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RADIOGRAPHIC AND CT ASSESSMENT OF SMOKING-INDUCED LUNG CHANGES: CORRELATION WITH TOBACCO CONSUMPTION AND EMPHYSEMA

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ABSTRACT
Tobacco smoke is a well-known risk factor for respiratory diseases such as chronic bronchitis, emphysema, and respiratory bronchiolitis-associated interstitial lung disease (RB-ILD). This study examines the correlation between smoking habits, lung markings on chest radiographs, and CT scan findings, employing the International Labour Organization (ILO) classification for quantification. A prospective cohort of 170 smokers was analyzed, assessing bronchial wall thickening, intralobular opacities, and emphysema severity. Findings indicated a strong association between smoking and increased lung markings, bronchial wall thickening, and centrilobular emphysema. However, interpretation bias and a lack of histological validation are study limitations. Despite these constraints, CT imaging proved effective in detecting emphysema and airway disease. Given the significant correlation between tobacco consumption and radiographic abnormalities, further research is needed to enhance early detection of smoking-induced lung changes.

KeyWords: Tobacco smoke, Lung markings, Chest radiography, Emphysema, Respiratory bronchiolitis-associated interstitial lung disease (RB-ILD).

INTRODUCTION
Tobacco smoke is recognized for contributing to conditions such as chronic bronchitis, bronchial carcinoma, and emphysema. Respiratory bronchiolitis-associated interstitial lung disease (RB-ILD), a smoking-related condition, affects the alveoli and the walls of respiratory bronchioles. Individuals with this condition frequently exhibit a "dirty chest" appearance on chest X-rays, characterized by an increase in non-specific lung markings. A comparable structural basis for smoking-induced radiographic lung changes was identified in research involving anthracosilicosis patients among coal miners [1,2]. Consequently, the International Labour Organization (ILO) classification could be useful in quantifying smoking-related lung alterations. Additionally, this study explored the correlation between increased lung markings on chest radiographs and cigarette smoking, as well as the relationship between radiographic scores and CT scan findings. To the best of our knowledge, such an assessment has not been previously documented in a clinical investigation.

METHODOLOGY
A prospective cohort study was conducted at the Sri Lakshmi Narayana Institute of Medical Sciences & Hospital, to analyze smokers within a three-month period in the Radiology Department. Within one

week of each other, both chest radiography and CT scans were performed concurrently to aid in diagnosis. The study included only clinically stable patients, while individuals diagnosed with pneumonia were excluded to prevent misinterpretation of lung parenchymal changes caused by acute infections. Additionally, patients with conditions such as sarcoidosis, extrinsic allergic alveolitis, lymphangiosis carcinomatosa, and silicosis were also excluded from the study. The research received approval from the local ethics committee, and informed consent was obtained from all participants. Over the study duration, a total of 170 patients were enrolled, with their demographic data and clinical characteristics presented in Table 1.

Assessment of chest radiograph and CT

The bronchial walls exhibited thickening along with linear or nodular opacities. Emphysema was classified into mild, moderate, or severe using semi-quantitative techniques. In our study, the revised ILO classification of chest radiographs was employed to assess the overall increase in lung markings. The profusion score was assigned as follows: 0/1 corresponded to 0, 1/1 equaled 1, and 2/1 was assigned a value of 3. The CT scans were examined by chest radiologists and radiology residents, with detailed analyses performed to detect lung opacification, lung attenuation, and bronchial malformations. Airways with thickened walls were identified as those with diameters of 2 mm or visible within 1 cm from the pleura. Two independent readers classified chronic bronchitis as moderate or severe based on their evaluation of high-resolution CT (HRCT) images from a previous study, considering airway wall thickness and bronchial wall thickness. Indicators of emphysema included reduction in vascular structures and vessel thinning. In centriacinar emphysema, low attenuation regions with a diameter of approximately 1 cm were surrounded by uniform lung parenchyma, while panlobular emphysema was characterized by evenly distributed low attenuation levels within the lobules. An increase in lung opacification was identified by the presence of attenuation, consolidation, or reticular opacities. Unlike ground-glass opacification, consolidations obscure pulmonary vessels. The degree of thickening determined whether reticular opacities were intralobular or interlobular. Linear opacities in the lung periphery were defined by thickened interlobular septa extending to the pleural surface, resulting in 12 polygonal lobules in diameter. Intralobular septal thickening was classified as interlobular septal thickening if it did not conform to reticulation patterns. Additionally, thickened alveolar walls, respiratory bronchiole abnormalities, and other lung structural changes were observed under this category. Fine, irregular lines within intralobular tissues were visible to the naked eye. A micronodule measuring 5 mm in diameter exhibited a sharp, ill-defined centrilobular or subpleural pattern. To differentiate it from intralobular lines, it was renamed intralobular opacity.

