

# Clinical Characteristics and Interstitial Findings on High-Resolution Computed Tomography in Patients with Coal Workers' Pneumoconiosis

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**KEYWORDS:** Coal Workers' Pneumoconiosis; Interstitial Lung Disease; High-Resolution Computed Tomography

## ABSTRACT

**Background:** Coal workers' pneumoconiosis (CWP) is a parenchymal lung disease resulting from the prolonged inhalation of coal dust. Coal dust exposure may also lead to a spectrum of airway and parenchymal disorders. This study aimed to investigate the clinical characteristics, radiological and functional findings of CWP, as well as the presence of interstitial changes and associated risk factors. **Methods:** Patients with CWP who were admitted to the occupational diseases clinic of a tertiary hospital between 2017 and 2023 were included. Demographic, radiological, and pulmonary functional data were analyzed. High-resolution computed tomography (HRCT) scans were reviewed for interstitial lung abnormalities (ILA) and interstitial lung disease (ILD). Factors associated with ILD were assessed using logistic regression analysis. Statistical significance was defined as  $p < 0.05$ . **Results:** One hundred male patients with CWP (mean age,  $61.7 \pm 11.7$  years) were evaluated. Large opacities were present in 37% of cases. Pulmonary function testing revealed restrictive impairment in 35% and obstructive impairment in 38% of patients. Compared with those working lignite mines, bituminous coal miners exhibited a significantly higher prevalence of large opacities and lower lung function values. ILAs were identified in 63% of patients, and ILD in 42%. ILD prevalence was significantly higher among those with exposure durations exceeding 10 years, working in bituminous coal mines, and with predominant small opacities larger than 3 mm. **Conclusions:** Coal dust exposure is a risk factor not only for CWP but also for impaired lung function, ILA, and ILD. Comprehensive surveillance of coal workers is essential for early detection and timely management of these conditions.

## 1. INTRODUCTION

Coal workers' pneumoconiosis (CWP) is a chronic parenchymal lung disease caused by prolonged inhalation of coal dust [1]. The prevalence and severity of CWP depend on coal type, dust concentration,

exposure duration, and the composition of carbon, silica, and other minerals [2]. Based on coal rank, sub-bituminous coal and lignite are classified as "soft coal," whereas bituminous coal and anthracite are considered "hard coal." Differences in mining methods also affect the particle surface area, free

radical potential, and silica content of the inhaled dust. The characteristic lesion of CWP is the coal macule, composed of dust-laden macrophages surrounding respiratory bronchioles, often associated with focal emphysema. Fibrotic nodules and progressive massive fibrosis (PMF) may also develop, reflecting more advanced disease [3, 4]. Because miners are typically exposed to mixed dusts containing coal, kaolin, mica, and silica, most patients also present with mixed-dust fibrosis and silicotic nodules. Together with CWP, silicosis, mixed-dust pneumoconiosis, coal dust-related diffuse fibrosis (DDF), emphysema, and chronic bronchitis, these entities constitute the clinical spectrum of coal mine dust lung disease (CMDLD) [5].

Diffuse interstitial pulmonary fibrosis may also be present in coal miners. Autopsy studies have demonstrated DDF in 15-20% of miners [6]. DDF is characterized by bridging fibrosis related to macular, nodular, or PMF lesions of CWP or silicosis, typically exhibiting pigmented interstitial septal thickening [5, 6]. Moreover, coal dust exposure has been associated with chronic interstitial pneumonias, desquamative interstitial pneumonia (DIP), and combined pulmonary fibrosis and emphysema (CPFE) [7-9]. Despite these observations, data regarding the prevalence and determinants of interstitial abnormalities on high-resolution computed tomography (HRCT) in CWP remain limited.

This study aimed to evaluate the clinical and radiological characteristics of patients with CWP in a tertiary occupational diseases clinic, to analyze pulmonary function findings, and to investigate interstitial abnormalities on HRCT and their associated risk factors.

