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Acute Pulmonary Edema Caused by Choking in An Adult Patient

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Abstract

Postobstructive pulmonary edema (POPE), otherwise known as negative pressure pulmonary edema (NPPE), is a potentially life-threatening complication following acute upper airway obstruction (UAO). It can develop rapidly, without warning, in persons who are otherwise well. Although POPE was first described nearly 30 years ago, yet this perplexing syndrome is rarely reported in adult. We report an adult patient who suffered from choking with a Chinese dessert. He developed a non-cardiogenic pulmonary edema due to acute upper airway obstruction and recovered well after conservative treatment. Clinicians should be alert to continued respiratory symptoms following relief of acute airway obstruction. Preventing re-obstruction and providing adequate ventilation and oxygenation are mandatory. Such patients should be observed in ED for at least 6 hours in order to avoid a catastrophic outcome. Awareness of this condition can avoid unnecessary intervention and possible iatrogenic complications coming from treating ALI/ARDS. (J Intern Med Taiwan 2007; 18: 108-112)

Key Words ：**Postobstructive pulmonary edema, Negative pressure pulmonary edema, Noncardiogenic pulmonary edema, Airway obstruction, Choking**

Introduction

Postobstructive pulmonary edema (POPE), otherwise known as negative pressure pulmonary edema (NPPE), is a potentially life-threatening complication following acute upper airway obstruction (UAO). It can develop rapidly, without warning, in persons who are otherwise well. Yet this perplexing syndrome is rarely reported in adult. We report an adult patient who developed such non-cardiogenic

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pulmonary edema after choking with a Chinese dessert.

Case Report

A 49-year-old male with a history of mental retardation suffered choking by Mua-Chee (a kind of hand-made sweet Chinese dessert made by sticky rice). The emergency medical technician who responded to the family's 119 call found him air-grasping; performed Heimlich maneuver and removed some food manually. He was still in respiratory distress and was sent to the emergency department (ED).

On arrival, he was alert but unable to talk fluently. His vital signs included blood pressure 128/78 mmHg, respiratory rate 26 breathes per minute, pulse rate 108 beats per minute and body temperature 36.4 ℃. Physical examination was remarkable for the orthopnea, bibasilar rales and the presence of a frothy pink pulmonary secretion. A chest radiography showed bilateral pulmonary edema with a normal heart silhouette (Figure 1A). An arterial blood gas showed a pH of 7.485, PaO_2 of 62.2 mmHg, $PaCO_2$of 28.2 mmHg, and $HCO3^-$ of 22.2 mmol/L when he was using a nonrebreather facemask. The remaining laboratory tests were normal except blood glucose of 212 mg/dL. He required the use of Bi-level Positive Airway Pressure® ventilator support. He was admitted to the ward and treated conservatively. On the second hospital day, he could use ambient air. Prophylactic antibiotic therapy whch had been given for the concern of aspiration pneumonia was discontinued after he was stabilized. A follow-up chest radiograph showed no air-space opacity and was deemed absence of pulmonary edema (Figure 1B). He recovered well and discharged on the 5th hospital day.

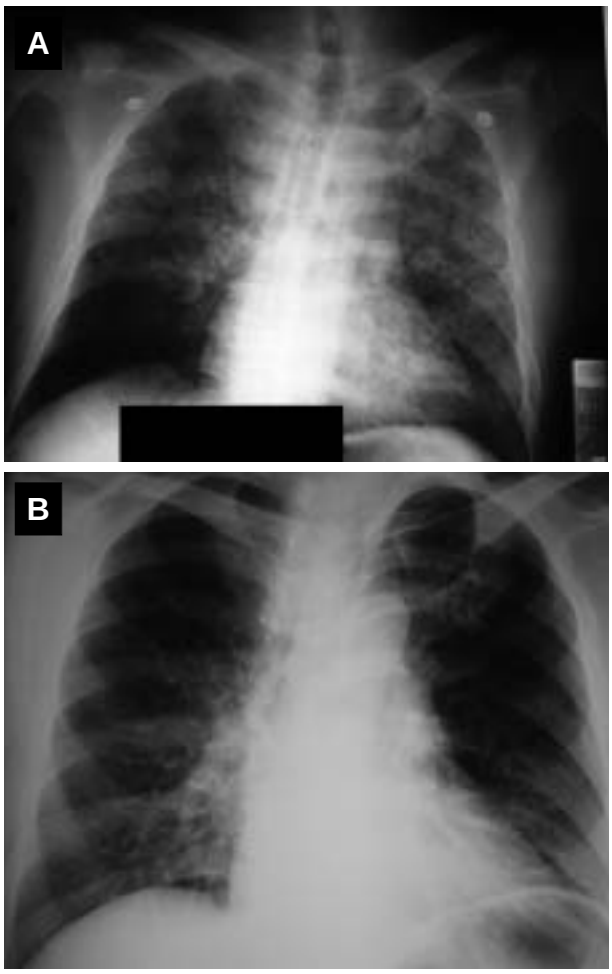

Figure 1 A: Chest radiography obtained on arrival; B: obtained on the 5th hospital day

