ACUTE NEGATIVE PRESSURE LUNG EDEMA AFTER EXTUBATION IN RHINOPLASTY: CASE REPORT

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ABSTRACT

Introduction: Negative pressure pulmonary edema (NPPE) is a complication that is rarely described in the literature and has an incidence of 0.1% in patients undergoing general anesthesia, with a higher occurrence in otorhinolaryngological procedures. Pulmonary edema is a complication that appears after anesthesia with glottis closure during the inspiratory period, which significantly reduces intrathoracic pressure. Case report: Individual undergoing elective rhinoplasty. In the operating room with stable vital signs. Subjected to balanced general anesthesia: Pre-oxygenation under a face mask was induced with dexmedetomedine, sulfentanila, ketamine, propofol and atracurium, and periglottic block was performed with ropivacaine and lidocaine. The intraoperative period continued without complications. At the end of the surgery, the patient was extubated in plane due to the onset of intense laryngospasm and coughing. After extubation, the patient developed a drop in oximetry (50%), with the installation of a Guedel cannula and the Jaw-thrust maneuver and with improvement. important aspect of oximetry with supplemental oxygen (O2). Discussion: The formation of non-cardiogenic pulmonary edema has been observed following various forms of upper airway obstruction. The frequency is, however, dependent on the surgical method and procedures. Most of them appear immediately after extubation. The vigorous inspiratory effort against the closed glottis generates negative pressures in the pulmonary interstitium, which favors fluid transudation. The therapeutic approach involves treating the cause with supportive measures, airway permeabilization, administration of supplemental O2 via face mask, bronchodilators and, if necessary, non-invasive, or invasive mechanical ventilation.

KEYWORDS: PULMONARY EDEMA; LARYNGISMUS; RHINOPLASTY; DYSPNEA; OXYGEN

INTRODUCTION

Negative pressure pulmonary edema (NPPE) is a seldom-discussed complication in the literature, with an incidence of 0.1% in patients undergoing general anesthesia. It occurs more frequently in otorhinolaryngological procedures.[1] Pulmonary edema is a complication that arises post-anesthesia with glottis closure during the inspiratory period, leading to increased subatmospheric intrathoracic pressure. This negative pressure promotes transudation of fluid into the alveoli, resulting in the entire clinical presentation of NPPE.[2] Despite few scientific reports in the literature, NPPE is a significant issue in anesthesia practice, with a higher incidence during increased manipulation of the patient's airways, especially in the presence of laryngospasm.1 This article aims to present a case of NPPE, its diagnosis, mechanism of action, and treatment.

CASE REPORT

Female patient, 17 years old, ASA I, with no history of prior surgeries, comorbidities, or drug allergies. She arrives at the surgical center for an elective rhinoplasty at a hospital in Goiânia, Goiás, performed by an otorhinolaryngologist. The patient in question experienced flu-like symptoms one day before the surgery, and the surgeon was informed promptly. Immediate initiation of antibiotic therapy and corticosteroids resulted in a substantial improvement of symptoms on the day of the surgical procedure.

In the operating room, the patient was adequately monitored with pulse oximetry with plethysmographic waveform, electrocardiogram, and non-invasive blood pressure, exhibiting stable vital signs before anesthesia induction. A venous line was established with a 20G needle in the right upper limb. The patient underwent balanced general anesthesia: Preoxygenation with a facial mask using 100% oxygen at 6L/min for 3 minutes. Anesthesia induction was carried out with dexmedetomidine (60 mcg) infused in 250 ml of 0.9% saline, sufentanil (15 mcg), ketamine (20 mg), propofol (120 mg), and atracurium (30 mg). Periglottic block was performed with 10 ml of 0.5% ropivacaine and 1% lidocaine without vasoconstrictor. Tracheal intubation was done with a 7.0 cuffed tube, Cormack-Lehane 2b view, direct and atraumatic laryngoscopy confirming proper tube placement by capnography. Mechanical ventilation was adjusted to maintain an end-tidal CO2 (PETCO2) close to 35 mmHg. Anesthetic maintenance included 1.5-2% sevoflurane with low fresh gas flows of 2L/min on the anesthesia machine and remifentanil target-controlled as per the physician's discretion. Adjuvant medications used were dipyrone 2g, cephalexin 2g, dexamethasone 10mg, ondansetron 8mg, tenoxicam 40mg, and tranexamic acid 750mg.

