Case Report

Negative Pressure Pulmonary Edema: A Case Report

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ABSTRACT

Negative-pressure pulmonary edema (NPPE) is a noncardiogenic pulmonary edema in which a large, negative intrathoracic pressure generated against an obstructed upper airway results in the shift of fluid into the interstitium of the lungs. It is the life-threatening complication if remains undiagnosed and untreated promptly. We present a case of NPPE that occurred after general anesthesia for ear surgery. After completion of the surgery and reversal of residual neuromuscular blockade, the patient was extubated. Immediately after extubation, the patient stopped breathing and was desaturated which was managed with positive pressure ventilation with bag and mask. Soon after this, the patient was breathing spontaneously and maintaining saturation. After 45 min in recovery room, the patient developed coarse bilateral crepitations and pink frothy sputum on coughing and tachypnea. A tentative diagnosis of NPPE was made. Continuous positive airway pressure was applied. In addition, furosemide and nebulization with salbutamol and budesonide were administered. By the night, the symptoms had almost disappeared and the next day, the patient was shifted to the ward.

Keywords: Continuous positive airway pressure, negative pressure pulmonary edema, upper airway obstruction

INTRODUCTION

Negative pressure pulmonary edema (NPPE) is an underreported phenomenon of an acute disturbance in normal lung physiology that can result in life-threatening respiratory failure.[1] It is the noncardiogenic pulmonary edema, caused by upper airway obstruction and rapid negative intrapleural pressure increasing due to attempts of inspiration against the obstruction. NPPE is a dangerous clinical complication during the recovery period after general anesthesia.[2] NPPE following tracheal extubation is uncommon (0.05–0.1%)[2,3] and acute upper airway obstruction occurred following laryngospasm is the main reported cause of NPPE.[4] We present a case of NPPE that occurred in an adult patient after general anesthesia for ear surgery.

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CASE REPORT

A 17-year-old male patient (weight 55 kg) was admitted to our hospital with chronic suppurative otitis media left ear and was scheduled to undergo left modified radical mastoidectomy. He had a history of cough and cold 15 days back which was treated. There was no significant medical history. General examination, systemic examination, and laboratory reports were unremarkable. The patient was classified with the American Society of Anesthesiologists physical status I. The patient was fasted for 10 h before surgery. The patient was monitored with pulse oximetry, non-invasive blood pressure (BP), $EtCO_2$, and electrocardiography throughout the surgery. The patient was premedicated with inj Glycopyrrolate 0.2 mg iv., inj Emset 4 mg iv, inj Fentanyl 100 mcg iv, inj Midazolam 1 mg iv and immediately after which the patient was induced with inj Propofol 100 mg iv and succinylcholine 100 mg iv. The patient was intubated with 8 no. portex cuffed

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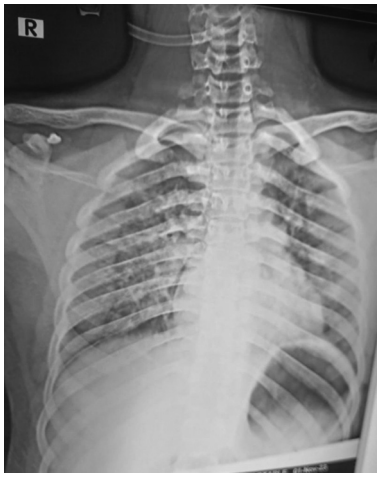
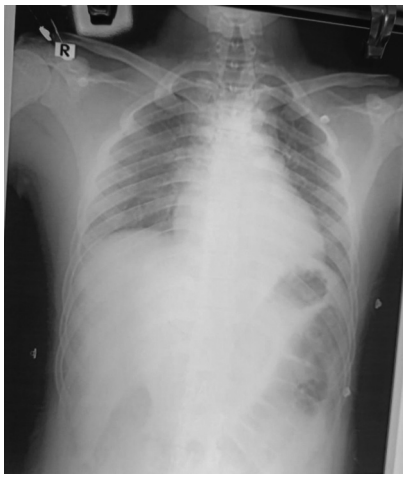
endotracheal tube. During the operation, anesthesia was maintained by regulating inhalation concentration of sevoflurane between 1.5% and 2%, intermittent doses of atracurium, O_2:N_2O 50:50. Tidal volume and rate were adjusted to maintain $EtCO_2$ between 35 and 45 mmHg. Surgery lasted for 4 h. (Completed at 2 pm) 1000 mL of ringer lactate was given throughout the surgery. Blood loss was minimal. As major blood loss was not expected, the patient was not catheterized. After surgery patient started breathing spontaneously hence patient was reversed with inj. Glycopyrrolate 0.4 mg iv and inj. Neostigmine 2.5 mg iv. After reversal patient was breathing spontaneously, regularly, and generating adequate tidal volume. The patient was sedated but arousable. The patient was extubated as the patient was coughing on the tube. Immediately after extubation patient developed respiratory distress, stridor with no movement of bag of bains circuit with sudden fall in saturation from 100% to 78%. Meanwhile, we tried to ventilate with positive pressure with bag and mask. There was increased resistance to bag which was gradually decreased and saturation improved to 100%. The patient was sedated, arousable, and maintaining saturation 99% at O_2 at 4 L/min with venti mask. After 5 min patient had one episode of vomiting and immediately suctioning and lateral position was given. On auscultation chest was clear and maintaining saturation 99% at O_2 at 4 L/min with venti mask. After 30 min in recovery room, it was observed that after discontinuation of oxygen, saturation dropped to 89–90%, tachypnoea up to 20–24/min and pink frothy sputum on coughing, heart rate 110–115, BP 100–110/80 and coarse bilateral crepitations on auscultation. Then propped-up position was given, oxygen continued and inj Lasix 60 mg i.v. given and then the patient was shifted to surgical intensive care unit (SICU). The patient was then catheterized in SICU. The patient started on non-invasive ventilation with FiO_2 50%, Positive end-expiratory pressure 5, respiratory rate- 18, and PS- 8. The patient was also started on nebulization with Salbutamol and Budesonide, Inj. Monocef and Inj. Metronidazole continued. Arterial blood gas analysis showed pH 7.40, PCO_2 31.5, and pO_2-214. Chest X-ray showed bilateral infiltrates.

