

Original Research Article

Role of high resolution computed tomography in the clinico-radiological study of diffuse parenchymal lung diseases and in disease progression of tuberculosis

Sumit Sharma, S. Shrinivasan*, R. Chidambaram

Department of Radiology, Sri Lakshmi Narayana Institute of Medical Sciences, Pondicherry, India

Received: 22 December 2016

Revised: 23 December 2016

Accepted: 03 February 2017

***Correspondence:**

Dr. S. Shrinivasan,

E-mail: drshrinivas@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: This study was undertaken to detect and study the various HRCT patterns of DPLD, to assess disease progression of TB, also to evaluate disease prognosis and reversibility by quantification of data and compare it with clinical and functional impairment as evaluated by PFT; and to finally correlate it with a histopathological diagnosis wherever necessary.

Methods: This prospective study was conducted in the Department of Radiodiagnosis, SLIMS from December 2014 to November 2016. Patients of all age groups (3-86 years) presenting with dyspnea at rest/exertion, dry cough and known cases of DPLD were included; known lung anomalies were excluded. Clinical history with relevant lab investigations was followed by computerized radiography and HRCT. The extent of disease on HRCT scans, assessed by the mean attenuation value of the lung, was correlated with the severity of dyspnea and PFT results. HPE was done for diagnostic confirmation.

Results: IPF including chronic interstitial pneumonia comprised the most common group of diseases accounting for 28.3% cases followed by TB (25%). 'Tree in bud' appearance (71.4%), consolidation (42.8%) and scattered nodules (28.5%) are features of active disease while fibrosis (50%), honeycombing (50%), traction bronchiectasis (37.5%) and calcified granuloma (37.5%) are features of inactive disease. The histogram of the relative frequency of pulmonary attenuation values was depressed and skewed to the right in most cases. There was a statistically significant positive correlation between the mean lung density and severity of dyspnea and the extent of functional impairment as measured by the reduction in DLCO. Seven cases of active TB, three of sarcoidosis, three of PSS and two each of ABPA and HP among others were biopsy proven.

Conclusions: The present study concludes that HRCT is an invaluable tool in the detection, characterization, diagnosis, and evaluation of disease prognosis and reversibility of DPLDs especially in the study of disease progression of TB, in an appropriate clinical setting; while HPE is the gold standard for confirmation of the diagnosis.

Keywords: Diffuse parenchymal lung disease, HRCT, Tuberculosis

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. It has been nearly 80 years since Hamman and Rich 1935 described the first case of progressive pulmonary fibrosis that resulted in death.¹ 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. 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.

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. Patients with suspected DPLD usually have a chest radiograph as the initial imaging investigation. The chest radiograph pattern is, however, not specific in most patients.^{2,3} Conventional computed tomography of the chest provides a two-dimensional representation of a three-dimensional cross-sectional slice of the lung. High resolution computed tomography (HRCT) is currently the most accurate non-invasive modality for evaluating the lung parenchyma.

The added value of HRCT in DPLD depends upon its ability to increase confidence of a specific diagnosis, to alter patient management and if possible, to influence outcome. The addition of volume algorithm 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.^{4,5} 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. The role of lung biopsy remains vital in understanding the pathogenesis of DPLDs and therapeutic intervention. The proposed research is an endeavour to study the clinico-radiological profile of DPLDs and evaluate the role of HRCT in forming a definitive diagnosis and to study the disease progression of Tuberculosis and to correlate the spectrum of findings with the functional impairment and histopathological findings, wherever possible in our hospital set up.

Aims and objectives

- 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.

METHODS

This prospective study was conducted in the Department of Radiodiagnosis in association with the Departments of Medicine and Pathology, Sri Lakshmi Narayana Institute of Medical Sciences (SLIMS), Pondicherry for a period of two years, included a total of 60 patients.

Patients of all age groups who presented with complaints of shortness of breath at rest/exertion and dry cough and known cases of idiopathic interstitial pneumonias and hypersensitivity pneumonias, pulmonary tuberculosis – primary, post-primary and TB sequelae, collagen vascular diseases, systemic vasculitides, industrial exposure related diseases, drugs and radiation exposure related cases, invasive aspergillosis, allergic bronchopulmonary aspergillosis (ABPA) and hematogenous/lymphangitic dissemination of tumours were included in the study. Patients with known lung anomalies were not included.

