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Diffuse alveolar hemorrhage secondary to plastic fume exposure: A case report

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Abstract

A 31-year non-smoker man, working in plastic making industry for 12 years presented with cough and streaking hemoptysis for 2 days. Computed tomography (CT) of chest showed patchy ground glass opacities with interlobular septal thickening in bilateral lung parenchyma. Fiber optic bronchoscopy (FOB) was done. Sequential lavage was taken which showed progressively increasing hemorrhagic fluid. His diffusion capacity for carbon monoxide (DLCO) was 38.08 mL/mmHg/Mi (126%) predicted on day 2 of admission, 32.36 ml/mmHg/Mi (106%) predicted on discharge and 39.63 mL/mmHg/Mi (130%) predicted on going back to work. He was diagnosed with plastic fume exposure related pulmonary alveolar hemorrhage.

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Introduction

In the era of industrialization, plastic industries have flourished. Being one of the relatively inexpensive polymers, plastic has taken over a significant place in our daily routine as it can be molded to any desired shape. Most of the cases of plastic fumes exposure are undiagnosed. The presentation is initially mild and non- specific. Persistent exposure can lead to devastating manifestations leading to significant respiratory impairment. Plastic fumes have been culprit since long in the etiopathogenesis of various diseases ranging from occupational asthma to diffuse alveolar hemorrhage secondary to capillaritis [1-4]. The poly vinyl chloride (PVC) is toxic and carcinogenic polymer of plastic [5]. Trimellitic anhydride (TMA) is low molecular weight chemical involved in the manufacturing of plasticizers for PVC resins [6]. Workers not using industrial respirators or personal protective equipment (PPE) typically get exposed to these toxic fumes causing diffuse alveolar hemorrhage (DAH) like in our case.

Case Report

A 31-year-old non-addict man, presented with hemoptysis for 2 days. There was no history of fever, atopy, childhood pneumonia or anti tubercular treatment (ATT) in past. There was no history of any medication or drug abuse. He did not give any history of trauma or surgical intervention. There was no significant family history of any disease. On examination his oxygen saturation was 94% at room air, blood pressure was 110/80mmHg, respiratory rate was 20/min and pulse rate 102/min. His respiratory system examination showed bilateral infra-scapular rhonchi. His general examination was normal. Chest radiograph was normal (Figure 1). Complete blood count and routine biochemical investigations were normal. Computed Tomography (CT) thorax showed bilateral ground glass opacities with interlobular septal thickening

(Figure 2). Fiber optic bronchoscopy (FOB) was done. No intraluminal growth seen on bronchoscopy. Sequential lavage was taken which showed progressively hemorrhagic fluid characteristic of alveolar hemorrhage (Figure 3) [7,8]. Bronchial aspirate showed alveolar macrophages positive for hemosiderin. The spirometry on day 2 of presentation showed forced expiratory volume in 1 second (FEV1) - 3.00 L (83%), forced vital capacity (FVC) - 4.67L (109%) and FEV1/FVC ratio of 64.40(79 %). The diffusion capacity of carbon monoxide (DLCO) was 38.08(126%).

Antinuclear antibody (ANA) and double standard deoxyribose nucleic acid (Ds DNA) antibody were negative. Cytoplasmic-anti nuclear cytoplasmic antibody (C-ANCA) and perinuclear antinuclear cytoplasmic antibody (P-ANCA) was 3.88 (N<7) and 4.32 (N<7) respectively. Anti-glomerular basement membrane (GBM) antibody 5.90 U/mL was negative (N=0-20 U/mL). His Non Structural protein 1(NS1) antigen and Dengue IgM and IgG were negative. Enzyme linked immune-sorbent assay for human immunodeficiency virus was also negative. Urine routine microscopy was normal. The two-dimensional echocardiography (2D ECHO) showed normal ejection fraction with no valvular abnormality. No significant abnormality was observed by otorhinolaryngologist, ophthalmologist and dermatologist on their respective consultation.

He was advised to refrain from work, which improved patient's condition: DLCO decreased to 32.36 mL/mmHg/Mi (106%). However the patient went back to work and developed hemoptysis again. Patient was readmitted, after stabilization DLCO increased to 39.63 mL/mmHg/Mi (130%) His serial spirometry with DLCO is given in Table 1. He was managed with steroid and other supportive therapy and complete abstinence from plastic fumes exposure. In subsequent follow up after 3 years he showed improvement in symptoms and no further episode of hemoptysis. He was diagnosed to have diffuse alveolar hemorrhage due to trimellitic dust used in plastic industry.

