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NEGATIVE PRESSURE PULMONARY EDEMA AFTER GENERAL ANESTHESIA

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Review

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Abstract

Negative pressure pulmonary edema is a type of non-cardiogenic pulmonary edema caused by strong inspiratory effort to overcome obstruction in the upper airways. The main mechanisms responsible for the pathophysiology are the increase in pulmonary capillary hydrostatic pressure and the increase in capillary membrane permeability that develops after the negative intrathoracic pressure increases with inspiratory effort. Although all causes that may lead to upper airway obstruction may play a role in the etiology, the most common factor is laryngospasm that develops during awakening from anaesthesia. Young male smokers under general anaesthesia are more at risk. Onset may vary from a few minutes to several hours after extubation or relief of laryngospasm. It leads to an acute respiratory failure that is potentially life-threatening and usually requires follow-up and treatment in the intensive care unit. The first step in treatment is to relieve airway obstruction and provide oxygen support. Positive pressure ventilation and use of diuretics are beneficial and usually tend to improve within 24-48 hours. The mortality rate is reported to be around 5% in case analysis reports of recent years. Since it is observed more frequently in anaesthetised patients, early diagnosis and treatment in the postoperative period is important. The aim of this review is to present a current perspective on negative pressure pulmonary edema in the light of the literature.

Key Words: Non-cardiogenic pulmonary edema, Laryngospasm, Positive pressure ventilation

Özet

Negatif basınçlı akciğer ödemi, üst hava yollarındaki tıkanmayı aşmak için sarfedilen güçlü inspiratuar efor sonucu meydana gelen kardiyojenik olmayan bir akciğer ödemi türüdür. Patofizyolojiden sorumlu temel mekanizmalar, inspiratuar efor ile artan negatif intratorasik basınç sonrasında gelişen pulmoner kapiller hidrostatik basınç artışı ve kapiller membran permeabilitesindeki artıştır. Üst hava yolunda tıkanmaya yol açabilecek tüm sebepler etyolojide rol oynayabilmekle birlikte en sık etken anesteziden uyanma sırasında gelişen laringospazmdır. Genel anestezi uygulanan, genç sigara içen erkek hastalar daha fazla risk altındadır. Başlangıcı ekstübasyon veya laringospazmın giderilmesini takiben birkaç dakikadan birkaç saate kadar değişebilir. Potansiyel olarak yaşamı tehdit eden ve genellikle yoğun bakım ünitesinde takip ve tedavi gerektiren akut bir solunum yetmezliğine yol açar. Tedavide ilk adım, hava yolu tıkanıklığının giderilmesi ve oksijen desteği sağlanmasıdır. Pozitif basınçlı ventilasyon ve diüretik kullanımı faydalıdır ve genellikle 24-48 saat içinde düzelme eğilimindedir. Son yıllara ait vaka analiz raporlarında mortalite oranının %5 civarında olduğu belirtilmektedir. Anestezi alan hastalarda daha sık gözlenmesi nedeniyle postoperatif süreçteki erken teşhis ve tedavisi önem arz eder. Bu derlemenin amacı, negatif basınçlı akciğer ödemine literatür eşliğinde güncel bir bakış açısı sunmaktır.

Anahtar Kelimeler: Kardiyojenik olmayan akciğer ödemi, Laringospazm, Pozitif basınçlı ventilasyon

1. Introduction

Negative pressure pulmonary edema (NPPE) is a non-cardiogenic type of pulmonary edema characterized by fluid accumulation in the lung interstitium and alveolar space as a result of rapidly increasing negative intrathoracic pressure triggered by a strong inspiratory effort to overcome upper airway obstruction. NPPE, which is of clinical importance in general anesthesia, most commonly occurs in patients with acute laryngospasm during removal of endotracheal tube or supraglottic airway devices (Ghofaily et al., 2013). In studies including large case series, the incidence after extubation has been reported to be 0.019-0.094% (19-94 per 100,000 cases) (Deepika et al., 1997; Tsai et al., 2018).

Several risk factors for NPPE have been proposed. These include young age, male gender, smoking, American Society of Anesthesia score I-II (ASA I-II), head and neck surgeries, difficult intubation, use of irritating anesthetic gas agents, obesity, recent upper respiratory tract infection and presence of reactive airway. According to the results of comparative case analysis, it has been reported that it is especially common in young male smokers. This is in line with the idea that young healthy men may generate higher negative intrathoracic pressure in case of airway obstruction (Ghofaily et al., 2013; Tsai et al., 2018).