All the patients were evaluated along the following lines and findings were recorded on a separate proforma. A detailed clinical history was elicited from each patient followed by relevant lab investigations and PFT. The results were expressed as a percentage of the predictive value. After the clinical assessment, an elaborate radiological examination was performed in each patient. This included standard chest PA computerized radiography (CR) using Fujifilm Dry Pix CR System followed by non-enhanced spiral axial computed tomographic scans on Siemens Somatom Scope spiral CT scanner. Images were obtained using helical data acquisition with 8 mm sections using a pitch of 1-1.5 mm in a caudo-cranial direction giving intravenous contrast bolus (60-80 ml of iodinated contrast) wherever necessary. Non-ionic contrast was used wherever indicated. 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. Quantitative estimation of the extent and distribution of lung disease was done by taking spirometrically gated scans of the chest. Each scan was later evaluated using the Pulmo-CT software (version: Syngo CT VC28). The mean attenuation value of the lungs and the standard deviation of the attenuation values around the mean were recorded. The results were tabulated.

Statistical analysis

A histogram was plotted between the attenuation value of the lung on the X-axis and the relative frequency of the attenuation values on the Y-axis. Segmentation of each

slice of the lung was done in an antero-posterior direction and the antero-posterior gradient of the lung attenuation values was calculated to evaluate the distribution of the disease. The findings from each of the three scans were used to calculate a mean value of the total lung density. A reference curve was obtained comparing the distribution of attenuation values in the patient's lung with that in a normal individual. 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 examination

HPE for confirmation of the pathology was done in 34 patients with DPLD at SLIMS central research lab and findings were recorded.

RESULTS

The study included 60 patients with a clinically suspected diagnosis of diffuse parenchymal lung diseases including TB and referred for thoracic CT followed by histopathology. Various parameters were measured, correlated, tabulated and processed in the form of the following Tables (1-7) and Figures (1 and 2) for statistical analysis and interpretation of results.

Table 1. Pathology based distribution of patients (n=60).

Pathology	Number of patients	(%)
Idiopathic pulmonary fibrosis (including chronic interstitial pneumonias)	17	28.3
Tuberculosis – primary, post-primary including miliary TB and TB Sequelae	15	25
Sarcoidosis	6	10
Connective tissue disorders (including systemic sclerosis)	5	8.3
Hypersensitivity pneumonitis (HP)	4	6.6
Bronchiectasis	4	6.6
Allergic bronchopulmonary aspergillosis (ABPA)	2	3.3
Bronchiolitis obliterans organizing pneumonia (BOOP)	2	3.3
Radiation induced ILD	1	1.6
Diffuse metastases	1	1.6
Silicosis	1	1.6
Langerhans cell histiocytosis (LCH)	1	1.6
Lymphangiomyomatosis (LAM)	1	1.6
Acute interstitial pneumonitis (AIP)	1	1.6
Total	60	100

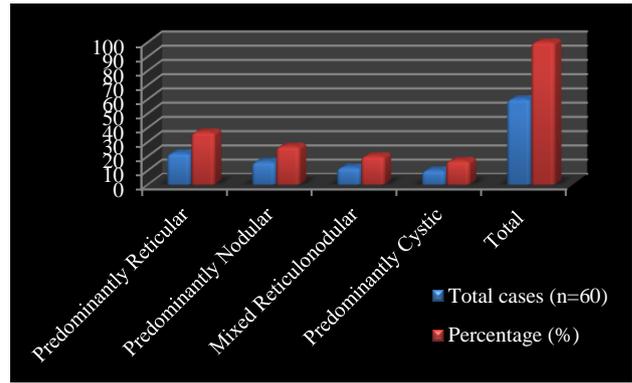


Figure 1: HRCT pattern based distribution of patients (N=60).