Discussion

Trimellitic anhydride (TMA) dust/fumes results in clinical syndromes ranging from upper respiratory symptoms or asthma to wheezing and dyspnea due to immunological and irritant phenomenon [9]. After inhalation TMA acts as a hapten joining human albumin to become an antigen (Figure 4). The main etiopathogenesis in diffuse alveolar hemorrhage is the disruption of the alveolar

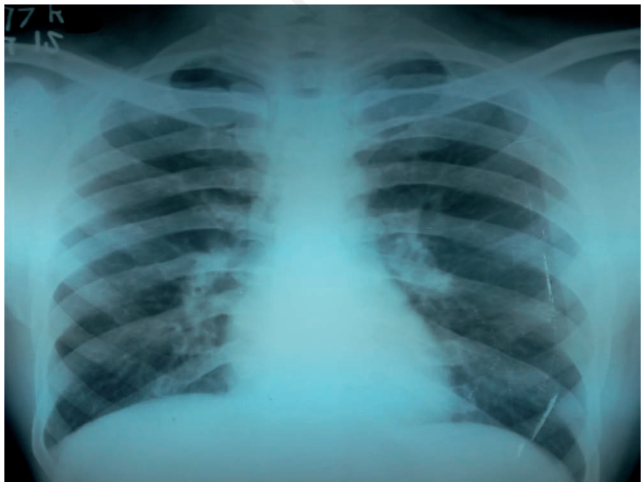

Figure 1. Chest radiograph (PA) view.

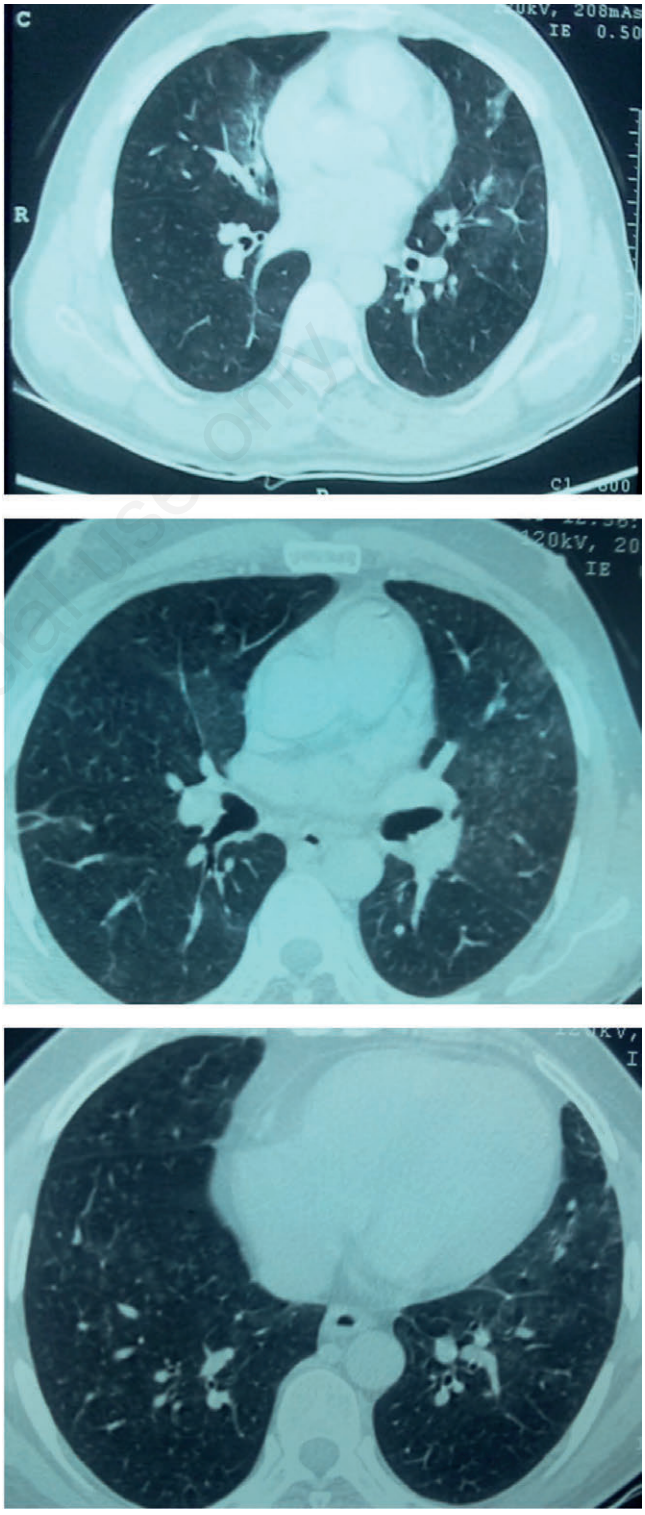

Figure 2. Computed tomography (CT) images showing bilateral ground glass attenuation with interlobular septal thickening.

capillary membrane resulting in entry of erythrocytes in the functional respiratory unit [10]. Clinical manifestations are cough, hemoptysis, dyspnea and diffuse alveolar hemorrhage as seen in our case. In our case, upon re exposure to the culprit agent patient again started having same symptoms with increased DLCO on spirometry providing a contributory evidence to the role of trimellitic dust causing alveolar hemorrhage.

