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LETTERS TO THE EDITOR

Wood charcoal dust pneumoconiosis



Dear Editor,

We report the case of a 51-year-old man, former smoker (30 packs/year), complaining of progressive dyspnea, which limited his capacity to work. The patient has been working in a wood charcoal manufacturing facility for the last 30 years, with an 8 h daily journey, without individual protection equipment. His activity involved carrying the charcoal from inside the masonry kilns and its fragmentation, invariably with heavy exposure to dense smoke and dust.

Physical examination and chest X-ray were unremarkable. A chest high resolution computed tomography (HRCT) showed diffuse ground glass opacities and ground glass

centrilobular opacities, besides lobular air trapping on the expiratory acquisition (Fig. 1A and B). Bronchoscopy revealed small amount of clear mucus with black anthracotic dots on trachea and main bronchi (Fig. 1C). In a transbronchial biopsy, abundant anthracotic pigment was observed within alveolar lumen, in some macrophages and alveolar septa, without significant inflammatory or fibrotic findings (Fig. 1D). A diagnosis of wood charcoal dust related pneumoconiosis was confirmed based on clinical, radiologic and pathologic correlation.

Wood is the biomass fuel most frequently used both as unprocessed wood and as charcoal.¹ Charcoal production involves carbonization of wood in masonry kilns, usually *Eucalyptus* or *Pinnus* spp. In Brazil, some facilities utilize a poorly mechanized process, highly dependent on human labor and associated with significant occupational hazards

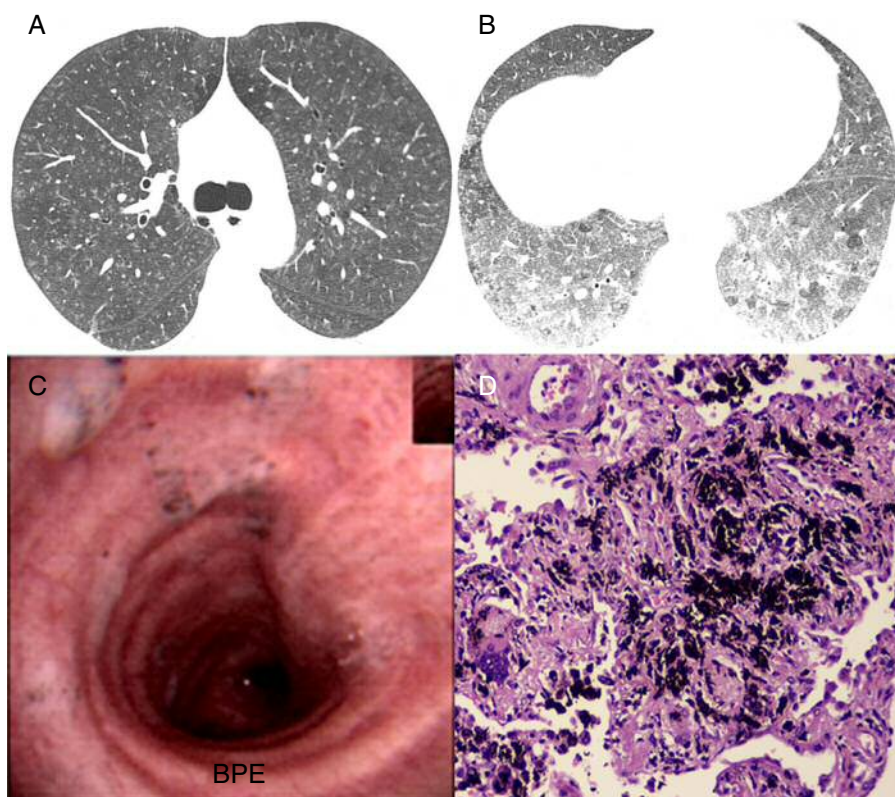


Figure 1 Inspiratory high resolution computed tomography axial scan (A) showing extensive centrilobular ground glass opacities and diffuse ground glass. Expiratory scan (B) showing ground-glass opacities and some lobular air trapping in the lower lobes. Bronchoscopy image showing small amount of secretion with anthracotic dots on trachea and main left bronchi (C). In (D) photomicrography showing interstitial anthracosis and multinucleated giant cell (hematoxylin-eosin, 200×).