DISCUSSION

A study of 60 patients was conducted in the Department of Radiodiagnosis, SLIMS, Pondicherry. The aim was to evaluate the clinico-radiological spectrum of patients with diffuse parenchymal lung diseases (DPLD) and disease progression of TB using high resolution computed tomography. Idiopathic pulmonary fibrosis including chronic interstitial pneumonias comprised the most common group of diseases accounting for 28.3% cases. Coultas et al reported that IPF accounts for 25-50% of total DPLDs.² Patients with tuberculosis and sarcoidosis formed the next common groups (Table 1). The age of patients in present study ranged from 3 years to 86 years. Most of the cases presented in the fourth and fifth decade of life. There was a preponderance of females in our study group. In the study reported by Todo G et al, the mean age of their patients was around 53 years with slight female preponderance.³ We graded the dyspnea from 0 to 4 depending on the severity, as suggested by Mawson et al.⁴ Majority of the patients presented with Grade II or III dyspnea (74%). Dyspnea was usually associated with dry cough at presentation. Fever, malaise, and other constitutional symptoms were present more often in patients with tuberculosis, hypersensitivity pneumonitis and acute interstitial pneumonia. A history of occupational exposure was elicited in 3 patients. Only 11 patients gave a history of smoking. Ryu et al described four lung disorders linked to smoking-desquamative interstitial pneumonia, respiratory bronchiolitis-associated lung disease, pulmonary Langerhans Cell Histiocytosis and Idiopathic Pulmonary Fibrosis.⁵

Bibasilar inspiratory crackles were the most common finding on physical examination (90% cases). Epler et al reported that bilateral fine crackles were common in patients with idiopathic pulmonary fibrosis and connective tissue disease, occurring in 60% of the patients, but generally reported in 90% of the patients with IPF.⁶ Digital clubbing and cyanosis were seen in patients with grade 3 or 4 dyspnea and advanced disease. Warwick et al showed that finger clubbing in seen in 49 - 66% of patients with IPF.⁷ Signs of heart failure were

present in patients with advanced disease and developing cor pulmonale. Pulmonary function tests were obtained in 41 patients presenting with dyspnea. Most patients had a purely restrictive pattern with reduced PVC and DLco. Three patients had a mixed restrictive obstructive pattern. Impairment of gas exchange as assessed by carbon monoxide diffusing capacity (DLco) was the most sensitive indicator of disease with a mean value of 53.74%. All the values were expressed as percentage of the predicted value. The radiological investigations performed in each patient consisted of a chest radiograph in the postero-anterior view and spiral unenhanced high resolution sequential computed tomography scans of the chest in each patient. Quantification of the disease extent by spirometrically gated CT lung density measurement was done in 30 patients. Patients with idiopathic pulmonary fibrosis had a basal reticular pattern as the predominant finding on chest radiographs. Staples et al reported similar findings in their study.⁸ On HRCT scans, interlobular and intralobular septal thickening (94%

cases) with peripheral and lower zone predominance was the most common finding. Nishimura et al reported the incidence of septal thickening in 94% of cases.⁹ 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 commonly in subpleural location was present in 88.2% cases.¹⁰ Staples et al reported 90% incidence of honeycombing in cases of IPF identified on HRCT.⁸ Ground glass opacities that suggest the presence of reversible disease were present in 94.1% cases. Similar findings were reported by Remy-Jardin et al.¹¹ Majority of the patients had both honeycombing and ground glass opacities on HRCT scans. Only ground glass haze was present in one case. These findings suggest the presence of active reversible disease as suggested by Leung et al.¹² Thus, findings of active alveolitis and irreversible fibrosis were coexistent in majority of cases suggesting acute on chronic disease. (Figure 1) (Table 2) (Figure 2a).

Table 2: HRCT findings in patients with IPF, Sarcoidosis, CTD and HP.

HRCT findings	IPF* (n=17)		Sarcoidosis (n=6)		CTD** (n=5)		HP*** (n=4)	
	Cases	%	Cases	%	Cases	%	Cases	%
Septal thickening								
Interlobular	16	94.1	4	66.6	5	100	2	50
Centrilobular	8	47	5	83.3	1	20	3	75
Peribronchovascular	10	58.9	6	100	2	40	2	50
Subpleural	14	82.35	4	66.6	2	40	0	0
Intralobular	16	94.1	2	33.3	4	80	1	25
Predominant zones								
Upper	1	5.8	4	66.6	1	20	2	50
Mid	3	17.6	3	50	2	40	1	25
Lower	6	35.29	0	0	4	80	1	25
Diffuse	9	53	4	66.6	0	0	2	50
Predominant distribution								
Central	0	0	3	50	1	20	0	0
Peripheral	7	41.2	0	0	4	80	0	0
Diffuse/random	10	58.9	5	83.3	0	0	4	100
Predominant pattern								
Reticular	14	82.35	0	0	5	100	1	25
Nodular	0	0	5	83.3	0	0	3	75
Reticulonodular	3	17.6	3	50	0	0	1	25
Honeycombing	15	88.2	4	66.6	3	60	1	25
Ground glass haze	16	94.1	6	100	5	100	4	100
Traction bronchiectasis	9	52.9	1	16.6	0	0	1	25
Conglomerate fibrosis	7	41.2	2	33.3	0	0	1	25
Pleural thickening	15	88.2	5	83.3	3	60	1	25
Cardiomegaly with prominent pulmonary artery	7	41.2	2	33.3	3	60	0	0
Mediastinal/hilar lymphadenopathy	6	35.3	5	83.3	3	60	2	50

