The Journal of Laryngology and Otology
May 1997, Vol. **111**, pp. 482–484

Acute pulmonary oedema complicating laryngospasm

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Abstract
Pulmonary oedema is an uncommon but important complication of laryngeal spasm which in turn occurs more commonly in ENT practice than in most other surgical specialities. A case is reported and the literature reviewed, with particular reference to the proposed pathophysiological mechanism of this phenomenon.

Key words: Pulmonary oedema; Larynx; Spasm

Introduction

Otolaryngologists and head and neck surgeons frequently share the upper airway with their anaesthetic colleagues. This usually causes no problems but from time to time complications can occur. Laryngeal spasm is relatively common in patients following ENT operations. Pulmonary oedema is a rare complication of such a spasm with which otolaryngologists should be familiar.

Case report

A previously well 44-year-old male was taken to theatre for examination of his nose under general anaesthetic after suffering a left-sided epistaxis. This had failed to respond to six days of conservative management and to formal bilateral anterior nasal packing with bismuth iodoform paraffin paste (BIPP) ribbon gauze under a previous general anaesthetic two days earlier. His haemoglobin concentration had fallen from 13.4 g/dl to 9.1 g/dl, although his platelet count and clotting indices (activated partial thromboplastin time, prothrombin time, international normalized ratio and thrombin time) were all within normal limits.

He was anaesthetized with a rapid sequence induction, paralysed with suxamethonium and intubated with a size 9 RAE™ endotracheal tube. Endoscopic examination revealed bleeding from the left superior meatus of the nose and his left middle turbinate was excised and haemostasis achieved. A formal post-nasal pack was inserted, and appropriately secured. Both sides of the nose were packed anteriorly with BIPP ribbon gauze. He was extubated uneventfully in the operating theatre and transferred to the recovery room. He then developed acute laryngeal spasm, with transcutaneous oxygen saturation falling to 40 per cent. Suxamethonium 25 mg was given intravenously and relieved the spasm but hand ventilation with 100 per cent oxygen via a face mask led to an increase in oxygen saturation to only 78 per cent. The patient was re-intubated and copious amounts of pink foamy liquid were sucked from the endotracheal tube. The patient's oxygen saturation improved on intermittent positive pressure ventilation with 4 cmH_2O positive end expiratory pressure (PEEP). He was then sedated and transferred to the intensive care unit for overnight ventilation.

On admission to the ITU his arterial blood gases with an FIO_2 of 60 per cent showed PaO_2 19.40 kPa, $PaCO_2$ 6.14 kPa, pH 7.39 with a standard bicarbonate concentration of 27.9 mmol/l. A chest radiograph (Figure 1) showed bilateral fluffy hilar shadowing. The appearances are perhaps more characteristic of aspiration changes, and are atypical for pulmonary oedema. However, there was a little ground glass shadowing in the perihilar region which was still present on the next day (Figure 2) with a reticular pattern in the bases that was felt to support a clinical diagnosis of pulmonary oedema. No aspiration had been noted at any stage of the patient's management.

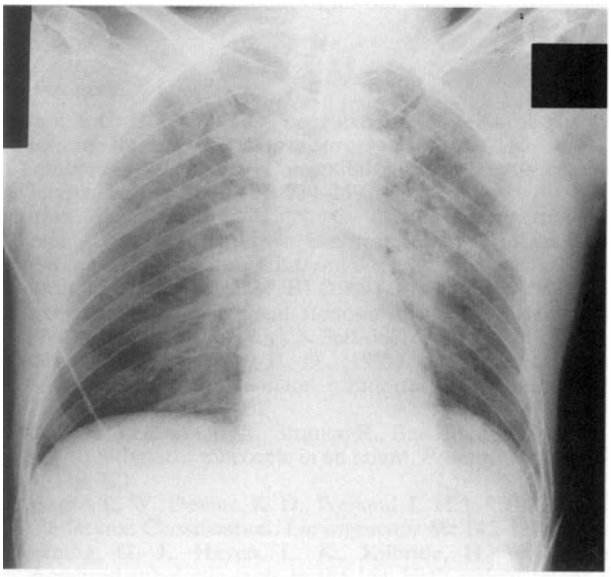

FIG. 1
Chest radiograph demonstrating bilateral hilar shadowing. There had been no clinical aspiration.

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Accepted for publication: 17 March 1997.

