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# CORRELATION OF SMOKING HISTORY WITH IMAGING SEVERITY IN COPD PATIENTS AGE GROUP BETWEEN 50 TO 70

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## ABSTRACT

*Background: The radiographic severity of chronic obstructive pulmonary disease (COPD), a progressive respiratory condition closely linked to smoking, can vary depending on a number of contributing factors. For more accurate disease assessment, it's critical to comprehend the connection between smoking history and imaging results. Objective: to assess the relationship between smoking history and imaging severity in patients with COPD between the ages of 50 and 70. Methodology: 135 individuals with a diagnosis of COPD participated in a cross-sectional study. In addition to recording smoking history, radiographic measures such as bronchial wall thickness, type, and severity of emphysema were evaluated. SPSS was used for statistical analysis. Regression analysis and Spearman's correlation were used to identify correlations and predictors. Results: Smoking history did not significantly correlate with bronchial wall thickness ($r = 0.024$, $p = 0.784$), emphysema type ($r = -0.065$, $p = 0.454$), or emphysema severity ($r = -0.056$, $p = 0.519$). Additionally, age, length of respiratory disease, and smoking history did not significantly predict the severity of emphysema, according to regression analysis ($p = 0.496$). Radiological results showed a balanced distribution of emphysema severity and common characteristics such air trapping, bronchial wall thickening, and bullae development. Conclusion: Imaging severity in COPD patients is not substantially influenced by smoking history alone. The results demonstrate the complex nature of COPD, where individual, environmental, and genetic factors may all have a significant impact. Accurately determining the severity of the condition requires a more comprehensive assessment than just smoking history.*

***Keywords:*** *COPD, emphysema, radiological findings, smoking history, SPSS*

## INTRODUCTION

The most vulnerable internal organ, the lungs have been a major cause of death for the past 200 years. The medical conditions changed over time, with some disappearing, others appearing, and others changing. Infections like pneumonia and tuberculosis have given rise to illnesses caused by contaminated air, such as lung cancer, asthma, and chronic obstructive pulmonary disease (Geddes, D 2020).

It is a prevalent, preventable, and manageable condition that is characterized by chronic respiratory symptoms and limited airflow due to abnormal changes in the airways and/or alveoli. These abnormalities are usually brought on by chronic pulmonary exposure to dangerous particles or gases. Its pathogenesis may begin much earlier, even before birth, since passive smoke exposure throughout the pregnancy is associated with an increased risk of adult COPD, untainted by active and passive smoke exposure during childhood, adolescence, or adulthood. Any disease's severity is determined by the target organ's degree of functional impairment; in the case of COPD, the degree of airflow obstruction has traditionally been used to gauge severity. (Soriano et al, 2018)

The two most common types of COPD and the two conventional subcategories are emphysema and chronic bronchitis. However, persons with COPD have been discovered to have varying degrees of co-existing chronic bronchitis and potentially significant vascular problems, casting doubt on this core premise. Other phenotypes or classifications have been identified as a result. (Myc LA et al, 2019)

Emphysema is characterized by enlarged airways (alveoli) with compromised walls that result in irreparable lung tissue damage. Emphysema is just one of the anatomical defects that can impede airflow; in a significant majority of people, it may exist without airflow restriction. (Martini K, Frauenfelder T, 2020)

A productive cough that lasts at least three months a year for two years in a row is the hallmark of chronic bronchitis.

Airflow restriction is not necessarily the outcome of chronic bronchitis. However, smoking increases the chance of developing it in young individuals with chronic bronchitis.

The pulmonary system is negatively impacted by COPD, which is linked to the respiratory system. Breathing is more difficult. Initial symptoms could be minor, starting with frequent coughing and dyspnea. (Chatreewatanakul B et al, 2022).

It is anticipated that by 2020, COPD will be the fourth most prevalent cause of death in the US and rank third internationally. Additionally, while fatality from other frequently occurring causes of death appears to be decreasing in the USA, death rates because COPD are increasing. COPD mortality rates are likely inflated due to underdiagnosis because co-morbidities are often reported as causes of death. (Berry et al, 2010).