in each stage of its manufacture.² Wood smoke is intensely released from the kilns, and represents a complex mixture of liquid, solid and gaseous particles, irritant and genotoxic, such as nitrogen and sulphur oxides, benzene, methanol, styrene, phenols, naphthalene, aldehydes, organic acids and polycyclic aromatic hydrocarbons.²

Both superior and inferior airway symptoms have been reported, including sneezing, nasal secretion, cough, expectoration, dyspnea, wheezing and even hemoptysis related to lower airway disease.³ Rhinitis, asthma, chronic obstructive pulmonary disease and respiratory infections may be observed in such workers.³ Literature is scant concerning interstitial lung disease/pneumoconiosis related to wood charcoal dust, especially regarding the HRCT findings. De Capitani et al.⁴ reported 3 cases of pneumoconiosis related to wood charcoal dust and activated carbon dust exposure. In all cases, HRCT descriptions match those observed in our cases, with centrilobular ground glass nodules. Our finding of air trapping on the expiratory scan probably representing small airway disease, was not mentioned in De Capitani et al.⁴ report. Similar findings have been described in carbon black pneumoconiosis, related to burning of natural gas and various petroleum products, with diffuse centrilobular ground glass nodules.⁵ We found no other papers describing HRCT appearance on charcoal dust exposure.

Histopathological description of black pigmented macules was observed both in our patient as in two of De Capitani et al.⁴ patients. No significant findings of fibrosis or inflammatory disease were observed in our patient's transbronchial or surgical biopsy, supporting the non-fibrogenic nature of this pneumoconiosis.⁴

In conclusion, we describe clinical, tomographic and histopathologic features of this uncommon interstitial lung pneumoconiosis related to wood charcoal smoke exposure. Considering the fact that biomass fuels are broadly disseminated as energy sources, especially in developing countries, prevention strategies in charcoal manufacturing should be encouraged in order to prevent such complications.

Conflicts of interest

The authors have no conflicts of interest to declare.

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Pleural effusion in AA amyloidosis – A rare involvement of a rare disease



Dear Editor,

Amyloidosis are a group of heterogeneous diseases due to protein misfolding and amyloid deposition in various organs and tissues, resulting in a wide range of clinical manifestations depending upon their chemical composition, location, and amount. The most frequent types of amyloidosis are the immunoglobulin light chain (AL) amyloidosis and the serum amyloid A, an acute phase reactant (AA) amyloidosis. The former is associated with plasma cell dyscrasia, while the latter results from longstanding chronic inflammatory/infectious diseases.¹ Reports about amyloid involvement of the respiratory tract are scarce.

We report the case of a 52-year-old female with type 2 diabetes mellitus and arterial hypertension who was admitted due to a 6-month history of anorexia, non-bloody diarrhea and weight loss. Upper gastrointestinal endoscopy revealed numerous lymphangiectasias in duodenum mucosa. Histological staining of duodenum biopsy samples showed macrophages containing bacilliform structures positive for periodic acid-Schiff (PAS) staining. The classic diagnosis of Whipple disease (WD) is based on positive PAS staining of duodenal biopsies, but PAS staining can be positive in other circumstances, such as *Mycobacterium* infection.² Real time PCR assays for *M. avium* and *M. intracellulare* were negative, and fungal structures were not identified in the biopsy specimen.

Despite the negative PCR for *Tropheryma whippelii*, the suggestive histological findings (but not entirely diagnostic) and the endoscopic characteristic observation, WD was assumed and the patient was treated accordingly. She