Hut Lung Disease: A Radiological and Pathological Correlation

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Abstract

Hut lung disease is a domestically acquired pneumoconiosis described in individual’s secondary to inhalation of particles from either grinding of maize, or when cooking on fires in a small dwelling, or hut. Palmer and Daynes first described the condition in 1967 seen in women in the Transkei district of South Africa, who had significant exposure due to traditional maize grinding practices [1]. Patients may be asymptomatic, or present with varying degrees of dyspnea, and/or chronic bronchitis which may progress into advanced interstitial lung disease, massive pulmonary fibrosis or malignancy. We describe the radiological, and histological findings associated with hut lung disease.

Keywords

Hut lung, Domestically acquired particulate lung disease, Mixed dust pneumoconiosis, Biomass fuel, Tomography, X-ray Computed, X-ray

Background

Hut lung, or domestically acquired particulate lung disease is a pneumoconiosis occurring secondary to inhalation of smoke derived from biomass fuels used for cooking in poorly ventilated huts or domestic settings [2]. The term mixed dust pneumoconiosis was historically used interchangeably particularly when the pneumoconiosis occurs following inhalation of other particulate matter such as mixed dust, and silicate which are known to arise from food grinding processes [3], and following the exclusion of other well-defined pneumoconiosis [4, 5], although more recent specific definitions have been described [4].

Presently, close to 3 billion people who make up approximately 50% of the world population, mainly from developing nations still depend on solid biomass fuels for cooking, and heating. Inadvertently, this results in increased exposure to household air pollutants (HAP) which not only results in various respiratory tract sequelae, but also other harms such as burns, and poisoning. This is potentially the largest environmentally attributable disorder in the world [5], and more troubling, women and children are most susceptible [6]. The term ‘hut lung’ had been coined to collectively represent a gamut of clinical conditions including chronic bronchitis, chronic obstructive pulmonary disease (COPD), and interstitial lung disease associated with exposures to biomass smoke [7].

Individuals are considered diagnosed with hut lung disease if the following are noted [3]:

• Individuals originating from rural dwellings,
• Exposure to biomass smoke from cooking fires, or who had ground maize
by traditional methods, or both,
  • No prior exposure to mining, or other industry,
  • No prior exposure to, or evidence of active tuberculosis, and
  • Presence of radiographic and histopathological evidence of pneumoconiosis.

Pathophysiology
Particulate matter from biomass smoke which is < 10 μm are of respirable size (PM_{10}) can penetrate deep into the lungs resulting in damaging health effects ranging from local reaction (airway inflammation or hyperresponsiveness), and COPD to carcinogenesis [8]. The cause-effect linkage between biomass smoke and chronic pulmonary disease is still unknown, limited by the scarce data. However, altered innate immunity due to macrophage dysfunction resulting in increased susceptibility to infection, and the increased activity of matrix metalloproteinase particularly with wood–smoke exposure have been reported [7, 9].

Symptomology
Symptoms can range from being asymptomatic, or more commonly, cough related symptoms, with or without dyspnea, to the more debilitating pulmonary hypertension, and cor pulmonale [5, 7]. Classically, disease identification occurs following a diagnostic work-up for emigration purposes. Considering the limited health resources available in developing nations which is compounded by the indigent patient socioeconomic status, patients often go undetected in their native countries.

The presence of bilateral lung nodules on the chest x-ray raises the suspicion of pulmonary tuberculosis, or chest infections which often leads to bronchoscopy, bronchoalveolar lavage, and lung biopsy. Following tissue diagnosis of extensive dust deposition suspicious for pneumoconiosis, a retrospective detailed environmental history will reveal prior exposure to biomass fuel which is usually difficult to obtain at referral due to the language barrier, and the purportedly distracting findings on radiography suggestive of pulmonary tuberculosis [2, 10].

Pulmonary function tests
A wide spectrum of pulmonary function tests has been documented ranging from normal, mild to severe airway obstruction, decrease diffusion capacity, mild restrictive pattern to a mixed picture [3, 5, 7].

Radiological findings
Characteristic diffuse pulmonary nodules resembling miliary tuberculosis (Figures 1 and 2) on the chest radiograph is generally the inciting finding [3, 5], although upper lobe predilection have also been reported (Figure 3) [2, 7]. Extensive fibrosis along with hilar, and mediastinal lymphadenopathy is not uncommon (Figures 4 and 5) [2, 7, 10, 11]. These findings can mimic healed granulomatous lung disease including pulmonary tuberculosis [3]. The CT chest imaging often demonstrates multiple centrilobular lung nodules sparing the pleural surfaces, patchy nodular bronchovascular thickening, nodular interlobular septal thickening, bilateral hilar with, or without mediastinal lymphadenopathy [2, 5, 7, 11].

