previously undergone an uncomplicated surgical procedure. Upon completion of the procedure, the majority of patients experience rapid oxygen desaturation, tachypnea, rales on auscultation, bradycardia, along with pink, frothy sputum immediately or within 30 minutes into the postoperative period. The diagnosis of NPPE is confirmed with a chest x-ray which demonstrates bilateral infiltrates in the pulmonary fields.

Once a patient is diagnosed with NPPE, an airway should be immediately secured via endotracheal intubation and supplemental oxygen. Mechanical ventilation may also be necessary. Often, patients require PEEP or continuous positive airway pressure. PEEP should be maintained at 5 to 10 cm H_20 because greater pressures may cause complications, such as tension pneumothorax and decreased cardiac output. The administration of diuretics and steroids in the treatment of NPPE has not been proven[4] and should be used at the physician's discretion.

There is no intervention proven to prevent NPPE, but avoiding laryngeal irritation that leads to laryngospasm is likely to reduce the occurrence of NPPE. For this reason topical laryngotracheal anesthesia (LTA) comprised of 2mL each of 1% lidocaine and 2% tetacaine is recommended.[4]

With prompt diagnosis and therapeutic action, NPPE resolves generally within 24 hours. However, when recognition is delayed, patients with NPPE have mortality rates ranging from 11% to 40%.[3] Therefore, early recognition of NPPE is crucial to decrease morbidity in these patients. A high index of suspicion for NPPE must be maintained for the patient who experiences post-extubation laryngospasm.

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