Turk Thorac J 2021; 22(1): 83-5 DOI: 10.5152/TurkThoracJ.2021.19137

Case Report

A Current Example of Historical Cases: Occupational Pulmonary Aluminosis

Eliz Kuman Oyman[1], Elif Altundaş Hatman[2], Duygu Acar Karagül[1], Zeki Kılıçaslan[3]

[1]Occupational Health Training Programme, Department of Public Health, İstanbul University, İstanbul School of Medicine, İstanbul, Turkey
[2]İstanbul Yedikule Chest Diseases and Thoracic Surgery Training and Research Hospital, İstanbul, Turkey
[3]Department of Chest Diseases, İstanbul University, İstanbul School of Medicine, İstanbul, Turkey

Cite this article as: Kuman Oyman E, Altundaş Hatman E, Acar Karagül D, Kılıçaslan Z. A current example of historical cases: occupational pulmonary aluminosis. Turk Thorac J 2021; 22(1): 83-5.

Abstract

Pulmonary aluminosis (PA) is a rare form of pneumoconiosis caused by aluminum powders and vapors. Although the pathogenesis is not fully elucidated, it is thought to make a number of changes in the lungs, resulting in fibrosis. Our patient, who had cough, sputum, and dyspnea and had thorax computed tomography results showing reticular density changes and symmetrical ground-glass opacity in the bilateral upper and middle zones, informed us that he had worked in aluminum casting for 20 years and was exposed to iron, aluminum, and zinc vapors, and dust in the workplace. The patient was scheduled for bronchoscopy; aluminum analysis in bronchoalveolar lavage revealed 0.256 mg/kg of aluminum. The patient, with a history of occupational exposure, was diagnosed with aluminum metal fume-induced PA. This case shows that, even if it is preventable, PA can still occur if the occupational health and safety regulations are not met and also emphasizes the importance of the detailed occupational history in interstitial lung diseases.

KEYWORDS: Interstitial Lung Disease, occupational lung disease, pneumoconiosis, pulmonary aluminosis

Received: *November 13, 2019* ***Accepted:*** *April 19, 2020*

INTRODUCTION

Aluminum is a metal obtained from aluminum-containing minerals such as bauxite and is the third most common element in the earth's crust. It is used in many fields of industry, and workers may be exposed to aluminum during bauxite refining and aluminum casting and production of waterproofing materials, fireworks, ceramics, glass, building materials, and textile dyeing products [1-3]. Aluminum and its compounds enter the body as a result of inhalation, ingestion with food or water, or contact with the skin [1]. Aluminum is discarded via the kidneys, and a large portion of it stored in various tissues, including the bones and lungs [1, 4].

Pulmonary aluminosis (PA) is a rare form of pneumoconiosis caused by aluminum powders and vapors [4]. Although the pathogenesis is not fully elucidated, it is thought to make a number of changes in the lungs, resulting in fibrosis [1, 4]. Like other pneumoconiosis, PA has no treatment [4].

CASE PRESENTATION

A 44-year-old male patient referred to the hospital with cough, sputum, and dyspnea ongoing for 3-4 months. He used to smoke 6 pack-years and had quit it 21 years ago. He was treated for pulmonary tuberculosis in 1997. He did not have any other chronic disease and did indulge in regular drug use. He was conscious, cooperative, and oriented, and his vital signs were stable. During the respiratory system examination, auscultation revealed bilateral rales in the basals, and other system examinations were normal. C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) were 18.4 mg/L (0-0.5) and 40 mm/h (0-10), respectively. Hepatitis markers were negative. Posteroanterior chest X-ray showed bilateral upper-middle zone predominant reticulonodular pattern, right diaphragm elevation, left hilar fullness, and distortion (Figure 1). Thorax computed tomography (CT) revealed calcified pathological size lymph nodes in the mediastinal, precarinal (15x11 mm), and infracarinal (12x10 mm) areas, and bilateral hilar regions (Figure 2). In the parenchyma, bilateral upper and middle zone reticular density changes, symmetrical ground-glass opacity, calcific pulmonary nodules with nonspecific millimetric dimensions, and fibrotic changes were observed (Figure 3). The test for sputum acid resistant bacterium (ARB) was negative. Pathogenic microorganisms did not grow in sputum gram staining and culture. Pulmonary function tests (PFT) revealed the following: forced expiratory volume in the first second (FEV1): 1.79 l (50%), forced vital capacity (FVC): 2.21 l (51%), FEV1/FVC: 81, and diffusion capacity of lung for carbon monoxide (DLCO) 57%. Collagen markers were negative. The patient was given nonspecific antibiotherapy treatment because of elevated ESR and CRP but no radiological regression after treatment.

