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Evaluation of Clinical, Functional, and Radiological Characteristics of Chronic Obstructive Pulmonary Disease Patients with Occupational Exposure

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ABSTRACT

Objective: In recent years, non-smoking factors contributing to the etiology of Chronic Obstructive Pulmonary Disease (COPD) have gained attention. Occupational exposures are known to account for 14% of the COPD burden. In this study, we aimed to document the demographic, clinical, functional, and radiological characteristics of COPD patients who presented to the occupational diseases outpatient clinic with respect to their occupational exposure.

Materials and Methods: The records of 33 patients with a final diagnosis of COPD, admitted to our outpatient clinic between 2013 and 2022, were analyzed retrospectively. The COPD diagnosis was made by an experienced occupational diseases specialist and two pulmonologists. Subgroups were created as non-occupational, work-exacerbated COPD, and occupational COPD.

Results: All patients were male. The patients had a history of working in various sectors, including mining, metal processing, textiles, ceramics, construction, dental prosthesis manufacturing, cement production, food production, denim sandblasting, transport, and brick production. Six patients (18.2%) were diagnosed with non-occupational COPD, 16 (48.5%) with work-exacerbated COPD, and 11 (33.3%) with occupational COPD. There were no associated effects of smoking and dust exposure on forced expiratory volume in one second (FEV1) and FE1/FVC (Forced Vital Capacity) levels. Furthermore, 69.7% of the cases had more than one type of exposure (multiple exposure), and there was no statistically significant difference between occupational status and multiple or single exposure. No significant association was found between COPD-related radiological findings and the duration of exposure to silica dust, coal dust, metal dust, or organic dust.

Conclusion: Vapors, dust, smoke, and gases in the workplace may contribute to COPD, even among non-smokers. Patients with suspected occupational exposure should be referred to occupational health and occupational diseases outpatient clinics.

Keywords: Chemicals, chronic obstructive pulmonary disease (COPD), dust, occupational exposure, smoking.

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a heterogeneous disease characterized by chronic respiratory symptoms, as well as airway and alveolar abnormalities that lead to persistent and often progressive airflow limitation. The global prevalence of COPD was estimated at 10.3% in 2019.[1] Chronic obstructive pulmonary disease is the third leading cause of death worldwide, accounting for at least 3 million deaths annually.[2] In our country, the prevalence of COPD ranges between 9.1% and 19.1%, with the disease ranking third among causes of death and eighth among causes of disability.[3] It is known that approximately 80–90% of COPD patients have a history of smoking, and half of smokers develop COPD.[4]

Inhalations of vapors, gasses, dust, or fumes (VGDF) in the workplace is known to contribute to the burden of COPD. In particular, coal dust, silica, construction dust, diesel exhaust, welding fumes, cotton dust, asbestos, and grain dust have been shown to increase the risk of developing COPD independently of smoking. It is also known that smoking combined with occupational exposure to VGDF presents a greater risk than the cumulative effects of both individually.[5] Although smoking is the predominant factor in the etiology of COPD, the occupational contribution is estimated to be approximately 14%. The pooled Population Attributable Fraction (PAF) for occupational contribution to COPD is 14% (95% Confidence Interval [CI], 10–18%). For non-smokers, the pooled PAF for occupational contributions to COPD is 31% (95% CI, 10–18%).[6] A 2019 systemic review found that exposure to occupational dust or smoke alone increased the risk of COPD by 1.4 times (Odds ratio [OR]: 1.4, 95% CI 1.3–1.6).[7] The fact that an estimated 12% of COPD deaths are caused by occupational exposure to airborne particles is significant for maintaining continuity in work life.[8]

Regarding respiratory diseases, workers exposed to dust may develop different conditions such as pneumoconiosis, asthma, and hypersensitivity pneumonitis. Due to deficiencies in detecting early signs and symptoms, the diagnosis of occupational COPD, which may coexist with these diseases, can often be overlooked.[9] According to the 2021 annual statistics from the Social Security Institution, no notifications have been made recently with the J44-ICD10 code.[10]

In our country, research on occupational COPD is insufficient. However, this issue should be addressed to prevent the additional adverse effects of occupational exposure on the disease. Therefore, this study aimed to examine the relationships between occupation-related exposures and the clinical, functional, and radiological characteristics of COPD patients evaluated at an occupational medicine outpatient clinic.

