

Role of High Resolution Computed Tomography (HRCT) in the Clinico-Radiological Study of Diffuse Parenchymal Lung Diseases (DPLD) and in Disease Progression of Tuberculosis (TB)

Sumit Sharma¹, R. Chidambaram²

^{1,2}Department of Radiodiagnosis, Sri Lakshmi Narayana Institute of Medical Sciences Affiliated to Bharath Institute of Higher Education and Research, Chennai, Tamil Nadu, India.

ABSTRACT

To detect and study the various computed tomographic patterns of diffuse parenchymal lung diseases. To assess disease progression of Tuberculosis based on the HRCT findings. To evaluate disease prognosis and reversibility by quantification of data derived from HRCT and to compare it with clinical and functional impairment as evaluated by pulmonary function tests. To correlate the HRCT profile with a histopathological diagnosis, wherever possible. The present study concludes that high resolution computed tomography is an invaluable tool in the diagnosis and characterization of diffuse parenchymal lung diseases especially in the study of disease progression in Tuberculosis, in an appropriate clinical setting.

Keywords:

high resolution computed tomography, lung diseases, tuberculosis

1. Introduction

Diffuse parenchymal lung disease (DPLD) describes a heterogeneous group of disorders of the lower respiratory tract characterized by inflammation and derangement of the interstitium and loss of functional alveolar units. The disease is not restricted to interstitium alone; as it involves epithelial, endothelial and mesenchymal cells with the disease process extending into the alveoli, acini and bronchioles. Thus, the entire pulmonary parenchyma is involved. [1,2] It has been nearly 70 years since Hamman and Rich described the first cases of progressive pulmonary fibrosis that resulted in death. Since then, many acute and chronic lung diseases with variable degrees of pulmonary fibrosis have been described and commonly referred to as interstitial lung diseases. [3]

Diffuse parenchymal lung diseases include a wide spectrum of diseases comprising more than 200 entities. Though the etiology may vary vastly, the clinical signs and symptoms differ little from one condition to another. Majority of patients are middle aged and present typically with progressive dyspnea and a dry unproductive cough. [4] While the rate of symptomatic progression in diffuse parenchymal lung disease is variable, the symptoms are usually chronic, ranging from few months to many years. Lung function tests typically show a reduction in the static lung volume, decreased pulmonary compliance and a reduction in diffusing capacity; but this may vary. [5,6]

Tuberculosis is the leading cause of morbidity and mortality in India and abroad. There is a resurgence of Tuberculosis infection seen now in view of the AIDS pandemic worldwide. Radiological imaging plays an important role in the evaluation of DPLDs including TB. [7-9] Patients with suspected DPLD usually have a chest radiograph as the initial imaging investigation. In most cases, this is abnormal and occasionally the radiographic appearances are

sufficiently characteristic to enable a specific diagnosis to be made in conjunction with the clinical and laboratory findings. The chest radiograph pattern is, however, not specific in most patients. Also in a small proportion of patients with DPLD, the chest radiograph may be normal. [10] Conventional computed tomography of the chest provides a two-dimensional representation of a three-dimensional cross-sectional slice of the lung. Although it allows assessment of the entire chest, it has limited ability to demonstrate fine parenchymal detail because all the structures within the thickness of the slice are averaged to produce the image.[11] High resolution computed tomography (HRCT) scanning is currently the most accurate non-invasive modality for evaluating the lung parenchyma. It is capable of imaging the lung parenchyma with excellent spatial resolution and providing anatomical detail similar to that seen by gross pathological examination. The modifications of the CT technique that make it one of high resolution are the use of thin sections and image reconstruction with a high spatial frequency algorithm. The added value of HRCT scanning in DPLD depends upon its ability to increase confidence of a specific diagnosis, to alter patient management and if possible, to influence outcome. However, optimal technique and knowledgeable pattern recognition of diseases are the prerequisites to use the potential of the modality to its full advantage.[12,13]

The addition of volume scanning allows data to be acquired through broad regions of interest during different phases of respiration or under different physiologic conditions. Quantitative image analysis of CT data may be useful in diffuse lung disease either to detect the nature of airway or parenchymal abnormality or to quantify the functional impairment and assess drug treatment efficacy. However, it requires meticulous imaging techniques to achieve accurate reproducible measurement.

Chest radiography remains the first imaging technique in the evaluation of pulmonary TB. HRCT is needed for further evaluation and confirmation of the radiographic findings. Histopathology has been regarded as the gold standard in the definite diagnosis of DPLDs including TB. AFB cultures are used to diagnose active M. tuberculosis infections or infections due to non-tuberculous mycobacteria. The role of lung biopsy remains vital in understanding the pathogenesis of DPLDs and therapeutic intervention. However, the cost effectiveness and risks associated with it must be weighed against the potential benefits in an individual case.[14,15] The proposed study is an endeavour to study the clinico-radiological profile of DPLDs in our hospital set up. It is also our purpose to evaluate the exact role of HRCT in forming a definitive diagnosis and studying disease progression of Tuberculosis; and to correlate the spectrum of findings with the functional impairment and histopathological findings, wherever possible.

2. Materials and Methods

Source of data

Study Population: The study group included a total of 60 patients of all age group with suspected clinical diagnoses of diffuse parenchymal lung diseases including Tuberculosis and AFB-positive Tuberculosis. The study was conducted in the Department of Radiodiagnosis, Sri Lakshmi Narayana Institute of Medical Sciences in association with the Department of Medicine and Department of Pathology.

Duration of Study:The study was conducted for a period of 2 years

Study Design: Prospective Study

Inclusion Criteria: Patients of all age group presenting with complaints of chest symptoms of shortness of breath at rest or with exertion and dry cough and known cases of -

- Idiopathic Interstitial Pneumonias and Hypersensitivity Pneumonias
- Pulmonary tuberculosis – primary, post-primary and TB sequelae including newly diagnosed cases on treatment, with or without positive chest radiograph findings and sputum positive / negative AFB.
- Collagen Vascular Diseases
- Systemic Vasculitides
- Industrial Exposure Related Diseases
- Medication, Drugs and Radiation Exposure Related Cases
- Allergic Bronchopulmonary Aspergillosis, Invasive Aspergillosis and Lymphangitic Spread of Tumours
- **Exclusion Criteria:**
- Lung Anomalies

Study method:

All the patients were evaluated along the following lines and findings were recorded on a separate proforma.

