International Journal of Innovative Research in Medical Science (IJIRMS)
Volume 03 Issue 03 March 2018, ISSN No. - 2455-8737
Available online at - www.ijirms.in

Open Access Journal | Research Article | DOI: 10.23958/ijirms/vol03-i03/06

Spirometry in Diffuse Parenchymal Lung Diseases

Dr. L. K. Mohanty[1], Dr. Rashim Vachhani[*2]

[1]Assistant Professor Department of Pulmonary Medicine, [*2]Assistant Professor Department of Anaesthesiology
Chandulal Chandrakar Memorial Medical College, Kachandur, Durg (Chhattisgarh) 490024

Corresponding Author

Dr. Rashim Vachhani
Assistant Professor Dept. of Anaesthesiology CCM Medical College Kachandur, Durg (CG)

Summary

A total 45cases diagnosed to have diffuse parenchymal lung disease were taken for study and compared with 70 normal control subjects. The mean age of presentation of diffuse parenchymal lung disease (ILD) was 50.15 + 13.16 years. Male Female ration were 1.6:1 the commonest presenting symptoms are breathlessness and commonest clinical sign was file mid to late in respiratory crackles.

A significant reduction (P<0.05) in mean value of FVC, FEU, FEF 25-75% PEF and MVU has been obtained in ILD in compared with normal controls.

The mean value of FEV1/FVC (%) has been observed slightly raised in ILD cases compared with normal. The apparent difference was statistically non significant (P>0.05). The commonest venltilatory defect observed in ILD cases restrictive defect.

Keywords: *- Interstitial Lung Diseases (ILD), Diffuse Parenaymal Ling Disease (DPLD), LILD.*

Introduction

Patients with diffuse parenchymal disease should undergo comprehensive pulmonary function testing, which includes, arterial or capillary blood gas analysis at best and under exertion, spirometry and body plethysmography, as well as measurement of the diffusion capacity using carbon monoxide on a tracer in single-breath method (DLCO).[1,2,3,4] In addition, measurement of compliance is pulmonary function tests are in general not able to support a specific LILD diagnosis but, they are necessary to assess the respiratory limitations and to monitor the disease during follow up.[2,3] Lung function abnormalities reflect the effects of interstitial inflammation and scarring resulting in a restrictive venltilatory deficit and impaired gas exchange. Airways obstruction and emphysema are not feature of ILD, may be present when chronic obstruct pulmonary disease or bronchial asthma coexists in the patient.[5]

During Follow-up, changes in lung function parameters are widely used and helpful for diseases monitoring.[6]

Marginal decline in forced vital capacity in IPF 5% to 10% during and observation period of 6 months is indicative of increased mortality.[7] DLCO and Blood gases are less established prognostic indicator but there parameter can support to the clinical relevance of marginal changes in forced vital capacity. Calculated lung function indices also helpful to objectify the course and prognosis of the disease.[8]

ILD refers to a large group of lung disorders that affect the interstitium, which is the connective tissue that forms the support structure of the alveoli (air sacs) of the lungs. Normally when you inhale, the alveoli fill with air and oxygen passes into the blood stream. When you exhale, carbon dioxide passes from the blood into the alveoli and is then expelled from the body. When interstitial disease is present, the lung becomes inflamed and stiff, preventing the alveoli from fully expanding. This limits both the delivery of oxygen to the blood stream and the removal of carbon dioxide from the body. As the disease progresses, the interstitium and the walls of the alveoli thicken, which further impedes lung function.

The most common symptoms of diffuse interstitial lung disease are shortness of breath and dry cough. As the disease progresses, weight loss, muscle and joint pain, and fatigue may also occur. At a more advanced stage, individuals may develop an enlarged heart, enlargement of the fingertips clubbing), and cyanosis (blue coloration in the lips, skin and fingernails as a result of reduced oxygen levels in the blood). Individuals might also experience nonrespiratory symptoms, such as muscle pain, joint pain, or thickening or

tightness of the skin, particularly in the presence of autoimmune disease.

Materials and Methods

Spirometry in diffuse parenchymal lung disease was conducted at Chandulal Chandrakar Memorial Medical College, in the department of Pulmonary Medicine, Durg. During the period of September 2015 to December 2017.

Patients with history of progressive cough, dyspnoe, chest X-Ray findings of bilateral reticular shadows at bases of lungs, peripheral reticular, cystic, linear, nodular lesions and ground glass density or non specific reticular modular pattern were the findings interpreted as consistant with DPLD, DPLD were considered as a case.

Patients diagnosed malignancy, pulmonary tuberculosis, diabetes mellitus, hypertension, acute coronary syndrome, acute haemoptysis, recent surgery of eye, abdomen, thorax, respiratory infection at least two weeks prior to test, patients with immune supressive therapy and patients below 14 yr of age were excluded.

