

Case report**Is my profession a risk for my lungs? - Unusual case of Hypersensitivity Pneumonitis.****Mohammad Ayaz Khan^{1,2,3}, Sarah Alrasheed³**¹College of Medicine, King Saud University for Health Sciences, Saudi Arabia²King Abdullah International Medical Research Centre, Saudi Arabia³Department of Medicine, Division of Pulmonary, Ministry of National Guard-Health Affairs, Saudi Arabia**Abstract**

Hypersensitivity pneumonitis (HP), previously known as extrinsic allergic alveolitis, is caused by inhalation of various antigens that lead to diffuse parenchymal lung infiltration of the interstitium, alveoli, and distal bronchioles. Currently, more than 200 inciting antigens have been identified as causative antigens. Dyspnea and cough are common symptoms of HP. The diagnosis is made based on clinical presentation, imaging findings, and bronchoscopic findings in such cases. The fundamental principle of management is to remove the causative allergen. This case report describes an unusual cause of HP in a surgeon secondary to surgical smoke exposure.

Case Report

A 68-year-old male was referred to the pulmonary clinic with progressive shortness of breath (SOB) for the last six months. The SOB has worsened in the two months prior to his presentation. His SOB was exertional, especially when he climbed stairs. He had undergone an extensive workup for cardiac causes, but the results were negative for such causes. The patient never smoked and reported no industrial exposure, pets, or pigeons at home. No other exposure history to any organic or inorganic agents was reported based on a detailed structured questionnaire. No symptoms suggestive of autoimmune disease were present. His medical history included diabetes mellitus (DM), hypertension (HTN), and dyslipidemia. He had no previous hospital admissions or exposures to drugs or radiation. No family history of any lung diseases was reported. No hobbies that would risk any other types of exposure were reported. The patient worked as a plastic surgeon and had been exposed to surgical smoke for more than 30 years on a daily basis. In his situation, this type of smoke was generated by surgical cautery using electric probes and debridement while dealing mostly with burn patients. He reported exposure to surgical smoke every day for an average of five hours per working week. Medication history included gliclazide, metformin, and liraglutide for DM. He took amlodipine and atenolol for HTN, and rosuvastatin for hypercholesterolemia. His nails were clubbed, and a chest examination revealed bilateral inspiratory squeaks and fine crackles at the lung bases.

At this stage, a differential diagnosis included interstitial lung disease, heart failure, and airway diseases. His basic labs values were within normal range, apart from high glycosylated hemoglobin (HbA1c) and lactate dehydrogenase (LD). A detailed connective tissue and autoimmune disease serological work up was negative. An echocardiogram showed a normal left ventricle size, mild concentric left ventricular hypertrophy, a normal left ventricular systolic function, ejection fraction of > 55%, impaired LV relaxation, and a normal right ventricle size and function. His pulmonary function test (PFT) results (All % predicted) confirmed the presence of restrictive pattern with reduced diffusion capacity for carbon monoxide (DLCO) of 62.5%, forced vital capacity (FVC) of 56.4%, forced expiratory volume in 1 s (FEV1)/FVC ratio of 86.06%, total lung capacity (TLC) of 61.8%, and residual volume (RV) of 71.3%. He was able to complete 312 m during the 6 min walk test (6MWT) with significant desaturation and nadir of 86% from an initial value of 96%.

He had undergone a chest x-ray four years ago for another reason before his current presentation. This previous X-ray showed bilateral lower zones with a faint reticulonodular shadow.

He underwent a high-resolution chest tomography (HRCT), which showed diffuse ground glass opacities, micronodules, mosaic attenuation, and areas of air trapping (Figures 1A and 1B).

**Figure.1A:** High-resolution CT thorax showing upper lobes.

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Figure 1B: HRCT shows diffuse ground glass opacities, micro nodules, mosaicattenuation and areas of air trapping..

