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Non-cardiogenic pulmonary oedema due to foreign body aspiration

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Abstract
A patient who developed non-cardiogenic pulmonary oedema secondary to acute airway obstruction caused by an aspirated foreign body is presented. The literature is reviewed, discussing the theories regarding the formation of non-cardiogenic pulmonary oedema. The case highlights the importance of this rare complication of foreign body aspiration and surgeons and anaesthetists should be alert to continued respiratory symptoms following relief of acute airway obstruction.

Key words: Pulmonary oedema; Foreign bodies; Aspiration

Introduction

Non-cardiogenic pulmonary oedema has been described in both adults and children secondary to acute upper airway obstruction caused by laryngospasm, post-intubation laryngeal oedema, epiglottitis and laryngo-tracheal foreign body. We present a case of pulmonary oedema following acute airway obstruction due to a balloon fragment lodging in the laryngeal inlet of a 12-year-old boy. This resulted in an ITU stay of 48 hours. Most cases in the literature describe a stormy course of events and surgeons should be aware of continued respiratory symptoms following upper airway obstruction.

Case report

A 12-year-old boy presented to the accident and emergency department in respiratory distress. He had been running around with a balloon in his mouth when he fell to the floor. His father witnessed him turning blue and carried out a Heimlich's manoeuvre after which the child coughed, spluttered and seemingly improved but remained markedly breathless. The child was rushed to the accident and emergency department. On initial examination the child was tachypnoeic with a respiratory rate of more than 30/minute and peripherally cyanosed. His pulse rate was 120 beats per minute. Pulse oximetry showed an oxygen saturation 70 per cent on air which improved to 96 per cent on 60 per cent inspired oxygen. Auscultation of his chest was difficult due to his stridulous breath sounds, but revealed bronchial breath sounds and bilateral course crepitations with decreased air entry on both sides. A portable chest X-ray taken at this stage showed hazy lung fields on both sides (Figure 1).

An initial diagnosis of tracheal or bronchial foreign body was made. The child was taken to theatre for emergency bronchoscopy which failed to reveal any foreign body in the respiratory tract. Flexible gastroscopy revealed the foreign body to be in the stomach. After bronchoscopy it was difficult to extubate the child because the saturations kept dropping to between 50 and 60 per cent SaO_2 on air.

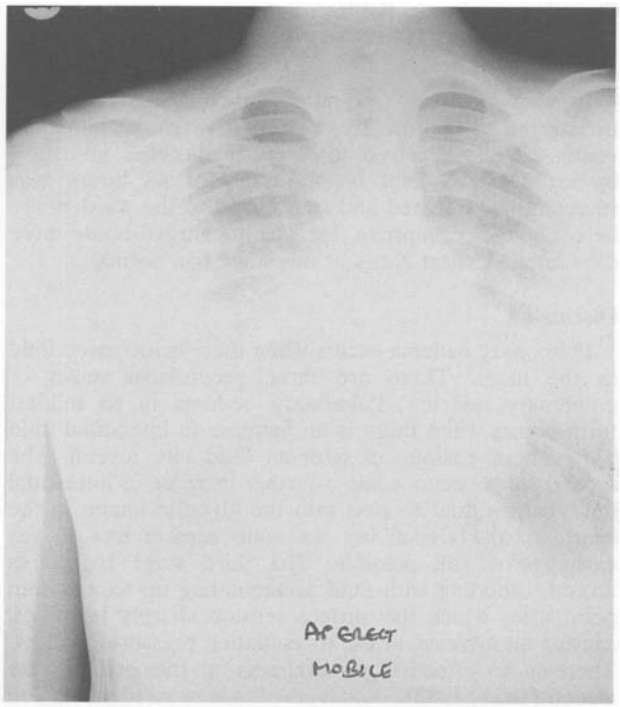

Fig. 1
Initial chest X-ray taken in the casualty department showing pulmonary oedema.

A repeat chest X-ray taken at this stage (Figure 2) revealed clear evidence of pulmonary oedema. With the benefit of hindsight, the initial portable chest film also showed evidence of pulmonary oedema, but this had been disregarded as a poor quality film. At no stage during admission and stay in casualty was the patient given any intravenous fluids.

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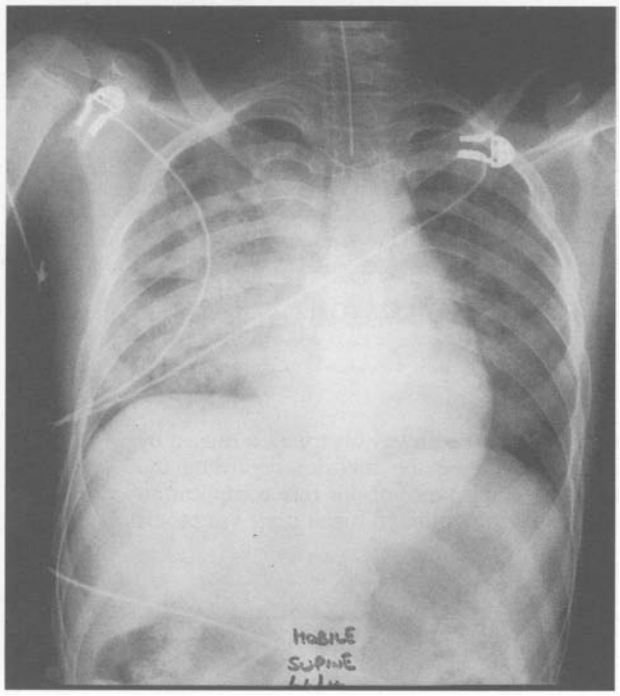

FIG. 2
Chest X-ray taken in the intensive care unit confirming the pulmonary oedema.

