

CASE STUDY

Reactive airways dysfunction syndrome following exposure to a fluorocarbon

R.E. de la Hoz

Reactive airways dysfunction syndrome following exposure to a fluorocarbon. R.E. de la Hoz. ©ERS Journals Ltd 1999.

ABSTRACT: This report describes the case of a 43-yr-old male who developed reactive airways dysfunction syndrome after exposure to a high level of bromotrifluoromethane (CF₃Br, Halon 1301), a fluorocarbon widely used in automatic fire extinguishing systems. The patient was a previously healthy, nonatopic male, who developed wheezing and intermittent and reversible obstructive ventilatory impairment starting immediately after a large accidental nonfire-related release of CF₃Br in a confined space.

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New York University School of Medicine and Bellevue Hospital Center, Chest Service and Occupational Medicine Clinic, New York, NY, USA.

Correspondence: R.E. de la Hoz, New York University School of Medicine, 550 First Avenue, Room MSB-C90D, New York, NY 10016, USA, Fax: 212 2631488

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The term reactive airways dysfunction syndrome (RADS) was first coined in 1985 [1] to describe a condition, clinically indistinguishable from asthma that develops in some individuals within 24 h after inhalation of a respiratory toxicant. The exposure is most characteristically of a high level and frequently a single episode. Patients with this condition have not had any prior history of respiratory disease, and complain of new onset of asthma symptoms after the exposure. The histopathological findings initially described in RADS based upon the scanty material obtained with bronchoscopic mucosal biopsies, were not very distinctive, and provide little justification for not using the term irritant-induced asthma to describe this condition [2].

Bromotrifluoromethane (CF₃Br, Halon 1301) is a colourless and odourless fluorocarbon widely used in automatic fire extinguishing systems. It is stored in liquid form, and becomes gaseous instantaneously upon release. As with other fluorocarbons, the acute toxicity of CF₃Br is usually considered to be related to central nervous system (CNS) dysfunction and cardiac sensitization to catecholamines with lowering of the threshold for arrhythmias [3].

Several lines of evidence (experimental animal studies [4, 5], controlled human exposure studies [6, 7], reports of large accidental exposures [8–10]) suggest that CF₃Br and other fluorocarbons can cause respiratory irritation. Despite this seldom recognized potential for adverse respiratory effects, no cases of asthma or RADS have been previously reported in relation to CF₃Br exposure.

This report describes the case of a 43-yr-old male who developed RADS after a sudden exposure to large amounts of CF₃Br in a confined space.

Case report

A 43-yr-old Caucasian male had developed symptoms of asthma after an episode in September 1990 (he was 36 yrs at the time). The patient was working in the telephone switch room at a local hospital. He was a technician, whose work consisted of repair and maintenance of computerized branch exchanges (computerized switches that regulate telephone communications). His duties consisted of running computerized diagnostic programs, and replacing faulty parts on computers as needed. No chemical handling or soldering was involved. The telephone switch was located in a large room at the basement of the hospital. Ventilation of the area (located in the basement of the building) depended on a central ducted system. On the day of the episode, he was the only person working in the area, except for two technicians who were repairing the CF₃Br-based automatic fire extinguishing system. They had left the room when one of the tanks of that system suddenly leaked greyish fumes from the ceiling immediately above the patient. The patient described a "rusty" smell. He immediately experienced eye irritation and shortness of breath. He had difficulty in exiting the room. From detailed documented descriptions of the latter, the exposure duration was estimated to have been ~10–15 min and to an initial concentration of at least 15%. When he finally left the room, he was already noticing increasing chest tightness, dizziness and lightheadedness. He was immediately taken to the emergency room of the same hospital. The medical staff documented the presence of conjunctivitis, as well as tachypnoea (28 breaths·min⁻¹) and wheezing. No hypoxaemia on room air was reported. Approximately 4 h later he was released to his home on no medication.

During the next few days the patient experienced severe tiredness, persistent eye irritation, chest tightness and some cough. He was seen by his personal physician 3–4 days later, who prescribed an inhaled bronchodilator, terfenadine, and a vasoconstrictor. Within a few more days, the patient was subsequently evaluated by a company physician, who found a decreased forced expiratory volume in one second (FEV₁) (50% of predicted) and forced vital capacity (FVC) (70% pred). Two weeks after the episode, he was treated with oral prednisone (50 mg·day⁻¹ for 1 week), then salbutamol and triamcinolone by metered-dose inhalers (MDI). The patient experienced partial improvement of his symptoms of cough, shortness of breath at rest or on exertion, eye irritation, and fatigue. Five weeks after the episode an examining pulmonologist reported the presence of conjunctivitis, but no wheezing. Spirometry, however, showed obstructive impairment, with a clear response to an inhaled bronchodilator (FEV₁ increased by 13% from 3.79 L). Besides the addition of ipratropium *via* MDI, the same treatment was continued for the next 7 yrs. Three yrs after the episode the patient was promoted to a managerial position. He continues to experience episodes of shortness of breath and mostly nonproductive cough. He also has perennial noninfectious rhinoconjunctivitis. His symptoms seem to worsen with cold weather and high environmental humidity, strong smells (*e.g.* from paints), heavy traffic smog, and dust. The respiratory symptoms have no seasonal pattern, and respond to inhaled medications.

