PULMONARY EDEMA DUE TO UPPER AIRWAY OBSTRUCTION
--- A CASE REPORT

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Abstract

Postobstructive pulmonary edema is an uncommon complication which immediately after the onset of acute airway obstruction and has been described in children and adults. Appropriate diagnosis and management are important in leading to a good outcome for the patient. We describe one case of postobstructive pulmonary edema caused by brief acute airway obstruction due to choking on bread. She developed symptom of complete upper airway obstruction and was managed initially with the Heimlich maneuver and subsequently developed increased work of breathing. The patient was managed in the intensive care unit and discharged after resolution of symptoms without sequel.

Key words: Postobstructive pulmonary edema, Acute airway obstruction, Heimlich maneuver

Introduction

Postobstructive pulmonary edema, also termed negative pressure pulmonary edema, is a relatively rare cause of pulmonary edema occurring at the time of or following the relief of an upper airway obstruction. The pathogenesis of pulmonary edema is multi-factorial, however the pathogenesis is predominantly focused on large negative intrapleural and transpulmonary pressure produced by forceful inspiration against a closes glottis that results in transudation of fluid from the pulmonary capillary into the interstitial and alveolar space. Appropriate therapy hinges upon quick recognition of the process and an understanding of underlying pathophysiology.

Case report

A 92-year-old female was sent to the emergency department after choking on bread at home. At the time of choking, the patient developed cyanosis, respiratory distress, and subsequently became less responsive. Her family performed the Heimlich maneuver, and the bread was expelled from the patient’s mouth. The patient recovery breathing, but consciousness was unclear.

Vital signs were temperature of 37.1°C, pulse rate of 87 beats/min, blood pressure 170/72 mmHg, respiratory rate of 24 beats/min with an oxygen saturation of 80% on room air. The patient was placed on 100% oxygenation by a non-rebreather facemask. On chest examination, inspiratory crackles was noted at bilateral lung. Her neurologic examination was nonfocal, and her Glasgow Coma Scale was E3V2M4.

Laboratory evaluation demonstrated a arterial blood gas with a pH of 7.356, pCO2 of 41.5mmHg, and PO2 59mmHg, HCO3 23.5mmol/
L, O₂ sat 89% under nonrebreather facemask. Electrolytes were sodium 140mEq/L, potassium 3.8 mEq/L. Blood urea nitrogen was 15mg/dl, and creatinine was 0.6 meq/L. A complete blood count showed a white blood cell count of 7,900 cells/mm³, hematocrit of 33.4%, and platelets of 257,000/uL. Troponin was 0.032 ng/ml, and creatinine phosphokinase-MB was 10 μL. Chest radiograph revealed bilateral diffuse infiltrates (Fig. 1).

The patient was admitted to intensive care unit for further management. Her condition continued to improved. The CXR taken at ICU on the next day showed clear lung. She was discharged on the fourth day.

Discussion

Pulmonary edema in association with upper airway obstruction was first described in the literature in 1927, when it was observed that prolonged inspiration against a fixed resistance resulted in pulmonary edema in an experimental dog model. Subsequently, pulmonary congestion was described in humans as and autopsy finding in victims of suicidal hanging. The process was described in children as early as 1966, when pulmonary edema was observed in pediatric patients with tonsillar and adenoid hypertrophy and was hypothesized to result, in part, from the variations in intrathoracic pressure produced by respiration against a partial airway obstruction. The first pediatric case reports describes postobstructive pulmonary edema were published in the 1970s and described pulmonary edema following foreign body aspiration and crop and epiglottitis. The first case series in adults was published in 1977 and described pulmonary edema in association with laryngeal tumor, strangulation, and interrupted hanging. Additional case series and reports have subsequently described pulmonary edema as a relative rare complication of acute and chronic upper airway obstruction from a multitude of cause. The variation in these reports regarding the timing and severity of the edema indicates that post-obstructive pulmonary edema likely represents a spectrum of a disease process.

The pathophysiology of pulmonary edema resulting from upper airway obstruction is still not fully understood. However, the mechanism proposed for its development is mainly focused on the generation of a negative intrathoracic pressure during inspiration against an obstructed upper airway. Inspiration against a closed glottis can lead to intrathoracic pressure of -110 cm H2O or more. Reduction of intrathoracic pressure causes an increase in left ventricular transmural pressure and therefore and increasing functional left ventricular afterload. Increased impedance to left ventricular ejection leads to incomplete emptying of the left ventricle, increasing left ventricular end-diastolic volume and left ventricular filling pressure, which in turn results in increasing hydrostatic pressure at the pulmonary micro-vascular level. Alveolar flooding, although always spectacular, occurs only in small number of patient with acute or chronic airway obstruction and seems to directly relate to

Fig. 1. Chest X-ray revealing bilateral interstitial and alveolar infiltrates.
the magnitude of the increase in hydrostatic pressure\textsuperscript{18}.

Hypoxia, which plays an important role in the etiology of neurogenic and high-altitude pulmonary edema, also contributes to edema formation in postobstructive edema. Hypoxia results from blocked alveolar ventilation and the V/Q mismatch which follows edema formation. Hypoxia is known to cause pulmonary vasoconstriction, and has been shown to elevate pulmonary artery pressure\textsuperscript{19}. Hypoxemia has also been shown to trigger an adrenergic response from the central nervous system. This sympathetic outflow results in peripheral vasoconstriction and the shift of a large volume of blood from the systemic to the pulmonary circulation, leading to increases in pulmonary vascular pressure.

Direct capillary damage may be another contributing factor in the development of postobstructive pulmonary edema. It has been suggested that the pressure changes after obstruction produce mechanical stresses that cause direct damage to the capillaries and that the increased permeability of the damaged capillaries results in leakage of fluid into the interstitium\textsuperscript{20}. It has been observed that postobstructive edema usually follows relief of the airway obstruction. One possible explanation for this observation is that expiration against the obstruction produces an effect to positive end expiratory pressure ventilation, which increases the lung volume and thereby masks the presence of edema. With the decrease in lung volume after relief of obstruction, pulmonary edema becomes clinically and radiographically apparent.

In summary, acute upper airway obstruction deserves greater recognition as a causative agent in pulmonary edema. Variant causes of upper airway obstruction have resulted in similar syndrome of rapid onset of pulmonary edema followed by quick resolution with supportive therapy. Aggressive hemodynamic monitoring, mechanical ventilation, or drug therapy is not need in most cases. Maintenance of adequate oxygenation and a patent airway are the mainstays of management.

References

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--- A case report

上呼吸道阻塞性肺水腫 --- 病例報告

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摘要

急性呼吸道阻塞性肺水腫是一種少見之後遺症，且在小孩及成人皆有報告被提出來。對於此種病症，適當的診斷與處置是適當預後的重要因素。我們報告一個因麪包哽住，造成急性上呼吸道阻塞性肺水腫之病例，症狀發生於完全上呼吸道阻塞之後，立即以哈姆立克法急救而恢復呼吸，此病患被送到加護病房照護，在症狀緩解之後出院，沒有留下後遺症。

關鍵詞：阻塞性肺水腫，急性呼吸道阻塞，哈姆立克法

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