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ORIGINAL ARTICLE

Role of Thin-Section Helical Computed Tomography for Categorizing Chronic Obstructive Pulmonary Disease by Quantitative Assessment of Airway and Parenchymal Components

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Abstract

Background: The lung disorder known as chronic obstructive pulmonary disease is typified by persistent, frequently worsening airflow restriction brought on by anomalies of the airways and/or alveoli. **Objectives**: To categorize COPD based on quantitative assessment of airway and parenchymal components by MDCT and to determine the relationship between bronchial wall thickening and spirometry. **Methods**: The study included patients aged 35-83, with an average age of 64.1±9.45 years. The study group was predominantly male, with 80 male and 20 female patients. All patients showed an obstructive pattern with varying degrees of severity. **Results**: The study showed a male-to-female ratio of 4:1; the average BMI was 18.70±2.57. Patients exhibited mild 39%, moderate39%, and severe 22% emphysematous changes based on quantitative assessment of CT. Most patients (37%) had mixed centrilobular and paraseptal, followed by 31% with centrilobular, 19% panacinar, and 13% paraseptal phenotypes of emphysema. The mean lung attenuation calculations revealed the lowest attenuation of -963 HU in the right lung of a patient with a large emphysematous bulla. **Conclusions**: Quantifying, prognosticating, and classifying patients into groups that recognize the precise distribution and severity of emphysema is quantitative HRCT. This allows for a personalized approach to diagnosis and therapy for each patient.

Key Words

Chronic Obstructive Pulmonary Airway, Parenchymal components, HCT

Introduction

Chronic Obstructive Pulmonary Disease (COPD) is characterized by dyspnoea, cough, sputum production, and exacerbations due to abnormalities of the airways (bronchitis, bronchiolitis) and/or alveoli (emphysema) that cause persistent progressive airflow obstruction.[1] COPD is caused by significant exposure to noxious gases and influenced by host factors, including abnormal lung development. COPD results from gene (G)-environment (E) interactions occurring over the lifetime (T) of the individual (GETomics) that can damage the lungs and/or alter their normal development/aging processes.[2]

The primary environmental exposures leading to COPD are tobacco smoking and the inhalation of toxic gases from household and outdoor air pollution. Other

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environmental[3,4], and host factors (including abnormal lung development and accelerated lung aging) can also contribute.[2] The most relevant (albeit epidemiologically rare) genetic risk factor for COPD identified to date is mutations in the SERPINA1 gene, leading to a1-antitrypsin deficiency; similarly, other genetic diseases are also associated with reduced lung function and risk of COPD.[5] COPD is a common, preventable, and treatable disease, but extensive under diagnosis and misdiagnosis lead to patients receiving no treatment or incorrect treatment. The main pathology is inflammation and deficient gas exchange. COPD is now one of the top three causes of death worldwide, and 90% of these deaths occur in low- and middle-income countries.[6] In emphysema, there is abnormal over-distension of alveoli and irreversible destruction of supporting structures, whereas in chronic bronchitis, there is inflammation and narrowing of airways. COPD's clinical diagnosis and severity are based on the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria, which rely on spirometry parameters.[7] GOLD criteria were designed as an epidemiological tool rather than for the assessment of severity in patients. It has some limitations in evaluating patients with early disease, and the inability of patients with poor clinical conditions to perform spirometry. HRCT can detect emphysema even in asymptomatic smokers with normal lung function.[7] The marked heterogeneity of the disease hinders patients' classification, diagnosis, treatment, and follow-up. In recent years, supervised deep learning (DL) techniques have been introduced to aid physicians in analyzing the diverse imaging features of COPD. Hence, there is a need to complement the Qualitative assessment of the airway and the Quantitative assessment of the lungs using radiological imaging to spirometry evaluation by GOLD criteria for phenotyping COPD. This helps in individualized diagnostic and treatment approaches for the patients. To categorize COPD based on quantitative assessment of airway and parenchymal components by MDCT. This study aims to categorize COPD based on quantitative assessment of airway and parenchymal components by MDCT. To evaluate the correlation between quantitative assessment of lung and pulmonary function tests in COPD.

