Case Report

Chronic Lung Diseases in Plastic Fumes Exposed Workers: A Case Series

Umesh Chandra Ojha[1], Amit Kumar[2], Sonam Spalgais[3], Omkar K. Choudhari[4] and Amol Anbhule[4]

Institute of Occupational and Environmental Health[1] and Department of Respiratory Medicine[1,4], Employee State Insurance Hospital, New Delhi, Department of Respiratory Medicine and Critical Care[2], Vardhman Mahavir Medical College and Safdarjung Hospital, New Delhi and Department of Pulmonary Medicine[3], Vallabhbhai Patel Chest Institute, University of Delhi, Delhi, India

This article is available on www.vpci.org.in

ARTICLE INFO

Received: August 5, 2020

Accepted after revision: December 31, 2020

Indian J Chest Dis Allied Sci 2021;63:193-197

KEY WORDS

Plastic, Fumes, Spirometry, Bronchiectasis, Poly vinyl chloride.

ABBRIVATIONS USED IN THIS ARTICLE

COPD = Chronic obstructive pulmonary disease

PVC = POLY vinyl chloride

PPE = Personal protective equipment

SpO_2 = Arterial oxygen saturation

RR = Respiratory rate

PR = Pulse rate

BP = Blood pressure

AFB = Acid-fast bacilli

Mtb = Mycobacterium tuberculosis

FVC = Forced vital capacity

FEV_1 =Forced expiratory volume in 1 second

HRCT = High resolution computed tomography

LABA = Long-acting β2 adrenergic agonist

ICS = Inhaled corticosteroid

PPE = Personal protctive equipment

Abstract

The work-place and household exposure to different pollutants involves airways as well as parenchyma of the lung. We evaluated four patients without comorbidities, working in plastic industries at least for 11 years. Pulmonary function testing of the patients revealed mixed airway disease. Computed tomography revealed bronchiectasis, centrilobular nodule, septal thickening and emphysema unrevealing the toxic potential of plastic fumes. The use of personal protective equipment with good work hygiene practises would certainly help in the prevention of these occupation-related morbidities.

Introduction

The environmental exposure including work-place and household exposure in the causation of airway disease is well documented along with lack of awareness and education among fume- and dust-producing occupation is also one of the reason. However, there are many lacunae which need to be addressed further. These include the various spectrums of disorders which may be caused by one agent or multiple agents, often present in the same environment. There is always a low voice for the effect of this exposure on lung parenchyma. We tried to ascertain the effect of plastic fume exposure on the respiratory system manifesting as chronic obstructive pulmonary disease (COPD), occupational asthma, or parenchymal destruction or pleural disease.[1-3] An occupational disease pertaining to exposure to chemicals, solvents, fumes, dust and vapours exhibit symptoms differently. Plastic fumes have the peculiarity that its human manifestation ranges from mild symptoms to devastating diffuse alveolar haemorrhage.[4] The poly vinyl chloride (PVC) is a toxic and carcinogenic polymer of plastic. The other various polymers of plastic causing impairment of lung function are polyethylene, polypropylene, polystyrene apart from PVC.[5] Plastic fumes exposure causes both obstructive and restrictive type pattern abnormality.[6] Workers get

Corresponding author: Dr Omkar Kalidasrao Choudhari, Department of Respiratory Medicine, ESI-PGMIR, Basaidarapur, New Delhi-110 015, India; E-mail: omkarchoudhari@yahoo.com.

exposed to fumes due to poor engineering control techniques and not using proper personal protective equipment (PPE). We are reporting a case series of four patients of mixed airway disease and emphysema (in 2 patients), resulting from the exposure to plastic fumes for at least 11 years.

Cases Reports

Case-1

A 31-year-old male, non-addict with no other co-morbidity presented with cough and expectoration for two months. There was no history of trauma or surgery in the past. There was no history of smoking or allergy in patient and family. Patient was working in plastic moulding industry for 11 years. On examination his oxygen saturation (SpO_2) was 96% at room air, respiratory rate (RR) was 16/min, pulse rate (PR) 84/min and blood pressure (BP) was 130/80mmHg. General physical examination was non contributory. Respiratory system examination showed bilateral infra-scapular rhonchi. Chest radiograph (postero-anterior view) was normal. Laboratory investigations did not show any abnormality. Sputum examination by direct acid-fast bacilli (AFB) staining was twice negative and culture for *Mycobacterium tuberculosis* (*Mtb*) was also negative. Spirometry showed forced vital capacity (FVC): 2.22L (57%), forced expiratory volume in one second (FEV_1): 1.38L (41%) and FEV_1/FVC: 62.11. High resolution computed tomography (HRCT) revealed traction bronchiectasis in bilateral upper lobes, right side more than left with right-sided pleural thickening and bilateral nodules (Figure 1). Patient was diagnosed as a case of occupational lung disease and treated with inhalational corticosteroids (ICS) and long-acting β2 adrenergic agonist (LABA) with other supportive therapy and advised proper use of PPE. His symptoms improved with the treatment.