A total of 45 cases were classified as Idopathic pulmonary fibrosis collagen vascular disease, sarcoidosis extrinsic allergic alveolities, pneumocuniosis, pulmonary eosinophilia, alveolar proteinases, and alveolar microlithiasis. All the above patients undergone computerized Spirometry with DATOSPIRO-70 Spirometry.

Spirometric test also carried out on 17 normal controls. All Patients consent was taken. Other special investigations like ABG, pulseoxymetery, HRCT, Chest X-Ray, Serum ACE, ECG, liver and Renal function, complete hemogram, Iron markers, sputum analysis have done from the various measured lung function test parameters FUC, FEV, PEF, FEF 25-75 and MUV were selected for study,

1) Where normal values of FEV,/ FBC was more than 75% and FVC as per % of predicated was more than 70%.
2) Obstructive Venltilatory defects =FEV,/EVC was > 75% and FUC was more than or equal to 70%
3) Restrictive Venltilatory defects = when the FEV,/FUC were 75% or above but FUC was below 70% of predicted.
4) Combined i.e. - obstructive as well as restrictive Venltilatory defects - when both FEV/FVC and FVC as a percentage of predicted was below normal.

Observations and Results

Age and sex distribution among 45 cases were 28 males and 17female and were healthy controls with 47 males and 23 females. The age distribution on her table no-1

Table -1 Age and Sex wise Distribution of normal and cases.

Age in Yr.	Normal		Cases	
	Male	Female	Male	Female
20-29	8	5	2	2
30-39	10	5	3	4
40-49	11	3	5	4
50-59	8	5	9	3
>60	10	5	9	4
Total	47	23	28	17

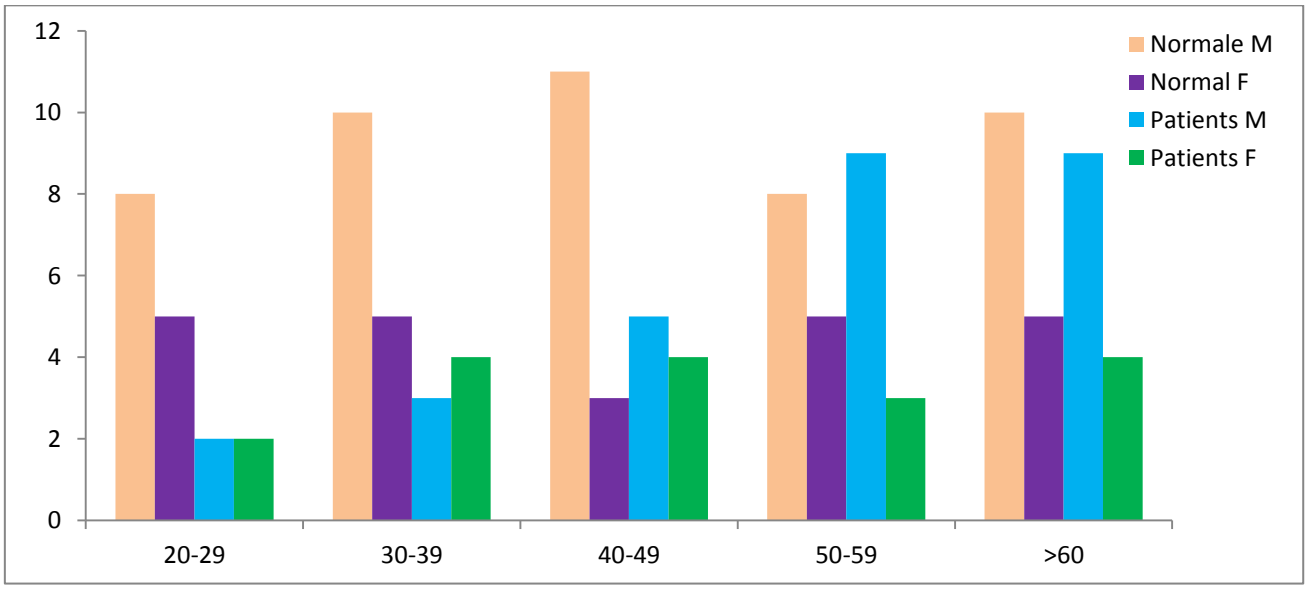

Table-2 Various Patterns of DPLD

Sr. No	Disease Group	No. of Patients N=45	Percentage
1	Collagen vascular disease	14	31.00
2	Idiopathic Pulmonary & fibrosis	12	26.67
3	Sarcoidosis	4	8.67
4	Pneumoconiosis	4	8.89
5	Pulmonary Eosinophillia	7	15.50
6	Extrinsic Allergic alveolities	2	4.40
7	Alveolar Proteinases	1	2.22
8	Alveolar Microlithiasis	1	2.22