Material and Methods

This is a hospital-based Cross-sectional study conducted for two years from 2020 to 2022 in a tertiary care teaching hospital. Considering the prevalence, precision of 9.75% and 95% confidence interval, the sample size is calculated as N = Z21-á/2 * p * (1 - p) / d2.A total of 100 patients were clinically diagnosed with COPD. After obtaining informed written consent, detailed histories, including the patient's name, age, sex, occupation, address, presenting symptoms, progression, and associated symptoms, were recorded. Subsequently, spirometry and MDCT chest imaging were performed.All the patients in our study, the sub-segmental bronchus was selected and assessed in the true transverse plane using Multiplanar capabilities of MDCT and Lung density analysis Software. The subsegmental bronchial wall thickness and wall area percentage were calculated automatically. Before the MDCT examination, data on anthropometric parameters, smoking exposure smoking index (S.I. = number of cigarettes/bidis per day × total duration in years), frequency and nature of exacerbations, exposure to biomass fuels, and Body Mass Index (BMI) were recorded.[8] Institutional Ethical Committee approval was obtained before the start of the study with letter number IEC No: ECR/627/Inst/OR/2014/RR-20.

Inclusion criteria

Patients showing clinical signs and Symptoms consistent with the diagnosis of COPD and who underwent MDCT of the thorax with given written consent were included.

Exclusion criteria

Patients with malignancy, Tuberculosis, Interstitial Lung diseases, and moderate to severe pleural effusion who are not willing to be part of this study and did not give written consent were excluded.

Statistical Analysis

Data was analysed using Statistical Package for the Social Sciences (SPSS version 20), IBM Corp., 2011. Crosstabulation was done to find out the relation between smoking and Body mass index. The mean was computed for all continuous data, while percentages were utilized for categorical categories. A P value of less than or equal to 0.05 was used to evaluate statistical significance.

Results

The present study included patients aged ranging from 35-83, with a mean age of 64.1±9.45.The study group showed male predominance with 80 Male patients against 20 Female patients. The mean BMI was 18.70±2.57, the minimum BMI was 13.05, maximum BMI was 27.97. Most of the patients had a smoking index exceeding 300. The non-smokers were primarily individuals exposed to passive smoking or biomass fuel. The smokers were

categorized based on the stratification method described by Jindal et al. in 1988, which is tailored for Indian subjects and analogous to pack years.[8] There was a significant correlation between smoking index and body mass index, with a p-value of 0.017. FEV1/FVC ratio below 70%, indicating an obstructive pattern with varying degrees of severity. The categorization into different groups was based on FEV1% values to assess the severity levels (Table 1).

Table 1: Mean and standard deviation of various parameters.

Parameter	Mean±SD	Range
Age	64.1 ±9.45	35-83
Height (CM)	160.9±78	1.45-1.81
Weight (KG)	48.5±7.45	33-78
BMI	18.69±2.5	13.05-27.97
Smoking Index	499.5±370.17	15-1400
FEV1	46.18±19.27	12-85
FVC	71.84±25.43	27-120
FEV1/FVC%	63.10±7.5	41.3-70.83
Total Lung Volume (mL)	4111.26±769.19	1968-6185
Mean Lung Attenuation (HU)	-852±28.39	-963--772.5
Mean Wall Area Percentage	80.70±6.74	51.06-88.36
Mean Bronchial Wall Thickness (MM)	1.93±0.35	1.4-3.1

Presence of emphysema was evaluated based on the severity of lung parenchymal involvement and categorized by phenotypes. Using specialized software, emphysematous areas in both lungs were quantitatively assessed, represented as red-colored regions. This provided measurements including volume and percentage of lung involvement, Mean Lung Attenuation (MLA) calculated separately for each lung and averaged across both lungs. Automated Bronchial wall thickening measurement in sub-segmental bronchi shows wall thickening (2.6mm) (Figure 1).

Axial, Coronal, Sagittal HRCT images in lung window, panacinar emphysematous changes involving all segments of both lungs. The emphysematous areas are represented by red-colored regions. Automated Bronchial wall thickening measurement in sub-segmental bronchi shows wall thickening up to 2.6 mm (Moderate).

Most of the patients showed the centriacinar + paraseptal pattern, followed by the centriacinar pattern. The severity of emphysema was stratified based on percentage of lung involvement: mild (<5%), moderate (5 to 30%), and severe (>30%). The total lung MLA values of all the patients included in the study were categorized into low and normal attenuation, keeping 840HU as the lowest limit of cut-off for normal lung attenuation. The lowest MLA value was -963HU in the right lung of one of the patients having a large emphysematous bulla. All the patients with severe emphysema and most of the patients with moderate emphysema had low MLA.

