v. 3, n. 20, 2023

International Journal of Health Science

NEGATIVE PRESSURE PULMONARY EDEMA AFTER OROTRACHEAL EXTUBATION IN EMERGENCY SURGERY

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Abstract: The objective is to report the case of a patient undergoing emergency partial colectomy due to acute perforative abdomen who developed negative pressure pulmonary edema after orotracheal extubation. Information was obtained through chart review, patient interview, photographic record of diagnostic methods, and literature review. MLR, 37 years old, male, previously healthy, admitted to the hospital complaining of abdominal pain. He was admitted to the operating room for laparotomy due to a diagnosis of pneumoperitoneum. Anesthetic induction was uneventful. Intraoperatively, the parameters remained stable. At the end, anesthetic weaning and extubation were performed. The patient evolved with superficial respiration and use of accessory muscles, with a rapid decrease in peripheral saturation. Based on intraoperative normality parameters, employed protective ventilation and altered auscultation, acute pulmonary edema due to negative pressure was identified. Positive pressure ventilation was performed using a face mask, with increased saturation and improvement of respiratory pattern. Ventilation was necessary until the patient ceased discomfort and remained stable with oxygenation through nasal catheter. Despite being a well-described clinical entity, this is a little known complication. Early identification and appropriate treatment are essential for clinical outcome.
Keywords: Negative pulmonary pressure edema, Post-anesthetic complication, Case report.

INTRODUCTION

Negative pressure pulmonary edema (NPPE) is a relevant complication of general anesthesia and may occur in situations considered daily in practice, such as in cases of laryngospasm or upper airway obstruction (SILVA LAR, et al., 2019). There is a literary report of 4% incidence due to general causes other than postoperative ones, while others point to NPPE as a serious complication in general anesthesia with orotracheal intubation in up to 0.1% of cases; but, due to the underdiagnosis of the clinical condition, which is unfamiliar, its true incidence may be even higher (MCCONKEY PP, 2000; HIGINO A, et al., 2017; RUIZHU L, et al, 2019).

NPPE is a potentially life-threatening condition due to a change in the ventilation/perfusion ratio that triggers rapid hypoxemia. It must be hypothesized in a patient with stable intraoperative conditions that, during or after orotracheal extubation, develops respiratory failure (MCCONKEY PP, 2000; LOUIS PJ and FERNANDES R, 2002).

METHODS

Information was obtained through review of the medical record, interview with the patient, photographic record of the diagnostic methods to which the patient was submitted and review of the literature.

RESULTS

MLR, 37 years old, male, physical educator, had been admitted to a regional referral public service due to abdominal pain for 2 days. Admitted to the operating room for surgical proposal of laparotomy due to pneumoperitoneum seen on computed tomography, confirming diagnosis of perforating acute abdomen.

In the pre-anesthetic evaluation, the patient denied comorbidities, reported practicing daily physical activity, and denied the use of continuous medications. He had undergone an appendectomy 10 years before, and denied intercurrences at the time.

Anesthetic induction was performed with fentanyl, lidocaine, propofol and rocuronium, and the patient was fasting. He remained throughout the intraoperative period with

parameters within normal limits, maintaining oxygen saturation (O2) measured through peripheral oximetry between 97 and 100%, tidal volume in weight-adjusted volume-controlled ventilation, partial pressure of carbon dioxide (EtCO2) between 30 and 40 millimeters of mercury (mmHg) in capnography and mean arterial pressure between 70 and 75 mmHg through non-invasive measurement. At the end of the procedure, anesthetic weaning was performed in the plane, and when a tidal volume of 600 ml was obtained in spontaneous ventilation and eye opening at the call, extubation was performed.

