Negative Pressure Pulmonary Oedema in a young adult after Cortical Mastoidectomy: A Case report'

WG Cdr Gaurav Pandey[1], Maj Madhukar Upadhyay[2]

ABSTRACT

Introduction: Negative pressure pulmonary oedema (NPPO) is a uncommon complication of acute upper airway obstruction post laryngeal spasm. It is potentially fatal condition with a multifactorial pathogenesis, which occurs due to exponential increase in negative intrathoracic pressure generated by forced inspiration against a closed glottis. NPPO is usually reported within few mins of tracheal extubation. We hereby report a rare case of NPPO in a young adult after 20 min post tracheal extubation in post op recovery unit after cortical mastoidectomy under general anaesthesia and its timely management.

Keywords: Negative Pressure Pulmonary Oedema, Laryngeal Spasm, Cortical Mastoidectomy

INTRODUCTION

Negative pressure pulmonary oedema (NPPO) is a well-documented yet uncommon complication of acute upper airway obstruction. It is potentially fatal condition with a multifactorial pathogenesis, which occurs due to exponential increase in negative intrathoracic pressure generated by forced inspiration against a closed glottis, leading to extravasation of fluid into the alveolar spaces. The onset is usually immediately after tracheal extubation, we hereby report a rare case of NPPO in a young adult after 20 min post tracheal extubation in post op recovery unit after cortical mastoidectomy, its timely recognition and management.

CASE REPORT

A 17 yrs. old 64 kg S/o Ex-serviceman, case of CSOM (Rt) was planned for cortical mastoidectomy under general anaesthesia. His medical history, physical examination and investigations revealed nothing unusual, except for symptoms and signs of hearing loss and ear discharge for duration of 1 year, he was accepted under ASA I under general anaesthesia.

The surgery was planned under general anaesthesia. The patient was given Tab Diazepam 5 mg and Tab Ranitidine 150 mg and kept nil orally after 2200h a night prior to surgery. He was premedicated in the operation theatre with Inj Glycopyrolate 0.2 mg, Inj Fentanyl 80 mcg IV and Inj Ondensetron 4mg IV. He was induced with Inj Propofol 100 mg IV. Endotracheal intubation and muscle relaxation were obtained with Inj Vecuronium 6 mg IV. The patient was maintained on oxygen, nitrous oxide and isofluorane in appropriate concentrations. The tracheal intubation, mechanical ventilation, and surgical procedure were uneventful. Total fluids given were 1300ml Ringer lactate. The patient remained haemodynamically stable throughout the surgery and maintained oxygen saturation of 99-100%. The surgery went for 2h20min, following which, the reversal was achieved with Inj Neostigmine 3.5 mg IV and Glycopyrrolate 0.6 mg IV and the tracheal extubation was done after the patient was fully awake. After 5 min of oxygenation via face mask in operation theatre, patient was shifted to recovery bay in Operation Theatre. Patient was fully conscious and was generating good tidal volumes, vitals were stable. Oxygen with face mask at 6 L/min was started and SpO2 monitoring was attached, which was around 99%. Within another 15 min of reaching in recovery bay, patient started having dry cough, became restless and had a breath-holding episode followed by frothing from mouth. He was immediately shifted back to Operation Theatre and monitors were connected, a rapid desaturation to a SpO2 of 56% was noticed in the following minutes. The patient had clenched his teeth and his mouth opening was impossible. Mask ventilation was difficult and seemed inefficient. Inj Succinylcholine 100 mg and Propofol 80 mg IV stat were given, the patient was immediately intubated and ventilation was assisted. In a few seconds after intubation pink frothy secretions appeared in the endotracheal tube. ABG done at this time of resuscitation showed severe respiratory acidosis with pH 7.05, PaO2 56, PaCO2 108, HCO3 32. On auscultation, he had fine crepitations in the mid and lower zones of both lungs. Inj furosemide 40 mg IV and Inj Hydrocortisone 200 mg stat were given. After observing the patient for 30 min in Operation Theatre he was shifted to ICU and put on mechanical ventilator on PSIMV (Pressure-synchronized intermittent mandatory ventilation) mode with PEEP of 8 cm of H_2O and Pinsp titrated to achieve tidal volumes of 360-400ml, Peak pressures were around 32. His SpO2 and ABG improved gradually. The chest radiograph revealed bilateral pulmonary infiltrates. The next morning after stabilization of ABG and reassessment, he was given T-piece trial for 30 min, and extubated uneventfully after

