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Negative Pressure Pulmonary Edema

Fazal Hameed Khan*
Aga Khan University Hospital, Pakistan

***Corresponding author:** Fazal Hameed Khan, Professor of anesthesia and Associate Dean for Faculty, Aga Khan University Hospital, Karachi, Pakistan, Tel: +92 21 34864533; Email: fazal.hkhan@aku.edu

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Abstract

Negative pressure pulmonary edema is a well-known clinical condition mostly affecting young healthy adults and at times pediatric patients. It occurs mostly acutely in patients who have experienced airway obstruction. Most common pathophysiology is the development of a negative intrathoracic pressure which results in increased preload and subsequently increase pulmonary hydrostatic pressures. The pulmonary edema develops once the airway obstruction is relieved and the forces opposing the transudation of fluid into the interstitial tissues are removed. Management includes a high index of suspicion in patients who have developed airway obstruction, early recognition, relief of obstruction, and maintenance of airway along with oxygen therapy. BIPAP or CPAP may be needed in patients who do not responds to these measures. Invasive mechanical ventilation with high FiO2 is reserved for refractory hypoxia.

Keywords: Edema; Pulmonary; Negative Pressure

Introduction

Pulmonary edema developing after airway obstruction known as Post obstructive pulmonary edema or negative pressure pulmonary edema is a well-established condition that usually manifests acutely in the Post operative period and is sometimes life-threatening. As early as 1927, researchers were knowledgeable about a clinical condition where pulmonary edema following prolonged inspiration develops in dog models who had fixed airway obstruction. It was in 1942 that the pathophysiology of this condition was described in the literature [1,2]. It took another thirty years for the researchers to start publishing case reports involving pediatric patients which mentioned pulmonary edema secondary to airway obstruction in these patients [3,4]. The first documented case report of negative pressure pulmonary edema developing after laryngospasm in the post-extubation period was published in 1980 [5]. The main cause of POPE in adult patients is laryngospasm while in the pediatric patient's population epiglottitis and croup are the common etiologies [6]. The incidence of post obstructive pulmonary edema in healthy adult patients is 0.05% to 0.1% [7] and the incidence in both adult and pediatric patients having upper airway obstruction requiring urgent tracheal intubation or tracheostomy has been quoted to be 11% to 12% [8].

Pathophysiology

The initiating event in the pathophysiology of NPPE is airway obstruction due to any reason. To overcome airway obstruction, patient respiratory efforts result in negative intrathoracic pressure [9]. This causes augmentation of venous return which increases the pulmonary capillary hydrostatic pressure. At the same time, the patient tries to exhale forcefully resulting in the generation of positive intrathoracic pressure [4] hence despite an increased capillary hydrostatic pressure, there is no transudation of fluids into the interstitial tissue. Once the obstruction is relieved, the positive intrathoracic pressure is also eliminated and thus the increased pulmonary hydrostatic pressure causes fluid transudation into the interstitial tissues and

the alveoli. As a result of the development of pulmonary edema, hypoxia develops, and this later causes a release of catecholamines. These effects cause pulmonary and systemic vasoconstriction. Increasing pulmonary vascular resistance further increases the pulmonary edema while increased systemic vascular resistance causes a decrease in the ejection fraction and a further increase in the pulmonary edema NPPE [10].

Management

Early recognition and Avoidance of airway obstruction are crucial in preventing the development of this crisis. Few precautions during anesthesia management such as adequate anesthetic depth during mask ventilation, extubating the patient once fully awake and use of awake fiberoptic intubation in patients with airway abnormalities may help [11]. Most commonly these patients present with shortness of breath, shallow breathing, increase respiratory rate, desaturation, and at times outpouring of pink frothy secretion either immediately after relief of airway obstruction or sometimes within hours. Chest X-RAY reveals bilateral alveolar and interstitial infiltrates [12].

Treatment starts with the treatment of the cause that results in airway obstruction and later maintenance of the open airway till the patient's condition improves. Other measures include oxygen administration through a tight-fitting facemask, diuretics, and if needed use of Bilevel positive airway pressures or continuous positive airway pressures to treat hypoxia. In very severe cases where there is unresolved hypoxia, invasive ventilation and one hundred% oxygen is needed [13].

Conclusion

Post obstructive pulmonary edema is often misdiagnosed. There should be a high index of suspicion in patients who are more prone to airway obstruction. These cases often present with acute respiratory distress usually occurring immediately after extubation. Whenever there is a suspicion that patients are likely to develop post obstructive pulmonary edema, they must be kept in a suitable setting for monitoring and further management. This can occur in adults and pediatric patients with or without any risk factors. The symptoms may vary from mild hypoxia to severe respiratory compromise needing mechanical ventilation and PEEP.

Conflicts of Interest

Not applicable

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