## **Original Research Article**

# **Role of HRCT in Smoking Related Interstitial Lung Diseases**

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	International Archives of Integrated M	ledicine, Vol. 6, Issue 8, August, 2019.
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	Available online at <u>h</u> t	tp://iaimjournal.com/
June 1	ISSN: 2394-0026 (P)	ISSN: 2394-0034 (O)
IAIM	<b>Received on:</b> 26-07-2019	Accepted on: 09-08-2019
AIN	Source of support: Nil	Conflict of interest: None declared.
How to cite this article: Sreedevi Meka, D Ankamma Rao. Role of HRCT in Smoking Related		

Interstitial Lung Diseases. IAIM, 2019; 6(8): 78-94.

## Abstract

**Background:** Smoking induced lung diseases constitute a complex group of disorders, varying from the well-known entity of Chronic Obstructive Pulmonary Disease (COPD) to the more recently described interstitial lung diseases.

Aim and objectives: The aim of the study was to delineate the relation between cigarette smoking and the development of interstitial lung disease with declaration of the different types of the interstitial lung associated with smoking.

**Materials and methods:** This was prospective study consisting of patients with smoking history referred to Department of Radiodiagnosis of NRIGH for HRCT with symptoms of progressive dyspnea. Inclusion criteria were smokers with progressive dyspnea, (age range 30-90 years, mean age 54 years  $\pm$  8). HRCT was done to all subjects using GE Light Speed Multislice 4 channels. CT scan examination was performed using GE Light Speed Multislice 4 channels present in our radiology department. The examination was done in supine position. A scout was taken with kV 120 and mA 120, then helical scanning was done in caudo-cranial direction to minimize respiration artifacts, using detector row 4, helical thickness 1.25, pitch 1.5:1, speed (mm/rot) 7.5, Detector configuration  $4 \cdot 1.25$ , beam collimation 5.00 mm, interval 1.00, gantry tilt 0.0, FOV depends on the patients' body build, but is about 35 cm, kV 120–140, mA 120–160, total exposure time about 16 s during breath hold in inspiration. The images acquired were sent to a separate workstation to be processed, manipulated and reconstructed. Reconstruction of the images was done using reconstruction software available at the workstation to attain HRCT axial, coronal and sagittal images. Also mediastinal window images were done for each case.

**Results:** In the present study, the most common seen HRCT pattern was Idiopathic Pulmonary Fibrosis, most affected gender were males and most affected age group was between 60-70 years.

**Conclusion:** In the appropriate clinical evaluation and in the presence of typical findings, high - resolution CT plays an essential role in evaluation and definite diagnosis of Smoking Related – Interstitial Lung Disease, and this may obviate further testing. However, lung biopsy may be needed when the high-resolution CT are relatively non-specific or when a confident definitive diagnosis is needed.

## Key words

Respiratory Bronchiolitis, Desquamative Interstitial Pneumonia, Pulmonary Langerhans Cell Histiocytosis, High Resolution Computed Tomography, Smoking Related Interstitial Lung Diseases.

## Introduction

Smoking induced lung diseases constitute a complex group of disorders, varying from the well-known entity of Chronic Obstructive Pulmonary Disease (COPD) to the more recently described interstitial lung diseases (ILDs) [1, 2]. Development of interstitial lung disease is a recognized risk factor for cigarette smoking [3]. Four Interstitial lung diseases (ILDs) have been related to cigarette smoking: Respiratory Bronchiolitis (RB)-associated ILD (RB-ILD), Desquamative Interstitial Pneumonia (DIP), Idiopathic Pulmonary Fibrosis (IPF) and Histiocytosis Pulmonary Langerhans Cell (PLCH) [4]. Among patients with these disorders there is a prevalence of current or ex-smokers: over 90% in RB-ILD, 60-90% in DIP and 41-83% in IPF. Thus, cigarette smoking is to be a risk factor of great significance.

Respiratory Bronchiolitis (RB) also called "smoker's bronchiolitis,". Importantly, RB may persist in some patients for many years after stopping smoking [10]. The most common highresolution CT findings in RB-ILD are centrilobular nodules, ground-glass opacities, and thickening of the bronchial walls, which predominate in the upper lobes. Upper lobe emphysema is also commonly present.