KEY MESSAGES

- Since smoking is a major factor in the etiology of chronic obstructive pulmonary disease (COPD), establishing a causal relationship for diagnosing occupational COPD is challenging.
- Currently, the number of patients diagnosed with occupational COPD within the Social Security Institution is limited due to insufficient occupational history and the predominant confounding effect of smoking.
- Workers with suspected obstructive pulmonary disease who are employed in high-risk environments (involving vapors, dust, gas, and fumes) should be referred to the occupational medicine outpatient clinic.

MATERIALS AND METHODS

This study was conducted at the occupational medicine clinic of a university hospital after receiving approval from the Local Ethics Committee (9 Eylul University Ethics Committee-20.07.2022-7315GOA). The medical records of 33 patients with a confirmed diagnosis of COPD, admitted between 2013 and 2022, were analyzed retrospectively.

The occupational exposures of the patients were evaluated based on how they were recorded in the work history, the physician's opinion, and sectoral information. The duration, dose, and frequency of exposure were considered to assess the intensity of exposure. Smoking was categorized as low (≤10 pack-years), medium (11–20 pack-years), and high (≥21 pack-years). Those with a low smoking history and sufficient occupational exposure, as determined by expert opinion, were classified as having occupational COPD. Those with a medium-to-high smoking history and insufficient occupational exposure, according to expert opinion, were placed in the non-occupational COPD group. Individuals with a medium-to-high smoking history and sufficient occupational exposure, according to expert opinion, were categorized as having work-exacerbated COPD (Table 1).

Independent variables included gender, age, occupational class, comorbidities, asbestos exposure status, respiratory symptoms, presence of VGDF exposure, employment status after the occupational diseases outpatient evaluation, pulmonary function test results (forced expiratory volume in one second [FEV1], forced vital capacity [FVC], FEV1/FVC ratio), and thorax computed tomography (CT) findings.[11] The main diagnostic criterion for COPD was an FEV1/FVC ratio of less than 70% in the post-bronchodilator pulmonary function test, following the Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2022 guidelines (For access to the report: https://goldcopd.org/2022-gold-reports/).

Table 1. Evaluation of the relationship between smoking and occupational exposure to dust and and vapors, gasses, dusts, and fumes (VGDF)

	Occupational exposure (duration x dose x frequency)	Cigarette smoking (pack/day x years)
Occupational COPD	Adequate	Low
Non-occupational COPD	Inadequate	Medium-high
Work-exacerbated COPD	Adequate	Medium-high

COPD: Obstructive pulmonary disease.

Statistical analysis was performed using the SPSS 24.0 (Statistical Package for the Social Sciences) program. The Shapiro-Wilk test was used to assess the suitability of the variables for normal distribution. For data that were not normally distributed, continuous numerical variables were presented as median (minimum–maximum). Categorical variables were displayed as the number of cases (n) and percentage (%). Spearman's correlation test was used to assess relationships between continuous or numerical variables, and the point-biserial correlation test was employed for relationships between binary or categorical variables. The results were analyzed with a 95% confidence interval, and a p value of less than 0.05 was considered statistically significant.

Ethical approval was obtained from the non-interventional ethics committee of our university (9 Eylul University Ethics Committee-20.07.2022-7315GOA). Verbal informed consent was obtained from the patients, and written consent was secured from the institution.

RESULTS

A total of 33 cases were included in the study. All patients were male, blue-collar workers, and low-income. The mean age was 48.8±8.41 years. The most common symptom at presentation was dyspnea (81.8%) and the Modified Medical Research Council (MMRC) score was 1 in 54.5% of the patients. According to the 2022 GOLD guidelines,[12] the distribution was as follows: A= 72.7%, B= 21.2%, and D= 6.1%.

While 57.6% (n=19) of the patients had only COPD, 33.3% (n=11) had one additional comorbidity, and 9.1% (n=3) had two or more comorbidities. In 48.5% of the patients, the diagnosis was limited to COPD, while the others had additional occupational diseases (pneumoconiosis, noise-induced hearing loss, lumbar disc herniation). When the employment status of 23 of our patients was analyzed, it was observed that four patients were unemployed at the time of admission, and five were unemployed after the admission.

Table 2. Evaluation of the sectors, levels of exposure to risk factors, and occupational classification of the cases based on work history

	n	%
Sectors		
Mining	9	27.3
Metal processing	9	27.3
Textile	3	9.1
Building construction	2	6.1
Ceramic	2	6.1
Transport	2	6.1
Dental prosthesis	1	3
Cement	1	3
Food	1	3
Denim sandblasting	1	3
Brick	1	3
Furniture	1	3
Total	33	100
Cigarette pack-year		
Low	11	33.3
Medium	8	24.2
High	14	42.4
Total	33	100
Occupational classification		
Occupational COPD	11	33.3
Work-exacerbated COPD	16	48.5
Non-occupational COPD	6	18.2
Total	33	100

COPD: Obstructive pulmonary disease.

