

Case Report

Occupational Emphysema Following Long-term Exposure to Metal Fumes During Electroplating in a Non-smoker

Richa Mittal, Pawan Gupta, Devi Jyoti Dash, Rajendra Prasad and Sunil Kumar Chhabra

Departments of Cardio-respiratory Physiology and Pulmonary Medicine, Vallabhbhai Patel Chest Institute, University of Delhi, Delhi, India

Abstract

Exposure to cigarette smoke is by far the most common causative factor for chronic obstructive pulmonary disease. Occupational exposure to fumes, chemicals, dusts and environmental pollution is also an important cause of chronic productive cough. Emphysema developing as a consequence of an occupational exposure is extremely rare. We describe the rare occurrence of severe emphysema in a non-smoker male who had worked for nearly three decades in nickel electroplating industry. [Indian J Chest Dis Allied Sci 2016;58:123-125]

Key words: Emphysema, Occupational exposure, COPD, Environmental pollution, Cough.

Introduction

Exposure to noxious environmental stimuli that incite an inflammatory response in the airways is the initiating event in the pathogenesis of chronic obstructive pulmonary disease (COPD).¹ The chronic inflammatory process in the airways and the development of emphysema are responsible for the characteristic airflow limitation that is only partially reversible. Exposure to cigarette smoke is by far the most common causative factor though occupational exposures to fumes, chemicals and dusts, as well as environmental pollution are also responsible for a substantial proportion of patients with COPD.² Usually, such occupational exposures result in chronic productive cough (chronic bronchitis phenotype).³ Emphysema developing as a consequence of an occupational exposure has been reported only rarely.^{4,7}

We describe the rare occurrence of severe emphysema in a non-smoker male who had worked for nearly three decades in nickel electroplating industry.

Case Report

An 80-year-old male patient, a life-time non-smoker, presented with persistent cough with scanty sputum, and progressively worsening dyspnoea on exertion that had become prominent for the past three years causing severe limitation of activities, currently grade 4 of the modified Medical Research Council scale.⁸ There was no history of wheezing, fever, haemoptysis and joint pains. There was no previous history of any significant respiratory or other systemic disease nor was there any significant family history of a similar

disorder. He had worked in an electroplating industry where he was exposed to metals fumes emanating from the use of nickel and aluminum for 30 years. He started developing symptoms while still in the job but quit a few months later and had now been symptomatic for three years. Detailed questioning did not reveal significant exposure to environmental tobacco smoke or any other noxious substance at home or at work. He had received oral bronchodilators with corticosteroids from time to time. Examination revealed evidence of a barrel-shaped chest with signs of hyperinflation, scattered end-inspiratory crackles and prolonged expiration with wheeze. There was no cyanosis, clubbing or oedema. Pulse oximetry showed oxygen saturation (SpO₂) of 90%.

Chest radiograph showed hyperinflated lung fields with a long tubular heart (Figure 1). Laboratory investigations, namely, blood counts, glucose, and serum biochemistry including hepatic and renal function were in the normal range. The lung function data are shown in the table. Spirometry revealed a mild obstructive ventilatory impairment with lack of any significant response to inhaled salbutamol. The functional residual capacity was increased suggesting hyperinflation. The single breath diffusion capacity for carbon monoxide (DLCO_{SB}) was severely reduced, even after correction for alveolar volume.

High resolution computed tomography (HRCT) of the chest (Figure 2) revealed bilateral extensive centrilobular emphysema, more in the upper lobes and a sabre-sheath trachea with a sagittal:coronal diameter > 2:1 (not shown). There was no evidence of mediastinal lymphadenopathy (Figure 2). The

[Received: January 8, 2015; accepted after revision: May 20, 2015]

Correspondence and reprint requests: Dr Sunil Kumar Chhabra, Professor, Department of Cardio-respiratory Physiology, Vallabhbhai Patel Chest Institute, University of Delhi, Delhi-110 007, India; E-mail: skchhabra@mailcity.com

