Respir Case Rep 2015;4(1):1-4 DOI: 10.5505/respircase.2015.63825

RESPIRATORY CASE REPORTS

Pneumosiderosis in a Welder Masquerading as Hypersensitivity Pneumonitis Caused by Pigeons

Kaynakçıda Güvercinlere Bağlı Hipersensitivite Pnömonisi ile Karışan Pnömosiderozis

Ayşe Baççıoğlu,[1] Füsun Kalpaklıoğlu,[1] Önder Bozdoğan,[2] Mikail İnal,[3] Ömür Güngör[4]

Abstract

A 43-year-old man with a dyspnea, and cough had been a welder for 20 years, and had raised pigeons for 30 years. A pre-diagnosis of hypersensitivity pneumonitis was proposed with multiple micronodular opacities in radiology, exposure to pigeons, and hypoxemia. However, it was ruled out with continuing lung symptoms though avoidance of pigeons, no alveolitis in the bronchial lavage, corticosteroid insensitivity, and normal diffusion lung capacity. A diagnosis of welder's lung disease was made with the occupational environment consisting of iron dust particles, compatible chest radiology, and positive staining for ferritin (Prussian blue stain) of the macrophages in the bronchial lavage. His hypoxemia was thought to improve after bronchial washing. He was recommended to quit his job and quit smoking, as well as feeding pigeons, since these factors potentially had an effect and predispose lung fibrosis, asthma, and cancer.

Key words: *Occupational lung disease, pigeon's lung, welding.*

Özet

Nefes darlığı ve öksürük şikâyetleri olan 43 yaşındaki erkek hasta, 20 yıldır kaynakçılık yapmakta ve 30 yıldır güvercin beslemekteydi. Radyolojide yaygın mikronodüler opasiteler, güvercin besleme öyküsü ve hipoksemi olması nedeniyle ile ön tanı olarak hipersensitivite pnömonisi düşünüldü. Ancak güvercinlerin uzaklaştırılmasına rağmen şikâyetlerinin devam etmesi, bronş lavajında lenfositik/nötrofilik alveolitis bulgusunun olmaması, akciğerde fibrozis olmamasına rağmen kortikosteroide yanıtsızlık ve normal akciğer difüzyon kapasitesiyle bu tanıdan uzaklaşıldı. Kaynakçı akciğeri hastalığı, meslek ortamındaki demir tozlarının varlığı, uyumlu radyoloji bulguları, serum ferritin yüksekliği ve bronş lavajında Prusya mavisi ile boyanmış demir granüllerinin görülmesiyle kondu. Hastanın hipoksemisinin yapılan bronş lavajı sonrası demir granüllerinin uzaklaştırılmasıyla düzeldiği düşünüldü. Hastaya tedavi olarak mesleğini değiştirmesi, sigarayı bırakması ve güvercinlerden uzaklaşması önerildi. Çünkü bu faktörler ileride akciğer fibrozisi, astım ve akciğer kanseri gelişimine sebep olabilmektedir.

Anahtar Sözcükler: *Mesleki akciğer hastalığı, güvercinci akciğeri, kaynakçılık.*

[1]Department of Pulmonary Diseases, Division of Immunology and Allergy Diseases, Kırıkkale University Faculty of Medicine, Kırıkkale, Turkey
[2]Department of Pathology, Kırıkkale University Faculty of Medicine, Kırıkkale, Turkey
[3]Department of Radiology, Kırıkkale University Faculty of Medicine, Kırıkkale, Turkey
[4]Department of Pulmonary Diseases, Kırıkkale University Faculty of Medicine, Kırıkkale, Turkey

[1]Kırıkkale Üniversitesi Tıp Fakültesi, Göğüs Hastalıkları Anabilim Dalı, İmmünoloji ve Alerji Hastalıkları Bilim Dalı, Kırıkkale
[2]Kırıkkale Üniversitesi Tıp Fakültesi, Patoloji Anabilim Dalı, Kırıkkale
[3]Kırıkkale Üniversitesi Tıp Fakültesi, Radyoloji Anabilim Dalı, Kırıkkale
[4]Kırıkkale Üniversitesi Tıp Fakültesi, Göğüs Hastalıkları Anabilim Dalı, Kırıkkale

Submitted (Başvuru tarihi): 24.05.2014 ***Accepted (Kabul tarihi):*** 16.07.2014

Correspondence (İletişim): Ayşe Baççıoğlu, Department of Pulmonary Diseases, Division of Immunology and Allergy Diseases, Kırıkkale University Faculty of Medicine, Kırıkkale, Turkey
e-mail: aysebaccioglu@gmail.com

Pneumosiderosis or welder's lung is a rare occupational lung disease that is seen after inhalation of iron dust particles in welders, and occurs in about 7% of arc welders (1). Herein we present a case of welder's lung with a differential diagnosis of hypersensitivity pneumonitis in the light of literature.