In the evening after 4–5 h of extubation, respiratory distress improved with minimal crepitations on auscultation. In the morning patient was comfortable with no tachypnoea, no crepitations with stable vitals and improved chest X-ray findings.

In the morning, the patient was shifted to ward and then discharged to home without any complications.



X ray (2 hours post extubation)



X ray (16 hours post extubation)

DISCUSSION

NPPE is an acute noncardiogenic pulmonary edema that can be a perioperative life-threatening complication without timely diagnosis and treatment.

The principle mechanism noted in literature is inspiration against an acute airway obstruction causing a profoundly negative intrathoracic pressure. As a result of this, venous return to the cardiopulmonary circuit increases significantly. Subsequent and/or concurrent hypoxia contributes as well. Hypoxia triggers the release of catecholamines, resulting in increased systemic vascular resistance and pulmonary vasoconstriction. Both increased negative intrathoracic pressures and increased systemic vascular resistance contribute to an increased afterload and decreased left ventricular ejection fraction.

Increased afterload and increased volume contribute immensely to raised hydrostatic pressures within the cardiopulmonary circuit. This imbalance in Starling Forces manifests as pulmonary capillary volume shifts from vascular space to interstitium.[1]

It is important to notice the potential causes, make a rapid differential diagnosis, and determine the effective treatment during the perioperative period before disease aggravation occurs. In this case, A total of 1000 mL of the ringer lactate was administered at a constant speed and was balanced with perioperative fluid loss. Therefore, pulmonary edema caused by fluid overload was prevented. Our patient was healthy with no history of cardiovascular disease, thus cardiogenic pulmonary edema was ruled out. And there was no drug-associated allergy during the entire perioperative period. Our patient developed pulmonary edema after one episode of acute airway obstruction which was developed after extubation hence differential diagnosis of NPPE was made along with aspiration pneumonia as the patient had one episode of vomiting after management of laryngospasm.

Acute laryngeal spasm during extubation is the most common reported cause of NPPE which was the causative factor in our case.[5,6] Our patient had a history of cough 15 days back that is one of the contributory factor for laryngospasm at extubation.[7] Airway hyperreactivity may require 6 weeks or more to heal.[8] The incidence of NPPE is more than 50% among men following laryngospasm.[6] Muscular healthy patients are at increased risk of NPPE because of their ability to generate significant inspiratory force.[9]

Prior to extubation, we must ensure that the patient is completely awake, thus regaining optimal upper airway muscle tone. In several cases, the development of pulmonary edema is delayed for several hours. Because of this delayed onset, patients who experience postanesthetic laryngospasm should be observed as long as 2–3 h.[10,11] Our patient developed crepitations and tachypnea after 45 min of extubation.

Although NPPE does not result from fluid overload, it is recommended gentle diuresis using low-dose furosemide in the treatment regimen. This clinical syndrome can usually improve after 12–24 h after treatment.[3,5,6,11] Our patient experienced a recovery after assisted ventilation via CPAP, and furosemide with rapid and complete resolution of both clinical and radiological features in <24 h, and there were no sequelae once recovery occurred. Therefore, a diagnosis of NPPE was made,[2,3] because such rapid recovery is not expected after aspiration pneumonia.

CONCLUSION

In conclusion, NPPE should be suspected in patients who develop acute respiratory distress following extubation, particularly when there is no history of cardiac illness or cardiovascular risk factors. This case report highlights the importance of early diagnosis and prompt management of NPPE which is essential to prevent morbidity and mortality in patient.

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