*ejpmr, 2019,6(4), 363-365*

**SJIF Impact Factor 4.897**

**EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH**

www.ejpmr.com

Case Report
ISSN 2394-3211
EJPMR

# SOLDERING FUME EXPOSURE AS A CAUSE OF ACUTE HYPERSENSITIVITY PNEUMONITIS

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**Article Received on 24/01/2019** **Article Revised on 14/02/2019** **Article Accepted on 06/03/2019**

## ABSTRACT
Hypersensitivity pneumonitis (HP) or extrinsic allergic alveolitis, is a complex syndrome caused by exposure to a wide variety of organic particles small enough to reach the alveoli (<5 μm). In susceptible individuals, these antigens provoke an exaggerated immune response of the small airways and lung parenchyma. The causative antigens include fungi; bacterial, protozoal, animal or insect proteins; low–molecular-weight chemical compounds etc among others. In this report, we describe the case of a 42 year old healthy gentleman, who developed acute hypersensitivity pneumonitis with respiratory failure following exposure to soldering fumes. He was treated with supplemental oxygen, systemic steroids and supportive measures. After clinical response, he was advised avoidance of further exposure. He remains asymptomatic and under follow up.

**KEYWORDS:** Hypersensitivity pneumonitis, soldering, occupational lung disease.

## INTRODUCTION
Hypersensitivity pneumonitis is the most common interstitial lung disease (ILD) in India, followed by connective tissue disease related ILD and idiopathic pulmonary fibrosis.[1] Hypersensitivity pneumonitis due to exposure to hay, pigeons and molds are frequently reported. Exposure to soldering fumes as a cause of acute HP is rare. We report a 42 year old healthy male, who developed acute hypersensitivity pneumonitis with respiratory failure following exposure to soldering fumes exposure. The presentation of acute HP has significant clinical and radiological overlap with a variety of infectious and non-infectious syndromes. A careful occupational history needs to be elucidated and focused battery of tests needs to be undertaken to rule out closely mimicking conditions. He recovered well with systemic steroids and supportive measures. He continues to fare well as further occupational exposure is avoided.

## CASE REPORT
A 42 year old gentleman, resident of Panachiyam, Kerala was referred to our hospital with the complaints of cough, small amount of mucoid expectoration and shortness of breath for one week. The breathlessness had progressed to grade 4 MMRC for the last 2 days. He denied history of fever, weight loss, chest pain or joint pain. He had no addictions. He was not atopic, had no medical comorbidities or drug intake. No significant illness in the past or in his family was noted. He was a soldering worker for past one year. He used to work for an average of 6 hours a day without any personal protective equipment. He had no exposure to pigeons or hay.

Upon physical examination, he was conscious, alert and afebrile. He was tachypneic with a respiratory rate of 30/minute and had a heart rate of 118/mt. Peripheral blood oxygen saturation of 86% was noted on breathing room air. Chest auscultation revealed bilateral vesicular breath sounds and bilateral fine crackles more pronounced at lung bases. No palpable peripheral lymphadenopathy or pedal edema was appreciated. Cardiac examination was unrewarding except for the presence of tachycardia.

His chest radiograph at the time of admission revealed bilateral extensive alveolar opacities with some basal reticulation.(Fig 1). His laboratory reports showed normal haemoglobin levels and total leucocyte counts and low CRP. Renal, thyroid and hepatic functions were within normal range. Serum IgE levels were in the acceptable range and cardiac biomarkers were negative. Serology for HIV was negative and urinalysis revealed no proteinuria or hematuria. Arterial blood gas analysis revealed type 1 respiratory failure and respiratory alkalosis.

An HRCT Thorax was done which revealed bilateral diffuse centrilobular nodules with ground glass opacities, interstitial thickening and minimal traction bronchiectasis. (Fig 2a and 2b). The provisional diagnoses entertained were Viral or atypical pneumonia**,** acute hypersensitivity pneumonitis**,** pulmonary tuberculosis**,** sarcoidosis**,** pulmonary alveolar proteinosis etc. He was started on antimicrobials (piperacillin-tazobactam, doxycycline and oseltamivir) with supplemental oxygen but exhibited no clinical response even after 48 hours. A bronchoscopy revealed non-inflamed airways and BAL microbial tests returned negative; BAL differential counts revealed 45% lymphocytes. Connective tissue disease serology was unrewarding. Echocardiography showed a structurally normal heart and good cardiac function with mild pulmonary artery hypertension.

