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CASE REPORT



Hypersensitivity pneumonitis in a slaughterhouse worker: A case report

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ABSTRACT

This case report describes a 60-year-old male, who presented to the Respiratory Outpatient Unit due to dyspnea on exertion and persistent dry cough, worsened during the preceding 6 months. He was nonsmoker with an otherwise unremarkable medical history and had been working in a sheep/goat slaughterhouse for the last 25 years. He recalled a number of episodes of flu-like symptoms in the past that subsided without any specific treatment. Given the compatible occupational history, the radiologic pattern in chest High-Resolution Computed Tomography and the Bronchoalveolar Lavage subpopulation analysis, hypersensitivity pneumonitis was diagnosed, and the patient was advised to leave temporarily his current occupational activity. Four weeks later, clinical and functional improvement was observed. A permanent job change was subsequently suggested, and sustained improvement was confirmed during his follow-up at 3 months.

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Hypersensitivity pneumonitis; occupational disease; occupational exposure; occupational respiratory disease; slaughterhouse worker

Background

Working in a slaughterhouse involves exposure to a significant number of biological hazards. A broad range of contaminants, such as bacteria and fungi, can be released during different processes of slaughtering while removing head hair or cleaning the colon of the animals. These contaminants can be the major cause of non-allergic inflammation in the respiratory system.^{1,2} Indeed, various respiratory disorders have been previously reported among sheep-shearing workers, exposed to microbial air contamination, namely, chronic bronchitis, asthma, and hypersensitivity pneumonitis (HP).³ The latter, also called *extrinsic allergic alveolitis* (EAA), is an interstitial lung disease (ILD) with a wide spectrum of clinical manifestations, as well as a varying intensity and natural history. Occupational and environmental exposures play a major role in its pathogenesis.

This is a presentation of a case of a middle-aged worker in a sheep/goat slaughterhouse with persistent dry cough and progressive dyspnea on exertion over the last six months. Prior to publication, written consent has been requested and provided by the patient.

Case presentation

A 60-year old, white/Caucasian, male presented to the Respiratory Outpatient Unit of our Institution, with progressively worsening dyspnea and nonproductive cough over the preceding 6 months. On presentation, the patient was alert, oriented and tempo-spatially collaborative. He was hydrated and afebrile, with a respiratory rate of 20 breaths per minute, blood pressure 135/75 mmHg and hemoglobin oxygen saturation (SaO₂) 95% while breathing room air. Pulmonary auscultation revealed bilateral, fine “Velcro” crackles while the cardiac auscultation and abdominal examination were normal. He had no palpable lymphadenopathy or peripheral edema, and rest of physical examination was normal. His medical history included only a number of episodes of flu-like symptoms that subsided without specific treatment in the past. A detailed occupational history was obtained, where he mentioned that he has been working in a sheep/goat slaughterhouse for the last 25 years. He did not have a pet and he did not report any hobbies or contact with animals in his free time, although he lived in a rural setting with his elderly mother. He was nonsmoker but reported a moderate consumption of alcohol.

