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Case Report

Acute pulmonary non-cardiogenic edema after extubation with laryngospasm: a case report

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ABSTRACT

Acute pulmonary edema post extubation due to negative pressure with laryngospasm in the early postoperative period has been reported and may occur at any time during anesthesia. The usual treatment consists of respiratory support and diuretics. We present the clinical case of a 15-year-old patient who underwent laparoscopic appendectomy, who presented acute non-cardiogenic pulmonary edema in the postoperative period. This complication can be presented in any surgical patient intubated, so it is important to know the pathophysiological basis to be able to diagnose and treat this pathology.

Keywords: Acute pulmonary edema, Laparoscopic surgery, Laryngospasm, Post extubation pulmonary edema

INTRODUCTION

Endotracheal intubation is one of the procedures more frequently performed in clinical practice. This procedure involves the introduction of a cannula through the patient's airway, which is the technique of choice to ensure a patent airway during most current surgical procedures. One of its immediate complications is acute pulmonary edema.[1-2] Acute pulmonary edema after negative pressure extubation with laryngospasm in the early postoperative period has been reported, laryngospasm can occur at any time during anesthesia (induction, intubation, extubation) and usually manifest after the postoperative period in the post-anesthesia care unit or recovery room.[3-5]

The incidence of acute pulmonary edema after negative pressure extubation with laryngospasm is not known with precision, a study derived from the Australian incident monitoring study reported 189 cases of laryngospasm in 4,000 anesthesia processes, five cases of acute pulmonary edema after negative pressure extubation, with an incidence of 3% of those patients with laryngospasm.[6] Other estimates report that 0.05 to 1% of healthy patients undergoing general anesthesia may have this entity, secondary to laryngospasm.[7] Deepika K et al, reported a higher incidence among men (80% of cases of acute

pulmonary edema after negative pressure extubation) with laryngospasm, and in patients categorized with ASA (American Society of Anesthesiology) class I or II (73% of cases). Patients with ASA class I are those healthy who have not undergone elective surgery and ASA class II are those patients with mild systemic disease, controlled and non-disabling, which may or may not be related to the cause of the intervention.[8] Men, particularly young people, athletes, can generate markedly higher intrathoracic pressures, therefore, the disorder may be more common in this group.[9] However, it generally occurs in young, healthy and middle-aged patients.[10] The clinical presentation should be recognized and controlled in time to avoid the complications of transient hypoxia. This is presented as acute respiratory distress syndrome, with cough, pink expectoration, auscultation of bilateral basal crackles, decrease in oxygen saturation in the pulse oximeter, and a diffuse interstitial alveolar image can be seen on the chest radiograph. Incomplete obstruction is usually associated with respiratory disturbances: an audible inspiratory or expiratory sound that can be heard better in the precordial area. If it worsens you can observe a tracheal pull, paradoxical breathing movements of the thorax and abdomen. The absence of respiratory sounds despite the existence of movements of the chest wall indicates a severe laryngospasm (complete obstruction).[11] Acute pulmonary edema after negative pressure extubation with laryngospasm is a form of acute non-cardiogenic pulmonary edema, which results from the generation of the high negative intrathoracic pressure needed to overcome upper airway obstruction, is a life-threatening complication that develops rapidly after obstruction of the airway superior in healthy young people who are capable of producing large negative intrathoracic pressures.[12]

In general, two mechanisms have been proposed to explain it: the first mechanism is in relation to the oscillations in intrathoracic pressure with the respective increase in negative pleural pressure, which leads to an increase in venous return while decreasing the pressure perivascular interstitial hydrostatics, which favors the movement of fluid from the capillaries to the interstitium and the alveolar spaces.[13] Pulmonary edema is caused by a hemodynamic cause by "distortion" of the pressure equilibrium (Starling's law) that maintains the plasma volume in the pulmonary intravascular compartment with passage to the alveoli. This distortion usually begins with inspiratory efforts with obstruction of the upper airway (Mueller's maneuver) that can produce negative intra-alveolar pressures of up to -100cm H_2O.[14] Acute obstruction with hypoxia occurs leading to an increase in sympathetic discharge produces systemic and pulmonary vasoconstriction with increased afterload of both ventricles. Hypoxic pulmonary arterial vasoconstriction further increases right ventricular afterload and the change in trans pleural pressure leads to increased venous return and increased hydrostatic pressure with consequent pulmonary edema.[15] Alveolar hemorrhage due to capillary rupture or hemoptysis is described or both, by rupture of bronchial vessels. In this situation the pathophysiology of the acute pulmonary edema after negative pressure extubation with laryngospasm would be combined, hemodynamic and lesional since there would be rupture of the alveolar capillary membrane. Despite the origin of the blood, it aggravates respiratory insufficiency and prolongs treatment.[16] The second mechanism is related to mechanical stress in the microvascular membranes of the alveolar and pulmonary epithelium by forced breathing with a blocked airway, which increases capillary permeability with high protein content in the transudate.[17]