Address for Correspondence: Eliz Kuman Oyman, Occupational Health Training Programme, Department of Public Health, İstanbul University, İstanbul School of Medicine, İstanbul, Turkey
E-mail: elizoyman@istanbul.edu.tr

When the anamnesis deepened, we learnt that he had applied to another health institution 3 months earlier, and bronchoscopy had been performed after imaging tests; yet no diagnostic results were obtained. He stated that he had worked in different aluminum foundries for 20 years and was exposed to iron, aluminum, and zinc vapors and dust in the workplace. There was no ventilation system in the workplace. He occasionally used a simple surgical mask. Bronchoscopy showed normal endobronchial system. Macrophages 85%, lymphocytes 10%, neutrophils 5%, and CD4/CD8 1.79 were detected in bronchoalveolar lavage (BAL). BAL-ARB was negative, and no pathological cells were observed. Aluminum analysis in BAL revealed 0.256 mg/kg of aluminum. The patient with a history of occupational exposure was diagnosed with aluminum metal fume-induced PA. He was started with methylprednisolone 32 mg/day peroral to slow the development of pulmonary fibrosis. Treatment was completed by reducing the dose after 9 months. There was no clinical, radiological, or functional improvement after the treatment. Symptomatic treatment was arranged and followed up. The patient's informed consent was obtained for the publication of this case report.

DISCUSSION

The first cases of aluminum-induced interstitial disease in the lungs were published about 90 years ago [5]. In 1939, Goralewski studied the damage to the lungs caused by the inhalation of aluminum-containing powders, used the term "aluminum lung" for the first time, and mentioned that exposure could cause serious progressive lung diseases [6]. Although PA cases are not frequently encountered in developed countries, they continue to be seen in small enterprises, which have adverse working conditions and where workers are exposed to high amounts of aluminum as in this case [7].

PA is a condition that does not have a specific clinical picture, usually occurring years after exposure to aluminum powders or vapors. Patients may present with any respiratory complaint such as dyspnea, cough, and/or wheezing [8]. In general, PFT results were normal in patients exposed to aluminum [1]. In this case, restrictive patterns and a decrease in DLCO tests were observed in PFT, consistent with interstitial lung disease.

The role of CT in the radiological evaluation of occupational lung diseases is increasing [9]. PA usually presents with nodular or irregular opacities, predominantly of the upper

MAIN POINTS

- Pulmonary aluminosis (PA) is a rare form of pneumoconiosis caused by aluminum powders and vapors.
- Although the pathogenesis is not fully elucidated, it is thought to make a number of changes in the lungs, resulting in fibrosis.
- Although PA is preventable, it still occurs because of adverse working conditions.
- Detailed questioning about occupational exposure in patients with pulmonary parenchymal disease is essential in diagnosing occupational diseases and clarifying the etiology.

