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NEGATIVE PRESSURE PULMONARY EDEMA: A CASE SCENARIO

Anesthesiology

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ABSTRACT

Negative pressure pulmonary edema (NPPE) is a form of noncardiogenic pulmonary edema (PE) that results from the generation of high negative intrathoracic pressure (NIP) needed to overcome upper airway obstruction. Negative pressure pulmonary edema (NPPE) is a dangerous and potentially fatal condition. The principal mechanism involved are upper airway obstruction, the large negative intrathoracic pressure generated by forced inspiration against an obstructed airway.Development of noncardiogenic pulmonary edema has been observed after a variety of inciting events including upper airway obstruction (negative pressure pulmonary edema [NPPE])[1], anaphylaxis[2], fluid maldistribution[3,4] and severe central nervous system trauma (neurogenic pulmonary edema).[5] Early diagnosis, proper understanding of its pathophysiology is necessary to provide optimal treatment and to avoid high mortality rate associated with it.

KEYWORDS

INTRODUCTION-

NPPE was first demonstrated in 1927 by Moore in spontaneously breathing dogs exposed to resistive load[6]. Negative-pressure pulmonary edema (NPPE) or postobstructive pulmonary edema is a well-described cause of acute respiratory failure that occurs after intense inspiratory effort against an obstructed airway, usually from upper airway infection, tumor or laryngospasm. The presentation of NPPE can be immediate or delayed, which therefore necessitates immediate recognition and treatment by anyone directly involved in the perioperative care of a patient[7]. This disorder is classified as Type I or Type II[8]. Type I NPPE develops immediately after onset of acute airway obstruction and Type II NPPE develops after the relief of chronic upper airway obstruction. As Type I NPPE develops usually with upper airway acute obstruction or after manipulation of the airway surgically, some authors call it laryngeal spasm-induced pulmonary edema.Other factors that increase the risk of Type I NPPE are hanging, strangulation, upper airway tumors, foreign bodies. The objective of this report is to describe a case of perioperative NPPE occurred during induction secondary to laryngospasm in an adult patient, who underwent a diagnostic laproscopy for primary infertility under general anesthesia and how we managed it.

PATHOPHYSIOLOGY-

The pathophysiology of NPPE has been extensively reviewed by several studies.NPPE begins with a significant upper airway obstruction, inspiratory efforts to overcome the obstruction generate highly negative intrapleural and alveolar pressures, and the high pressure gradient causes fluid to move out of the pulmonary capillaries and into the interstitial and alveolar spaces.[9] The pathophysiology of NPPE is attributed to four major mechanisms: Disturbances of pulmonary fluid homeostasis can be induced by four pathways that can lead to increased interstitial fluid, increased hydrostatic pressure in the pulmonary capillary bed, decreased osmotic pressure of plasma, increased permeability of the membrane and decreased return of fluid to the circulation.

CASE PRESENTATION-

A 28-yr-old female (weight 56 kg, height 163 cm) presented to the gyaenecology department of PGIMS Rohtak for diagnostic laproscopy for primary infertility. She was in American Society of Anesthesiologists grade I and her blood investigations and chest x-ray were within normal limits. The patient was premedicated with injectable ondansetron 0.1 mg/kg, injectable glycopyrrolate 10 µg/kg, injectable midazolam 0.05 mg/kg, and injectable fentanyl citrate 2 µg/kg. Intraoperative monitoring included electrocardiography, blood pressure monitoring, blood oxygen saturation by pulse oximetry (SpO_2), and end-tidal capnography. The patient was preoxygenated with 100 % oxygen through a face mask. Injection propofol 120 mg given through 18 G IV canula slowly. Then after checking for able to ventilate she was given injection atracurium 25 mg. On continuing bag mask ventilation her saturation started falling and pink, frothy sputum was noticed coming through her mouth.She was intubated immediately. Bilateral air entry checked and endotracheal tube fixed at 19 cm.Bilateral crepts were present on auscultation. A probable diagnosis of NPPE was made. She was treated with endotracheal suctioning,positive pressure ventilation and injection frusemide 40 mg.Patient regained 100% saturation. Diagnostic laproscopy completed uneventfully and patient shifted to intensive care unit where she was treated with positive pressure ventilation and injection frusemide. She was extubated next day.

Disscussion -

NPPE is defined as a form of noncardiogenic pulmonary edema that results from the generation of high negative intrathoracic pressure following spontaneous breathing against upper air way obstruction. The incidence of developing Type I NPPE associated with acute postoperative upper airway obstruction is 9.6–12%, whereas the incidence of developing Type II NPPE is 44%[10].In adult population about 50% of NPPE occurrences are due to postoperative laryngospasm. Risk factors associated with NPPE include obese individuals with difficult intubation, presence of any airway lesions, history of nasal, oral or pharyngeal surgery. The most common cause of postobstructive pulmonary edema is laryngospasm during intubation or after anesthesia in the postoperative period[11]. Patient usually present with decreased oxygen saturation, pink frothy secretions. Our patient also had tachypnea, pink, frothy secretions and rapidly decreasing oxygen saturation. The differential diagnosis includes aspiration of gastric contents, acute respiratory distress syndrome, volume overload, anaphylaxis, and airway obstruction. Proper diagnosis and rapid treatment are essential to alleviate this respiratory complication. The first treatment priority should be relief of the airway obstruction and correction of hypoxemia. Pulmonary edema is than addressed with a diuretic unless the patient is hypovolemic. Most of cases can be treated with effective airway management,positive pressure ventilation and diuretics. There is no effective way to prevent negative pressure pulmonary edema but can be minimized with minimal irritation of larynx.

CONCLUSION-

Negative pressure pulmonary edema is a common condition seen in anaesthesia practice. Early diagnosis and prompt intervention may significantly improve overall outcome.

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