The usual treatment consists of mechanical ventilation with positive pressure and diuretics, which usually resolves in a few hours, with an excellent prognosis.[11] The medications are usually administered under the following dose: Oxygen at 10 liters per minute and Furosemide at 0.5 to 1mg/kg.[18]

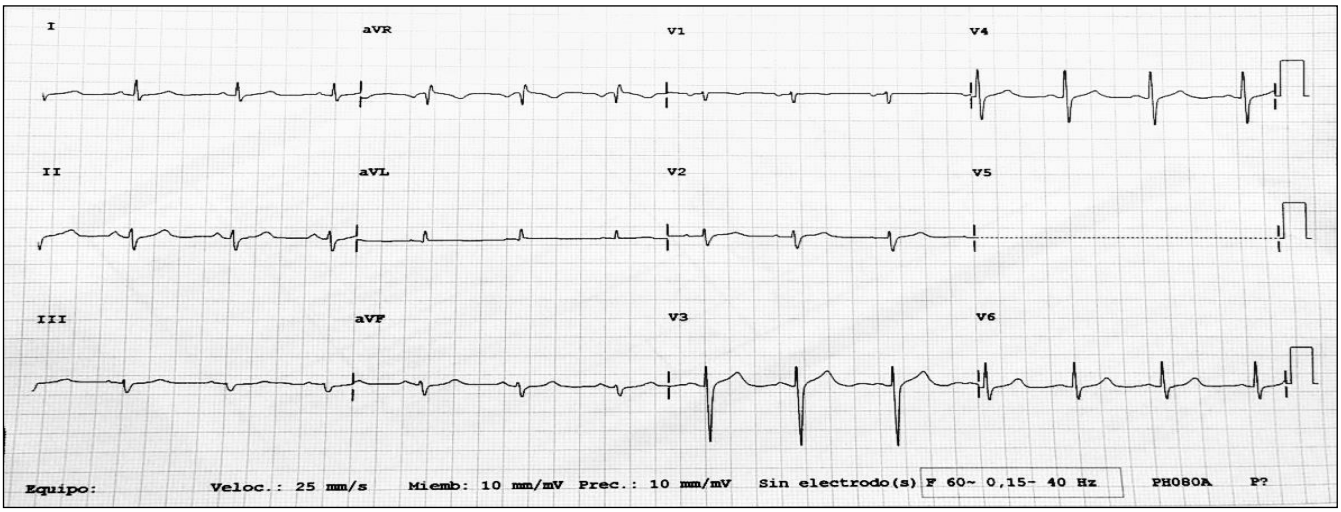
CASE REPORT

A 15-year-old female patient admitted to the emergency room for abdominal pain of 12 hours of evolution in the epigastrium that migrates and subsequently localizes in the right iliac fossa, fever of 38.5ºC, asthenia, and adynamia. On admission she reported abdominal pain with stable vital signs, abdomen soft, tender to the right iliac fossa, Mc Burney sign positive, psoas positive, Rovsign positive with no peritoneal signs. WBC 14.2, 83% neutrophils, abdominal USG with acute appendicular process. Treatment was initiated with Physiological Solution 1000 cc for 12 hours, ceftriaxone 1gr IV every 12 hours, ketorolac 30mg IV every 8 hours, omeprazole 40mg IV every 24 hours. Transferred to the OR where general endotracheal intubation was performed. Medication: Midazolam 1mg, Fentanyl 150mcs, Propofol 100mg, Rocuronium 36mg. Intubation ET tube 7.0 pneumatic tamponade with 3cc. of air, tidal volume 325 ml, respiratory rate 15x', PEEP 4. Maintenance: Defluorine 6 Vol% for 1 CAM. Fentanyl in total perfusion 192.37 mcs. Clinical and hemodynamically stable transanesthetic. A laparoscopic appendectomy was performed through 3 ports, using harmonic for cutting and hemostasis of the mesoappendix, with 2 endoloops to ligate the appendicular base and 1 distal staple.

The appendix was extracted by endobag. Reversal of neuromuscular blockade with sugammadex 200mg. Secretions were aspirated before extubation. Extubated and placed supplemental oxygen. Exits to post anesthesia care unit, with 99% saturation, respiratory rate 16, Aldrete 10, Ramsay 2. Patient is in his first postoperative hour of laparoscopic appendectomy, post-anesthetic, non-invasive monitoring was performed in the post-anesthetic care unit, withdraw supplemental oxygen with saturation at 80%, oxygen is administered again and withdrawal is attempted; maintaining low saturation, so oxygen is continued. The pulmonary fields were explored, bilateral

hypoventilation and crackles were found, causes were investigated and EKG was requested (Figure 1) with a chest plate (Figure 2).

