CASE REPORT

Wood-Smoke Exposure : An Unusual Cause of Miliary Mottling on X-ray Chest

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ABSTRACT

A case of wood smoke inhalation related lung disease presenting with miliary mottling on radiography is described. Transbronchial lung biopsy showed the presence of coal macules.

Key words: Wood-smoke, Transbronchial lung biopsy, Coal macule.

INTRODUCTION

Wood is still being used in many parts of India, especially in the villages as fuel. In many cases, due to lack of knowledge about the significance of proper ventilation and exposure to smoke, it leads to many untoward health effects. In this report we present unique findings in a patient after exposure to fine particles generated by wood burning in a closed environment.

CASE REPORT

A 26-year-old married woman has been a resident of a remote village in Tehsil Pangi of District Chamba in Himachal Pradesh (India) since her childhood. The village is situated at an altitude of 8000 feet in absolutely pollution free surroundings and is snow bound for most part of the year. She enjoyed good health until about 15 months prior to her hospital visit. Earlier at a medical camp organised near their village, her chest radiograph was taken; she was prescribed bronchodilators and vitamins and was advised to report to a tertiary care hospital for further investigations.

Patient remained active as a home maker. She was a non-smoker. There was no past history of tuberculosis or contact with a case of tuberculosis. Her parents had died of some respiratory illness.

She presented with the complaints of progressive breathlessness for the last 15 months. Initially, the patient used to get dyspnoeic while climbing up a hill but now she was breathless even during a brisk walk on a level ground. She also complained of occasional cough with greyish mucoid expectoration.

Besides this, there was no complaint of any fever, night sweats, haemoptysis, fatigability, loss of appetite or weight, chest pain, palpitation, parorysmal nocturnal dyspnoea.

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orthopnea, myalgias, arthralgias, dryness of mouth or bluish discoloration of fingers. The menstrual history was also normal.

Physical examination was unremarkable. Ophthalmologic evaluation including slit lamp examination revealed no abnormality.

Initial laboratory findings showed a normal hemogram, serum biochemistry and urinalysis. Thyroid function, rheumatoid factor, antinuclear antibody, fungal serology were all negative. She was human immunodeficiency virus (HIV) sero-negative. Sputum examination was repeatedly negative for acid-fast bacilli (AFB), fungus and malignant cells. The resting electrocardiogram was normal.

Ultrasound for abdominal and pelvic organs revealed no abnormality. Skin tests for various fungi (Candida, Histoplasma, Coccidia) and tuberculin were negative.

Pulmonary function tests (PFT) demonstrated a mild obstructive airways disease. Diffusion studies could not be carried out. The resting arterial blood gas analysis was normal.

Chest radiograph (PA view) (Figure 1) and HRCT scan of the chest (Figure 2) revealed diffuse discrete nodular miliary shadows in both lung fields more on right side. Radiographs of hand, lumbosacral spine and hips were normal.

Bronchoscopy revealed anthracotic staining of airways. Bronchoalveolar lavage (BAL) and transbronchial lung biopsy (TBLB) were subsequently performed and multiple biopsies were obtained from the right middle and lower lobes. Results of BAL fluid analysis showed carbon pigment laden macrophages (70%), neutrophils (20%) and lymphocytes (10%). Stain for AFB was negative. Transbronchial lung biopsy showed thickening of alveolar septa due to infiltration by lympho-mononuclear cells, carbon-laden macrophages and fibrosis. In addition there were coal macules, i.e., broad fibrous scars infiltrated by carbon laden spindle cells (Figure 3). There was no granulomatous pathology.

Figure 1. Chest radiograph (PA view) showing diffuse discrete miliary shadows in both lung fields, more on the right side.

Figure 2. HRCT of thorax showing diffuse discrete miliary shadows in both lung fields.