Respiratory complications of general anesthesia are significant. NPPE leads to an acute respiratory failure that is potentially life-threatening and usually requires follow-up and treatment in the intensive care unit. While the mortality rate was higher in the past years, it has been reported to be 5% in recent case studies (Mehta et al., 2006; Din-Lovinescu et al., 2021). Early diagnosis and treatment is extremely important in anesthetized patients, especially in the postoperative period. The aim of this review is to provide an up-to-date overview of NPPE and information about its diagnosis and treatment.

2. Etiology

Although all causes of upper airway obstruction can lead to NPPE, the most common etiologic factor is laryngospasm occurring in the postoperative period after anesthesia. Laryngospasm is responsible for 50% of NPPE after obstruction (Louis and Fernandes., 2002). Biting the endotracheal tube or laryngeal mask during awakening from anesthesia is another possible cause (Koh et al., 2003; Devys et al., 2000). In otolaryngology procedures in adults, it has been reported to occur most commonly after septoplasty, rhinoplasty and sinus surgery (Din-Lovinescu et al., 2021). In the pediatric population, it is seen after surgeries to remove chronic causes of airway obstruction (tonsillectomy, adenoidectomy) (Ahmed and Almutairi., 2018; Thiagarajan and Laussen., 2007).

The etiology is not limited to anesthetized patients. In the literature, there are various case reports (tumor, suffocation, hanging, epiglottitis, croup, Ludwig's angina, goiter, hiccups, hematoma, obstructive sleep apnea, obesity, foreign body aspiration, mediastinal tumor) of NPPE developing as a result of airway obstruction (Oswalt et al, 1977; Lopez et al., 1990; Koh et al., 2003; Stuth et al., 2000; Deepika et al., 1997; Louis and Fernandes, 2002).

3. Pathophysiology

The two main mechanisms responsible for the pathophysiology of NPPE are pulmonary capillary hydrostatic pressure and increased capillary membrane permeability. In lung physiology, where hydrostatic pressure is in equilibrium and permeability is normal, fluid flow is minimal from the capillaries to the interstitium. The fluid is then absorbed and passes into the pulmonary lymphatics. When the rate of interstitial fluid formation exceeds the reabsorption capacity, pulmonary edema results.

The event that initiates the process of NPPE development is the increase in negative intrathoracic pressure due to the patient's strong effort to breathe in case of partial or complete obstruction of the upper airway. While the normal NIP during inspiration is between (-2) - (-8) cmH_2O, in such a situation it can increase up to (-140) cmH_2O in young healthy people (Wu., 2015).

Due to increased NIP; venous return to the right heart increases, pulmonary blood flow (volume and pressure) increases, pulmonary capillary hydrostatic pressure increases and alveolar interstitial pressure decreases. This pressure gradient allows the movement of fluid from the pulmonary capillaries to the interstitium and alveolar spaces. Increased right ventricular filling with increased venous return pushes the interventricular septum to the left, resulting in a decrease in left ventricular (LV) compliance and volume. In addition, NIP may increase LV afterload, increasing LV end-diastolic volume and pressure. This decreases LV ejection fraction. The decrease in ejection fraction further increases pulmonary capillary hydrostatic pressure by successively increasing end-diastolic pressure, left atrial pressure and pulmonary venous pressure and contributes to edema formation (Lemyze and Mallat., 2014).

The other mechanism underlying edema formation is the disruption of pulmonary capillary membrane integrity and increased permeability. The factors responsible for pulmonary capillary membrane damage are elevated NIP due to airway obstruction, acute hypoxemia and increased sympathetic activity. Acute hypoxemia and secondary hyperadrenergic state increase systemic and pulmonary vascular resistance, leading to vasoconstriction in the microcirculation, damage to the pulmonary capillary membrane and impaired permeability. This increase in capillary permeability predisposes to the formation of protein-rich pulmonary edema (Ma et al., 2023). Edema fluid/plasma protein ratio is a method used to differentiate cardiogenic and noncardiogenic pulmonary edema. An edema fluid/plasma protein ratio of < 0.65 suggests

hydrostatic pulmonary edema, while a ratio of > 0.75 suggests high-permeability pulmonary edema and acute lung injury (Fremont et al., 2007).