*Idiopathic pulmonary fibrosis **Connective tissue disorder ***Hypersensitivity pneumonitis.

Among the 15 patients with pulmonary tuberculosis, 7 were sputum positive for the acid-fast bacilli. HRCT scan

helped in assessing the pattern of involvement of lung parenchyma and predicting disease progression.

In this study, ‘tree in bud’ appearance, scattered nodules and consolidation was found to be features of active disease Figure 3 (a-c) and traction bronchiectasis, honeycombing, fibrotic bands, collapse and calcified granuloma were features of inactive disease Figure 4 (a-c) (Table 3).

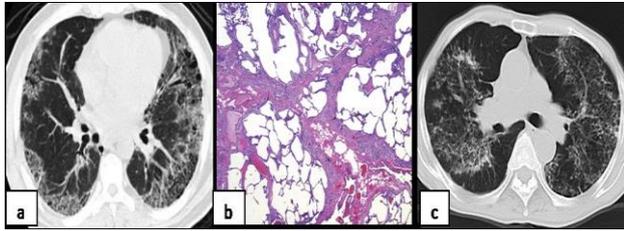


Figure 2: Predominantly reticular findings (a) HRCT lung shows sub-pleural distribution of reticular opacities in a case of UIP; (b) Histology shows irregular septal fibrosis with relative centrilobular sparing and traction emphysema; (c) HRCT image of anot.

Patients with miliary TB had randomly distributed small nodular opacities measuring 1 to 3 mm seen throughout

the lung fields. Similar findings were described by Hong et al.¹³ In patients with other forms of pulmonary tuberculosis, active disease was characterized by patchy alveolar space consolidation (42.8%). Fibrotic opacities with poorly defined nodules (few of them showing cavitation) and interlobular septal thickening was also seen.

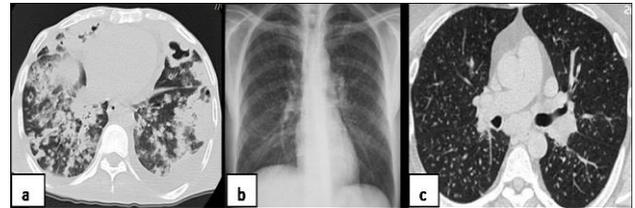


Figure 3: Active TB images (a) HRCT in a patient with TB shows fluffy alveolar opacities and ‘tree in bud’- centrilobular nodules suggestive of active disease (b, c) Chest radiograph PA view and HRCT of another patient shows multiple diffuse micronodules.

Post TB sequelae included fibrosis and honeycombing identified in 50 % of cases. Im JG et al reported the prevalence of HRCT findings in tuberculosis (Table 3).¹⁴

Table 3: Distribution of HRCT findings based on sputum AFB test (Active Vs Inactive TB).

HRCT Findings	Active TB (n=7)	%	Inactive TB (n=8)	%
Ill-Defined Nodules	2	28.5 %	1	12.5%
Consolidation	3	42.8 %	1	12.5%
Tree In Bud	5	71.4%	2	25%
Ground Glass Opacity	1	14%	1	12.5%
Traction Bronchiectasis	0	0%	3	37.5%
Calcified Granuloma	0	0%	3	37.5%
Peribronchial Cuffing	1	14%	2	25%
Cavitation	3	42.8%	1	12.5%
Atelectasis	0	0%	2	25%
Emphysema	1	14%	3	37.5%
Fibrosis	0	0%	4	50%
Honeycombing	0	0%	4	50%

(Sputum: AFB positive = Active TB, AFB negative = Inactive TB)



Figure 4: Inactive TB HRCT images show (a) honeycombing pattern in the right lung (b) confluence of large bullae causing complete collapse of the right upper lobe resulting in pneumothorax (Vanishing lung syndrome) (c) shows few calcified granulomas.