Desquamative interstitial pneumonia (DIP) was initially described by Liebow, et al. [12]. This disease is characterized by the widespread accumulation of intra-alveolar macrophages, with usually mild interstitial reaction, although some conditions may evolve to fibrosis and endstage lung disease. Currently, it is well known that the histopathologic patterns of RB-ILD and DIP may overlap, and the key feature to differentiate both disorders is the distribution and extent of the lesions: Bronchiolocentric in the RB-ILD, or diffuse in the DIP [4]. It was previously considered that DIP was the early, cellular phase and UIP the later, fibrotic phase of the same disease [19, 20], but they are now accepted as separate clinical-pathologic entities. The predominant abnormality at high-resolution CT in patients with DIP is ground-glass opacity, which may be peripheral, patchy, or diffuse in distribution [2, 14]. A peripheral subpleural and basal predominance of ground-glass opacity is commonly seen. Honeycombing most is uncommon. Coexistent emphysema may be present. At follow-up high-resolution CT of patients receiving treatment, the ground-glass opacity may show partial or complete resolution [15, 16].

Langerhans cell Histiocytosis refers to a group of diseases of unknown etiology often recognized in childhood, in which Langerhans cell accumulations involve one or more body systems, including bone, lung, pituitary gland, mucous membranes, skin, lymph nodes, and liver. This disease is also referred to as Histiocytosis X or Eosinophilic Granuloma. The term Pulmonary Langerhans Cell Histiocytosis refers to disease in adults that affects the lung [17]. The condition is uncommon, with a prevalence of 3.4% in a series of 502 patients undergoing surgical lung biopsy for chronic diffuse infiltrative lung disease [18]. The peak occurrence is at 20-40 years of age. Approximately 25% of patients are

asymptomatic, and the disease is uncovered on a routine chest radiograph. In patients with PLCH, typical findings on chest radio- graphs include nodular or reticulonodular opacities most prominent in the middle and upper lung zones [19]. There is usually sparing of the costophrenic angles and the lung volume appears normal or increased. High-resolution CT is sensitive and specific for the diagnosis of PLCH, the characteristic finding being a combination of nodules and cysts predominating in the upper and mid lungs, sparing the bases. Early in the disease, nodules with irregular borders predominate, mainly in a peribronchiolar distribution. As the disease evolves, thick- or thin-walled cysts predominate and are often irregular. Longitudinal studies with high-resolution CT show that the solid nodules progress to cavitatory nodules, then thick-walled cysts, and finally thin-walled cysts [13].

A relationship between cigarette smoking and Idiopathic Pulmonary Fibrosis has been recognized for many years [6]. IPF is also called cryptogenic fibrosing alveolitis in Europe [5]. A high prevalence of current or former smokers is noted in a series of IPF patients, varying from 41% to 83% [5]. Recent work suggests that smoking may have a detrimental effect on IPF with survival and severity-adjusted survival being higher in nonsmokers than in former smokers or in a combined group of former and current smokers. IPF is the most common form of idiopathic ILD, manifesting in the 6<sup>th</sup> -7<sup>th</sup> decades with a slight male predominance [8]. PFTs usually demonstrate a restrictive defect with reduced lung volumes and diffusing capacity [7]. The typical chest radiograph in IPF shows bilateral basal and peripheral reticular opacities. Progressive fibrosis leads to a reduction in lung volumes and honeycombing. Typical high resolution CT features allowing confident diagnosis are irregular reticular opacities, traction bronchiectasis, and honeycombing in a basal peripheral and subpleural distribution.

Identifying and determining the cause of interstitial lung disease can be challenging. CT is the key to, and sometimes the first step in, the diagnosis of ILD's. CT scanners use a computer to combine X-ray images taken from many different angles to produce cross-sectional images of internal structures. A high-resolution CT scan can be particularly helpful in differentiating various patterns of smoking related interstitial lung diseases, determining the extent of lung damage caused by interstitial lung disease. It can show details of the fibrosis, which can be helpful in narrowing the diagnosis.

## Materials and methods

This study was conducted in the Department of Radio-Diagnosis at NRI General Hospital, Chinakakani, Guntur, in 63 patients with smoking history referred for HRCT with symptoms of progressive dyspnoea and cough. The duration of study was from December 2015 to October 2017. 63 patients with smoking history referred for HRCT with symptoms of progressive dyspnoea, cough. Descriptive observational study was performed.

## Inclusion criteria

- Smokers with progressive dyspnoea and cough
- Age range 30 90 years.

## **Exclusion criteria**

- Non smokers
- Age less than 30 years.

## Study parameters being monitored

- Incidence of various pathologies among the sample of study.
- Percentage distribution of sex among various pathologies.
- Percentage distribution of age group among various pathologies.

## **Brief procedure**

Patients clinically with history of smoking and with progressive dyspnoea, cough  $\rightarrow$  HRCT Chest examination with desired protocol  $\rightarrow$ (A) Clinical diagnosis of cause for progressive dyspnea, cough; (B) Imaging findings  $\rightarrow$  Statistical analysis  $\rightarrow$  Conclusion.