1) Clinical assessment:

A detailed clinical history was elicited from each patient which included relevant symptoms, occupational and environmental history and smoking habits. Findings of general and systemic examination were noted in each patient.

2) Lab Investigations:

Relevant laboratory investigations were recorded in each case. In case of known and suspected cases of TB, sputum AFB examinations / bronchoalveolar lavage followed by culture was done to segregate active from inactive TB.

3) Pulmonary function testing:

Detailed pulmonary function tests were done in the Department of Medicine in all patients presenting with dyspnea. The tests included-

- a) Forced Vital Capacity (FVC) and Forced Expiratory Volume (FEV₁) in one second measured by spirometry.
- b) Total lung capacity measured by helium dilution method.
- c) Diffusion capacity for carbon monoxide measured by a single breath method (DL_{co})

Out of the 47 patients presenting with dyspnea, only 41 could perform the spirometric examination. 6 patients were unable to perform the spirometric examination because of marked dyspnea. 4 patients were not able to perform DL_{co} because of very low forced vital capacity.

The results were expressed as a percentage of the predictive value.

4) Radiological evaluation:

After the clinical assessment, a detailed radiological examination was performed in each patient. This included the following imaging studies –

A) COMPUTERISED RADIOGRAPHY (CR)

Standard postero-anterior radiographs of the chest were obtained in all cases using Fujifilm Dry Pix CR System.

Radiographs were evaluated to detect the involvement of both lung fields by the diffuse lung diseases. The following features were noted:

1. Zonal distribution of disease- Upper, middle, lower or diffuse involvement
2. Predominant pattern of disease – reticular, nodular, reticonodular, alveolar opacities, cystic lesions and/or bronchiectatic changes.
3. Presence of honeycombing.
4. Presence of pleural thickening/effusion.
5. Volume of the lung fields.
6. Associated abnormalities like the presence of cardiomegaly and mediastinal and/or hilar lymphadenopathy.

B) COMPUTED TOMOGRAPHY OF THE CHEST

Non-enhanced spiral axial computed tomographic scans of the chest were obtained in each patient on Toshiba Asteion and Siemens Somatom ScopeSpiral CT scanners. Patients were instructed to come after overnight fasting on the day of the examination. Images were obtained using helical data acquisition with 8 mm sections using a pitch of 1-1.5 mm in a caudocranial direction giving intravenous contrast bolus (60-80ml of iodinated contrast) wherever necessary. Non-ionic contrast was used wherever indicated. Patients were asked to inspire fully and hold their breath while the data acquisition was completed. Thinner sections were taken in children. Images were evaluated on both mediastinum and lung window settings. The images were reconstructed using a high spatial frequency or bone algorithm. The exact technique was customised for individual cases.

The following aspects of the lung parenchyma were evaluated:-

- 1) Large bronchi and vessels
- 2) Secondary pulmonary lobule and its components.
 - a) Interlobular septae
 - b) Centrilobular region and core structures
 - c) Lobular parenchyma
 - d) Pulmonary acinus
- 3) Lung interstitium
 - a) Axial –
 - I. Peribronchovascular interstitium

- II. Centrilobular interstitium
- b) Peripheral –
- I. Subpleural interstitium
- II. Interlobular interstitium
- c) Intralobular interstitium

The following features of lung involvement by diffuse parenchymal lung diseases were noted: -

1. Distribution of the disease – central, peripheral or diffuse.
2. Predominant lobe involvement – upper, middle, lower or diffuse
3. Predominant pattern of disease – reticular, nodular, reticulonodular, alveolar opacities, bronchiectasis or presence of cystic lesion.
4. Presence and type of septal thickening.
5. Presence and distribution of honeycombing and its associated findings – traction bronchiectasis and conglomerate fibrosis.
6. Presence of associated findings – pleural thickening or effusion, pneumothorax, mediastinal and / or hilar lymphadenopathy, presence of cardiomegaly and the size of pulmonary arteries and presence of esophageal dilatation in patients with progressive systemic sclerosis.
7. The findings were recorded in a tabulated manner and a differential diagnoses based on the HRCT findings were made.

Quantitative estimation of the extent and distribution of lung disease was done by taking spirometrically gated scans of the chest at three levels- carina, and 5 cm above and below it, in 30 patients with diffuse parenchymal lung diseases. The scans were spirometrically triggered at 50% of the vital capacity. Each scan was later evaluated using the Pulmo- CT software.

The extent of disease on HRCT scans, assessed by the mean attenuation value of the lung, was correlated with the severity of dyspnea and pulmonary function results.

Histopathological evaluation

Histopathological examination was done in 20 patients with DPLD. Sputum AFB smear test followed by culture was done in suspected and known cases of TB. Out of the 15 such patients, seven were found to be sputum positive for AFB.

Three cases of progressive systemic sclerosis were proved by skin biopsy. Two cases of suspected hypersensitivity pneumonitis were proven by bronchial aspirate findings.

One case each of UIP, Histiocytosis X and Sarcoidosis were proven on biopsy.

