

Case Report

Wood Charcoal and Activated Carbon Dust Pneumoconiosis in Three Workers

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Background Data on prevalence of lung diseases due to inhalation of carbonaceous materials other than mineral coal is very limited.

Methods We present three cases of wood charcoal pneumoconiosis, two due to activated carbon, and one from wood charcoal artisan handling. To our knowledge, no clinical cases of wood charcoal pneumoconiosis, from artisan handling has been published so far.

Clinical Cases The three cases had their X rays classified by two B-readers as p/q round opacities with profusion ranging from 2/2 to 3/3. HRCT of two of them showed a diffuse centrilobular ground glass nodular pattern with subpleural small areas of consolidations. Transbronchial biopsies showed deposition of black pigment in the bronchiolar interstice similar to the histological appearance of simple coal workers pneumoconiosis, with no signs of fibrosis. Spirometry showed no abnormalities in the three cases.

Conclusions The authors point out to a probably underestimated respiratory occupational risk related to wood charcoal manipulation, which must be addressed mostly in developing countries, where deficient workplace conditions can lead to exposure above limit levels. Am. J. Ind. Med. 50:191–196, 2007. © 2007 Wiley-Liss, Inc.

KEY WORDS: wood charcoal; activated carbon; pneumoconiosis

INTRODUCTION

The use of activated carbon in medicine dates back to ancient Egypt, around 1500 BC. Since then it has been used

for treatment of wounds and internal ailments. Nowadays, it is still used as an internal agent for gastrointestinal decontamination in the treatment of intoxicated patients [Yehaskel, 1978; AACT-EAPCCT, 1997]. It is manufactured

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from charcoals produced by carbonization of wood, coconut shells, peat, lignite, and coal [Yehaskel, 1978; Parkes, 1994; AACT-EAPCCT, 1997]. Industrially, activated carbon is used in sugar purification, gas adsorption, dry cleaning, alcoholic beverages production, organic solvents recovery, and air and water purification. In Brazil, activated carbon is produced from wood or coconut and other nut shells. Due to its low rank, high quartz content (more than 10%), and high levels of sulfur and volatile hydrocarbons, Brazilian bituminous coal is not suited for activated carbon production [Algranti, 1998].

Data on prevalence of lung diseases due to inhalation of carbonaceous materials other than mineral coal is very limited. We present three cases of wood charcoal pneumoconiosis, two of them due to exposure in the activated carbon manufacturing process, and one at a wood charcoal packing process at a storehouse. To our knowledge, no clinical case of wood charcoal pneumoconiosis due to this last exposure condition has been published so far.

CASE REPORTS

Case 1

A 53 years old Afro-Brazilian male, was first seen in January 2001, complaining of insidious and progressive shortness of breath (SOB) for the last 2 years. A previous chest X-ray lead a general practitioner to refer him to further differential diagnosis. He suffered from essential hypertension and had cardiac arrhythmia with diagnosis of cardiomyopathy. Normal spirometry results are shown in Table I. Dyspnea in this case was considered to be secondary to his cardiomyopathy. For 12 years he had worked in an activated carbon plant, in the state of Paraná, Brazil, packing the grounded material before further delivery. The plant processed *Pinnus spp*, as wood source for the activated carbon manufacturing. Chest X-ray showed micro nodular opacities diffusely distributed with the cardiac shadow slightly oversized. It was interpreted as 3/3 q/q [ILO, 2000]. Thorax High Resolution Computed Tomography (HRCT) showed an intense and diffuse distribution of centrilobular ground glass nodules and foci of posterior subpleural consolidations extending from upper to lower lobes (Fig. 1).

A transbronchial biopsy was performed showing dense deposits of carbon around terminal bronchioles and no signs

of fibrosis (Fig. 2). Scanning Electron Microscopy (LEO 430i) of a bulk sample of dust collected in the plant during a technical visit by two of the authors (EMDC and AZH), showed a characteristic image of vegetal structures reminiscent of the wood from which charcoal was made (see Discussion section; Fig. 3). EDAX analysis of this material showed no other elements besides Al, Si, Fe, Ti, K, P, C, and Ca. No silica analysis was performed.