Patients with sarcoidosis revealed nodular or reticulonodular opacities with an upper and mid zone predominance in most of the cases on chest radiographs. Lymphadenopathy was suggested in 66% of cases. On HRCT, a nodular thickening of the peribronchovascular, centrilobular and subpleural thickening was the most common pattern observed. Grenier et al reported a similar incidence of nodular septal thickening in patients with sarcoidosis.¹⁵ Mediastinal and / or hilar lymphadenopathy was identified in 87.5% cases (Table 2) Figure 5(a).

In patients with hypersensitivity pneumonitis, diffuse ground glass haze was present in all patterns on HRCT

scans with ill-defined, centrilobular nodules seen in 75% cases. Hansell et al reviewed similar HRCT findings (Table 2) (Figure 5c).¹⁶

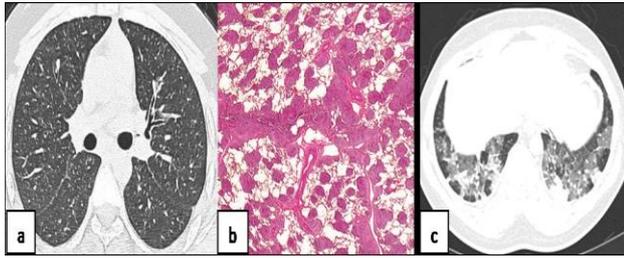


Figure 5: Predominantly nodular findings (a) HRCT image of a patient with sarcoidosis shows small well-defined centrilobular nodules with perilymphatic distribution and symmetrical lymph node enlargement (b) HPE shows extensive pleural and interlobular segments.

In patients with connective tissue disorders, a typical pattern of peripherally distributed reticular opacities with a lower zone predominance was seen in 4 out of 5 cases on HRCT. Schurawitzki et al reported similar finding in patients with PSS.¹⁷ Esophageal dilatation was seen in one patient with progressive systemic sclerosis. Grenier et al reported the incidence of esophageal dilatation as 40-80% in PSS (Table 2) (Figure 2c).¹⁸

Table 4: Pathology-wise syngo Pulmo-CT findings (n=30)*.

Diseases	Mean attenuation value of the lung (HU)
Allergic bronchopulmonary aspergillosis (n=1)	-821.4
Tuberculosis and post-tubercular disease (n=4)	-780.8
Miliary tuberculosis (n=1)	-731.5
Idiopathic pulmonary fibrosis incl. chronic interstitial pneumonias (n=5)	-680.3
Hypersensitivity pneumonitis (n=1)	-663.7
Sarcoidosis (n=3)	-661.5
Connective tissue disorders (including PSS and other diseases) (n=3)	-626.4
Bronchiolitis obliterans organizing pneumonia (n=1)	-580.5
Acute interstitial pneumonia (n=1)	-556.5

*Normal mean attenuation value was noted in 10 cases (n=10).

Bronchiectatic changes were identified in 100% cases by HRCT as against only 50% cases on chest radiograph patients with allergic bronchopulmonary aspergillosis had a proximal bronchiectasis with patchy alveolar opacities seen in both cases. Similar findings were found in a study by Kullnig et al.¹⁹ A tree-in-bud pattern suggestive of mucus impacted bronchioles was seen in 60% patterns

with bronchiectasis and 50% with ABPA. Nodular opacities measuring more than 3 mm in diameter some of them showing conglomeration with upper and mid zone predominance were seen in one case with silicosis. Mediastinal and bilateral hilar lymphadenopathy was also identified. Ill-defined discrete nodules of varying sizes with no relationship to lobular structures and interlobular septa as per Murata et al, were seen in one case with diffuse hematogeneous metastases to lung fields.²⁰

Cystic lesions of varying sizes were present in patient with Langerhans Cell Histiocytosis (LCH) and Lymph-angioliomyomatosis. In LCH, relative sparing of the bases was noted on HRCT whereas a more diffuse pattern of spread was seen in Lymphangioliomyomatosis. Grenier et al reported the presence of both thin and thick walled cystic lesions in 51 patients with Histiocytosis X.¹⁵ Both were associated with pneumothorax in the right upper chest Figure 6 (a, b).

