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Case Report

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Alveolitis as a result of dust chlorine exposure

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ABSTRACT

Hypersensitivity pneumonia of the lung, also called "extrinsic allergic alveolitis" or "hypersensitivity pneumonitis", is a condition in which the lung tissue becomes inflamed for reasons other than microbial causes. There may be many different reasons. Among the most common reasons were bird feeding, agricultural works and air conditioners. There are acute, subacute or chronic (slowly progressing) forms. Acute and subacute forms may recur, while the chronic form progresses and causes permanent and irreversible damage such as fibrosis and emphysema. Bird proteins, mammalian proteins, fungi, bacterial proteins and small molecular weight chemicals are generally blamed for the formation of the disease. Sometimes the causative agent may not be identified. In this case, we aimed to present a patient who applied to our clinic due to complaints of sudden respiratory distress, rapid fatigue and cough as a result of dust chlorine exposure, and was diagnosed with alveolitis, hospitalized and followed up.

Keywords: Hypersensitivity pneumonia, dust chlorine exposure, alveolitis

INTRODUCTION

Hypersensitivity pneumonia of the lung, also called "extrinsic allergic alveolitis" or "hypersensitivity pneumonitis", is a condition in which the lung tissue becomes inflamed for reasons other than microbial causes. There may be many different reasons. Among the most common reasons were bird feeding, agricultural works and air conditioners. There are acute, subacute or chronic (slowly progressing) forms. Acute and subacute forms may recur, while the chronic form progresses and causes permanent and irreversible damage such as fibrosis and emphysema. Bird proteins, mammalian proteins, fungi, bacterial proteins and small molecular weight chemicals are generally blamed for the formation of the disease. Sometimes the causative agent may not be identified.

In this case, we aimed to present a patient who applied to our clinic due to complaints of sudden respiratory distress, rapid fatigue and cough as a result of dust chlorine exposure, and was diagnosed with alveolitis, hospitalized and followed up.

CASE

A 47-year-old male patient was admitted to our clinic with complaints of sudden respiratory distress, fatigue and cough. There was no known history of additional disease. He had a history of 16 pack-year cigarette smoking dating back to 10 years ago. He had no history of alcohol use. He had no history of tuberculosis or contact. He was working as a pool chemical worker in hotel services. The patient had been exposed to chlorine before, and the last time he had a history of heavy dust chlorine use. There was no history of chemical exposure other than chlorine. The lung tomography showed that he was in the upper lobes of both lungs. There were peribronchial focal ground-glass-shaped density increases. Septal thickenings were observed in the lower lobes. Diffuse central ground-glass-shaped density increases were observed in both lungs, and a peripheral mass-like consolidation area was observed in the anterior upper lobe of the right lung. The findings were compatible with alveolitis. On examination, breathing sounds, gross oxygen saturation, temperature was 36.5°C, blood samples were within normal limits. Pulmonary Function Test result was FEV1: 86% FVC: 81% FEV1/FVC: 84 (Figure 1).

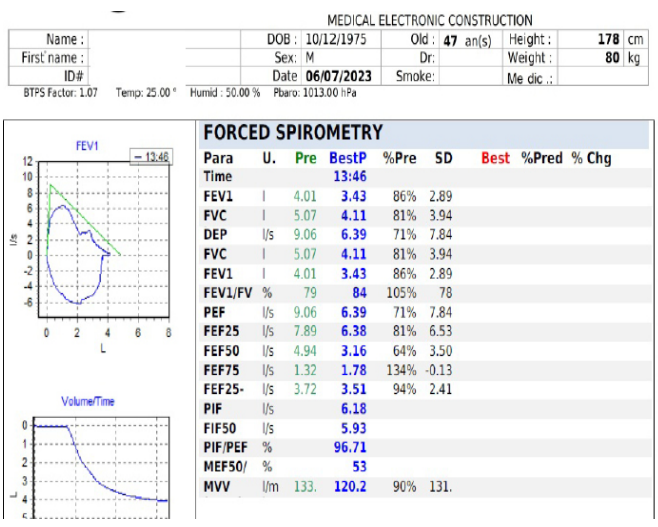

Figure 1. Pulmonary function test result.

During the hospitalization period, the patient was given 1x80 mg of prednol and bronchodilator treatment for 4 days. On the 5th day, 40 mg of prednol was given. Prednol 16 mg 20 tablets 1x1 was started for the next day and he was discharged. Tomography image comparisons at first arrival and 1 month later were seen at Figure 2 and Figure 3.

