Adiaspiromycosis

An Uncommon Disease Caused by an Unusual Pathogen

Adiaspiromycosis is a pulmonary disease caused in humans by the fungus *Chrysosporium parvum var crescens* (*Emmonsia crescens*), a soil saprophyte of cosmopolitan distribution. The disease was first described in Arizona rodents by Emmons and Ashburn in 1942, and the first human case was reported from France in 1964. Subsequently, human infection was reported from Czechoslovakia, where most of the work characterizing this disease and its etiologic agent has been done, and from the USSR, Central America, and Venezuela. Although adiaspiromycosis occurs in rodents and other small mammals throughout the world, no autochthonous human cases have been reported from the United States.

The term “adiaspiromycosis” derives from the conidia of this fungus, the adiasconidia, which exhibit the unique property of progressive enlargement, without replication, at elevated temperature (approximately 37°C). An inhaled conidium, 2-4 μm in diameter, can grow to 200-400 μm or more, which results in a millionfold expansion of its volume. The pulmonary lesions produced by the enlarging conidia consist of discrete or confluent fibrotic granulomas, each containing one or more spherical, thick-walled adiasconidia that, when mature, are just large enough to be seen with the unaided eye. Proliferation or replication of the adiasconidia does not occur in human tissues. The granulomas therefore maintain a bronchiolocentric pattern of distribution indicative of an inhalational portal of entry, and secondary dissemination within or beyond the lungs does not appear to happen, despite an unconvincing claim to the contrary. Symptomatic disease has been attributed to inflammation and fibrosis associated with the granulomatous inflammatory reaction, and to displacement and compression of terminal airways and alveoli by massive numbers of progressively enlarging but sterile adiasconidia.

Because adiaspiromycosis occurs so infrequently in humans, little clinical experience with it has accumulated. The Czechoslovakian workers have proposed a clinicopathologic classification based upon the density of granulomas found in the lungs. Solitary adiaspiromycotic granuloma is an incidental finding in lung tissue removed for another reason; patients with this form of the disease have no symptoms or radiographic abnormalities attributable to the fungal infection. Disseminated granulomatous pulmonary adiaspiromycosis is a localized or diffuse bilateral pulmonary disease that has a finely nodular or reticulonodular radiographic pattern; only those patients with the densest, most severe bilateral involvement have had symptoms, which include fever, cough, and progressive dyspnea. No more than half a dozen symptomatic patients have been described. The diagnosis is usually established by lung biopsy. Pathologists unfamiliar with this disease are likely to mistake the adiasconidia for helminthic parasites, aspirated starch granules of lentil pneumonia, or more familiar large-form fungal pathogens such as *Coccidioides immitis* or *Rhinosporidium seeberi*.

In the present issue of *Chest* (see page 1171), Valente Barbosa Filho et al report two Brazilian patients with progressive respiratory failure attributed to disseminated granulomatous pulmonary adiaspiromycosis, demonstrated by lung biopsy. Both patients recovered, and both were asymptomatic with normal chest radiographs and pulmonary function studies six months and one year later. The authors are appropriately cautious about advocating the use of systemic antifungal chemotheraphy even for symptomatic disease. Published evidence indicates that the thick-walled adiasconidia are sterile cells incapable of replication in host tissues. Resolution of the disease without adequate therapy in these two patients further supports a cautious attitude about the need for specific antifungal therapy. The role of corticosteroids in the treatment of adiaspiromycosis remains largely unexplored. There is clearly much that remains to be learned about this uncommon and fascinating disease.

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Toronto, Ontario, Canada

October 22-26, 1990