

Gujjar Lung: A Disease Mimicking Miliary Tuberculosis

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Abstract: Gujjar lung is a chronic lung disease caused due to the long-term exposure to pinewood smoke inhalation in Gujjar community and the people residing at the hilly regions of the Indian sub-continent. This is characterized clinically by progressive cough and dyspnea, distinct radiological patterns and pathological features of anthracotic nodules and fibrosis. A typical case with miliary mottling on chest radiograph is presented and the relevant literature reviewed.

Keywords: Pine wood smoke, Miliary shadows, Anthracotic nodules, Interstitial lung disease.

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Introduction

Gujjar lung is an environmental interstitial lung disease caused due to the indoor air pollution with pinewood smoke, occurring predominantly in members of the Gujjar community—a social and an ethnic group residing at high altitude hilly regions of the Indian sub-continent (viz Jammu and Kashmir, Himachal Pradesh, Rajasthan, Pakistan, Pakistan Administered Kashmir and Tibet). These people typically live in very small poorly ventilated dwellings called *kothas*, made of mud, stones and wood with very low roofs made of wood and crop residues covered with clay. Inside, under the same roof, there is a compartment of cattle and another where the family members live. In addition, there is a *Chullah* (fixed mud hearth) which is made of clay and stone and is used for cooking where by wood, dried cow dung and crop residues are burnt for an average of 12 to 16 hours a day. A high oleoresin containing part of the pine wood called a *lash* is burnt for lighting purpose and is usually kept burning during morning and evening hours creating a dense smoky atmosphere in the dwellings, which as already mentioned are very poorly ventilated.

The disease is characterized clinically by progressive cough and dyspnea, with onset usually beyond the fourth decade of life; the characteristic radiological pattern of miliary mottling, reticulonodular shadowing, fibrosis and/or cor pulmonale and presence of anthracotic nodules with carbon-laden macrophages and fibrosis on histopathological examination. The entity was introduced in medical literature in 1991^[1], and since then few more cases have been reported^[2, 3]. We again describe a case of miliary mottling on chest radiograph misinterpreted as miliary tuberculosis.

Case Scenario

A 48-year-old male Gujjar presented with a history of progressive dyspnea and cough with the production of blackish sputum. He had no history of fever or loss of weight and there was no other history suggestive of tuberculosis or any other significant illness in his past. He had lived in a *kotha* since his early childhood. His general physical examination and systemic examination were normal, except for the presence of a few crackles in the lower lobes of both lungs on the auscultation of the chest. The chest radiograph revealed a miliary shadowing in all zones on both sides (Fig. 1).



Fig. 1. Chest radiograph showing miliary shadows bilaterally, more dense in mid zones peripherally.

All other baseline investigations including hemogram, serum biochemistry and serological studies (including rheumatoid factor, anti-nuclear antibodies, serum electrophoresis and immunoglobulin assay) were normal. Fiberoptic bronchoscopy revealed the anthracotic staining of main bronchi. Bronchoalveolar lavage (BAL) fluid on microscopic examination predominantly showed macrophages laden with carbon pigment and cultures of the fluid for mycobacterium tuberculosis and fungi proved negative. The serology for human immunodeficiency virus (HIV) was negative. Resting arterial blood gas analysis was normal. Pulmonary function testing by spirometry (spirolab II Italy) showed moderate restriction. High resolution computed tomography (HRCT) scan of the chest showed well defined miliary shadows on both sides (Fig. 2).

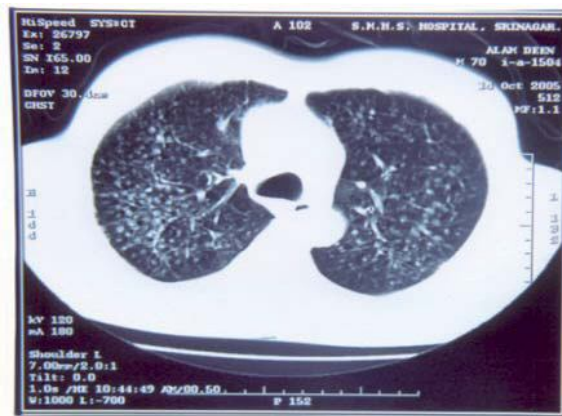


Fig. 2. High resolution computed tomography (HRCT) scan of chest showing well defined miliary shadows, with diffuse distribution.

Percutaneous lung biopsy was performed under CT guidance. On the histopathologic examination, it revealed anthracotic nodulation with carbon laden macrophages, with fibrosis (Fig. 3).