Because it develops gradually, older age is a known risk factor for the condition. The majority of exposures, including biomass smoke, cigarette smoking, and occupational exposures, require time to cause COPD. Nonetheless, there is considerable overlap between the molecular mechanisms that cause COPD and the characteristics of aging. Therefore, it's also probable that COPD develops more easily as people age. (Kukrety et al, 2018).

However, chest X-rays (CXRs) are easily accessible and could be used as a screening tool to find COPD patients who need additional testing or treatment.

Electromyography and fluoroscopy are intrusive, radiation-exposing, or require patients to leave the room in order to diagnose diaphragmatic weakness or paralysis. Additionally, they can be time-

consuming, indirect, and uncomfortable (plethysmography and transdiaphragmatic pressure measurement) or challenging and expensive (dynamic ecoplanar magnetic resonance imaging). (Evrin et al, 2019).

High-resolution CT (HRCT) is now the recommended method for the sensitive and non-surgical assessment of pathological changes in emphysema since it has been shown to have a strong connection with pathology. With modern multislice CT (MSCT) technology, the entire lung can be covered in a superior resolution mode and thin slice (1 mm or less) with the same breath-hold (3D-HRCT). The distribution of emphysema can be more easily observed with multiplanar reformations. Compared to inspiratory CT scans, analysis of expiratory scans revealed stronger relationships with pulmonary function test findings. (Ley-Zaporozhan et al, 2008).

The incidence of COPD is directly correlated with smoking frequency. The primary risk factor for COPD is cigarette smoking. However, about 25% of patients have never taken up smoking. Different rates of COPD have been found in studies on nonsmokers. Research on COPD usually includes smokers and/or ex-smokers. Other factors that contribute to the onset of COPD, though to a lesser degree, include occupational exposure, respiratory conditions from childhood or the past, outdoor and indoor air pollution, passive tobacco exposure to radiation, biomass exposure, age, female gender, socioeconomic status, nutrient deficiencies, inadequate lung morphology, inherited conditions, and asthma. (Guldaval F, et al, 2021).

The main cause of COPD is known to be smoking, and a person's susceptibility is a constant rather than an identifiable characteristic that may cooperate with other indicators of risk. There is strong evidence that most smokers suffer respiratory harm from COPD. 50% of smokers eventually develop COPD. Because it provides smokers with a scientific basis for the advice that they have a one in two chance of developing COPD if they persist in smoking for their lives, this study has important treatment implications. (Marsh et al, 2006).

Because of its well-established causal link to cigarette smoking, COPD stands out among the debilitating chronic illnesses; approximately 90% of patients with an assessment of COPD are either former or current smokers (Hansen and others, 2007).

Tobacco usage is the biggest risk factor to develop COPD, however not all individuals with COPD have smoked in the past. Only 50% of COPD occurrences worldwide are associated with smoking, while an estimated 10% to 12% of patients have never smoked. In other words, nonsmokers also experience irreversible airway restriction. (Zhang et al, 2014)

The most common and obvious risk factor for COPD, which develops in about 15% of smokers, is cigarette smoking. Smokers are three to five times as likely than nonsmokers to develop COPD. Target cell types may produce more endogenous reactive oxygen species due to the approximately 4,700 chemical components found in cigarette smoke. Nevertheless, little is known about the underlying molecular pathways responsible for the considerable variation in COPD development in response to cigarette smoking.

Neutrophils, macrophages, and lymphocytes can invade lung tissue as a result of aberrant pulmonary inflammation brought on by cigarette smoking. Proinflammatory mediators are released by these cells. Damage to the pulmonary architecture may result from a complicated network of inflammation that includes different inflammatory cells and mediators. Autonomic

nervous system dysfunction, oxidation/antioxidant imbalance, and protease/anti-protease imbalance. (Bai et al, 2017)