The most common disease was Collagen vascular disease 14 (31%) followed by Idiopathic Pulmonary & fibrosis 12 (15.5%), sarcoidosis 4 (8.89%) and other as shown in above Table-II

Table 3: Ents (spirometric value in control)

Parameters	Control Group N=70		Cases (N=45)		Significance
	Mean	SD	Mean	SD	P<0.05
FUC (Ctrs)	3.27	0.70	2.26	0.85	P<0.05
FEV_1 (Ctrs)	2.64	0.56	1.87	0.66	P<0.05
FEF Ltrs (25.75%)	2.99	0.67	1.92	0.84	P<0.05
PEF (Ltrs)	5.07	1.62	3.82	1.39	P<0.05
MUV (Ltr/Min)	80.33	18.24	63.31	21.78	P<0.05
FEV/FVC (%)	82.00	3.11	83.54	11.07	P<0.05

Pulmonary Function test (Spiro meters) value obtained in as per Table III

Table 4: Types of venltilatory defects in dpld

Type of Defects	No. of Patient (N=45)	Percentage
Restrictive	37	82.22
Combined	5	11
Obstructive	2	4.44
Normal	1	2.22

It was obscured that the highest number of patients had a restrictive venltilatory defects followed by combined obstructive and normal venltilatory functions.

Discussion

In our study median age of male and female was 52.5 and 48 years. Minimum patients (26.61%) were age group of 50-59 years. Most of the patients (75.59) were aged 40 yr and above. Maheshawari et.al.[9] studied 76 patients: their mean age was 50.5 ± 11.9. Median age of male and female was 52 and 49 year respectively. Similar observation had been made by Abul et.at Subhas et.al and Agrawal et.al.[10-12] Male to female ratio in our study were 1.6:1 whereas study by Gupta et.al,[13] Alhuwalia et.al,[14] Abul et.al[10] was 1.5:1, 4:1, 1.20:1 respectively.

In our study the collagen vascular disease is the most common variant of DPLD that in 31% and this correlate with previous workers Sharma et.al, Jindal et.al[15-16] and the percentage of IPF was 26.67% correlated with Sharma et.al. they reported 29% cases. In our study Pneumoconiosis alveolar proteinosis, alveolar Micro lithiasis was observed that probably because our patients are coming from industrial belt of Chhattisgarh. In our study the mean value of FVC, FEV, FEF, 125-75%, PEF and MVV were found to be significant reduced (P<0.05) in comparison to normal controls. DPLD are characterized by restrictive lung function by which is mean a reduction in lung volumes with ratio of post expiratory volume in 1 sec. to forced vital capacity (FVC) is normal or greater than normal Cushley M.J., Chetta et.al, Martinez et.al[17-19] reported that the static lung volumes are typically reduced in DPLD. Similar observations was made by boros et. At.[20] In our study the mean FVC of DPLD cases was 2.36± 0.85 and mean FVC% of predicatyed was 58.92±13.86. In study done by Jindal et.al.[16] and Sharma et.al.[15] the mean vital capacity among Indian subject with DPLD in noted to be less than 55 to 66% for the predicted values this correlated with present study.

Based on the valued of FVC and FEV/FUC % the venltilatory defect in DPLD has been obsered to be of three types namely obstructive, restrictive and combination of

both mixed pattern. The commonest venltilatory defect observed in our study in the restrictive one.

The percentage of patients with each type if venltilatory defect as observed by previous workers as well as present study has been shown in below table.

Table 5

Type of Defect	Abal Etal (200 lt) as %	Maheshwari etal as %	Present Study as %
Restrictive	87	81.2	82
Combined	1N	-	11
Obstructive		1.56	4.44
Normal		17.21	2.22

A restrictive patter of lung function in probably the commonest pattern, but a proportion of patients have preserved lung volumes or air flow obstruction.[21] A mixed pattern of obstructive and restrictive abnormalities may also be found in DPLD associated with COPD or asthma or recurrent bronchospasm including Churg-Strauss syndrome, allergic bronchopulmonary aspergillosis and tropical pulmonary eosinophilia.[21]

Our study is probably one of the few studies from India that has looked at the spectrum of DPLD prospectively and has attempted a comparison between two of its largest contributors. However, the study has certain limitations, in that, the diffusing capacity of the lung could not be done for monitoring the disease progress; triple drug therapy versus supportive therapy in the IPF group was not randomized.