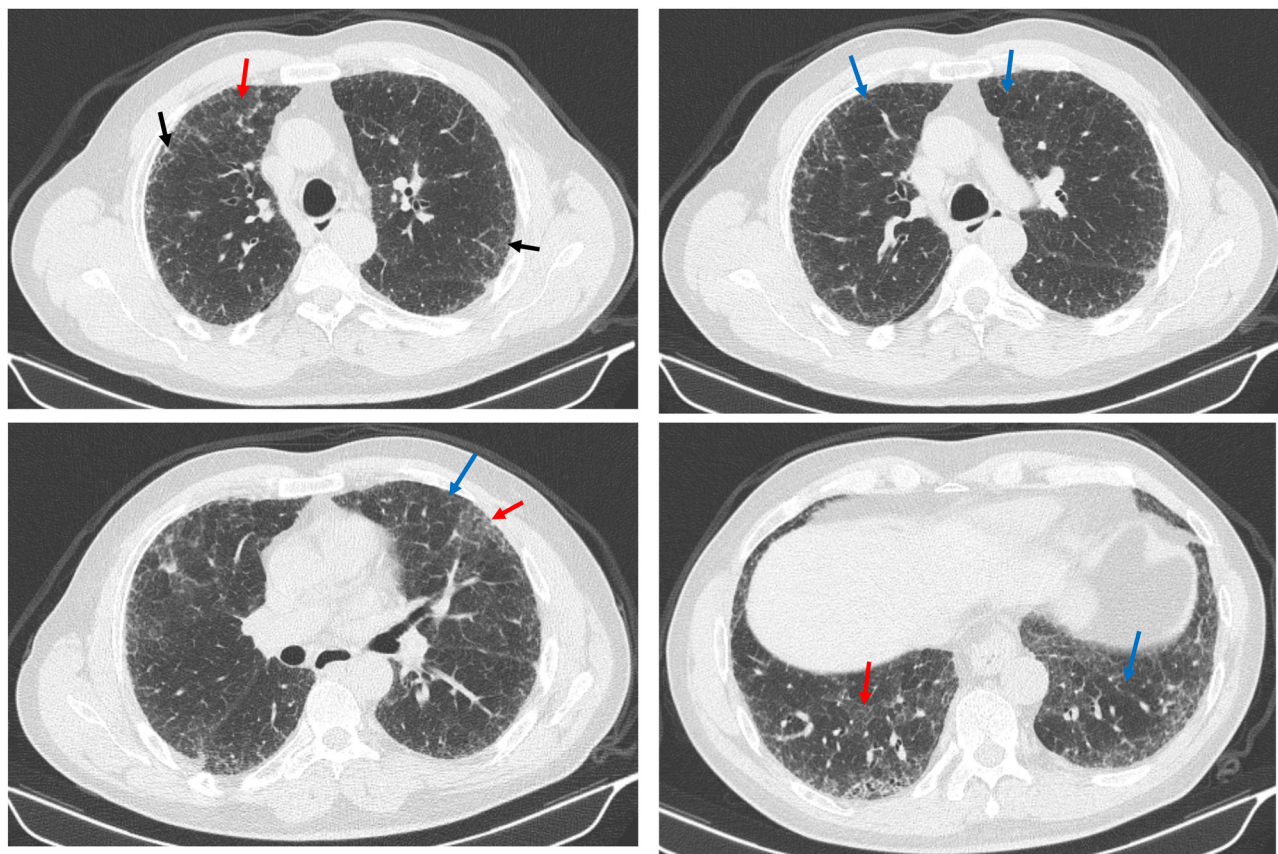


Figure 1. Chest HRCT scans, depicting sub-pleural reticular opacities (black arrows), areas of increased attenuation (ground glass, red arrows) and areas of decreased attenuation (air trapping, blue arrows) without zonal predominance.

Pulmonary Function Tests (PFTs) were performed according to the 2019 ATS/ERS statement on standardization of spirometry⁴ and revealed a restrictive respiratory disorder (Forced Vital Capacity – FVC: 60% predicted; Total Lung Capacity – TLC: 63% predicted; Diffusion Capacity for Carbon Monoxide – DLCO: 50% predicted). Six-minute walking test (6MWT) distance walked was 460 m (75.06% of predicted distance of 612.8 m). Chest High-Resolution Computed Tomography (HRCT) was typical for HP, demonstrating bilateral, sub-pleural reticular opacities, ground glass opacities and areas of decreased attenuation, representing air trapping. There was no zonal predominance of the distribution of these lesions.⁵ Honeycombing was absent. (Figure 1).⁶ All serologic tests were negative for auto-immune disorders. An extended panel for specific IgG antibody testing of most common antigens, including *Aspergillus fumigatus*, *Micropolyspora faeni*, *Thermoactinomyces vulgaris* was negative. A bronchoscopy with Bronchoalveolar Lavage (BAL) was performed and BAL fluid analysis demonstrated a lymphocyte predominance (60%).

A diagnosis of Hypersensitivity Pneumonitis (HP) was made with high confidence (positive exposure,

BAL lymphocytosis and typical findings in HRCT) according to the ATS/JRS/ALAT criteria.⁵ Since exposure at the occupational setting was considered responsible, he was instructed to abstain completely from work. At his follow-up, 3 months later, he reported elimination of his symptoms. In addition, PFTs and 6MWT were improved (FVC: 70% predicted; TLC: 67% predicted; DLCO: 58% predicted; distance walker during the 6MWT: 510 m). He was advised to consider a permanent occupational change.

Discussion

To the best of our knowledge, this is the first report in the literature of a previously unknown occupational exposure that led to the development of HP. Work in a slaughterhouse involves exposure to a number of biological hazards as a result of the interaction with animals, predisposing the slaughterhouse workers to the development of respiratory disorders.⁷ A wide range of “bioaerosols” including bacteria and fungi that are produced during slaughtering can be the major cause of non-allergic inflammation of the lung, as it is the case in HP.

HP is an ILD that represents an excessive, non-IgE-mediated immunological reaction to a variety of organic materials or low molecular weight inhaled agents in occupational settings or the environment. Although its actual burden in the general population is uncertain, HP is considered a rare and rather underdiagnosed condition. Variations in the prevalence estimations are due to disparities upon case definitions, severity of exposure to inciting antigens, seasonal and geographical variability, local practices and customs, proximity to industrial zones, and host factors.^{8–10} According to a recent analysis of a large healthcare database in the US, with 150 million participants, the one-year prevalence rate of HP ranged from 1.67 to 2.71 per 100,000 persons for participants under the age of 65. An 11.2 per 100,000 persons rate was noted for those aged 65 and older, with 7,498 cases being identified in total.¹⁰

In the past, HP was classified as acute, subacute or chronic depending on time from exposure to clinical manifestations. This categorization is under scrutiny since subacute form is difficult to define and is usually overlapping with the other two forms.¹¹ The acute is the most common form of presentation and usually develops within 4–8 hours of an intense exposure to the offending antigen. It manifests with flu-like symptoms such as fever, malaise, chills, cough, dyspnea and myalgia that automatically subside after 1–3 days.¹² Chronic HP is characterized by breathlessness on exertion and dry cough. Differential diagnosis embraces the large spectrum of disorders that cause ILDs, especially IPF. According to a previous study, in almost half of the patients that were initially diagnosed with IPF, the diagnosis changed to chronic HP after careful reevaluation.¹³ Most of the cases were attributed to exposure to avian antigens, highlighting the importance of meticulous assessment of the occupational and environmental exposures.¹³