#### **Examination technique**

A CT scan examination was performed using GE Light Speed Multislice 4 channels present our radiology department. The examination was done in supine position. A scout was taken with kV 120 and mA 120, then helical scanning was done in caudo-cranial direction to minimize respiration artifacts, using detector row 4, helical thickness 1.25, pitch 1.5:1, speed (mm/rot) 7.5, Detector configuration  $4 \cdot 1.25$ , beam collimation 5.00 mm, interval 1.00, gantry tilt 0.0, FOV depends on the patients' body build, but is about 35 cm, kV 120-140, mA 120-160, total exposure time about 16 s during breath hold in inspiration. The images acquired were sent to a separate workstation to be processed, manipulated and reconstructed. Reconstruction of the images was done using reconstruction software available at the workstation to attain HRCT axial, coronal and sagittal images. Also mediastinal window images were done for each case. Various imaging parameters were considered which include:

- Interlobular septal thickening
- Honey combing
- Centrilobular nodules
- Centrilobular Emphysema
- Bronchiectasis
- Cystic lesions
- Centrilobular Emphysema
- Ground glass opacities
- Reticular Lesions

#### **Results**

The present study was a prospective study carried out in the Department of Radio diagnosis, NRI Medical College, to analyze clinically suspected smoking related interstitial lung diseases using HRCT and comparing its efficacy and significance in diagnosing and characterizing various HRCT patterns. The present study sample included 63 patients who were clinically suspected have smoking related interstitial lung disease (**Table – 1 to 12, Case 1 to 8**).

<u>Table</u> -	<u>1</u> :	Percentage	Distribution	of	Age
Among H	Patier	nts Studied.			

AGE	FREQUENCY	PERCENTAGE
30-40	2	3
40-50	10	16
50-60	14	22
60-70	22	35
70-80	14	22
80-90	1	2
Total	63	100

## <u>**Table - 2:**</u> Percentage Distribution of Gender Among Patients Studied.

GENDER	FREQUENCY	PERCENTAGE
MALE	48	76
FEMALE	15	24
TOTAL	63	100

## <u>**Table - 3:**</u> Percentage Distribution of Total Cases.

TYPES OF DISEASES	FREQUENCY	PERCENTAGE
Respiratory bronchiolitis	6	10
Desquamative interstitial pneumonia	2	3
Idiopathic pulmonary fibrosis	10	16
Langerhans cell histiocytosis	1	1
Other diseases(emphysematous changes,malignancies,infective pathologies)	44	70
Total	63	100

## <u>**Table - 4:**</u> Percentage Distribution of Smoking Related Interstitial Lung Diseases.

Distribution Of Smoking Related Interstitial Lung Diseases	Frequency	Percentage
Respiratory Bronchiolitis	6	32
Desquamative Interstitial Pneumonia	2	10
Idiopathic Pulmonary Fibrosis	10	53
Langerhans Cell Histiocytosis	1	5
Total	19	100

<u><b>Table - 5</b></u> : Frequency of HRCT Findings in Total	
6 Respiratory Bronchiolitis Cases.	

HRCT FINDINGS	FREQUENCY
Centrilobular Nodules	6
Faint ground glass opacities	3
Centrilobular Emphysema	4

<u>**Table - 6:**</u> Percentage Distribution of Gender predominance in total 6 cases of Respiratory Bronchiolitis.

Gender	Frequency	Percentage
Male	6	100%
Female	0	0%
Total	6	100%

<u>**Table - 7**</u>: Percentage Distribution of HRCT Findings in Total 2 Cases of Desquamative Interstitial Pneumonia.

HRCT FINDINGS	FREQUENCY
Ground Glass Opacities	2
Prominent Interstitial Markings	1
Total cases	2

<u>**Table - 8:**</u> Percentage Distribution of Gender Predilection In Desquamative Interstitial Pneumonia.

Gender	Frequency	Percentage
Male	1	50%
Female	1	50%
Total	2	100%

## DISCUSSION

Cigarette smoking is a recognized risk factor for the development of interstitial lung disease (ILD). There is strong evidence supporting a causal role for cigarette smoking in the development of respiratory bronchiolitis ILD (RB-ILD), Desquamative interstitial pneumonitis and pulmonary Langerhans (DIP), cell Histiocytosis (PLCH). In addition, former and current smokers may be at increased risk for developing idiopathic pulmonary fibrosis (IPF). The combination of lower lung fibrosis and upper emphysema being lung is increasingly recognized as a distinct clinical entity in smokers. High-resolution computed tomography is sensitive for the detection and characterization of ILD and may allow recognition and classification of the smoking related ILDs (SR-ILDs) into distinct individual entities [7]. High resolution CT is the imaging modality of choice in patients with suspected smoking-related interstitial lung disease [11]. This is a prospective study carried out in NRI General Hospital, in 63 patients who are suspected to have smoking related interstitial lung disease. Our study was planned to recognize the different interstitial lung diseases related to cigarette smoking with the ratio of eachpathology, the incidence and the ability of HRCT to properly diagnose different lesions effectively.