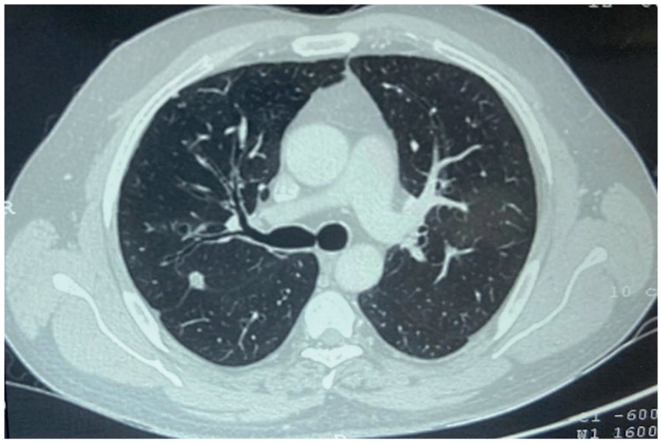

Figure 1. Computed tomography of chest showing bronchiectasis and pleural thickening with nodules (Case 1).

Case-2

A 53-year-old male, non-addict with no other co-morbidities presented with breathlessness for six months. No history of trauma or surgery in the past. History of smoking or allergy in patient and family was negative. Patient was working in plastic moulding industries for 32 years. On examination, his SpO_2 was 94% at room air, RR was 18/min, PR 94/min and BP was 138/80mmHg. General physical examination was normal. Respiratory system examination showed bilateral infra-scapular rhonchi. Chest radiograph (postero-anterior view) showed sign of hyperinflation. His routine blood investigations were within normal limits. Sputum examination by direct AFB staining and *Mtb* culture was also negative. Spirometry showed FVC: 2.60L (79%), FEV_1: 0.35L (35%) and FEV_1/FVC: 34.59. The HRCT showed bilateral lower lobe traction bronchiectasis and cystic lesion with emphysematous changes (Figure 2). Patient was diagnosed to have occupational lung disease and was treated with ICS and LABA with other supportive therapy and advised proper use of PPE during working which improved his symptoms.

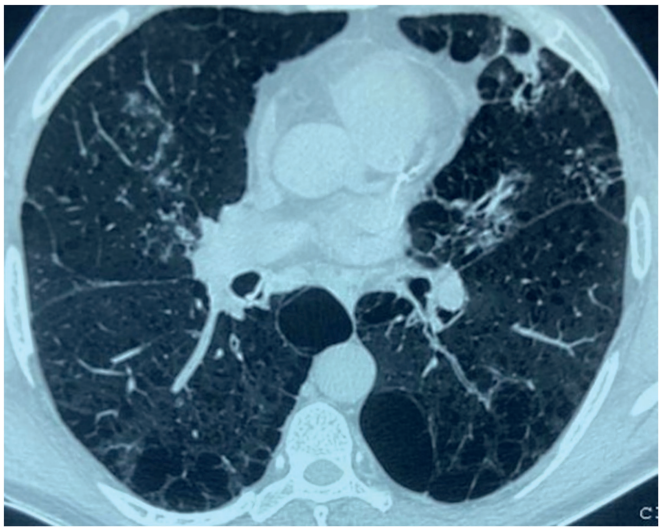

Figure 2. High-resolution computed tomography of chest showing bronchiectasis and emphysema changes with multiple cystic lesions (Case 2).

Case-3

A 48-year-old male, non-addict with no other co-morbidity presented with cough and expectoration for six months. There was no significant past history. Family history of allergy and smoking was not evident. Patient was working in plastic granules making industry the last for 24 years. On examination, his SpO_2 was 96% at room air, RR was 18/min, PR 110/min and BP was 138/80mmHg. General examination

was normal. Electrocardiography showed sinus tachycardia. Respiratory system examination showed reduced bilateral air entry with no added sound. Chest radiograph (postero-anterior view) depicts flattened diaphragm and hyperinflation. Laboratory investigations were within normal limits. The sputum examination for direct AFB smear and *Mtb* culture was negative. The spirometry showed FVC: 1.33L (44%), FEV_1: 0.53L (22%) and FEV_1/FVC: 39.77.The HRCT revealed left upper, lingular and right middle lobe bronchiectasis with centrilobular nodule and pleural thickening (Figure 3). Patient was diagnosed to have occupational lung disease and treated with ICS and LABA with short-term antibiotics and other supportive therapy. He was also advised regarding proper use PPE during working and his symptoms improved.