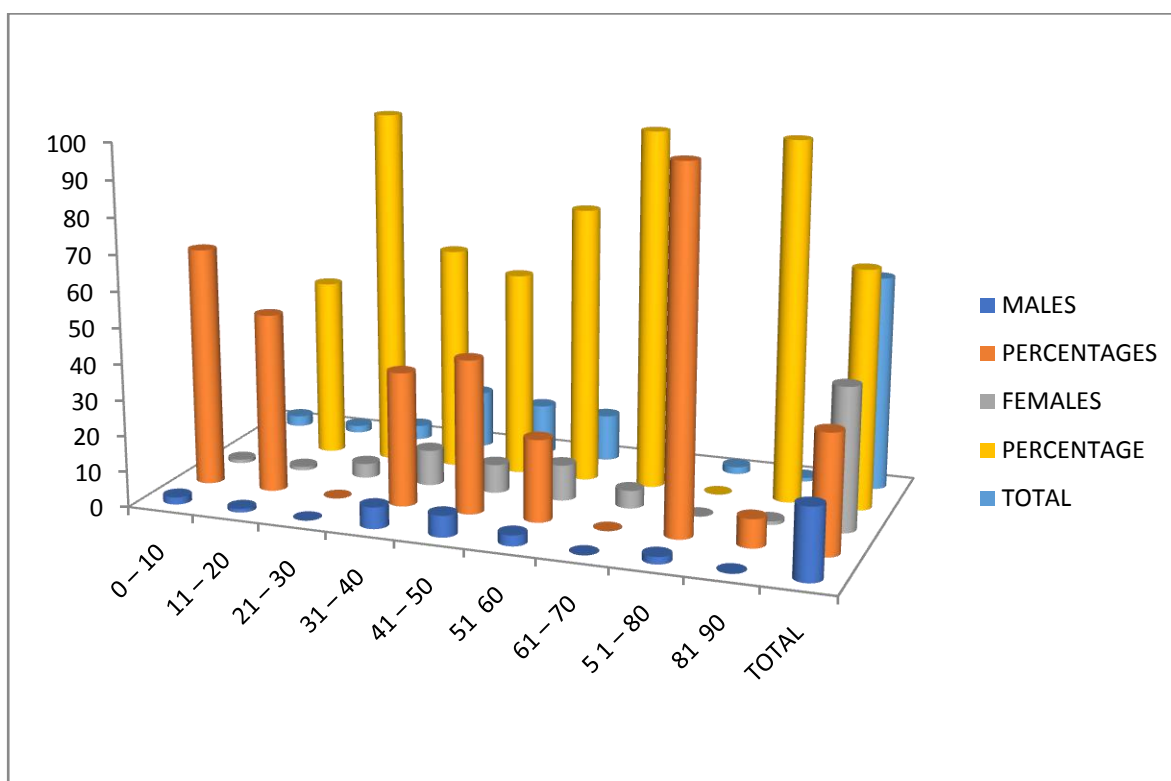
3. Results

The study included 60 patients with a clinically suspected diagnosis of diffuse parenchymal lung disease including TB and referred for thoracic computed tomography.

Table1. Distribution of patients (n=60)

PATHOLOGY	NO. OF PATIENTS	PERCENTAGE(%)
IDIOPATHIC PULMONARY FIBROSIS (INCLUDING CHRONIC INTERSTITIAL PNEUMONIAS)	17	28.3
TUBERCULOSIS – PRIMARY, POST-PRIMARY INCLUDING MILIARY TUBERCULOSIS AND TB SEQUELAE	15	25
SARCOIDOSIS	6	10
CONNECTIVE TISSUE DISORDERS (INCLUDING SYSTEMIC SCLEROSIS AND OTHER DISEASES)	5	8.3
HYPERSENSITIVITY PNEUMONITIS	4	6.6
BRONCHIECTASIS	4	6.6
ALLERGIC BRONCHOPULMONARY ASPERGILLOSIS	2	3.3
BRONCHIOLITIS OBLITERANS ORGANIZING PNEUMONIA	2	3.3
RADIATION INDUCED ILD	1	1.6
DIFFUSE METASTASES	1	1.6
SILICOSIS	1	1.6
HISTIOCYTOSIS X	1	1.6
LYMPHANGIOLEIOMYOMATOSIS	1	1.6
ACUTE INTERSTITIAL PNEUMONITIS	1	1.6

Figure 1. Age and Sex distribution of patients

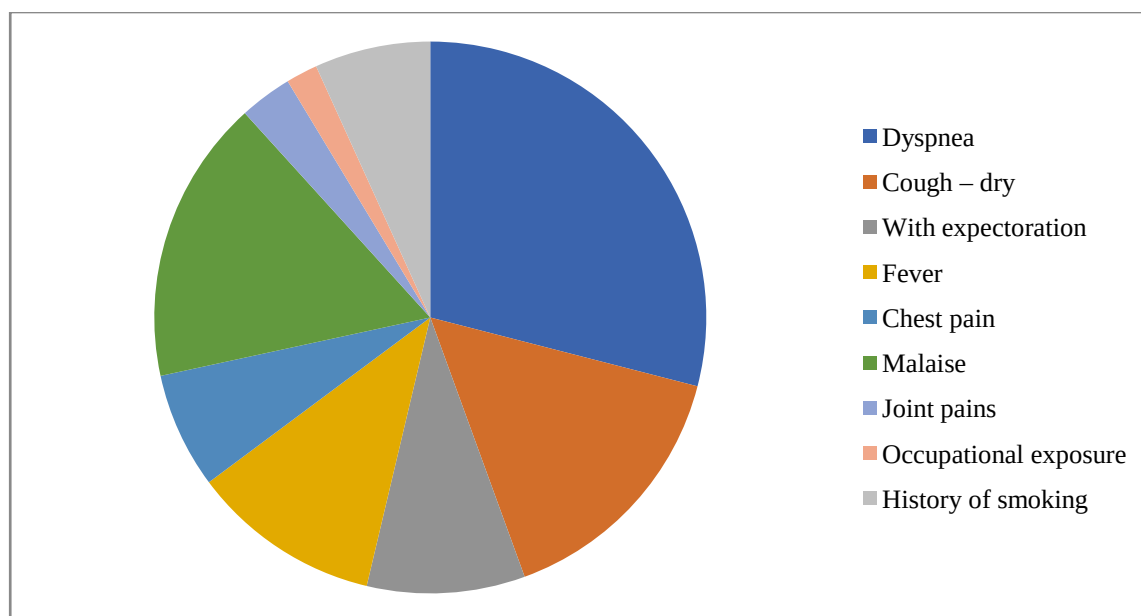


- Most of the patients presented in the fourth and fifth decades of life.
- The mean age of the patients in our study was 44.5 years.
- There was a preponderance of female patients in our study group comprising 66.7% of the total patients.

Table2.Clinical symptoms present in patients

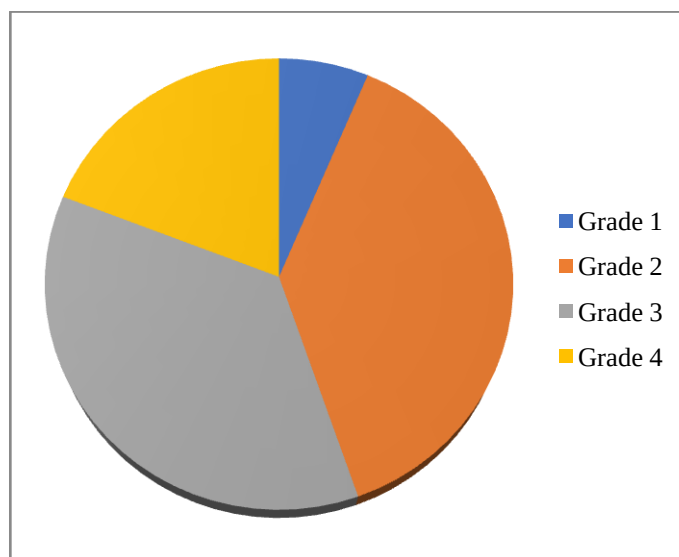
CLINICAL SYMPTOMS	NO. OF PATIENTS (n=60)	PERCENTAGE
Dyspnea	47	78.3
Cough – dry	25	40.6
With expectoration	15	25
Fever	18	30
Chest pain	11	18.3
Malaise	27	45
Joint pains	5	8.3
Occupational exposure	3	5
History of smoking	11	18.3

