Intensive Care Med (1988) 14:241–243

Intensive Care Medicine

Pulmonary barotrauma including orbital emphysema following inhalation of toxic gas

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Received: 15 December 1986; accepted: 1 Juli 1987

Abstract. Severe pulmonary barotrauma occurred following smoke and toxic gas inhalation in a 20-year-old male. He developed pneumothorax, pneumomediastinum, and extensive facial subcutaneous emphysema which intensified during treatment with positive pressure ventilation. Following the appearance of diplopia and exotropia, orbital emphysema was demonstrated radiologically. The diplopia and exotropia were manifestations of mechanical interference in extra-ocular muscle function by the intra-orbital air, an unusual expression of pulmonary barotrauma.

Key words: Pulmonary Barotrauma – Smoke Inhalation – Intra-orbital Emphysema

Pneumothorax is the most serious expression of pulmonary barotrauma, but subcutaneous emphysema is the most common manifestation. Intra-orbital air has not previously been reported in association with pulmonary barotrauma, and has not been implicated as a cause of diplopia and exotropia. We report a case of severe pulmonary barotrauma associated with orbital emphysema. The resulting diplopia and exotropia slowly resolved as the intra-orbital air was absorbed.

Case report

A 20-year-old male was brought to our emergency room with second degreee burns of both arms and complaining of shortness of breath following his rescue from a burning building. Arterial blood gases showed mild hypoxemia and the chest x-ray was normal. He was treated with 8 mg dexamethasone iv daily, supplemental oxygen therapy by mask, iv penicillin and fluids. On the second day, he became febrile and his heart rate increased to 140 beat/min and respirations to 40 breath/min. Expiration was prolonged and wheezing was heard on auscultation. Subcutaneous emphysema was noted on the anterior chest and abdominal wall. Chest x-ray now showed pneumomediastinum and a diffuse micronodular infiltrate in both lung fields. Arterial blood gases were PO_2 93 torr, PCO_2 50 torr, and pH 7.31 using a mask supplied with an FiO_2 of 0.5. Intravenous aminophylline and aerosolized salbutamol were then administered with no improvement. On the third hospital day despite nasotracheal intubation and mechanical ventilation, P_aCO_2 increased and reached a peak of 121 torr without significant hypoxemia. Peak inspiratory pressure (PIP) during mechanical ventilation was 50 cm H_2O in spite of paralysis with pancuronium bromide, and sedation with midazolam. The subcutaneous emphysema rapidly spread to the neck, both arms, and face such that both eyelids were crepitant and swollen shut. A left-sided pneumothorax developed and a tube thoracostomy was performed, followed immediately by full expansion of the lung. P_aCO_2 did not significantly decrease until expiratory retard was effected using a 3 mm diameter resistor in the expiratory limb of the ventilator circuit.

On the fifth post-injury day, PIP began to decrease and the subcutaneous emphysema began to subside. When he was able to open his eyes, the patient immediately reported double vision, though he was able to see clearly with either eye. On examination, crossed diplopia was noted in all cardinal gazes (Fig. 1). Using the red-green spectacles method, a clear V-pattern deviation was recognized, that is the exotropia was greatest in upward gaze and least in downward gaze. There was a mild right proptosis without signs of injury to the eye or the orbit. Pupillary reflexes were in-

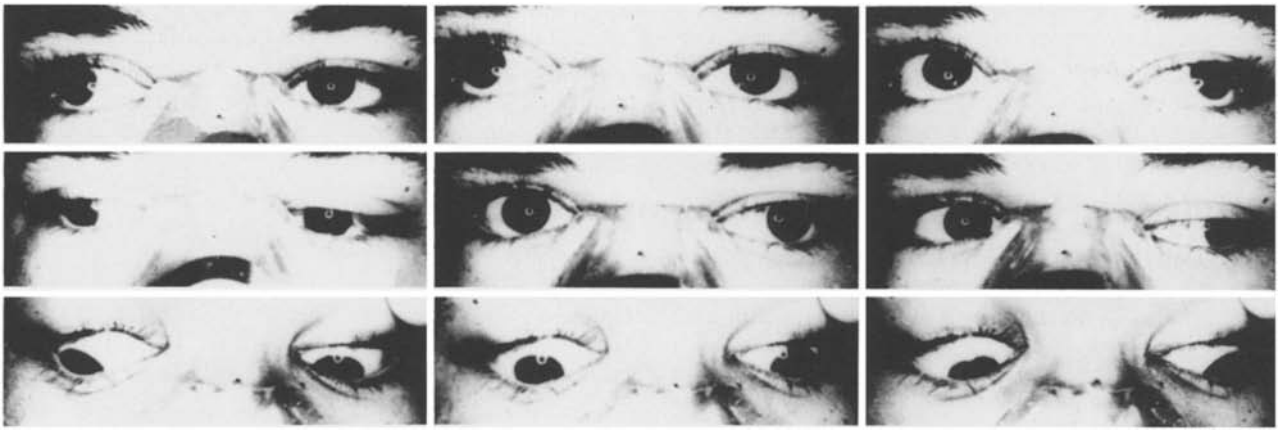

Fig. 1. Cardinal gazes showing V-pattern exotropia

tact and convergence was totally absent. Visual acuity was 6/6. An orbital x-ray demonstrated the presence of air in the superior aspect of both orbits (Fig. 2).