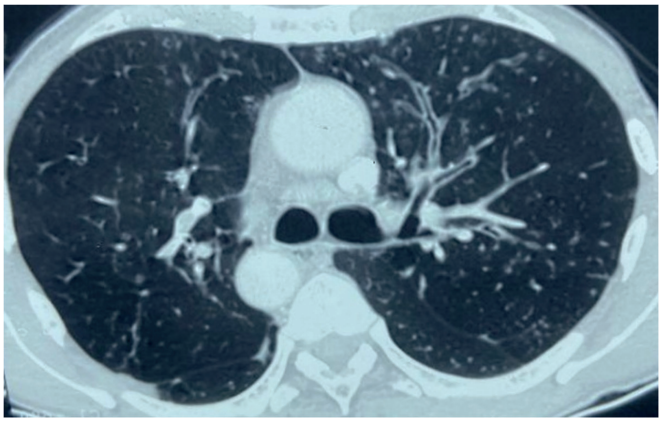

Figure 3. Computed tomography of chest showing bronchiectasis, pleural thickening and bilateral multiple nodules (Case 3).

Case-4

A 44-year-old male, non-addict with no co-morbidity presented with cough with expectoration for one year which increased since last one month. There was no significant past history. There was no history of smoking or allergy in patient and family. He was working in PVC pipes manufacturing industries for the last 21 years. On examination, his SpO_2 was 93% at room air, RR was 18/min, PR 100/min and BP was 148/86mmHg. General examination was normal. Respiratory system examination revealed bilateral few expiratory crepitation. Chest radiograph (postero-anterior view) showed hyperinflation. Blood investigation did not show any abnormality. Sputum smear examination for AFB and culture for *Mtb* were negative. Spirometry showed FVC: 137L (30%), FEV_1: 1.01L (35%) and FEV_1/FVC: 73.54. The HRCT revealed bilateral upper, right middle and lingular lobe traction bronchiectasis with emphysema and pleural thickening (Figure 4). Patient was diagnosed to have occupational lung disease and treated with ICS and LABA with other supportive therapy. He was advised to proper use PPE during working, which improved his symptoms.

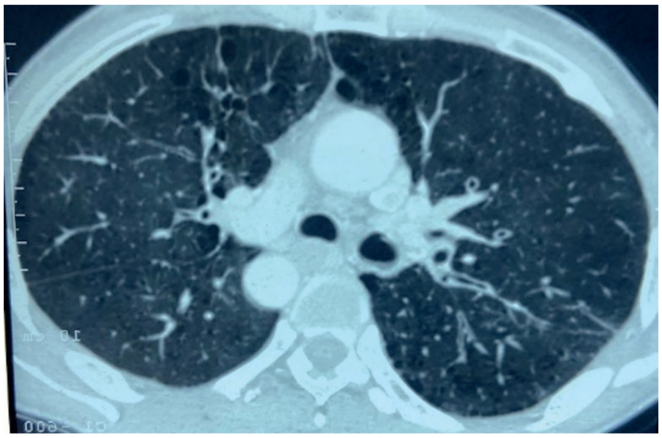

Figure 4. High-resolution computed tomography of chest showing bronchiectasis and septal thickening with emphysematous changes (Case 4).

Discussion

Plastic fume exposure and its sequel on pulmonary function test and lung parenchyma are known since a long time. With the industrial revolution and invention of new techniques, the use of versatile property bearing plastic has increased. The plastic we use and re-use is mainly available in two forms, one is PVC while other is styrene apart from polyethylene and polypropy lene.[5] With increasing awareness, more cases pertaining to plastic fumes induced respiratory diseases are coming in light which improves our understanding of the multi-factorial cause of respiratory disability apart from smoking and biomass fuel exposure. These toxic fumes have a variable effect on humans depending upon the duration of exposure. However, acute inhalational injuries are also reported.[7]

Globally PVC business valued at 70 billion US dollars.[8] In India, PVC market valued at 3,159 million dollars in 2016 and projected to rise 10.2% to reach 6,224 million US dollars by 2023.[9] In India, 4 million people are employed in the plastic industry in around 25000 companies and among plastic exporters, 85% to 90% industries are small and medium-sized enterprises and in financial year 2020, plastic and linoleum export from India valued at 7.55 billion US dollars.[10] The growth of plastic industry is expected to rise 16% every year, thus involving human lives and it is our responsibility to protect the valuable human resources.[11]

