

Acute postobstructive pulmonary edema caused by a massive substernal thyroid goiter: a case report and a brief literature review

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Abstract

Acute Postobstructive Pulmonary Edema (APOPE) is a rare form of non-cardiogenic pulmonary edema. APOPE is a manifestation of acute airway obstruction usually described following anesthesia in post-operative phase due to laryngospasm. We present a case of 58-year-old lady who developed a severe APOPE induced by a

massive thyroid goiter. The subsequent imaging studies revealed the compressing mass-effect of the thyroid goiter on the upper airway. The prompt recognition of this rare clinical event is crucial to rapidly restore the patency of the airway and correct hypoxemia.

Key words: postobstructive pulmonary edema, substernal goiter, airway obstruction

Introduction

Acute Postobstructive Pulmonary Edema (APOPE) is a rare clinical event presenting a multifactorial pathogenesis. APOPE is most widely reported in the anesthesia literature related to perianesthetic-induced laryngospasm. Review of the literature suggests an incidence of one case per thousand anesthetics. (1)

APOPE resulting from upper airway obstruction in the setting of tumor, hanging/strangulation, and goiter has been reported sporadically in children and adults since 1977. (2) Acute airway obstruction from a chronic substernal goiter is exceedingly rare, and may be caused by idiopathic

hemorrhage into the gland with resultant hematoma and mass effect. (3) We report a case of APOPE related to massive substernal extension of a clinically benign appearing chronic thyroid goiter.

Case report

A 58-year-old woman was found unresponsive shortly after going to bed. She was in the supine position with copious amounts of pink, frothy nasal and oral discharge. Basic life support and advanced cardiac life support were initiated and a definitive airway was placed, in the field, by emergency medical services providers. Past medical history was significant for a chronic, long-standing cervical thyroid goiter. She was also noted to have intermittent asthma, not responsive to bronchodilator therapy, and obstructive sleep apnea (OSA). On presentation, she was noted to be afebrile with a heart rate of 79 beats/minute, BP of 100/50 mmHg, respiratory rate of 24 breaths/minute and an arterial oxygen saturation of 97% on 100% FiO₂. Neck examination revealed a palpable cervical goiter (**Figure 1a**). Lung examination revealed diffuse bibasilar crackles extending halfway up the posterior lung fields bilaterally. The remainder of the physical examination was unremarkable.

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CBC count, electrolyte levels, and coagulation studies were normal. Thyroid-stimulating hormone, free T4 and total T3 were normal. Cardiac troponin T was 0.11 ng/dL (normal, 0.0-0.03 ng/dL). Brain natriuretic peptide level was normal. An electrocardiogram revealed no acute changes. A trans-thoracic echocardiogram revealed preserved systolic and diastolic function. A portable anterior-posterior chest radiograph revealed bilateral areas of peri-hilar air space densities consistent with pulmonary edema and a widened mediastinum with displacement of the trachea to the left and narrowing of the left mainstem bronchus (**Figure 1b**). Computed tomography (CT) scan of the neck and chest demonstrated a massively enlarged thyroid with substernal/mediastinal extension to the level of the carina. There was associated mass effect with tracheal narrowing and displacement to the left (**Figure 2 a,b**). Initial attempt to liberate from mechanical ventilation was unsuccessful related to recurrent airway obstruction and acute pulmonary edema. Subsequently, a total thyroidectomy was performed with removal of a thyroid measuring 11.7 cm x 8.4 cm x 5.3 cm and weighing 295 grams. On pathological evaluation, the thyroid demonstrated areas of focal hemorrhage, hematoma, and hemosiderin deposits. No malignant cells were identified. Extubation was achieved on postoperative day number one. Fiber-optic bronchoscopy was performed during the extubation and revealed no evidence of significant tracheomalacia. The patient progressed well without incident and was discharged on postoperative day number five.

Discussion

APOPE is an uncommon medical emergency, mostly described following anesthesia in post-operative phase. Post-anesthetic laryngospasm has been implicated as the most frequent cause of this syndrome in adult. (4) The incidence of APOPE is less than 0.1% but mortality can be as high as 40% in case of delayed diagnosis. (5) It has been rarely described as complication of acute airway obstruction due to thyroid goiter. (6) In our case, the history and clinical presentation characterized by tachypnea, frothy pink pulmonary secretions, rales, and progressive oxygen desaturation suggested the diagnosis of APOPE. Our patient demonstrated rapid clinical and radiographic improvement

after 24-hours of mechanical ventilation followed by total thyroidectomy to prevent recurrence of acute respiratory distress.

The pathogenesis of APOPE is multifactorial and risk factors such as obesity, palatal mass, short thick neck, obstructive sleep apnea syndrome, and post-nasal surgery have been associated with its development. Careful review of the literature indicates that there appears to be two distinct types of APOPE. Type 1 follows acute airway obstruction, which then induce forceful respiratory efforts leading to an extremely negative intrapleural/intrathoracic pressure and to the entirely perivascular interstitium. The markedly negative intrapleural pressure increases venous return, pulmonary blood volume, and pulmonary capillary hydrostatic pressure while lowering the perivascular interstitial hydrostatic pressure. (7) The decline in pulmonary interstitial hydrostatic pressure coupled with the increased pulmonary capillary hydrostatic pressure leads to accumulation of fluid in the interstitial space. (8) Other factors may play contributing roles in the development of edema related to airway obstruction. Hypoxic vasoconstriction, hyper-adrenergic state associated with catastrophic airway obstruction and physical damage to alveoli and capillaries may also increase capillary pressure and favor movement of fluid into the interstitium. (2) In type 2 APOPE, pulmonary physiology has been set at a different baseline by a chronic upper airway obstruction. Relief of the obstruction causes a sudden drop in airway pressure that leads to the formation of pulmonary edema by the aforementioned mechanisms. It is postulated that the airway obstruction creates more positive pressures during expiration, which serves as a form of "auto-PEEP" to oppose transudate formation. (2) Although recent data suggest that endotracheal intubation could be avoided through the use of noninvasive respiratory support, management of APOPE is based on the rapid relief of the airway obstruction and correction of hypoxemia. The use of diuretics is controversial and not routinely warranted. (9-10)

