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Research Article

QUANTIFICATION OF RADIOLOGICAL CHANGES IN SMOKING-INDUCED AIRWAY DISEASE: A PROSPECTIVE STUDY

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ABSTRACT

In this prospective study, we investigated the quantification of radiological changes associated with smoking-induced airway disease, commonly referred to as "dirty chest," using the International Labour Organization (ILO) classification of pneumoconiosis. The morphological similarity between this condition and anthracosilicosis of coal workers led us to hypothesize that the ILO classification could effectively quantify these changes. Participants' chest radiographs were analyzed for lung markings, emphysema, and thickened bronchial walls by a board-certified radiology team. The ILO profusion score, ranging from zero to nine, was utilized to correlate smoking habits with lung markings. Revised ILO standards were applied to the radiographic ratings. Results revealed higher lung markings in 74% of smokers, with 37% exhibiting a profusion score of 1-4 and 29% a score of 2-2. There was a significant correlation (r=0.68) between pack years, a measure of smoking exposure, and lung markings on chest radiography. Additionally, heavy smokers (those with more than 40 pack years) showed a higher prevalence of emphysema on both CT and chest radiography. However, the prevalence of emphysema did not significantly differ between the two imaging modalities. CT scans further revealed intralobular opacities in 64% of cases and thickened bronchial walls in 62% of cases, with only 7% exhibiting ground-glass opacity. Notably, radiographic bronchial wall thickening and intralobular opacities on CT were inversely correlated with lung markings. These findings underscore the utility of the ILO classification in quantifying radiological changes associated with smoking-induced airway disease, and highlight the complementary role of CT imaging in evaluating associated pathological features.

Keywords:-Smoking-induced airway disease, Chest radiography, International Labour Organization (ILO) classification, Emphysema, Computed tomography (CT) imaging..

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INTRODUCTION

Among other things, tobacco smoke is known to cause chronic bronchitis, bronchial cancer, and emphysema. The alveoli and walls of respiratory bronchiales are affected by respiratory bronchiolitis-associated interstitial lung disease (RB-ILD) caused by smoking. Such patients are often observed to have a "dirty chest," which appears on chest radiography as a proliferation of non-specific lung marks. A similar morphological substrate for the cigarette smoking-induced chest radiographic changes was identified by a study in patients with anthracosilicosis from coal miners [1, 2]. Therefore, the ILO classification might help quantify smoking-induced lung changes. Also this study examined the relation between increased lung markings on chest radiography and cigarette smoking, as well as chest radiography scores and CT scan results. To our knowledge, this evaluation has never been described in a clinical study.

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METHODOLOGY

Within a 3-month period, a prospective cohort of smokers was analyzed in the Radiology Department. Within a week of each other, a chest radiography and CT scan were performed simultaneously to determine the diagnosis. The study was conducted on all clinically stable patients. Patients with pneumonia were excluded from this evaluation in order to prevent misinterpretation of parenchymal changes caused by acute infectious processes. As well as these diseases, sarcoidosis, extrinsic allergic alveolitis, lymphangiosis carcinomatosa, and silicosis could also exclude the patient. A local ethics committee approved the study and informed consent was obtained from all patients. During the observation period a total of 170 patients were enrolled. The demographic data and clinical features are shown in Table 1.

Assessment of chest radiograph and CT

Bronchial walls showed thickening and linear or nodular opacities. It was possible to classify emphysema as mild, moderate, or severe using semi-quantitative techniques. Our study evaluated the overall increase in lung markings using the revised ILO classification of chest radiographs. A profusion score of 0/1 equals 0, a profusion score of 1/1 equals 1, a profusion score of 2/1 equals 3. These CT scans were reviewed by chest radiologists and radiology residents. Analyses of CT scans were performed to detect lung opacification, lung attenuation, and bronchial malformations. Airways with thick walls have diameters of 2 mm or can be seen as 1 cm from the pleura. Based on their analysis of representative high resolution CT (HRCT) images published by a study, two readers categorized chronic bronchitis as moderate or severe based on the measured thickness of the airway walls and the thickness of the bronchial walls. A decrease in vessels, as well as a thinning of the vessels, were also considered signs of emphysema. Attenuation areas with an approximate diameter of 1 cm are surrounded by homogenous lung parenchyma in circulating emphysema. A uniform low attenuation level within panlobules characterized panlobular emphysema during this time period.