Figure2.Presenting Clinical Symptoms



Dyspnea was the most common presenting symptom in the patients (78.3% of cases). Dry cough was the most frequently associated symptom with dyspnea present in 41.6% of patients. Joint pains were mainly present in patients with connective tissue disorders and sarcoidosis. Fever was the main presenting symptom in patients with tuberculosis. Acute onset chest pain presenting symptom in patients with tuberculosis. Acute onset chest pain along with breathlessness was present in patients with Histiocytosis X and Lymphangioleiomyomatosis secondary to pneumothorax. Three patients presented with a history of occupational exposure – one was a tin factory worker, one had a history of exposure to silica dust and another worked in an asbestos tile factory. History of smoking was present in 11 of the 60 patients. All of these patients were male patients.

Figure 3. Grades of Dyspnea



- The severity of dyspnea was graded from 0 to 4 (Staples et al,1987)-
Grade 0 - No dyspnea
Grade 1 - Dyspnea following strenuous activity
Grade 3 - Dyspnea following moderate activity
Grade 4 - Dyspnea at rest
- 74 percent of the patients had grade 2 or 3 dyspnea at presentation.

Table3.Physical findings present in patients

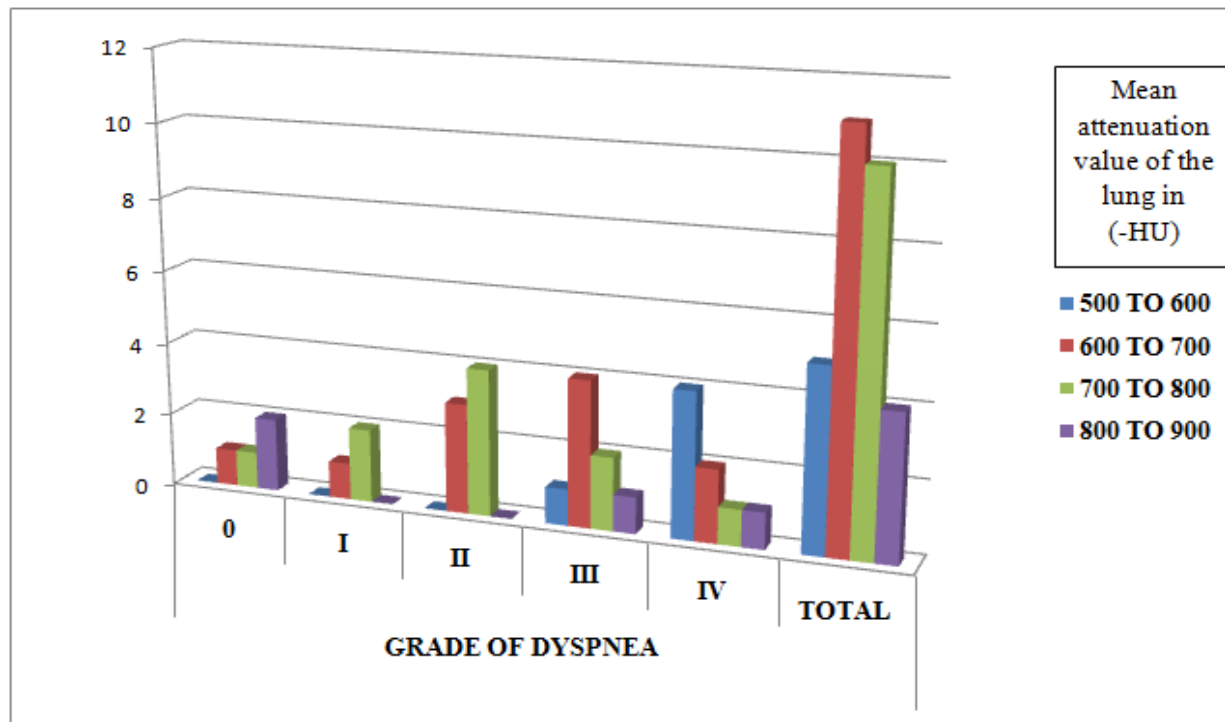
PHYSICAL EXAM FINDINGS	NO. OF PATIENTS	PERCENTAGE
Inspiratory crackles	54	90
Rhonchi	4	6.6
Clubbing	11	18.3
Cyanosis	12	20
Raised JVP	9	15
Hepatomegaly	6	10
Ascites	1	1.6
Cervical, axillary lymphadenopathy	1	1.6

Table4. Clinico-functional correlation between PFT findings and HRCT findings(n=26)

MEAN LUNG DENSITY (- HU)	DLco(% PREDICTED)				
	<40%	41-60%	61 – 80%	>80%	TOTAL
-500 TO- 600	3	2	0	0	5
-600 TO -700	2	6	4	0	12
-700 TO- 800	1	2	4	1	8
-800 TO -900	0	0	0	1	1

- Dyspnea was the presenting symptom in 47 out of 60 patients with DPLD Out of this pulmonary function tests were performed in 41 patients. 6 patients were unable to perform the spirometric test.
- Out of the 41 patients with PFT, DLco was performed in 37 patients, 4 patients were unable to perform this test.
- Spirometric gated Pulmo-CT was performed in 26 patients with PFT and HRCT evidence of DPLD.
- A statistically significant but low correlation between the mean attenuation value of the lung and diffusion capacity for carbon monoxide (DLco) was found in patients with DPLD. (r value – 0.452).
- An increase in lung density corresponded with reduced gas transfer measured by DLco.
- 76% of patients with lung density above – 700 HU had DLco below 60%.