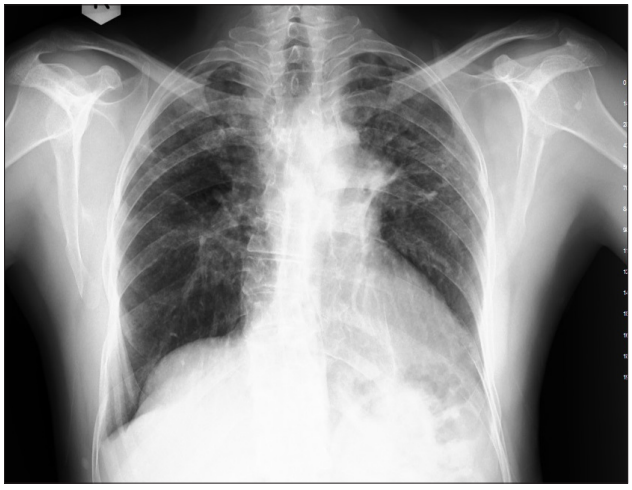

Figure 1. Posteroanterior chest X-ray showing bilateral upper-middle zone predominant reticulonodular pattern

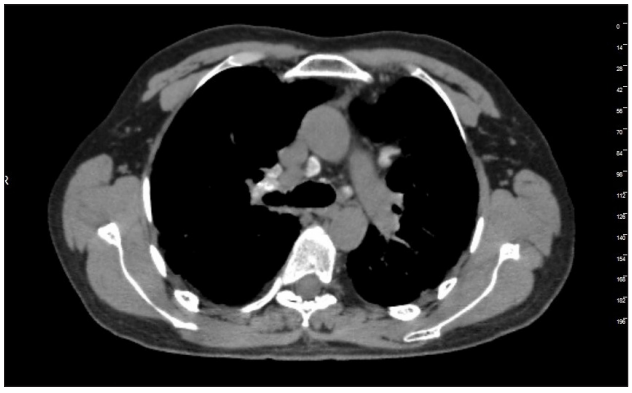

Figure 2. Thorax computed tomography (mediastinal window) reveals lymph nodes with calcified-pathological size

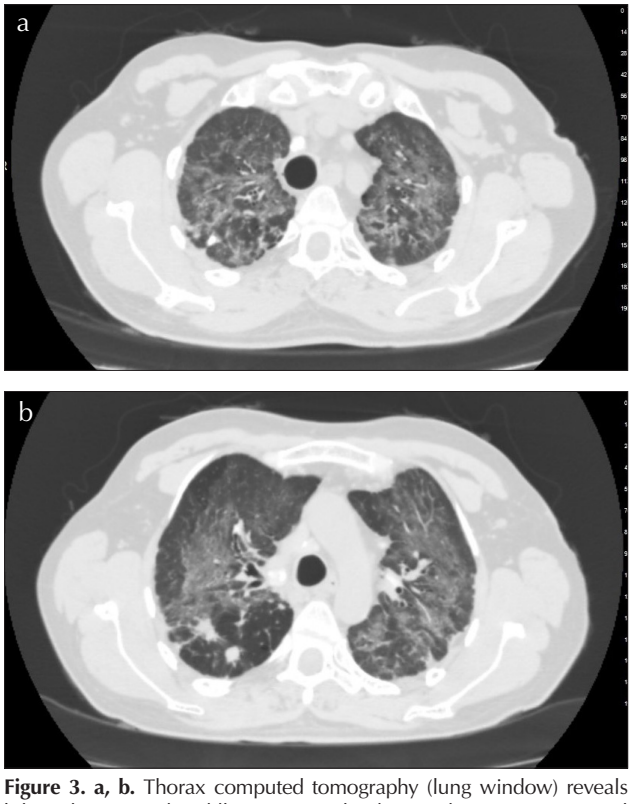

Figure 3. a, b. Thorax computed tomography (lung window) reveals bilateral upper and middle zone reticular density changes, symmetrical ground-glass opacity, calcific pulmonary nodules and fibrotic changes

zone, leading to fibrotic changes. In advanced disease, severe pulmonary fibrosis with honeycomb appearance may be seen [10]. A case of aluminosis diagnosed with pleural effusion and mediastinal lymphadenopathy has also been reported in recent years [11]. In a study evaluating radiological findings in patients with occupational aluminum exposure, CT showed reticular or nodular predominant fibrosis in the upper lung zones in 5 of the 6 patients [12]. In this case, reticular changes and symmetrical ground-glass opacities spreading to the upper and middle zones were observed, and the findings were thought to be because of aluminum exposure as they are not limited to the upper lobes.