CASE

A 43-year-old-man reported a one-year history of dyspnea and non-productive cough that was nonresponsive to antibiotics. He was an active smoker (20 packs/year), and had been feeding pigeons out of his house for 30 years. On admission, the physical examination revealed no crackles or rhonchi. The chest radiograph showed a pattern of reticular-linear opacities bilaterally in all zones, and computed tomography showed bilateral diffuse multiple micronodular opacities and peribronchial thickening accompanied by lymphadenopathies (< 1cm) in the right hilar, paratracheal, and precarinal area (Figure 1). His complete blood count, renal, and liver function tests, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), and urine analysis were within normal limits. Some laboratory findings are summarized in Table 1, and were characterized by a mild hypoxemia, mould sensitivity, normal pulmonary functions, and diffusion lung capacity (DL_{CO}).

The bronchoscope visual examination was normal. Bronchial lavage culture was negative for bacteria except *Candida non-albicans*, and lavage cytology was clear for malignancy with a normal cell profile (Table 1). However, oxygen saturation increased to 97% and pO_2 to 80.9 mmHg.

Hypersensitivity pneumonitis (HP) was presumed with three (symptoms, radiology, exposure to pigeons) of six major criteria (biopsy not taken, natural provocation test not completed, but no increase in symptoms nearby pigeons, and no lymphocytic / neutrophilic alveolitis in bronchial lavage), and one minor criteria of hypoxemia (normal DL_{CO} and no crackles) (2). However, HP was ruled out with persistent lung symptoms and radiology, despite four weeks of inhaled 1000 µg fluticasone/day, followed by 4 weeks of methyl-prednisolone 0.5 mg/kg/day concomitant with 8 weeks of avoidance of pigeons.

He was a welder for 20 years, and was trimming/scarfing metal objects to build heating boilers. His occupational environment had an ineffective exhaust-ventilated-hangar with welding fumes, where approximately 2 of 100 colleagues quit due to respiratory symptoms. Moreover, his previous bronchial lavage fluid preparations were reinvestigated, and revealed numerous aggregates of iron particles stained with Prussian blue, both outside and within the alveolar macrophages (Figure 2). Finally, our patient was diagnosed with welder's lung (WL) due to exposure to iron fumes, micronodular lung pattern, iron granules stained with Prussian blue in bronchioalveolar lavage (BAL), and semi-high serum ferritin levels.

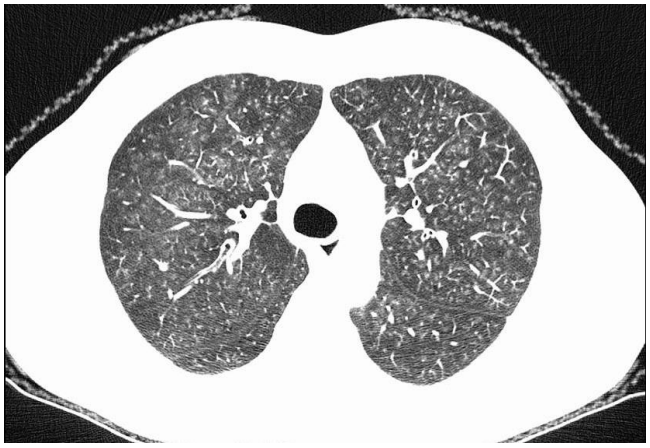

Figure 1: *High-resolution computed tomography (CT) showed bilateral diffuse multiple center-asiner ground-glass nodular opacities, and peribronchial thickening*