In the Figure 2, the image A, immediate postoperative. Soft tissues and bone structures are appreciated without apparent alterations. There are diffuse, diffuse bilateral opacities of peri-hilar predominance. There is cephalization of the flow with thickening of the vascular paths towards the pulmonary apices. The Kerley lines are observed in the left apex (A) and in the middle lobe of the right lung (B). In this same image, slight prominence of the pulmonary arch is also observed as an overload data. Also decrease in the radio lucidity pulmonary so that ventilation decreases. In image B, 12 hours postoperatively. There is an increase in lung lucidity as a result of increased ventilation. There is a decrease in the hilar and infiltrating opacity (in this image and in the previous one it is the typical image of infiltrate in butterfly wings). The cephalization of the flow with prominence of the vascular trajectories persists. Decreases the prominence of the pulmonary arch. In image C, 24 hours postoperatively. The perihilar infiltrate and the cephalization of the flow can no longer be observed. Vascular pathways conserved. Cardiac profiles within normality. The pulmonary radiographic pattern is homogeneous with the exception of observing a right basilar radiopaque linear image that suggests a passive band of atelectasis. Clinically healthy patient not tachypneic, nor diaphoretic. Management was started with furosemide 10mg IV, semifowler position with supplemental oxygen, therapy with microcirculation ATROVENT 1 every 6 hours, hydrocortisone 50mg, lung physiotherapy in turn. Patient was observed for 24 hours, maintained vital signs within the normal limits and discharged.



Note: 12-Lead electrocardiogram in sinus rhythm, heart rate of 75 bpm, P: 80ms PR: 160ms A ° QRS: -30 ° QRS: 80ms QT measured: 360ms QT corrected by BAZET: 402ms, FRIEDERICIO: 388ms. Findings: Discrete deviation of the axis to the left, without any other abnormal electrocardiographic data

Figure 1: Electrocardiogram.

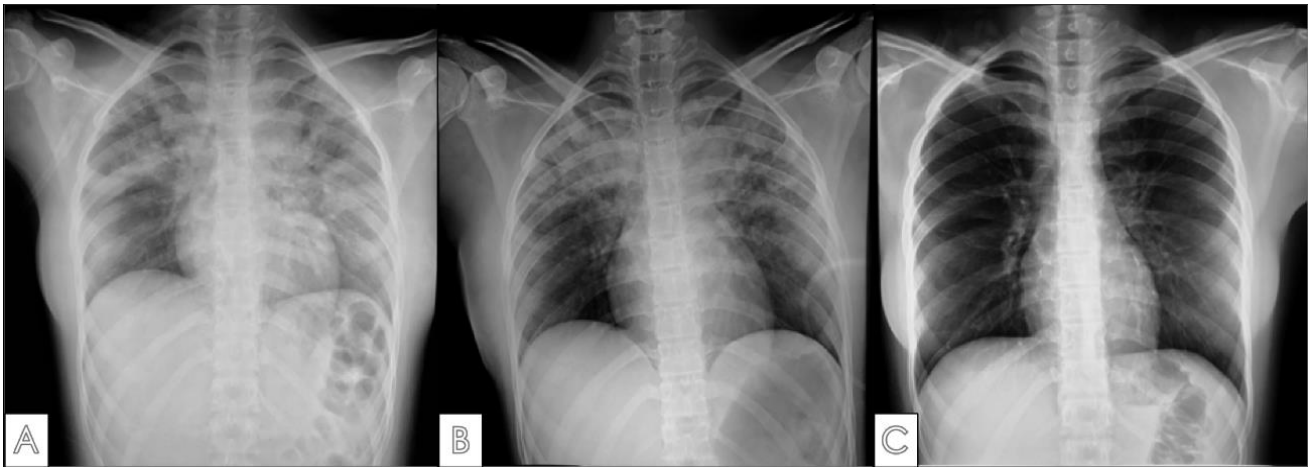


Figure 2: A) Immediate postoperative. B) 12 hours postoperatively. C) 24 hours postoperatively.