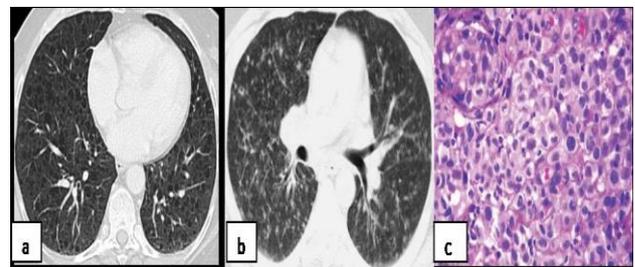
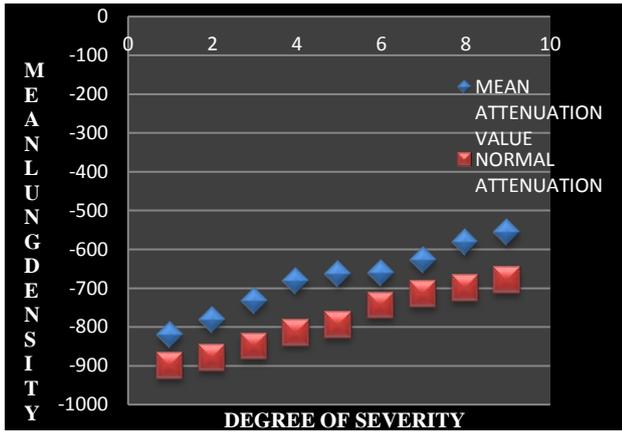


Figure 6: Predominantly cystic findings (a) Imperceptible thin walled cysts of varying sizes, present throughout bilateral lung fields in a HRCT of a 60-year-old male patient with LAM; (b) HRCT of a 7-year-old with dyspnea and cough, shows thin/thick walle.

Dense consolidation due to conglomerate fibrosis with evidence of volume loss confined to the high lung field was seen in a patient with radiation induced fibrosis. Libshitz et al and Ikezoe et al also described similar findings of dense fibrotic consolidation confined to areas of irradiated lung.^{21,22} Two cases of bronchiolitis obliterans organizing pneumonia had patchy area of air space consolidation in a peribronchial and subpleural distribution on HRCT scans. Several authors have described similar HRCT findings of BOOP.²³⁻²⁶

Well-marginated areas of ground glass attenuation in both lung fields with mediastinal and bilateral hilar lymphadenopathy were seen in one case with acute interstitial pneumonia. Similar findings have been described by Webb et al and Primack et al.^{27,28} The histogram of the relative frequency of pulmonary attenuation values was depressed and skewed to the right in most cases. These findings were similar to those described by Goldin et al and reflect the increase in the interstitial cellular or fluid content of the pulmonary parenchyma. Similar findings were reported by Hartley et al (Table 4) (Figure 7).^{29,30}



The histogram of the relative frequency of pulmonary attenuation values in the patients' lung with DPLD was depressed and skewed to the right in comparison with the curve of a normal individual of the same age. These findings reflect the increase in the interstitial cellular or fluid content of the pulmonary parenchyma.

Figure 7: Degree of severity of various diffuse parenchymal lung diseases.

There was a statistically significant positive correlation between the mean lung density and severity of dyspnea and the extent of functional impairment as measured by the reduction in DLCO. Similar findings were reported by Remy-Jardin et al.³¹ 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 (Table 5) (Table 6).³²

Findings of non-specific interstitial pneumonia, interstitial fibrosis or normal lung tissue were found in 3 out of the 5 transbronchial biopsy specimens. Histopathological examination was specific for seven cases of active TB, three of sarcoidosis, three of PSS and two each of ABPA and HP. Wall et al carried out a transbronchial and open lung biopsies in 176 patients with interstitial lung disease and showed that the transbronchial biopsy was diagnostic in 38% of cases whereas open lung biopsy was unreliable and often misleading. (Table 7) Figure 2 (b), 5(b), 5(c).³³

Table 5: Clinico-functional correlation between PFT and HRCT findings (n=26).