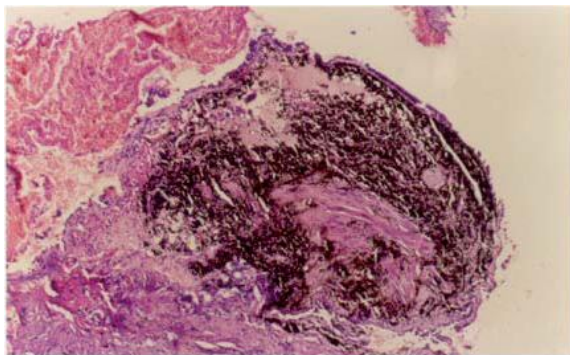


Fig. 3. Lung biopsy showing anthracotic nodule with carbon laden macrophages and fibrosis, (Hematoxylin & eosin x-250).

On the basis of typical radiological features and histopathology, the diagnosis of Gujjar lung was made.

Discussion

The chronic exposure to wood smoke and its long-term inhalation has been associated with chronic obstruction pulmonary disease, pulmonary arterial hypertension, corpulmonale, recurrent lower respiratory infections, lung cancer and interstitial lung disease [4-9].

The entity Gujjar lung was first introduced in 1991 by Dhar and Pathania [1] from Kashmir when they noticed miliary mottling and reticulonodular pattern in the chest radiographs of patients belonging to Gujjar community. These were empirically put on therapeutic trials with antituberculosis treatment, but the shadows remained unchanged despite adequate dosage and duration of treatment. Finally, the lung biopsy was taken which revealed the findings of anthracotic nodules, carbon laden macrophages and fibrosis. The authors attributed it to the indoor air pollution with smoke from biomass combustion, mainly pine wood. Subsequently, Raison et al. [2] from the George Washington University, USA reported HRCT findings in a case of Gujjar lung with similar histopathological features in a Kashmiri baker, who was working in Saudi Arabia. The chest radiographs were taken on the patients as part of routine health checkup prior to his immigration,

which showed significant miliary shadows. The history of exposure to pinewood smoke was established. By 2001, Saiyed et al. [10] from Ladakh region of Jammu and Kashmir reported miliary shadowing in the chest radiographs of residents of this high altitude region. They described this entity as non-occupational pneumoconiosis and attributed it to the exposure to free silica from dust storms or domestic wood smoke inhalation. However, histopathologic examination was not performed [7].

More recently, Saini et al. in 2003 [11] from the Postgraduate Institute and the Government Medical College, Chandigarh, India reported similar radiological and histopathological findings in a woman of Himachal Pradesh and described the disorder as wood smoke inhalation associated with lung disease. We have been observing and studying such cases for the last eight years now, and the most probable mechanism of lung injury seems to be the long-term inhalation of pine wood smoke, as the disease was not found in any of the 4,124 individuals who belonged to Gujjar community, but were different in that lived in a different region and were also exposed to intense indoor air pollution with smoke from other biomass fuel (crop residues, dried dung cakes and wood) combustion but were not exposed to pinewood smoke.

Pinewood on combustion yields sulphur dioxide, benzopyrene, carbon monoxide, nitrogen oxides, polycyclic hydrocarbons and low molecular weight aldehydes including acrolein and albeitic acid. These gases, individually or along with carbon, could be responsible for lung injury and fibrogenic reaction [1, 2, 7, 11]. Coal macules formed in the lungs following a prolonged exposure to carbonaceous dusts and the resulting fibrosis is postulated to give rise to the characteristic radiological appearance of miliary and reticulonodular shadows [1, 9, 11].

The entity needs to be differentiated from coal worker's pneumoconiosis which has similar histopathological appearance, but the patient's ethnic origin, history, exposure and occupation help to differentiate it from the latter. Cryptogenic organizing pneumonia is differentiated by the absence of polypoid granulation tissue within the lumen of bronchioles and alveolar ducts [1, 2]. Gujjar lung is of paramount importance from the treatment point of view because such cases are empirically being subjected to anti-tuberculosis

drugs resulting in an unnecessary wastage of resources and exposure to adverse effects.

Further studies involving large samples are needed to see the injury patterns, genetic predisposition and the reactions evoked in the lungs. Moreover, as the data suggests for now, prevention involves changing the living standard of this community, so as to prevent the exposure to pinewood smoke inhalation. The provision of electricity as the safest and cleanest source of fuel, through appropriate government policy, is expected to prevent the occurrence of this disorder.

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