Figure 4. Correlation of HRCT with Dyspnea



- Dyspnea was present in 47 out of the total 60 patients – CT was performed in 30 out of the total 60 patients including those who did not present with dyspnea. Patients with very advanced lung disease and those who were very dyspneic were not able to perform this test.
- An increase in the grade of dyspnea was manifested by an increase in lung density measurement.
- 80% of patients with attenuation value between -500 to -600 HU presented with Grade 4 dyspnea
- 11 out of 16 patients with grade 3 and 4 dyspnea had lung density above 700 HU.
- A statistically significant correlation was found between the mean lung density and the severity of dyspnea. (r value- 0.488).
- Histopathological findings
- Histopathological examination was done in 20 patients with DPLD: 15 of these were via the transbronchial route.
- Seven cases of active tuberculosis showed acid fast bacilli in their sputum smear.
- Three cases of progressive systemic sclerosis were proved by skin biopsy.
- Two cases of hypersensitivity pneumonitis were proven by bronchial aspirate findings of chronic inflammatory cells (lymphocytes) and intra-alveolar exudates.
- One case of carcinoma lung was proven by transbronchial biopsy earlier before radiation therapy.
- Two cases of allergic bronchopulmonary aspergillosis were proven by serum positive for IgG for *Aspergillus fumigatus*. In one case, the sputum culture was positive for *Aspergillus fumigatus*.

Table5.Findings on Histopathology

HISTOPATHOLOGICAL FINDING	NO. OF CASES
Normal lung tissue	4
Non-specific interstitial pneumonitis	6
Acid Fast Bacilli	7
Interstitial fibrosis	8
Non-caseating granulomas	3
Chronic inflammatory cells	5
Stellate cells	1
Squamous metaplasia of the bronchial epithelium	1

4. Pattern Wise Presentation

Figure 5: Predominantly Reticular

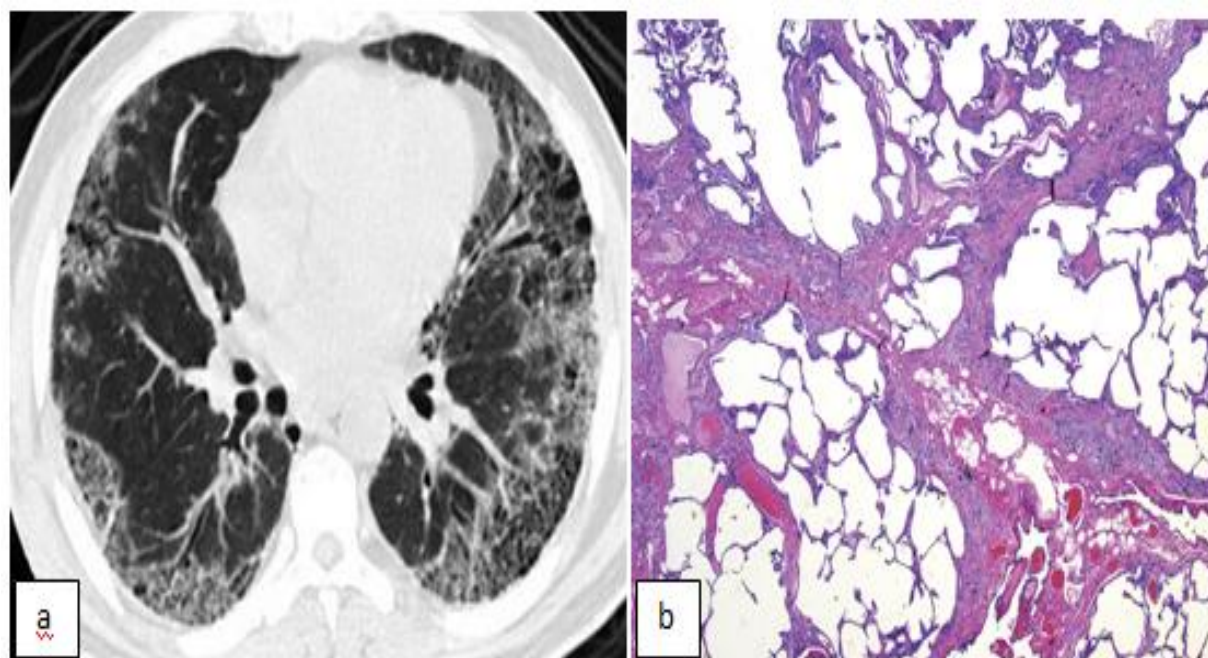


Figure 5a) HRCT lung shows usual interstitial pneumonia basal and subpleural distribution of reticular opacities, traction bronchiectasis and honeycombing in case of a patient with Usual Interstitial Pneumonia. b) Histology shows irregular septal fibrosis, with relative centrilobular sparing. Traction emphysema is present within the lobules, causing dilatation of alveolar spaces.

Figure 6HRCT image shows thick walled cystic bronchiectasis in a patient with a known history of TB largely limited to the right paracardiac region.

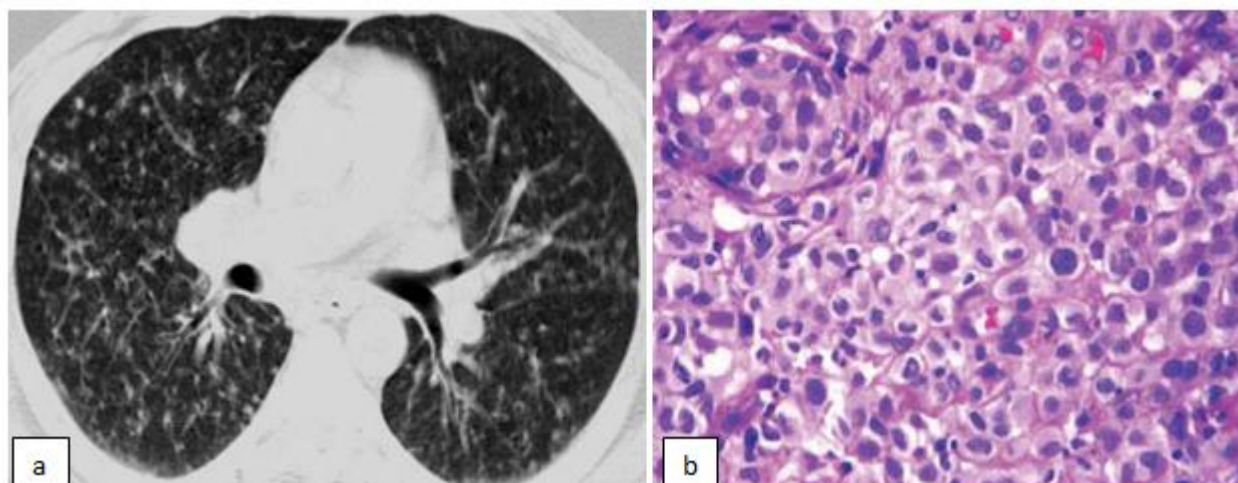


Figure 7a) HRCT image of a 7-year-old with dyspnea and cough , shows thin/thick walled cysts in various shapes smaller than 4mm present in the bilateral mid and upper lung zones. b) Diffuse infiltrates by the Langerhans cells permeated by lymphocytes, neutrophils and eosinophils are seen in this photomicrograph. Findings are suggestive of Histiocytosis X.