Given the history of exposure to soldering fumes and the characteristic radiology, low titres of infection markers, lymphocytosis in BAL and lack of a microbial yield, acute hypersensitivity pneumonitis was settled as the most probable diagnosis. Systemic steroids were initiated which led to rapid improvement in symptoms and oxygenation at 48 hours.

Chest radiograph showed reasonable resolution at 72 hours of steroid initiation. He was discharged on the 5$^{th}$ day after starting steroids and was discharged on oral prednisolone. He remains asymptomatic at 4 weeks post discharge with an oxygen saturation of 97%. Chest radiograph at follow up shows good resolution of shadows. (Figure 3) Spirometry at 3 weeks of follow up reveals a moderate restrictive ventilatory defect. He has been instructed to change his profession and remains under follow up.

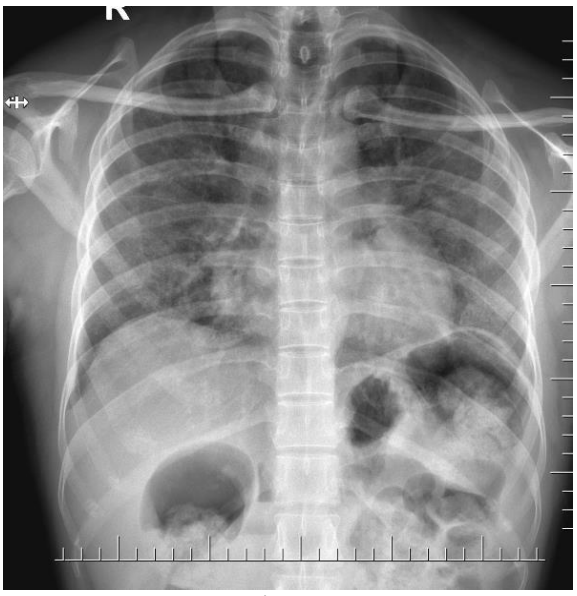

**Figure1**

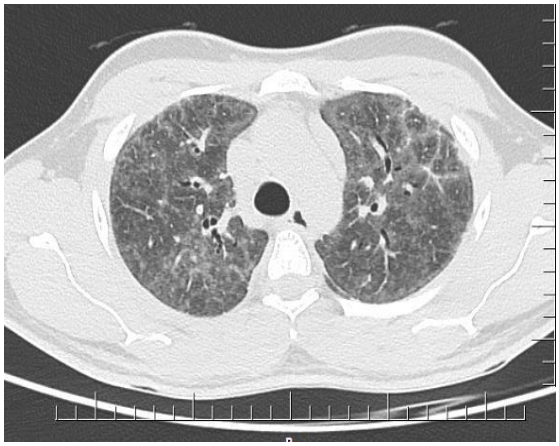

**Figure2a**

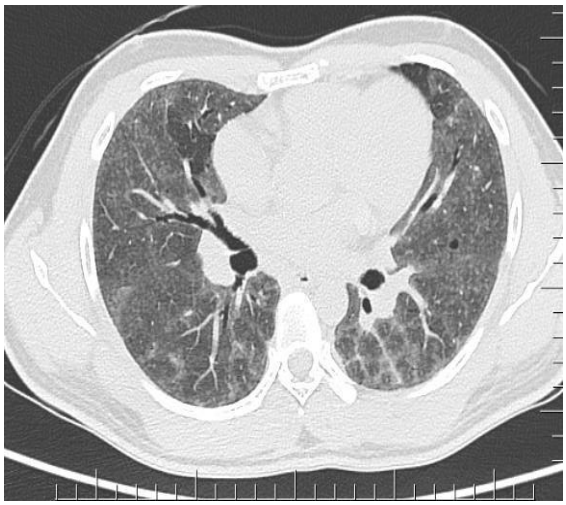

**Figure 2b**

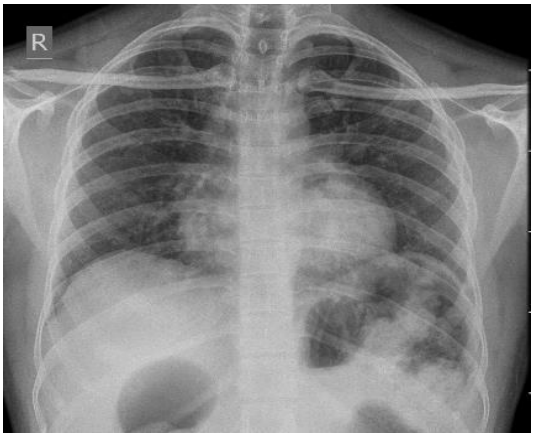


**Figure 3**

## DISCUSSION

Awareness of hypersensitivity pneumonitis (HP) as a unique disease entity dates back to the 18th century.[2] Since then, numerous inciting agents have been attributed to inducing HP, and the pathogenesis of the disease has now been better elucidated.[2,3] Medical progress has been forthcoming in imaging, bronchoalveolar lavage (BAL) study, and pathology since the original classification of HP, and these progresses has been variably incorporated into clinical

practice.

Acute HP in soldering workers has been rarely reported. Soldering is a process in which two or more items (usually metal) are joined together by melting and putting a filler metal (solder) into the joint, the filler metal having a lower melting point than the adjoining metal. In electronics assembly, the eutectic alloy of 63% tin and 37% lead (or 60/40, which is almost identical in melting point) has been the alloy of choice. The charcoal is used as a flux in soldering, the purpose of which is to facilitate the soldering process which helps in reducing the impurity at the site of the joint. Other metals that can be found in solders include cadmium, silver, copper, nickel, zinc, arsenic, beryllium, antimony, indium, and bismuth. The colophony of soldering flux contains 90% resin acid and other 10% includes various hydrocarbons.