4. Clinical and Diagnosis

The onset of NPPE can range from a few minutes to several hours following extubation or resolution of laryngospasm. Initial signs and symptoms of airway obstruction include respiratory distress, stridor, decreased oxygen saturation, use of auxiliary respiratory muscles including suprasternal-intercostal retractions and a panicked facial expression. As pulmonary edema develops, progressive decrease in oxygen saturation, dyspnea, tachypnea, tachycardia and foamy sputum are observed. Some patients may have haemoptysis. Crepitant rales are heard on lung auscultation. Arterial blood gas analysis is useful to demonstrate hypoxaemia. Hypercapnia and acidosis may be observed depending on the severity of respiratory dysfunction. Bilateral interstitial and alveolar infiltrations on chest radiography and bilateral central interstitial alveolar consolidations and ground glass appearance on computed tomography are remarkable (Bhaskar and Fraser, 2011).

Although NPPE usually occurs immediately after surgery, late-onset cases are rare. A patient who underwent vocal cord polypectomy under general anaesthesia was reported to develop NPPE 3 hours after surgery. Depending on the degree of pulmonary damage, some patients with a mild course may not be recognised. Patients with previous airway obstruction or risk factors for NPPE should have a longer post-anaesthetic observation period (Koide et al., 2020).

5. Differential Diagnosis

In patients who show signs of respiratory distress (decreased oxygen saturation, dyspnea, tachypnea) in the postoperative period, the perioperative period and past medical history of the patient should be rapidly evaluated for possible causes. If the echocardiogram is unknown, new-onset arrhythmia, ST changes on electrocardiogram, cardiomegaly on radiography and presence of peripheral edema should suggest cardiogenic pulmonary edema. Fluid inbalance should be evaluated, plasma sodium level and osmolarity should be controlled in patients who are given excessive fluid during surgery, hypotonic fluid is administered and urine output is not adequate. It should be kept in mind that neurogenic pulmonary edema may develop due to increased sympathetic activity in patients with intracranial mass, haemorrhage and head trauma. Vomiting,

presence of gastric contents in the oropharynx or witnessed aspiration may lead to respiratory distress (aspiration pneumonia) in the postoperative period. In case of anaphylaxis, cardiogenic pulmonary edema may develop due to cardiac dysfunction. Atopic history, allergen exposure, presence of rash, bronchospasm and hypotension should be warning in such patients.

In the presence of a triggering event (acute obstruction) in the postoperative period, in patients with respiratory distress symptoms (decreased oxygen saturation, dyspnea, tachypnea), hearing rales with auscultation, foamy sputum and bilateral interstitial infiltrations on chest radiography support the diagnosis of PPPE. In the presence of severe respiratory distress findings occurring without signs of acute airway obstruction, evaluations for acute respiratory distress syndrome should be performed (Bhaskar and Fraser, 2011).

6. Treatment

The first step of NPPE treatment is to relieve airway obstruction and provide oxygen support. Due to the etiological diversity, the way oxygenation is provided may vary from a simple face mask, high-flow nasal cannula, intubation or even surgical procedures (cricothyrotomy, tracheotomy) in case of difficult airway.

In patients who develop extubation-induced laryngospasm, the obstruction usually has a good prognosis and NPPE tends to resolve within 24-48 hours. Patients should be closely monitored under oxygen support. Initially, noninvasive positive pressure ventilation provides faster resolution of edema. For this purpose, the use of positive end-expiratory pressure (PEEP), continuous positive airway pressure (CPAP) and bilevel positive airway pressure (BiPAP) reduces respiratory workload, improves oxygenation and reduces the need for intubation. In patients with severe hypoxaemia and the need for invasive mechanical ventilation, low tidal volume ventilation with PEEP support (lung protective ventilation strategy) is recommended. The use of paralytic agents may be considered to relieve upper airway obstruction in intubated patients (Jaber et al., 2010; Bhattacharya et al., 2016).

Although diuretics are frequently used in the treatment of cardiogenic edema, their use in the treatment of NPPE is controversial. In a study of 87 case reports of 87 patients, it was reported that the rate of diuretic use in primary treatment was 63% (55 patients). Their use is recommended in non-hypovolemic patients (Din-Lovinescu et al., 2021; Cerna-Viacava et al., 2023). B2-agonist bronchodilators are used because they are thought to improve alveolar fluid

clearance and help resolve pulmonary edema (Berthiaume et al., 2002). Steroids are thought to be particularly useful in reducing airway events after extubation and reducing interstitial damage (Westreich et al., 2006; Kuriyama et al., 2017).

7. Conclusion

NPPE is one of the respiratory complications that can be seen rarely in the postanesthetic period. Early diagnosis and treatment is important to reduce morbidity. Since the diagnosis may be delayed in some patients due to its late onset and atypical nature, the post-anaesthetic observation period of patients in the risk group should be prolonged. Patients who develop NPPE should be followed closely and supported with positive pressure ventilation in intensive care unit.

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