The frequencies regarding the sectors, smoking classification levels, and occupational COPD groups are shown in Table 2. It was noted that the applicants had a history of working in various sectors (mining, metalworking, textile, ceramics, construction, dental prosthesis, cement, food production, denim grinding, transport, and brick production). A history of exposure to silica dust was present in 54.5% of cases, metal dust in 45.5%, coal dust in 12.2%, and organic dust in 30.3% (Table 3). In addition, exposure to solvents, paints, or resins was identified in 51.5% of cases, and smoke exposure in 30.3%. In 48.5% of cases, in addition to dust exposure, exposure to a chemical substance perceived by the subjects as an odor or other smoke was also reported. One patient had asbestos exposure. The median duration of dust exposure was 20 years (range: 5–33 years). There

Table 3. Types of dust encountered by the subjects during their work life

Type of dust	n	%
Silica	18	54.5
Metal dust	15	45.5
Coal	2	12.1
Organic dust	10	33.3

were 9.1% (n=3) never smokers, 39.4% (n=13) former smokers, and 51.5% (n=17) current smokers (to access Centers for Disease Control and Prevention [CDC] definition: https://www.cdc.gov/nchs/nhis/tobacco/tobacco_glossary.htm). The proportions of patients with low, medium, and high smoking history were 33.3% (n=11), 24.2% (n=8), and 42.4% (n=14), respectively. The median FEV1 level was 1760 mL (range: 800–3410 mL), and the median FEV1/FVC ratio 60.9% (range: 40–69.5%) at the time of presentation. Thorax CT/HRCT (high-resolution computed tomography) showed emphysema in 17 (51.5%) patients, bronchial thickening in nine (27.3%) patients, sequelae/fibrotic changes in eight (24.2%) patients, atelectasis in six (18.2%) patients, and bronchiectasis in four (12.1%) patients. In two patients, lung cancer was detected following investigations performed due to a suspicion of malignancy (Table 4).

The correlation of cigarette pack-years and dust exposure with FEV1 and FE1/FVC levels is presented in Table 5. Additionally, there was more than one exposure (multiple exposure) in 69.7% of the cases. There were no statistically significant differences between occupational status and multiple or single exposures. No significant correlation was found between radiological findings, particularly emphysema and bronchial thickening, and exposure time to silica dust, coal dust, metal dust, or organic dust (p>0.05) (Table 6).

DISCUSSION

This study emphasizes that occupational exposure to air pollutants in the workplace, particularly in cases where the impact of smoking is low, should be carefully considered.

Table 4. Thorax high-resolution computed tomography (HRCT) radiologic findings of patients

Radiologic findings	n	%
Emphysema		
Yes	17	51.5
No	16	48.5
Bronchial thickening		
Yes	9	273
No	24	72.7
Sequelae fibrotic changes		
Yes	8	24.2
No	25	75.8
Atelectasis		
Yes	6	18.2
No	27	81.8
Bronchiectasis		
Yes	4	12.1
No	29	87.9
Malignant lesion		
Yes	2	6.1
No	31	93.9
Mediastinal lymphadenomegaly		
Yes	7	21.2
No	26	78.8

Moreover, cases with a low smoking history and sufficient occupational exposure are suggested to be classified as occupational COPD.

Occupational diseases are nearly 100% preventable. Although COPD is often prioritized among occupational lung diseases, diagnosing occupational COPD is challenging because smoking and occupational exposures frequently coexist in the etiology.[13] It is estimated that approximately 12% of COPD deaths are caused by occupational exposure to airborne particles.[8] After

Table 5. Correlation of cigarette pack-years and dust exposure with forced expiratory volume in the first second (FEV1) and forced expiratory volume in the first second/forced vital capacity (FEV1/FVC) levels at admission

	Cigarette pack-years p*	Cigarette pack-years r	Dust exposure per year p*	Dust exposure per year r
FEV1 level at admission	0.444	0.138	0.752	0.605
FEV1/FVC level at admission	0.460	0.135	0.057	0.095

FEV1: Forced expiratory volume in the first second; FVC: Forced vital capacity; *: Spearman correlation test.