# <u>**Table - 9:**</u> Frequency of HRCT Findings in Idiopathic Pulmonary Fibrosis.

HRCT FINDINGS	FREQUENCY
Subpleural prominent interlobular septae	6
Traction Bronchiectasis	10
Honeycombing	10
Reticular Opacities	5
Centrilobular Emphysema	3
Total	10

# <u>**Table – 10**</u>: Percentage Distribution of Gender Predilection In Idiopathic Pulmonary Fibrosis.

GENDER	FREQUENCY	PERCENTAGE
MALE	1	10
FEMALE	9	90
TOTAL	10	100

Out of total 63 cases of the present study, most common age group of presentation was in three

peaks. First peak was between 60-70 years of age (35%). Second and third peaks were in between 50-60 and 70-80 years (22%). 16% of cases were in between 40-50 years. 3% were between 30-40 years and 2% between 80-90 years. In present study 76% of cases were males and 24% were females.

<u>**Table - 11**</u>: Frequency of HRCT Findings in Langerhans Cell Histiocytosis.

HRCT FINDINGS	FREQUENCY
Hyperinflated lung fields	1
Variable sized cysts	1
Total cases	1

<u>**Table - 12</u>**: Frequency of Gender Predominance in Langerhans Cell Histiocytosis.</u>

GENDER	FREQUENCY
Male	1
Female	0
Total	1

Out of 63 patients, 70% were negative cases, 16% cases were idiopathic Pulmonary Fibrosis, 10% cases were Respiratory Bronchiolitis, 3% cases were Desquamative Interstitial Pneumonia, 1% cases were Langerhans Cell Histiocytosis.

Out of total 18 positive cases, 33% cases were respiratory bronchiolitis, 11% cases were Desquamative Interstitial Pneumonia, 56% cases were Idiopathic Pulmonary Fibrosis, 0% cases were Langerhans Cell Histiocytosis.

According to the literature, RB-ILD usually affects current smokers of 30-40 years of age. There is a slight male predominance. Mild cough and dyspnea are the most common presenting symptoms. Inspiratory crackles are present in one- half of patients, and digital clubbing is rare [5]. PFT results may be normal or show a mixed obstructive-restrictive pattern. Chest radiographs show diffuse, fine reticular or reticulonodular opacities in over two-thirds of the patients with RB -ILD. Ground-glass pattern may be the predominant abnormality in some patients [5]. Few studies also report normal chest radiographs in up to 28% cases.

In HRCT, respiratory bronchiolitis-associated interstitial lung disease often manifests with centrilobular ground-glass nodules, thickening of central and peripheral airways with associated centrilobular emphysema and air trapping on HRCT. These findings appear predominantly in the upper lobes [9, 10].

According to Attili, et al. [7], the most common high-resolution CT findings in RB-ILD are centrilobular nodules, ground-glass opacities, and thickening of the bronchial walls, which predominate in the upper lobes. Upper lobe emphysema is also commonly present. A small percentage of patients have a reticular pattern due to fibrosis in the absence of honeycombing and traction bronchiectasis [7].

Out of our 6 smoker patients diagnosed as RB-ILD had an age range of 44-67 years (mean age 55 years  $\pm$  5). Their main presenting symptom was progressive dyspnea. General and chest examinations revealed no significant abnormality. Blood gases were slightly impaired in all patients. All patients had mild restrictive pulmonary function changes. Three of the patients only with centrilobular emphysema had associated mild obstructive PFT changes. HRCT showed hyper inflated upper lobes and all six of patients have typical changes the of predominantly upper lobar centrilobular nodules. Three patients had also upper lobar faint ground glass opacities. Four had associated upper lobar centrilobular emphysema. One patient had small reticular lesions. None of our cases showed traction bronchiectasis or bronchiolectasis. Also, none of our patients had fibrosis.