## 2. METHODS

### 2.1. Study Population

This retrospective, cross-sectional study was approved by the Ethics Committee of Ankara Atatürk Sanatorium Training and Research Hospital (Dated 12 July 2023 and numbered 2012-KAEK-15/2749). Patients diagnosed with CWP in the occupational diseases outpatient and inpatient clinics between January 2017 and January 2023 were screened. Those

with available HRCT scans in the hospital information system were included. Patients with suspected interstitial lung diseases (ILDs) due to other causes (e.g., DIP, RB-ILD, hypersensitivity pneumonitis) were evaluated using laboratory and pathological findings; those with alternative ILD diagnoses were excluded. Demographic data, smoking status, occupational history, pulmonary function test (PFT) results, chest radiographs, and HRCT scans were obtained from patient files and hospital records.

### 2.2. Chest Radiography Assessment

Chest radiographs were evaluated according to the International Labour Organization (ILO) classification system for pneumoconiosis [10]. Small opacities <1 cm were classified as irregular (s, t, u) or rounded (p, q, r) based on shape, and graded into categories 1, 2, or 3 according to profusion. Large opacities  $\geq 1$  cm were considered PMF and classified as A, B, or C. Patients with PMF were categorized as having complicated pneumoconiosis, whereas those without PMF were categorized as having simple pneumoconiosis. Radiographs were independently assessed by three certified ILO readers (all pulmonologist and occupational medicine specialists). In cases of disagreement, consensus was reached through joint review.

### 2.3. Pulmonary Function Testing

Pulmonary function testing was performed by experienced technicians in accordance with the American Thoracic Society / European Respiratory Society (ATS/ERS) standards. Spirometry results were expressed as percentages of predicted values. An FEV1/FVC ratio <0.7 was defined as obstructive impairment [11], while an FEV1/FVC ratio  $\geq 0.7$  with FVC <80% predicted was defined as restrictive impairment [12]. Patients with both obstruction and reduced FVC were classified as having mixed impairment.

### 2.4. HRCT Imaging and Evaluation

Thin-section CT images with a slice thickness of 1 mm, obtained using a 128-slice multidetector

Philips Ingenuity CT scanner, were evaluated with a bone reconstruction algorithm in the lung window settings (window width: 1500, window level: -600) by a radiologist with 30 years of experience in thoracic radiology. HRCT patterns were interpreted using standardized descriptors for occupational and environmental lung diseases, consistent with the International Classification of HRCT for Occupational and Environmental Respiratory Diseases [13].

### 2.5. Assessment of Interstitial Abnormalities

In addition to CWP-specific findings, interstitial lung abnormalities (ILA) and ILD were assessed according to the 2025 ATS Clinical Statement (14). Bilateral, nondependent ground-glass opacities, reticular abnormalities, architectural distortion, traction bronchiectasis, and/or honeycombing involving >5% of a lung zone were considered ILA. Consistent with the ATS definition of ILD in individuals with ILA, a diagnosis of ILD required the presence of at least one additional criterion beyond HRCT abnormalities. These criteria included: respiratory symptoms attributable to ILD (dyspnea and/or cough); objective physiological impairment on pulmonary function testing, defined as reduced FVC and/or DLCO (<80% of predicted); radiological progression on serial chest CT; or pathological evidence of fibrotic ILD. Nodular and macular CWP-related lesions were not classified as ILA/ILD.