The patient had been a smoker of about half a pack of cigarettes daily for 10 yrs until 3 yrs before the exposure episode. He had no previous history of any lung disease before the episode. Before coming to the USA in 1985, the patient had worked as an electrical engineer in his home country (Egypt) and had no significant history of exposure to dusts or fumes. He did not have any known allergies, and this was confirmed by both skin prick allergy testing and serum radioallergen sorbent test (RAST) panel for common allergens.

Recent (August, 1997) pulmonary function tests revealed an FVC of 3.72 L (80% pred), slow vital capacity (SVC) of 4.7 L (101%) FEV₁ of 2.88 L (75%), FEV₁/FVC of 78%, and flow at 50% of VC (V'₅₀) of 2.95 L·s⁻¹ (61%). Airway resistance and conductance, and diffusion capacity were normal. After inhaled salbutamol, FEV₁ and FVC improved by 31 and 24%, respectively. A chest radiograph was normal. Blood tests revealed normal eosinophil count and immunoglobulin (Ig)E levels, and absence of RAST antibodies to a panel of 20 common allergens.

Discussion

Halon 1301 (bromotrifluoromethane, CF₃Br) is a fluorocarbon extensively used in automatic fire extinguishing systems. After those systems are triggered by a smoke detector, the gas immediately floods the area where the fire is taking place. In order to achieve an optimal air concentration of 4–7% of CF₃Br, confinement of the space is required. The mechanism for fire extinction is not precisely known, but may be related to removal of the active chemical species in flame chain reactions [3]. Although Halon 1301 reacts with combustion products, its almost complete lack of reactivity with computer parts makes it a very attractive choice for use in computer and communication equipment rooms.

As with other fluorocarbons, human toxicity of Halon 1301 has traditionally been related to facilitation of cardiac arrhythmias and to depression of the CNS. Halon 1301 can decompose into byproducts, primarily upon heating, but also upon reaction with hydrogen from water vapour. These decomposition products include hydrogen fluoride and hydrogen bromide, carbonyl-fluoride and carbonyl-bromide. They are all well-known respiratory irritants, capable of causing injuries at all levels of the respiratory tract. Controlled human exposure to concentrations not exceeding 7% have not documented evidence or complaints of adverse respiratory effects [11–13]. Investigators have noted, however, that the effects of high concentrations upon the initial release of the gas could not be predicted from those studies [12]. Studies in experimental animals have suggested that prolonged or higher level exposures, or exposure to pyrolysis products from fluorocarbons are associated with adverse health effects, including pneumonitis [4, 5]. Human exposure studies with fluorocarbons used as MDI propellants have documented acute obstructive effects (increased airway resistance [6], and decreased airway conductance [14] or respiratory flows at low lung volumes [7]). Controlled human exposure to hair sprays has been reported to cause respiratory obstruction [15, 16], but it is not clear that fluorocarbons were the specific cause of those effects.

Reports of large accidental releases of CF₃Br in confined environments documented the occurrence of conjunctival and/or upper respiratory irritation in exposed individuals [8–10]. MALO *et al.* [17] reported a case of asthma in a subject who was chronically exposed to a heated fluorocarbon at work. Although they could not establish a specific pathogenic mechanism, and the time course of the exposure and of the development of disease would be consistent with either an irritation or a sensitization mechanism, they favoured the latter possibility [17]. The present patient declined specific challenge tests. In view of his documented one-time high level exposure, lack of previous lung disease or atopy, irritant-induced disease seems the more likely scenario. It is unclear what the direct irritant was in this case, whether it was CF₃Br or a decomposition product that may have built up during the repair work on the tanks. Decomposition of CF₃Br, however, is unlikely except at high temperatures (over 800°C). Given the widespread use of this agent in fire extinguishing systems, it is important to realize its potential to cause a spectrum of irritative symptoms and disease, of which asthma may be a long-term outcome, as this case illustrates.

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