ABSTRACT

Introduction: Secondary pulmonary edema causing airway obstruction is an uncommon condition. Although the physiopathology of edema is still unclear, the predominant mechanism is likely forced inspiration against a closed glottis, generating a negative intrapulmonary and transpleural gradient, which favors fluid transudation from the pulmonary capillaries into the interstitium. Case Report: We report a case of barotrauma and acute pulmonary edema in a young patient after undergoing surgical treatment for gynecomastia under general anesthesia.

Keywords: Acute pulmonary edema; Gynecomastia; Laryngospasm; Postoperative complications.

INTRODUCTION

Secondary pulmonary edema causing airway obstruction is a rare condition, with an estimated incidence of 11%, although it may be observed in a wide variety of clinical situations. Health professionals need to be aware of this condition, to recognize the early signs and symptoms and to establish treatment as soon as possible.

Although the pathophysiology of edema is still unclear, the predominant mechanism is likely forced inspiration against a closed glottis, generating a negative intrapulmonary and transpleural gradient, which favors fluid transudation from the lung capillaries into the interstitium.

The objective of this study is to describe the case of a patient who developed a barotrauma clinical scenario and acute pulmonary edema after undergoing general anesthesia for surgical treatment of gynecomastia.
CASE REPORT

F.M.X., 21 years old, male, was suffering from glandular gynecomasty and bilateral lipodystrophy without other comorbidities. He was not a drug user, had no history of drug allergy, and was above the ideal weight for his age.

In May 2009, he was admitted for surgical treatment. The pre-pectoral subcutaneous tissue was infiltrated with a physiologic saline solution with adrenaline (1: 500,000), after which pre-pectoral liposuction and excision of the mammary gland was performed through a Webster incision. The procedure was performed under general anesthesia without complications, lasting 50 minutes.

Towards the end of surgery, he was agitated and biting on the oral tracheal tube, making extubation difficult. The tube was not protected by the Guedel oral cannula. Recurarization and ventilation were performed under pressure, which resulted in clinical improvement. After recovery from anesthesia, the patient was clinically stable without any complaints. However, six hours later, the patient developed dyspnea and neck discomfort. Physical examination revealed mild cervical crackles, diminished vesicular murmur on bilateral auscultation, cough with pinkish secretion, increased respiratory effort, especially in the supine position, and oxygen saturation of 85% on room air. The patient was referred for evaluation to a specialty clinic, where a chest radiograph showed interstitial infiltrate, especially in the perihilar regions (figure 1), and a computed tomography showed a frosted glass lung opacity as well as bilateral peribronchovascular, pneumomediastinum, and subcutaneous emphysema in the chest wall (figure 2).

DISCUSSION

The first case of secondary pulmonary edema caused by airway obstruction was described by Oswalt et al. in 1977. The laryngospasm that occurs during oral tracheal intubation or after anesthesia is described as the most common cause of this condition, accounting for approximately 50% of cases. Other causes include strangulation, epiglottitis, foreign body aspiration, hypothyroidism, hiccups, cervical hematomas, difficult intubation, thyroid goiter, croup, cancer, and obesity.

The pathophysiology of edema is still uncertain. There are two proposed mechanisms. One theory is that the edema is caused by major fluid shifts due to changes in intrathoracic pressure. Negative intrathoracic pressure is generated during inspiratory effort against resistance – in this case, the airway obstruction. The pressure drop causes an increase in venous return to the right ventricle, which in turn increases the pulmonary venous pressure. This increase in pulmonary venous pressure generates a transpulmonary hydrostatic gradient that causes fluid displacement from high pressure zones (pulmonary venous system) to low pressure zones (pulmonary interstitium)

The second proposed mechanism involves rupture of the alveoli capillaries and pulmonary microvascular membrane due to intense mechanical stress, leading to increased vascular permeability and ultimately an edema with a high protein content. Another factor involved in the genesis of acute pulmonary edema is the lack of alveolar ventilation in the obstructive period, resulting in hypoxemia and hypercapnia which, along with the patient’s state of anxiety, leads to the release of catecholamines.

This condition causes an increase in hyperadrenergic systemic vascular resistance (SVR) and consequently increased left ventricular afterload (LV). The sum of the de-
crease in the left ventricular compliance (interventricular sep-
tum shift) and the increase in afterload cause reduction of LV
ejection volume, which results in an increase in systolic and
diastolic final ventricular volumes\textsuperscript{2,3,4,7,8}.

This condition affects children and young adults more
often. The prognosis is better for younger patients than for
older ones. The potential factors influencing the development
of intrapleural negative pressure pulmonary edema (NPPE)
are: young patients, male, muscular body type, short neck,
with a Malampatti rating of 3 on physical examination, and
having a history of apnea\textsuperscript{2,3}.

Classically, NPPE includes sanguineous or pinkish se-
cretion, demonstrating extravasation of hair cells as a result of
mechanical failure of alveolar–capillary membrane, leading to
alveolar edema or frank bleeding. The diagnosis is made based
on the sudden development of dyspnea, tachypnea, hypox-
emia, hypercapnia, or pink secretion after airway obstruction is
relieved. The chest radiograph usually reveals increased vas-
cular pedicles with bilateral interstitial infiltrates. The beginning
of NPPE is fast (within minutes), but it may be delayed up to
4 hours after the occurrence of an obstructive\textsuperscript{3} phenomenon.

The literature suggests that the speed of edema de-
velopment is associated with the beginning of the obstruction
and its severity. The condition is self-limiting, generally resolv-
ing in 12 to 24 hours. In most cases, treatment is supportive. In
about 85\% of patients, both children and adults, tracheal intu-
bation is necessary for maintenance of the airways, followed
by ventilation with positive expiratory pressure. Once the di-
agnosis is established, aggressive hemodynamic monitoring
is not necessary, nor is the use of vasoactive drugs. However,
there may be serious morbidity and mortality, ranging from
11\% to 40\% of reported\textsuperscript{2,3} cases.

**CONCLUSION**

Acute pulmonary edema associated with obstruction
of the upper airways is a clinical condition that can aggravate
surgical procedures considered to have low morbidity. The
condition appears mostly in young patients. Its recognition and
prevention are of great importance. Patient extubation with
fully reversed neuromuscular blockade and ensuring respon-
siveness to simple commands seems to be the best form of
prevention, where the routine use of Guedel cannula at the
time of extubation prevents inadvertent obstruction of the en-
dotracheal tube by biting, especially in younger patients. If the
condition occurs, treatment should be initiated early, because
the resolution is rapid, and in most cases, without sequelae.

**REFERENCES**

1. Oswalt CE, Gates GA, Holmstron FG. Pulmonary edema as a com-
2. Louis PJ, Fernandes R. Negative pressure pulmonary edema. Oral
Sury Oral Mee Oral Pathol Oral Radiol Endoy. 2002;93(1):4-6. Re-
view.
3. Scarbrough FE, Wittenberg JM, Smith BR, Adcock DK. Pulmonary
edema following postoperative laryngospasm: case reports and
MP. Postextubation pulmonary edema: a case series and review.
Respiy Mee. 2008;102(11)9165966.
5. Visvanathan T, Kluger MT, Webb RK, Westhorpe RN. Crisis man-
6. Lang SA, DuncanPG, ShephardDA, HaHC. Pulmonary edemaassos-
Review.
7. Jackson FN, Rowland V, Corssen G. Laryngospasm–induced pul-
8. Halow KD, Ford EG. Pulmonary edema following post–operative-
1993;59(7):44347.