

Acquired progressive asthma in a fire-fighter

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ABSTRACT: A fire-fighter, who developed chronic severe asthma after exposure to decomposition products of plastics during routine fire-fighting, is described. The outcome was fatal 25 months after onset of the disease.

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Fire-fighters are exposed to toxic and irritant combustion products from fires [1]. Several authors have reported on chronic lung function loss in fire-fighters [2-4]. Development of severe airway obstruction in relation to fire-fighting has previously been reported [5]. We describe a fire-fighter who developed severe progressive asthma after fire-fighting.

Case report

The patient was a 42 yr old fire-fighter, with no previous history of allergy or pulmonary disease, who had stopped smoking one year earlier. In October 1977, when fighting a fire without wearing any respiratory protection, he was suddenly engulfed by smoke from burning plastics under high pressure. After the fire he immediately started to cough, and after a few days he complained of tightness in the chest with dyspnoea and wheezing. In a short time, he developed all the classical signs of asthma.

He was referred to our department in April 1978 with severe breathing difficulties, coughing and wheezing. Physical examination on admission revealed normal breathing rate but prolonged expiration. Heart rate was 93 and blood pressure 140/80 mmHg. Chest X-ray showed obliteration of the right pleural sinus but was otherwise normal. Arterial blood gases showed a slight hypoxia (pH 7.41, arterial oxygen tension (P_{aO_2}) 8.5 kPa, arterial carbon dioxide tension (P_{aCO_2}) 4.8 kPa, standard bicarbonate 23 mmol/l). Routine blood analyses were normal, but before admission to our department he had 19% eosinophils in a differential count of white blood cells, and also eosinophils in the sputum. Serum protein analysis showed an increase in haptoglobin (4.1 g/l, reference value <2.5 g/l), the levels of immunoglobulins and alpha-1-antitrypsin were normal. Immunologic investigation showed normal IgE, and no specific antibodies against various allergens including pollens, dust, house mite, mould and animals could be detected by means of radio allergosorbent test

(RAST). Spirometry varied between normal values (vital capacity (VC) 4.73 l, forced expiratory volume in one second (FEV_1) 3.76 l, maximum ventilation volume (MVV) 182 l/min) and pronounced obstructive ventilatory impairment (VC 3.0 l, FEV_1 1.1 l, MVV 50 l/min). Ventilation and perfusion radionuclide studies (technetium^{99m}, thallium²⁰¹) showed similar patterns with scattered defects.

For further investigation of the cardio-pulmonary function and alveolar gas exchange, a catheterization of the pulmonary and brachial arteries was performed, in connection with a constant infusion of biologically inert gases. On account of the clinical status, intravenous infusions of terbutaline, theophylline and hydrocortisone were given during the investigation. There was a high cardiac output (13.6 l/min, stroke volume 136 ml, heart rate 100/min), which was probably an effect of terbutaline after vasodilation caused by theophylline and terbutaline, and an elevated oxygen uptake (397 ml/min, arteriovenous oxygen difference 29 ml/l), which was thought to be caused by terbutaline-induced tremor. The arterial hypoxaemia (P_{aO_2} 8.8 kPa, P_{aCO_2} 4.7 kPa) was mainly due to perfusion of poorly ventilated regions of the lungs; the venous admixture was calculated to 13%.

The patient was treated with intravenous infusions of hydrocortisone, terbutaline and theophyllamine together with inhalation of terbutaline. His symptoms disappeared, and the blood gases and spirometry became normal. He was discharged from the hospital after two weeks with a maintenance therapy consisting of terbutaline 5 mg three times daily, choline-theophyllinate 200 mg four times daily, bromhexine 8 mg three times daily, prednisolone 15-5 mg daily and aerosol inhalations of fenoterol 0.4 mg four times daily and beclomethasone 100 microgram four times daily.

Attacks of coughing, followed later by dyspnoea and wheezing, reappeared soon after release from hospital and gradually grew worse. Intensive intra-

venous treatment with corticosteroids and bronchodilators became necessary more frequently, and at times he was coughing continuously day and night and arrived at hospital totally exhausted. During the following 18 months, he was never able to stay discharged from hospital for more than seven weeks at a time. As a last attempt first azothioprine and then chloroquine was tried with no effect. Artificial respiration was considered on account of severe hypoxaemia but did not become necessary until November 1979, when he was admitted to hospital in a severe status asthmaticus. Despite artificial ventilation and intensive intravenous medication, his clinical status deteriorated, and after two hours ventricular fibrillation developed. Resuscitation was unsuccessful, and he died 25 months after the onset of the asthma. Autopsy showed emphysema of high grade and tenacious mucus in the bronchi.

Discussion

The patient had been completely free from respiratory symptoms until the occasion reported, and we believe that there is no doubt about the causal connection between the exposure to smoke and development of persistent asthma. During the fire he was exposed to decomposition products of plastics, and it has been shown that many toxic and irritant gases are produced in such conditions [6-8]. MUSK *et al.* [9] found an acute decline in FEV₁ related to the severity of smoke exposure in fire-fighters, and SHEPPARD *et al.* [10] likewise found acutely lowered FEV₁ after fire-fighting. Measurement of metacholine sensitivity suggested that this was due to an acquired increase in airway responsiveness. Persistent asthma 2½ yr after exposure to smoke has earlier been described by LOKE *et al.* [5], who reported a fire-

fighter developing severe obstructive lung disease after being trapped in a fire. The risk of developing severe pulmonary diseases during fire-fighting underlines the necessity for always wearing sufficient respiratory protection equipment in any situation when exposure to smoke may occur.

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RÉSUMÉ: Description du cas d'un pompier chez qui un asthme chronique et sévère s'est développé après exposition à des produits de décomposition des matières plastiques au cours de ses activités professionnelles de routine. L'évolution fut fatale, 25 mois après le début de la maladie.