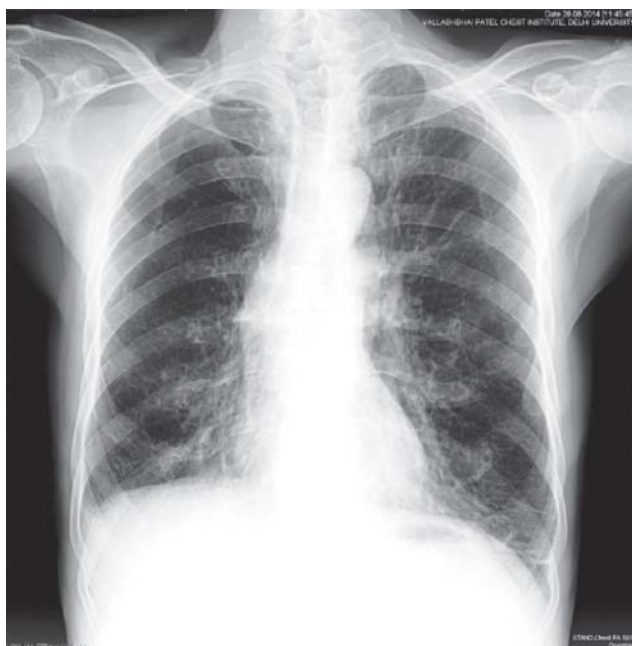


Figure 1. Plain chest radiograph (postero-anterior view) showing hyperinflated lung fields with a long tubular heart, prominent hila and peripheral pruning of vasculature.

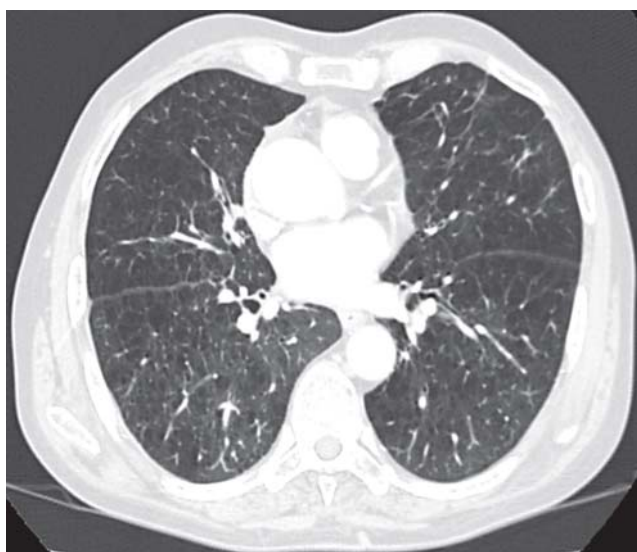


Figure 2. HRCT chest showing bilateral centrilobular emphysema in affecting all lobes.

electrocardiogram showed low voltage complexes and poor progression of R waves. Arterial blood gas analysis revealed pH of 7.44, arterial carbon dioxide tension 49.5 mmHg, arterial oxygen tension 69.6 mmHg and bicarbonate 32.9 mmol/L, suggestive of mild hypoxaemia with metabolic alkalosis.

Based on the history of occupational exposure to heavy metal fumes, absence of any other exposures, clinical features, and characteristic lung function and HRCT findings, a diagnosis of occupational emphysema was established. He was advised inhaled long-acting bronchodilators that provided some relief in symptoms.

Table. Lung function data

	Pre-bronchodilator	Post-bronchodilator
FVC (L)	2.79 (104%)	2.75 (103%)
FEV ₁ (L)	1.23 (72%)	1.29 (75%)
FEV ₁ /FVC	44	47
TLC (L)	4.67 (103%)	
FRC (L)	3.51 (119%)	
RV (L)	1.86 (82%)	
DLCO _{SB} (mL/min/mm ³ Hg)	10.03 (33%)	
DLCO/VA	2.11 (50%)	

Definitions of abbreviations: FVC=Forced vital capacity; FEV₁=Forced expiratory volume in one second; TLC=Total lung capacity; FRC=Functional residual capacity; DLCO_{SB}=Diffusion capacity for carbon monoxide; VA=Alveolar volume.

Discussion

Unlike occupational and work-associated asthma, the role of occupational factors as the main or contributory cause in the development of emphysema has received remarkably little attention. The true population-attributable risk due to occupational exposure is unclear as occupational COPD is rarely clinically diagnosed.⁹ Establishing a true cause-effect relationship is difficult as it takes years of exposure to the offending agent. Off-work amelioration of symptoms and recurrence on re-exposure as well objective lung function monitoring on and off work that are the standard tools to diagnose occupational asthma are not applicable to occupational COPD. Further, smoking often confounds the estimation of the risk of developing COPD.

The association of dusts, fumes, and chemicals of industrial origin with the development of COPD has been documented in some studies.¹⁰⁻¹² A systematic epidemiological review by the American Thoracic Society showed that about 15% of COPD cases might be attributable to workplace exposure.¹⁰ Findings of seven population-based studies on occupational COPD revealed that a population-attributable risk for occupational COPD to be 15%.¹² The population-attributable fraction for COPD associated with occupational exposure has been estimated between 9% and 31%.¹²

Industrial exposures to metal fumes have also been associated with airways obstruction. A high prevalence of COPD has been reported in iron steel and ferrochrome industry workers.¹³ Metal smelting activities have also been related to worsening annual decline of forced expiratory volume in one second (FEV₁), suggesting that it may result in an increased risk of COPD.¹⁴ The main observations in these studies have been a higher prevalence of chronic productive cough and lower FEV₁ in the exposed workers after taking into account the confounding effect of smoking. In contrast, the development of emphysema following prolonged industrial exposure is less well

established.⁴⁻⁷ It has been suggested that heavy exposure to cadmium may lead to emphysema.⁵ *In vivo* diagnosis of emphysema requires HRCT of the chest or measurement of diffusion capacity. Both are usually not feasible in the settings in which occupational COPD has been studied. Hence, the true prevalence of occupational emphysema and specific occupation associated with it has never been established.