### 2.6. Statistical Analysis

All statistical analyses were performed using IBM SPSS Statistics for Windows, version 22.0 (IBM Corp., Armonk, NY, USA). Categorical variables were presented as counts and percentages, whereas continuous variables were expressed as medians with interquartile ranges (IQR). The chi-square or Fisher's exact tests were used for comparisons of categorical variables, and the Mann-Whitney U or Kruskal-Wallis tests were used for continuous variables, as appropriate. Univariate logistic regression analyses were conducted to assess the associations between independent variables and ILD. Variables with a p-value <0.20 in the univariate analysis were

considered potential candidates and subsequently included in the multivariate logistic regression model. Before multivariate analysis, correlations among independent variables were examined using a correlation matrix. To avoid multicollinearity and ensure model stability, variables with a pairwise correlation coefficient ( $r$ ) greater than 0.6 were considered highly correlated. In such cases, the variable deemed less clinically relevant or statistically significant was excluded from the final model. The results of logistic regression analyses were expressed as odds ratios (ORs) with 95% confidence intervals (CIs). A p-value <0.05 was considered statistically significant.

## 3. RESULTS

Between January 2017 and January 2023, 130 patients were diagnosed with CWP, of whom 100 with available HRCT scans were included in the analyses. All patients were male, with a mean age of  $61.7 \pm 11.7$  years. Sixty-seven had worked in bituminous coal mines and 33 in lignite coal mines. Comorbidities other than pneumoconiosis were present in 67 patients. COPD was the most common comorbidity ( $n=46$ ), followed by hypertension ( $n=11$ ) and malignancy ( $n=10$ ). Of those with malignancy, five had lung cancer. Other descriptive findings are summarized in Table 1.

The presence of PMF was about three times more common in bituminous miners than in lignite miners ( $p=0.025$ ; OR=3.012; 95% CI: 1.149–7.893). Mean exposure duration was  $20.5 \pm 6.6$  years in patients with complicated pneumoconiosis and  $19.4 \pm 6.5$  years in those with simple pneumoconiosis. Among PMF patients, 3 (8.1%) had  $\leq 10$  years of exposure, 10 (27%) had 11–20 years, and 24 (64.9%) had >20 years. Exposure duration was significantly linked to PMF development ( $p=0.031$ ; OR=1.077; 95% CI: 1.007–1.152). Age and smoking were not significantly associated.

Pulmonary tests showed obstruction in 38 (38%) patients and restriction in 35 (35%). Of those with obstruction, 12 (31.6%) had pure obstruction, and 26 (68.4%) had mixed impairment. No significant difference in obstruction or restriction prevalence between smokers and non-smokers ( $p=0.394$  and

**Table 1.** Descriptive Findings.

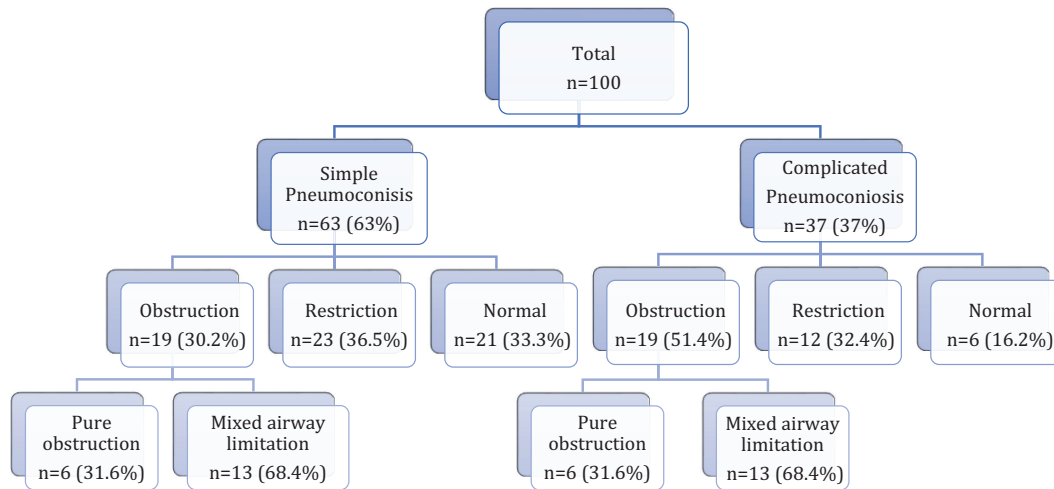
		n	%
<b>Age</b> (mean ± SD)		61.66±11.66	
<b>Smoking (Pack-Years)</b> (mean ± SD)		26.72±17.76	
<b>Smoking Status</b>	Non-Smoker	23	23
	Current or Former Smoker	77	77
<b>Exposure Duration (Years)</b> (mean ± SD)		20.50±6.63	
<b>Coal Type</b>	Bituminous	67	67
	Lignite	33	33
<b>Predominant Small Opacity</b>	p	20	20
	q	39	39
	r	16	16
	s	12	12
	t	13	13
	u	0	0
<b>Predominant Opacity Shape</b>	Irregular	25	25
	Rounded	75	75
<b>Small Opacity Profusion</b>	Category 1	31	31
	Category 2	60	60
	Category 3	9	9
<b>Large Opacities</b>	Absent	63	63
	Present	37	37
<b>Size of Large Opacities</b>	A	22	59
	B	12	32
	C	3	8.1
<b>FEV<sub>1</sub></b> (mean ± SD)		72.47±20.13	
<b>FVC</b> (mean ± SD)		75.11±20.01	
<b>FEV<sub>1</sub>/FVC</b> (mean ± SD)		73.80±12.42	
<b>Comorbidity</b>	Absent	33	33
	Present	67	67
<b>Respiratory Symptoms</b>	Absent	39	39
	Present	61	61