The PVC is the most widely used plastic all over the world. It is formed by polymerisation of mono vinyl chloride and are involved in the aetiopathogenesis of simple rhinitis, constrictive

bronchiolitis, bronchiectasis to malignancies.[12,13] It was earlier considered as one of non-toxic material and used general anaesthesia as well.[14] The first report of PVC toxic effects resulting in liver angiosarcoma and acroosteolysis enforced to investigate its similar effects in other neoplastic entities.[15] The inhaled PVC dust component of less than 5μm may travel to respiratory bronchiole and lodge inside the alveoli. The PVC component gradually releases the residual monomer, as PVC after polymerisation has approximately 10% of an unpolymerised monomer which with the help of cytochrome P450 2E1 induces neoplastic changes or lung fibrosis.[16] The relative increase in lung cancer risk is 20% per extra year of work among PVC exposed individuals.[13]

The plastic industries include both the manufacturing and recycling industries. During the recycling of plastics, it is melted at a high temperature and the resultant fumes result in respiratory dysfunction (anatomical and physiological). These fumes contain dioxins, furans, mercury and polychlorinated biphenyls.[17] Dioxins and furan have risk of malignancy and dioxins also affects reproductive system[18,19] while polychlorinated biphenyls have neurotoxic potential and responsible for childhood respiratory infections.[20] Apart from the mentioned effects, being environmental pollutants all of them lead to pulmonary function test abnormality.

Styrene exposure causes in memory loss, brain and liver abnormalities, bronchiolitis, occupational asthma and also has neoplastic potential.[21] Polyethylene and polypropylene affecting the respiratory abnormality is also a known phenomenon.

In many studies, the cause of the impairment in pulmonary function test and respiratory interstitial morbidity is attributed to oxidative stress secondary to toxic fumes mediated by a cascade of pro-inflammatory cytokines. The role of oxidative stress and redox reaction in respiratory diseases, independent of occupational exposure, is attributed in COPD, pulmonary fibrosis or asthma, whereas the occupational contribution for the burden of COPD is 15%.[22-24] The role of plastic fumes is also seen in constrictive bronchiolitis, interstitial lung disease to diffuse alveolar haemorrhage as well as in auditory dysfunction.[12,25,26] Lead is added to the PVC as stabilizer and workers are exposed to lead in various forms, depending upon the nature of job leading to accumulation of this toxic metal and bringing oxidative stress apart from anaemia.[27]

In conclusion, plastic industries are growing exponentially, hence, the exposure. Most of the workers are from poor socio-economic background and are unaware of potential hazards of the occupation. Hence, good work hygiene practices must be taught and followed. Engineering control methods to be adopted by all industries to curb the toxic fumes. Early detection and use of PPE is the key for prevention of these occupational induced morbidities. Plastic fume exposure is yet another cause of chronic lung disease in non-smokers. This type of respiratory disease is characterised by mixed pattern of airway abnormality on spirometry with the evidence of bronchiectasis and small airway disease involvement on HRCT. All people in plastic industry should regularly use proper PPE and may be screened with spirometry at least every 2-3 years to prevent respiratory morbidities and early intervention.

References

1. Mehta JN, Gupta A, Bhatt K, Vasani K. Pulmonary function in petrol pump workers in Anand district. *Natl J Physiol Pharm Pharmacol* 2018;8:23–7.
2. Rajendra KC, Shukla SD, Gautam SS, Hansbro P, Otoole RF. The role of environmental exposure to non-cigarette smoke in lung disease. *Clin Trans Med* 2018;7:39.
3. Vandenplas O, Malo JL. Definitions and types of work-related asthma: a nosological approach. *Eur Respir J* 2003;21:706–12.
4. Kaplan V, Baur X, Czuppon A, Rilegger M, Russi E, Speich R. Pulmonary hemorrhage due to inhalation of vapor containing pyromellitic dianhydride. *Chest* 1993;104:644–5.
5. Maddah HA. Polypropylene as a promising plastic: a review. *Am J Polym Sci* 2016;6:1–11.
6. Khaliq F, Singh P, Chandra P, Gupta K, Vaney N. Pulmonary functions in plastic factory workers: a preliminary study. *Indian J Physiol Pharmacol* 2011;55:60–6.
7. Gorguner M, Akgun M. Acute inhalation injury. *Eur-Asian J Med* 2010;42:28–35.
8. $70 Billion PVC Market by Product, Application and Geography - Global Forecast to 2028 – Research and Markets.com [Internet]. Financialbuzz.com. 2020. *Available from URL:* https://www.financialbuzz.com/70-billion-pvc-market-by-product-application-and-geography-global-forecast-to-2028-researchandmarkets-com/. Accessed on July 27, 2020.
9. India PVC Pipes Market Industry Analysis Report Forecast - 2023| AMR [Internet]. Allied Market Research. 2020. *Available from URL:* https://www.alliedmarketresearch.com/india-pvc-pipes-market. Accessed on July 30, 2020.
10. Indian Plastic Industry and exports Exports I. Indian Plastic Manufacturing & Export Data, Information and Reports | IBEF [Internet]. Ibef.org. 2020. *Available from URL:* https://www.ibef.org/exports/plastic-industry-india.aspx#:~:text=The%20industry%20spans%20the%20country,stood%20at%20US%24%207.55%20billion. Accessed on August 3, 2020.

