

CASE REPORT

Acute Pulmonary Edema in a Patient undergoing Appendectomy under Spinal Anesthesia

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ABSTRACT

Acute pulmonary edema, accumulation of large amount of fluid in the interstitial and alveolar space, in patients undergoing surgeries poses a great challenge to the Anesthetist. If not treated well, it could be associated with high mortality rate up to 40%. We report a case of a patient aged 21 years undergoing emergency appendectomy under spinal anesthesia who developed acute Negative Pressure Pulmonary Edema and was successfully managed. His preoperative blood investigations were normal and had no other comorbidities.

Keywords: Negative pressure pulmonary edema, Pulmonary aspiration, Spinal anesthesia.

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INTRODUCTION

Negative pressure pulmonary edema (NPPE) is a rare but dangerous form of noncardiac pulmonary edema with a mortality of 40%. Recognition of patients with predisposing factors for upper airway obstruction is important in the diagnosis of NPPE, which is often confused with pulmonary aspiration. Signs and symptoms are subtle and edema is usually self-limiting. Here, we describe a case of an adult male coming for emergency appendectomy, who was administered spinal anesthesia and developed acute pulmonary edema and was managed successfully.

CASE REPORT

A 21-year-old patient visited the emergency department with complaint of pain in abdomen. The patient was

diagnosed with acute appendicitis and was posted for emergency open appendectomy. His laboratory investigations were within normal limits. On examination, patient was moderately built with height of 168 cm and moderately nourished with weight of 78 kg and was scheduled for surgery under American Society of Anaesthesiologists physical status 1 "E". He did not receive any premedication. After securing intravenous (IV) line with 20G cannula, Ringer lactate solution was started. Routine mandatory monitors namely pulse oximeter, noninvasive blood pressure, and 3-lead electrocardiogram were instituted. Under strict aseptic precautions, spinal anesthesia was given at L3–4 (lumbar) interspace by midline approach in sitting position with 16 mg of 0.5% hyperbaric bupivacaine and 60 µg of buprenorphine, using 25G Quincke's spinal needle after confirming clear and free flow of cerebrospinal fluid. Immediately after injection of local anesthetic agent, the patient was placed in supine position. We achieved and confirmed a sensory segmental level of T8 (thoracic) by cold sponge method. Oxygen at 5 L/min was given through disposable face mask. Intraoperatively, his vital signs were within normal limits. Patient complained of pain at the surgical field after 20 minutes of starting the surgery, following which he developed bradycardia and his heart rate was 40 bpm. Surgeon was asked to stop manipulation of bowel and bolus of injection atropine 0.6 mg was administered and 100% oxygen at 5 L/min with Bain's circuit was initiated. Patient had one episode of vomiting for which injection Emiset (ondansetron) 8 mg was administered. Two minutes later, patient developed hiccups and started complaining of difficulty in breathing. Patient was restless, tachypneic with nasal flaring, and was maintaining a saturation of 80% with 100% oxygen. On auscultation, bilateral crepitations were heard all over the lung fields. Suspecting aspiration, intubation was planned. Patient developed cough following which pink frothy sputum was noticed from nostrils as well as from mouth and patient began losing consciousness. Patient was immediately intubated with 8 mm cuffed endotracheal tube and ventilated with 100% oxygen and positive end-expiratory pressure (PEEP) of 8 cm H₂O. His oxygen saturation came down to 60%. Injection furosemide 40 mg and injection morphine 5 mg were administered IV. Intermittent endotracheal tube suctioning was carried out to clear the frothy secretion. Patient was rapidly desaturating and developed second episode of bradycardia which

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failed to respond to injection atropine 0.6 mg and heart rate dropped to 20 bpm. Pharmacological intervention with injection adrenaline 1 mg and cardiopulmonary resuscitation (CPR) was initiated simultaneously. After one cycle of CPR, heart rate improved to 170 bpm, peripheral oxygen saturation 92%, and blood pressure (BP) 180/110. Controlled ventilation with 100% oxygen and PEEP was continued. We administered a second dose of injection furosemide 40 mg IV and also repeated 3 mg of injection morphine IV. In order to facilitate elective ventilation, a nondepolarizing muscle relaxant, injection vecuronium 4 mg, was administered. Saturation improved to 96% and BP came down to 150/90. However, patient had persistent tachycardia with heart rate ranging from 100 to 130 bpm. Postoperatively, patient was shifted to surgical intensive care unit (SICU) for monitoring. He was ventilated overnight and in the morning when rhonchi and crepitations had cleared, he was put on synchronized intermittent mandatory ventilation mode and planned extubation was performed. On the 2nd postoperative day, he was shifted to surgical ward for further management.