During the following ten days there was a gradual improvement in gas exchange and the tube thoracostomy and endotracheal tube were removed. The diplopia decreased and the convergence partially returned by 2 weeks. A repeat x-ray 14 days after the initial complaint showed no air in the orbits. At this time the patient was symptomless, but residual diplopia could be elicited in right lateral and right upper gaze. One month after the initial complaint, the eye movements returned to normal.

The patient was gradually weaned from oxygen enrichment but continued to have mild hypoxemia and hypercarbia breathing room air due to chronic bronchiolitis obliterans.

Discussion

The clinical course of the patient described here involved a short latent period before the development of severe respiratory failure, suggesting the development of bronchiolitis obliterans from the inhalation of gases, especially phosgene gas and oxides of nitrogen [1], given off by burning furniture and plastics. Corticosteroid therapy, which has been shown to be of benefit in bronchilotis obliterans [1] was administered throughout the hospital course. In spite of aggressive drug therapy and mechanical ventilation, hypercarbia

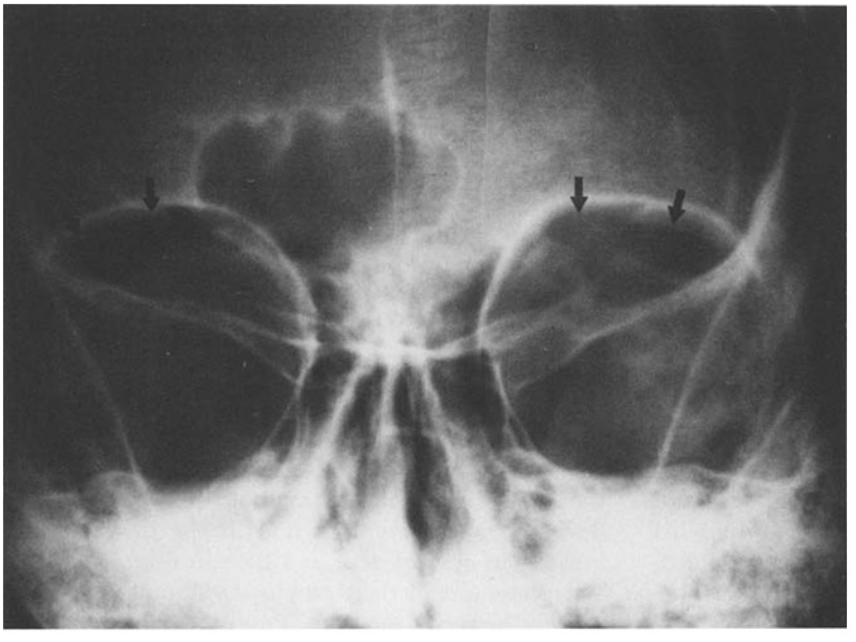

Fig. 2. Orbital x-ray showing bilateral orbital emphysema (arrows)

could only be reduced by use of expiratory retard, a technique previously shown to be efficacious in reducing severe air trapping [2]. PIP reached 50 cm H_2O and the subcutaneous emphysema overlying the face, chest, and trunk became tense. Tense subcutaneous air is a rare cause of ptosis [3] and can apparently enter the orbit resulting in proptosis [4]. We therefore believe that the air which was found in both orbits of the patient described here was a direct result of the tense facial subcutaneous emphysema, though the precise site of entry was not identified.

Intra-orbital air which remains peripheral to the cone of muscles is benign [5]. In order to penetrate through the muscle cone to the central orbital compartment, the air must be under considerably greater pressure, and this pattern of orbital emphysema has serious consequences [5]. The orbital x-ray of this patient (Fig. 2) showed air peripheral to the muscle cones and this indicated a good prognosis with no specific therapy required.

Diplopia has many possible causes, including an orbital space-occupying lesion or a blow-out fracture of the medial wall or the floor of the orbit with entrapment of extra-ocular muscles. The absence of history and signs of trauma, and the temporary nature of the diplopia in the case reported here make the diagnosis of blow-out fracture unlikely. We attribute the diplopia and exotropia to interference with extra-ocular muscle function by the intra-orbital air pocket acting as a space-occupying lesion. The gradual and simultaneous disappearance of the diplopia, the exotropia, and the x-ray of orbital emphysema supports a causal relationship.

References

1. Cahalane M, Demling RH (1984) Early respiratory abnormalities from smoke inhalation. JAMA 251:771
2. Weng JT, Smith DE, Graybar GB, Kirby RR (1984) Hypotension secondary to air trapping treated with expiratory flow retard. Anesthesiology 60:350
3. Widder D (1982) Ptosis associated with iatrogenic pneumothorax. Arch Intern Med 142:145
4. Schneider SM, Goodman D (1984) Spontaneous rupture of the oesophagus presenting with unilateral proptosis. Ann Emerg Med 13:374
5. Bertelsen TI (1962) Pneumotomography of the orbit. Arch Ophthalmol 68:446

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