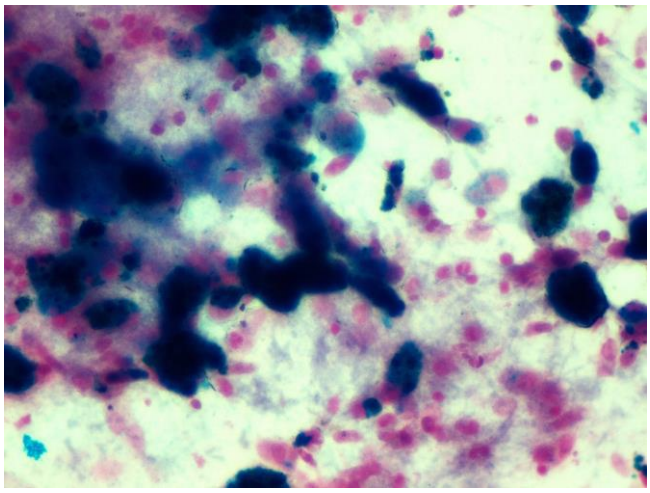

Figure 2: *Bronchial lavage preparations revealed numerous intracytoplasmic iron accumulations in macrophages stained with Prussian blue. Granular blue material is also seen in the background (x 40)*

Hypoxemia was improved by bronchial washing. He was recommended to quit his job and quit smoking to prevent lung fibrosis and cancer. Furthermore, he had a high risk of developing lung cancer with his long duration of exposure to iron fumes besides smoking. Even though he was reported to have a good prognosis with strict avoidance of iron dust, the risk of lung cancer remained even after controlling for the smoking status. Alternative employment with no risk of the inhalation of toxic fumes was advised,

such as a desk job. On the other hand, his employer was informed about the use of respiratory protective equipment and local exhaust ventilation in the workplace. Written informed consent was obtained from the patient prior to publishing his story.

Table 1: Laboratory findings of the patient

Variable	Value
Serum:	
Ferritin, ug/L (Normal: 20-250 ug/L).	254
Total IgE, kU/L,	404
Specific IgE for mold mix, grade (kUA/L)	Grade 2 positive (1.27)
Specific IgE for Aspergillus, Alternaria, feathers mix, occupational allergen mix (Isocyanates, phthalic anhydride	0
Bronchial lavage:	
Specific IgE for mold mix, grade (kUA/L)	0 (0.05)
Arterial blood gases:	
pO_2, mmHg (pre-post bronchoscopy)	63-80.9
pCO_2, mmHg (pre-post bronchoscopy)	38-40
Sa,O_2 % (pre-post bronchoscopy)	93-97
Pulmonary function test:	
FEV_1 L (% pred)	3.53 (94)
FEV_1/FVC % (% pred)	73 (100)
$DL,_{CO}adj$ (% pred)	96
Bronchial lavage:	
Non-specific culture	1. Bacteria (-) 2. Acid fast bacilli (-) 3. *Candida non-albicans* (+)
Cytology	1. Negative for malignancy, 2. Cell profile : 40% of alveolar macrophage, 30% of neutrophils and 30% of lymphocytes, 3. Numerous aggregates of iron particles stained with Prussian blue, both outside and within alveolar macrophages.

IgE: immunoglobulin E; FVC: forced vital capacity; % pred: % predicted; FEV1: forced expiratory volume in one second; DL,COadj: diffusing capacity of the lung for carbon monoxide adjusted for haemoglobin; mold mix: *Penicillium chrysogenum, Cladosporium herbarum, Aspergillus fumigatus, Candida albicans, Alternaria alternata, Setomelanomma rostrata*, PO_2: partial pressure of oxygen; PCO_2: partial pressure of carbon dioxide; Sa,O_2: arterial oxygen saturation.

DISCUSSION

WL is a type of pneumoconiosis caused by the accumulation of iron particles in the lung macrophages, in addition to "industrial bronchitis" by the irritant effect (1). The symptoms are usually nonspecific, such as shortness of breath and cough. Although 20 years seems to be a mile stone in WL, some welders might have symptoms even after five years, whereas some never become ill due to low exposure intensity of iron fumes, and phagocytic capacity of macrophages (3). The diagnostic tests are staining with Prussian blue of ferritin granules in the alveolar macrophages, and an elevation of ferritin levels in bronchial lavage followed by serum. Even though the patient's pulmonary function tests were normal, it was reported that there might be a decline in pulmonary functions in the future with continuing exposure (1). The treatment is usually symptomatic, and bronchial washing might improve symptoms and radiology, as in the current case (3). Prognosis is generally favorable if strict avoidance of the occupational environment is adopted (4). On the other hand, continued inhalation of iron dust particles may lead to the development of progressive massive fibrosis, and spontaneous pneumothorax/fungus ball (5,6). Furthermore, the risk of lung cancer among welders is associated with the duration and density of welding particulate exposure, which remains even after smoking cessation (4,7).