Although there is no medical treatment option for PA, there have been cases in which antifibrotic or immunosuppressive therapy was used to slow the progression of fibrosis [4, 7]. In this case, methylprednisolone treatment did not have any effect on clinical, radiological, or functional parameters.

It can be thought that the first PA cases were because of the high amount of aluminum exposure in the working environment; in the following years, there have been fewer cases as a result of the progress made in the field of occupational health and safety. In conclusion, although PA is preventable, it still occurs because of adverse working conditions. Etiologic diagnosis with detailed questioning about occupational exposure in patients with pulmonary parenchymal disease can at least help in the diagnosis of occupational diseases and help provide compensation to the patient and ensure that protective and preventative measures are taken or made more efficient for workers who have not presented with clinical findings yet.

Informed Consent: Informed consent was obtained from the patient.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - E.K.O., E.A.H., Z.K.; Design - E.K.O., E.A.H., Z.K., D.A.K.; Supervision - E.K.O., E.A.H., Z.K.; Resources - E.K.O., E.A.H., D.A.K.; Materials - E.K.O., E.A.H., Z.K., D.A.K; Data Collection and/or Processing - E.K.O., E.A.H., Z.K., D.A.K.; Analysis and/or Interpretation - E.K.O., E.A.H., D.A.K.; Literature Review - E.K.O., E.A.H., D.A.K; Writing- E.K.O., E.A.H., Z.K., D.A.K; Critical Review - E.K.O., E.A.H., Z.K., D.A.K.

Conflict of Interest: The authors have no conflicts of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

REFERENCES

1. Ferguson G, Hopkins R, Poortinga K, McCallum L. Systematic Review of Occupational Aluminum Exposure and Adverse Health Conditions. Revised Final Report. March 2018
2. Krewski D, Yokel RA, Nieboer E, et al. Human health risk assessment for aluminium, aluminium oxide, and aluminium hydroxide. J Toxicol Environ Health B Crit Rev 2007;10 Suppl 1(Suppl 1):1-269. [Crossref]
3. Ansari HA, Al-Bahrani G, Vishwanat M, et al. Thoracic scintigraphy in aluminosis: lymph node uptake on bone scan. Clin Nucl Med 2009;34:24-6. [Crossref]
4. Smolkova P, Nakladalova M, Tichy T, et al. Occupational pulmonary aluminosis: A case report. Ind Health 2014;52:147-51. [Crossref]
5. Doese M. Industrial Medicine Studies on Injury to Health Caused by Aluminium, in Particular Lung Disease From Aluminium Dust. Arch Gewerbepathol 1938, 8:501-31. [Crossref]
6. Goralewski G. The aluminium lung: A new industrial disease. Br J Ind Med 1948;6:53-4.
7. Yabuuchi Y, Goto H, Nonaka M, et al. A case of airway aluminosis with likely secondary pleuroparenchymal fibroelastosis. Multidiscip Respir Med 2019;14:15. [Crossref]
8. Wesdock JC, Arnold IMF. Occupational and environmental health in the aluminum industry: key points for health practitioners. J Occup Environ Med 2014;56(5 Suppl):S5-11. [Crossref]
9. Kraus T, Schaller KS, Angerer J, et al. Aluminosis - Detection of an almost forgotten disease with HRCT. J Occup Med Toxicol 2006;1:4. [Crossref]
10. Smolkova P, Nakladalova M. The etiology of occupational pulmonary aluminosis - the past and the present. Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub 2014;158:535-8. [Crossref]
11. Vahlensieck M, Overlack A, Müller KM. Computed tomographic high-attenuation mediastinal lymph nodes after aluminum exposition. Eur Radiol 2000; 10:1945-46. [Crossref]
12. Akira M. Uncommon pneumoconioses: CT and Pathologic Findings. Radiology 1995; 197:403-409. doi: 10.1148/radiology.197.2.7480684. [Crossref]