FEV<sub>1</sub>: Forced Expiratory Volume in 1 second, FVC: Forced Vital Capacity.

p=0.980). Obstruction was more common in complicated pneumoconiosis (p=0.035), but restriction prevalence was similar (p=0.680). The distribution of respiratory abnormalities is shown in Figure 1.

When factors associated with pulmonary function were analyzed, FEV<sub>1</sub>, FVC, and FEV<sub>1</sub>/FVC values

were significantly lower in bituminous coal miners compared with lignite miners (Table 2). No significant differences in pulmonary function parameters were observed between patients with and without a history of smoking (Table 2). Pulmonary function parameters and other related factors are presented in Table 2.



**Figure 1.** Distribution of respiratory dysfunction in simple and complicated pneumoconiosis.

When HRCT findings of patients diagnosed with CWP were evaluated, the most common findings were nodules, lymph node enlargement, and interlobular septal thickening. All HRCT findings observed in the cases are presented in Table 3.

Among patients with nodules, 66 had a peribronchovascular distribution, 8 had a centrilobular distribution, and 24 had a nonspecific distribution. Among patients with emphysema, 37 had paraseptal, 7 had bullous, 10 had centrilobular, and 3 had panacinar emphysema. The prevalence of emphysema was 47.8% in never-smokers and 59.7% in smokers; however, the difference between the groups was not statistically significant ( $p=0.311$ ).

ILA were identified in 63 (63%) cases. Of these, 42 patients (66.7%) also exhibited features consistent with ILD. Patients with ILD were significantly older than those without ILD ( $p=0.034$ ), whereas there was no statistically significant difference in exposure duration between the two groups ( $p=0.341$ ). Figure 2 shows HRCT images of a patient with ILD.

Univariate logistic regression analyses revealed that the risk of ILD was approximately 3.2-fold higher in workers employed in bituminous coal mines, 5.2-fold higher in those with more than 10 years of exposure, and 4.8-fold higher in those

with small opacities  $>3$  mm in diameter (Table 4). Factors associated with ILD and the results of the univariate logistic regression analysis are presented in Table 4. In the multivariate logistic regression analysis including exposure duration, coal type, and small opacity size, only the presence of small opacities  $>3$  mm remained independently associated with ILD. Patients with predominant small opacities  $>3$  mm had a 4.66-fold increased risk of ILD ( $p=0.026$ ; OR=4.661; 95%CI: 1.202–18.078).