11. The Plastics Industry in India: An Overview [Internet]. Bpf.co.uk. 2020. *Available from URL:* https://www.bpf.co.uk/article/the-plastics-industry-in-india-an-overview446.aspx#:~:text=The%20Indian%20plastics%20market%20is,5.72m%20tonnes%20in%202009.&text=Despite%20India%20having%20a%20population,reported%20problems%20with%20labour%20shortages. Accessed on July 27, 2020.
12. Kreiss K. Occupational cause of constrictive bronchiolitis. *Curr Opin Allergy Clin Immunol* 2013;13:167–72.
13. Mastrangelo G, Fedeli U, Fadda E, Milan G, Turato A, Pavanello S. Lung cancer risk in workers exposed to poly (vinyl chloride) dust: a nested case-referent study. *Occup Environ Med* 2003;60:423–28.
14. Eckardt RE. Occupational and environmental health hazards in the plastic industry. *Environ Health Perspect* 1976;17:103–06.
15. Sherman M. Viny l chloride and the liver. *J Hepatol* 2009;51:1074–81.
16. Crawford EL, Weaver DA, DeMuth JP, Jakson CM, Khuder SA, Frampton MW, *et al.* Measurement of cytochrome P450 2A6 and 2E1 gene expression in primary humanbronchial epithelial cells. *Carcinogenesis* 1998;19:1867–71.
17. Verma R, Vinoda KS, Papireddy M, Gowda ANS. Toxic pollutants from plastic waste- a review. *Procedia Environ Sci* 2016;35:701–08.
18. Kogevinas M. Human health effects of Dioxins: cancer, reproductive and endocrine system effect. *Hum Reprod Update* 2001;7:331–9.
19. Knutsen H, Alexander J, Barregård L, Bignami M, Brüschweiler B, Ceccatelli S, *et al*. Risks for public health related to the presence of furan and methylfurans in food. *EFSA J* 2017;15:5005.
20. Dallaire F, Dewailly E, Vézina C, Muckle G, Weber JP, Suzanne B, *et al.* Effect of prenatal exposure to polychlorinated biphenyls on incidence of acute respiratory infections in preschool inuit children. *Environ Health Perspect* 2006;114:1301–5.
21. Gibbs BF, Mulligan CN. Styrene toxicity: an ecotoxicological assessment. *Ecotoxicol Environ Saf* 1997;38:181–94.
22. Hecker L. Mechanisms and consequences of oxidative stress in lung disease: therapeutic implications for an aging populace. *Am J Physiol Lung Cell Mol Physiol* 2018;314:L642–L653.
23. Schünemann HJ, Muti P, Freudenheim JL, Armstrong D, Browne R, Klocke RA, *et al*. Oxidative stress and lung function. *Am J Epidemiol* 1997;146:939–48.
24. Balmes J, Becklake M, Blanc P, Henneberger P, Kreiss K, Mapp C, *et al*. Environmental and Occupational Health Assembly, American Thoracic Society: American Thoracic Society statement: occupational contribution tothe burden of airway disease. *Am J Respir Crit Care Med* 2003;167:787–97.
25. Banks D, Bolduc C, Ali S, Morris M. Constrictive bronchiolitis attributable to inhalation of toxic agents. *J Occup Environ Med* 2018;60:90–6.
26. Chen CH, Tsai PJ, Wang WC, Pan CH, Ho JJ, Guo YL. Obliterative bronchiolitis in workers laying up fiberglass-reinforced plastics with polyester resin and methylethyl ketone peroxide catalyst. *Occup Environ Med* 2013;70:675–76.
27. Mazumdar I, Goswami K. Chronic exposure to lead: a cause of oxidative stress and altered liver function in plastic industry workers in kolkata, India. *Indian J Clin Biochem* 2014;29:89–92.