

Chronic airflow limitation after inhalation of overheated cooking oil fumes

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Summary: A patient who developed severe irreversible airways obstruction with features of bronchiolitis obliterans after exposure to fumes from overheated cooking oil is described.

Introduction

Inhalation burns or inhalation of toxic gases such as the oxides of nitrogen and sulphur, phosgene, ammonia and chlorine may result in the development of severe airflow limitation predominantly due to obliterative bronchiolitis (Epler & Colby, 1983). We report a case in which severe airflow obstruction with features of obliterative bronchiolitis developed after inhalation of fumes from overheated cooking oil.

Case report

A 22 year old non-smoking white man with cerebral palsy associated with mild mental subnormality and frequent generalized epileptic seizures presented in April 1982 having suffered a grand mal fit whilst cooking. A chip-pan containing vegetable oil was on the stove and became overheated, filling the room with fumes though neither the oil nor anything else ignited. The patient was found in the fume-filled room after about 1 h. On examination in hospital he was noted to have injected conjunctivae and a slightly reddened throat but there was no dyspnoea, or cyanosis. The chest was clinically clear and chest radiograph was normal. There was no history of any previous chest complaints or of aspiration during seizures. One week later he attended the Accident and Emergency Department complaining of dyspnoea but examination and the chest radiograph were again normal, peak expiratory flow rate (PEFR) was 380 litres/min and he was referred to the psychiatric department.

Two weeks after the inhalation of fumes he was admitted with severe dyspnoea associated with wheeze. He was cyanosed with pulsus paradoxicus of 20 mmHg, PEFR was 200 litres/min, chest radiograph

was normal and arterial blood gas tensions on 35% O₂ were PO₂ 7.9 kPa, PCO₂ 5.4 kPa, with a pH of 7.43. Despite intravenous aminophylline, nebulized salbutamol and ipratropium bromide, antibiotics and prednisolone (initially 40 mg/d, then 100 mg/d) he deteriorated over the following 10 d. Arterial PO₂ fell to 5.9 kPa with PCO₂ 8.8 kPa, and he was mechanically ventilated for 9 d. Subsequently he has remained dyspnoeic with chronic wheeze and productive cough. Serial pulmonary function tests (Table I) show severe airways obstruction with reduced transfer factor for carbon monoxide. Total lung capacity is increased but lung volume accessible to a single inhalation of helium [V_A] is markedly reduced, suggesting poor gas mixing in the lungs. Flow-volume loops were technically unsatisfactory because of the patient's mental state but suggested increasing airways obstruction at low lung volumes. No significant reversibility to beta₂ agonists, or ipratropium bromide was found.

Values for total lung capacity (TLC) and residual volume (RV) in July 1982 were obtained by helium

Table I Respiratory function tests over 2 y after inhalation of cooking oil fumes

	Predicted	July 1982	March 1983	May 1984
PEFR (l/min)	650	200	—	75
FEV ₁ (l)	4.7	0.8	0.9	0.6
FVC (l)	5.7	2.4	2.1	2.1
FEV ₁ /FVC (%)	83	33	43	29
TLC (l)	7.0	6.1†	8.7*	8.5*
V _A (l)	7.0	—	3.8	3.6
RV (l)	1.3	3.7	6.6	6.6
RV/TLC (%)	19	61	76	78
TLCO (mmol/min/kPa)	13.7	10.0	9.0	8.6

† Helium rebreathing; * Body plethysmograph.

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Accepted: 23 May 1985

rebreathing and are probably under-estimates because of poor gas mixing. There was probably no real change in these values between 1982 and 1983, though by 1984 there seems to have been some deterioration.

Discussion

There can be little doubt that inhalation of the cooking oil fumes caused the severe airflow limitation in this patient. Though there is no direct evidence from histology or bronchography to confirm that the predominant lesion is that of obliterative bronchiolitis the pattern of lung function tests with gas trapping and markedly uneven ventilation (as shown by the disparity between TLC and V_A) suggest that the small airways are abnormal, though large airway involvement is also certainly present.

Obliterative bronchiolitis may be seen after viral infections especially in children (Epler & Colby, 1983), in association with collagen vascular disease (Geddes *et al.*, 1977), associated with chronic bronchitis (Hogg *et al.*, 1968), and rarely may be idiopathic (Turton *et al.*, 1981). In adults, inhalation of toxic fumes is the most frequently recognized cause but the fumes from overheated vegetable oil consist mainly of vaporized glycerides and do not contain any of the toxic gases recognized as causing this syndrome (Smith, R.W., Scientific Services Branch, GLC, personal communication). There was no evidence of airway burns in this case or of any of the associations listed above.

The effects of fats and oils on the lungs after

injection into the trachea of animals were studied by Pinkerton (1928). On the whole, vegetable and mineral oils are relatively non-toxic whereas oils of animal origin cause severe inflammation and fibrosis. Perhaps of more relevance to this case, however, are observations on the chronic lipoid pneumonia seen in blackfat tobacco smokers in Guyana (Miller *et al.*, 1971). Blackfat tobacco is impregnated with a light mineral oil (canopus oil) and petroleum jelly as flavouring. Chronic smoking of this tobacco produces a widespread pulmonary fibrosis with severe irreversible airways obstruction and increased residual volume without increase in total lung capacity. Histologically there is an interstitial fibrosis with lipid deposition in alveolar macrophages and with granuloma formation in severe cases. Interestingly, bronchiolitis obliterans may also be associated with interstitial infiltrates or fibrosis (Seggev *et al.*, 1983).

It seems, therefore, that inhalation of the fumes of even non-toxic oils may cause severe lung damage. Domestic fires involving cooking oil are common and whilst brief exposure to fumes in this situation has not been reported to have long-term adverse effects we would suggest that patients exposed to such fumes for more than a few minutes should be carefully assessed.

Acknowledgements

We thank Dr R. Hainsworth for the pulmonary function test data and for helpful advice and Mrs K. Marsden for secretarial assistance.

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