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The intraoperative period was uneventful and the patient maintained stable vital signs throughout the period. At the end of the surgery, the patient was extubated in a flat plane due to the onset of intense laryngospasm and coughing, and after extubation the patient developed a drop in oximetry (50%). A Guedel cannula was installed and the Jaw-thrust maneuver was performed with a significant improvement in oximetry, 90-92% (with oxygen supplementation). Then, after clinical stabilization, she was taken to the post-anesthesia care unit (PACU), awake, conscious, using a mask with an oxygen reservoir at 7 L/min. After oximetry stabilization, SpO2 of 92-93%, eupneic, she was discharged home. On the same day of hospital discharge, the patient returns to the hospital emergency room due to dyspnea and a drop in O2 saturation (80%) verified by the mother at home. Due to significant clinical worsening and the need for supplemental O2, the patient continued to be admitted to the intensive care unit (ICU). Upon admission to the ICU, already clinically stable, however using supplemental oxygen, the patient underwent laboratory tests, electrocardiogram, viral panel, chest x-ray and chest tomography angiography for diagnostic purposes, the latter being represented by figure 01 bellow:

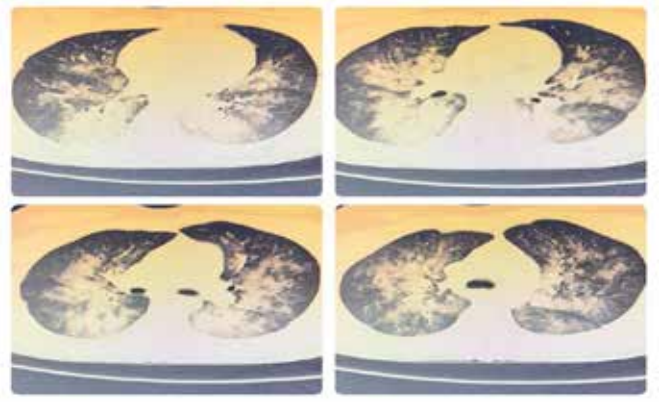

Figure 01: Angiotomography image of the patient's chest (parenchyma) showing confluent acinar and lobular opacities of consolidative and ground-glass nature, at times configuring a mosaic pattern.

The chest angiotomography revealed bilateral and diffuse pulmonary involvement, with a predominance in the right lung. It is characterized by confluent acinar and lobular opacities of consolidative and ground-glass nature, at times forming a mosaic pattern. No filling defects were identified in the evaluated pulmonary arterial branches that could suggest acute pulmonary embolism (PE), as shown in Figure 02. The angiotomography report also mentioned that the pulmonary findings require close clinical and laboratory correlation, considering infectious/inflammatory processes as a hypothesis. To rule out any suspicions of lung disease, the patient underwent a viral panel, COVID-19 test, and other infectious control exams.

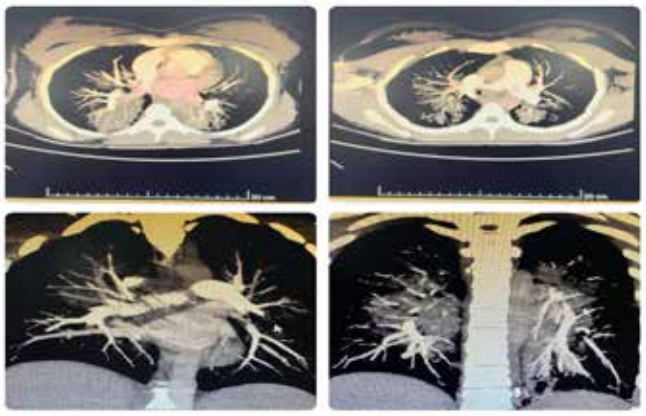

Figure 02: Image of angiotomography of the patient performed upon admission with the objective of ruling out acute pulmonary embolism (PE).

