Aluminum-associated Lung Disease: A Case Report

Nejdiye Mazıcan,1 Seher Kurtul,2 Deniz Öngel Harbiyeli3

1Department of Chest Diseases, Division of Occupational Diseases, Istanbul University-Cerrahpaşa Faculty of Medicine, Istanbul, Turkey
2Department of Occupational Diseases, Izmir Bozyaka Training and Research Hospital, University of Health Sciences, Izmir, Turkey
3Department of Chest Diseases, Istanbul University-Cerrahpaşa Faculty of Medicine, Istanbul, Turkey

ABSTRACT

Aluminum-associated fibrosis is a form of lung disease characterized by mixed-dust pneumoconiosis and interstitial pneumonia. Pneumoconiosis is a progressive disease resulting in non-nodular interstitial fibrosis located mainly in the upper and middle lobes of the lungs. It is primarily an occupational injury. This report describes the case of a patient who had worked in an aluminum sanding environment for 10 years and developed pneumoconiosis due to exposure to aluminum dust.

Keywords: Aluminum, fibrosis, pneumoconiosis

INTRODUCTION

The harmful effects of aluminum and its compounds on humans are well established. Aluminum that enters the body through respiration via the mouth or the skin can accumulate in tissues, such as the brain and lungs, though much is excreted by the kidneys. Aluminum exposure may lead to acute or chronic respiratory conditions, such as potroom asthma, chronic bronchitis, pulmonary fibrosis, granulomatous lung disease, acute tracheobronchitis, pneumonia, or pulmonary edema.[1] Aluminum-associated nonnodular fibrosis (also known as Shaver’s disease, aluminosis, or aluminum lung) is defined as mixed-dust pneumoconiosis accompanied by interstitial pneumonia caused by the inhalation of aluminum dust. Early reports by Shaver and Riddell recognized the role of aluminum particles in lung disease, including a 1947 study that investigated lung disease in plant workers involved in the production of corundum, an aluminum-based abrasive.[2] Although the pathogenesis of aluminum lung is not fully clear, like other pneumoconioses, it starts as alveolitis. Aluminum dust may remain in the lungs of exposed workers for years.[3] Aluminosis is characterized by diffuse interstitial fibrosis, mainly in the upper and middle lobes. In patients with prominent radiological findings, the most common symptom is dyspnea. Histologically, severe subpleural and interstitial fibrosis appear as scar emphysema and giant cell granulomatous pneumonitis.[1] Workers with aluminum exposure are often employed in smelting, preparing/processing aluminum powder, aluminum electric welding, grinding or polishing aluminum products, the use and manufacture of aluminum sanding tools, and the production of explosives, fireworks, glass, ceramics, and rubber.[1]

Increased occupational health and safety measures in workplaces have reduced the incidence of aluminosis, however it remains a potential problem.
This case report describes an instance of aluminosis caused by exposure to aluminum dust in an aluminum sanding worker who did not use personal protective equipment.

**CASE REPORT**

A 30-year-old male was admitted to the clinic with complaints of dyspnea, cough, intermittent sputum, and chest pain ongoing for 6 years. He had no history of smoking, chronic disease, or drug use, and he had no family history of any relevant disease. A physical examination was normal. The patient’s routine hemogram and biochemistry test results were within normal limits and his autoimmune serology tests were negative. Serum antinuclear antibody and anti-cyclic citrullinated peptide tests to investigate rheumatological pathologies were negative. The patient’s respiratory function test and carbon monoxide diffusion test results were forced expiratory volume in 1 second (FEV1)/forced vital capacity (FVC)= 80%, FEV1= 2.37 L (67%), FVC= 2.96 L (72%), diffusing capacity for carbon monoxide (DLCO)= 22.90 mL/mmHg/min (78%), and DLCO/veolar volume= 5.57 mL/mmHg/min/L (109%). The evaluation of the chest radiograph according to the International Labour Organization International Classification of Radiographs of Pneumoconioses was interpreted as 2/1 pq (Fig. 1). High-resolution computed tomography (HRCT) of the lungs revealed bilateral centrilobular nodular patterns in the apical and apicoposterior regions and ground-glass densities in the lower lobe superior segments, interlobular septal thickening, traction bronchiectasis, and pleuroparenchymal sequelae (Fig. 2). The occupational history of the patient revealed that he had previously worked at a metallic coating/polishing job for 10 years at a workplace that manufactured door handles and he was exposed to dust from aluminum sanding while working without personal protective equipment. For 6 years prior to presentation, the patient had been working at a packaging job that did not involve exposure to aluminum dust. Material safety data sheets of the chemical substances or personal ambient air aluminum exposure results of the previous workplace were not available, as it was a small business. The patient’s occupational history and clinical status suggested pneumoconiosis due to occupational aluminum dust exposure.