**Figure 1:** Posterior–anterior (PA) chest radiograph (left) demonstrates bilateral diffuse nodular opacities while the coronal reformats of the chest computed tomography (CT) (right) reflect the chest x-ray findings.

**Figure 2:** PA chest radiograph (left) demonstrates bilateral diffuse miliary nodules with multiple areas of confluent inflammatory scarring. The coronal reformat of the chest CT (right) confirms the presence of multifocal areas of nodularity.

**Figure 3:** PA chest radiograph (left) demonstrates bilateral diffuse nodularities with more focal confluent inflammatory change in the right lung apex which is also seen of the coronal reformat of the chest CT (right).

**Bronchoscopy, bronchoalveolar lavage and tissue biopsy**
Macroscopically, bronchoscopy demonstrates large blackish-dark blue plaques in the large airways, commonly involving the bifurcation of the lobar bronchi [5, 7]. Bronchoalveolar lavage usually demonstrates normal differential cell count [5, 7] with cytology revealing numerous dust-filled macrophages [2]. Although, tissue biopsy is the gold standard for the diagnosis, only a few authors report histological confirmation in their series [2]. Microscopically, characteristic anthracotic pigment (carbon) deposition around terminal bronchioles, the presence of dust macules, and mixed dust fibrosis are confirmatory (Figure 6) [3, 5, 7]. Grobbelaar et
al. [3] described three typical histological patterns which were observed in their study which includes simple anthracosis, anthracosis with macule formation, and mixed dust fibrosis which were similarly noted in other studies [2, 4, 7, 10, 11]. More recently, Mukhopadhyay et al. [2] have described precise classification of the deposited particles utilizing scanning electron microscopy with energy dispersive x-ray spectroscopy (SEM/EDS), given that crystalline silica particle may be too small to be detected by polarized light microscopy alone. This added information may provide an insight into the aetio-pathogenesis but ultimately does not affect treatment.

• close radiological resemblance to pulmonary tuberculosis, and the difficulties in differentiating the two especially when there is a high prevalence of pulmonary tuberculosis in areas where hut lung disease is commonly seen, and

• the possible malignant transformation potential.

Association with pulmonary tuberculosis

The findings associating hut lung disease with pulmonary tuberculosis have been varied with several authors reporting insufficient, and weak evidence associating the two [12-14], while Sumpter et al. argues that the more recent body of evidence suggests otherwise [15]. Exposure to tobacco smoke, poverty and over-crowding are several plausible reasons associated with both biomass fuel use, and pulmonary tuberculosis with a reasonable inference that the most indigent population from developing nations who can only afford biomass fuel as a form of combustion is the very same cohort at the highest risk for tuberculosis for aforementioned reasons [12, 13, 15, 16]. Nevertheless, it was agreed that further studies are required to understand the association of HAP with pulmonary tuberculosis.

Association with lung cancer

The products of fuel combustion are known to contain carcinogens, thus the association between HAP with lung cancer risk seems justified [17, 18]. Several studies worldwide have shown, and suggested that there is an increased lung cancer risk associated with the inhalation of smoke from wood fuel combustion, with limitations related to the extent, duration, and intensity of exposure [19]. The known carcinogen implicated includes polyaromatic hydrocarbons [18, 19]. Lung cancer risks in exposed women were also shown to increase by almost two-fold [19, 20]. A novel study also demonstrated close association of solid biomass fuel with squamous cell cancer histopathological subtype. However, it was acknowledged that the effect estimates from a large number of studies in this report which did not adjust for smoking, was accountable for this [20]. Additionally, it is well established that cigarette smoking is the leading cause for squamous cell carcinoma and potentially confound this association. It was also reported that there was close association between coal, and lung cancer when compared to any other types of biomass fuel [20]. Other authors have demonstrated a genomic association of glutathione S-transferases (GSTM1) null genotype with a more significant lung cancer risk in populations with coal exposure in certain Asian population [17].

Conclusion

Globalization and continued immigration of individuals from developing nations to developed countries pose new health challenges to health care providers in the latter. Although a thorough work-up is mandatory and justified when there is a high clinical suspicion for pulmonary tuberculosis, health care providers in developed countries should be aware, and consider the diagnosis of hut lung syndrome, particularly when involving patients with the aforementioned demographics, and findings.
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Conflict of Interest

None declared.

References