The patient's viral panel was negative for all viruses it included, including Coronavirus, Parainfluenza, Adenovirus, Influenza, Metapneumovirus, Rhinovirus, Bordetella, Mycoplasma, Respiratory Syncytial Virus, and all various subtypes of the mentioned viruses. Among the other conducted tests, it is important to highlight the negative Bacterioscopy and a Procalcitonin level of 0.18 ng/ml. This significantly reduced the possibility of both viral and bacterial infections.

The patient was kept in the intensive care unit (ICU) until clinical stabilization. At the request of the attending physician, a new tomography was performed for added safety before discharge from the ICU, and thus, we obtained the following image:

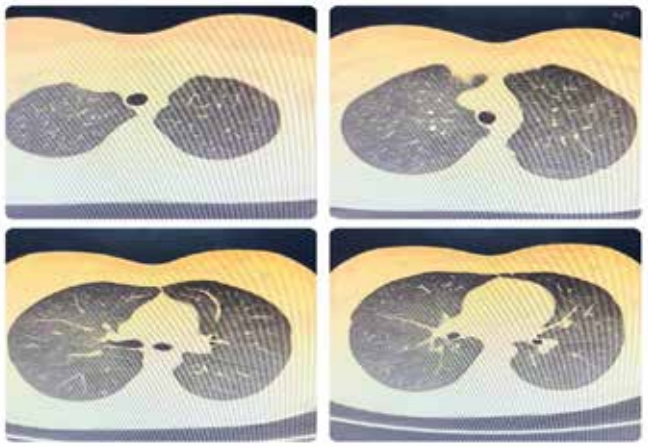

Figure 3: Image of patient, control chest tomography, performed on the 5th day of illness.

The patient's clinical progression in the ICU, demonstrating hemodynamic stability, the absence of respiratory discomfort, and no supplemental oxygen for the past three days with an adequate SpO2 (99%), coupled with a significant improvement in the radiological pattern, led to her

medical discharge from the ICU on 05/09/2023 to a general ward while maintaining favorable parameters. She was ultimately discharged from the hospital on 05/10/2023.

DISCUSSION

The formation of non-cardiogenic pulmonary edema has been observed following various forms of upper airway obstruction, becoming more common in conjunction with laryngospasm (reported in 50% of cases), with an incidence ranging from 0.05% to 5% of anesthetic procedures. The frequency, however, depends on the surgical method, with otorhinolaryngological procedures showing a higher incidence. Approximately 11% of individuals experiencing laryngospasm progress to non-cardiogenic pulmonary edema (NPPE). The latter is a complication that typically occurs with an estimated incidence of 0.1% in general anesthesia practice. [1,3]

The majority of adults who develop acute pulmonary edema (APE) have laryngospasm as the primary triggering factor. The diagnosis is based on clinical manifestations, including ventilatory difficulty followed by inspiratory stridor. Aspiration of a foreign body, bronchospasm, tracheal tube obstruction, laryngospasm, diphtheria, epiglottitis, and strangulation are likely acute causes of non-cardiogenic pulmonary edema (NPPE).[2]

The definition of laryngospasm is the occlusion of the glottis after chemical, mechanical or extrinsically painful stimuli that lead to contraction of laryngeal constrictor muscles. The middle vagus nerve promotes this protective reflex, most of which occurs during an anesthetic emergency, generally in the period prior to extubation. Which makes it possible to increase levels of negative intrapleural pressure, causing APE.[2]

