Respir Case Rep 2014;3(2):86-89 DOI: 10.5505/respircase.2014.97268

Hypersensitivity Pneumonitis due to Glue Inhalation

Yapıştırıcı İnhalasyonu Sonrası Gelişen Hipersensitivite Pnömonisi

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Abstract

A 31-year-old male patient was admitted to the hospital with coughing and dyspnea. One month ago he was treated with inhaler drugs with the diagnosis of occupational asthma. His history revealed that he smoked five packets of cigarettes per year (he was an ex-smoker for 8 years) and worked in the furniture industry for 3 years. He also added that he was exposed to glue inhalation in the work place; he did not use a mask. The physical examination revealed normal vital signs, and oxygen saturation was 95%. Rales in the base of right lung were also noted. Diffusion capacity was found to be very low. The thoracic CT revealed ground-glass densities in the right lower lobe and air trapping in the left lung. Fiber optic bronchoscopy and transbronchial biopsy were performed. The transbronchial biopsy specimen was insufficient for a specific diagnosis. Right thoracotomy and wedge resection were performed for the specific diagnosis. The pathological diagnosis was reported to be hypersensitivity pneumonitis (HP). The patient was treated with methylprednisolone (1mg/kg/day) for six months. After the treatment and removal from the antigen exposure, clinical and radiological improvements were obtained. In conclusion, detailed working anamnesis is important in the patients with suspicion of occupational HP.

Key words: *Hypersensitivity pneumonitis, occupational asthma, glue.*

Özet

Otuzbir yaşında erkek hasta nefes darlığı, öksürük şikâyetleri ile başvurdu. Hasta 1 ay önce mesleksel astım tanısıyla inhaler tedavi kullanmıştı. Özgeçmişinde 5 paket yıl sigara hikâyesi (8 yıldır sigara içmiyor) vardı. Hasta 3 yıldır mobilya fabrikasında çalışıyor ve çalışma ortamında maske kullanmadığını ve yapıştırıcı inhalasyonu olduğunu tarifliyordu. Fizik muayenesinde vital bulguları normaldi. Oksijen saturasyonu %95, sağ akciğer bazalinde raller vardı. Difüzyon kapasitesi ise çok düşüktü. Toraks BT'de sağ alt lobda buzlu cam densitesi, sol akciğerde hava hapsi mevcuttu. Fiberoptik bronkoskopi yapıldı ve transbronşiyal biyopsi alındı. Ancak biyopsi materyali kesin tanı için yeterli değildi. Sağ torakotomi yapılan hastadan wedge biyopsi alındı. Histopatolojik tanı ise hipersensitivite pnömonisi (HP) olarak raporlandı. Hastaya 6 ay metilprednizon (1mg/kg/gün) tedavisi verildi. Tedavi sonrası ve antijen maruziyetinin kesilmesi ile belirgin klinik ve radyolojik düzelme sağlandı. Sonuç olarak detaylı meslek anamnezinin mesleksel HP şüphesi durumunda önemli olduğunu vurgulamak istiyoruz.

Anahtar Sözcükler: *Hipersensitivite pnömonitis, mesleksel astım, yapıştırıcı.*

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Submitted (Başvuru tarihi): 29.10.2013 **Accepted (Kabul tarihi):** 23.12.2013
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Hypersensitivity pneumonitis (HP) is a pulmonary disease with symptoms of dyspnea and coughing, resulting from the inhalation of an antigen to which the patient has been previously sensitized (1,2). Chemical-induced hypersensitivity pneumonitis has been so far rarely described. Reactive low molecular weight chemicals, many of which are known causes of occupational asthma, may act as haptens and cause HP (1). Occupational exposure to some low-molecular-weight chemicals such as isocyanate vapors, and aerosols that are found in foam, glue, and spray paint may induce typical hypersensitivity pneumonitis (3). The current report presents a case of HP due to glue inhalation that was misdiagnosed as occupational asthma.