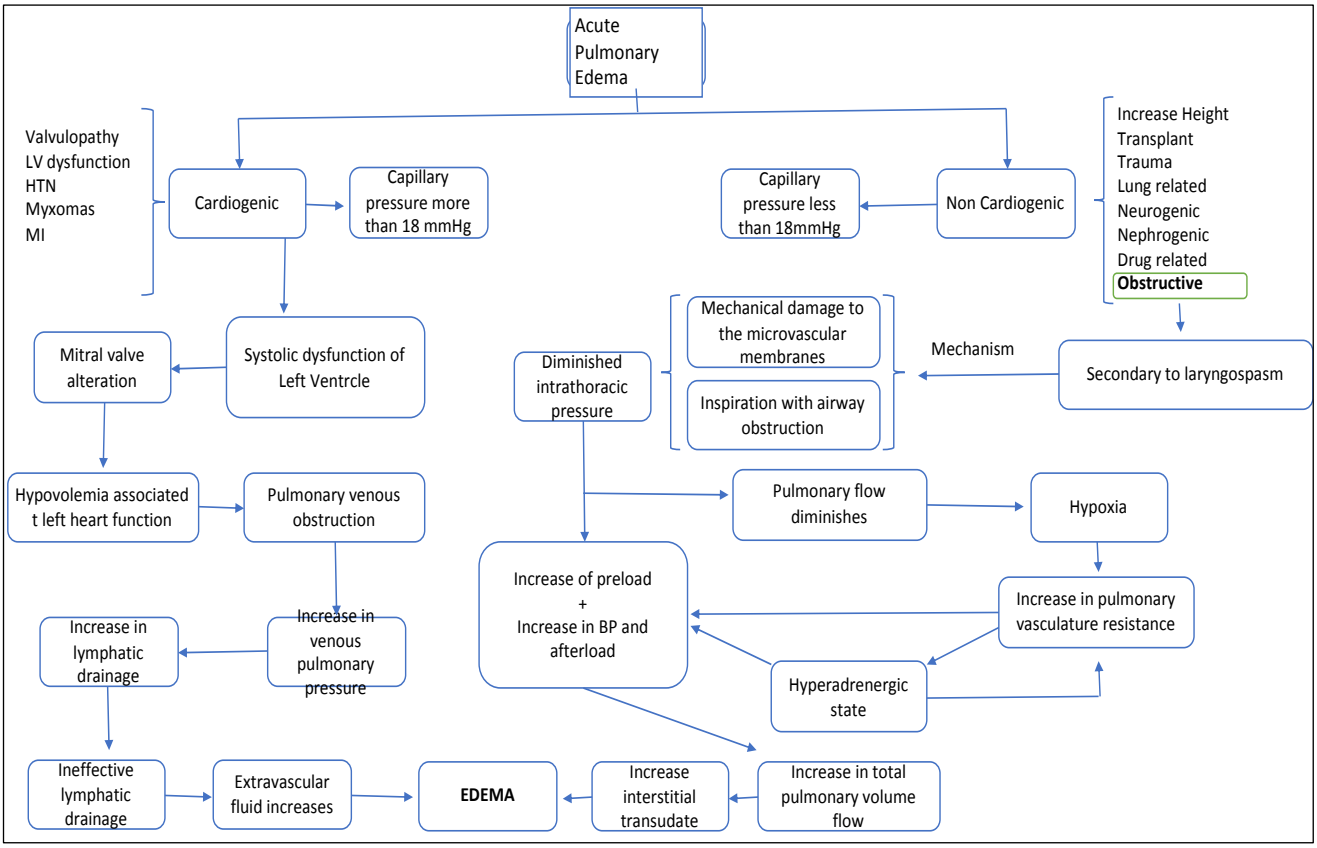


Figure 3: Pathophysiology of acute pulmonary edema.

DISCUSSION

Acute non-cardiogenic pulmonary edema is common due to the increase in the incidence of laparoscopic surgery since its appearance is implemented in several disciplines and specialties.[19] It is crucial to immediately recognize the clinical presentation of acute non-cardiogenic pulmonary edema after anesthesia, as there may be a delayed clinical presentation of up to 24 hours, systematically knowing the pathophysiological events that occur in post-extubation (Figure 3).[20-22] On radiography there is a rapid onset with characteristic changes with cotton-like infiltrates, and the resolution of concomitant radiological and clinical changes, most resolving within the first 24-36 hours.[23] In addition, an electrocardiogram can also be included (to identify arrhythmias, ischemia or infarction) or an echocardiogram (to evaluate the performance of the myocardium) to help determine if pulmonary edema is due to a cardiogenic etiology since it is the differential diagnosis.[22] Acute pulmonary edema by negative pressure is one of the main causes of acute pulmonary edema in the peri-operative period and with initial pathology of a laryngospasm that triggers increased intra-thoracic pressures after forced inspiration, with the subsequent plasma flow into the interstitial space.[24]

CONCLUSION

It is important to have tools for the correct diagnosis and treatment (chest x-ray and electrocardiogram), keeping in mind the clinical suspicion with a history of non-fortuitous extubation. The diagnosis and effective treatment achieves a complete resolution of the frame in 24 hours.

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