CASE REPORT

Acute respiratory symptoms following massive carbon black exposure

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Background
Chronic carbon black exposure in the work environment can cause both respiratory symptoms and changes in lung function. There is limited information on the respiratory effects of acute exposure to carbon black.

Methods
Case report and literature review.

Results
A 44-year-old man had intense exposure to carbon black when his crane ran into a truck with a trailer filled with carbon black. One week after this exposure he developed shortness of breath and cough with sputum production. These symptoms persisted and increased in intensity. Physical examination revealed expiratory wheezes when in the supine position. Pulmonary function tests revealed a mild obstructive ventilatory defect with a reduced FEV1/FVC ratio. The patient responded to treatment with fluticasone and salmeterol with a reduction in symptoms and improvement in his spirometry to a normal range.

Conclusions
Acute exposure to carbon black can cause respiratory symptoms and an obstructive ventilatory defect. This presentation suggests a small airway disease which improved over time with inhaled steroids and long-acting beta-agonists. Patients with intense carbon black exposure following industrial accidents will need frequent evaluation to manage any related respiratory tract injury.

Key words
Carbon black; chemical exposure; chest; cough; dyspnoea; industrial accident; lung disease; spirometry.

Introduction
Carbon black (CB) is one of the most commonly used nanomaterials, especially in the tyre industry [1]. Some studies have suggested that chronic low dose CB exposure in the work environment causes respiratory symptoms and changes in lung function [2–4]. Information on the effects of acute high-dose CB exposure is limited, and we describe a patient who developed pulmonary disease following an acute exposure to CB.

Case report
A 44-year-old man presented to the pulmonary clinic for evaluation of dyspnoea and cough in late January 2012. He had been in good health until December 2011. At that time he came over a hill at approximately 40 miles/h driving a crane and ran into a truck with a trailer filled with CB. His crane cut the trailer in half, and there was an immediate intense exposure to CB dust (Figure 1). The patient estimates that he inhaled CB for approximately 2 to 3 min. He noted some chest discomfort at that time but had no other immediate effects. Approximately 1 week later he noted shortness of breath and cough which steadily increased. During his clinic visit he reported shortness of breath when walking on flat ground, cough with yellow sputum production, wheezing and increased symptoms with second-hand smoke. The patient had no history of any chronic lung disease and had not smoked since age 18. He did not think that any bystanders developed respiratory symptoms.

The patient presented as a well-nourished man in no acute distress. Vital signs were normal, with O2 saturation of 95% in room air. Thoracic examination revealed normal expansion on inspiration, normal expiratory flow rates and normal percussion notes. Breath sounds were...
slightly increased in intensity. There were high-pitched expiratory wheezes in the anterior fields when supine but not when sitting. Cardiac examination was within normal limits.

Complete blood count and chest x-ray were within normal limits. Pulmonary function tests revealed FVC 5.5 l (96% of predicted), FEV₁ 3.70 l (82%), FEV₁/FVC 0.67 (predicted 0.79), FEF₂₅₋₇₅% 2.44 l/s (60%), total lung capacity 7.92 l (105%), residual volume 2.67 l (128%), RV/TLC 0.34 and a normal diffusion capacity (106%).

The patient was started on fluticasone (500 micrograms per puff)/salmeterol (50 micrograms per puff) one puff twice daily. After 6 weeks his symptoms improved but had not resolved. He had no wheezing. His spirometry was within normal limits with FEV₁ of 4.91 l (122% predicted) and FVC of 6.07 l (110% predicted).

Discussion

CB is a chemical product composed of elemental carbon that is produced by incomplete combustion and vapour-phase pyrolysis of hydrocarbons under controlled conditions. It is a black, extremely fluffy, fine powder and is mostly used in rubber applications [1]. CB particles have a large surface area with aggregate dimensions ranging from ten to a few hundred nanometers [1]. Particles this size can easily enter the lower respiratory tract, and several studies have suggested that there is a relationship between CB exposure and respiratory symptoms and impaired pulmonary function [2–4]. Neghab et al. recently completed a cross-sectional study on 72 male rubber workers with a past history of and current exposure to CB and 72 healthy unexposed office workers [3]. They reported that exposure to CB exceeding its current threshold limit value was associated with a significant increase in respiratory symptoms, including cough, sputum production, wheezing and dyspnoea and with decreases in both FVC and FEV₁ in exposed workers. Harber [4] also found that cumulative exposure to CB caused small reductions in the FEV₁ but not in other spirometric parameters. These studies evaluated the effect of the chronic exposure to low-dose CB in the work environment. In contrast, our case had a single intense exposure to CB. He had an FEV₁ at the lower limit of normal and a reduced FEV₁/FVC ratio consistent with an obstructive defect. He improved with inhaled steroids and a long acting beta-agonist, and his spirometry returned to normal. This presentation might suggest reactive airway dysfunction syndrome, but symptoms in this syndrome typically develop within hours after the exposure and usually involve exposure to fumes. Workers at the 9/11 World Trade Center collapse had exposure to multiple particulates and fumes, and some developed bronchial hyper-reactivity and chronic airway disease [5]. Pathological studies in these workers have revealed sarcoid-like granulomatous disease, eosinophilic pneumonia and obliterative bronchiolitis. Our patient could also have had bronchiolitis with delayed onset. This is a rare disease reported in patients exposed to some gases. King [6] reported constrictive bronchiolitis diagnosed by lung biopsy in soldiers returning from Iraq and Afghanistan who had exposures to smoke in a sulphur mine fire, dust storms and incinerated waste. Other studies have also reported delayed bronchiolitis following exposure to acetic acid steam and mustard gas.

CB can cause acute pulmonary inflammation in animal models. Li [7] instilled fine CB (260 nm diameter) and ultrafine CB (14 nm diameter) intra-tracheally into rats and found that bronchoalveolar lavage fluid from these animals contained neutrophils, lactate dehydrogenase, and increased concentration of protein. The neutrophil influx persisted at least 7 days. Vesterdal [8] challenged mice with CB nanoparticles using intratracheal instillation and found that monocyte chemoattractant protein-1 increased 24h after instillation only in mice receiving higher doses (in the range of 0.9–2.7 mg/kg). Stoeger [9] also reported a dose-dependent inflammatory response by measuring the number of neutrophils, interleukin 1 β and macrophage inflammatory protein in lavage fluid in mice 24 h after exposure to ultrafine CB. Barlow reported that type II epithelial cell lines exposed to nanoparticle CB released macrophage chemotaxins in vitro [10]. These
chemotaxins could initiate inflammatory responses after in vivo exposures that lead to bronchial disease.

In conclusion, our case demonstrates that an acute exposure to a high dose of CB can cause respiratory symptoms and changes in pulmonary function and that both symptoms and spirometry improved with treatment. One case does not necessarily establish causation but is potentially relevant to workers with similar exposures in industrial accidents.

Key points
• Acute intense carbon black exposure can cause respiratory symptoms and an obstructive ventilatory defect.
• These symptoms and changes in pulmonary function are probably explained by small airway disease.
• Respiratory symptoms following acute carbon black exposure responded to inhaled steroids and beta agonists in this patient.

Conflicts of interest
None declared.

References