CASE

A 31-year-old male patient was admitted to a reference hospital for chest diseases with the symptoms of coughing and dyspnea. He had effort-associated dyspnea and a nonproductive cough for one year. Symptoms progressed over the past three months. He had no sputum, hemoptysis, chest pain, or fever. He had an involuntary 6 kg weight loss in the last year. One month prior, he was treated with inhaler drugs with the diagnosis of asthma, with only mild improvement. His history revealed that he smoked five packets of cigarettes per year (he was an ex-smoker for 8 years) and worked in the furniture industry for 3 years. He also added that he was exposed to glue inhalation in the work place; he did not use a mask. The physical examination revealed normal vital signs, and oxygen saturation (SO_2) was 95%. The chest examination revealed rales in the base of the right lung. The examination of the other systems was normal. The chest radiograph revealed non-homogenous infiltration in the paracardiac area and the lower zone at the right side. Hemogram, biochemical tests, CRP level, total IgE and urine test were all normal; sedimentation rate was 30 mm/hr. Results of the pulmonary function test were as follows: forced expiratory volume (FEV1): 42 % (1.57 liters), forced vital capacity (FVC): 44% (1.92 liters), FEV1/FVC: 82%, FEF 25-75: 37% (1.70 liters/sec), and diffusing capacity: 36%. The thoracic CT revealed ground-glass densities in the right lung basal segments and air trapping in the left lung (Figure 1). Fiberoptic bronchoscopy was performed; vocal cords and trachea were normal. Both bronchial systems were open up to the subsegmental levels. Transbronchial biopsy and bronchoalveolar lavage (BAL) were obtained from the right lung basal segment. The BAL analysis produced the following, total cell count: 1020/mm^3, lymphocytes 70%, macrophages 20%, neutrophils 9%, and eosinophils 1%, and CD4/CD8: 0.32. Transbronchial biopsy was insufficient for the specific diagnosis. Bronchial lavage cytology was negative for malignancy. Bronchial lavage was negative for acid resistant bacilli (ARB). For a specific diagnosis, a wedge resection was performed through right thoracotomy. The histopathological examination revealed lymphocyte infiltration and non-necrotizing granulomas with the diagnosis of hypersensitivity pneumonitis (extrinsic allergic alveolitis) (Figure 2). No clinical and radiological improvements were obtained in spite of removal from antigen exposure. Thus, the patient was treated with methylprednisolone (1mg/kg/day) for six months. Following treatment, the patient experienced clinical and radiological improvement. In the thoracic CT, ground-glass densities in the right lower lobe were regressed (Figure 3). The pulmonary function test results, in particular, significantly improved after the treatment as follows: FEV1: 69% (2.56 L), FVC: 75% (3.25 L), FEV1/FVC: 79%, FEF 25-75: 54% (2.45 L/sec) and diffusing capacity was 93%.

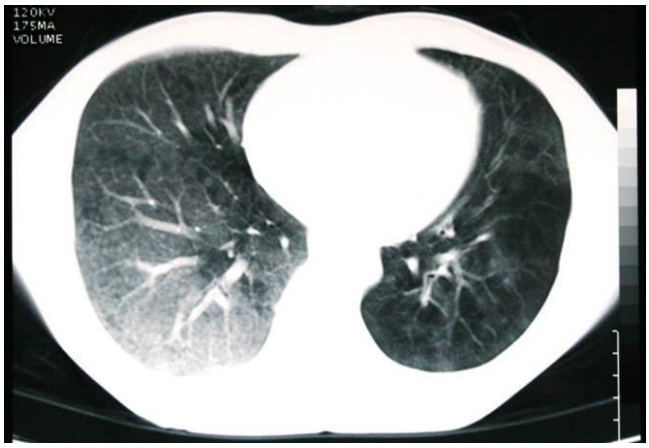

Figure 1: *Ground-glass densities in the right lower lobe and air trapping in the left lung in the thoracic CT before treatment*

DISCUSSION

Patients with HP might be misdiagnosed with occupational asthma, as they have similar symptoms and a history of exposure in the work place. Low molecular weight chemicals, especially isocyanates, are known to cause occupational asthma (1). In the current report, the patient was working in the furniture industry and had nonspecific symptoms as dyspnea and cough, and thus he was treated with bronchodilator drugs with the diagnosis of asthma. However, his chest radiograph was not normal and the diffusion capacity for carbon monoxide was low. Furthermore, the thoracic CT revealed ground-glass densities in the right lower lobe, which is not expected in patients

with asthma. In the BAL analysis, the CD4/CD8 level was low and the histopathological examination was compatible with HP.