On Imaging YoussriahYahiaSabriet al. reported In 25 patients (n = 25, 62.5%) (Age range 32–61 years, mean age 47 years  $\pm$  5) HRCT showed predominantly upper lobar hyperinflation with centrilobular nodules and peribronchial thickening, 16 of whom had also upper lobar faint ground glass opacities and 9 of whom had associated upper lobar centrilobular emphysema. On Imaging in present study, in 6 patients (n=6, 10%) (age-range 44-67 years, mean age 59 years  $\pm$  5). HRCT showed predominantly upper lobar hyperinflation with centrilobular nodules, 3 of them have upper lobar faint ground glass opacities, 4 patients have associated upper lobar centrilobular emphysema



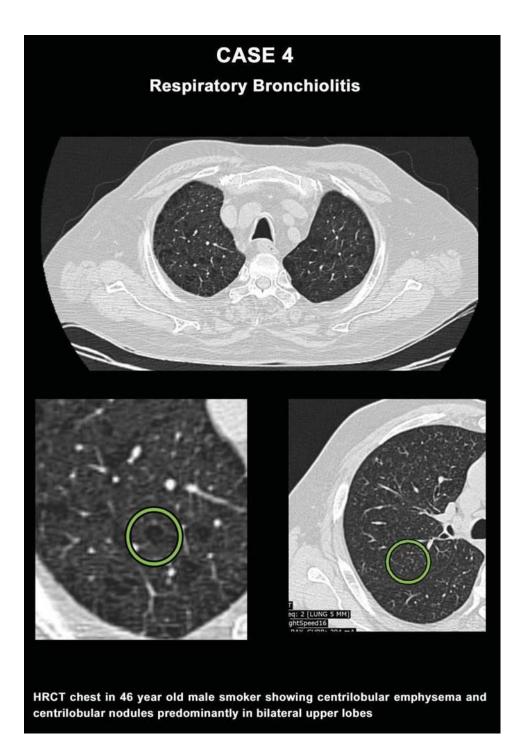


#### Desquamative interstitial pneumonia

Although the term Desquamative interstitial pneumonitis has been retained in the consensus classification of IIPs, it is considered a misnomer, as the predominant pathologic feature is the intraalveolar accumulation of pigmented macrophages and not desquamation of epithelial cells as previously thought. The condition represents the end spectrum of RB-ILD with similar pathologic findings and an almost invariable association with smoking. DIP affects cigarette smokers in their  $4^{th}$  or  $5^{th}$  decades. Males are affected nearly twice as often as females.

Dyspnea and dry cough are the most common presenting symptoms, and the onset is usually insidious. Inspiratory crackles are heard in 60% of patients, and digital clubbing occurs in nearly one-half of patients. The most common and striking PFT abnormality is marked reduction in diffusing capacity, with reductions of 50% or more being common. Restrictive defects are also common. Patients with advanced disease may have hypoxemia at rest or with exertion. Radiographically, lung volume appears reduced unless there is a co-existing obstructive airway disease such as in smokers with emphysema. Bibasilar opacities of hazy, ground-glass appearance are present in about one quarter of patients. The predominant abnormality at highresolution CT in patients with DIP is groundglass opacity, which may be peripheral, patchy, or diffuse in distribution. A peripheral sub pleural and basal predominance of ground-glass opacity is most commonly seen. Honeycombing is uncommon. Coexistent emphysema may be present [7].





Koyama, et al. [11] and Lynch, et al. [22] stated that the abnormality has a lower-zone and peripheral distribution in the majority of cases. Irregular linear opacities and a reticular pattern are frequent but are limited in extent and are usually confined to the lung bases. Honeycombing is uncommon, but well defined cysts may occur within the areas of ground-glass opacification. The cysts are usually round, thinwalled, and less than 2 cm in diameter. The ground glass opacification usually regresses with

treatment. Progression of ground glass opacification to a reticular pattern occurs infrequently (<20% of cases).

Present study involved two patients with HRCT changes suggestive of DIP. One patient is male and other is female, age range 50-80 years-old. All of the patients had upper limb clubbing, bibasal crepitation on auscultation, mixed PFT changes of obstructive and restrictive types and impaired blood gases.



HRCT showed bilateral predominantly lower lobar ground glass opacities in all of the patients. This was not associated with any cysts .One patient (50% of the cases) showed sub pleural prominent interlobular septae with mild traction bronchiectasis and traction bronchiolectasis. None of the patient showed honeycombing or centrilobular emphysema. On Imaging Youssriah Yahia Sabri, et al. [21] reported in eight patients (n = 8, 20%), (32–82 years-old, mean age 63 years $\pm$  5). 8 patients had Bilateral lower lobar subpleural ground glass opacification,3 patients had small cysts, 5 patients had upper lobar centrilobular emphysema and 3 patients had bilateral predominantly lower lobar subpleural prominent interlobular septae with traction bronchiectasis and traction bronchiolectasis, associated with multiple cysts results were all diagnostic of DIP changes.