Figure 7: Predominantly Nodular pattern

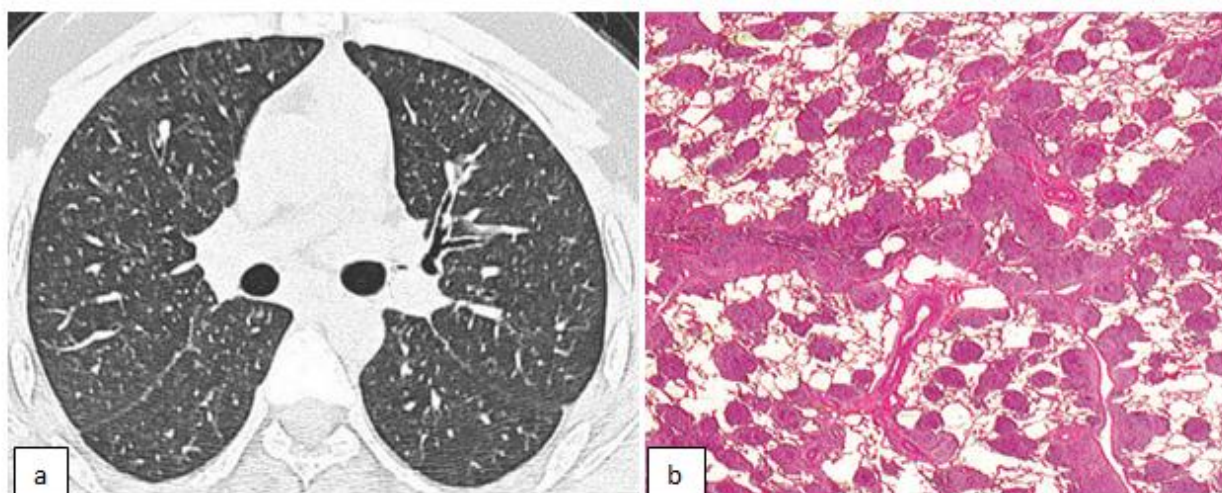


Figure 8 a) HRCT image of a patient with sarcoidosis shows small well-defined centrilobular nodules with perilymphatic distribution and symmetrical lymph node enlargement. b) Extensive pleural and interlobular septal lymphatic granulomas of sarcoidosis can be seen in this photomicrograph of the same patient. Nodules are also present at the center of lobules along bronchovascular bundles where lymphatics also traverse the lung.

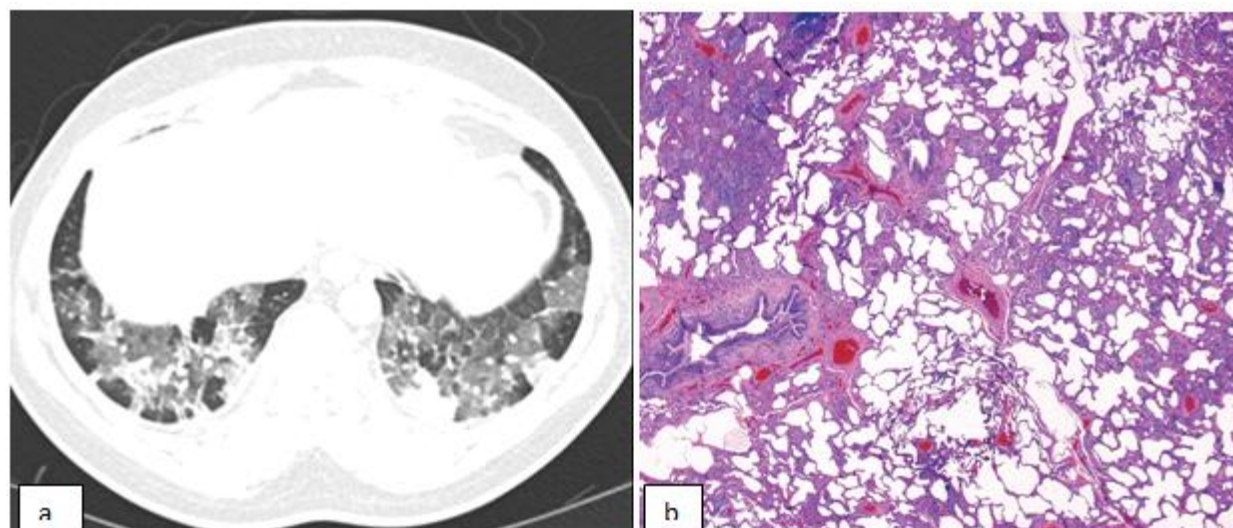


Figure8 a) HRCT image in a sand-quarry worker with Hypersensitivity Pneumonitis shows patchy ground glass opacity with small centrilobular nodules and lobular areas of mosaic perfusion (Headcheese Sign). b) In this photomicrograph from the same patient, the primarily lymphoplasmacytic infiltrates are irregularly distributed around the terminal airways.

5. Discussion

The present study was undertaken to evaluate the clinico-radiological profile of diffuse parenchymal lung diseases using high-resolution computed tomography in our hospital set up. Sixty patients with suspected clinical diagnosis of DPLD including TB and those referred for thoracic CT were included in the study. [16] The most common diffuse parenchymal lung disease encountered in our study group was idiopathic pulmonary fibrosis (IPF) including chronic interstitial pneumonias accounting for 28.3% of cases. Coultas et al reported that IPF accounts for 25-50% of total DPLDs. (123). Tuberculosis formed the second major group constituting 15 cases of sputum positive/negative for AFB. Miliary tuberculosis accounted for 35% of these cases and the rest were other forms of active tuberculosis or sequelae of old disease. Six cases of sarcoidosis accounting for 10 % of cases were also seen.[17] The youngest patient in our study was 3 years old and the oldest 86 years. The mean age of the patients in our study was 45 years. There was a female preponderance in our study (66.7% of the total cases).Dyspnea was usually associated with cough at presentation. Dry cough, was present in 42% of the patients. 25% patients had associated expectoration that was either mucoid, mucopurulent or hemoptysis. Purulent sputum was associated with the presence of bronchiechasis. Hemoptysis was present in 4 of the 7 patients (57%) with tubercular or post-tubercular DPLD. One case with a history of carcinoma lung had complained of hemoptysis earlier at presentation.[18,19] 30% of our patients had fever at the time of presentation, it was the predominant symptom in all the cases of miliary tuberculosis, 1 case with hypersensitivity pneumonitis and 1 with acute interstitial pneumonia.18% of our patients had chest pain at presentation. Acute onset chest pain and breathlessness secondary to pneumothorax was present in each case of Histiocytosis X and LAM. The other constitutional symptoms of malaise (45%) and joint pains (8.3%) were more frequently found in patients with suspected connective tissue disorders and sarcoidosis.[20]