The primary cause of emphysema and COPD is unquestionably smoking. Since smoking contributes 80–90% of the risk in industrialized nations, these illnesses may be referred to as "chronic smoking diseases." However, it takes ten to twenty years for the condition to manifest. The worrisome estimates of the burden of COPD are mostly a reflection of the spread and growth of smoking. Long-term cigarette smoking is linked to an increased risk of respiratory symptoms, a lower FEV1, and a higher death rate from COPD compared to nonsmokers, according to both cross-sectional and longitudinal studies. (Bartal et al, 2007)

Although smoking cigarettes has long been thought to be the most common and significant cause of COPD, other causes are becoming more widely acknowledged globally. Understanding the factors that play a role in the progression of COPD in non-smokers, the processes of illness progression, and the relationship between specific risk factors and the symptoms and course of the disease will inform strategies for disease avoidance and the development of standard therapies to lessen or treat the effects of COPD. We want to provide information on COPD to people who have never smoked. According to a 2009 Review of COPD in non-smokers published in The Lancet, 3 Salvi and Barnes conducted a thorough analysis of the literature and found that variables other than tobacco use account for 25–40% of all cases of COPD worldwide. Since then, more proof of the increasing prevalence of COPD in nonsmokers has surfaced. (Yang et al, 2022)

Due, at least in part, to varying lung morphologic abnormalities, COPD shows considerable variation in its clinical presentation and rate of disease development across affected individuals. Chest computed tomography (CT) offers more information on structural and pathophysiologic lung parameters, which helps to better understand disease variability and characterize COPD subtypes. (Labaki et al, 2017)

The most precise imaging method for determining whether structural changes of the chest, such as those affecting the heart, lung parenchyma, mediastinum, thoracic cage bone structure, and respiratory muscles, are present in vivo and non-invasively is computed tomography (CT). (Alvar Agusti et al, 2022)

It was utilized to identify the main morphological characteristics of emphysema, bronchial wall thickening, and gas trapping in chronic obstructive pulmonary disease (COPD). Lung CT already has significant clinical uses, such as identifying patients with severe emphysema who should undergo lung volume reduction treatments and diagnosing concurrent disease. Furthermore, new quantitative analysis methods provide objective measurements of the disease's peripheral and pulmonary symptoms. These techniques can help explore the wide range and underlying causes of COPD and offer insightful information about the condition. (Kristoffer Ostridge et al, 2016)

In actuality, COPD is a complex collection of conditions with a variety of lung pathological alterations, extrapulmonary consequences, and comorbidities, all of which may exacerbate the illness. Airflow restrictions in the lungs are caused to varied degrees by parenchymal deterioration (emphysema), airways disease, and perhaps other reasons. Quantitative computed tomography (CT) may be an extremely interesting modality to detect these pathologies in vivo because its own independent analysis of pathological features may enable morphologic characterization, and visual evaluation of CT images for a typical ailment is

time-consuming and subject to significant observer variability. Over the past few decades, the use of quantitative CT to assess lung structure has increased, and emphysema quantification has drawn a lot of research interest. (Mets et al, 2012)

**MATERIAL AND METHOD:**

This is a descriptive cross-sectional study designed to determine the correlation of smoking history with imaging severity in COPD patients. The study will be conducted at Rahim Yar Khan Hospital, Pakistan. The data collection was carried out over a period of 3 months, from [Jan, 2026] to [Mar,2026]. Elderly Man (aged between 50 to 70) undergoing CT scan for the evaluation of respiratory conditions, including assessment of suspected COPD, will be included in the study. The study was involved total 135 patients.

**Data Analysis Procedure:**

- SPSS version 27.0 will be used for data entry and analysis.
- To summarize clinical and demographic information, descriptive statistics (mean, the standard deviation, and frequencies) will be employed.
- The coefficient of correlation Spearman will be used to assess the relationship between bronchial wall thickness and the type and severity of emphysema.
- A statistically significant p-value is one that is less than 0.05.

**RESULT & STATISTICAL ANALYSIS:**

There was a total of 135 patients who collected in this study. The data was analyzed using SPSS version 27 software. Results are presented in the form of frequency, percentage, and other descriptive statistics.

