Acute Respiratory Failure from Cement Exposure: A Case Report and Review of the Literature

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ABSTRACT

Cement is widely used in construction. Acute exposures with immediate sequelae have been infrequently described. This case report describes a man who developed multifocal pneumonitis with acute respiratory distress syndrome (ARDs) and respiratory failure one day after cement dust exposure. Chromium, cobalt, and nickel components in cement may cause pulmonary tissue irritation. Sand and gravel in cement may cause direct abrasive injury. Inhalation may cause direct thermal injury through an exothermic reaction. The silicon dioxide component has been shown to cause pulmonary injury through cytokine-mediated inflammation. Cement batches for smaller-scale construction jobs are often mixed onsite increasing exposure risk. Implementation of personal protective equipment has been shown to reduce respiratory symptoms among cement workers, underscoring the need for occupational health standards and further research.

KEYWORDS: acute, respiratory, failure, cement, exposure

INTRODUCTION

Concrete is one of the main substances used in the modern construction process in developed countries. Concrete is a composite material composed of coarse gravel, crushed granite or limestone, and sand (collectively called the aggregate) bonded together with cement and water. Portland cement is the most widespread form of cement.[1] It is a mixture made primarily of calcium oxide and silicon dioxide with smaller amounts of chromium, cobalt, and nickel.[2] When the aggregate is mixed with dry Portland cement and water, the mixture forms a slurry that can be easily molded into shape. The mixture chemically reacts with water to gradually harden over time.[1]

Concrete workers are exposed to concrete and cement dust and its potential toxicologic effects during the mixing, pouring, and cleaning processes.[3-5] Cement dust is irritating to the respiratory tract.[4] Long-term exposure to cement dust inhalation is correlated with increased prevalence of chronic cough and wheezing and lower peak expiratory flow rates, forced expiratory volume in 1 second (FEV1), and vital capacity.[6-9] Environmental exposure to cement dust may also be an independent risk factor for developing respiratory tract cancers.[10] There are limited published cases on the acute effects of cement dust inhalation. We present a case of multifocal pneumonitis induced by acute cement-dust inhalation which resulted in acute respiratory distress syndrome.

CASE REPORT

A 47-year-old male construction worker with a history of diabetes, hypertension, hyperlipidemia and cigarette smoking presented to the Emergency Department (ED) with shortness of breath that began the day prior. He reported that the day before he mixed concrete all day and was repeatedly enveloped in a cloud of concrete dust. Later at home he developed progressively worsening shortness of breath and associated chest pain with continued cough. He had no prior history of lung disease.

On arrival, his vital signs were: blood pressure, 145/86 mmHg; heart rate, 115/min; respiratory rate, 24/min; O_2 saturation of 84% on room air; temperature, 99.2° F. He was in mild respiratory distress with diffuse basilar rhonchi. He had no wheeze.

His oxygen saturation did not improve with oxygen via non-rebreather, and he was placed on bilevel positive pressure ventilation (BPPV) with improvement in his oxygen saturation to 97% and mild improvement in his work of breathing.

An electrocardiogram demonstrated sinus tachycardia. Initial basic metabolic panel, B-type natriuretic peptide (BNP), and troponin were normal. Lactate was 1.0 mEq/L. Complete blood count was notable for leukocytosis to 25 x10^9/L without any left shift. D-dimer was elevated at 423 ng/mL. His venous blood gas was pH 7.39, pCO2 45 mmHg, and pO2 < 30 mmHg with venous O_2 saturation of 49%. His HIV returned negative. His initial chest x-ray showed bilateral, multifocal airspace disease (**Figure 1**). A chest computed tomography angiogram (CTA) was read as "bilateral multifocal airspace disease with ground-glass opacities" (**Figure 2**). No pulmonary emboli were identified. The radiologic differential diagnosis included pneumonia, aspiration, pulmonary hemorrhage, and acute respiratory distress syndrome (ARDS).

The patient's respiratory distress worsened and he was intubated in the ED. Post-intubation arterial blood gas was

Figure 1. Chest X-rays: Anteroposterior (A) and lateral (B) views showing multifocal airspace disease.

