Negative Pressure Pulmonary Edema Post-Appendicectomy

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Postoperative pulmonary edema is a rare life-threatening complication associated with general anesthesia. Laryngospasms lead to forced inspiration, generating a large amount of negative intrathoracic pressure which leads to negative pressure pulmonary edema (NPPE). It usually develops immediately after extubation.

We report a case of a young fit adult who developed NPPE after an uneventful appendectomy in the postoperative period. This rare condition in the postoperative period is lethal if immediate remedial steps are not taken. The anesthetist and paramedical staff in the recovery room should be well aware of this condition.

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Pulmonary edema is an abnormal accumulation of fluid in the extravascular compartment of the lung. Negative pressure pulmonary edema (NPPE) is a potentially lethal medical emergency seen usually in the immediate postoperative period following extubation. Forceful inspiration against a closed epiglottis is postulated to be the main reason for this condition. It is also known as 'post-obstructive pulmonary edema' and 'laryngospasm-induced pulmonary edema[1]. NPPE is a rare but well-known complication associated with upper airway obstruction. It usually occurs just after extubation[2]. However, the presentation may sometimes be delayed[3].

The aim of this presentation is to report a rare condition of NPPE in the postoperative period.

THE CASE

A thirty-two-year-old Saudi male presented with typical signs and symptoms of acute appendicitis. His weight, height and BMI were 80 kg, 163 cm, and 30.1, respectively. There was no history of smoking, asthma or any other chronic medical illness. He had an uneventful open appendectomy. The surgery lasted for approximately 35 minutes. He was extubated and was in good condition when he was transferred to the recovery room. In the recovery room, the oxygen saturation suddenly decreased to 86% on room air. He was dyspneic and having pink frothy sputum from his mouth. He was immediately placed on non-invasive ventilation and given 20 mg Lasix, and transferred to the Intensive Care Unit (ICU). X-ray chest revealed features of diffuse bilateral pulmonary edema, see figure 1. Echocardiogram revealed an ejection fraction of 60%. Further Lasix was given 12 hourly. He was transferred to the general ward after 24 hours, see figure 2.

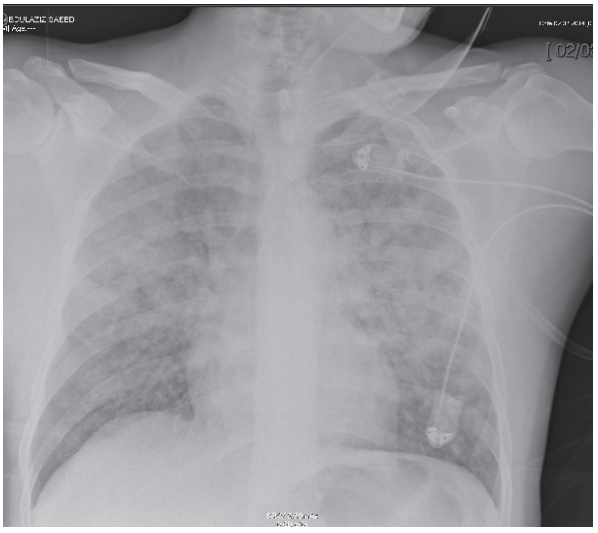

Figure 1: X-ray Chest Showing Features of Pulmonary Edema

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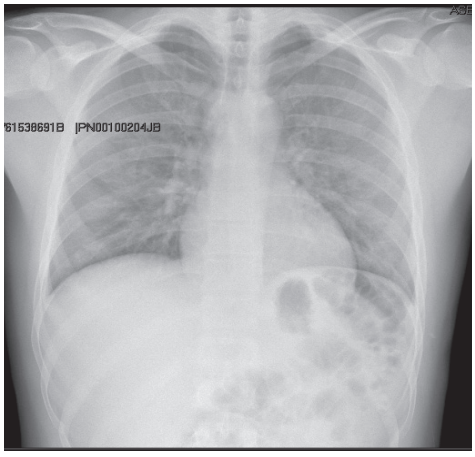

Figure 2: X-ray Chest PA View Showing Resolved Edema after 24 Hours

The patient was kept under observation for another 24 hours and was discharged. He was instructed to inform the anesthetist of this complication in case of any future surgery, as recurrences of NPPE have been reported.