**DISCUSSION**

Aluminum-associated lung diseases are very now rare in occupational medicine. The first cases of lung disease due to industrial exposure to aluminum were reported in Germany in the 1930s. The impact of aluminum dust inhalation on the lungs was comprehensively studied in the 1940s, when Goralewski first used the term “aluminum lung”. He explored several pathophysiological mechanisms and found that other environmental factors and mixed exposures may also contribute to the etiology of the disease. In the 1940s, Shaver and Riddell also reported 35 cases of pulmonary fibrosis among 344 aluminum potroom workers in Canada who were exposed to aluminum dust during the manufacture of aluminum abrasives. The present case is a rare example of aluminosis due to prolonged exposure to aluminum dust in an aluminum sanding environment. Similar cases of aluminosis is due to exposure to the grinding and polishing of aluminum materials have been reported in the literature.
Particle manufacturing technologies involving aluminum and its compounds are essential. Pulmonary aluminosis is characterized by pneumoconiosis caused by the presence of aluminum dust in lung tissue, which may result in pulmonary fibrosis. It can be caused by exposure to aluminum and its compounds under a diverse range of occupational circumstances and factors. It has been noted that the inhalation of fine dust with respirable aluminum particles sized 0.5-5µm played a significant and threatening role in the disease's development. Last but not least, the level of exposure is a particularly significant factor, especially the constant presence of aluminum particles in the organism. Pulmonary diseases pose a risk in poorly ventilated workplaces within adequate personal protective equipment. In Turkey, the permissible exposure limit for the total quantity of aluminum dust in a workplace is 15 mg/m³ (time weighted average), and the quantity of respirable dust is 5 mg/m³. Routine engineering testing should be conducted in the workplace to eliminate or minimize exposure.

Aluminum oxide, silicon carbide, abrasive paper, garnet, and flint are used in sanding and polishing belts. Jederlinic et al. described workers involved in the production of grinding wheels and tools using aluminum oxide. Three of the 1000 workers examined in the study were diagnosed with severe histologically confirmed pulmonary fibrosis, and 6 other workers had abnormalities suggesting interstitial changes. According to the authors, this prevalence was 300 times greater than that of idiopathic pulmonary fibrosis. Although mixed-dust exposure could not be completely excluded, dust containing aluminum oxide was the most likely cause. In our case, the patient had been exposed to dust containing aluminum oxide while engaging in aluminum sanding for 10 years.

The initial clinical manifestations of pulmonary aluminosis include exertional dyspnea with a dry, non-productive cough as well as crepitation. Restrictive ventilatory defects are observed and sometimes accompanied by decreased diffusing capacity. Radiographical findings vary, revealing nodular or slightly irregular opacities which may assimilate into more prominent formations, mainly in the upper lungs. Consistent with the literature, our patient had a dry cough and dyspnea, HRCT imagery revealed ground-glass densities in the upper fields and interlobular septal thickening, and a restrictive pattern was observed in respiratory function tests. Since our patient had been working at a different job that did not involve exposure to aluminum dust for 6 years, aluminum levels were not detected in the blood or urine.

CONCLUSION

Pulmonary aluminosis, though very rare, still occurs. With the rising significance of the aluminum industry, lung damage caused by exposure to respirable aluminum particles should not be overlooked, but rather, monitored, and prevented. To protect the health of employees, a safe working environment and routine health inspections should be conducted. General engineering controls are fundamental. Recent studies recommend early detection of the disease using modern methods such as HRCT of the lungs. Measurement of aluminum concentration in the blood and urine of exposed workers may also prove valuable.

Disclosures

**Informed Consent:** Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

**Conflict of Interest:** None.

**Peer-review:** Externally peer-reviewed.

**Financial Disclosure:** None.


**REFERENCES**

8. Ueda M, Mizoi Y, Maki Z, Maeda R, Takada R. A case of alumi-