After about 2 minutes, the patient evolved with a change in breathing pattern, with shallow breathing and the use of subcostal and abdominal muscles, elevation of the sternal notch, and a rapid decrease in peripheral saturation to approximately 60%, despite the installation of a cannula with Supplemental O2. Auscultation revealed the presence of bilateral bullous stertoration. Due to normal intraoperative parameters and protective ventilation employed, the hypothesis of acute pulmonary edema due to negative pressure was raised. Imaging tests confirmed the diagnostic suspicion **(Figure 1), (Figure 2).**

Positive pressure ventilation with a face mask was then promptly started with increased saturation and improved breathing pattern.

Ventilation was continuously required for two hours after the onset of the event, until the patient's respiratory distress ceased and remained stable with supplemental oxygen through a nasal catheter. The patient was then kept in post-anesthetic recovery for 20 hours and sent to the ward after resolution of the condition.

DISCUSSION

Acute negative pressure pulmonary edema, also known as post-obstructive pulmonary edema, occurs due to a process of homeostatic imbalance between pulmonary fluids (BHATTACHARYA M, et al., 2016; LIM D, et al., 2023). The variables of Starling's law, which determine the fluid dynamics along the capillary bed are the hydrostatic and oncotic pressure and the permeability of the capillary membrane. The influx of liquids in the interalveolar space is limited by the alveolar epithelium, whose narrow intracellular junctions act as a barrier (LOUIS PJ and FERNANDES R, 2002).

The adjacent interstitial space is drained into the systemic circulation by the lymphatic system; The development of negative pressure pulmonary edema may occur due to increased hydrostatic pressure in the pulmonary capillary bed, increased permeability in the alveolar epithelium, decreased plasma oncotic pressure, or decreased absorption of interstitial fluid via the lymphatic route (FREMONT RD, et al., 2007; LEMYZE M and MALLAT J, 2014).

In the genesis of acute negative pressure pulmonary edema, the elevated intrapleural and alveolar negative pressure gradient occurs due to significant upper airway obstruction, as in laryngospasm, the presence of a large tumor, or the occurrence of upper airway infection - the latter entity being statistically important particularly among the pediatric population (BHATTACHARYA M, et al., 2016). Such a gradient generates increased venous return, decreased cardiac output due to reduced pulmonary drainage into the left atrium, and fluid transudation into the alveoli through the epithelium as the pressure in the pulmonary capillary increases while the interalveolar pressure reduces. Congestion is maintained due to fluid accumulation in the interstitial space, even after resolution of the airway obstruction condition (HIGINO A, et al., 2017).

There is greater susceptibility to acute negative pressure pulmonary edema in athletic men, due to the fact that the better developed muscles are able to generate extremely high negative inspiratory pressures. The mechanical stress of negative pressure could induce injury and rupture of intravascular membranes in the pulmonary capillaries (MCCONKEY PP, 2000; HIGINO A, et al., 2017; MOREIRA BL, et al., 2022).

The pathogenesis of pulmonary edema due to negative pressure appears to be multifactorial, with reports in the literature of both the biochemical characteristics of transudative and exudative edema (MATTHAY MA, et al., 2002). Normally, the patient who develops NPPE has no intraoperative intercurrences. Therefore, it is necessary to have a high degree of suspicion for the diagnosis. During or shortly after extubation, attention must be payed to signs of acute airway obstruction such as stridor, suprasternal and supraclavicular retractions, use of accessory muscles for inspiration, and panic in facial expression (KOH MS, et al., 2003).

Suspicion is higher in patients without intraoperative complications such as aspiration of gastric contents, volume overload, hypertension and without known personal factors that cause cardiogenic pulmonary edema. The clinical presentation involves rapidly onset of hypoxemia due to changes in the ventilation/perfusion ratio, pink foamy sputum, and radiographic findings (generally, interstitial and alveolar infiltrate). On auscultation, crackles and wheezing can be observed (VISVANATHAN T, et al., 2005; FREMONT RD, et al., 2007; ERDEN B, et al., 2020).