There was a significant correlation between the severity of emphysema and mean lung attenuation, with a p-value of 0.02. Based on the severity of emphysema and bronchitis, the 100 patients were further categorized into various COPD phenotypes. It was observed that the patients with a severe Smoking index >300 had findings suggestive of severe and very severe obstruction on spirometry. There was a significant correlation between smoking index and spirometry, with a p-value of 0.0001.The patients with moderate and severe smoking index showed maximum changes in emphysema. There was a significant correlation between the smoking index and severity of emphysema obtained by quantitative

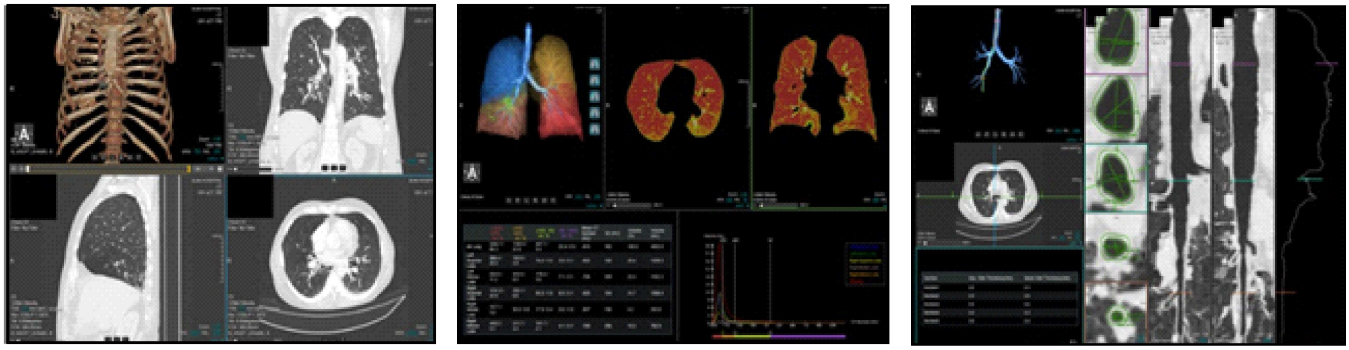

Figure 1 : Moderate emphysematous changes and moderate bronchitis (MEMB PHENOTYPE).

assessment on CT, with a p-value of 0.001.On correlation of severity grading of emphysema with spirometry, there was a significant correlation with a p-value of 0.0001.On correlation of bronchial wall thickening with spirometry, there was no significant correlation with a p-value of 0.21.In all 100 patients, the mean lung attenuation was cross-tabulated with emphysema phenotypes to find out whether there was any relation between them. On correlation of emphysema phenotypes with mean lung attenuation, there was a significant correlation with a p-value of 0.0001.The mean lung attenuation was cross-tabulated with spirometry, and it showed that most of the patients with severe and very severe obstruction on spirometry had low mean lung attenuation.

Discussion

Chronic obstructive pulmonary disease (COPD) is a multifaceted condition that impacts the airways, lung parenchyma, and vasculature. Multidetector CT has significantly advanced in localizing, quantifying, prognosticating, and phenotyping both COPD and emphysema.[9,10]

Patients' ages ranged from 35 to 83 years, with a mean age of 64.1±9.45. The COPDGene study by Han et al, the largest study on COPD to date, examined CT scans of 9,317 patients and developed the emphasize score.[11] This score includes age as a crucial factor in predicting the presence of clinically significant COPD and future morbidity.

The mean BMI of all COPD patients was 18.70±2.57, with the lowest recorded at 13.05 and the highest at 27.97. Our study found a significant correlation between BMI and the Smoking Index, with a value of 0.017.A study according to Chao et al, current smokers tend to have a lower BMI compared to non-smokers and former smokers.[12] Out of the 57 patients, 31 had severe obstruction and 13 had a very severe obstructive pattern on spirometry. Our studyalso found a significant correlation between the smoking index and severity based on spirometry, with a p-value of 0.0001. Ding et al. found in their study that significant reductions in PFT values correlate with higher pack-year histories, indicating more severe airflow obstruction and lung damage in heavy smokers.[13] MDCT findings in these patients predominantly showed mixed centrilobular and paraseptal emphysema in 21 cases, and only centrilobular emphysema in 19 cases. All the patients showed an obstructive pattern with different degrees of severity. Our study found that 38 patients out of 100 had severe obstruction, followed by 27 patients showing moderate obstruction based on spirometry.

According to CT, 39% of patients had moderate degree and 39% had mild emphysematous changes. Cross-tabulation and correlation of severity grading of emphysema with spirometry showed a significant correlation, with a p-value of 0.0001. It was observed that most patients 37% had mixed centrilobular and paraseptal, 31% of patients with centrilobular, 19% panacinar, and 13% paraseptal emphysema.

Cross-tabulation showed a significant correlation between the severity of emphysema and MLA, with a p-value of 0.026.When compared with spirometry, it was observed that patients with severe (26D) and very severe (12D) obstruction had low MLA values. In a study by Gietema et al., concluded that estimates of emphysema are not only determined by the extent of low attenuation areas but also by the lesion size, predominant type and distribution of emphysema, and the presence or absence of small airway disease.[14] Our study measured MLA in all patients and determined the phenotype of emphysema, its distribution, quantitative evaluation, and the presence or absence of small airway disease.21 patients with mixed centrilobular and paraseptal emphysema phenotypes and 20 patients with centrilobular phenotypes had low MLA values. There was a significant correlation between emphysema phenotypes and MLA, with a p-value of 0.0001.

