Original Article

Correlation between clinical characteristics, spirometric indices and high resolution computed tomography findings in patients of chronic obstructive pulmonary disease

Anubhuti Singh, Santosh Kumar, Ashwini Kumar Mishra, Manoj Kumar[1], Surya Kant, S K Verma, R A S Kushwaha, Rajiv Garg

Departments of Pulmonary Medicine and [1]Radio diagnosis, King George Medical University, Lucknow, Uttar Pradesh, India

ABSTRACT

Introduction: Chronic obstructive pulmonary disease (COPD) is a chronic inflammatory disease affecting the airways, leading to significant morbidity and mortality throughout the world. There is a need to have a holistic evaluation of COPD patients, other than just measuring the level of obstruction as performed by spirometry. High resolution computed tomography (HRCT) scan of thorax partly fulfills this requirement. **Materials and Methods:** Fifty patients of COPD (confirmed on spirometry as per the GOLD guidelines 2014 guidelines) were enrolled, out of which 35 patients got a HRCT done. Complete clinical evaluation was done. The Philips computer program for lung densitometry was used with these limits (−800/−1, 024 Hounsfield unit [HU]) to calculate densities, after validating densitometry values with phantoms. We established the area with a free hand drawing of the region of interest, then we established limits (in HUs) and the computer program calculated the attenuation as mean lung density (MLD) of the lower and upper lobes. **Results:** There was a significant correlation between smoking index and anteroposterior tracheal diameter ($P = 0.036$). Tracheal index was found to be decreasing with increasing disease severity which was statistically significant ($P = 0.037$). Mean upper lobe MLD was −839.27 HU, mean lower lobe MLD was −834.91 HU and the mean MLD was −837.08 HU. The lower lobes MLD were found to be decreasing with increasing disease severity. A mild linear correlation of pre forced expiratory volume in the first second (FEV1) was observed with lower lobe and total average MLD while a mild linear correlation of Post-FEV1 was observed with both coronal ($P = 0.042$) and sagittal ($P = 0.001$) lower lobes MLD. In addition, there was a linear correlation between both pre ($P = 0.050$) and post ($P = 0.024$) FEV1/forced vital capacity with sagittal lower lobe MLD. A predictive model can be derived to quantify obstruction severity (FEV1). **Conclusion:** HRCT may be an important additional tool in the holistic evaluation of COPD. HRCT can well be correlated with the spirometric and clinical features and the level of obstruction can be indirectly derived from it by measuring the MLD.

KEY WORDS: Chronic obstructive pulmonary disease, high resolution computed tomography thorax, spirometry

Address for correspondence: Dr. Ashwini Kumar Mishra, Department of Pulmonary Medicine, King George Medical University, Lucknow, Uttar Pradesh, India.
E-mail: ashwini486@gmail.com

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a chronic inflammatory disease affecting the airways, leading to significant morbidity and mortality throughout the world. The prevalence of Stage II or higher COPD is 10.1% worldwide.[1] For establishing a diagnosis of COPD, according to the Global Initiative for Chronic Obstructive

Access this article online

Quick Response Code:

Website: www.lungindia.com

DOI: 10.4103/0970-2113.173064

How to cite this article: Singh A, Kumar S, Mishra AK, Kumar M, Kant S, Verma SK, *et al*. Correlation between clinical characteristics, spirometric indices and high resolution computed tomography findings in patients of chronic obstructive pulmonary disease. Lung India 2016;33:42-8.

Lung Diseases (GOLD) guidelines, spirometry showing fixed airflow obstruction is essential. Spirometry is an inexpensive, easily reproducible and readily available test. However there are certain drawbacks of spirometry, for instance, it cannot be used in patients presenting for the 1st time in an exacerbation (being difficult to perform and less reliable). In addition, spirometry is an effort dependent procedure, so elderly patients and patients with neurological or psychiatric disorders who are unable to follow commands face difficulty in performing spirometry. Patients with oro-facial trauma or tumors are not able to perform too. Hence, in this group of patients, an alternative test to spirometry is required for establishing a diagnosis of COPD.

There is the need to have a holistic evaluation of COPD patients, other than just measuring the level of obstruction as done by spirometry. High resolution computed tomography (HRCT) scan of thorax fulfills this requirement. It provides information about the extent and distribution of emphysema, the presence of chronic bronchitis, or other associated findings such as bullae, bronchiectasis and cysts. In addition, it is essential in the patients of COPD for ruling out alternative diagnoses and for presurgical assessment before lung volume reduction surgeries or bullectomy. There is increasing role of HRCT in evaluation of early emphysema in asymptomatic smokers, in patients of chronic bronchitis and in assessment of the various phenotypes of COPD.

In this study, we have tried to evaluate the usefulness of HRCT in the patients with COPD and relate it to clinical and spirometric values for future correlation and derive a means for evaluation of obstruction in absence of spirometry.

MATERIALS AND METHODS

It was a prospective observational study carried out at a tertiary care centre to study the correlation among clinical characteristics, spirometric indices, and HRCT findings in patients with COPD. The duration of study was 1-year.

Fifty patients of COPD (confirmed on spirometry as per the GOLD 2014 guidelines) were enrolled, out of which 35 patients got a HRCT done and were included in the study after an explicit, written consent. Patient's detail history was taken, including smoking status and pack years. They underwent physical examination, routine blood investigations including arterial blood gas (ABG) analysis and a chest radiograph (postero-anterior view).