In view of poor respiratory function the child was transferred to the intensive care unit where he remained ventilated and received intravenous diuretics and antibiotics. He was kept on the unit for 48 hours, was successfully extubated and transferred to the ward where he continued to improve. He was discharged home three days later. A chest X-ray at this stage was normal.

Discussion

Pulmonary oedema occurs when there is increased fluid in the lungs. There are three progressive stages of pulmonary oedema. Pulmonary oedema in its mildest form occurs when there is an increase in interstitial fluid but without passage of oedema fluid into alveoli. The second stage occurs when a further increase in interstitial fluid, causes fluid to pass into the alveolar lumen in the centre of the alveoli but the walls remain free so gas exchange is still possible. The third stage represents alveolar flooding with fluid accumulating up to a certain point after which the surface tension sharply increases, causing an increase in the transudation pressure gradient. There is no effective gas exchange at this point in the alveoli (Issak, 1982).

The aetiology of pulmonary oedema can either be cardiogenic, or non-cardiogenic. Cardiogenic pulmonary oedema is secondary to heart failure. An increase in the back pressure causes an accumulation of fluid in the lungs. Non-cardiogenic pulmonary oedema is based on Starling forces which keep the alveoli open (Galvis *et al.*, 1980). During acute airway obstruction large negative pressures develop causing fluid to leak out from the capillaries into the interstitial space. Neurogenic pulmonary oedema occurs due to an imbalance of the sympathetic nervous system where an increased adrenergic discharge leads to peripheral vasoconstriction with elevation of blood pressure and shift of fluid to the central circulation (Theodore and Robin, 1975; Staubb, 1988). The effect of the circulating catecholamines is more pronounced in the systemic circulation. This causes an increase in blood volume within the pulmonary circulation leading on to pulmonary hypertension and oedema (Oswalt and Gates, 1977).

In any type of pulmonary oedema when interstitial fluid increases it reduces lung compliance, and large inspiratory pressures must then be generated which increase the work of breathing. This results in fatigue of muscles causing decreased tidal volumes and worsening gas exchange (Issak, 1982; Kanter and Watchko, 1984).

Although in upper airway obstruction the exact pathophysiology of pulmonary oedema is uncertain, it may be an interaction between the various mechanisms postulated. We believe that the problem of fluid transudation occurs due to a combination of increased pulmonary vascular volumes secondary to high negative pressures of inspiration, alveolar hypoxaemia constricting the arterioles and anxiety-related sympathetic discharge shifting the circulating blood volume into the pulmonary system. Hypoxia also directly affects the permeability of the basement membrane.

The formation of pulmonary oedema can either occur during acute airway obstruction or after its relief. In the acute stage it is because of the mechanical stresses to the basement membrane, whereas delayed pulmonary oedema could be because the changes in surfectant levels in the alveoli following hypoxia. Clinically apparent pulmonary oedema may occur up to several hours after airway obstruction is relieved. This is dependent on the severity of the obstruction, the degree of hypoxaemia, the inspiratory efforts of the patient in attempting to overcome the obstruction, and the underlying cardiopulmonary status of the patient. Thus patients with acute upper airway obstruction should be observed for several hours even after the obstruction has been relieved (Issak, 1982).

Mild pulmonary oedema can be treated with inspired oxygen and diuretics. More severe pulmonary oedema may require intravenous diuretics and intermittent positive airway pressure ventilation (Oswalt and Gates, 1977; Travis *et al.*, 1977; Rao and McNiece, 1986). This has relevance in ENT in patients with tracheostomies. In the post-operative period after tracheostomy, the tube may block with secretions or crusts. This can result in pulmonary oedema due to the increased respiratory effort. This should be kept in mind if patients do not improve after tracheostomy tube changes.

At no stage during the patient's stay in hospital were any intravenous fluids given. It is well known that fluid overload is a common cause of non-cardiogenic pulmonary oedema in intensive care and casualty departments.

Clinicians should not disregard X-ray findings even when the films are obtained using portable equipment and the quality of films is not ideal. All films of doubtful reliability should be reported by a radiologist. We postulate that this child aspirated the balloon fragment into the laryngeal inlet causing acute upper airway obstruction. His father managed to expel this balloon into his pharynx using Heimlich's manoeuvre. The patient then swallowed the balloon. He may have developed pulmonary oedema at the time of the obstruction, or shortly thereafter, but his low oxygen saturation present in Casualty is likely to have been due solely to pulmonary oedema and not obstruction. Clinically, the laryngoscopy and bronchoscopy was the correct course of action, in view of the strong history of aspiration.

Conclusion

This case highlights the dangers of pulmonary oedema associated with acute airway obstruction. The initial portable chest X-ray film did demonstrate features of pulmonary oedema but these were thought to be because the film was of poor quality. A clinician should not ignore X-ray findings, even if the films are of doubtful quality.

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