Our study found that 58% of patients had mild bronchial wall thickening, followed by 38% of patients with moderate thickening.All the patients with severe bronchial wall thickening had a severe smoking index, and most patients with a moderate smoking index showed moderate bronchial wall thickening. There was a significant correlation between the smoking index and severity of bronchitis obtained by quantitative assessment on CT, with a p-value of 0.036. Based on the degree of severity of emphysema and chronic bronchitis, the patients were categorised into various COPD phenotypes. The maximum number of cases (26%) had a mixed mild phenotype, followed by moderate emphysematous (20%).

Conclusion

MDCT also plays an important role in the detection of early/mild cases with normal or borderline spirometry study and clinical profile. Smoking cessation and prompt medical treatment can prevent the progression of COPD in such cases. Quantitative HRCT is an emerging and powerful tool to quantify, prognosticate, and stratify

patients into categories that acknowledge the exact severity and distribution of emphysema, leading to an individualized diagnostic and treatment approach for the patients.

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References

1. Celli B, Fabbri L, Criner G, Martinez FJ, Mannino D, Vogelmeier C, et al. Definition and Nomenclature of Chronic Obstructive Pulmonary Disease: Time for Its Revision. Am J Respir Crit Care Med 2022;206(11):1317-25.
2. Agustí A, Melén E, DeMeo DL, Breyer-Kohansal R, Faner R. Pathogenesis of chronic obstructive pulmonary disease: understanding the contributions of gene-environment interactions across the lifespan. Lancet Respir Med 2022;10(5):512-24.
3. Sin DD, Doiron D, Agusti A, Anzueto A, Barnes PJ, Celli BR, et al. Air pollution and COPD: GOLD 2023 committee report. Eur Respir J 2023;61(5):2202469.
4. Yang IA, Jenkins CR, Salvi SS. Chronic obstructive pulmonary disease in never-smokers: risk factors, pathogenesis, and implications for prevention and treatment. Lancet Respir Med 2022;10(5):497-511.
5. Cho MH, Hobbs BD, Silverman EK. Genetics of chronic obstructive pulmonary disease: understanding the pathobiology and heterogeneity of a complex disorder. Lancet Respir Med 2022;10(5):485-96
6. Halpin DMG, Celli BR, Criner GJ, Frith P, López Varela MV, Salvi S, et al. The GOLD Summit on chronic obstructive pulmonary disease in low- and middle-income countries. Int J Tuberc Lung Dis 2019;23(11):1131-41.
7. Global Initiative for Chronic Obstructive Lung Disease. "Global strategy for prevention, diagnosis and management of COPD: 2023 report." Bethesda: GOLD, Avialble at : 2023https://goldcopd.org/2023-gold-report-2/
8. Singh SP, Kaur K, Meena K, Saraya PK, Kaur K,Singh CS. Smoking index- A measure to quantify cumulative smokingexposure.Panacea J Med Sci 2020;10(2):71-4.
9. Lozano R, Naghavi M, Foreman K. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 2012;380(9859):2095-128.
10. American Lung Association Epidemiology and Statistics Unit. Trends in COPD (Chronic Bronchitis and Emphysema): Morbidity and Mortality. 2013 https://www.lung.org/assets/documents/research/copd-trend-report.pdf (accessed Oct 2021).
11. Han MK, Tayob N, Kim V, Murray S, Barr G, Rennard, SI, et al. C37 COPD: WHAT IS NEW IN IMAGING?: CT Emphysema Score (emphysema, Age, Smoking, Size-Emphasize) Identifies Clinically Significant COPD. American Journal of Respiratory and Critical Care Medicine 2014;189:1.
12. Chao AM, Wadden TA, Ashare RL, Loughead J, Schmidt HD. Tobacco smoking, eating behaviors, and body weight: a review. Current addiction reports 2019;6:191-9.
13. Ding Qi, Li J, Xu S, Gao Y, Guo Y, Xie B,et al. Different smoking statuses on survival and emphysema in patients with acute exacerbation of chronic obstructive pulmonary disease.International Journal of Chronic Obstructive Pulmonary Disease 2022:;17:505-15.
14. Gietema HA, Müller NL, Fauerbach PV, Sharma S, Edwards LD, Camp PG, et al. Quantifying the extent of emphysema: factors associated with Radiologists' Estimations and quantitative indices of emphysema severity using the ECLIPSE cohort. Academic Radiology 2011:18(6):661-71.