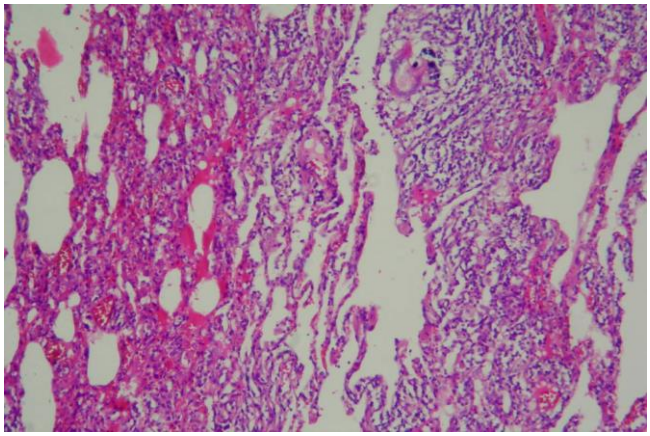

Figure 2: *Lymphocyte infiltration and non-necrotizing granulomas with the diagnosis of hypersensitivity pneumonitis*

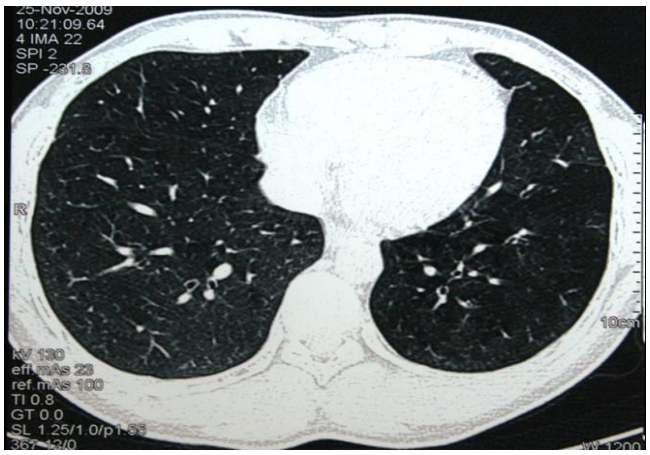

Figure 3: *Thoracic CT showing improvement in ground-glass densities in the right lower lobe after treatment*

Some low-molecular-weight chemicals that by themselves are not solely antigenic may combine with the host's proteins to form haptens, which then may provoke hypersensitivity pneumonitis. For example, the agents implicated in a chemical worker's lung are isocyanates, and are found in products such as foam, glue, and spray paint (4). Clinical suspicion is important in the diagnosis of occupational HP. In particular, the finding of immunoglobulin G precipitating antibodies in the serum of patients with suspected HD is a helpful diagnostic clue (2,5). However, the researchers of the current study could not measure precipitins in the serum of the patients.

Hypersensitivity pneumonitis is not an atopic disease. There is no increase in IgE levels and eosinophil count. Precautions to decrease the antigen exposure must be taken both at home and in workplaces. In particular, the use of filtered masks, air conditioning, and the regular measurement of dust concentration are important. Many agents may cause HP and the most important factor in the treatment is the cessation of the antigen exposure (5). The decision to initiate steroid therapy should be based on the severity of symptoms and physiological abnormalities. The effect of antigen type on the prognosis is unclear. Genetics and the duration of antigen exposure are important for the prognosis (5-7). In the current patient, there was no clinical and radiological improvement despite of removal from antigen exposure. Thus, he was treated with methylprednisolone for six months, which resulted in clinical and radiological improvement.

In conclusion, recognizing HP is clinically challenging because among different patients, the disease might vary according to the clinical presentation, severity, and natural history. Detailed working anamnesis is important in patients with suspicion of occupational HP.

CONFLICTS OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Concept - E.Ü., S.C., R.O., A.E., N.Ç., M.G.; Planning and Design - E.Ü., S.C., R.O., A.E., N.Ç., M.G.; Supervision - E.Ü., S.C., R.O., A.E., N.Ç., M.G.; Funding - E.Ü., S.C., R.O.; Materials - E.Ü., S.C., R.O.; Data Collection and/or Processing - E.Ü., S.C., R.O.; Analysis and/or Interpretation - E.Ü.; Literature Review - E.Ü.; Writing - E.Ü.; Critical Review - E.Ü., S.C., N.Ç.

YAZAR KATKILARI

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