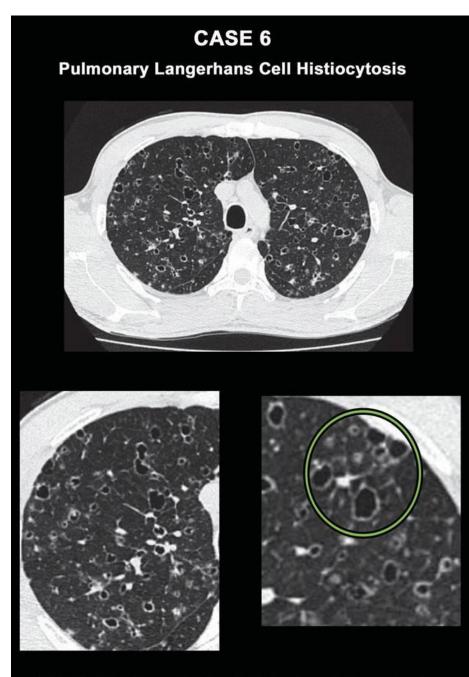
On Imaging in present study in two patients  $(n=2, 3\%), (50-80 \text{ years old}, \text{ mean age } 65 \text{ years } \pm$ 

5), 2 patients have Bilateral lower lobar subpleural ground glass opacification and 1 patient have bilateral predominantly lower lobar subpleural prominent interlobular septae.

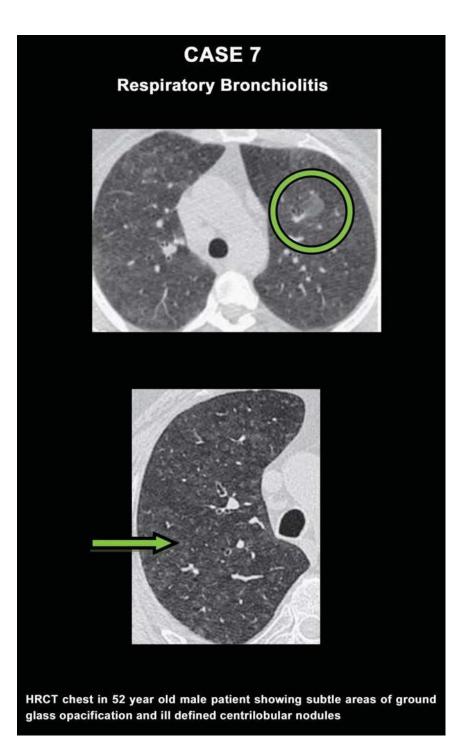
#### Idiopathic pulmonary fibrosis

Epidemiologic studies have suggested that there is some relationship between smoking and

idiopathic pulmonary fibrosis, the clinical entity associated with the pathologic diagnosis of usual interstitial pneumonia (UIP), this association is less strong than for PLCH, DIP, and RB-ILD [3, 5]. IPF is the most common form of idiopathic ILD, manifesting in the  $6^{th} - 7^{th}$  decade with a slight male predominance.



HRCT chest in 39 year old male patient showing variable sized cysts in bilateral lung fields

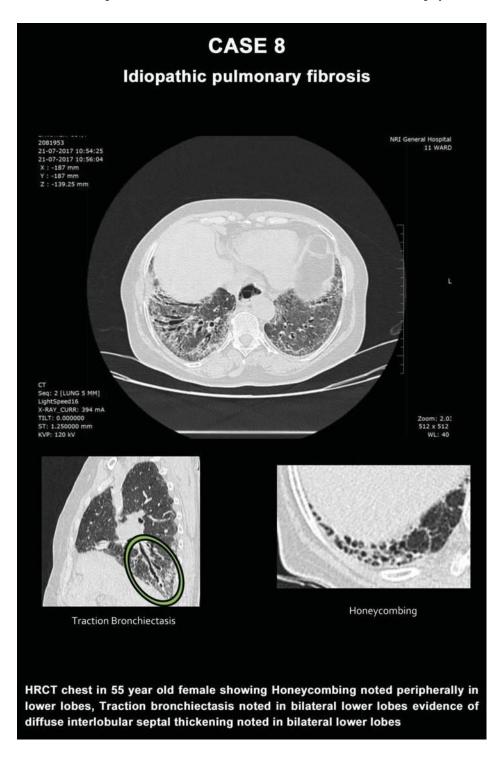


Clinical features include gradually progressing dyspnea, chronic cough, and bibasilar Inspiratory Digital clubbing crackles. is seen in approximately two thirds of patients. PFTs usually demonstrate a restrictive defect [7, 8]. radiographs demonstrates Chest bilateral reticular or reticulonodular opacities with small lung volumes [9]. There is a lower lung zone and peripheral predominance in the distribution of these infiltrates.

High-resolution CT findings consist of honeycombing, traction bronchiectasis and intra lobular interstitial thickening with sub pleural and lower lung predominance [6]. When these HRCT features are present in a proper clinical context, the diagnosis of IPF is correct in 85%-90% of patients.