The spirometry was carried out on computerized Spiro 232 (PULMOLAB 435, Morgan Medical Limited, England). Spirometric indices were measured using the best out of three satisfactory performances. The parameters recorded were forced expiratory volume in the first second (FEV1) in liters, forced vital capacity (FVC) in liters and FEV_1/FVC% (FEV1/FVC).

HRCT was carried out using GE CT/e Single Slice Spiral CT machine, without contrast. The features assessed on HRCT were tracheal index (TI) (ratio of transverse to anteroposterior [AP] diameter of trachea 1 cm above the aortic arch), thoracic cage ratio (TCR) (ratio of AP to transverse diameter at TCR carina [TCRC]) and 5 cm below carina (TCR5C), sterno-aortic distance (SAD) (distance from posterior surface of sternum to anterior margin of aorta at the level of carina, vascular attenuation (thinning and decreased number of pulmonary vessels) and other features such as bronchiectasis, cysts, bullae, pulmonary hypertension, evidence of fibrosis, and mediastinal lymphadenopathy. The Philips computer program for lung densitometry was used with these limits (−800/−1, 024 Hounsfield units [HU]) to calculate densities, after validating densitometry values with phantoms. We established the area with a free hand drawing of the region of interest, then we established limits (in HU), and the computer program calculated the attenuation as mean lung density (MLD) of the lower and upper lobes.

Patients requiring mechanical ventilation or those having co-existing cardiac disorder leading to breathlessness such as congestive heart failure, cardiomyopathy, or coronary artery disease were excluded from the study.

RESULTS

Thirty five male patients of COPD were enrolled in our study. On the basis of disease severity based on the GOLD classification, the patients were divided into four groups. Maximum patients were in GOLD Stage II ($n = 17$, 48.57%). The mean age of patients was 58.43 ± 9.72 (ranging from 38 to 82 years). The duration of illness ranged from 1 to 20 years. Overall, mean duration of illness was 5.92 ± 4.62 years. Regarding smoking status, almost all patients were smokers ($n = 31$, 88.57%), and smoking was significantly associated with disease severity. Mean pack years was 22.76 pack years (range = 6.5–60 pack years). Breathlessness was the most common complaint and grade of breathlessness increased with increasing disease severity. On clinical examination, three (8.57%) patients had cyanosis and clubbing was observed in one (2.86%). All except one, that is, 34 (97.14%) had rhonchi on auscultation. Signs of heart failure, namely pedal edema were noticed in seven (20.0%) patients, raised jugular venous pressure in eight (22.8%) patients, tender hepatomegaly in three (8.57%) patients, and basal crepts in nine (25.71%) patients. These features were found more frequently in Stages III and IV than Stages I and II.

While comparing hematological and biochemical parameters, four (11.4%) patients had polycythemia, while a significant number of patients (15 [42.88%]) had anemia. Among other parameters, values of serum creatinine were found to be significantly increasing with disease severity. On evaluating oxygenation status, more than 50% patients were found to be hypoxic on ABG analysis (54.28%), while 12 (34.28%) patients had hypercapnia. No other correlation was found between

blood gases and disease severity. On chest radiography, signs of hyperinflation which were subjectively assessed were flattening of diaphragm in 22 patients (62.58%), tubular heart in 20 (57.14%) and pruning of vascular markings in 18 (51.4%) patients. Four (11.43%) patients had bullae identified on chest radiograph and five (14.28%) patients had prominent pulmonary trunk.

The important CT features are shown in Table 1. Vascular attenuation and emphysema were the most common finding (n = 31, 88.57%, each). Our study revealed bronchiectasis in a significant proportion of patients (n = 19 [54.28%]). In addition, patients with bronchiectasis were more often smokers and had lower FEV1. Regarding signs of hyperinflation on HRCT, our study found a mean TI of 0.96 (range = 0.36–1.95). Saber sheath trachea was seen in two (5.71%) patients.

Tables 2a and b demonstrate the correlation of HRCT parameters with patient characteristics and pulmonary function parameters. Mean TCR at carina was 0.77, while mean TCR at a level 5 cm below the carina was 0.69. Increased TCR was found in 11 (31.4%) patients, while barrel shaped chest (TCR > 0.9) was seen in six (17.1%) patients. Mean SAD was 2.87 cm (range = 1.13–4.26). SAD of >4 cm was seen in two (5.71%) patients. In our study, there was significant correlation between smoking index and AP tracheal diameter (P = 0.036). TI was found to be decreasing with increasing disease severity (GOLD stage), and this was statistically significant (P = 0.037).

The data of correlation of average MLD with patient characteristics and pulmonary function parameters is shown in Table 3. Mean upper lobe MLD was −839.27 HU, mean lower lobe MLD was −834.91 HU and the mean MLD was −837.08

