Case Report

Pneumomediastinum and acute pulmonary edema after surgical treatment of gynecomastia

Pneumomediastino e Edema agudo de pulmão pós tratamento cirúrgico de ginecomastia

CARLOS EDUARDO GUIMARAES LEÃO[1]
PAULO CÉSAR DE ABREU SALES[2]
DREYFUS SILVA FABRINI[3]
GISELLE LELIS BURGARELLI[4]

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 .

RESUMO

Introdução: O edema pulmonar secundário à obstrução das vias aéreas é uma condição incomum. Apesar da fisiopatologia do edema ser ainda obscura, é provável que o mecanismo predominante seja a inspiração forçada contra a glote fechada, gerando um gradiente intrapulmonar e transpleural negativos, o que favorece a transudação de fluidos dos capilares pulmonares para o interstício. **Relato de Caso:** Os autores relatam um caso de barotrauma e edema agudo de pulmão em um paciente jovem após ter sido submetido a tratamento cirúrgico de ginecomastia, sob anestesia geral.

Descritores: Edema agudo de pulmão; Ginecomastia; Laringoespasmo; Complicações pós-operatórias.

Institution: PHD - Pace Hospital.

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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[1-5] 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[1].

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.

1- Full member of the SBCP – Head of the Plastic Surgery and Burns Service of the FHEMIG Network.
2- Full member of the Brazilian Society of Anesthesiology – Head of Anesthesia Service of the São José University Hospital - Belo Horizonte.
3- Physician - Resident of the Plastic Surgery Service of the FHEMIG Network.
4- Physician - Resident of the Plastic Surgery Service of the FHEMIG Network .

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).

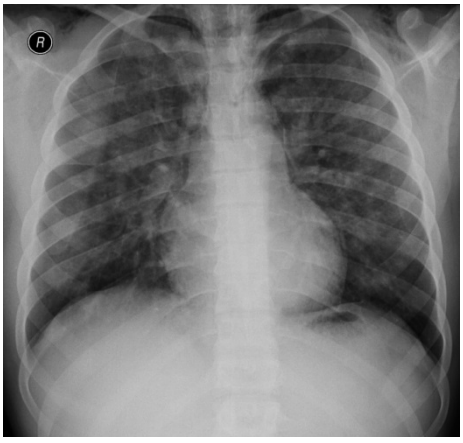

Figure 1. *Interstitial infiltrate, especially in the perihilar region*

After evaluation, the patient was admitted with a diagnosis of acute pulmonary edema and treated conservatively with oxygen by nasal cannula 3 L/min. He showed progressive improvement and was discharged on the 4th postoperative day without any symptoms.

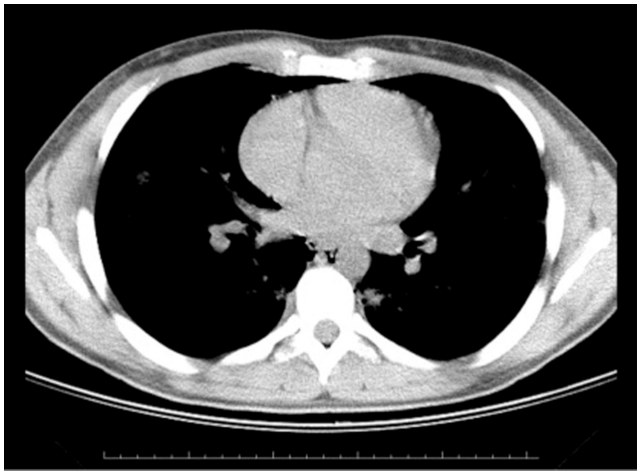

Figure 2. Frosted glass pulmonary opacity, bilateral peribronchovascular, pneumomediastinum and subcutaneous emphysema in the chest wall.

DISCUSSION

The first case of secondary pulmonary edema caused by airway obstruction was described by Oswalt et al. in 1977[1]. 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[1,2,3].

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)[2].

The second proposed mechanism involves rupture of the alveoli capillaries and pulmonary microvasculature membranes 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 hypercarbia 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 septum shift) and the increase in afterload cause reduction of LV ejection volume, which results in an increase in systolic and diastolic final ventricular volumes[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[2,3].

Classically, NPPE includes sanguineous or pinkish secretion, 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, hypoxemia, hypercapnia, or pink secretion after airway obstruction is relieved. The chest radiograph usually reveals increased vascular 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[3] phenomenon.

The literature suggests that the speed of edema development is associated with the beginning of the obstruction and its severity. The condition is self-limiting, generally resolving in 12 to 24 hours. In most cases, treatment is supportive. In about 85% of patients, both children and adults, tracheal intubation is necessary for maintenance of the airways, followed by ventilation with positive expiratory pressure. Once the diagnosis 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[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 responsiveness 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 endotracheal 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.

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Corresponding Author: **Carlos Eduardo Guimarães Leão**
Rua Ceará 1986/10° andar. Funcionários. Belo Horizonte-MG. CEP 30150311
E-mail: leao@leao.med.br