In conclusion the spectrum and clinical presentation of DPLD is largely similar to that in the western countries. Although Indian patients seem to develop the disease a decade earlier than their western counterparts. The function abnormalities ere typical but not specific. Pulmonary function test can aid in the diagnosis of DPLD although it is not diagnostic. Base line PFTS may provide an estimate of Prognosis. It is possible to detect lung function abnormalities in diffusing parenchymal lung diseases by periodic spirometry before systptoms and radiological signs appear.

References

[1] American Tllnacidc society/europeahrespiratory society international multidisciplinary consensus classification of the idiopathic intestinal pueaurnies. Ame. J. Respir. Cril. Care Med 2002 1651277-3041

[2] Rashu c =, 10011aidHR, Egan JJ etal- An official ATS/RS/JRS/ALAT Statement: Idopathic pulmonary fibrosis evidence based guidelines for diagnosis and management Am. Jr. Respire. Crit care med 2011:183:788-824

[3] Bradley po, branley HM, Engan JJ etal+ British Twnacic spciety interstitial lung disgnois guidelines committee: Tholacic societs of Australia Newzeland;. Twnacic society, Irish Twracic society: intestital lung disease guideline-thorax-2008 b, (Suple-5) V-1-58.

[4] Rashu G, Mageto yn, Lochhart D etal-The accuracy of clinical diagnosis of new onset idorthic pulmonary & Brosis and other interstitial lung disagree_ A prospective studs-chest-1999:116:1168-74.

[5] King T.E. Jr. Tooje Ja Etal:- Predictins survival in idiopathic pulmonary & Brosis scorzing system and survival model-AM. JR. Resp. Crit. Care Med.- 2001; 164 1171-81

[6] Behrj. FurstDe=Pulmonay Fuuction tests Rbeumatology-2008:47 (Suppl-5) V -64-67

[7] Zappela CJ< Latsi PL, Nicholoson AG, Etal:- Marginal Decline in force Vital Capacity is associated with poor outcome in idiopathic Pulmonary fibrosis:- EOR, RRESP. J-2010:35:830-5.

[8] Robeitson H. Clinical Application of Pulmonary Function and exercise tests in management of patients cvith intestitical lug disesase-Seman Resp. Crit. Care Med. 1994; 15:1-9.

[9] Maneswan iV, Gupta d, Agawal AN and Jindas S.K. Speetrum and Diagniosis of Idiorathic pulmonary Fibross- Indian J Chest Dis. All:ed Sci-2004; 46-23-26.

[10] Abul A, Onadeko Bo, Khadadah Me Etal- Clinical Kaherms of diffuse palenchgmal Lung disease in kucuant. Med Princ Pract- 2004; 13:78-83.

[11] Subhash HS. Ashwin I, Solomon sk Etal:- A comparatively study on idiopathic pulmonary fibrosis and secondacy diffuse parenchjmal lung disease-Indian JMed Sa 2004; 58; 185-190.

[12] Agrawal R, Gupta D. Vermajs, Jindal S.K. Etal Non inuasiul Estimationof dinicalls asyrpts matic pulmonary hspertension in didiopatic pulmonary thiosis. Ind. J Chest dis-Allied Sci-2005; 47:267-271.

[13] Gupta D, Benerad, Joshik, Jindalsk-Role of fiberoptic bronchoscopy in diagnosis of paleuchgmal lung diseses JAP. 1997; 45:371-373.

[14] Sharma S.K. Pande JN, GuleriaJS= Diffuse insterstital pulmonary fibroses Indian J OBest Dis. Allied Sci. 1984:26:214-219.

[15] Ahlumalia 4, Sharma S.K. Dattagupta S,Pande Jn+ FOle of Transbronchial Lung Biopsy in diffuse pulmonary disease – A review of 25 cases ducing One year- Ind. J.Chest Dis. Allied sc, - 1999:4:213:217.
[16] Jindal s.K.Dhaskat bu. Behia D. Respiratory Disease Pattern in a large nefterql aspitel in diania+ 1989:7:119-121.
[17] Cushley MJ Davidson AG, Dubosis RM etal-The diagnosis, assessment and treatment of diffuse paleudymal lung disease in adults british Thopiecic societs keconsmendatians-THORAY-1999:54 (Supl) S,-514.
[18] Chetta Marangio E. Olivie ric-Pulmonary function testing in interstitial lung diseases respiration-2004: 71:209-213.
[19] Nartinez j Fernando and Flaherty K- Pulmonary Function testing in Idiopathic inserstifial pueumoniss-The ploceeding of the American Thopacic Society-2006; 3;:315=321.
[20] BOROS PW,FIanc Zakm, Wesolowsk S Etal:- Value of spilometry in detecting Vloume rewestriction in insterstital ungs disease patiests Respilation 2004; 71:314-379.
[21] Mohajqur, dagamK, Tiwari-N= Diffuse palenchimal Lung disease: AN approach disagnossi JAPI-2002, 50:1285-1294.