Table 1: Distribution of important CT features

Variable	Stage I (n=5) n (%)	Stage II (n=17) n (%)	Stage III (n=8) n (%)	Stage IV (n=5) n (%)	Statistical significance (Chi-square test)		Total (%)
					χ^2	P	
Vascular attenuation (n=31)	4 (80.00)	16 (94.12)	7 (87.50)	4 (80.00)	1.251	0.741	88.57
Vascular distortion (n=2)	1 (20.00)	1 (5.88)	0 (0.00)	0 (0.00)	2.683	0.443	6.5
Mosaic attenuation (n=8)	1 (20.00)	3 (17.65)	3 (37.50)	1 (20.00)	1.281	0.734	22.85
Emphysema (n=31)	5 (100.0)	15 (88.24)	6 (75.00)	5 (100.0)	2.748	0.432	88.57
Bullae (n=10)	2 (40.00)	3 (17.65)	3 (37.50)	2 (40.00)	1.947	0.584	28.57
Cyst (n=6)	1 (20.00)	4 (23.53)	1 (12.50)	0 (0.00)	1.673	0.643	17.14
Bronchiectasis (n=19)	1 (20.00)	11 (64.71)	4 (50.00)	3 (60.00)	3.237	0.356	54.28
Fibrosis (n=22)	1 (20.00)	11 (64.71)	6 (75.00)	3 (60.00)	4.24	0.237	62.85
Pulmonary hypertension (n=4)	0 (0.00)	2 (11.76)	1 (12.50)	1 (20.00)	1.02	0.797	11.42

CT: Computed tomography

Table 2a: Correlation of thoracic CT parameters with patient characteristics and pulmonary function parameters

	Tracheal index						Thoracic cage ratio at carina						Thoracic cage ratio 5 cm below carina					
	AP		Transverse		Index		AP		Transverse		Index		AP		Transverse		Index	
	r	P	r	P	r	P	r	P	r	P	r	P	r	P	r	P	r	P
Age	−0.06	0.745	0.40	0.017	0.25	0.146	−0.04	0.839	0.25	0.152	−0.20	0.257	0.13	0.461	0.05	0.770	0.10	0.576
Duration of disease	0.04	0.840	0.05	0.786	−0.03	0.875	−0.07	0.690	0.01	0.937	−0.06	0.738	−0.01	0.939	0.10	0.555	−0.04	0.808
Smoking index	0.36	0.036	0.07	0.691	−0.27	0.122	−0.04	0.836	0.17	0.316	−0.11	0.524	0.03	0.878	0.31	0.070	−0.07	0.698
Pack years	0.33	0.052	0.05	0.784	−0.26	0.130	−0.05	0.758	0.18	0.305	−0.12	0.485	0.01	0.936	0.30	0.080	−0.08	0.659
mMRC	−0.10	0.561	0.28	0.100	0.23	0.189	0.00	0.996	0.12	0.509	−0.04	0.832	0.01	0.936	0.17	0.315	−0.05	0.763
GOLD stage	−0.15	0.396	0.13	0.455	0.20	0.256	−0.30	0.081	0.09	0.588	−0.16	0.364	−0.26	0.130	0.24	0.171	−0.35	0.042
Hb	−0.42	0.013	0.08	0.644	0.43	0.010	−0.04	0.820	−0.08	0.629	0.06	0.716	−0.11	0.513	0.13	0.448	−0.17	0.334
TLC	0.26	0.136	0.09	0.605	−0.14	0.418	−0.05	0.794	0.25	0.154	−0.17	0.340	−0.01	0.975	0.07	0.689	−0.04	0.832
RBS	−0.10	0.580	0.09	0.595	0.12	0.475	0.07	0.697	−0.33	0.052	0.33	0.055	−0.31	0.074	0.02	0.892	−0.29	0.093
Urea	0.11	0.518	0.18	0.312	−0.04	0.809	0.06	0.748	0.07	0.676	0.01	0.962	−0.01	0.932	0.15	0.397	−0.06	0.744
Creatinine	−0.07	0.684	0.05	0.787	0.05	0.761	−0.19	0.274	0.09	0.612	−0.09	0.588	−0.24	0.172	0.24	0.156	−0.30	0.080
pH	−0.13	0.449	0.21	0.223	0.22	0.210	−0.01	0.947	−0.01	0.942	0.10	0.555	−0.15	0.375	0.21	0.223	−0.21	0.227
PO_2	−0.05	0.763	−0.04	0.806	−0.06	0.736	0.38	0.025	0.12	0.491	0.10	0.565	0.45	0.006	0.20	0.238	0.35	0.040
PCO_2	−0.18	0.299	0.11	0.544	0.22	0.207	−0.22	0.204	0.07	0.708	−0.18	0.305	−0.11	0.540	−0.07	0.683	−0.08	0.663
HCO_3	−0.23	0.175	0.13	0.449	0.27	0.117	−0.21	0.226	0.07	0.689	−0.13	0.451	−0.16	0.357	0.00	0.982	−0.14	0.409
SpO_2	0.02	0.889	−0.02	0.928	−0.08	0.630	0.40	0.017	−0.07	0.690	0.23	0.184	0.35	0.042	0.03	0.845	0.31	0.066
Pre-FEV_1	0.09	0.608	−0.31	0.070	−0.29	0.089	0.40	0.017	−0.21	0.227	0.32	0.061	0.24	0.172	−0.17	0.336	0.30	0.085
Post-FEV_1	0.01	0.966	−0.38	0.024	−0.27	0.113	0.42	0.012	−0.26	0.134	0.36	0.036	0.21	0.231	−0.23	0.179	0.29	0.097
Pre-FVC	0.03	0.853	−0.16	0.372	−0.12	0.489	0.34	0.047	−0.19	0.285	0.30	0.077	0.14	0.419	−0.07	0.701	0.17	0.342
Post-FVC	0.03	0.853	−0.07	0.685	−0.06	0.724	0.32	0.059	−0.22	0.198	0.33	0.056	0.10	0.578	−0.10	0.560	0.13	0.452
Pre-V/C	0.10	0.566	−0.43	0.011	−0.35	0.038	0.24	0.171	−0.20	0.250	0.26	0.135	0.04	0.841	0.00	0.983	0.05	0.793
Post-V/C	−0.10	0.563	−0.47	0.004	−0.23	0.176	0.26	0.134	−0.19	0.284	0.25	0.145	0.08	0.656	0.00	0.980	0.09	0.619