Case 2

During the plant visit of case 1 a new suspected case was detected and referred to clinical and radiographic evaluation. This patient was a 65 years old Afro-Brazilian male who had worked for the last 15 years at the same plant, at the same section. Patient showed no dyspnea, and spirometry test was normal (Table I). Chest X-ray showed a similar pattern of diffuse bilateral micronodules, interpreted as 3/3 p/q [ILO, 2000], and HRCT showed centrilobular ground glass opacities diffusely distributed. No lung biopsy was performed in this case.

Case 3

White male, 49 years, worked for 24 years in a wood charcoal store house, opening the big bags, shoveling and packing it in smaller packs for domestic use in a very dusty environment. He was referred in 1997 to a specialized clinic in occupational lung diseases with a suspicion of pneumoconiosis and Grade III shortness of breath. His chest X-ray was 2/2 p/q with a normal spirometry test (Table I). HRCT showed a diffuse centrilobular ground glass micronodular pattern, thickening of bronchial walls and some thickened interlobular septa (Fig. 4). A transbronchial biopsy revealed black pigmented macules with birefringent structures and foreign body type giant cells. The patient gradually developed episodes of wheezing with SOB that demanded medication. From 1997 to 2004 profusion of small opacities remained stable with a mean loss of 100 ml/year of FEV1.

DISCUSSION

Solid carbonaceous materials, like charcoal and carbon black, are formed by the process of carbonization, that is, the increasing of carbon content from organic materials by pyrolysis [Parkes, 1994]. This is the process by

TABLE I. Spirometric Results From 3 Cases of Pneumoconiosis Due to Wood Charcoal Dust, Brazil

Patient	FVC (FVC%)	FEV ₁ (FEV ₁ %)	FEV ₁ /FVC ratio	FEF _{25-75%}	X-ray ILO classification
1	4.30 L (105)	3.29 L (101)	78% (81%)	64%	3/3 q q
2	3.83 L (113)	3.29 L (125)	90% (89%)	92%	3/3 p q
3	3.65 L (108)	2.76 L (101)	75% (93%)	71%	2/2 p q



FIGURE 1. HRCT of case #1 showing centrilobular ground glass opacities, some of them in the subpleural parenchyma.

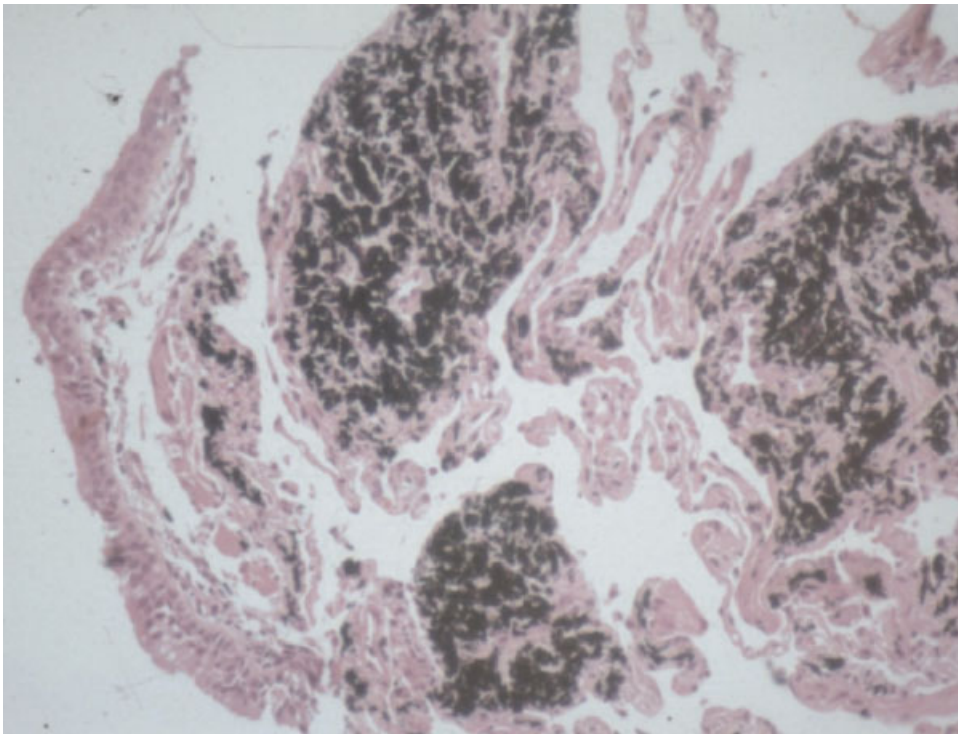


FIGURE 2. Transbronchial biopsy specimen showing deposits of black pigment in the bronchiolar interstice.

