Case Report

Cement dust exposure-related emphysema in a construction worker

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ABSTRACT

Although, smoking is considered the most important predisposing factor in development of emphysema; environmental exposures also play an important role. There have been several studies on work related respiratory symptoms and ventilatory disorders among employees of cement industry. We report a case of cement exposure related emphysema in 75 years old woman construction worker.

KEY WORDS: Cement, construction material, COPD, risk

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INTRODUCTION

Respiratory disorders related to inhalation of airborne dust are the most important group of occupational diseases. Workplace exposures are strongly associated with an increased risk of chronic obstructive pulmonary disease (COPD).[1] Chronic bronchitis often associated with emphysema has been reported as the most frequent respiratory disease, with cement exposure followed by silicosis and mixed dust fibrosis.[2] There are very few studies assessing the actual cement dust exposure and related risk in construction workers.

CASE REPORT

A 75-year-old woman—nonsmoker, and construction laborer involved in mixing concrete and tiling—had exposure to cement dust for 30 years. There was no history of exposure to biomass fuel or passive smoking. She had been using gas stove for cooking for approximately 1-2 hours daily. She presented with a history of cough with expectoration and breathlessness on exertion since 20 years. This condition was Medical Research Council (MRC) grade 1 to begin with, and gradually progressed to MRC grade 4 at the time of presentation. She had repeated hospitalizations for type II respiratory failure. There was no history of any systemic disease. On examination, she was tachypnoeic and had bilateral pedal edema and elevated jugular venous pressure. There were changes of hyperinflation and reduced breath sounds with rhonchi. Pulse oximetry showed oxygen saturation of 80% in room air. Arterial blood gas analysis was 7.32/77/41/38.5/73, suggesting type 2 respiratory failure. Chest X-ray [Figure 1] showed low flat diaphragms, hyperinflation, and cardiomegaly, with dilated pulmonary arteries. Biochemical investigations were normal including alpha-1 antitrypsin levels (226 mg/dl). Management with controlled oxygen therapy, inhaled corticosteroids, and bronchodilators along with noninvasive ventilation stabilized her condition. She was further investigated in a stable state. On two-dimensional echocardiography, mean pulmonary artery pressure (PAP) was 77 mm Hg, and there was dilatation of the right ventricle and right atrium, with good left ventricular function and no valvular or septal defects detected. High-resolution computed tomography (HRCT) of the thorax [Figure 2] showed changes in centrilobular emphysema, with diffuse air trapping. Spirometry showed forced vital capacity (FVC - 1.36 L) (60% predicted), forced expiratory volume in 1 second (FEV_1 - 0.62 L) (32% predicted), and FEV_1/FVC ratio at 45%, suggesting severe obstructive abnormality with poor post-bronchodilator reversibility. A diagnosis of COPD with right heart failure was made.

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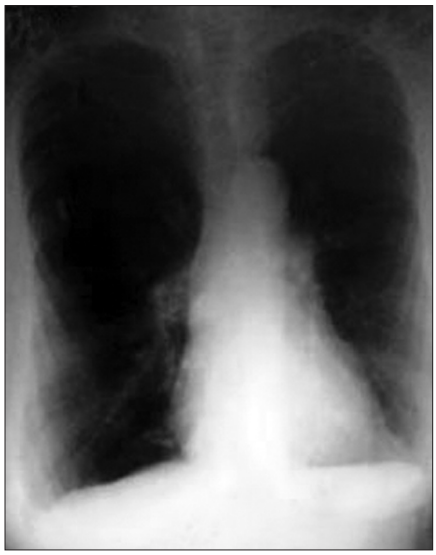