The diagnosis of HP is challenging since there is no gold standard test and therefore it relies on a combination of detailed history of occupational and environmental exposures, diagnostic tests and high index of clinical suspicion. Both the clinical and the occupational history are key to the diagnosis. Importantly, BAL should be performed in all patients with suspected HP, since a high lymphocytes count (>50%) is highly suggestive of HP, while a lymphocyte count of <30% makes the diagnosis unlikely.¹⁴ A recent statement by the American Thoracic Society (ATS), Japanese Respiratory Society (JRS) and Asociación Latinoamericana de Tórax (ALAT) represents a first collaborative attempt to develop a systematic approach

for the diagnosis of HP.⁵ Although clinical presentation of HP does not differentiate according to an occupational or another environmental cause, the timing of symptoms may differ. In acute episodes, the symptoms occur 4–8 hours after the exposure and usually resolve in 1–3 days after the exposure cessation. Therefore, when triggered by a workplace antigen, acute symptoms typically begin toward the end of a work shift or following a work shift and improve near the end of a weekend off work. In contrast, chronic HP related to a workplace antigen may have no clear pattern related to short periods off work.

HP's work-relatedness is well-established throughout history. The first account of the disease, dating back to 18th century, was described by the Italian physician Bernardino Ramazzini in grain workers in 1713.¹⁵ In the beginning of 20th century, the most common form of the disease known as "farmer's lung" was acknowledged.¹⁶ Currently, there are over 300 known occupational exposures responsible for developing HP. Due to the improvement of work practices, some causes, such as farmer's lung have markedly declined, while other, new agents/exposures are emerging.¹⁷ Metal working fluids (MWF), water-aerosolized procedures produced by air-conditioners, humidifiers and water-purifiers have been increasingly implicated as causative agents of HP.^{17,18} Laboratory workers, furriers, veterinarians are among the occupations struck by HP related to animal handling. Awareness of new occupational, causal agents is prerequisite for clinicians to suspect possible causes of HP.

Although occupational history of animal handling is a well-known cause of HP, little is known concerning abattoir workers. Since the body of literature referring to the slaughterhouses focuses mainly on foster-keeping animals and the animals' health, its contribution to occupational health is ambiguous.^{19,20} A study of prevalence estimation of respiratory diseases among 81 slaughterhouse workers with pre-shift and post-shift lung function tests revealed a high predisposition of slaughterhouse workers for lung diseases.⁷ In addition, in the same study samples of "bioaerosols" revealed a high burden of bacterial and fungal species with a known respiratory pathogenicity such as *Staphylococcus epidermidis*, *Staphylococcus aureus*, *Penicillium* sp., *Aspergillus flavus*, *Aspergillus fumigatus*, and *Aspergillus niger* respectively.⁷ *Aspergillus* spp. were also detected in slaughterhouses in Portugal.²¹ Another study of environmental sampling in slaughterhouses reported high fungal burden on the floor, the surfaces, and in the air of the

slaughterhouses.^{2,22} Since the majority of studies refer to poultry and cattle slaughterhouses, the need for similar studies in sheep/goat settings is imperative.

The cornerstone of the management of HP is removal from exposure to the eliciting antigen, as early as possible, although deterioration may continue even after avoidance of exposure to the causal agent. The prognosis of chronic HP largely depends upon the extent of permanent lung damage at the time of diagnosis.¹¹ In the small percentage of patients with advanced pulmonary fibrosis, progression of their disease may continue even though they completely avoid re-exposure to the inciting antigen, for example change in the occupational setting.^{11,12} These patients usually develop severe, chronic hypoxemia and may require oral corticosteroid treatment.¹¹

In our case, although the patient had a typical clinical, radiological and BAL pattern of HP, a well-known, identifiable stimulus that would immediately explain these findings was lacking. Rather than considering an alternative diagnosis, a thorough occupational history was decisive for the diagnostic process. The clinical and radiological presentation as well as the improvement of patient's symptoms after withdrawal from the occupational setting confirmed the diagnosis.

Conclusions

Hypersensitivity pneumonitis is a rather uncommon, underdiagnosed disease that requires a high index of clinical suspicion and knowledge of potential causes, such as occupational exposures. Awareness of alarming symptoms, as well as periodic evaluation of the respiratory system is a prerequisite for early detection of HP and other lung diseases that have an association with occupational exposures. Prompt referral to a respiratory unit for further investigation, as well as immediate removal of the worker from the harmful environment can aid in the confirmation of the diagnosis in the absence of identifiable cause or negative precipitin tests. In addition, it is our suggestion that an occupational physician is included in the Multidisciplinary Discussion (MDD) for unidentified ILDs.

Conflict of interest

The authors declare no conflicts of interest, and the work was not supported or funded by any drug company.

Ethical statement

This work received no funds.

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