Mean lung density (- HU)	DLCO (% Predicted)				Total
	<40%	41- 60%	61- 80%	>80%	
-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
Total	6	10	8	2	26

Table 6: Correlation of HRCT findings with grade of dyspnea (n=30).

Mean attenuation value of lung (-HU)	Grade of dyspnea					Total
	0	I	II	III	IV	
-500 to -600	0	0	0	1	4	5
-600 to -700	1	1	3	4	2	11
-700 to -800	1	2	4	2	1	10
-800 to -900	2	0	0	1	1	4
Total	4	3	7	8	8	30

Table 7: Findings seen on histopathological examination (n=34).

Histopathological Finding	Number of cases
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
Subepidermal collagen and epidermal atrophy	3
Total	34

CONCLUSION

The following conclusions be drawn from the study-

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. Pulmonary function tests typically reveal a restrictive pattern in most cases. Certain HRCT features are diagnostic for individual diseases in a proper clinical setting, like interlobular septal thickening and peripheral honeycombing in IPF, diffuse ground glass haze for hypersensitivity pneumonitis and cystic lesions in Langerhans cell histiocytosis and Lymphangioleiomyomatosis.

In case of Tuberculosis, HRCT findings of ill-defined nodules, consolidation, tree-in-bud appearance and cavitations 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.

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.

Histopathology helps in the confirmation of diagnosis of various DPLDs like UIP, NSIP, TB, Sarcoidosis, PSS and ABPA. The present study concludes that high resolution computed tomography is an invaluable tool in the detection, characterization, diagnosis, and evaluation of disease prognosis and reversibility of diffuse parenchymal lung diseases especially in the study of disease progression of Tuberculosis, in an appropriate clinical setting; while histopathology is the gold standard for confirmation of the diagnosis.

ACKNOWLEDGEMENTS

Authors would like to thank the professors of Department of Medicine and Pathology, SLIMS, Pondicherry for their clinico-pathological and moral support during the course of this study.

Funding: No funding sources

Conflict of interest: None declared

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

REFERENCES

1. Hamman L, Rich AR. Acute diffuse interstitial fibrosis of the lungs. Bull Johns Hopkins Hosp. 1944;74:177-212.
2. Coultas DB, Zumwalt RE, Black WC, Sobonya RE. The epidemiology of interstitial lung diseases. Am Resp Crit Care Med. 1994;150:967.
3. Todo G, Ito H, Nakano Y, Dodo Y, Maeda H, Murata K, et al. HRCT for the evaluation of pulmonary peripheral disorders. Jpn J Clin Rad. 1982;7:1319-26.
4. Mawson JB, Muller NL, Mathieson JR. Sarcoidosis-correlation of extent of disease at CT with clinical, functional and radiographic findings. Radiology. 1989;171:613-8.
5. Ryu JH, Colby TB, Hartman TE. Smoking related interstitial lung diseases: a review. Eur Respir J. 2001;17:122-32.
6. Epler GE, Carrington CB, Gaensler EA. Crackles (rales) in interstitial pulmonary diseases. Chest. 1978;73:333-9.
7. Turner-Warwick M, Burrows B, Johnson A. Cryptogenic fibrosing alveolitis: clinical features and their influence on survival. Thorax. 1980;35:171-80.
8. Staples CA, Muller NL, Vedral S. UIP – Correlation of CT with clinical functional and radiologic findings. Radiology. 1987;162:377-81.
9. Nishimura K; Kitaichi M, Izumi T. UIP – histologic correlation with high resolution computed tomography. Radiology. 1992;182:337-42.
10. Austin JH, Müller NL, Friedman PJ, Hansell DM, Naidich DP, Remy-Jardin M, et al: Glossary for terms for CT of lungs: Recommendations of Nomenclature Committee of Fleishner society. Radiology. 1996;200:327.
11. Remy-Jardin M, Remy J, Wallaert B, Bataille D, Hatron PY. Pulmonary involvement in progressive systemic sclerosis: sequential evaluation with CT, Pulmonary function tests and bronchioalveolar lavage. Radiology. 1993;188:499-506.
12. Leung AN, Miller RR, Muller NL. Parenchymal opacities in chronic infiltrative lung diseases: CT-pathologic correlation. Radiology. 1993;188:209-14.
13. Hong SH, Im JG, Lee JS, Song JW, Lee HJ, Yeon KM. HRCT study of miliary tuberculosis Journal of Comput Assist Tomography. 1998;22:220-4.
14. Im JG, Itoh H, Shim YS, Lee JH, Ahn J, Han MC, et al. Pulmonary tuberculosis –CT findings – early disease and sequential change with anti-tubercular therapy. Radiology. 1993;186:653-60.
15. Grenier P, Valeyre D, Cluzel P, Brauner MW, Lenoir S, Chastang C. Chronic diffuse infiltrative lung disease: Diagnostic value of chest radiography and HRCT. Radiology. 1991;179:123-32.
16. Hansell DM, Moskovic E. HRCT in extrinsic allergic alveolitis. Clin Rad. 1991;43:8-12.
17. Schurawitzki H1, Stiglbauer R, Graninger W, Herold C, Pölzleitner D, Burghuber OC, et al.

