Organic antigen-induced interstitial lung disease: Diagnosis and Management

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BACKGROUND: Traditionally, chronic idiopathic interstitial pneumonia/fibrosis (IIP/F) had included usual interstitial pneumonia, desquamative interstitial pneumonia, and nonspecific interstitial pneumonia (NSIP). More recent classifications have included bronchiolitis obliterans-organizing pneumonia (BOOP), respiratory bronchiolitis-associated interstitial lung disease, and acute interstitial pneumonia. Some chronic eosinophilic pneumonias (CEP)/pulmonary infiltrate with eosinophilia (PIE) have obvious causes, but many lack an identifiable etiology. We felt that hypersensitivity pneumonitis (HP) was being underdiagnosed and was hidden within this large heterogeneous group of interstitial lung disorders of unrecognized cause.

OBJECTIVE: We sought to prove that detailed environmental histories and investigations would reveal causative contaminations in the home or workplace of some patients with idiopathic interstitial lung disease and remediation of the contamination would stabilize the disorder.

METHODS: Consecutive cases of IIP/F were investigated. Patients were identified by compatible signs and symptoms, roentgenographic studies, pulmonary function tests, and lung biopsies. They were further evaluated with detailed environmental histories, serologic tests, and investigation into the suspected causative environment. Environmental and specific antigen challenges were done in some cases. Remediation of contaminations or moving into another environment were the methods used as therapy.

RESULTS: Eighty-six consecutive patients with IIP/F were evaluated. Twelve patients were subsequently diagnosed with specific causes for interstitial lung disease. Fifty-seven of 74 patients were identified by clinical evaluation and lung biopsy with HP, CEP/PIE, NSIP, BOOP, UIP, and nonclassifiable morphologic patterns. Seventeen patients were not biopsied or had an inadequate transbronchial biopsy but had consistent findings radiographically and clinically of idiopathic interstitial lung disease. Contamination of the home was causative in 69 of 74 and the workplace in 3 of 74 cases. There were 9 positive and 33 negative environmental challenges with 4 positive and 1 negative specific challenges. Fifty of 74 (67%) patients are receiving no treatment and are free of active disease after remediation of the environmental contamination, with a mean survival of 8.2 years.

CONCLUSIONS: Our data show that UIP, BOOP, NSIP, CEP/PIE, and nonclassifiable morphologic patterns represent a spectrum of interstitial lung disease that may be caused by inhalation of organic dusts in the home or workplace as described with HP. Remediation of, or moving from the contamination, can lead to arrest of the active inflammatory process and stability of the lung disorder.