Electroplating is a process that uses electrical current to reduce dissolved metal cations so that these form a coherent metal coating on an electrode. Metals used for plating include nickel, cadmium, platinum, gold, silver, chromium etc. Throughout the electroplating process, operators are exposed to hazardous chemical vapour, fume and mist. In addition, metal dust is generated. Chromium, nickel and cadmium are all on the International Agency for Research on Cancer's (IARC) list of known human carcinogens. Some studies^{15,16} have shown the risk of development of asthma upon exposure to nickel dust but none has shown an association with COPD.

A limitation in proving cause-effect relationship in occupational emphysema is the prolonged exposure required to produce the characteristic features. Amelioration and recurrence of symptoms on avoidance and re-exposure or objective lung function monitoring on and off work are of little help in diagnosing occupational COPD. Diagnosis remains circumstantial. In this case report, we illustrate the association of emphysema with nickel exposure. The patient worked in an electroplating industry for 30 years with exposure of nickel fumes in the working place. We based our diagnosis on the history of occupational exposure to nickel fumes and absence of any other exposures, clinical features and characteristic lung function and HRCT findings.

References

1. MacNee W. Pathogenesis of chronic obstructive pulmonary disease. *Proc Am Thorac Soc* 2005;2:258-66.
2. Blanc P, Toren K. Occupation in chronic obstructive pulmonary disease and chronic bronchitis: an update. *Int J Tuberc Lung Dis* 2007;11:251-7.
3. Rodríguez E, Ferrer J, Zock JP, Serra I, Antó JM, de Batlle J, et al. Lifetime occupational exposure to dusts, gases and fumes is associated with bronchitis symptoms and higher diffusion capacity in COPD patients. *PLoS One* 2014;9:e88426.
4. Kjuus H, Istad H, Langård S. Emphysema and occupational exposure to industrial pollutants. *Scand J Work Environ Health* 1981;7:290-7.
5. Davison AG, Fayers PM, Taylor AJ, Venables KM, Darbyshire J, Pickering CA, et al. Cadmium fume inhalation and emphysema. *Lancet* 1988;1:663-7.
6. Hnizdo E, Vallyathan V. Chronic obstructive pulmonary disease due to occupational exposure to silica dust: a review of epidemiological and pathological evidence. *Occup Environ Med* 2003;60:237-43.
7. Kuempel ED, Vallyathan V, Green FHY. Emphysema and pulmonary impairment in coal miners: quantitative relationship with dust exposure and cigarette smoking. *J Phys Conf Ser* 2009;151:012024.
8. Schilling RSF, Hughes JPW, Dingwall-Fordyce I. Disagreement between observers in an epidemiological study of respiratory disease. *Br Med J* 1955;1:65-8.
9. Decramer M, Sibille Y. European conference on chronic respiratory disease. *Lancet* 2011;377:104-6.
10. Balmes J, Becklake M, Blanc P, Henneberger P, Kreiss K, Mapp C, et al. American Thoracic Society Statement: Occupational contribution to the burden of airway disease. *Am J Respir Crit Care Med* 2003;67:787-97.
11. Blanc PD. Occupation and COPD: a brief review. *J Asthma* 2012;49:2-4.
12. Blanc PD, Iribarren C, Trupin L, Earnest G, Katz PP, Balmes J, et al. Occupational exposures and the risk of COPD: dusty trades revisited. *Thorax* 2009;64:6-12.
13. Bala S, Tabaku A. Chronic obstructive pulmonary disease in iron-steel and ferrochrome industry workers. *Cent Eur J Public Health* 2010;18:93-8.
14. Becklake MR, Irwig L, Kielkowski D, Webster I, de Beer M, Landau S. The predictors of emphysema in South African GOLD miners. *Am Rev Respir Dis* 1987;135:1234-41.
15. McConnell LH, Fink JN, Schlueter DP, Schmidt MG. Asthma caused by nickel sensitivity. *Ann Intern Med* 1973;78:888-90.
16. Malo JL, Cartier A, Doepner M, Nieboer E, Evans S, Dolovich J. Occupational asthma caused by nickel sulfate. *J Allergy Clin Immunol* 1982;69:55-9.