- Interstitial lung disease in progressive systemic sclerosis: HRCT versus radiography. *Radiology.* 1990;176:755-9.
18. Grenier P, Chevret S, Beigelman C, Brauner MW, Chastang C, Valeyre D. Chronic diffuse infiltrative lung disease: Determination of diagnostic value of clinical data. Chest radiography and CT with Bayesian analysis. *Radiology.* 1994;191:383-90.
 19. Kullnig P, Pangratz M, Kopp W. CT in the diagnosis of allergic bronchopulmonary aspergillosis. *Radiology.* 1989;29:228-31.
 20. Murata K, Takahashi M, Mori M. Pulmonary metastatic nodules – CT pathologic correlation. *Radiology.* 1992;182:331-35.
 21. Libshitz HI, Shuman LS. Radiation induced pulmonary change- CT findings. *J Comput Assist Tomo.* 1984;8:15-9.
 22. Ikezoe J, Takashima S, Morimoto S, Kadowaki K, Takeuchi N, Yamamoto T, et al: CT appearance of acute radiation induced injury to the lung. *AJR.* 1988;150:765-70.
 23. Bouchardy LM, Kuhlman JE, Ball WC. CT findings in BOOP with clinical, radiographic and histological correlation. *J Comput. Assist Tomo.* 1993;17:352-7.
 24. Muller NL, Staples CA, Miller RR: BOOP – CT features in 14 patients. *AJR.* 1990;154:983-87.
 25. Muller NL, Gueny-Force ML, Staples CA. Differential diagnosis of UIP and BOOP: clinical, functional and radiological findings. *Radiology.* 1987;162:151-6.
 26. Lee KS, Kullnig P, Hartman TE, Müller NL. Cryptogenic organizing pneumonia – CT findings in 43 patients. *AJR.* 1994;162:543-46.
 27. Webb WR, Muller NL, Naidich DP. *HRCT of the lung* Ed 2 Philadelphia. Lipincott – Raven. 1996.
 28. Primack SL, Hartman TE, Ikezoe. Acute interstitial pneumonia – radiological and CT findings in 9 patients. *Radiology.* 1993;188:817-20.
 29. Goldin JG. Quantitative CT of the lung. *RCNA.* 2002;40(1):145-61.
 30. Hartley PG, Galvim JR, Hunninghahn GW. HRCT derived measures of lung density are valid indexes of ILD. *J Appl Physiology.* 1994;76:271-7.
 31. Remy-Jardin M, Giraud F, Remy J. Pulmonary sarcoidosis- role of CT in the evaluation of disease activity and functional impairment and in prognosis assessment. *Radiology.* 1994;191:675.
 32. Kalender WA, Rienmüller R, Seissler W, Behr J, Welke M, Fichte H. Measurement of pulmonary parenchymal attenuation: use of spirometric gating with quantitative CT. *Radiology.* 1990;175:256-68.
 33. Wall CP, Gaensler EA, Carrington CB, Hayes JA. comparison of transbronchial biopsy for chronic diffuse infiltrative lung disease. *Am Rev Respir Dis.* 1981;3:491-501.

Cite this article as: Sharma S, Shrinuvasan S, Chidambaram R. Role of high resolution computed tomography in the clinico- radiological study of diffuse parenchymal lung diseases and in disease progression of tuberculosis. *Int J Res Med Sci* 2017;5:956-64.