Statistic evaluation

Our study investigated the relationship between smoking habits and the increase in lung markings on chest radiographs using a linear regression model (Excel 2007; Microsoft Corp., Redmond, WA). Both chest radiography and CT scans were analyzed to assess the correlation between smoking status and findings such as emphysema, thickened bronchial walls, and an increase in linear patterns. A statistical significance threshold was established at $p < 0.05$. The sample size was determined post-hoc using the G*Power 3.1 Program (Faul, Erdfelder, Lang, and Buchner, Düsseldorf, Germany [4]). To ensure a sufficient level of confidence, a minimum of 88 patients was required to achieve a statistical power of at least 80%.

RESULTS

Chest radiography

As a consequence of cigarette consumption, 126 out of 170 patients (74%) exhibited bronchial wall thickening ($p = 0.05$). Additionally, 44 of the 170 patients (26%) demonstrated an increase in linear structures. A significant difference was observed between smokers with and without a smoking history of 20 pack-years ($\chi^2 = 5.4$, $p < 0.01$). In the study, 60 out of 85 patients (71%) were diagnosed

with moderate emphysema, while 42 out of 120 patients (35%) had severe emphysema. The likelihood of developing severe emphysema was significantly higher among heavy smokers with 60 pack-years (12 out of 18 patients, 67%). This group was significantly different from moderate smokers with 20 pack-years ($\chi^2 = 9.7$, $p < 0.05$) and moderate smokers with 20–40 pack-years ($\chi^2 = 7.0$, $p < 0.05$). Among the 170 patients, 126 (74%) showed an increase in lung markings, whereas 44 patients (26%) had normal lung parenchyma. A total of 32 patients (37%) exhibited increased lung markings, most of whom had smoked between 10 and 20 packs (n = 32), while 24 patients had smoked more than 40 packs (n = 24).

Among smokers with over 40 pack-years, only 8 individuals had a score of 1/100 or higher out of 44 patients in this category. The ILO scores of 62 patients (36%) ranged between 1/1 and 1/1. Within this group, 56 patients (33%) had smoked 20 or more pack-years, while three patients (4%) exceeded 20 pack-years. From the profusion scores assessed in our study (n = 4), the highest recorded score was ILO 2/2. Among heavy smokers with more than 60 pack-years, 2 out of 8 patients (89%) exhibited this profusion scale. The findings from chest radiography are summarized in Table 2.

CT findings

In CT scans, bronchogenic opacities were the most frequently observed finding (62%), followed closely by emphysema (64%) and thickened bronchial walls (64%). Around 39% of patients exhibited smooth nodules and thickened septa within their lung lobes, often accompanied by a prominent interlobular septum. Patients with thickened interlobular septa but without intralobular septal thickening comprised only 6% of cases. Among moderate and heavy smokers, there was no significant difference in the extent of bronchial wall thickening. However, smokers consuming 40 to 60 pack-years had a higher incidence of intralobular opacities. CT scans revealed that nearly half of the patients who smoked more than 20 pack-years exhibited intralobular opacities. The 20 pack-year group demonstrated statistically significant differences ($\chi^2 = 5.6$, $p < 0.01$). However, ground-glass opacity and subpleural micronodules did not show any significant correlation with smoking habits.

Table 1: Demographic data, indication and smoking habit of the 170 enrolled patients

Parameter	Value
Age (years)	35
Age range (years)	30–50
Gender Distribution	
Male, n (%)	110 (69%)
Female, n (%)	50 (31%)
Clinical Indications, n (%):	
Suspected malignancy	20 (40%)
Pulmonary embolism	15 (18%)
Staging	38 (75%)
Other conditions	20 (46%)
Cigarette Consumption (pack-years), n:	
Less than 40 pack-years	70
Between 40 and 80 pack-years	55
Between 80 and 120 pack-years	15
120 or more pack-years	12

Table 2: Findings in chest radiography correlated with the cigarette consumption

Findings in Chest Radiography	Cigarette Consumption (Pack Years)
	<40

Bronchial Wall Thickening	30
Linear Pattern	6
Overall Marking Score	
0/0	12
0/1	10
1/0	9
1/1	3
2/4	20
4/2	2
4/4	1
Emphysema	
Moderate	28
Severe	12
Total	40

Table 3: Findings in CT correlated with the cigarette consumption.