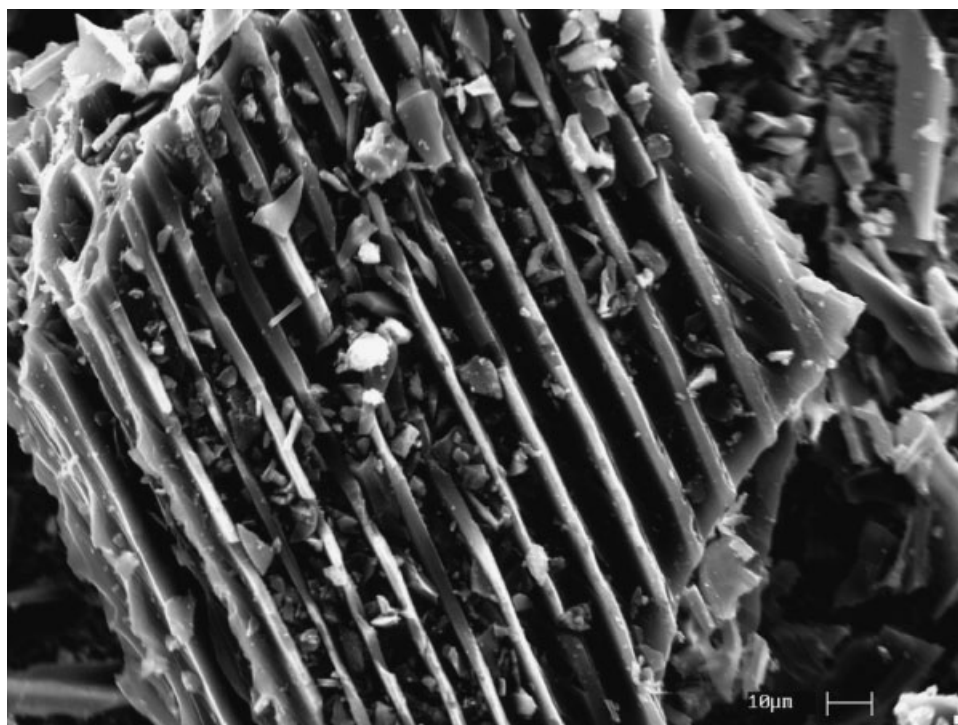


FIGURE 3. SEM image of dust showing characteristic vegetal anatomic structure of the particulate material collected at the activated carbon plant.

