Distinct Characteristics Of Bronchiolar Disorders Related To Occupational And Environmental Exposures

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Rationale:
Bronchiolar disorders encompass a spectrum of diseases involving the small airways (<2 mm in diameter). Pathology may be limited to the bronchioles or also involve other components of the respiratory tract, including the alveoli. Etiologies include infection, immunologic mechanisms, and inhalational exposures; many cases are categorized as idiopathic. We sought to determine whether bronchiolar disorders related to occupational and environmental exposures could be distinguished from other causes on the basis of clinical, functional, and radiographic findings and clinical course.
Methods:
We searched PubMed for articles on bronchiolar disorders using the following terms: “bronchiolitis,” “cellular bronchiolitis,” “bronchiolitis obliterans,” “obliterative bronchiolitis,” “constrictive bronchiolitis,” “proliferative bronchiolitis,” “bronchiolitis obliterans organizing pneumonia (BOOP),” and “organizing pneumonia.” We also consulted textbook chapters on bronchiolar disorders and the reference lists of identified information sources. Case reports, case series, and epidemiologic investigations were included. Although hypersensitivity pneumonitis often has bronchiolar involvement, we considered it to be a separate entity and excluded it from our review.
Results:
We included 146 information sources, the majority of which were original reports in peer-reviewed journals. A wide range of gaseous, volatile, and particulate exposures has been reported to result in bronchiolar disorders, including diacetyl, methyl isocyanate, nitrous oxide, nylon flock, sulfur mustard, textile dye, World Trade Center dusts, and humidifier disinfectant. The symptoms of exposure-related bronchiolar disorders are generally non-specific respiratory complaints: dyspnea, particularly with exertion; cough; and in some cases, wheeze. Reported functional abnormalities include obstruction and restriction with or without impaired diffusing capacity. HRCT findings include centrilobular nodules, ground glass opacification, and mosaic attenuation. However, recent histopathologic series have demonstrated bronchiolar disease in the face of normal lung function and/or normal imaging. Biopsy has demonstrated classic features of known histopathologic entities with some exposures and novel patterns with others, and highlighted a spectrum of responses to a particular exposure in some series. Exposure-related bronchiolar disorders including BOOP appear to be less responsive than their non-exposure-related counterparts to therapies like steroids. However, exposure cessation has been found to halt progression of disease and, for some exposures, leads to recovery.
Conclusions:
Clusters of bronchiolar disorders in workplaces and communities have identified new etiologies, drawn attention to indolent disease that may otherwise have been categorized as idiopathic, and expanded the spectrum of histopathologic responses to an exposure. Cessation of exposure is the most important therapeutic measure and may result in improved outcomes compared to bronchiolar disorders unrelated to exposures.

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