10 of our patients have HRCT pattern of Idiopathic Pulmonary Fibrosis, age range

between 41-75 years, with female preponderance. HRCT features showed bilateral predominantly lower lobar sub pleural prominent interlobular septae with traction bronchiectasis and traction bronchiolectasis and areas of honeycombing. Some of the patients (30%) patients had associated upper lobar changes of centrilobular emphysema.



On Imaging Youssriah Yahia Sabri, et al. [21] reported six patients (n = 6, 15%) (32–66 years, mean age 54 year  $\pm$  5). HRCT showed bilateral predominantly lower lobar sub pleural prominent interlobular septae with traction bronchiectasis

and traction bronchiolectasis and areas of honeycombing. All those patients had associated upper lobar changes of centrilobular emphysema. On Imaging In present study in ten patients (n=10, 16%), (41-75 years, mean age 62 years  $\pm$ 

5), 6 patients had bilateral predominantly lower lobar subpleural prominent interlobular septae, 10 patients had traction bronchiectasis, 10 patients had areas of honeycombing, 5 patients had reticular opacities and 3 patients had associated upper lobar changes of centrilobular emphysema.

### Langerhan's cell histiocytosis

Adult pulmonary Langerhans' cell histiocytosisis a rare disorder of unknown etiology that occurs predominantly in young smokers, with an incidence peak at 20-40 years of age. In adults, pulmonary involvement with Langerhans' cell histiocytosis usually occurs as a single system disease and is characterized by focal Langerhans' cell granulomas infiltrating and destroying distal bronchioles.

Typical findings on chest radiograph include nodular or reticulonodular opacities most prominent in the middle and upper lung zones with sparing of the costophrenic angles. The lung volumes are either normal or increased. As the disease progresses, cystic changes and bullae appear.

High-resolution computed tomography (HRCT) of the chest is essential to the diagnosis, typically showing a combination of nodules, cavitated nodules, and thick and thin-walled cysts In this study only one patient (39 year-old) was diagnosed as PLCH. HRCT showed marked hyperinflation of both lung fields with total substitution of normal lung parenchyma by variable sized cysts. The patient had second-tothird degree clubbing of both upper limbs, markedly impaired pulmonary function with mixed obstructive and restrictive types changes, and blood gases were markedly impaired. The HRCT findings together with the clinical and laboratory results; all favored diagnosis of PLCH.

On Imaging Youssriah Yahia Sabri, et al. [21] reported one patient (30 year-old) showed marked hyperinflation of both lung fields with total substitution of normal lung parenchyma by variable sized bizarre shaped cysts, features consistent with pulmonary Langerhan's Cell Histiocytosis.

On Imaging in present study one patient (39 vear-old) showed marked hyperinflation of both lung fields with total substitution of normal lung parenchyma by variable sized cysts. Cottin and colleagues have described a "syndrome" of combined emphysema and fibrosis. Their study involved 61 patients; all smokers, with a mean age of 65 years. All had PFT changes. They named the syndrome 'combined pulmonary fibrosis and emphysema (CPFE)'. The combination of emphysema in the upper lobes and fibrosis in the lower lobes (CPFE) is being increasingly recognized as a distinct entity in smokers. Patients are almost exclusively men in their 6<sup>th</sup> and 7<sup>th</sup> decades. Lung volumes are relatively preserved despite markedly impaired diffusion capacity and hypoxemia during exercise. Honeycombing, reticular opacities, and traction bronchiectasis are the most frequent findings at high resolution CT in the lower lungs, while the upper lungs exhibit paraseptal and centrilobular emphysema [7]. In some cases of CPFE, emphysema and fibrosis may co-occur in the same area of the lung. There is a high prevalence of pulmonary hypertension in CPFE, and this is a critical determinant of prognosis. Median survival is reported to be6.1 years, better than in patients with IPF alone but worse than expected for emphysema in the absence of fibrosis. None of our cases however had pulmonary hypertension. In the present study, there are 0% cases of combined pulmonary fibrosis and emphysema

## Conclusion

In the appropriate clinical evaluation and in the presence of typical findings, high -resolution CT plays an essential role in evaluation and definite diagnosis of SR-ILD, and this may obviate further testing. However, lung biopsy may be needed when the high-resolution CT are relatively non-specific or when a confident definitive diagnosis is needed.

The effects of cigarette smoking on the development of interstitial lung diseases are poorly understood. Due to rarity of some of these interstitial lung diseases, it is difficult to firmly establish a direct causative role for smoking in the pathogenesis of these diseases. Nonetheless, available epidemiological data suggest that smoking is causally related to the development of certain interstitial lung diseases, including RBILD, DIP and PLCH. The preponderance of smokers has been consistently observed in the reported case series of patients with these disorders. In these patients smoking cessation may prove to be the most important and effective therapeutic option and should be strongly encouraged.