CT: Computed tomography, FEV_1: Forced expiratory volume in the first second, FVC: Forced vital capacity, AP: Anteroposterior, GOLD: Global Initiative for Chronic Obstructive Lung Disease, Hb: Hemoglobin, TLC: Total lymphocyte count, RBS: Random blood sugar, mMRC: Modified Medical Research Council, V/C: Vital capacity

Table 2b: Correlation of other HRCT parameters with patient characteristics and pulmonary function parameters

	SAD		APD		TCSA		MLD															
							CRUL		CRLL		CLUL		CLLL		SRUL		SRLL		SLUL		SLLL	
	r	*P*	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*	*r*	*P*
Age	−0.25	0.178	−0.06	0.739	0.10	0.587	−0.02	0.911	−0.12	0.530	−0.10	0.609	−0.10	0.596	0.07	0.723	−0.12	0.526	−0.04	0.832	−0.08	0.650
Duration of disease	0.23	0.221	−0.03	0.885	0.10	0.605	−0.06	0.740	−0.04	0.833	0.03	0.893	0.12	0.519	0.12	0.523	−0.06	0.735	0.16	0.385	−0.11	0.571
Smoking index	0.20	0.273	0.08	0.660	0.20	0.290	0.08	0.684	0.06	0.736	0.07	0.703	0.07	0.714	0.10	0.577	0.11	0.540	0.09	0.630	0.05	0.800
Pack years	0.22	0.239	0.07	0.725	0.18	0.320	0.11	0.545	0.08	0.658	0.11	0.572	0.10	0.601	0.14	0.462	0.14	0.468	0.13	0.491	0.08	0.660
mMRC	0.08	0.676	0.02	0.902	0.07	0.702	0.20	0.290	0.11	0.543	0.21	0.257	0.09	0.614	0.22	0.236	0.15	0.412	0.08	0.663	0.07	0.694
GOLD stage	−0.19	0.307	−0.17	0.351	−0.13	0.495	−0.02	0.912	0.01	0.975	−0.10	0.579	−0.17	0.364	0.11	0.568	0.03	0.874	−0.02	0.899	−0.13	0.485
Hb	−0.01	0.961	0.01	0.937	−0.07	0.720	0.11	0.548	0.23	0.220	0.03	0.881	−0.01	0.973	0.31	0.091	0.38	0.036	0.07	0.695	0.05	0.805
TLC	0.04	0.846	−0.08	0.650	0.06	0.749	0.16	0.382	0.12	0.524	0.16	0.379	0.00	0.983	0.06	0.731	−0.03	0.865	0.03	0.891	−0.04	0.824
RBS	−0.18	0.339	0.09	0.614	−0.24	0.188	−0.35	0.054	−0.34	0.063	−0.50	0.004	−0.40	0.024	−0.27	0.149	−0.19	0.311	−0.29	0.112	−0.28	0.126
Urea	0.08	0.668	0.05	0.782	−0.01	0.967	−0.06	0.739	−0.10	0.607	−0.24	0.199	−0.19	0.314	−0.07	0.698	−0.03	0.872	−0.18	0.345	0.02	0.909
Creatinine	−0.11	0.538	−0.14	0.468	−0.12	0.536	−0.24	0.201	−0.18	0.324	−0.34	0.062	−0.34	0.061	−0.12	0.508	−0.11	0.572	−0.33	0.072	−0.23	0.222
pH	−0.08	0.666	0.04	0.816	−0.06	0.745	0.18	0.325	0.07	0.722	−0.12	0.523	−0.30	0.105	0.10	0.610	−0.02	0.895	−0.06	0.750	−0.22	0.235
PO_2	−0.12	0.522	0.39	0.029	0.48	0.006	−0.06	0.734	−0.09	0.645	0.06	0.750	−0.01	0.962	−0.22	0.237	−0.06	0.764	−0.16	0.385	−0.16	0.383
PCO_2	0.00	0.992	−0.18	0.322	−0.09	0.615	0.01	0.944	0.02	0.935	0.06	0.746	0.30	0.101	0.18	0.321	0.13	0.476	0.33	0.066	0.39	0.028
HCO_3	−0.03	0.855	−0.18	0.346	−0.12	0.534	0.11	0.570	0.10	0.599	0.05	0.778	0.19	0.303	0.24	0.192	0.15	0.415	0.34	0.058	0.33	0.068
SpO_2	−0.18	0.336	0.40	0.028	0.37	0.039	−0.18	0.335	−0.14	0.464	−0.06	0.737	−0.07	0.710	−0.34	0.064	−0.24	0.186	−0.25	0.180	−0.27	0.148
Pre-FEV_1	0.00	0.987	0.25	0.184	0.15	0.428	−0.40	0.026	−0.31	0.090	−0.21	0.266	−0.27	0.146	−0.40	0.024	−0.33	0.067	−0.21	0.254	−0.43	0.017
Post-FEV_1	−0.04	0.845	0.23	0.204	0.11	0.573	−0.42	0.018	−0.37	0.042	−0.28	0.127	−0.37	0.042	−0.43	0.016	−0.40	0.026	−0.32	0.078	−0.56	0.001
Pre-FVC	0.08	0.666	0.34	0.063	0.23	0.209	−0.32	0.082	−0.15	0.433	−0.12	0.504	−0.16	0.385	−0.34	0.065	−0.21	0.247	−0.11	0.561	−0.25	0.175
Post-FVC	0.17	0.368	0.32	0.078	0.18	0.337	−0.28	0.123	−0.17	0.371	−0.14	0.461	−0.15	0.436	−0.35	0.052	−0.24	0.194	−0.10	0.586	−0.24	0.187
Pre-V/C	−0.07	0.701	0.16	0.382	0.06	0.734	−0.34	0.065	−0.32	0.075	−0.21	0.256	−0.22	0.239	−0.33	0.069	−0.28	0.123	−0.18	0.322	−0.35	0.050
Post-V/C	−0.18	0.331	0.17	0.364	0.12	0.525	−0.31	0.087	−0.25	0.167	−0.20	0.272	−0.22	0.233	−0.28	0.133	−0.22	0.237	−0.24	0.194	−0.40	0.024