An increase in lung opacification is characterized by attenuation, consolidation, or reticular opacities. The pulmonary vessels are obscured by consolidations, as opposed to ground-glass opacification. The extent of thickening determines whether reticular opacities occur intralobularly or interlobularly. An interlobular septum thickened to the pleural surface is considered a linear opacity in the lung periphery. There were 12 polygonal lobules in diameter due to the thickened interlobular septa. Intralobular septal thickenings were classified as interlobular septal thickenings if they did not conform to reticulation patterns. The alveolar wall was thickened, there were respiratory bronchial abnormalities, and other abnormalities were present under this heading. It is possible to see fine and irregular lines in intralobular tissues since they are visible to the naked eye [3]. A 5 mm diameter micronodule had a sharp, ill-defined, centrilobular or subpleural appearance. To distinguish it from intralobular lines, it has been renamed intralobular opacities.

Statistic evaluation

Our research examined the association between smoking habits and increased lung markings on chest radiographs using a linear regression model (Excel 2007; Microsoft Corp., Redmond, WA). In chest radiography and CT scans, emphysema, thickened bronchial walls, and linear pattern increases were correlated with smoking status. Statistical significance was set at $p<0.05$. It was estimated the sample size post-hoc using the G*Power3.1 Program (Faul, Erdfelder, Lang and Buchner, Düsseldorf, Germany [4]). In order to achieve an acceptable level of confidence, it is necessary to have at least 88 patients with statistical power of at least 80%.

RESULTS

Chest radiography

As a result of cigarette consumption, 126 out of 170 (74%) patients experienced thickening of the bronchial walls (p 0.05). 44 of the 170 patients (26%) had linear structures that had increased. Among smokers with and without 20 pack years, there was a significant difference ($\chi2=5.4$, $p<0.01$). 60/85 (71%) of the patients in the study had moderate emphysema and 42 of 120(35%) had severe emphysema. The odds of developing serious emphysema were higher for heavy smokers with 60 pack years (12 of 18 patients, 67%); this group was significantly different from moderate smokers with 20 pack years ($\chi2=9.7$, $p<0.05$) and moderate smokers with 20–40 pack years ($\chi2=7.0$, $p<0.05$). Of 170 patients, 126(74%) had an increase in lung markings. There were only 44 patients (26% of our patients) with normal lung parenchyma. A total of 32 (37%) patients had increased lung markings, most having smoked between 10 and 20 packs (n=32), but 24 had smoked more than 40 packs (n=24). There were only 8 smokers with over 40 pack years with a score of 1/100 or higher out of 44 who had over 40 pack years. The ILO scores of 62 patients (36%) ranged from 1/1 to 1/1. 56 (33%) patients in this group had 20 or more pack years, but three (4%) had more than 20 pack years. Among the profusion scores assessed by our evaluation (n = 4), ILO 2/2 was the top score. Among heavy smokers with more than 60 pack years, there were 2 out of 8 profusion scales (89%). Chest radiography findings are listed in Table 2.

CT findings

In CT scans, bronchialogenic opacities were most common (62%), followed by emphysema (64%), and thickened bronchial walls (64%). Approximately 39% of patients exhibit smooth nodules and thickened septa in their lobes. This was often accompanied by a prominent interlobular septum. Patients with thickened interlobular septums but not thickened intralobular septums accounted for only 6% of the cases. In smokers with moderate and heavy pack years, bronchial wall thickening was not significantly different. There is a high rate of intralobular opacities among smokers who consume 40 to 60 packs per year. Patients who smoked over 20 packs of cigarettes a year were found to have intralobular opacities on nearly half of their CT scans. The 20 year group showed statistically significant differences ($\chi 2=5.6$, $p<0.01$). Ground-glass opacity and subpleural micronodules were not associated with significant differences in smoking habits.

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

Age (years)	30			
Age (years), range	28–44			
Gender				
Male, *n* (%)	122			
Female, *n* (%)	48			
Indication, *n* (%)	Suspected malignancy: 18 (42)	Pulmonary embolism: 18 (22)	Staging: 40 (82)	Others: 18 (44)
Cigarette consumption, *n*	<40 py:66	40–<80 py: 60	80–<120 py: 13	≥120 py: 18

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

Findings in chest radiography			Cigarette consumption (pack years)			
			40	40	80	120
Bronchial wall thickening			28	19	10	6
Linear pattern			4	11	4	3
Overall marking score	0	0/0	14	5	3	0
	1	0/1	8	5	0	1
	2	1/0	8	7	3	0
	3	1/1	1	5	5	2
	8	2/4	24	4	2	24
	10	4/2	0	6	0	6
	12	4/4	0	2	0	2
Emphysema	Moderate		26	32	16	4
	Severe		10	12	8	12
	Total		36	44	24	16