The role of smoking in the pathogenesis of IPF is controversial. Cigarrette smoking appears to increase the risk of development of IPF, but there is no convincing evidence that smoking directly leads to development of IPF.

We can conclude from this study that HRCT is highly recommended for diagnosing and follow up of smoking related interstitial lung disease and is considered to be the best diagnostic modality.

## References

- Baumgarten KB, Samet JM, Sitdley CA, et al. Cigarette smoking: a risk factor for idiopathic pulmonary fibrosis. Am J RespirCrit Care Med., 1997; 155: 242–8.
- Heynemann LE, Ward S, Lynch DA, et al. Respiratory bron-chiolitis, respiratory bronchiolitis-associated interstitial lung dis-ease, and desquamative interstitial pneumonia: different entitiesor part of the spectrum of the same disease process? AJR Am J Roentgenol., 1999; 173: 1617–22.
- Caminati A, Harari S. Smoking-Related Interstitial Pneumonias and Pulmonary Langerhans Cell Histiocytosis. Proceedings of the American Thoracic Society, 2006; vol.3.

- 4. Selman Moise's. The spectrum of smoking-related interstitial lungdisorders the never-ending story of smoke and disease. Chest, 2003; 124: 1185–7.
- Ryu JH, Colby TV, Hartman TE. Smoking-related interstitial lung diseases: a concise review. Eur Respir J., 2001; 17: 122–32.
- 6. Hidalgo A, Franquet T, Gime´nez A, Bordes R, Pineda R, et al. Smoking related interstitial lung diseases: radiologic-pathologic correlation. Eur Radiol., 2006; 16(8): 2463–70.
- Attili AK, Kazerooni EA, Gross Barry H, Flaherty Kevin R, Myers Jeffrey L, Martinez Fernando J. Smoking-related intersti-tial lung disease: radiologic clinical-pathologic correlation. Radio Graphics, 2008; 28: 1383–96.
- Gross TJ, Hunninghake GW. Idiopathic pulmonary fibrosis. N Engl J Med., 2001; 345: 517–25.
- Hubbard R, Venn A, Lewis S, et al. Lung cancer and cryptogenic fibrosing alveolitis: a population-based cohort study. Am J Respir Crit Care Med., 2000; 161: 5–8.
- Fraig M, Shreesha U, Savici D, et al. Respiratory bronchiolitis: a clinicopathologic study in current smokers, ex-smokers, and never smokers. Am J Surg Pathol., 2002; 26: 647–53.
- Koyama M, Johkoh T, Honda O, et al. Chronic cystic lung disease: diagnostic accuracy of high-resolution CT in 92 patients. AJR Am J Roentgenol., 2003; 180: 827–35.
- Liebow AA, Steer A, Billingsley JG. Desquamative interstitial pneumonia. Am J Med., 1965; 39: 369–404.
- Brauner MW, Grenier P, Tijani K, et al. Pulmonary Langerhans cell histiocytosis: evolution of lesions on CT scans. Radiology, 1997; 204: 497–502.
- 14. Hartman TE, Primack SL, Swensen SJ, et al. Desquamative interstitial pneumonia: thin-section CT findings in

22 patients. Radiology, 1993; 187: 787-90.

- Akira M, Yamamoto S, Hara H, et al. Serial computed tomographic evaluation in desquamative interstitial pneumonia. Thorax, 1997; 52: 333–7.
- Hartman TE, Primack SL, Kang EY, et al. Disease progression in usual interstitial pneumonia compared with desquamative interstitial pneumonia: assessment with serial CT. Chest, 1996; 110: 378–82.
- Vassallo R, Ryu JH, Colby TV, et al. Pulmonary Langerhans' cell histiocytosis. N Engl J Med., 2000; 342: 1969–78.
- Gaensler EA, Carrington CB. Open biopsy for chronic diffuse infiltrative lung disease: clinical, roentgenographic, and physiological correlations in 502 patients. Ann Thorac Surg., 1980; 30: 411–26.

- Harari S, Comel A. Pulmonary Langerhans cell histiocytosis. Sarcoidosis Vasc Diffuse Lung Dis., 2001; 18: 253–62.
- Sundar KM, Gosselin MV, Chung HL. Pulmonary Langerhans cell histiocytosis: emerging concepts in pathobiology, radiology, and clinical evolution of disease. Chest, 2003; 123: 1673–83.
- Youssriah Yahia Sabri, et al. Smoking Related Interstitial Lung Disease-High Resolution Computed Tomography (HRCT) findings in 40 smokers. The Egyptian Journal Of Radiology and nuclear medicine, 2014; 45(2).
- 22. Lynch DA, Travis WD, Muller NL, et al. Idiopathic interstitial pneumonias: CT features. Radiology, 2005; 236: 10–21.