

INTRODUCTION

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Hypersensitivity pneumonitis has been known as an occupational disease for well over two centuries, but it was only recently recognized as an allergic disease (*Stites et al., 1997*).

Hypersensitivity pneumonitis is defined as an allergic disease of the lung parenchyma with inflammation in the alveoli and interstitial spaces induced immunologically by acute or chronic inhalation of a wide variety of inhaled materials (*Fuller, 1992*). The disease may present as an acute, subacute, or chronic form. It is not currently possible to describe all features of the illness to a single immunological mechanism. There is evidence for several different immune pathways that operate separately or concurrently, but the most compelling evidence favors allergen-specific cell mediated hypersensitivity as the mechanism of pathogenesis (*Fink, 1992*). Interstitial pneumonitis is the primary clinical manifestation for all forms of the disease (*Sharma, 1995*).

It was first thought to be a pulmonary arthus reaction caused by immune complexes of inhaled allergen and precipitating IgG antibodies, but persuasive evidence from clinical, pathologic, epidemiologic, and experimental studies has shown that the disease is mediated predominantly by T-lymphocyte (cellular) effector mechanisms (*Zenz, 1994*).

It shares some pathologic features with sarcoidosis and the pneumoconiosis, but it differs from the former disease by having a

recognized environmental cause and from the latter group of diseases by the immune responses to inhaled material (*Reynolds, 1992*).

Like allergic asthma, it is produced by inhaled allergens, and in fact there are allergens that can cause either disease. IgE antibodies, however, play no known role in the pathogenesis of hypersensitivity pneumonitis (*Stites, 1997*).

According to the epidemiology of hypersensitivity pneumonitis cases have been reported worldwide. The disease is most frequently associated with occupational allergens, which determine its prevalence and geographic, age, and sex distribution. According to the age workers 30-50 years are usually affected. Sex males more affected. Geographic distribution damp organic material and standing water provide ideal condition for the amplification and proliferation of microbial antigens Summer-type has been identified as the most prevalent form of disease (*Salvaggio, 1990*). Farmer's lung, the prototype and most widely reported form of hypersensitivity, is caused by thermophilic actinomycetes, usually from warm, moist, moldy hay and therefore the disease predominates in wet regions and especially among dairy farmers (*Novey, 1993*).

Several surveys suggest that 2-4% of farmers have been affected. Bird handler's disease (also known as bird fancier's lung, pigeon breeder's disease, and bird breeder's disease) has been diagnosed in 15-21% of exposed individuals (*Stites et al., 1997*).