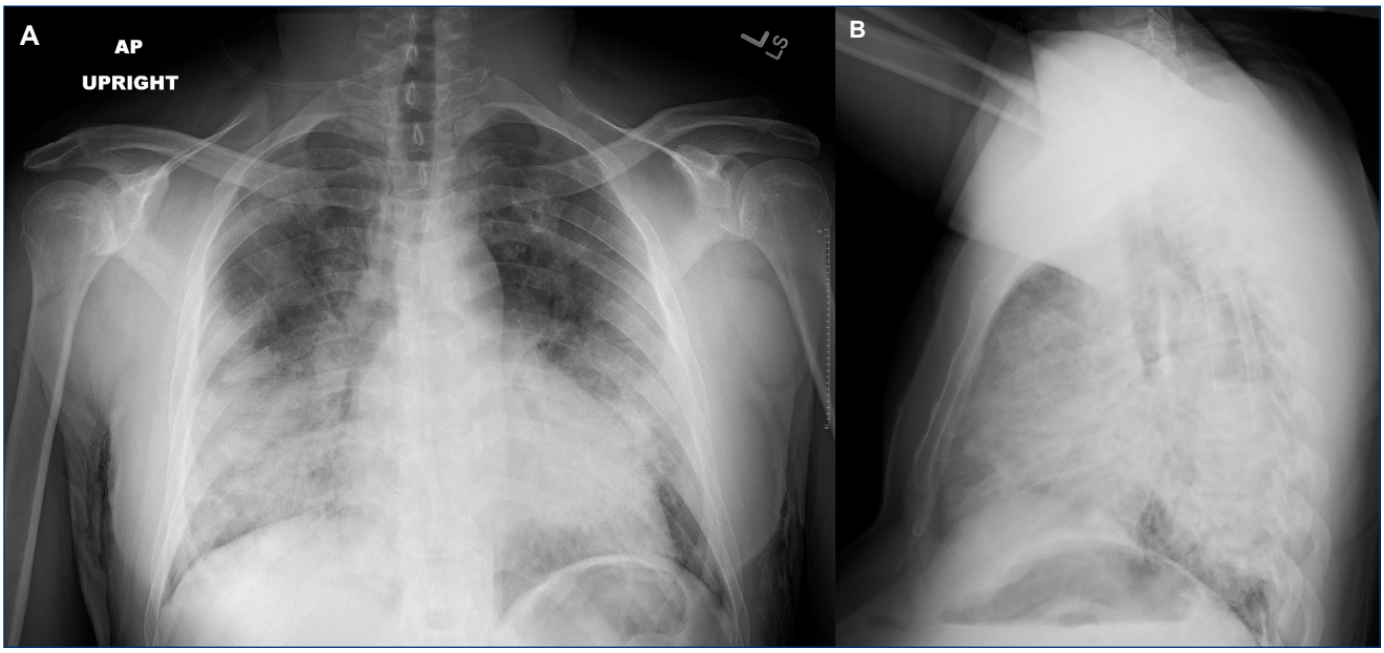
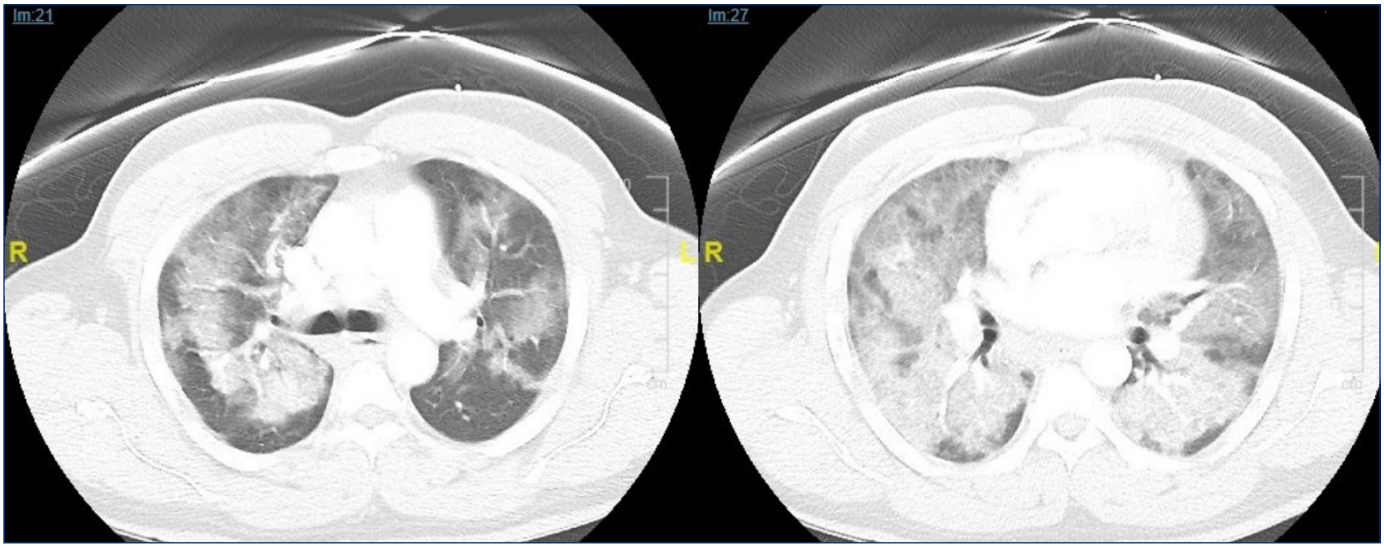