Raised jugular venous pressure and signs of cardiac failure were present in 15% of the patients, mostly with advanced disease and developing cor pulmonale. Four patients were known cases of

heart disease. [21] Diffuse parenchymal lung diseases are characterized by a restrictive lung function, with reduction in lung volumes with preserved ratio of forced expiratory volume in one second (FEV1) to forced vital capacity (FVC) together with a reduction in carbon monoxide transfer factor (DLco). In our series, DLco was found as the most sensitive indicator of diffuse parenchymal lung disease with a mean value of 53.74% the mean value of DLco in patients the IPF was 43.2% and in patients with Sarcoidosis was 61%, the mean value of FVC was 61% and TLC 68.3% in our study group. [22] Koegh et al in their series reported the mean value of FVC as 66%, TLC 63.2% and DLco 59%, the slightly higher values a compared to our study are due to the fact that a large number of patients in our study were having severe disease. Three of our patients had FEV1 /FVC value below 70% indicating in obstructive pattern. Two of these patients were chronic smokers and one was a known case of bronchial asthma showing hyperinflated lung fields on radiological examination. [23]

Our study included 17 patients with idiopathic pulmonary fibrosis, comprising 28.3% of total cases. The age of patients ranged from 30 to 86 years with most of the cases between 50-65 years. There was a preponderance of females in patients with IPF. On chest radiographs, a reticular pattern spread throughout the lung fields was the most commonly observed finding. The lower zones were involved more often (23.5%) as compared to upper and mid zones: and in cases of diffuse disease, the findings were more profuse in the lower zones. Staples et al reported similar findings in their study. Honeycombing was observed more frequently due to the fact that most of the cases presented with advanced disease. No abnormality was identified in one case with early IPF. On HRCT scans, the predominant features observed were interlobular and intralobular septal thickening (94% cases) in a reticular pattern throughout the lung fields. Thickening of the interstitial network of the lung by field, fibrous tissue or cells primarily results in this appearance. Nishimura et al reported the incidence of septal thickening in 94% of cases. [24] though a diffuse pattern of disease was observed in 53% of cases, the lower zones and the peripheral lung fields were more commonly involved. Muller et al reported similar findings. Honeycombing was present in 88% of cases, more commonly present in the subpleural and peripheral location. Honeycombing is pathologically defined by the presence of small air-containing cystic spaces, generally lined by bronchiolar epithelium, and having thickened walls composed of fibrous tissue (Primack et al) Staples et al reported 90% incidence of honeycombing in cases of IPF identified on HRCT. [25] Traction bronchiectasis and conglomerate fibrosis were associated with the presence of honeycombing. Ground glass opacities were present in 82.35% of cases with IPF. Similar findings were reported by Remy-Jarden et al (55) Patchy ground glass opacities were the only findings in 1 case, suggestive of early disease. Ground glass haze was more frequently present in patients with lesser degrees of fibrosis. It results from volume averaging of morphologic abnormalities below the resolution of HRCT. It can reflect minimal thickening of the septal or alveolar interstitium, or presence of the cells or fluid partially filling the epithelium (Webb et al). (31) These findings suggest the presence of active reversible disease as suggested by Leung et al. [26] The chest radiographs in these patients revealed nodular or reticulonodular opacities in 62% cases with an upper and mid zone predominance (37.5% cases only). Hilar and right paratracheal lymphadenopathy was identified in 50% of the cases on chest radiography. it was the only finding on chest radiograph in 2 cases, with pulmonary involvement proven on HRCT. [27]

In one case with progressive systemic sclerosis, evidence of esophageal dilatation was seen in the mediastinal window scans. Grenier et al reported the incidence of esophageal dilatation as 40-

80% in PSS. Enlarged mediastinal nodes were seen in 60% of the cases. Similar findings were found by Bhalla et al. Cardiomegaly with a prominent pulmonary artery was seen in 3 cases. Primack et al (54) and Aroliga et al found that PSS is commonly associated with pulmonary vasculitis and pulmonary hypertension. HRCT images show enlargement of the pulmonary arteries and bilateral diffuse mosaic patterns of lung attenuation..[28]

LPG against *Aspergillus fumigatus*. Patients with bronchiectasis as the predominant finding on imaging constituted 4 cases. Most patients presented with purulent sputum production and recurrent pulmonary infections. Chest radiographic findings were non-specific in most of the cases. Multiple discrete cystic lesions with evidence of air-fluid levels were seen in two cases. Other findings included peribronchial cuffing, alveolar opacities and a reticulonodular pattern in 40% of cases. [29]

Similarly, a correlation between the mean lung density and the pulmonary function test findings (DLco) was seen in patients presenting with dyspnea. An increase in the lung density corresponded with a reduction in the diffusion capacity for carbon monoxide suggesting reduced gas transfer. 76% of the patients with mean lung density above -700 HU had reduced DLco below 60%. Statistically significant but low correlation was found between the extent of parenchymal abnormalities identified on CT and measures of functional impairment like diffusing capacity (r value -0.45). Similar findings were reported by Remy-Jardin et al. Rienmuller et al in a study of 63 patients found significant positive correlation between the CT attenuation values with the vital capacity and DLco in patients with diffuse pulmonary parenchymal diseases. [30-33]

6. Conclusion

The following conclusions can be drawn from the study diffuse parenchymal lung diseases commonly occur in the middle age, the presenting complaint being unremitting dyspnea of long duration in most cases. Idiopathic pulmonary fibrosis forms the first major group of diffuse parenchymal lung diseases in our society. Various forms of tuberculosis constitute the second major group. Bibasilar inspiratory crackles are the most common finding on physical examination. In case of Tuberculosis, HRCT findings of ill-defined nodules, consolidation, tree-in-bud appearance and cavitation are best indicators of active disease. While traction bronchiectasis, atelectasis, calcified granulomas and peribronchial cuffing are indicators of inactive disease. HRCT findings can help in the assessment of disease activity and reversibility: e.g. the presence of ground glass opacities indicates active and potentially reversible disease whereas the presence of septal thickening and fibrotic opacities indicates irreversible disease. The extent and distribution of disease identified on HRCT scans correlate well with the functional and clinical impairment in patients and with the histopathological findings. Quantitative histogram analysis of spirometrically standardized HRCT of the lungs provides objective quantitative data that may be helpful in the early diagnosis and staging of patients with diffuse parenchymal lung disease. HRCT is a useful tool in the diagnosis and management as it can differentiate active from inactive disease with greater sensitivity in case of DPLDs especially TB.