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

CT findings	Smoke burden (pack years)			
	40	40	80	60
Intralobular	34	44	22	10
Interlobular	60	42	20	12
A micronodule	4	4	2	4
Gravel	2	6	2	2
Thickness of the respiratory tract wall	20	32	22	14
Asthma	13	40	24	16

DISCUSSION

The health risks caused by cigarette smoke remain acute in western countries despite the decline of coal and steel industries [5]. Studies have linked tobacco smoke exposure to respiratory illnesses and cancer [5, 6]. It is commonly called "smokers' bronchiolitis" since smokers are almost exclusively affected. In contrast, abnormal CTs caused by emphysema or altered bronchial walls receive less attention than those caused by chronic bronchitis or chronic RB-ILD [7, 8]. Due to cigarette

smoking, a greater number of lung markings appear on chest radiographs. This may not be surprising since it has been assumed in literature for many years [9, 10]. There is little lung parenchyma present in smokers, as confirmed by other investigators. In 75 percent of cases with RB-ILD, chest radiographs are abnormal [11, 12]. Chest radiography suggests thickened walls in approximately 75% of RB-ILD patients [13]. A chest radiograph showed reticulonodular patterns in two of ten patients with RB-ILD [22]. Among smokers with a pack consumption of over 20 cigarettes, only eight (15%) had reticular pattern - ILO 0/1. The body resistance of tobacco smoke would seem to be lower than it actually is. The growth of lungs has never been demonstrated by chest radiographs. The diagnosis and management of chronic obstructive pulmonary disease (COPD) is not aided by chest radiographs or HRCTs [13-16]. A CT scan can differentiate between COPD phenotypes dominated by airways and emphysema, and determine the appropriate treatment plan. Based on their study of 98 healthy asymptomatic smokers, HRCT can detect micronodules in the parenchyma and mild emphysema. 57 smokers were examined for changes in CT findings caused by smoking. There were emphysematous and ground glass changes in 40% of patients after 5.5 years of follow-up. Micronodules with poor definition were common in most cases. There have been numerous CT findings associated with RB-ILD, including atelectasis, ground glass opacity, and distortions in linear and reticular structures, according to Holt et al. Researchers found that smokers and ex-smokers suffer from thickened bronchial walls 76 percent of the time and ground glass opacity of 57%. Among the results of a CT scan, 90% of the central and peripheral bronchial walls were thickened and 71% of them were nodules, as well as 67% of them were opacities. There was an opacity of 61 percent caused by centrilobular opacities, whereas just seven percent was caused by ground-glass opacities. Possibly, our results can be explained by the wide window widths and high window levels we used in our displays. A CT measurement of airway wall dimensions reveals a reduction in airflow in COPD. According to this study, bronchial wall thickening is found more frequently on CT scans or radiographs than on chest radiographs. Our conclusion is substantially the same. Smokers with pleural dystelectasis, centrilobular emphysema, and panlobular emphysema were evaluated to see if their lung parenchyma had changed. Micronodules in the centrilobular region of the bronchi did not change as a result of tobacco use. According to the findings, centrilobular nodules on CT do not seem to be associated with chronic inflammation in respiratory bronchials and macrophages. It was found that smoking had no effect on the thickness of the bronchial wall.

A nearly 90% chest radiograph and 95% CT scan of these patients revealed emphysema, despite heavy smoking being associated with a high prevalence. Studies have shown that both modalities are effective for diagnosing emphysema early. As a means of detecting emphysema, HRCT has a better sensitivity and specificity than functional tests. The results of our study indicate that chest radiography can be a more reliable method of detecting emphysema than CT. The study sample in our study had a relatively low rate of early emphysema, as implied.

CONCLUSION

As a final consideration, we need to identify which morphological substrates increase lung markings overall. CT imaging showed a significant association between tobacco consumption and emphysema and intralobular opacities. Small airway disease is likely to be a major underlying cause of the increased intralobular opacities and overall lung markings in chest radiography. The small sample size prevented us from generalizing our results. In addition, smoking and nonsmoking of the same age were not comparable due to nonspecific lung markings on chest radiographs. The findings of our study are not verified by a gold standard histological examination. Invasive investigations were not justified by the study's design. There may be interpretation bias in CT scans when diagnosing interstitial lung disease. The ability to detect subtle abnormalities in thin sections is enhanced when inspiration conditions are defined. The findings presented here are preliminary in nature, therefore.

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