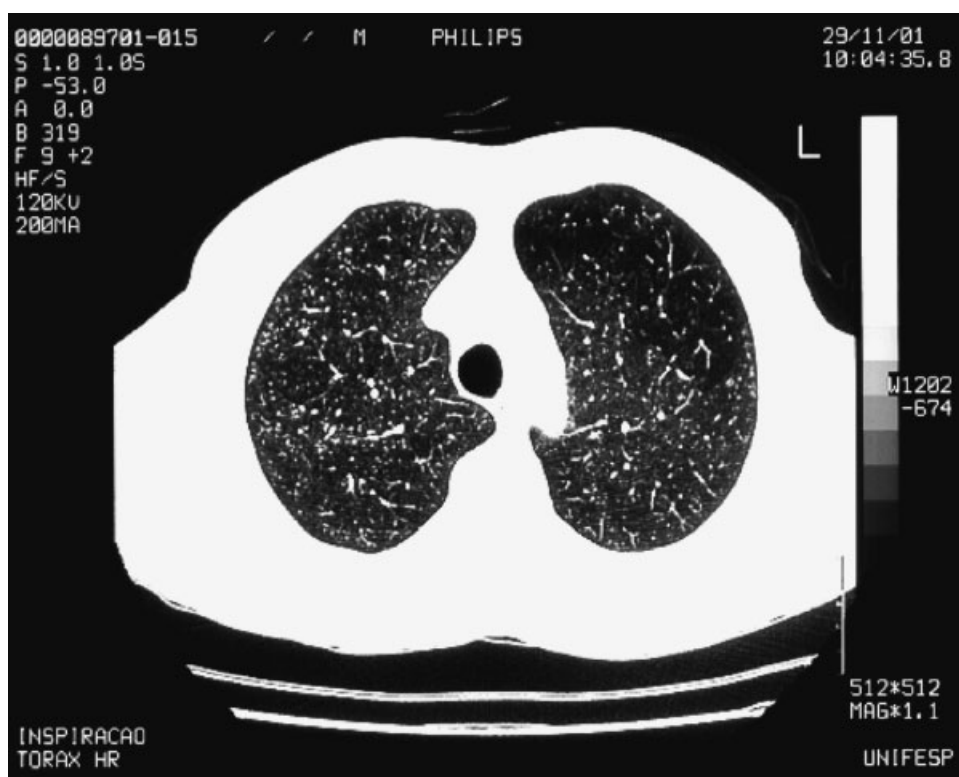


FIGURE 4. HRCT of case #3 showing intense and diffuse centrilobular well defined nodules.

which charcoal is formed from wood. The pyrolysis process is usually developed in small earth kilns where the wood is slowly and partially burned in an anaerobic atmosphere extracting most of the impurities, leaving behind almost pure carbon [Freese, 2003]. Activated carbon is produced from charcoal (made from wood, coconut shells, lignite, peat, or coal), which is "activated" through steam, CO₂ or pressured air, cleaning the charcoal from volatile hydrocarbons and other impurities, increasing its porosity and capacity of adsorption [Yehaskel, 1978; Uragoda, 1989; Parkes, 1994].

The work place where the activated carbon patients worked was dusty, the process of production involved heating the charcoal at 900°C before crushing by rollers in rotating kilns at lower temperatures. The packing section was the dustier area with no adequate ventilation system. Uragoda [1989] showed that airborne dust particles in a similar plant were between 12.5 and 3.5 µm, with a small proportion between 3.5 µm and less than 1.2 µm [Uragoda, 1989]. Quartz content in wood is negligible and absent in coconut shells, with carbon responding for more than 90% of the dust produced during the industrial process [Parkes, 1994]. Crystalline silica content of USA activated carbon was found to be about 0.12% [Wehr et al., 1975]. Vegetable anatomic structure of activated carbon dust content was confirmed by SEM. As showed in Figure 3, an image taken during SEM analysis shows the characteristic configuration of the stem secondary xylem (principal part of the plant vascular system) of *Pinnus spp*, with its parallel tracheid units [Fahn, 1990].

The inhalation of dust from coal or other carbonaceous materials can produce pneumoconiosis with similar pathological appearances and clinical behavior. Differences may be present depending on the intensity of exposure and the percentage of contaminants in the inhaled material. The lesions would be classified as simple pneumoconiosis or progressive massive fibrosis (PMF). The reported cases are classified as simple pneumoconiosis: small, discrete dust macules or nodules not larger than 5 mm in diameter, concentrated mainly in the peribronchiolar region. PMF, not seen in these cases, are mostly related to coal exposure [Parkes, 1994]. Simple pneumoconiosis due to carbon deposition in the lungs has also been diagnosed in graphite workers [Lister, 1961; Parkes, 1994], and carbon-black workers in rubber plants [Crosbie, 1986; Parkes, 1994].

To our knowledge, case 3 is the first case of pneumoconiosis in a worker from this kind of wood charcoal low technology process. The pathological appearance and clinical behavior is quite similar to the two activated carbon cases described.

Activated carbon pneumoconiosis was first described by Wehr et al. [1975]. Studying 397 workers, they found 9.6% of them showing radiographic alterations compatible with pneumoconiosis interpreted as *p* round opacities according

to ILO [1980]. Two workers had been submitted to lung biopsies and showed extensive carbon interstitial deposits, but minimal associated fibrosis [Wehr et al., 1975]. Lung biopsy specimen of a man who had worked for 11 years exposed to activated carbon dust, showed alveolar walls thickened by carbon deposits and anthracotic pigment concentrated beneath the pleura and in perivascular lymphatic [Wehr et al., 1975]. Studying 66 exposed workers in Sri Lanka, Uragoda [1989] found no cases of radiographic alterations consistent with pneumoconiosis. Gross and Nau [1967] have shown that animals experimentally exposed to activated carbon from lignite presented extensive peribronchial and interstitial deposits of carbon dust with minimal fibrosis, like the pathologic features of our cases. The pathologic alterations from the two biopsy specimens described here match the ones found in the studies of Wehr et al. [1975] and Gross and Nau [1967]. After 7 years of clinical and radiological follow up, case 3 did not show any change in radiographic classification [ILO, 2000].