The purpose of this article was to present a rare case and a clinical review of the literature of APOPE in order to promote signs and symptoms recognition and earlier diagnosis and to improve the correct management.

Figure 1 a-b. Thyroid goiter (1a); CXR (1b) showed bilateral peri-hilar air space densities consistent with pulmonary edema and a widened mediastinum with displacement of the trachea to the left and narrowing of the left mainstem bronchus

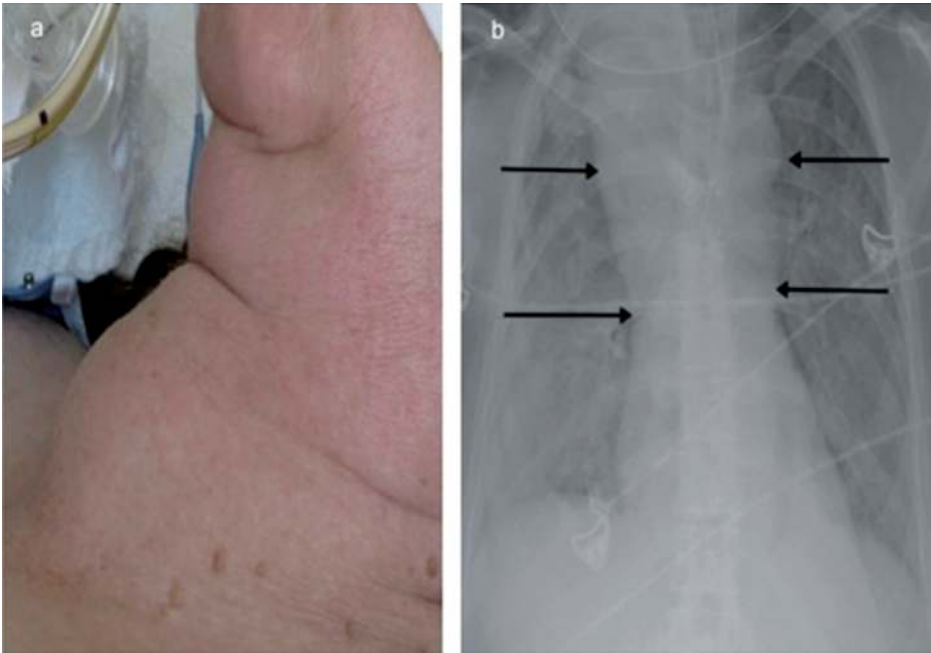
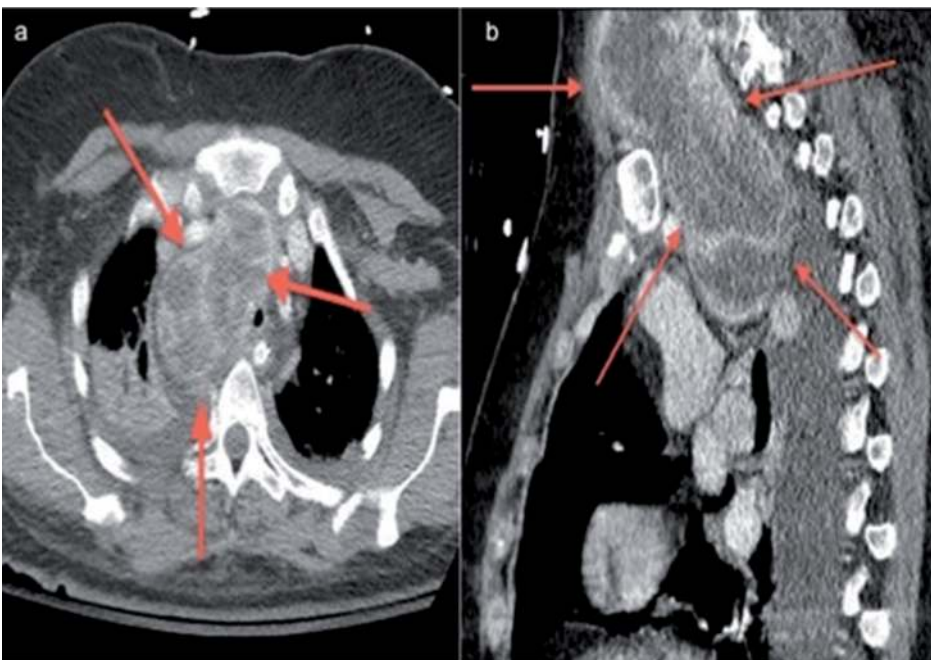


Figure 2 a-b. CT scan of neck and chest demonstrated a massively enlarged thyroid with substernal and mediastinal extension to the level of the carina, inducing narrowing and displacement to the left of trachea due to mass-effect



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