Table 6. Correlation between emphysema, bronchial thickening, and duration of exposure to silica dust, metal dust, and organic dust

	Silica dust p*	Silica dust r	Coal dust p*	Coal dust r	Metal dust p*	Metal dust r	Organic dust p*	Organic dust r
Emphysema	0.624	0.89	0.272	-0.197	0.240	-0.210	0.912	-0.020
Bronchial thickening	0.491	-0.124	0.203	-0.227	0.945	-0.012	0.551	-0.108

*: Spearman correlation test.

adjustment for gender, age, smoking status, and socioeconomic status, the etiological role of occupational factors in COPD was found to be 19% in smokers and 31% in non-smokers.[14] The results highlight the importance of early detection of the interaction between COPD and occupational exposure, as early recognition of the occupational component can help prevent the onset of the disease. Unfortunately, there are currently two critical barriers to effective prevention. The first involves the identification of COPD, which typically occurs by diagnosing bronchial obstruction at a stage when it is already irreversible. The second obstacle is the socioeconomic consequences of changing jobs before the disease reaches an advanced stage and/or recurrent exacerbations occur. Therefore, if occupational exposure is not eliminated, the pathophysiological vicious circle continues, diminishing the effectiveness of treatment.[9] However, stopping occupational exposure in the early phase of COPD is the most effective approach.

In this study, patients with occupational COPD were more frequently employed in industries (mining, construction, ceramics, etc.) associated with air pollutants, and the socioeconomic status and social welfare of workers in these sectors were low.

While smoking is the main risk factor, other factors such as air pollution and workplace pollutants are also associated with COPD. Occupational exposure to vapors, gasses, dusts, and fumes contributes to the development and progression of COPD, accounting for a population attributable fraction of 14%.[15] A 20-year prospective follow-up study of a large population demonstrated that exposure to metals and mineral dust is particularly associated with chronic sputum production and chronic bronchitis.[16]

Consequently, the differential diagnosis of occupational lung disease is necessary to identify workplace risks and to ensure that appropriate warnings are issued to take preventive measures. Just as smoking cessation is the best therapeutic management for classic COPD, eliminating exposure is the most effective intervention for occupational COPD. There continues to be a challenge in under-diagnosing occupational COPD, even when the term "compensated occupational COPD" is used for cases where other strong risk factors, such as smoking, are not present. This approach may discourage occupational medicine specialists from reporting occupational COPD to avoid affecting the workers' compensation insurance. Additionally, employees themselves may sometimes deny workplace exposure out of fear of possible consequences to their employment. Moreover, some smokers may not want to quit smoking, preferring that the blame for their illness remains on cigarettes.[15] Despite these challenges, we believe that at least patients with multiple workplace exposures and a low smoking history should be diagnosed with occupational COPD, considering the importance of preventive disease management and the future socioeconomic burden.

In 2022, the Lancet COPD commission report revealed that more than 70% of 22,000 participants surveyed via social media reported not having received any training on protecting lung health at work, and over 90% reported having to leave their previous job due to respiratory symptoms.[17] In a population-based cross-sectional study, the likelihood of having COPD was found to be 5.8 and 6.9 times higher, respectively, in subjects exposed to tobacco and occupational smoke for more than 20 years compared to reference subjects exposed for less than 10 years.[18] As noted in the Lancet COPD commission report of 2022, occupational exposures have a significant relationship with the pathophysiological features of COPD, such as chronic cough, airway flat hypertrophy, persistent airflow limitation, and parenchymal destruction.[17] Consistent with the industries represented in our cases, occupation-related causes of COPD have been identified in a wide range of industry-based studies. In alignment with our findings, these sectors include coal mining, construction, tunneling, brick manufacturing, pottery and ceramic production, silica sand production, iron and steel foundry work, welding, cotton production, and agriculture.[19]

In a study of 100,000 people from the UK Biobank cohort, the occupations most at risk of developing COPD included sculptor, painter, carver, art restorer, gardener-park keeper, food, drink, and tobacco processor, plastics processor, mold maker, agricultural and fishing occupations, and warehouse stock keeper and stacker.[20] In the changing and developing world,

occupations have started to diversify, and these changes shape risks according to the socioeconomic development status of countries. For example, the inhalation of nanoparticles, which is a current concern, has been associated with COPD in animal experiments.[21] Recently, we have had limited clinical experience with nanoparticle-associated COPD. Therefore, since occupational COPD has a national or country-specific dimension, each country should contribute to the literature by presenting its own findings.