GOLD: Global Initiative for Chronic Obstructive Lung Disease, mMRC: Modified Medical Research Council, Hb: Hemoglobin, TLC: Total lymphocyte count, RBS: Random blood sugar, FEV_1: Forced expiratory volume in the first second, FVC: Forced vital capacity, V/C: Vital capacity, HRCT: High resolution computed tomography, SAD: Sterno-aortic distance, TCSA: Thoracic cross-sectional area, APD: Anteroposterior diameter, MLD: Mean lung density, CRUL: Coronal right upper lobe, CRLL: Coronal right lower lobe, CLUL: Coronal left upper lobe, CLLL: Coronal left lower lobe, SRUL: Sagittal right upper lobe, SRLL: Sagittal right lower lobe, SLUL: Sagittal left upper lobe, SLLL: Sagittal left lower lobe

HU. The lower lobes MLD were found to be decreasing with increasing disease severity. For spirometry parameters, a mild linear correlation of pre-FEV_1 was observed with lower lobe and total average MLD while a mild linear correlation of post-FEV_1 was observed with both coronal ($P = 0.042$) and sagittal ($P = 0.001$) lower lobes MLD. In addition, there was a linear correlation between both pre ($P = 0.050$) and post ($P = 0.024$) FEV1/FVC with sagittal lower lobe MLD. In an attempt to quantify obstruction severity (FEV1) on the basis of MLD values, a predictive model was prepared [Table 4] where FEV_1% was considered as a dependent variable while MLD of coronal right upper lobe (RUL), right lower lobe (RLL), left upper lobe (LUL), left lower lobe (LLL) and sagittal RUL (SRUL), sagittal RLL (SRLL), sagittal LUL (SLUL), and sagittal LLL (SLLL) were taken as an independent variable, MLD of SRUL, SRLL, and SLLL were found to be significantly associated with the dependent variable ($P < 0.05$). The model had a fair explanatory ability ($r^2 = 0.439$).

The equation will be as follows

Dependent Variable: FEV1%, $r^2 = 0.439$.

$FEV_1\% = -44.700 + 0.184$ CRUL $- 0.230$ CRLL $- 0.106$ CLUL $+ 0.219$ CLLL $- 0.350$ SRUL $+ 0.439$ RLL $+ 0.173$ LUL $- 0.218$ LLL.

DISCUSSION

COPD is a disease of old age and its association with prolonged duration of exposure to smoke and noxious particles, is a well-known fact. Our findings were also in conformity with this data.[2] According to GOLD staging, the patients in our study were divided in four groups. Maximum patients in our study were in GOLD Stage II ($n = 17$, 48.57%). The mean age of patients in our study was 58.43 ± 9.72 (ranging from 38 to 82 years). The mean age was observed to be increasing with increasing GOLD stage. The duration of illness ranged from 1 to 20 years. Overall, mean duration of illness was 5.92 ± 4.62 years and was found to be increasing with increasing GOLD stage. The mean duration of illness of Stage IV patients was found to be lesser than Stage III, which can be explained by the fact that patients with severe disease have frequent exacerbations, leading to steep fall in FEV1 and thus having shorter illness duration.

Smoking as the prime and modifiable cause of COPD is an established fact.[3] In accordance, we also found a significant number ($n = 31$, 88.%) of our patients to be smokers (either current or previous). Patients were nonsmokers. Among the smokers, 13 (37.14%) patients had quit smoking. Statistically, significantly higher proportion of patients with smoking habit (smoker/ex-smokers) had

Table 3: Correlation of average MLD with patient characteristics and pulmonary function parameters