The subject was working in the same factory since last 12 years and was involved in the process of addition of plasticizers in PVC pipes. They are added to PVC to add flexibility. The complete process of manufacturing of PVC pipes includes melting of PVC granules into tubular pipe form after introducing to high temperature, by the process of extrusion follow by addition of plasticizers on heated pipes, cooling and then cutting them according to desired length. Our patient was involved in the process of spraying the plasticizers on PVC pipes since joining of this unit. The spraying modality consists of compounds, having TMA to be sprayed on PVC pipes when they are heated, resulting in inhalation of fumes. Our case presented after acute onset of haemoptysis, following exposure to the fumes after doing his routine duty. The TMA concentration at the work place however, was unknown .

DAH is caused primarily by vasculitis secondary to pulmonary capillaritis. Apart from vasculitis, cases pertaining to drugs, *e.g.* antiplatelets and low molecular weight heparin (LMWH) [11], amiodarone or narcotics abuse like marijuana or cocaine have been described in literature [12-14]. Infective etiology causing DAH includes viruses such as adenovirus, influenza virus, dengue virus, bacterial causes like leptospirosis, mycoplasma and *staphylococcus* and histoplasmosis [15-17]. In our case these causes were ruled out.

Figure 3. Progressive hemorrhagic nature of sequential lavage.

Alveolar hemorrhage secondary to plastic fumes is just tip of iceberg. Many studies have demonstrated its detrimental effect in human life. Other manifestations of plastic fumes include obliterative bronchiolitis, occupational asthma, hearing loss and end stage renal disease (ESRD) due to high exposure to solvents used in plastic industries [18-22]. Recent studies have demonstrated that phthalates which is used in PVC for softening has an impact on cardiovascular and autonomic function. Some reports have also suggested risk of diabetes, obesity, insulin resistance due to exposure to bisphenol-A, a type of plastic [23,24].

Engineering techniques to reduce exposure, at source of generation and use of personal protective equipment (PPE) can only help to diminish the risk of exposure to the occupationally exposed workers. In our case, the PPE were not used, only hand gloves

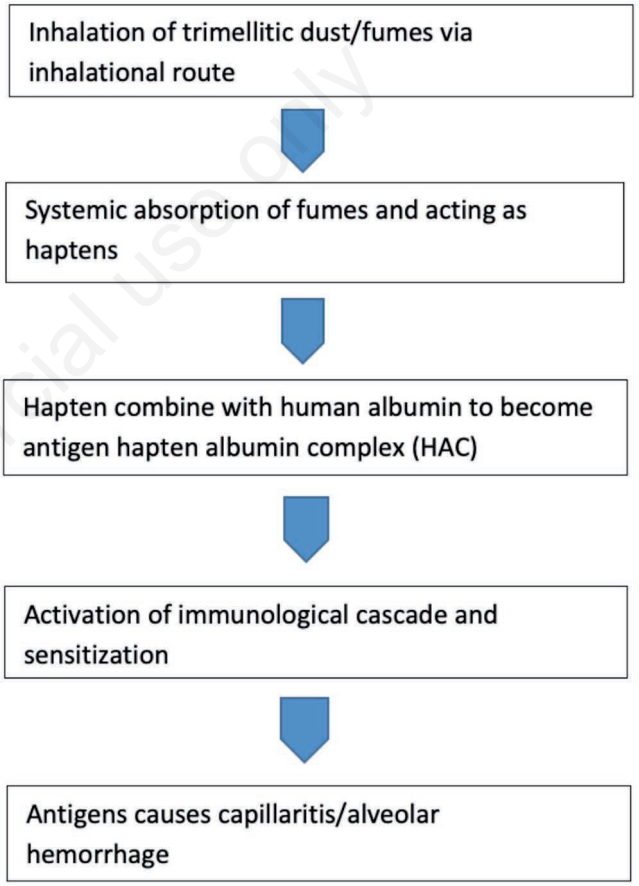

Figure 4. Pathogenesis of Trimellitic dust/fumes in the disruption of alveolar capillary membrane.

Table 1. Sequential spirometry with diffusion capacity of carbon monoxide.

Parameters	On presentation (post 48 h no hemoptysis period)	After abstinence from work for 10 days	48 h of re-exposure
FEV1	3.00 L (83%)	2.94 L (80%)	3.05 L (83%)
FVC	4.67 L (109%)	3.88 L (90%)	3.99 L (92%)
FEV1/FVC	64.40 (79%)	75.87 (93%)	76.42 (94%)
DLCO	38.08 (126%)	32.36 (106%)	39.63 (130%)

DLCO, diffusion capacity of carbon monoxide.

were used and localized engineered control measures to curb the dust/fumes were not installed at the manufacturing unit, unrevealing the glaring reality in plastic industry.

To conclude, plastic exposure is an important cause of alveolar haemorrhage as it is preventable and requires complete abstinence to treat it.

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