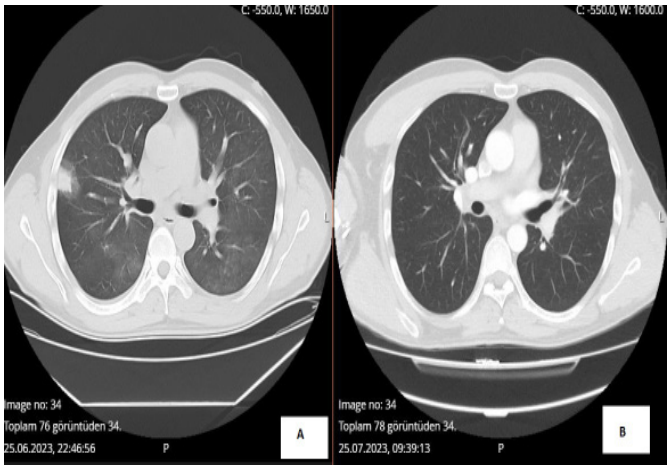

Figure 2. A: Peripheral mass in the anterior upper lobe of the right lung. Scattered consolidation area fibroatelectatic changes in both lungs. B: After using corticosteroids for one month, a considerable response was seen

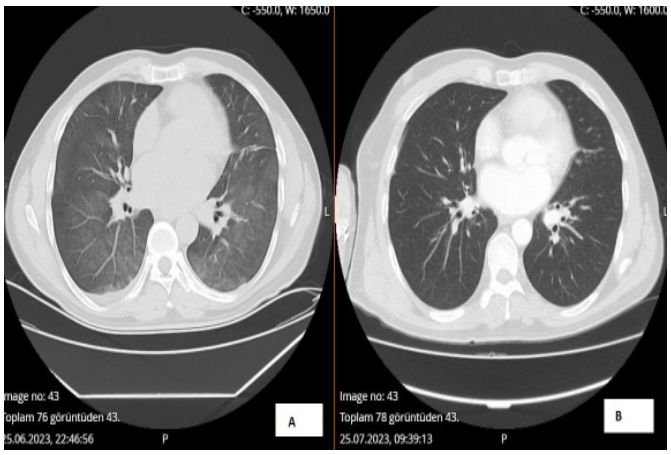

Figure 3. A: Widespread central ground glass pattern in both lungs. B: After using corticosteroids for one month, a considerable response was seen

DISCUSSION

The annual prevalence of hypersensitivity pneumonia is 1.6-2.7/100000. Its incidence varies according to age, profession and geographical region. It is seen at a rate of 1.3-12.9% in farmers, 3.7-10.4% in bird breeders, and 3.5-29% in mushroom workers. In our country, it has been detected in the third place after IPF and interstitial lung diseases due to collagen tissue diseases. Bird proteins, mammalian proteins, fungi, bacterial proteins and small molecular weight chemicals are generally blamed for the formation of the disease. Sometimes, the causative agent may not be identified. HP is seen in 80-95% of non-smokers.[1]

Chlorine is a green-yellow gas, heavier than air, with a characteristic odor. It is used in industry to make alkali and bleach, as a disinfectant, and as a whitening agent in the paper and textile industry. Although its irritating effects on the lungs occur throughout the entire airway due to its moderate water solubility, it is especially evident in the bronchioles and alveoli.[2-4]

Exposure to chlorine gas often occurs in the home environment, either from mixing household cleaning agents or, as in our case, during pool or spa maintenance. In a retrospective study on chlorine poisonings in a poison center, it was determined that 73% of the admissions were due to mixing cleaning products containing acid and hypochlorite bleach, 14% were related to swimming pools, and 7% were related to industrial exposure.[5] If the exposure time is long and ventilation is insufficient, the patient complains of eye and respiratory system irritation. In mild exposures, the main symptoms are nasal irritation, conjunctivitis, dry throat, cough and mild shortness of breath. In more severe exposures, as in our case, symptoms such as obvious shortness of breath, headache, cough, white-pink sputum production, chest pain, and vomiting are added. Since the odor threshold value is above the threshold value of respiratory irritation, the absence of odor does not indicate the absence of exposure. On physical examination, there is no exposure in the lungs. rales and rhonchi may be heard; In cases of heavy exposure, it may result in noncardiogenic pulmonary edema.[6,7] In the treatment of these patients, they generally respond to eye irrigation, oxygen, cough suppressants, bronchodilators, and, in cases where airway obstruction does not improve, steroid administration, as in our case.[7,8] There are studies reporting that sodium bicarbonate application by nebulization may be beneficial. Theoretically, sodium bicarbonate neutralizes the acid formed as a result of contact of chlorine with water.[5,7] It has been reported that permanent bronchial hyperreactivity and RADS develop after single exposure to high amounts of chlorine gas.[4]