Funding: No funding sources

Ethical approval: The study was approved by the Institutional Ethics Committee

Conflict of interest

The authors declare no conflict of interest.

Acknowledgments

The encouragement and support from Bharath University, Chennai is gratefully acknowledged. For provided the laboratory facilities to carry out the research work.

References

- [1] Scadding JG, Hinson KF: Diffuse fibrosing alveolitis: Correlation with histology at biopsy with prognosis. *Thorax*; 1976;22;291.
- [2] Carrington CB, Gaensler EA, Fitzgerald MX, Gupta RG: Natural history and treated course of usual and desquamative interstitial pneumonias. *N Eng J Med*;1978;298;801-09.
- [3] Fulmer JD, Crystal RG: interstitial lung disease. *Curr pulmonology*: 1979;1;1- 65.
- [4] Burkhardt A: Alveolitis and collapse in the pathogenesis of pulmonary fibrosis. *Am Rev Respir Dis*: 1989;140;513-524.
- [5] Diagnosis, assessment and treatment of diffuse parenchymal lung diseases in adults. *Thorax*; Apr 1999;54;suppl 1;S1-14.
- [6] Gaensler EA, Carrington CB; Open biopsy for chronic diffuse infiltrative lung disease. *Ann Thorac Surg*; 1980;30;411.
- [7] David A, Lynch: HRCT of idiopathic interstitial pneumonias. *RCNA*, Nov 2001;39(6) 1153-70.
- [8] Heard BE: Pathology of interstitial lung diseases with particular reference to terminology, classification and trephine lung biopsy. *Chest*; 1976;69;252.
- [9] Schwartz DA, Van Fossen DS, Davis CS et al: Determinants of progression in IPF. *Am J Respir Crit Care Med*: 1994;149;444-9.
- [10] Spencer P: Pathology of the lung. *Pathology of the lung*; 1985; 4th edition, New York. Pergamon press; 832-835.
- [11] Friedman PJ: Pulmonary patterns – the concepts of alveolar and interstitial lung disease. In: potchen EJ; *Curr concepts in radiography*; 1990; St Louis Mosby: 64-106.
- [12] Hartman TE, Primack SL, Hansell DM, Muller NL: End-stage lung disease: CT findings in 61 patients. *Radiology*; 1993;189;681-86.
- [13] Crystal RG, Fulmer JD, Roberts WC: Idiopathic pulmonary fibrosis: Clinical, Histologic, Radiographic, Physiology and Scintigraphic aspects. *Ann Intern Med*, 1976;85;769-88.
- [14] Colby TV, Carrington CB: Lymphoreticular tumors and infiltrates of the lung. *Pathol Ann*; 1983;18;27-70.
- [15] Soler P, Morean A, Besset F; Cigarette smoking induced changes in the number and differentiated state of Pulmonary Langerhan's cells. *Am Rev Respir diseases*; 1989;139;1112-17.
- [16] Ryu JH, Colby TB, Hartman TE et al: Smoking related interstitial lung diseases: a review. *Eur Respir Journal*; 2001;17;122-32.
- [17] Koegh BA, Crystal RG: Clinical significance of pulmonary function tests in interstitial

- lung disease.Chest; 1980;78(6).
- [18]Pritchard MG, Finucaine KE: Lung function in diffuse interstitial lung disease of unknown cause.Aust NZ J Med; 1984;14;749-54.
 - [19]Mc Cloud TC, Carrington CB, Gaensler EA: Diffuse infiltrative lung diseases: a new scheme for description.Radiology: 1983;149;353-63.
 - [20]Epler GR, Mc Cloud TC, Gaensler EA et al: Normal chest roentgenograms in chronic diffuse infiltrative lung diseases. N Eng J Med; 1978;298;801-09.
 - [21]Hansell DM, Kerr IH: The role of high resolution computed tomography in the diagnosis of interstitial lung disease.Thorax; 1991;46;77-84.
 - [22]Kreel L: CT of interstitial pulmonary diseaseJ Comput Assis Tomog: 1982;6;181-99.
 - [23]Todo G, Ito H, Nakano Y et al: HRCT for the evaluation of pulmonary peripheral disorders. Jpn JClin Rad: 1982;27;1319-26
 - [24]Mayo JR, Webb WR, Gould R: HRCT of the lungs: an optimal approach.Radiology: 1987;163;509-10
 - [25]Murata K, Khan A,Rojas KA: Optimization of CT technique to demonstrate fine structures of the lung.Invest Rad; 1988;23;170-95.
 - [26]Zwirewich CV, Territt B, Muller NL: High spatial frequency (bone) algorithm improves quality of standard CT of the thorax AJR; 1989;153;1169-73.
 - [27]Griffin CB, Primack SL: HRCT – Normal anatomy, techniques and pitfalls.RCNA: Nov 2001;39(6);1073-30.
 - [28]Evans SH, Davis R, Cook J et al: A comparison of radiation doses to the breast in CT chest examination for two scanning protocols.Clin Rad; 1989;40;45-46.
 - [29]Webb WR, Stein MG, Finkbeiner et al: Normal and disease isolated lungs: HRCT.Radiology; 1988;166;81-87.
 - [30]Wang NS; AnatomyIn Dail DH, Hamman SP: Pulmonary pathology ed 2: New York; Springer Verlag; 1994;21-25.
 - [31]Webb WR, Muller NL, Naidich DP: HRCT of the lung Ed 2 Philadelphia; 1996;Lipincott – Raven.
 - [32]Gamsu G, Klein JS: HRCT of diffuse lung disease. Clin Rad: 1989;40;554-56.
 - [33]Muller NL, Miller RR: CT of chronic diffuse infiltrative lung disease. Am Rev Resp Dis; 1990;142;1206-25.