	Average MLD					
	Upper average MLD		Lower average MLD		Total average MLD	
	r	*P*	*r*	*P*	*r*	*P*
Age	−0.03	0.873	−0.12	0.533	−0.08	0.684
Duration of disease	0.06	0.741	−0.02	0.902	0.02	0.922
Smoking index	0.09	0.620	0.08	0.671	0.09	0.639
Pack years	0.13	0.479	0.11	0.561	0.12	0.513
mMRC	0.25	0.182	0.12	0.524	0.18	0.322
GOLD stage	−0.07	0.692	−0.15	0.421	−0.12	0.536
Hb	0.14	0.469	0.17	0.358	0.16	0.400
TLC	0.12	0.523	0.02	0.920	0.07	0.714
RBS	−0.39	0.028	−0.34	0.059	−0.38	0.038
Urea	−0.15	0.405	−0.08	0.654	−0.12	0.518
Creatinine	−0.29	0.116	−0.24	0.190	−0.27	0.143
pH	0.02	0.904	−0.13	0.482	−0.06	0.756
PO_2	−0.10	0.609	−0.09	0.637	−0.09	0.616
PCO_2	0.16	0.403	0.23	0.209	0.20	0.283
HCO_3	0.20	0.291	0.21	0.248	0.21	0.258
SpO_2	−0.22	0.240	−0.19	0.294	−0.21	0.257
Pre-FEV_1	−0.33	0.069	−0.37	0.040	−0.36	0.047
Post-FEV_1	−0.40	0.028	−0.47	0.007	−0.44	0.012
Pre-FVC	−0.24	0.195	−0.21	0.252	−0.23	0.214
Post-FVC	−0.24	0.20s2	−0.22	0.239	−0.23	0.211
Pre-V/C	−0.29	0.114	−0.33	0.070	−0.32	0.083
Post-V/C	−0.28	0.124	−0.31	0.093	−0.30	0.100

MLD: Mean lung density, mMRC: Modified Medical Research Council, GOLD: Global Initiative for Chronic Obstructive Lung Disease, Hb: Hemoglobin, TLC: Total lymphocyte count, RBS: Random blood sugar, FEV_1: Forced expiratory volume in the first second, FVC: Forced vital capacity, V/C: Vital capacity

Table 4: Quantification of obstruction of severity (FEV_1) on the basis of MLD values

Model	Unstandardized coefficients		Standardized coefficients	*t*	Significant
	B	SE	Beta		
Constant	−44.700	53.334		−0.838	0.410
CRUL	0.184	0.127	0.544	1.455	0.158
CRLL	−0.230	0.122	−0.793	−1.888	0.070
CLUL	−0.106	0.115	−0.331	−0.919	0.367
CLLL	0.219	0.128	0.694	1.719	0.098
SRUL	−0.350	0.138	−0.894	−2.537	0.018
SRLL	0.439	0.145	1.234	3.029	0.005
SLUL	0.173	0.147	0.493	1.179	0.249
SLLL	−0.218	0.104	−0.703	−2.103	0.045

Dependent variable: FEV_1%, r^2=0.439. SE: Standard error, FEV_1: Forced expiratory volume in the first second, MLD: Mean lung density, CRUL: Coronal right upper lobe, CRLL: Coronal right lower lobe, CLUL: Coronal left upper lobe, CLLL: Coronal left lower lobe, SRUL: Sagittal right upper lobe, SRLL: Sagittal right lower lobe, SLUL: Sagittal left upper lobe, SLLL: Sagittal left lower lobe

Stage IV of COPD as compared to Stages I, II, and III of COPD (P = 0.053). Mean pack years was 22.76 pack years (range = 6.5–60 pack years).

Among the clinical features, breathlessness was found to be the most common presenting complaint and was present in all the patients. Twenty six (74%) patients were presented with cough and 23 (65.71%) patients complained of expectoration. These findings were similar to the data from the third National Health and Nutrition Examination Survey which showed that a large majority of patients with severe COPD (FEV1 <50% of predicted) may be asymptomatic. The symptoms reported most frequently were wheezing and shortness of breath.[4]

While comparing hematological and biochemical parameters, four (11.4%) patients had hemoglobin percentage >17 g/dl, thus classifying as polycythemia,[5] most probable cause being chronic hypoxia. According to the WHO definition, anemia in males is classified as Hb <13 g/dl.[6] In our study, 15 (42.88%) patients qualified as anemia according to this definition. This finding can be explained by the fact that the inflammatory mediators in circulation in patients with COPD lead to either initiation or worsening of co-morbidities such as normocytic anemia, metabolic syndrome, and diabetes. The values of serum creatinine were found to be significantly higher in Stage IV as compared to other stages (P = 0.016), the most probable causes being increasing age, recurrent infections and the use of antibiotics. On evaluating oxygenation status among COPD patients, 19 (54.28%) patients had hypoxia, while 12 (34.28%) patients had hypercapnia (Pco_2 >45 mm Hg). For all the other parameters, the difference among different stages was not significant statistically (P > 0.05).

Although a chest radiograph is not essential in the diagnosis of COPD, yet it has an important role. There are certain radiological features which can be suggestive of emphysema, namely flattening of diaphragm, pruning of vascular markings, tubular heart, and increased retrosternal air space. Among others and certain features suggestive of chronic bronchitis, such as prominence of markings (dirty chest), cardiomegaly, and prominent pulmonary trunk can also be found. However, chest X-ray has limited sensitivity and specificity.[7,8] In our study, four (11.42%) patients had bullae detected on chest X-ray, while HRCT helped detect bullae in 10 (28.57%) patients. On evaluating the level of agreement between chest X-ray and HRCT in diagnosing bullae, it was found to be of moderate order (κ > 0.2) which was also significant statistically (κ =0.317; P = 0.029). Similarly, bronchiectasis was reported in only two (5.71%) patients on CXR, while 19 (54.28%) patients had bronchiectasis on HRCT scan.