Exposure to carbon-rich, non-fibrogenic dust appears similar, regardless of the source, and can raise the possibility that activation of the carbon does not affect the pathogenesis of these lesions. Coal workers pneumoconiosis (CWP) caused by the inhalation of coal dust with variable amounts of silica can have some similarities regarding HRCT appearance, like the presence of small ill-defined centrilobular nodules. However, focal centrilobular emphysema generally accompanies the nodular lesions [Webb et al., 2001]. The microscopic macules seen in CWP are usually composed by dust-laden macrophages within the walls of the respiratory bronchioles and adjacent alveoli, associated with variable amounts of reticulin and collagen fibers intermingled with the macrophages. The presence of reticulin and collagen fibers in CWP reflects the proportion of silica in the inhaled dust [Green and Vallyathan, 1998]. Other carbonaceous materials such as graphite and carbon black can potentially cause similar radiographic and histopathological appearances like the one described here, depending on the proportion of fibrogenic material content of the inhaled dust [Green and Vallyathan, 1998].

Despite the few cases published so far, pneumoconiosis due to wood charcoal might be an underestimated occupational risk, and early diagnosis and prevention must be addressed mostly in developing countries, where low industrial hygiene standards might expose workers to dust above threshold limits.

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REFERENCES

- AACT-EAPCCT. American Association of Clinical Toxicologists and European Association of Poison Control Centres and Toxicologists. 1997. Position statement: Single-dose activated charcoal. *Clin Toxicol* 35:721–741.
- Algranti E. 1998. Occupational lung diseases in Brazil. In: Banks D, Parker J, editors. *Occupational lung disease—an international perspective*. London: Chapman & Hall Medical. p 105–115.
- Crosbie W. 1986. The respiratory health of carbon black workers. *Arch Environ Health* 41:346–358.
- Fahn A. 1990. *Plant anatomy*, 4th Edition. Oxford: Pergamon Press. p 335.
- Freese B. 2003. *Coal, a human history*. Cambridge, MA: Perseus Publishing. p 308.
- Green FYH, Vallyathan V. 1998. Coal worker's pneumoconiosis and pneumoconiosis due to other carbonaceous dusts. In: Churg A, Green FYH, editors. *Pathology of occupational lung disease*, 2nd Edition. Baltimore: Williams & Wilkins. p 129–207.
- Gross P, Nau CA. 1967. Lignite and the derived steam-activated carbon. The pulmonary response to their dusts. *Arch Environ Health* 14:450–460.
- ILO. International Labor Office. 1980. *Guidelines for the use of the ILO International Classification of Radiographs of Pneumoconiosis—Revised Edition 1980*, Occupational Safety and Health Series no 22, Rev 80, Geneva: ILO. 48 p.
- ILO. International Labor Office. 2000. *Guidelines for the use of the ILO international classification of radiographs of pneumoconiosis—revised edition 2000*. Occupational safety and health series no 22. Geneva: ILO. p 43.
- Lister W. 1961. Carbon pneumoconiosis in a synthetic graphite worker. *Br J Ind Med* 18:103–113.
- Parkes W. 1994. Pneumoconiosis associated with coal and other carbonaceous materials. In: Parkes W, editor. *Occupational lung disorders*. Oxford: Butterworth-Heinemann. p 340–410.
- Uragoda CG. 1989. Clinical and radiographic study of activated carbon workers. *Thorax* 44:303–304.
- Webb WR, Muler NL, Naidich DP. 2001. *High resolution CT of the lung*, 3rd Edition. Philadelphia: Lippincott-Raven Publishers. p 149–191.
- Wehr KL, Johanson WG, Jr., Chapman JS, Pierce AK. 1975. Pneumoconiosis among the activated-carbon workers. *Arch Environ Health* 30:578–582.
- Yehaskel A. 1978. *Activated carbon—manufacture and regeneration*. New Jersey: Noyes Data Corporation. p 51.