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Brief Report

Acute Toxic Pneumonitis Complicating Chronic Obstructive Pulmonary Disease (COPD) in a Farmer

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Background *Toxic pneumonitis or Organic Dust Toxic Syndrome (ODTS) is a common, usually self-limiting condition among agricultural workers, occurring after high level exposure to agricultural dust.*
Case Report *We describe the case of a 68-year-old smoker and farmer who became ill with dyspnea, flu-like symptoms, and hypoxia after cleaning out a grain bin.*
Results *Chest radiograph was normal. He was treated with steroids, antibiotics, and oxygen supplementation and recovered over 3 months, but continues with lingering exercise intolerance after 2 years.*
Discussion *Although toxic pneumonitis usually follows a relatively benign course with spontaneous recovery and no permanent sequale it can lead to the exacerbation of underlying lung conditions, resulting in incomplete or delayed recovery. Prevention is aimed at limiting exposure by avoidance, by increasing ventilation in closed spaces and by appropriate use of personal protective equipment.* Am. J. Ind. Med. 46:393–395, 2004.
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KEY WORDS: toxic pneumonitis; Organic Dust Toxic Syndrome; agricultural dust; Chronic Obstructive Pulmonary Disease

INTRODUCTION

Toxic pneumonitis or Organic Dust Toxic Syndrome (ODTS), is a systemic response to inhalation of high concentrations of agricultural dust [Von Essen and Donham, 1997]. While the term ODTS has been most widely used in the US, the condition has also been called inhalation fever, atypical farmer's lung disease, and silo unloaders' disease. It was initially described by Emanuel et al. [1975] as pulmonary mycotoxicosis. Rylander and Malmberg [1992] proposed the term toxic alveolits or toxic pneumonitis for this condition, as there is evidence of pulmonary inflammation based on experimental studies in humans and animals. Patients typically present with history of flu-like symptoms including fever and dyspnea, 4–6 hr after exposure. The clinical course is generally self-limiting with complete recovery within 24–72 hr [Von Essen and Donham, 1997].

CASE REPORT

A 68-year-old male farmer and 30 pack year smoker developed dyspnea, fever, chills, myalgias, and malaise,

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4–5 hr after using a grain vacuum to unload a grain bin. During the unloading process, he remained within the semi-enclosed bin. He was previously in good health but reported a 2-year history of mild but progressive exertional dyspnea as well as several episodes of dyspnea and fever following work in dusty agricultural environments such as grain storage and swine confinement facilities. Because the symptoms related to dust exposure and always resolved spontaneously, he never sought medical attention, but sometimes used a two-strap dust mask to reduce the likelihood of discomfort. On the day of the incident exposure, he used the mask infrequently due to the hot working environment and increased work of breathing. When symptoms did not resolve one day after the exposure, he sought medical attention. On evaluation, he was found to be tachypneic, in mild respiratory distress with a low-grade fever (38.2°C). Physical exam was otherwise remarkable for diffuse wheezing and a prolonged expiratory phase on lung exam. A chest roentgenogram demonstrated pronounced right lower lobe bronchial markings (consistent with bronchiectasis), and hyperinflation but no other abnormalities. Laboratory evaluation revealed mildly elevated white blood cell count of 14.2×10^3 with a left shift on the differential. Arterial blood gas on 3 L oxygen showed hypoxia and combined respiratory acidosis and metabolic alkalosis (pH 7.45, PCO2 45, PO2 58). The patient was hospitalized and treated with supplemental oxygen, steroids, and antibiotics. He gradually improved although oxygen continued to be needed over the next 3 months. Spirometry showed a fixed airflow obstruction with FEV1 of 1.35 (34% predicted), FVC of 3.06 (60% predicted), and FEV1/FVC ratio of 44% with no significant response to bronchodilators. Lung volumes showed hyperinflation, while DLCO was well preserved at 82% predicted.

DISCUSSION

Toxic pneumonitis is perhaps the most common pulmonary response to inhaled agricultural dust; with an annual prevalence among American farmers of 20–30%, compared with a lifetime prevalence of 1–2% for hypersensitivity pneumonitis (HP) and 5–12% for occupational asthma [Von Essen and Donham, 1997; Kline and Hunninghake, 2001; Hoppin et al., 2002]. Endotoxin has been implicated as an important causative factor in toxic pneumonitis. Other potential agents that may act in concert with endotoxin include fungal spores, thermophilic actinomycetes, and peptidoglycans [Anonymous, 1998]. Toxic pneumonitis requires no previous exposure, whereas both occupational asthma and HP require previous sensitization and then may be expressed following exposure to low concentrations of antigens [Anonymous, 1998]. Hypoxia and PFT abnormalities are uncommon, but elevation of the alveolar-arterial gradient and transient airflow obstruction have been described in clinical exposure studies [Anonymous, 1998]. While the initial clinical presentation of toxic pneumonitis is similar to the acute presentation of HP, HP is generally characterized by interstitial infiltrates on the chest radiograph and abnormal (restrictive pattern) lung function testing. Examination of lung lavage in patients with toxic pneumonitis reveals predominantly neutrophils while lung lavage in HP patients shows a predominance of lymphocytes (>40%) with a high CD4/CD8 ratio [Kline and Hunninghake, 2001]. Pathological examination of biopsy specimens from patients with HP reveals noncaseating granulomas while biopsy specimens from patients with toxic pneumonitis demonstrate acute interstitial pneumonitis [Emanuel et al., 1975; Von Essen and Donham, 1997; Kline and Hunninghake, 2001]. Serum precipitins are positive in a large number of agricultural workers and are not of value in distinguishing between the two conditions.

The patient described in this report had apparent pre-existing, (though undiagnosed), Chronic Obstructive Pulmonary Disease (COPD). This case demonstrates that, in patients with underlying lung disease, toxic pneumonitis may present as a cause of exacerbation and that recovery may be prolonged and complicated. Preventive efforts to limit dust exposure are thus important such as increasing ventilation in enclosed spaces, increasing fat content in feeds and using automated equipment to avoid the need to enter dusty areas. Personal protective equipment should only be considered if engineering controls are not possible; minimal acceptable protective equipment includes two-strap disposable facemasks, quarter of half-face particle filtering respirators, and ventilation hoods [Donham et al., 1990]. Patients with chronic lung conditions frequently experience difficulty wearing air-filtering respirators because of underlying respiratory insufficiency. In this particular case, the use of a powered air-purifying respirator, such as the Air Stream helmet would have been appropriate and might have prevented this patient's illness.

In addition to smoking cessation, the patient was advised to employ engineering controls in his work environment, including frequent power washing of hog barns and increasing fat content in animal feeds. He was furthermore advised to avoid work in dusty areas and use a powered air-purifying respirator when entering such places. He has not had a recurrence of acute symptoms of toxic pneumonitis. However, he continues to have daily symptoms of chronic bronchitis, including productive morning cough and dyspnea on exertion.

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