**Table 1:** ***Descriptive Statistics of Age and Duration of Respiratory Disease***

| Variable | N | Minimum | Maximum | Mean | Std. Deviation |
|---|---|---|---|---|---|
| **Age (years)** | 135 | 50 | 70 | 60.82 | 6.18 |
| **Duration of respiratory disease (years)** | 135 | 2 | 5 | 3.27 | 1.03 |

Total of 135 patients were studied. Mean age of the patient was 60.82 ± 6.18, with an age range of 50 to 70 years. It can be said that majority of the subjects belonged to the older age group.

Mean duration of the respiratory disease was 3.27 ± 1.03, with a range of 2 to 5 years. It is evident that the duration of illness was moderate for majority of the subjects.

**Table 2:** ***Demographic and Clinical Characteristics of Study Participants (N = 135)***

| Variable | Category | n | % |
|---|---|---|---|
| Gender | Male | 135 | 100.0 |
| | Female | 0 | 0.0 |
| Smoking History | Smoker | 105 | 77.8 |
| | Non-Smoker | 30 | 22.2 |
| Main Symptoms | Dyspnea | 42 | 31.1 |
| | Sputum Production | 32 | 23.7 |
| | Wheezing | 31 | 23.0 |
| | Chronic Symptoms | 30 | 22.2 |
| Comorbidities | Diabetes Mellitus | 53 | 39.3 |

| | | | |
|---|---|---|---|
| | Hypertension | 51 | 37.8 |
| | Ischemic Heart Disease | 31 | 23.0 |

Total participants involved in the study were 135 out of which 100% were males. The majority 77.8% of participants were cigarette smokers while non-smokers were only 22.2%. Among the clinical presentation, dyspnea was the most common 31.1% followed by sputum production 23.7%, wheezing 23.0%, and chronic complaints 22.2%. In terms of concomitant diseases, diabetes mellitus was the most prevalent 39.3% followed by hypertension 37.8%.

**Table 3:** ***Imaging Characteristics of Study Participants (N = 135)***

| Variable | Category | n | % |
|---|---|---|---|
| Emphysema Type | Panlobular | 54 | 40.0 |
| | Centrilobular | 42 | 31.1 |
| | Paraseptal | 39 | 28.9 |
| Emphysema Severity | Mild | 46 | 34.1 |
| | Moderate | 45 | 33.3 |
| | Severe | 44 | 32.6 |
| Air Trapping | Absent | 65 | 48.1 |
| | Present | 70 | 51.9 |
| Bronchial Wall Thickness | Absent | 66 | 48.9 |
| | Present | 69 | 51.1 |
| Bullae | Absent | 70 | 51.9 |
| | Present | 65 | 48.1 |

Imaging features indicated that panlobular emphysema was the predominant type (40.0%), followed by centrilobular and paraseptal types (31.1% and 28.9%, respectively). The distribution of the severity of the condition was quite balanced among mild (34.1%), moderate (33.3%), and severe cases (32.6%). Air trapping was found in 51.9% of the participants, whereas thickened bronchial walls were seen in 51.1% of patients.

**Table 4:** ***Spearman's Correlation between Smoking History and Emphysema Severity (N =135)***

| Variables | Smoking History | Emphysema Severity |
|---|---|---|
| **Smoking History** | 1.000 | -0.056 |
| **Emphysema Severity** | -0.056 | 1.000 |
| **Sig. (2-tailed)** | — | 0.519 |
| **N** | 135 | 135 |

A Spearman's rank correlation analysis was done to examine any correlation between smoking history and emphysema severity for 135 subjects. A very low correlation was found between smoking history and emphysema severity ($r = -0.056$, $p = 0.519$).

There was no statistical significance for the correlation ($p > 0.05$).

It can be concluded that there is no relationship between smoking history and emphysema severity. No significant correlation was found between smoking history and emphysema severity ($r = -0.056$, $p = 0.519$).

