Negative Pressure Pulmonary Edema

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Negative Pressure Pulmonary Edema (NPPE) or Post-Obstructive Pulmonary Edema (POPE) is one of the most life-threatening emergencies in anesthesia. Immediate diagnosis with NPPE leads to better outcome. Usually, it affects healthy young patients. It occurs after upper airway obstruction, such as laryngeal spasms, tumor, and infection; it follows strong inspiratory effort leading to low negative pressure in the lungs which results into fluid infiltration and precipitates interstitial and alveolar edema.

We report a case of unusual presentation of Negative Pressure Pulmonary Edema during and after extubation, which highlights the importance of early recognition and timely intervention to prevent further deterioration.

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Desaturation and pink frothy discharge from the mouth or nose is a red flag, suggestive of negative pressure pulmonary edema. It has been noted as one of the complications of upper airway obstruction, which might be misdiagnosed[1].

The aim of this report is to highlight the unusual presentation of Negative Pressure Pulmonary Edema (NPPE) during and after extubation and to highlight the importance of early recognition and timely intervention to prevent further deterioration and fatal outcome.

THE CASE

A thirty-one-year-old male weighing 87 kg and Grade 2 ASA; he was posted for left anterior cruciate ligament reconstruction; he was a football player. He was non-allergic, non-smoker and had good exercise tolerance. His airway examination showed Mallampatti grading 2; large tongue and Thyromental distance was approximately 4 to 6 cm. He had a history of esophageal surgery performed under general anesthesia without complications. The patient has chest pain occasionally and dyspnea on severe exertion. He was referred to the cardiologist for further evaluation. He had an echocardiogram which revealed ejection fraction of 50% to 55%. He was advised a nuclear scan, which revealed positive reversible stress ischemia in the inferior wall. Coronary angiography was within normal. The respiratory system was normal. Investigations revealed Hb: 14 gm/dl, platelet: 254, serum sodium: 137, potassium: 4.6 and coagulation profile was within normal.

Induction was done with Propofol 2 mg/kg, fentanyl 2 mcg/kg, Atracurium 0.5 mg/kg and the patient was intubated with Portex cuffed endotracheal tube size 8. Maintenance was achieved with oxygen and air (50/50) and sevoflurane as an inhalational agent with MAC 1. He was given morphine 7 mg, paracetamol 1 g and diclofenac 75 mg for analgesia. Ondansetron 4 mg and dexamethasone 8 mg were given as routine prophylaxis. He received 1,500 ml of Ringers lactate in the intraoperative period. Postoperatively, ultrasound guided femoral block with 10 ml of bupivacaine 0.25% and 10 ml of lidocaine 1% with epinephrine 1:100000 was performed with use of peripheral nerve stimulator for analgesia. The reversal was accomplished with Neostigmine 2.5 mg and Glycopyrrolate 0.5 mg at the end of the operation. The operation time was 150 min with minimal blood loss. He was extubated with good tidal volume and was stable hemodynamically.

Shortly after extubation, the patient had an extreme episode of cough and tongue fall; after which, desaturation occurred, and SpO2 fell gradually from 65% to 70%. Mask ventilation was attempted immediately with 100% oxygen with positive airway pressure and the SpO2 elevated up to 90% to 94%. A provisional diagnosis of negative pressure pulmonary edema was made. The patient desaturated to SpO2 70%. He was ventilated with BiPAP. Chest X-ray findings were suggestive of pulmonary edema, see figure 1. The patient was given furosemide 60 mg IV, urinary catheterization was performed and Nitroglycerine infusion was started. The patient was conscious, oriented, hemodynamically stable and maintaining saturation above 95% with BiPAP. ECG showed sinus tachycardia, ABG results were pH: 7.3, PaCO2: 48 mmHg, HCO3: 28 mmol/L and cardiac enzymes were normal. ECG revealed normal left ventricular dimensions with apparently preserved contractility with ejection fraction 55%. He stayed in recovery area 4 hours with intermittent BiPAP and then transferred to high dependency unit where he maintained saturation of 100% with nasal cannula on 6 L/min for 24 hours. Gradually, his saturation was 100% on room air and chest X-ray improved, see figure 2. The patient was discharged.