RADS is a condition of bronchospasm that occurs within hours after a person who has no previous respiratory complaints is exposed to a respiratory irritant intensely.[9] It has been reported that permanent bronchial hyperreactivity and RADS develop after single exposure to high amounts of chlorine gas. The diagnosis of RADS is made if there is no previous respiratory complaint, the symptoms begin after a single accidental or incidental exposure, the respiratory irritant such as gas, smoke or vapor is in high concentration, the symptoms start within 24 hours after the exposure, and lasting at least 3 months, symptoms such as cough, shortness of breath, wheezing consistent with asthma, presence of nonspecific bronchial hyperresponsiveness, normal respiratory function tests or airway obstruction, and other respiratory symptoms It is diagnosed by ruling out diseases.[4,10,11] In our case, it was observed that he responded to steroid treatment and RADS did not develop.

CONCLUSION

As a result, although chlorine gas inhalation is frequently observed, it may cause mild effects on the respiratory tract, but may also cause the development of RADS in more severe exposures. The most important issue in treatment is to avoid exposure to the agent. Corticosteroids are indicated for the treatment of acute, subacute and chronic HP. Corticosteroids may also be useful in the treatment of severe or progressive chronic HP.

ETHICAL DECLARATIONS

Informed Consent: All patients signed and free and informed consent form

Reviewer Evaluation Process: Externally peer-reviewed.

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

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Author Contributions: All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Raghu G, Remy-Jardin M, Ryerson CJ, et al. Diagnosis of hypersensitivity pneumonitis in adults an official ATS/JRS/ALAT clinical practice guideline. *Am J Respir Crit Care Med*. 2020;202(3):e36-e69.
2. Blanc PD. Acute pulmonary responses to toxic exposures. In: Murray JF, Nadel JA, Mason RJ, et al, eds. Textbook of respiratory medicine. 3rd ed. Philadelphia, W.B. Saunders, 2000;1903-1914.
3. Schwartz DA, Blaski CA. Toxic inhalations. In: Fishman AP, Elias JA, Fishman JA, Grippi MA, Kaiser LR, Senior RM, eds. Fishman's pulmonary diseases and disorders. 3rd ed. McGraw-Hill: 1998:925-940.
4. Graham DR. Noxious gases and fumes. In: Baum GL, Crapo JD, Celli BR, Karlinsky JB, eds. Textbook of pulmonary disease. 6thEd. Lippincott-Raven Publishers: 1998:741-753.
5. Bosse GM. Nebulized sodium bicarbonate in the treatment of chlorine gas inhalation. *J Toxicol Clin Toxicol*. 1994;32(3):233-241.
6. Weiss SM, Lakshminarayan S. Acute inhalation injuries. *Clin Chest Med*. 1994;15(1):103-115.
7. Vinsel PJ. Treatment of acute chlorine gas inhalation with nebulized sodium bicarbonate. *J Emerg Med*. 1990;8(3):327-329.
8. Mrvos R, Dean BS, Krenzelok EP. Home exposures to chlorine/chloramine gas: review of 216 cases. *South Med J*. 1993;86(6):654-657.
9. Brooks SM, Weiss MA, Bernstein IL. Reactive airways dysfunction syndrome (RADS): persistent asthma syndrome after high level irritant exposure. *Chest*. 1985;88(3):376-384.
10. Shusterman D. Sequelae of respiratory tract exposures to irritant chemicals. Pulmonary and critical care update. American College of Chest Physicians 2001;15(Lesson 2): 1-4. Available at: http://chestnet.org/education/online/pccu/vol15/lessons12/lesson02.php.Accessed July 26, 2011.
11. Gautrin D, Boulet LP, Boutet M, et al. Is reactive airways dysfunction syndrome a variant of occupational asthma? *J Allergy Clin Immunol* 1994; 93(1):12-22.