Decrease in size as well as number of pulmonary vessels presents as vascular attenuation. It is a common accompaniment to emphysema and airway destruction and is an important HRCT feature. Various studies have demonstrated significant correlation between vascular attenuation and FEV1% and dyspnea scale.[9,10] Vascular attenuation was the most common HRCT finding (n = 31, 88.57%), although no correlation with disease severity was found.

Emphysema on HRCT is defined as areas of abnormally low attenuation. In our study, emphysema was noted in 31/35 patients (88.57%), which is comparable to the findings of the study by Gupta *et al.*,[10] who reported that 25 patients had at least one type of emphysema (sensitivity 62.5%).

Bullae are a common associated feature of emphysema, which develop as a result of progressive destruction of respiratory bronchioles and alveoli. This has an important impact on both the symptomatology and the overall morbidity and prognosis. Sometimes the mode of therapy may totally change toward a surgical option. Bullectomy may be beneficial in patients with large bullae and predominantly bullous emphysema.[11] This can be detected with confidence only by HRCT, it assesses the extent of bullous disease and the degree of compression and emphysema in the remaining lung parenchyma.[12] Bullae were detected in 10 (28.57%) patients in our study population. Similar findings have been found by Mostafa also who reported bullae in 7/50 (14%) patients.[13]

Other features detected were cyst in six (11.14%) patients, which is similar to that reported by Aydin *et al*.,[14] who found cyst formation in four (8%) subjects.

Our study revealed bronchiectasis in 19 (54.28%) patients. In addition, patients with bronchiectasis were more often smokers and had lower FEV1. This high percentage of incidental diagnosis of bronchiectasis in patients of COPD raises a number of questions, namely, whether this bronchiectasis phenotype can be included in the spectrum of chronic bronchitis and emphysema or whether COPD is late sequelae of primary bronchiectasis. This warrants further large scale studies.

Patients with COPD tend to have shorter transverse than AP diameter of the trachea, leading to lower TI. TI has been significantly correlated with the functional residual capacity values.[15] TI had significant inverse correlations with duration of illness, smoking pack years, and dyspnea scale; and had direct correlations with FEV1, peak expiratory flow rate, FEV1/FVC ratio, and FEV1/slow vital capacity (SVC) ratio.[10] Our study found a mean TI of 0.96 (range = 0.36–1.95). Saber sheath trachea (TI < 0.67) was seen in two (5.71%) patients. TI was found to be decreasing with increasing disease severity (GOLD stage) and this was statistically significant ($P = 0.037$).

The normal TCR is 0.71, with the transverse thoracic diameter being more than the AP diameter. However in COPD patients, due to lung hyperinflation, the AP diameter increases, causing the TCR to increase. In our study, mean TCR at carina was 0.77, while mean TCR at a level 5 cm below the carina was 0.69. TCR at carina >0.75 (suggestive of hyperinflation) was seen in 11 (31.4%) patients, while barrel shaped chest (TCR >0.9) was seen in six (17.1%) patients. TCR at 5 cm below carina >0.75 was seen in 12 (34.28%) patients while TCR >0.9 was seen in five (14.28%) patients. We also found significant inverse correlation between TCR at 5 cm below carina and GOLD stage ($P = 0.042$). Our findings are exactly opposite to that reported by Gupta *et al*. who found that both these values had direct correlations with duration of illness, smoking pack years and dyspnea scale; and had inverse correlations with FEV1, FEV1/FVC ratio, and FEV1/SVC ratio. This discrepancy can be explained by the fact that the GOLD staging uses only spirometric indices as disease severity. If we consider the combined assessment score which includes the symptoms (modified Medical Research Council [mMRC]) as well as exacerbation history, we find mMRC increasing with severity. Thus we need a broader parameter for disease severity assessment.

Another sign of hyperinflation in patients with COPD is the increased distance from the posterior sternal surface to the anterior margin of the ascending aorta,[16] depicted by the SAD. Mean SAD was 2.87 cm (range = 1.13–4.26) in our study. SAD of >4 cm was seen in two (5.71%) patients. In our study, no correlation was found between SAD and severity of disease, the cause being that most of the patients reporting to our Tertiary Care Center present with exacerbation and it can be assumed that these patients having frequent exacerbations have rapid decline in FEV1 and proceed from Stage I to IV, without developing the physical signs of hyperinflation.

Various studies have found correlation between MLD and FEV1%.[17,18] The MLD of a normal lung without any ventilation defect has been shown to be −800 HU.[19] As the severity of emphysema goes on increasing, the MLD decreases. In our study, mean upper lobe MLD was −839.27 HU, mean lower lobe MLD was −834.91 HU, and the mean MLD was − 837.08 HU. For spirometry parameters, a mild linear correlation of pre-FEV_1 was observed with lower lobe and total average MLD while a mild linear correlation of post-FEV_1 was observed with both coronal ($P = 0.042$) and sagittal ($P = 0.001$) lower lobes MLD. In addition, there was a linear correlation between both pre ($P = 0.050$) and post ($P = 0.024$) FEV1/FVC with sagittal lower lobe MLD. These findings are consistent with the findings of Torres *et al*.,[19] who reported that the lower lobe MLD on inspiration and expiration were lower in patients with very severe or severe COPD than in those with moderate disease. Heremans *et al*. also found that the mean MLD values in patients having COPD were much less than the lung density in normal persons.[20]

In an attempt to calculate the severity of disease (FEV1) on the basis of MLD, a predictive model was prepared where FEV_1% was considered as a dependent variable while MLD of coronal RUL (CRUL), coronal RLL (CRLL), coronal LUL (CLUL), coronal LLL (CLLL) and SRUL, SRLL, SLUL and SLLL were taken as an independent variable. The MLD of SRUL, SRLL, and SLLL were found to be significantly associated with the dependent variable ($P < 0.05$) and the

model had a fair explanatory ability ($r^2 = 0.439$). Using this model, FEV1 can be calculated as:

FEV1% = −44.700 + 0.184 CRUL − 0.230 CRLL − 0.106 CLUL + 0.219 CLLL − 0.350 SRUL + 0.439 SRLL + 0.173 SLUL − 0.218 SLLL.