On the other hand, having COPD significantly reduces patients' work productivity and employment rates. A recent study of approximately 2,500 patients aged 45–67 found that about 40% of patients retired early due to COPD at an average age of 54. The same study emphasized that people with COPD are more likely to be unemployed due to the disease.[20] When the employment status of 23 of our patients was analyzed, 15.2% were unemployed at the time of admission, and 21.2% were unemployed after admission. This is particularly important for COPD cases exacerbated by occupational factors, as it is crucial to protect these individuals both from becoming ill due to their work and from being fired because of workplace exposures.

Although it has not been definitely determined that occupational COPD occurs directly, a study reported a significant decrease in spirometry measurements among furniture workers exposed to organic wood dust in furniture factories compared to workers with six years of experience who were not exposed to dust, in terms of pulmonary function.[22] These data highlight the importance of monitoring pulmonary function parameters during periodic examinations and implementing protective measures against air pollutants, even in asymptomatic patients working in high-risk environments.

In one study, a significant decrease in FEV1 and the FEV1/FVC ratio was observed in individuals with both occupational dust exposure and a smoking history compared to non-smokers, with a greater decrease observed in current smokers.[23] Similarly, in another study on airflow limitation, patients exposed to VGDF had lower levels of current and cumulative smoking exposure than COPD patients who were never exposed to occupational hazards.[24] A study examining the association between cigarette pack-years (low (≤20) and high (>20)) and occupational exposures found that the odds ratios (ORs) for low pack-years alone and VGDF exposure alone were approximately equal, while the combined effect of both exposures was greater than the sum of the individual effects.[25]

Although smoking is the leading cause of COPD, there is no established link between occupational exposure and COPD in smokers.[26] Understanding the burden of occupational exposures on COPD will only be possible if challenges such as the lack of access to adequate cohorts and the effects of multiple risk factors, including concurrent smoking, are addressed. A 2015 cross-sectional study showed that occupational exposures increase the likelihood of COPD even among heavy smokers. The same study also found that occupational exposures were associated with higher rates of COPD and increased morbidity in patients with COPD. This effect was observed even in patients who had a significant smoking history and had quit smoking.[13] In 2021, the Workplace Safety and Insurance Board conducted a rapid literature review, which concluded that although a decrease in the rate of lung impairment decline is expected with the cessation of occupational exposure, there is limited evidence from the last five years, and it is not possible to distinguish between the causes of COPD with our current knowledge.[27] While the causal relationship can be established more clearly in non-smokers within the field of occupational diseases, there are serious problems in establishing this relationship in smokers. In the study by Fischwick et al.,[28] increasing pack-years of tobacco smoking was associated with a significant decrease in proportional occupational causality ratings. A significant decrease in the assessment of occupational disease was observed in proportion to the increase in cigarette pack-years. Increasing weighted occupational exposure was associated with a 0.28% increase in occupational causality ratings per unit change. For COPD, 20 pack-years of cigarette smoking and 20 years of exposure to high occupational risk were considered similar. Although some evaluations can be made based on the relationship between years of dust exposure and cigarette pack-years, there is currently insufficient data available.[29] In occupational disease outpatient clinic practice, while the causal relationship can be more clearly established in non-smokers, significant problems remain in establishing this relationship in smokers.

The limitations of our study include the small sample size, lack of detailed knowledge of environmental exposures other than asbestos, and lack of objective occupational exposure levels over the years. Prospective studies with large cohort groups are needed to better evaluate occupational exposure, particularly in cases with major confounding factors such as smoking. The small sample size in this study is due to the limited number of COPD patients without pneumoconiosis being referred to the occupational diseases outpatient clinic. Since smoking is the leading etiologic factor in COPD, many physicians do not take an occupational history and do not refer the majority of cases to occupational medicine. Therefore, in our study, the cause-effect relationship could not be analyzed in detail, and the results should be interpreted with caution. Based on the findings of this preliminary study, future cohort studies should evaluate both occupational exposures, including subjective risk assessments such as the Job Exposure Matrix, and smoking exposure.

CONCLUSION

This study emphasizes that even employees who do not smoke or have low levels of smoking but are exposed to workplace pollutants would rather be diagnosed with occupational COPD. In addition, smoking is quite common among blue-collar workers, as it is more prevalent among individuals with lower socioeconomic status. Therefore, the risk of developing COPD due to exposure to occupational air pollutants may be considerably higher than previously recognized. Early diagnosis and referral of occupational COPD cases to relevant clinics are crucial to prevent further decline in lung function due to exposure and to reduce the health and socioeconomic burden on workers.

Ethics Committee Approval: The Dokuz Eylul University Non-Interventional Research Ethics Committee granted approval for this study (date: 20.07.2022, number: 2022/23-03).

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