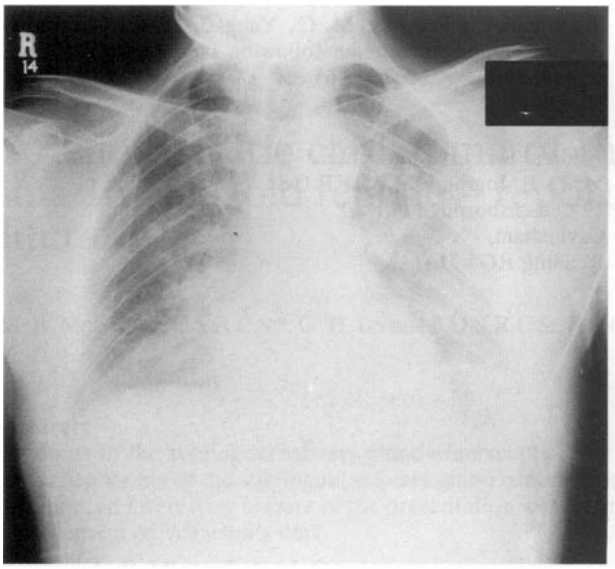

FIG. 2
Chest radiograph taken 12 hours later showing persistence of ground glass shadowing in the perihilar region consistent with pulmonary oedema.

Over the next 12 hours his condition improved rapidly, and FIO_2 was reduced gradually to 28 per cent; arterial blood gases were PaO_2 12.79 kPa, $PaCO_2$ 5.57 kPa, pH 7.43 with a standard bicarbonate concentration of 27.3 mmol/l. In the light of his rapid improvement a decision was made to not give diuretics.

He was subsequently extubated uneventfully and had no further difficulty maintaining satisfactory oxygen saturation. There was no further nasal bleeding before or after pack removal four days later. He was well on discharge home, five days after the episode of pulmonary oedema and did not rebleed over a three month follow-up period.

Discussion

Pulmonary oedema is an uncommon but well recognized complication of laryngospasm (Oswalt *et al.*, 1977). Laryngospasm is an exaggerated, prolonged glottic closure in response to direct glottic or supraglottic stimulation (Stone and Gal, 1990). The latter is mediated by the superior laryngeal nerves in response to stimulation from inhaled agents, secretions, foreign bodies, etc. (Donlon, 1986; Stone and Gal, 1990). Spasm persists after mucosal irritation ceases. As hypoxia and hypercapnia develop there is a reduction in the output from the brainstem via the vagus nerve, and the larynx relaxes (Donlon, 1986).

Noncardiogenic pulmonary oedema associated with intubation of the trachea has been described in both adults (Lorch and Sahn, 1986; Knutrud Meidel and Skulberg, 1989) and children (Lee and Downes, 1983). It is also well recognized that it may occur following relief of upper airway obstruction (Weissman *et al.*, 1984; Halow and Ford, 1993). In addition it has been reported in children following general anaesthesia using manually assisted ventilation via a mask (Lee and Downes, 1983; Guinard, 1990). The condition seems to be more commonly associated with otolaryngological procedures. In a recent review of 19 cases, 10, the largest single group, were in ENT patients (Halow and Ford, 1993).

The pathogenesis of pulmonary oedema in post-operative laryngospasm is multifactorial. The initial event is triggered by the large intrapleural negative pressure created by attempted inspiration against a closed glottis. This leads to an increase in blood flow to the right side of the heart, and a decrease in flow from the left side due to increased afterload. The combined effect of these is increased pulmonary blood volume and raised pulmonary venous pressure, which together with negative interstitial pressure promotes the formation of pulmonary oedema. There is in addition a central redistribution of blood volume secondary to hypoxia, hypercarbia and cerebral ischaemia which increases pulmonary venous congestion. Left ventricular function is also compromised by acidosis and hypoxia, further predisposing to oedema. There may also be an increase in capillary permeability secondary to hypoxia (Lang *et al.*, 1990).

Several hypotheses have been advanced to explain the onset of oedema following relief of obstruction, rather than during the obstruction itself (Galvis *et al.*, 1980). It has been proposed that during complete obstruction the intermittent negative intrapleural pressures on attempted inspiration are balanced by equal positive pressure on attempted expiration (Masa-Jimeney *et al.*, 1985). In addition, there may be an element of auto 'PEEP', a positive airway pressure at the end of expiration being created by air trapped in the alveoli (Pepe and Marini, 1982).

Treatment of pulmonary oedema secondary to laryngeal spasm is dependent on its severity. In most cases it is self-limiting, resolving spontaneously within 12 to 24 hours and requiring supportive therapy only, e.g. oxygen via a face mask. Mechanical ventilation may be required, but can usually be withdrawn within 24 hours. In 40 per cent of cases less than 12 hours of ventilation is required (Halow and Ford, 1993). PEEP of 5 to 10 cmH_2O is used. The efficacy of pharmacological agents such as diuretics and bronchodilators has not been clearly demonstrated (Halow and Ford, 1993).

Acknowledgement

The authors would like to thank Dr Penny Shaw, Consultant Radiologist, The University College London Hospitals.

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