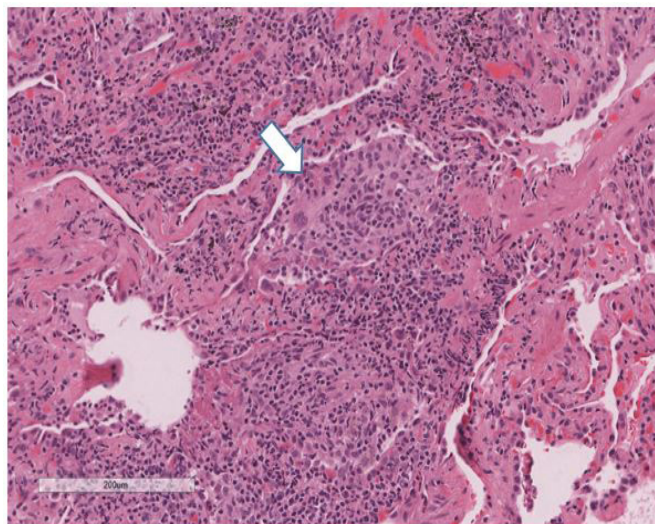


Figure 2C: Ill defined aggregate of epithelioid histiocytes, ill defined granuloma (arrow).

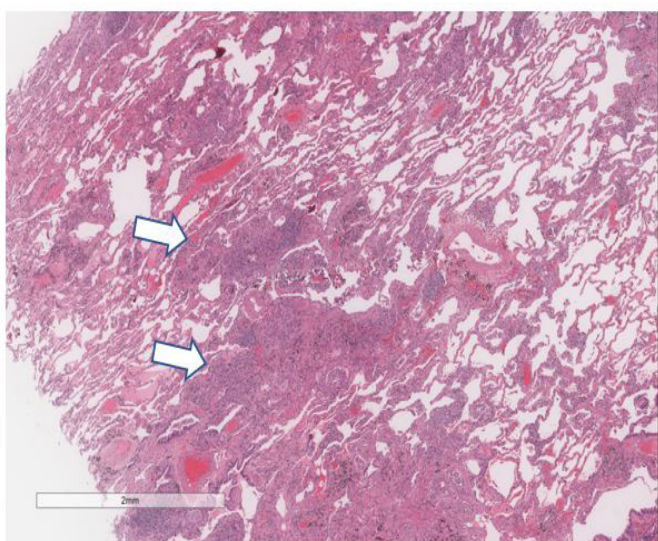


Figure 2A: Low power showing the centrilobular distribution of the inflammation (arrows).

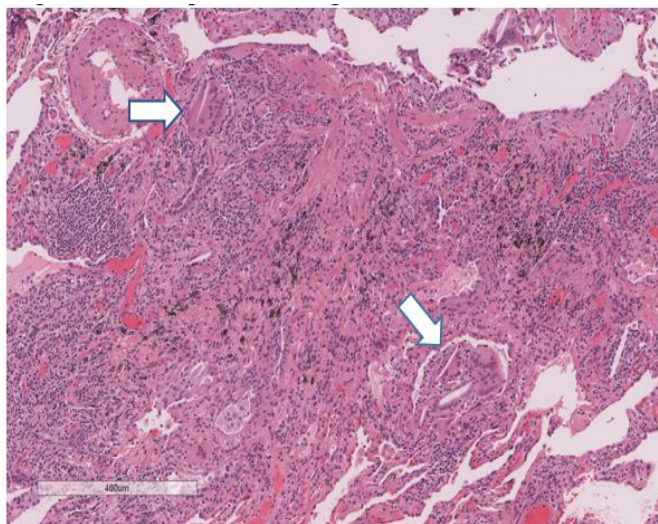


Figure 2B: The inflammatory cells consist mainly of lymphocytes with scattered giant cells (arrows).

Serum immunoglobulin IgG was negative for mold mix, weed and grass pollen mixes, house dust, epidermal and animal proteins, chicken feathers, and pigeon droppings..