Figure 3. Photomicrograph showing collection of fibroblasts rich in carbon pigment extending into the alveolar septae (H&E × 550).
It was found that people in that region live in small houses with little or absolutely no ventilation. Due to the extreme cold and non-availability of other fuels, they burn wood of a tree locally known as ‘Dayar’ (Cedar) to keep themselves warm and also to cook their food. To conserve fuel during non-cooking periods, the wood is not allowed to burn quickly but is kept smouldering to prolong its slow heating effect, leading to very high concentration of soot. All the inhabitants are exposed to this extremely smoky atmosphere throughout the day for months and years together. She also disclosed that many of their neighbours suffer from respiratory complaints. It was also observed that men who keep away from home to earn their livelihood do not suffer from similar ailments.

In this patient all other causes of fine miliary mottling in an afebrile patient1, such as sarcoidosis, metastases, pneumoconiosis, eosinophilic granuloma, alveolar microlithiasis, haemosiderosis, fungal infections and miliary tuberculosis were carefully considered and ruled out. A final diagnosis of ‘wood smoke inhalation associated lung disease’ was made. Patient was sent back with advice to switch over to alternative modes of cooking and warming and also to improve the ventilation in their house and also to advise their neighbours regarding the significance of the same. She came back one year later and was keeping fine with no further deterioration in her lung functions.

**DISCUSSION**

Wood smoke is known to contain compounds like carbon monoxide, nitrogen oxides, sulfur oxides, aldehydes, polycyclic aromatic hydrocarbons and fine respirable particulate matter2. Chronic exposure to emissions from wood burning has been associated with chronic obstructive pulmonary disease3-5. It has also been associated with pulmonary arterial hypertension and cor-pulmonale6, lower respiratory illness7, and lung cancer8-10 in various studies.

There are reports of patient developing interstitial lung disease following prolonged exposure to chronic domestic wood smoke inhalation6,11. In both the reports the patients were mostly females, over 60 years of age with a long-standing and intense indoor wood smoke exposure. Dyspnoea and cough were the main complaints and the chest radiograph showed a diffuse bilateral reticulonodular pattern. Most patients showed a mixed restrictive-obstructive pattern with severe hypoxaemia and variable degree of hypercapnia. Histopathology showed fibrosis and inflammatory focal thickening of the alveolar septa as well as diffuse parenchymal anthracotic deposits5. It was suggested that the carbonaceous particles produced by wood burning have a fibre-like character and iron coating, and enables them to incite chronic inflammation6. Probably these patients represented one end of the spectrum of the wood smoke inhalation associated lung disease.

In comparison, our patient was much younger and breathlessness and cough were not that marked. Pulmonary function showed only a mild obstructive airway disease. Chest radiograph revealed bilateral miliary mottling and histopathology showed coal macule with interstitial inflammation. In our opinion, this patient represents the early stage of the spectrum of wood smoke inhalation associated lung disease. Prior to this case which has been fully worked up at a tertiary care hospital, many cases of unexplained miliary shadows detected during a camp organized at a remote snow bound area in the state of Himachal Pradesh (HP) were brought to the notice of the author (VS) who had worked for a short while at the Indira Gandhi Medical College, Shimla. It is possible that those cases were also having early stages of wood smoke inhalation associated lung disease.

Also, cases with similar radiological findings from Ladakh another extremely cold and high altitude region of India have been reported by Saiyed et al12. However in these cases, the diagnosis was not confirmed by histopathology. Since the radiological findings in these cases resembled those seen in pneumoconiosis, and since there were no industries or mines in any
part of Ladakh, they termed the entity as ‘non-occupational pneumoconiosis’ and gave two probable reasons to explain these findings: (i) exposure to free silica from dust storms occurring in these regions; and (ii) exposure to soot from domestic fuel (wood). Also, low oxygen levels and some unknown factors associated with high altitude were considered as important contributing factors in causation of this type of picture.

However in the case being reported, there was no history of any exposure to the dust storms. Moreover exposure to dust only for a few days every year is not likely to lead on to such radiological changes. It is prolonged exposure to high concentration of soot, which is responsible for this. Regarding mechanism of miliary mottling in such cases, it has been suggested that coal macules formed in the lungs following a prolonged exposure to carbonaceous dusts are seen as fine opacities on radiography as a result of effect of summation.

It summary, wood-smoke inhalation associated lung disease is still a poorly understood entity and the nomenclature has not been properly defined. The authors suggest a more detailed and coordinated multicentric study in such areas to get more information about it.

REFERENCES


