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Acute upper airways obstruction and pulmonary oedema – Case reports

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Summary

Attention is drawn to acute upper airways obstruction as an uncommon but important cause of non-cardiogenic pulmonary oedema. Two cases managed by the authors recently are reported. Possible mechanisms in the pathogenesis of this condition are reviewed and the role of ventilation and diuretics in treatment is discussed.

Key words – Acute upper airway obstruction, non cardiogenic pulmonary oedema.

Introduction

Non-cardiogenic pulmonary oedema is an uncommon condition. Causes include drugs, sepsis, head injury and high altitude. Pulmonary oedema following acute upper airway obstruction is a recently recognised phenomenon. We describe two cases managed recently in the intensive care unit at our hospital.

Case One

M.F., a two-year old male, was admitted with severe respiratory distress following accidental inhalation of a peanut. On examination he was semi-conscious, cyanosed and had severe sternal and intercostal retractions associated with a tracheal tug. Several attempts to dislodge the peanut using Heimlich's manoeuvre and back blows were unsuccessful. The child was then brought to the operating theatre where emergency bronchoscopy under general anaesthesia with halothane and oxygen was attempted. However, he developed severe bradycardia and was immediately intubated and ventilated with 100% oxygen, with marked improvement in his condition. Immediately following intubation, copious pinkish froth was suctioned from the trachea and widespread crepitations were heard over both lungs. Bronchoscopy was attempted again and the peanut removed 'piece-meal' from the child's upper airways.

The child was subsequently managed in the Intensive Care Unit. Intravenous Frusemide 10mg, followed by a further 5mg was given with good diuretic response. A Chest X-ray (Fig. 1) supported the diagnosis of pulmonary oedema. The heart size was normal. Initial blood gas analysis showed metabolic acidosis and hypoxaemia with PaO_2 of 50 Torr. In view of the intraoperative episodes of hypoxia with bradycardia and subsequent pulmonary oedema with hypoxaemia, the child was put on intermittent positive pressure ventilation using a 'Baby Bird' venti-

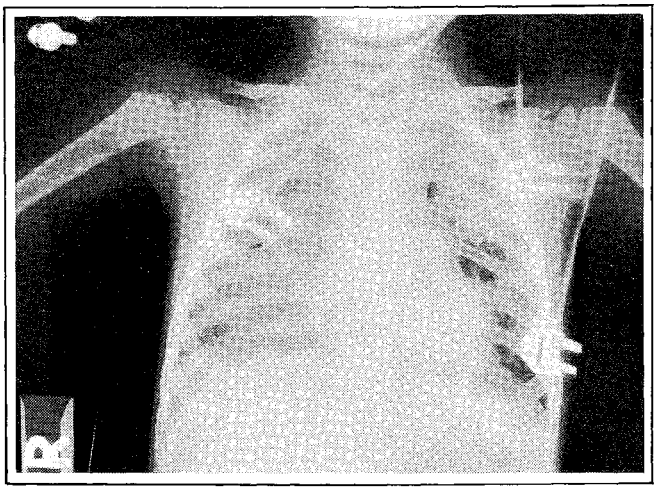

Fig. 1:
Chest radiograph of case one.

lator with F_1O_2 of 0.5. Muscle relaxation was maintained with intermittent 0.4mg bolus doses of pancuronium. Sedation was provided with 3mg bolus doses of diazepam six hourly. No further doses of diuretic was given. There was rapid resolution of the pulmonary oedema with improvement in oxygenation. The child was weaned off the ventilator after 48 hours when the PaO_2 was maintained at 90 Torr with F_1O_2 of 0.4. He subsequently made a complete recovery.

Case two

P.A., a 39-year old female, was admitted with respiratory distress of five days' duration. A near-total thyroidectomy had been performed on her 10 years previously for benign multinodular goitre. Five years ago, her thyroid gland had started to enlarge again and she had since suffered recurrent episodes of respiratory difficulty. On examination, she had severe inspiratory stridor and was using her accessory muscles of respiration. A CT scan of the thyroid region showed a large retrosternal goitre compressing the trachea, which appeared like a slit on the film (Fig. 2).

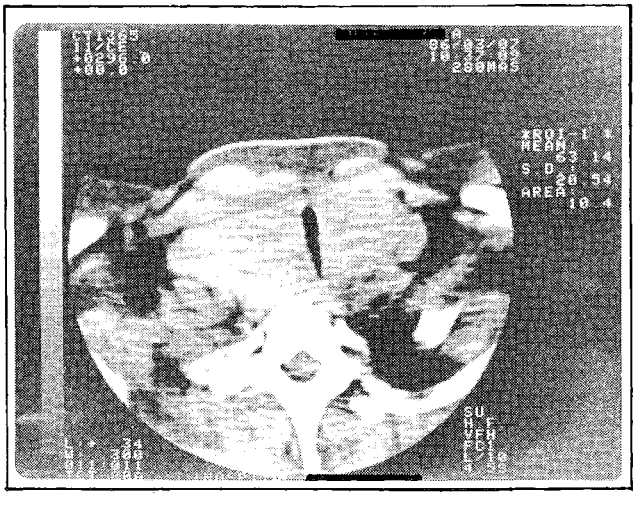

Fig. 2:
CAT scan of thyroid region, Case two.