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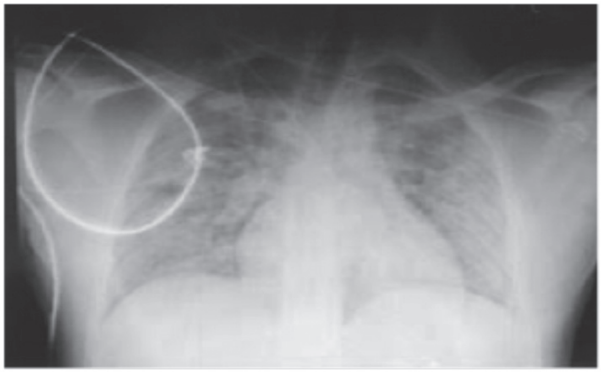

Figure 1: Chest X-ray Immediately after Intubation Suggestive of Pulmonary Edema

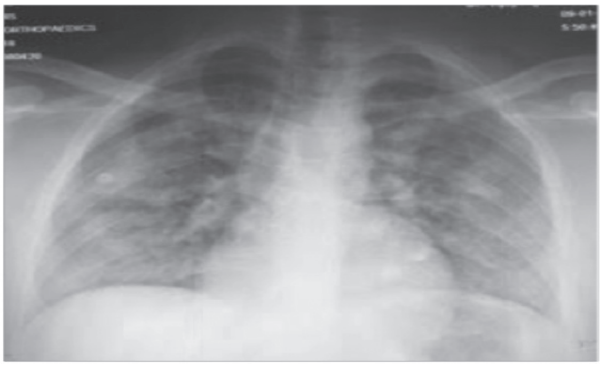

Figure 2: Chest X-ray 24 Hours after Extubation showing Improvement of Pulmonary Edema after Management

DISCUSSION

NPPE is an uncommon complication of anesthesia. It was first described in 1927 and 1977 by Morre and Oswalt respectively[2]. NPPE is classified into two types: type 1, laryngeal spasm induced pulmonary edema, which occurs immediately after onset of acute airway obstruction and type 2, which develops after relief of chronic upper airway obstruction[3]. Risk factors for developing NPPE are hanging, strangulation, foreign body, upper airway tumor, epiglottitis, croup, choking, large tonsils and hypertrophic adenoids[3]. The general incidence of NPPE is 0.05% to 0.1% of all anesthetic practice. However, other studies found that the incidence of NPPE varies according to the types of NPPE: type one, 9.2% to 12% and type two, 44%[2,4].

NPPE occurs after obstruction of the airway leading to an increase in intrathoracic negative pressure for approximately -50 to -100 mmHg (normal range -3 to 5 mmHg) which results in an increased systemic venous return, leading to high left ventricular afterload and preload pressure. That leads to an increase in pulmonary microvascular pressure which results in pulmonary edema. Hypoxia could mimic neurogenic pulmonary edema[3]. In chronic obstruction, there would be an auto Positive End-Expiratory Pressure (PEEP) with an increased end-expiratory lung volume. After removal of the obstruction, the lung volume and pressure return to normal level. If the pressure severely drops, it will result in pulmonary edema[5]. Our patient showed signs and symptoms of type 1 NPPE. Differential diagnosis of NPPE could be lymphatic insufficiency, aspiration of gastric contents, acute respiratory distress failure, acute radiation pneumonitis and acute hemorrhage pancreatitis[2].

Management of NPPE is to relieve airway obstruction. Supplementation of oxygen, positive end expiratory pressure or non-invasive positive pressure ventilation with Continuous Positive Airway Pressure (CPAP) or BIPAP is helpful along with diuretics unless the patient is hypovolemic. Mechanical ventilation with endotracheal intubation may be required in severely sick patients, elderly, Acute Respiratory Distress Syndrome (ARDS) or patients with pneumonia[6]. Early recognition of the problem would decrease mortality rate by 90%. In general, NPPE resolves after 12 to 24 hours[7].

CONCLUSION

NPPE is a non-cardiogenic disease which may occur after a severe laryngeal spasm, but early identification and treatment significantly reduce morbidity and mortality.

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Competing Interest: None

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