CONCLUSIONS

The current evaluation of COPD patients requires only spirometry. However, quantifying this complex and multisystem disease just on the basis of measuring airway obstruction is not justified and not feasible in all cases. HRCT may be an important additional tool in the holistic evaluation of this disease, and it can be a substitute to spirometry in certain situations. Our study found that HRCT can be well correlated with the spirometric and clinical features and the level of obstruction can be indirectly derived from it by measuring the MLD. Further large scale studies are warranted to consolidate these findings.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global Strategy for the Diagnosis, Management and Prevention of COPD; 2014. Available from: http://www.goldcopd.org/. [Last accessed on 2015 Apr 11].
2. Chugh T, Goel N, Bhargava SK, Kumar R. Correlation of physiological and radiological characteristics in COPD. Indian J Chest Dis Allied Sci 2012;54:235-42.
3. Remy-Jardin M, Remy J, Gosselin B, Becette V, Edme JL. Lung parenchymal changes secondary to cigarette smoking: Pathologic-CT correlations. Radiology 1993;186:643-51.
4. Mannino DM, Gagnon RC, Petty TL, Lydick E. Obstructive lung disease and low lung function in adults in the United States: Data from the National Health and Nutrition Examination Survey, 1988-1994. Arch Intern Med 2000;160:1683-9.
5. Adamson JW. Anemia and polycythemia. In: Fauci AS, Braunwald E, Kasper DL, Hauser SL, Longo DL, Jameson JL, *et al*., editors. Harrison's Principles of Internal Medicine. 18th ed. New York: McGraw-Hill; 2010. p. 456.
6. WHO, UNICEF, UNU. Iron deficiency anemia: Associations, prevention and control: A guide for programme managers. Geneva: WHO; 2001. Available from: http://www.who.int/nutrition/publications/micronutrients/anemia. [Last accessed on 2015 Apr 11].
7. Thurlbeck WM, Simon G. Radiographic appearance of the chest in emphysema. AJR Am J Roentgenol 1978;130:429-40.
8. Müller NL. Clinical value of high-resolution CT in chronic diffuse lung disease. AJR Am J Roentgenol 1991;157:1163-70.
9. MacNee W, Gould G, Lamb D. Quantifying emphysema by CT scanning. Clinicopathologic correlates. Ann N Y Acad Sci 1991;624:179-94.
10. Gupta PP, Yadav R, Verma M, Agarwal D, Kumar M. Correlation between high resolution computed tomography features and patients' characteristics in chronic obstructive pulmonary disease. Ann Thorac Med 2008;3:87-93.
11. Fishman A, Martinez F, Naunheim K, Piantadosi S, Wise R, Ries A, *et al*. A randomized trial comparing lung-volume-reduction surgery with medical therapy for severe emphysema. N Engl J Med 2003;348:2059-73.
12. Snider GL. Reduction pneumoplasty for giant bullous emphysema. Implications for surgical treatment of nonbullous emphysema. Chest 1996;109:540-8.
13. Mostafa M. Role of chest radiography, spirometry and HRCT in the early diagnosis of emphysema. Egypt J Radiol Nucl Med 2010;41:509-75.
14. Aydin O, Dursun AB, Kurt B, Aloglu V, Alpar S, Uc¸ar N, *et al*. Correlation of functional and radiological findings of lung in asymptomatic smokers. Turk Respir J 2008;9:15-9.
15. Trigaux JP, Hermes G, Dubois P, Van Beers B, Delaunois L, Jamart J. CT of saber-sheath trachea. Correlation with clinical, chest radiographic and functional findings. Acta Radiol 1994;35:247-50.
16. Copley SJ, Wells AU, Müller NL, Rubens MB, Hollings NP, Cleverley JR, *et al*. Thin-section CT in obstructive pulmonary disease: Discriminatory value. Radiology 2002;223:812-9.
17. Nakano Y, Sakai H, Muro S, Hirai T, Oku Y, Nishimura K, *et al*. Comparison of low attenuation areas on computed tomographic scans between inner and other segments of the lungs in patients with chronic obstructive pulmonary disease: Incidence and contribution to lung function. Thorax 1999;54:384-9.
18. Camiciottoli G, Bartolucci M, Maluccio NM, Moroni C, Mascalchi M, Giuntini C, *et al*. Spirometrically gated high-resolution CT findings in COPD: Lung attenuation vs lung function and dyspnea severity. Chest 2006;129:558-64.
19. Torres I, Allona M, Martínez M, Lores V, Rojo B, García-Río F. Severity-related differences in lung attenuation in men with COPD. Arch Bronconeumol 2010;46:56-63.
20. Heremans A, Verschakelen JA, Van fraeyenhoven L, Demedts M. Measurement of lung density by means of quantitative CT scanning. A study of correlations with pulmonary function tests. Chest 1992;102:805-11.