Occupational asthma has been reported in soldering workers. Work related respiratory symptoms in persons engaged in soldering have been attributed to the fumes from colophony, which is the main constituent in the electronic soldering flux and lead fumes. The patient's history can be supplemented by Material Safety Data Sheets (from the employer or poison-control centers), which list the chemical components of commercial materials. The results of previous measurements of air from the patient's work area may be obtained[5] or the physician may advise the employer to consult a certified industrial hygienist for air measurements and remediation.[6] The clinical syndrome develops only in a small minority of exposed workers. This may be accounted by polymorphisms in genes leading to variation in the rate and pathway of metabolism, which in turn results in marked differences in susceptibility to occupational substances among workers. Interactions among occupational exposure, atopic predisposition, nutrition,[7] home and host factors (such as gastroesophageal reflux, cigarette smoking, and viral infections) are plausible, and they help to explain the occurrence of disease in certain persons.

Because of the high risk of worker/operator exposure during soldering, OSHA suggests ventilation as a key to controlling lead exposures. Ventilation, either local or dilution (general), is probably the most important engineering control available to the safety and health professional to maintain airborne concentrations of lead at acceptable levels. If a local exhaust system is properly designed, it will capture and control lead particles at or near the source of generation and transport these particles to a collection system before they can be dispersed into the work environment.[8] Source capture also works effectively for soldering that does not involve lead-based solder. To prevent the inhalation and ingestion of lead in soldering fume exposure, use of protective face masks is advocated; hands should be washed with soap and water before work-breaks and lunch interval, at completion of soldering efforts, and at the end of the workday. Work areas should be kept clean and wiped with a damp paper towel to minimize the accumulation of lead dust in the work area. Food is not permitted in laboratory work areas.[9]

## CONCLUSION

Exposure to soldering fumes can rarely result in HP. Early identification and avoidance of further exposure coupled with supportive management can reverse acute respiratory compromise; prevention of subsequent exposures is the key to avoid recurrence. Adequate workplace safety measures like proper ventilation, fume extraction system and personal protection devices are needed to prevent this occupational lung disease.

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