The patient underwent a bronchoscopy, and bronchoalveolar lavage (BAL) and a biopsy were obtained. Microbiological results were obtained from negative for infectious causes, including fungus, mycobacterial tuberculosis polymerase chain reaction (PCR), and acid-fast bacilli. Cell counts showed lymphocyte predecessor of 57% (normal average, 10%–15%) and 8% eosinophil (normal average, 1%). Bronchoalveolar lavage cytology was negative for malignant cells and infectious agents (acid-fast bacilli, fungus, and pneumocystis).

The interstitial lung disease multidisciplinary team meeting (ILD-MDT) discussion led to the decision that the patient should undergo a surgical video assisted thoracoscopic biopsy to clarify the diagnosis. Surgical lung biopsy showed diffuse inflammatory cells, mainly consisting of lymphocytes with scattered giant cells and ill-defined aggregate epithelioid cells with ill-defined granuloma (Figure 2 A, B and C).

The patient was advised to avoid surgical smoke exposure by changing role in his department and wear proper fine particulate avoidance respirator mask. Since he was close to his retirement, he stopped his job to avoid any further exposures. In view of his symptoms and lung function defects he was started on pulse IV methylprednisolone followed by oral corticosteroids prednisolone 40 mg OD that was tapered over next few months. He felt much better and his PFTs improved with an improvement in his repeat CT scan. He was started on oral mycophenolate that was gradually increased. His clinical follow up with PFTs and HRCT showed stability of his underlying CHP.

Discussion

Hypersensitive pneumonitis (HP) was first described in 1932 after an outbreak of illness in 10 employees of company that railroads ties [2]. Later, numerous occupations and hobbies have been associated with an increased risk of HP.

In the medical profession, HP was reported only in dental technicians after exposed to methyl methacrylate (MMA) and hard metal dust during work [3].

Although several studies discuss the potential hazards of surgical

smoke, especially for the respiratory system, it was first reported in 1985 by National Institute for Occupational Safety and Health (NIOSH) in United States.

Surgical smoke contains 95% water and 5% cellular debris [4]. The amount and the content of smoke determined by the targeted tissue [5] in addition to the type of imparted energy (such as electrocautery versus ultra-sonic scissors [6]).

The size of the generated particles depends on the type of cautery machine. Electrocautery has the smallest plume sizes with diameters around 0.7 μm , which affects the staff in an operating room even at a wide distance, and it can also affect the non-scrubbed staff. Potential hazards are caused by chemical-generated materials, such as hydrocarbons, nitriles, fatty acids and phenols [8], and these materials might also contain infectious materials such viruses or bacteria, especially in laser procedures or ultra-sonic scalpel use [9]. These materials cause a numerous of adverse effects as reported by Alp et al. (7), which include sneezing, throat irritation, and acute and chronic inflammatory changes in respiratory tract (emphysema, asthma, chronic bronchitis). Pulmonary fibrosis has been shown in a rat model.

To minimize exposure to surgical smoke, the Centers for Disease Control and Prevention (CDC) recommends providing smoke evacuation systems during procedures at a minimum of 100 to 150 feet [10]. The CDC and National Institute for Occupational Safety and Health (NIOSH) suggest personal protective equipment, such as N95 or N100 respirators, for adequate protection (10).

Interstitial pneumonia, bronchiolitis, and emphysema were reported in animals' modules after exposure carbon dioxide (CO₂) laser smoke [11]. In this case, surgical smoke would be the most likely causative agent responsible for chronic HP (CHP). While evaluating the underlying causes for CHP in suspected individuals, this cause should also be kept in mind and appropriate enquiry about the occupation should be made.

Conclusion

In conclusion, we are presenting a case of CHP where extensive search and investigations have led us to believe that surgical smoke is the most likely culprit in a surgeon with long history of this particular exposure. His clinical and radiological improvement after avoidance and treatment further strengthen this belief. Human case of CHP due to surgical smoke has not been described before in scientific literature. We propose this cause should be kept in mind while screening for potential causes of CHP in relevant professions.

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