Discussion

Devastating events following relief of choking or aspiration are aspiration pneumonia or chemical pneumonitis resulting in acute lung injury/acute respiratory distress syndrome (ALI/ARDS) and lung abscess. The rapid improvements in both chest radiography as well a benign clinical course had made the diagnosis of aspiration or chemical pneumonitis unlikely. Nonetheless, NPPE had been described to be an imitator of ALI/ARDS and failure to consider NPPE in the differential diagnosis of acute clinical, physiologic, and radiographic changes that fit the criteria for ALI/ARDS may lead to unnecessary and potentially deleterious iatrogenic complications. While early recognition and specific precipitating conditions may herald NPPE, an obvious underlying cause may be absent particularly after emergence from unwitnessed UAO or anesthesia[1]. Majority of POPE was recognized postoperatively by anesthetists as a consequence of laryngospasm[2]. A myriad of causes had

been reported, including croup, epiglottitis, mass effect secondary to tumor, strangulation, interrupted hanging[3], endotracheal tube occlusion by patient biting on it[4], biting a laryngeal mask airway, hiccups during induction[5], sleep apnea, attempted suffocation of an infant[6], and diagnostic fiber-optic bronchoscopy in an infant[7]. The true incidence of POPE is unable to be determined because of the divergence clinical setting, but it could be expected to be underestimated. The incidence was estimated to be between 0.05 to 0.1% and 0.094% in surgical adult patients and in all anesthetics, respectively[8,9]. Ringold et al had reported two children, one who choked on a hot dog and the other on a "jawbreaker" (a hard candy with multiple layers) developed POPE[10]. On reviewing medical literature, POPE caused by choking had been reported even rarer in adult patients.

POPE may occur within minutes in majority of patients, following either the development or relief of acute severe UAO with a significant clinical and radiographic improvement in 12 to 24 hours[11]. Late-onset POPE can develop up to 6 hours postoperatively. Therefore, we suggest that patients should be observed for at least 6 hours after suffering from acute severe UAO. Patients may be asymptomatic or rapidly self-resolving but severe respiratory distress or even negative pressure pulmonary hemorrhage, where there is alveolar-capillary integrity compromise and global pulmonary hemorrhage in the airways. The diagnosis of non-cardiogenic pulmonary edema could be made based on the history, physical examinations, routine laboratory examination and characteristic of chest radiography (e.g. normal heart size, peripheral distribution, and absence of Kerley's B lines) without other adjunct examinations[12]. The absence of gastric content in pulmonary secretions together with a history of normal cardiac function, particularly for a young, active individual, favors the diagnosis of POPE[2]. Once the diagnosis has been established, the causes should be eliminated as possible and the treatments could be given accordingly.

POPE can be divided into two types. Type 1 POPE, which follows acute airway obstruction, is more frequently encountered in the ED. Type 2 POPE follows surgical relief of chronic airway obstruction[13]. The pathophysiology of type 1 POPE is the markedly negative intrathoracic pressure caused by forced inspiration against a closed airway, resulting in transudation of fluid from capillaries to the interstitium and alveoli[15]. Vigorous inspiratory effort against a totally obstructed airway is a common factor. Mechanical disruption, shear force or stress failure of the alveolar-capillary membrane can also result in diffuse alveolar injury and diffuse alveolar hemorrhage[14,15]. Young athletic men are more susceptible than general subjects because their muscular chest wall may generate extremely high inspiratory pressures[16]. The pathophysiology of type 2 POPE may be attributed to sudden removal of positive end-expiratory pressure may lead to interstitial fluid transudation and pulmonary edema. Re-expansion pulmonary edema, another rare complication that share may similarities with NPPE, has been reported following treatment of pneumothorax, drainage of large pleural effusion, single lung ventilation and transthoracic endoscopic sympathectomy. The pathophysiology of re-expansion pulmonary edema remains unclear. A number of mechanisms have been suggested, including increased pulmonary vascular permeability, a decrease in perivascular pressure, ischemia-reperfusion injury, free radical injury, hypoperfusion, hypoxia, the effect of a high negative pressure, tissue injury seconady to stretching, decreased surfactant and lymph flow in the collapsed lung[17,18].

Preventing re-obstruction and providing adequate ventilation and oxygenation are mandatory after relief of UAO. Treatments include supplemental oxygen and support cares[19] but positive end-expiratory pressure and mechanical ventilation may be required for a prolonged period of time[20]. Non-invasive continuous positive airway pressure (NIPPV) has been used in both NPPE and re-expansion pulmonary

edema[21], though its effects still require further studies. Full and rapid recovery can be expected with appropriate management.

In summary, adult patients can develop POPE after choking. Clinicians should be alert to continued respiratory symptoms following relief of acute airway obstruction. Besides, awareness of this condition can avoid unnecessary intervention and possible iatrogenic complications coming from treating ALI/ARDS. Such patients should be observed in ED for at least 6 hours in order to avoid a catastrophic outcome.

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急性肺水腫於一上呼吸道阻塞的成人病患

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摘 要

阻塞後肺水腫（post-obstructive pulmonary edema）或稱爲負壓性肺水腫（negative pressure pulmonary edema）是病患發生上呼吸道阻塞後潛在致命的併發症。此症可能急性無預警地發生在狀況似乎良好的病患。此病症於成年人仍鮮少被報導。在此我們報告一於吃麻糬嗆到之病患，予以暢通呼吸道後，卻發生肺水腫的臨床表徵，此病患經保守治療後迅速恢復，判斷爲因呼吸道阻塞後所發生之非心因性肺水腫。阻塞後肺水腫之處置以避免再度阻塞，輔助換氣，提供氧氣及支持治療爲主，通常預後良好：即使在上呼吸道阻塞緩解之後，臨床醫師仍應持續注意病患的呼吸狀況，並應留觀至少六小時。瞭解此狀況亦可避免與急性肺損傷/急性呼吸窘迫症候群混淆，而導致不必要的治療甚至醫源性的併發症。