DISCUSSION

NPPE is an uncommon but potentially lethal complication of upper airway obstruction. This is most commonly seen in patients during emergence from anesthesia[2]. In our case, NPPE was not seen during emergence, rather it was seen in the recovery room. The presentation of NPPE can be delayed for up to a few hours in the postoperative period[3]. The reason for delayed presentation may be positive pressure, which is created by forceful expiration against a closed glottis and this positive pressure opposes fluid transudation[3]. It emphasizes the importance of good observation and good care of the patients in the postoperative period a few hours after surgery.

NPPE is a pure form of hydrostatic edema[1]. The incidence is reported to be less than 0.1% of those undergoing general anesthesia[4]. However, the incidence may be largely unreported. The etiology of this condition is a large inspiratory force generated against a closed airway. The decreased intrathoracic pressure leads to an increased venous return to the right side and increased pulmonary capillary pressure. Pulmonary capillary pressure is further increased by decreased left ventricular compliance. The combination of increased venous return and increased pulmonary capillary wedge pressure leading to the shift of fluid into the pulmonary interstitium causing pulmonary edema. The main etiology of NPPE is negative intrathoracic pressure. Young athletic males who are capable of producing large amounts of negative intrathoracic pressure are thus at increased risk of this condition[4].

Patients predisposed to airway obstruction have an increased risk of airway complications post-extubation after general anesthesia[5]. The risk factors for airway obstruction are obstructive sleep apnea syndrome, obesity, palatal mass, short thick neck, and postnasal surgery[5].

Signs and symptoms of NPPE include hypoxemia, tachypnea, dyspnea, pulmonary rales, pink frothy sputum, hemoptysis, decreased oxygen saturation, and evidence of upper airway obstruction. The important differential diagnoses are fluid overload, pulmonary embolus, acute respiratory distress syndrome, and cardiac abnormalities[6].

Chest radiographs may show features of pulmonary edema. Though NPPE is the most common cause of pulmonary edema in a healthy patient in the postoperative period; pulmonary edema should be considered in aspiration of gastric contents, acute respiratory distress syndrome (ARDS), congestive heart failure (CHF), volume overload, and pulmonary embolism. Our patient was a healthy young patient with no history of major illness and all tests including echocardiogram, EKG was normal. We ruled out the other causes of pulmonary edema. Radiologically, NPPE presents with Kerley lines, peribronchial cuffing, and in severe cases, central alveolar edema without cardiomegaly on X-ray chest[7].

Our main line of management was bag and mask ventilation and furosemide injection. Most patients who develop NPPE are managed with oxygen and furosemide, and monitoring in the ICU, some patients require intubation. Short-acting muscle relaxants such as succinylcholine can be used to relieve laryngospasm if manual ventilation is ineffective. In some cases, re-intubation and mechanical ventilation are required for a short duration to maintain airway patency and adequate oxygenation. The role of furosemide is controversial but is being used by most physicians in such cases. Since NPPE is believed to be secondary to leaky capillaries and not to excess fluid or fluid overload, the role of furosemide is debatable[4].

Our patient improved within 24 hours clinically and radiologically. In other studies, patients usually have an uncomplicated course and the resolution of edema is rapid. Rarely, patients may develop adult respiratory distress syndrome[8-9].

To prevent the occurrence of NPPE, the use of intravenous lidocaine before extubation and lidocaine spray in the endotracheal cuff is advocated by anesthetists[9]. These measures are not being routinely used. In our case, these steps were not taken. In those who are high risk, intraoperative muscle relaxants, and steroids should be used[10]. Other preventive steps are to clear the airway of the retained blood or secretions by suctioning and extubation.

CONCLUSION

NPPE is a complication which is usually seen in the immediate postoperative period, but can be seen up to a few hours after. There should be proper observation of patients in the recovery room. It can be lethal if not treated on time.

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