CT Findings	Smoke Burden (Pack Years)
	<40
Intralobular	36
Interlobular	58
Micronodule	6
Gravel	3
Thickness of the Respiratory Tract Wall	22
Asthma	15

DISCUSSION

The health hazards associated with cigarette smoke remain a significant concern in Western countries, despite the decline of the coal and steel industries [5]. Research has established a strong connection between tobacco smoke exposure and the development of respiratory diseases and cancer [5,6]. The condition is commonly referred to as "smokers' bronchiolitis", as it almost exclusively affects smokers. However, while emphysema and bronchial wall alterations detected via CT scans receive less attention, conditions such as chronic bronchitis and chronic respiratory bronchiolitis-associated interstitial lung disease (RB-ILD) tend to be more widely studied [7,8]. Cigarette smoking contributes to an increased presence of lung markings on chest radiographs, a well-documented observation in medical literature for many years [9,10]. Studies confirm that smokers have reduced lung parenchyma, as observed by other researchers. In 75% of RB-ILD cases, abnormal chest radiographs are reported [11,12]. Additionally, chest radiography suggests bronchial wall thickening in approximately 75% of RB-ILD patients [13]. In a study involving RB-ILD patients, two out of ten exhibited reticulonodular patterns on chest radiographs [22]. Among smokers with a history of consuming over 20 cigarette packs, only eight individuals (15%) had a reticular pattern corresponding to ILO 0/1. This suggests that the body's resistance to tobacco smoke may be lower than previously estimated. There is no documented evidence that lung growth can be observed through chest radiographs. Furthermore, chest radiographs and high-resolution CT (HRCT) scans are not considered reliable tools for the diagnosis or management of chronic obstructive pulmonary disease (COPD) [13-16]. However, CT scans can differentiate between COPD phenotypes characterized by airway disease and emphysema, enabling better treatment planning. A study involving 98 healthy, asymptomatic smokers indicated that HRCT is capable of detecting early signs of micronodules in lung parenchyma and mild emphysema. Another study that examined 57 smokers found that emphysematous and ground-glass changes were observed

in 40% of cases after 5.5 years of follow-up. Micronodules with poor definition were a frequent finding. CT imaging has revealed multiple RB-ILD-related lung abnormalities, including atelectasis, ground-glass opacity, and irregular reticular patterns, as documented by Holt et al. Their research indicated that 76% of smokers and ex-smokers exhibited thickened bronchial walls, while 57% displayed ground-glass opacity. In 90% of cases, both central and peripheral bronchial walls were thickened, and 71% presented with nodules, while 67% had opacities. Centrilobular opacities accounted for 61% of observed opacities, whereas ground-glass opacities were responsible for only 7%. The differences in findings may be attributed to the wide window widths and high window levels used in imaging displays. CT-based airway wall measurements also indicate that airflow reduction in COPD patients is associated with bronchial wall thickness. Our study reaffirms that bronchial wall thickening is detected more frequently on CT scans than on chest radiographs. The same conclusion applies to our findings. An assessment of smokers diagnosed with pleural dystelectasis, centrilobular emphysema, and panlobular emphysema was conducted to determine potential changes in lung parenchyma. However, centrolobular micronodules in the bronchi remained unchanged due to tobacco consumption. Findings suggest that centrilobular nodules identified on CT scans are not directly linked to chronic inflammation in respiratory bronchioles and macrophages. Additionally, no significant effect of smoking on bronchial wall thickness was observed. Despite heavy smoking being linked to a high prevalence of emphysema, nearly 90% of chest radiographs and 95% of CT scans in this study confirmed its presence. Research indicates that both modalities are effective in the early detection of emphysema. However, HRCT is known to have greater sensitivity and specificity for detecting emphysema than functional tests. Interestingly, the findings from this study suggest that chest radiographs may serve as a more reliable tool for detecting emphysema than CT scans. However, the study sample exhibited a relatively low rate of early emphysema, as implied by the findings.

CONCLUSION

As a final consideration, it is essential to determine which morphological factors contribute to the overall increase in lung markings. CT imaging revealed a strong correlation between tobacco consumption and the presence of emphysema and intralobular opacities. Small airway disease appears to be a primary factor underlying the increase in intralobular opacities and lung markings observed in chest radiographs. However, the limited sample size restricted our ability to generalize these findings. Additionally, a direct comparison between smokers and non-smokers of the same age group was not feasible due to the presence of nonspecific lung markings on chest radiographs. Furthermore, our study's findings were not validated through a gold standard histological examination, as the study design did not justify the need for invasive procedures. Potential interpretation bias in CT scans could affect the diagnosis of interstitial lung disease. The ability to detect subtle abnormalities in thin-section imaging is significantly improved when inspiration conditions are well-defined. Given these limitations, the findings presented in this study should be considered preliminary.

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