Risk factors for the development of negative pressure pulmonary edema are patients with airway injuries, upper airway surgery, obesity, obstructive sleep apnea, heart disease, orotracheal intubation, active smoking and emergency surgery (FREMONT RD, et al, 2007; TSAI PH, et al., 2018; WATANABE Y, et al., 2020). Studies also confirm the higher incidence of this complication among men (about 80%) after suffering laryngospasm after extubation and in patients classified as ASA I or II (73% of cases), again relating this complication to the high intrathoracic negative pressures produced by adult men healthy (BHATTACHARYA M, et al., 2016). It is also possible to observe in the literature reports of severe complications subsequent to the occurrence of NPPE.

Some of the main differential diagnoses are pulmonary thromboembolism, septic shock secondary to a previous infectious process and previously silent infection by SARS-COV-2; Imaging findings on radiography or CT scan are helpful in diagnosing NPPE. Common findings include extensive central consolidation associated with ground-glass opacity, suggestive of pulmonary edema (HOLZGREVE A, et al., 2020; SUZUKI Y, et al., 2022), in addition to a compatible clinical history.

The treatment priority is to relieve the airway obstruction and correct the hypoxia with supplemental oxygen. Positive pressure ventilation can be used as an alternative to orotracheal intubation, and may be necessary in 9 to 18% of cases. The use of ventilation measures, when necessary, aim to increase alveolar recruitment, reduce left ventricular afterload and increase cardiac output (MCCONKEY PP, 2000).

There is no evidence regarding the usefulness of using diuretics, which can exacerbate hypovolemia and hypoperfusion (FREMONT RD, et al., 2007).

Pulmonary edema is usually self-limited and resolves within 12 to 48 hours. No intervention has proven effective to prevent negative pressure pulmonary edema, but it

is recommended to avoid laryngeal irritation and the use of topical laryngotracheal anesthesia, performing careful laryngeal aspiration (VISVANATHAN T, et al., 2005).

FINAL CONSIDERATIONS

The case report aims to discuss factors related to the development of non-cardiogenic pulmonary edema after the end of anesthetic induction and recovery of spontaneous ventilation, its diagnosis and treatment. It is noted that, despite being a well-described clinical entity, it is a little-known complication. Early identification and institution of adequate treatment are essential in the clinical outcome.

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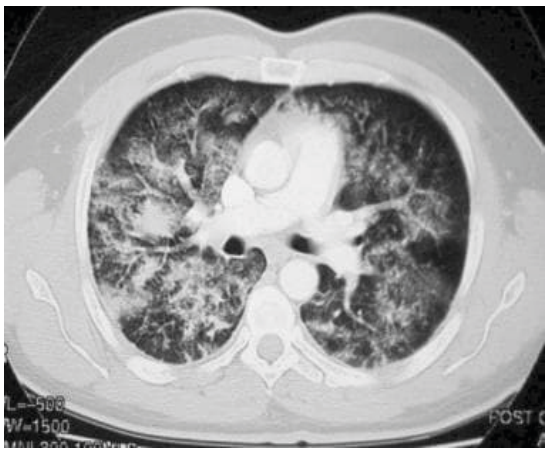

Figure 1. Non-cardiogenic pulmonary edema. Interstitial and alveolar congestion. (Source: own file)

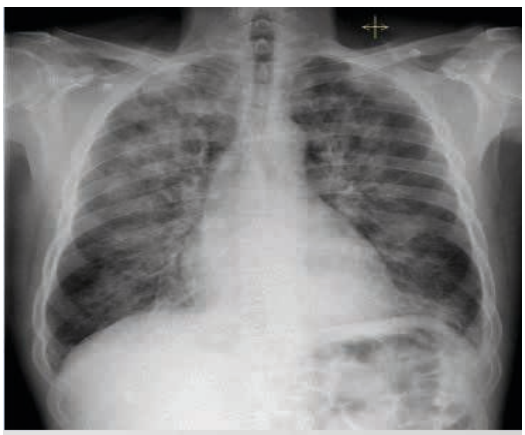

Figure 2. Chest X-ray demonstrating pulmonary congestion: Alveolar opacities distributed around the pulmonary hila, symmetrically (butterfly sign). (Source: own file)