Table 5: ***Spearman's Correlation Between Smoking History and Emphysema Type (N =135)***

| Variables | Smoking History | Emphysema Type |
|---|---|---|
| **Smoking History** | 1.000 | -0.065 |
| **Emphysema Type** | -0.065 | 1.000 |
| **Sig. (2-tailed)** | — | 0.454 |

Spearman rank correlation was done to measure the degree of correlation between the smoking history of a patient and their emphysema type for 135 patients. There was a very low negative correlation between the smoking history and the emphysema type ($r = -0.065$, $p = 0.454$), which was not statistically significant ($p > 0.05$). This means that smoking history has no significant correlation with emphysema type.

Table 6: ***Spearman's Correlation Between Smoking History and Bronchial Wall Thickness (N=135)***

| Variables | Smoking History | Bronchial Wall Thickness |
|---|---|---|
| **Smoking History** | 1.000 | 0.024 |
| **Bronchial Wall Thickness** | 0.024 | 1.000 |
| **Sig. (2-tailed)** | — | 0.784 |

To determine the degree of association between smoking status and bronchial wall thickness, a Spearman's rank correlation test was conducted on 135 patients. The outcome revealed that there was a very weak positive correlation between smoking status and bronchial wall thickness ($r = 0.024$, $p = 0.784$). There was no statistically significant association between the two variables ($p > 0.05$).

Table 7: ***Smoking History Correlation Summary Table (N=135)***

| Variable | Correlation ($r_s$) | p-value |
|---|---|---|
| **Emphysema severity** | -0.056 | 0.519 |
| **Emphysema type** | -0.065 | 0.454 |
| **Bronchial wall thickness** | 0.024 | 0.784 |

Spearman's correlation coefficient revealed that the relationship was quite poor between the smoking status and the severity of emphysema, as well as the presence of different types of emphysema and bronchial wall thickening. The findings indicated no statistical significance for any of these correlations ($p > 0.05$).

Table 8: *ANOVA for Predictors of Emphysema Severity*

| Model | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|
| **Regression** | 1.618 | 3 | 0.539 | 0.800 | 0.496 |
| **Residual** | 88.352 | 131 | 0.674 | — | — |
| **Total** | 89.970 | 134 | — | — | — |

A multiple regression analysis (ANOVA) was conducted to examine whether age, respiratory illness duration, and smoking history were significant predictors of emphysema severity. However, the results were not statistically significant ($F(3,131) = 0.800$, $p = 0.496$), showing that these variables as a whole do not explain variance in emphysema severity. Therefore, the conclusion can be made that age, smoking history, and respiratory illness duration were not good predictors of emphysema severity.

**DISCUSSION:**

The goal of the current study was to assess the relationship between smoking history and imaging severity in patients with COPD who were between the ages of 50 and 70. Although it is often known that smoking is a major contributing factor to the progressive respiratory condition known as chronic obstructive pulmonary disease (COPD), the results of this study offer an intriguing viewpoint on this relationship, especially with regard to radiological severity.

The study comprised 135 male patients with a mean age of 60.82 ± 6.18 years. This suggests that the majority of the research population was older, which is in line with the natural history of COPD since the illness usually shows symptoms after extended exposure to risk factors like smoking. The majority of patients appeared to be in the early to moderate stage of disease progression, as shown by the average duration of respiratory illness of 3.27 ± 1.03 years.

Regarding smoking history, 22.2% of participants did not smoke, compared to 77.8% who did. This is consistent with the body of research that shows smoking to be the main risk factor for developing COPD. The existence of a sizable percentage of non-smokers, however, also emphasizes the significance of additional contributing elements such genetic vulnerability, occupational exposure, exposure to biomass fuels, and environmental pollution.

According to the patients' clinical presentation, the most prevalent symptom was dyspnea (31.1%), which was followed by wheezing, sputum production, and persistent respiratory symptoms. These results align with the traditional symptomatology of COPD reported in earlier research. Given the older age group and the systemic character of COPD, concomitant diseases such diabetes mellitus (39.3%) and hypertension (37.8%) were also very common.

According to radiological results, the most prevalent type of emphysema was panlobular (40.0%), followed by centrilobular (31.1%) and paraseptal (28.9%). While panlobular emphysema is frequently connected to hereditary disorders such alpha-1 antitrypsin deficiency, centrilobular emphysema has historically been more frequently linked to smoking. The prevalence of panlobular emphysema in this study may indicate problems in classification, regional variations, or variations in exposure patterns.