Figure 2. Chest computed tomography: Axial slices on lung windows demonstrating multifocal airspace disease with ground glass opacities.



pH 7.31, pCO2 48 mmHg, pO2 64 mmHg, O2 sat 90%. His PAO2/FiO2 was 160, meeting the definition for ARDS. He was admitted to the medical intensive care unit with ARDS secondary to multifocal pneumonitis versus pneumonia.

Although afebrile, the patient was empirically treated with antibiotics with ceftriaxone and azithromycin and with intravenous steroids. Blood cultures were negative. He was maintained on lung-protective ventilation with tidal volumes of 6 ml/kg of ideal body weight and gradually improved and was extubated on hospital day three. By hospital day six, the patient was on room air, and he was discharged on a prednisone taper with a discharge diagnosis of multifocal pneumonitis. On a follow-up visit approximately one month after discharge the patient continued to feel well and denied any ongoing shortness of breath or respiratory complaints.

DISCUSSION

The long-term respiratory effects of chronic exposure to concrete dust have been previously reported in multiple studies.[3-10] In addition to respiratory disease, topical exposure to concrete can cause severe second- to third-degree burns due to the severe alkalinity of cement mixed with water.[11] Although

not an occupational exposure, ingestion of cement can cause significant gastrointestinal injury through alkaline burns, an exothermic heat reaction, and solidification. Ingested cement should be emergently removed with endoscopy.[2]

This case describes the acute effects of cement-dust exposure in which a patient with no prior pulmonary disease developed multifocal pneumonitis with acute respiratory distress syndrome one day after exposure to cement dust. There are few existing reports on the acute respiratory effects of cement exposure. A study involving workers in a cement factory in Iran found that the most common acute respiratory symptoms among exposed workers were subjective shortness of breath and nasal congestion.[12] Cross-sectional studies in cement factories in Ethiopia[13] and in Tanzania[14] found that workers exposed to cement dust had an acute decrease in their peak expiratory flow (PEF). None of these prior studies reported any cases of acute respiratory failure or ARDS from cement exposure.

Given the paucity of reports on acute respiratory failure from cement inhalation, we can only theorize on the mechanism of lung injury. There may be an allergic or irritant component; cement exposure is known to cause allergic dermatitis and the chromium, cobalt, and nickel components are known irritants. Furthermore, the exact composition of metals can be variable across brands and individual batches, with some batches containing more irritants. The sand and gravel in concrete may cause direct abrasive injury.[2,4] When mixed with water, cement undergoes an exothermic reaction that may cause direct thermal injury to lung tissue.[2] Chromium can cause pulmonary irritation through local deposition of chromium salts.[15] Silicon dioxide is a component of Portland cement.[1,2] Inhaled silica can cause pulmonary toxicity through cytokine-mediated inflammation. Chronic exposure can cause progressive lung fibrosis.[16]

Most modern concrete is prefabricated at offsite facilities using machinery, thereby limiting exposures at this stage. Cement workers are exposed to cement dust primarily during the pouring and cleaning processes.[4] However, batches for small jobs are often mixed onsite, increasing exposure risk. Internationally, dust exposure to workers without sufficient personal protective equipment and workplace safety standards is a major concern.[10,12,14]

CONCLUSION

Implementation of proper personal protective equipment (PPE) has been shown to reduce the rates of chronic respiratory symptoms among cement workers, underscoring the need for dust control measures and occupational health standards.[17] Given the widespread use of cement and concrete in the industrialized world, additional research is needed to identify the acute and long-term health effects of cement dust exposure on concrete workers and to develop appropriate preventative countermeasures for safety.

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