This case is interesting with multiple exposures, including welding and pigeons, to cause lung disease. The common findings of both diseases are nonspecific lung symptoms and micronodular opacities. HP can present with flu-like symptoms, weight-loss, fatigue, and loss of appetite, fine rales in the lung, increases in ESR and CRP, lymphocytosis in the bronchial lavage, restriction in pulmonary function tests, decrease in DLCO, and respiratory failure caused by the progressive fibrotic lung disease (8); all symptoms were absent in this case. The diagnosis of HP can be made certain after the improvement in symptoms and radiology by avoidance of pigeons for at least four weeks and/or corticosteroid therapy (2). On the other hand, there were mold species in the bronchial lavage, and the patient was sensitive to some mold types with increased IgE, that might be due to exposure to dampness in the pigeons' nests. Pigeon's disease is a type III hypersensitivity reaction related to IgG, whereas IgE positivity to mold was a hyper-sensitization, but not a cause of lung symptoms (8). Furthermore, in the literature, pulmonary aspergillum was found to be related to WL (9).

In conclusion, as exposure both to metal fumes and pigeons had the potential to develop lung diseases with a similar clinic and radiology, a careful environmental history and laboratory examination are import in highlighting the diagnostic dilemma.

CONFLICTS OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Concept - A.B., F.K., Ö.B., M.İ., Ö.G.; Planning and Design - A.B., F.K., Ö.B., M.İ., Ö.G.; Supervision - A.B., F.K., Ö.B., M.İ., Ö.G.; Funding - Materials - F.K.; Data Collection and/or Processing - Ö.B., Ö.G.; Analysis and/or Interpretation - M.İ.; Literature Review - A.B.; Writing - A.B.; Critical Review - F.K.

YAZAR KATKILARI

Fikir - A.B., F.K., Ö.B., M.İ., Ö.G.; Tasarım ve Dizayn - A.B., F.K., Ö.B., M.İ., Ö.G.; Denetleme - A.B., F.K., Ö.B., M.İ., Ö.G.; Kaynaklar - Malzemeler - F.K.; Veri Toplama ve/veya İşleme - Ö.B., Ö.G.; Analiz ve/veya Yorum - M.İ.; Literatür Taraması - A.B.; Yazıyı Yazan - A.B.; Eleştirel İnceleme - F.K.

REFERENCES

1. Ozdemir O, Numanoğlu N, Gönüllü U, Savaş I, Alper D, Gürses H. Chronic effects of welding exposure on pulmonary function tests and respiratory symptoms. Occup Environ Med 1995; 52:800-3. [CrossRef]
2. Schuyler M, Cormier Y: The diagnosis of hypersensitivity pneumonitis. Chest 1997; 111:534-6. [CrossRef]
3. Yamada G, Igarashi T, Sonoda H, Morita S, Suzuki K, Yoshida Y, et al. Use of bronchopulmonary lavage for eliminating inhaled fume particles from a patient with arc welder's lung. Intern Med 1998; 37:962-4. [CrossRef]
4. Ji C, Chen G, Cai HR, Meng FQ, Chen YB, Guo LC, et al. An unusual case of Welder's siderosis with local massive fibrosis: a case report. Chin Med J (Engl) 2012; 125:552-4.
5. Khalid I, Khalid TJ, Jennings JH. A welder with pneumosiderosis: a case report. Cases J 2009; 2:6639. [CrossRef]
6. Fidan F, Esme H, Unlu M, Acar M, Albayrak R, Dilek FH. Welder's lung associated with pneumothorax. J Thorac Imaging 2005; 20:120-2. [CrossRef]
7. Sørensen AR, Thulstrup AM, Hansen J, Ramlau-Hansen CH, Meersohn A, Skytthe A, et al. Risk of lung cancer according to mild steel and stainless steel welding. Scand J Work Environ Health 2007; 33:379-86. [CrossRef]
8. Hirschmann JV, Pipavath SN, Godwin JD. Hypersensitivity pneumonitis: a historical, clinical, and radiologic review. Radiographics 2009; 29:1921-38. [CrossRef]
9. Ashizawa H, Fujita A, Inami N, Tsuchiya T, Yoshitomi A, Masuda M, et al. Case of pulmonary aspergilloma in the apices of both lungs which was associated with welder's lung. Nihon Kokyuki Gakkai Zasshi 2010; 48:860-5.