Her condition deteriorated and she had respiratory arrest with cyanosis and severe bradycardia. Her trachea was immediately intubated, whereupon copious, pinkish frothy sputum issued forth from the endotracheal tube. She was given a bolus dose of intravenous Frusemide 80mg. Arterial

blood gas analysis showed metabolic acidosis with hypoxaemia, and she required intermittent positive pressure ventilation, using an OHIO CCV2 ventilator, with an initial F_1O^2 of 0.5. Muscle relaxation was maintained with intermittent bolus doses of pancuronium, and she was kept sedated with a morphine infusion.

The pulmonary oedema cleared within 24 hours with improvement in oxygenation, and she underwent total thyroidectomy the following day. Operative findings were an enlarged thyroid gland with marked distortion of anatomy. It was not possible to identify the right recurrent laryngeal nerve, which had to be sacrified. In addition there was tracheomalacia resulting from longstanding compression by the goitre. For these reasons the patient required a permanent tracheostomy.

Discussion

Non-cardiogenic pulmonary oedema may result from a number of causes, which include head injury, sepsis, drugs and high altitude. Upper airway obstruction is an uncommon but important cause, and was first described in children with acute epiglottitis[1] and croup. Other causes of upper airways obstruction associated with pulmonary oedema were reviewed recently,[2] and include laryngospasm, malignancy, goitre, sleep apnoea and strangulation. Pulmonary oedema secondary to airway obstruction caused by a foreign body, as seen in Case One, has not been reported before.

The underlying mechanism causing pulmonary oedema in such cases are unclear. Hypoxia is thought to play a major role, as the resulting pulmonary vasoconstriction leads to increased pulmonary capillary pressure. Neurogenic stimulation with resultant systemic vasoconstriction, and negative pleural pressures generated by breathing against an airway obstruction are also thought to be important, as both factors increase pulmonary blood volume and pulmonary capillary pressure, thus increasing the transmural gradient which drives fluids from the pulmonary capillaries into the interstitial space. In addition injury to the pulmonary capillary endothelium caused by hypoxia, and markedly negative intrapleural pressures are factors which directly favour movement of fluid across the pulmonary endothelium.

It has been noted that in more than 50% of documented cases pulmonary oedema occurred after relief of obstruction.[2] This was seen in Case Two and possibly also occurred in Case One (where the foreign body may have been dislodged by the endotracheal tube, resulting in at least partial relief of obstruction). Why this should be so is unclear. One theory is that the balance of intra-thoracic forces which prevent the oedema initially are overcome upon relief of the obstruction.[3] It is important to suspect this complication if relief of airway obstruction by endotracheal intubation does not immediately improve the patient's respiratory status.

There appears to be no clear relationship between the duration of asphyxia and detection of pulmonary oedema. In Case One, there was an interval of three hours between inhalation of the foreign body and relief of obstruction in hospital. In Case Two, it is likely that longstanding hypoxia was present. A similar situation is seen in children with reversible cor pulmonale and pulmonary oedema secondary to chronic upper airway obstruction, usually caused by hyper-trophied tonsils and adenoids.[2] It is postulated that mechanisms responsible for pulmonary oedema are similar whether it follows acute or chronic airway obstruction.

Treatment is aimed at maintaining adequate arterial oxygen tensions. The majority of cases will require assisted ventilation with appropriate oxygen enrichment of inspiratory gas. Positivie pressured ventilation will help to drive fluid out of alveoli back into capillaries. Froth interferes with ventilation of alveoli and should be suctioned out. The use of diuretics in non-cardiogenic pulmonary oedema has been discouraged as intracardiac pressures may be normal.[2] In addition

diuretics may decrease cardiac output and impair oxygen delivery to tissues.[4] However, other workers have argued that in pulmonary oedema due to increased pulmonary capillary permeability, and level of left atrial or pulmonary capillary wedge pressure causes more filtration than normal, and thus it may be justified to use diuretics even if pulmonary vascular pressures are within the normal range.[5] In the two cases reported here, diuretics were used empirically, as intracardiac pressures were not measured initially, the relative contribution of positive pressure ventilation and diuretics to resolution of pulmonary oedema in these cases is difficult to ascertain.

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