[1]Assistant Professor, Department of Anaesthesiology & Critical Care, 158 Base Hospital, Bagdogra – 734014, West Bengal, [2]Assistant Professor, Department of ENT, 158 Base Hospital, Bagdogra – 734014, West Bengal, India

Corresponding author: Wg Cdr Gaurav Pandey, Assistant Professor, Department of Anaesthesiology & Critical Care, 158 Base Hospital, Bagdogra – 734014, West Bengal, India

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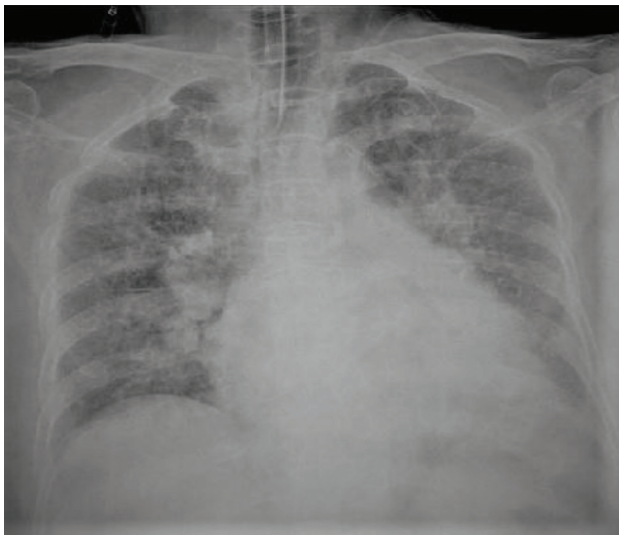

Figure-1: X-Ray chest: Showing B/L infiltrates post re-intubation

being fully awake. He was observed for a day in ICU and then shifted to the ward.

DISCUSSION

Negative pressure pulmonary oedema (NPPO) represents a form of hydrostatic oedema.[1] NPPO has also been described as 'laryngospasm - induced pulmonary oedema' and 'post-obstructive pulmonary oedema'.

It occurs characteristically after relief of upper airway obstruction, generally following laryngospasm.

Oswalt et al are accredited with the first description of negative pressure pulmonary oedema (NPPO). [7]

The incidence of NPPO, as a complication of all anaesthetics, is found to be 0.05-0.1%.[2] Approximately 11% of patients who develop laryngeal spasm go on to develop NPPO.[9] The morbidity and mortality of an un-recognized event of NPPO is as high as 40%.[3] The frequency depends mainly on the type of surgery, with greater incidence observed in post ENT procedures. [9]

Emergence laryngospasm is the most commonly reported anaesthetic cause of airway obstruction and negative pressure pulmonary oedema. Patients are usually young, athletic males who are capable of generating profound negative intra-thoracic pressure.[4] In young healthy patients, this negative pressure may be as high as -100 cm water.

Negative intra-thoracic pressure is the primary pathophysiological event in the initiation of NPPO; hypoxia, hypercarbia, acidosis, and hyperadrenergic state contribute to its development. Increased right heart filling, decreased left heart filling, increased left ventricular afterload and decreased LV ejection lead to increased pulmonary capillary hydrostatic pressure.[6] These factors increase the movement of fluid out of the pulmonary capillaries, resulting in alveolar and interstitial oedema.

Clinically after acute airway obstruction the events are followed by the rapid onset of respiratory distress, haemoptysis and clinical / radiological features. NPPO radiological manifests as Kerley lines, peribronchial cuffing and, in severe cases, as central alveolar oedema. Cardiac size is generally unchanged as hypervolaemia is usually not present.[5]

Differential diagnoses of NPPO include fluid overload, anaphylaxis, aspiration pneumonitis, acute lung injury, cardiogenic pulmonary oedema and drug-induced non-cardiogenic pulmonary oedema.

Laryngospasm during extubation can be prevented by extubating patients either deep or fully awake.[10]

Early relief of laryngospasm with use of neuromuscular blockers has been suggested to "break" the laryngospasm and put an end to the sustained negative intra-thoracic pressure, hyperadrenergic drive and hypoxia, implicated in the pathogenesis of NPPO. Most episodes of NPPO resolve within a few hours with requirement of oxygen therapy alone. A short period of mechanical ventilation and positive end expiratory pressure may speed up recovery. The role of loop diuretics is not certain. [8]

CONCLUSION

Negative pressure pulmonary oedema is an alarming perioperative emergency but with anticipation of this complication during endotracheal extubation, adequate preparedness, timely diagnosis and prompt management, the outcome is highly rewarding.

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