Figure 1: X-ray chest, posteroanterior (PA) view

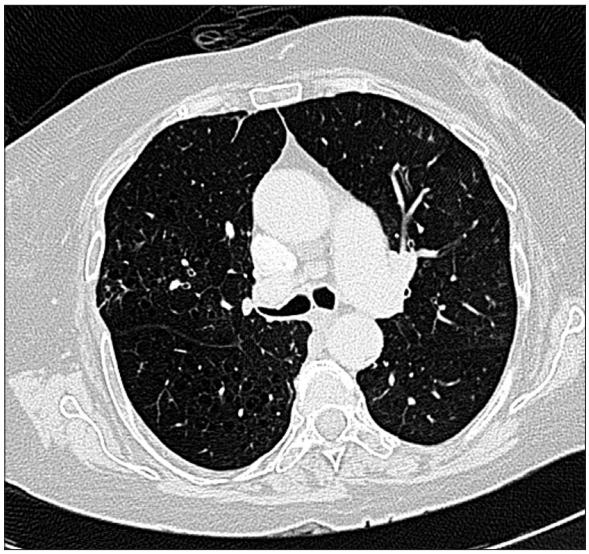

Figure 2: High-resolution computed tomography of the thorax

DISCUSSION

COPD is a leading cause of morbidity and mortality worldwide, with smoking being the most important predisposing factor, followed by environmental exposure. Data from the FLOW study of 1,202 subjects with COPD suggest that workplace exposures are strongly associated with an increased risk of COPD.[3] Workers at increased risk for chronic obstructive lung diseases include coal miners, construction workers handling cement, metal workers exposed to heat from furnaces, transport workers, grain handlers, cotton workers, and workers in paper mills. Unfortunately, the impact of smoking as a risk factor for COPD was perceived as so overwhelming that the interest in other risk factors faded away. Recognition of work-relatedness for airway diseases is difficult since these are multifactorial diseases that are strongly associated with nonoccupational exposures. American Thoracic Society conducted a systematic epidemiologic review and concluded that approximately 15% of COPD cases may be attributable to workplace exposures.[4] A recent study by Dement *et al.* showed statistically significant associations for COPD, with exposures to asbestos, silica, welding, cement dusts, and some tasks associated with exposures to paints, solvents, and removal of paints.[5]

Portland cement commonly known as cement is a fine, grayish green powder with an aerodynamic diameter in the range of 0.05-5 μm, which is produced by heating ground cement rock or other limestone-bearing materials into a fused clinker, which is subsequently ground into a fine powder. Various studies have shown the evidence of COPD in cement factory employees, few with conflicting results.[6-9] Increased mortality has been noted from COPD among construction workers exposed to organic dust as compared to unexposed construction workers.[10] However, the exact mechanism by which it affects the lungs is unknown. It is yet to be determined whether these effects are directly due to cement dust or mediated by a metabolic product of cement dust.

Cement dust particles can cause a disease due to the chemical nature of cement dust and its irritant, sensitizing and pneumoconiotic properties. The main routes of entry of cement dust particles into the body are the respiratory and gastrointestinal tracts. The deposition of inhaled particles is influenced by the physical and chemical properties of the inhaled agent and also by various host factors. The physical properties of importance include particle size and density, shape and penetrability, surface area, electrostatic charge, and hygroscopicity. The deposition of inhaled material is primarily dependent on particle size in the forms of an aerodynamic diameter.[11] It has been suggested that the reduced FEV_1% in cement factory workers may be due to reflex bronchospasm triggered by inhaled irritant cement dust or as a result of type 1 hypersensitivity reaction. The severity of the impairment of respiratory function has been shown to depend on years of exposure.[12]

Animal studies reveal that cement dust induces atrophic and hypertrophic changes in the nasal and pharyngeal mucosa as well as chronic exfoliative bronchitis.[13] One of the studies suggests that cement dust exposure may decrease lung and thoracic compliance by impairing intercostal muscle performance.[14] A significantly higher percentage of neutrophils was observed in cement production workers during the exposed period compared with the non-exposed period.[15] Another study concluded that exposure to cement dust decreased the physiological activity of polymorphonuclear neutrophils in cement mill workers.[16] A study carried out in 2006-07 to assess the actual cement dust exposure among construction workers concluded that the highest level of inhalable dust is at the construction site observed for concrete repairs, floor screed layers, and tile setters; in comparison, these exposures were lower than those found in cement plants.[17]

Cement mortar for setting ceramic tiles is composed of Portland cement and sand. The joints between the tiles filled with a grout, which may be Portland cement base, epoxy base, furans, or latex. Tile grout is again a mixture of Portland cement, fillers, pigments, and additives, which when mixed with water is used to fill the gaps between the tiles on walls and floors. Our patient has used both white or black cement and masonry sand at her workplace. She was a nonsmoker, presented with a history of chronic airway obstruction after 30 years of cement exposure and had normal alpha-1 antitrypsin level. There was no exposure to biomass fuel or passive smoking, and hence emphysema is most likely cement exposure-related. Although there are many studies showing a relationship between cement exposure in industrial workers and respiratory disorders, no case of emphysema related to cement exposure working at a construction site has been reported earlier.

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