The mild (34.1%), moderate (33.3%), and severe (32.6%) categories of emphysema severity were very evenly distributed. This equal distribution shows that patients from a range of illness severity stages were included in the study sample, allowing for a thorough evaluation of imaging results. Additionally, around half of the patients had imaging characteristics like air trapping (51.9%), bronchial wall thickening (51.1%), and bullae development (48.1%), which further supported the variety of COPD symptoms.

This study's main goal was to ascertain whether smoking history and imaging severity are related. However, the findings showed that smoking history and the severity of emphysema had a very weak and statistically negligible connection ($r = -0.056$, $p = 0.519$). Similarly, smoking history did not significantly correlate with either bronchial wall thickness ($r = 0.024$, $p = 0.784$) or emphysema type ($r = -0.065$, $p = 0.454$).

Given that smoking is generally thought to be a significant factor in determining the severity of COPD, these results are quite surprising. Nonetheless, a number of explanations are possible. First, the study's measurement of smoking history may have been restricted to a binary classification (smoker vs. non-smoker), which fails to account for the duration or intensity of smoking exposure. Stronger associations may have been obtained from variables like pack-years, age of smoking initiation, and cessation status, which are more reliable measures of cumulative exposure.

Second, smoking is not the only element that affects the severity of COPD; it is a complex disease. These include comorbid conditions, socioeconomic status, genetic predisposition, environmental exposures, and access to healthcare. This study's lack of substantial link raises the possibility that smoking is not the only factor contributing to the variation in imaging severity among COPD patients.

Third, the comparatively short duration of respiratory illness (mean ~3 years) may suggest that many patients had not yet reached advanced radiological abnormalities, which would limit the discernible correlation between smoking and the severity of imaging. The course of COPD is usually sluggish, and early clinical symptoms may not be closely correlated with radiological findings.

This conclusion is further supported by the multiple regression analysis (ANOVA) results, which showed that age, smoking history, and length of respiratory disease were not significant predictors of emphysema severity ($F = 0.800$, $p = 0.496$).

**CONCLUSION:**

According to the study's findings, smoking does not significantly correlate with imaging severity in individuals between the ages of 50 and 70, despite being a major risk factor for COPD. Smoking history did not significantly correlate with bronchial wall thickness, type, or severity of emphysema, according to statistical analyses. Furthermore, the length of the respiratory infection and age did not significantly predict the severity of the sickness. These results suggest that radiological abnormalities in COPD cannot be explained by smoking alone. Genetic, environmental, and personal factors all seem to play significant roles in the complex nature of the illness. The participation of additional risk variables is further supported by the existence of COPD among non-smokers. Common imaging characteristics were seen, including bronchial wall thickening, bullae development, and air trapping. In general, smoking history should not be the only factor used to determine the severity of COPD.

**RECOMMENDATION:**

To increase the reliability and generalizability of the results, a larger multi-center investigation ought to be carried out in the future.

To more accurately assess gender-related variations in imaging severity, future studies should include both male and female COPD patients.

Instead than only classifying people as smokers or non-smokers, comprehensive smoking exposure metrics such pack-years, smoking duration, and smoking cessation status should be evaluated.

Since COPD can also affect non-smokers, environmental and occupational risk factors such exposure to biomass fuels, air pollution, and passive smoking should be assessed.

To further understand their impact in the severity and course of COPD, genetic variables and family history should be studied.

It is advised to conduct longitudinal studies to track the course of the disease over time and ascertain whether extended smoking exposure alters the severity of imaging.

For a more precise evaluation of air trapping, airway remodeling, and the severity of emphysema, advanced imaging methods and quantitative CT analysis should be used.

To obtain a more thorough assessment of the severity of COPD, pulmonary function tests (PFTs) and HRCT results should be correlated.

To lessen the burden of the disease and enhance patient outcomes, awareness campaigns on quitting smoking and early COPD screening should be supported.

When assessing the severity of COPD imaging, medical professionals should take into account a variety of contributing factors instead of only smoking history.

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