DISCUSSION

The NPPE is a noncardiac pulmonary edema which is rare. It could be a potentially life-threatening complication. Generation of high negative intrathoracic pressure against closed glottis (Müller's maneuver) creates a large increase in venous return leading to increased preload and increased pulmonary capillary pressure.¹ This causes the extravasation of fluid and red blood cells, due to disruption of capillary membrane, into the interstitial and alveolar spaces.² The NPPE as a complication of upper airway obstruction has been reported in 1977 by Oswald et al.³ The reported incidence of NPPE varies from 0.05 to 1%. NPPE usually develops after upper airway obstruction, also called as laryngeal spasm.³⁻⁶ Factors that may

predispose patients to laryngospasm are recent upper airway infection, history of reactive airway disease, male gender, young age, dry cough, laryngeal irritation due to secretions, blood, gastric regurgitation, and pressure and temperature changes. Stimulation of airway results in coughing, bronchospasm, apnea, and vocal cord closure. In an anesthetized patient, this may prolong causing marked desaturation, fluctuations in BP and heart rate, vagal-mediated bradycardia, and hypoxia. This is followed by sympathetic stimulation causing tachycardia and hypertension. Ultimately, severe hypoxemia sets in, causing severe bradycardia and cardiac arrest.^{7,8} Signs of respiratory distress, such as cough and tachypnea, along with decrease in arterial oxygen saturation, inability to maintain saturation >90%, pink and frothy sputum through nostrils and mouth (if intubated, through the endotracheal tube) follow.^{8,9} Patients will have sweating and prominent lung signs like rhonchi and coarse crepitations.

X-ray of the chest may reveal diffuse bilateral interstitial and alveolar infiltrate (white out area).⁹ Arterial blood gas can be normal or show a picture of acute respiratory acidosis.⁹ This condition has to be differentiated from pulmonary aspiration, acute respiratory distress syndrome, pulmonary embolism, and administration of large volumes of IV fluids. The ratio of total protein concentration between pulmonary edema fluid and plasma differentiates between hydrostatic pulmonary edema and acute lung injury. Ratio of <0.65 is characteristic of hydrostatic pulmonary edema, whereas >0.75 to 1% is characteristic of acute lung injury.¹⁰ Early diagnosis of NPPE is very crucial, as it affects postoperative morbidity and mortality. In our case, vasovagal shock caused by handling of bowels might have initiated the sequence of events leading to laryngospasm and NPPE. In our patient, postoperatively, X-ray chest revealed typical findings of pulmonary edema (Fig. 1), which got resolved by 2nd postoperative day (Fig. 2). Strategic treatment of NPPE

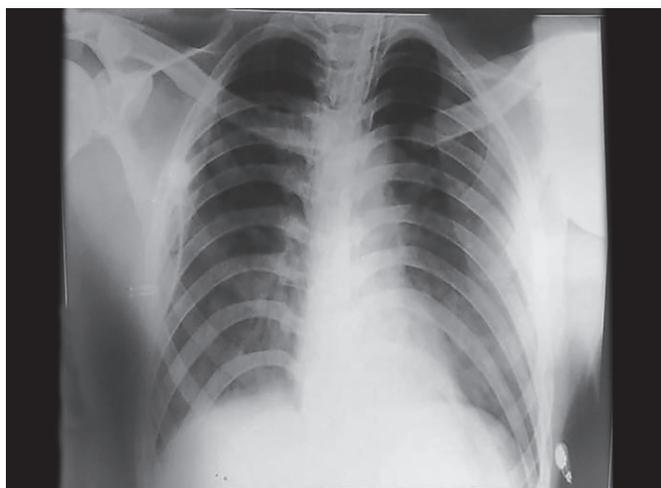


Fig. 1: Posteroanterior view of chest X-ray in the SICU on the first postoperative day



Fig. 2: Posteroanterior view of chest X-ray on the second postoperative day

includes maintenance of airway by tracheal intubation, providing supplemental oxygen, needing controlled ventilation, use of diuretics, good analgesia, and use of phosphodiesterase inhibitors (aminophylline) whenever needed as we did in our case.

CONCLUSION

The NPPE has a mortality rate ranging from 11 to 40%.¹¹ We must aim at early diagnosis and management in these patients, which helps in resolving NPPE within 24 to 48 hours which in turn decreases mortality and morbidity.

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