The pulmonary edema, in most cases, manifests immediately after extubation. However, it can occur after a variable period, between two and three hours after airway obstruction, as reported in the second case by Silva et al., where the patient developed dyspnea two hours after extubation. This later manifestation may be explained by an initial protective mechanism. NPPE has a multifactorial physiology. It occurs with forced inspiration against a closed glottis, known as the Müller maneuver, creating a subatmospheric pressure between -50 to -100 mmHg that is propagated to the pulmonary interstitium. This raises venous return to the right side of the heart, resulting in an increase in capillary pulmonary hydrostatic pressure, followed by the transudation of fluid into the alveoli. The negative intrathoracic pressure during left ventricular systole increases afterload, leading to an elevation of end-diastolic volume, reduction of systolic volume, and a decrease in ejection fraction of the left ventricle, promoting an increase in pulmonary vascular pressure. The displacement of the ventricular septum to the left reduces the ejection fraction of the left ventricle (LV) and further elevates microvascular pulmonary pressure due to increased venous return.[2,3,4]

The clinical manifestations include inspiratory stridor, hypoxia, hypercapnia, tachycardia, tachypnea, reduced tidal volume, paradoxical breathing, or uncoordinated ventilatory patterns. Pulmonary edema is accompanied by wheezing and bubbling rales on auscultation, dyspnea, cyanosis, and frothy pink-tinged secretions in the oropharynx.[2]

Complementary diagnostic measures for NPPE include a simple chest radiograph. The typical radiological appearance of this type of edema suggests a predominance of a hydrostatic mechanism. In our case, similar to the case mentioned by Pinhal et al., the images are generally central, bilateral, and located in non-dependent lung zones where the most negative intrathoracic pressures are reached. However, there may be a preferential involvement of a specific lung field. After the regression of the APE, the radiographic pattern may take 12 to 24 hours to return to the previous condition.[2,5]

The screening of symptomatic respiratory diseases is a key element in the preoperative anesthesia assessment. Upper respiratory diseases undergoing general anesthesia carry a higher risk of respiratory complications, including laryngospasm, bronchospasm, and desaturation. However, evidence of lower respiratory tract disease (such as productive cough, wheezing) or systemic disease (such as fever, toxic appearance) constitutes relative contraindications for elective anesthesia. Research suggests that patients with uncomplicated upper respiratory tract infections can undergo general anesthesia without a significant increase in anesthetic complications. Data supporting the risk of perioperative complications related to specific etiological agents are scarce in the literature.[6]

The therapeutic approach involves treating the underlying cause with supportive measures, ensuring airway patency, administering supplemental oxygen through a facial mask, using bronchodilators, and, if necessary, applying non-invasive or invasive mechanical ventilation. Non-invasive ventilation (CPAP/BIPAP) has progressively taken on a prominent role in the treatment of acute respiratory failure in the perioperative period, serving as an effective alternative to invasive ventilation. NPPE, a complication that primarily occurs postoperatively, is recognized in the literature as a well-defined entity but often underdiagnosed, possibly due to its frequently transient and self-limiting course. Invasive monitoring or vasoactive drugs are necessary only in cases of significant hemodynamic changes, which are rare. In the reported case, similar to the majority of cases described in the literature, the patient developed pulmonary edema immediately after extubation, showing significant improvement in dyspnea with the administration of oxygen through a mask, without severe hemodynamic repercussions.[5,7]

CONCLUSION

The present article aims to present a case of NPPE in a young teenager undergoing elective surgery, in which the anesthesiologist reported atraumatic endotracheal intubation with due care for a balanced and higher-quality general anesthesia. The need and importance of continuous support and vigilance with the patient during anesthesia and the time in the post-anesthesia care unit (PACU) were highlighted. It can be concluded that NPPE is still underdiagnosed and underreported in the scientific community. Its treatment involves supplemental oxygen and supportive measures as needed.

Conflicts of interest: The authors have no conflicts of interest to declare.

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