The presence of ILA or ILD was significantly associated with a higher prevalence of respiratory symptoms (ILA:  $p=0.001$ ; OR=6.943; 95%CI: 2.099–22.964; ILD:  $p=0.001$ ; OR=34.286; 95%CI: 4.276–274.930). While no significant differences in pulmonary function parameters were observed between patients with and without ILA, those with ILD had significantly lower FEV<sub>1</sub> and FVC values (both  $p<0.001$ ).

#### 4. DISCUSSION

Coal dust exposure, due to its complex composition, is a major cause of respiratory disease. All respiratory disorders potentially associated with coal dust are collectively termed Coal Mine Dust Lung Disease (CMDLD) [5]. In CWP, a principal

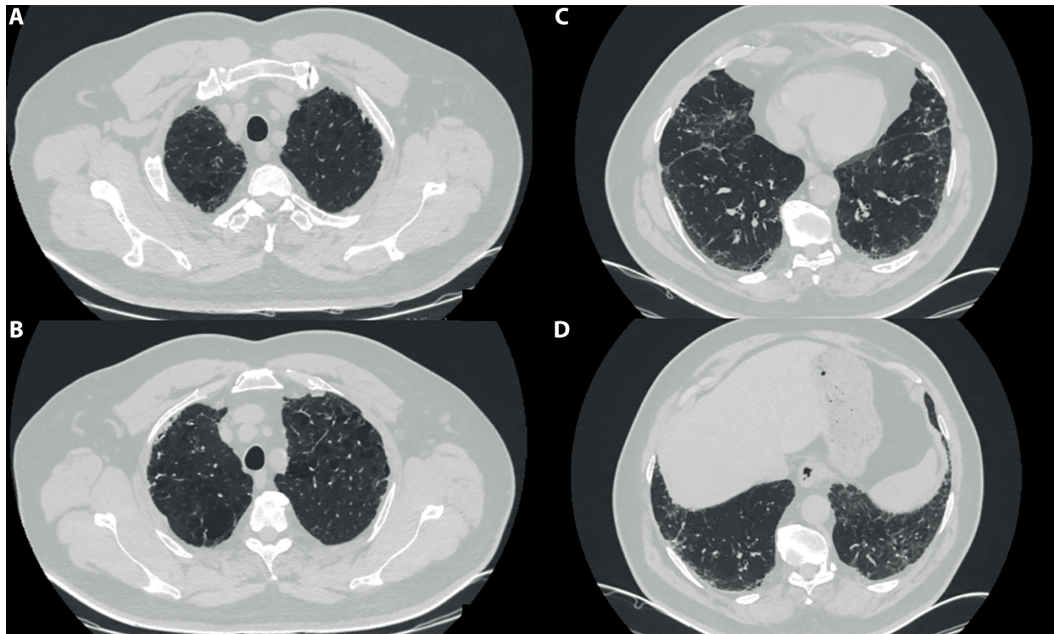
**Table 2.** Factors associated with pulmonary function parameters.

		FEV <sub>1</sub>	p*	FVC	p*	FEV <sub>1</sub> /FVC	p*
		Median (IQR)		Median (IQR)		Median (IQR)	
<b>Coal Type</b>	Bituminous	73 (55-83)	0.020	73 (63-86)	0.049	74 (61-79)	0.043
	Lignite	82 (68-94)		81 (72-93)		79 (70-85)	
<b>Smoking Status</b>	Never-smoker	77 (62-87)	0.734	78 (57-86)	0.756	77 (69-87)	0.231
	Current/ ex smoker	73 (59-87)		74 (64-90)		74 (67-80)	
<b>Size of Small Opacity</b>	<1.5 mm	79.5 (69.5-93.5)	0.001	79.5 (72.5-87.5)	0.024	79 (71.5-84.5)	0.027
	1.5-3 mm	75 (58-87.5)		77.5 (62.5-92)		74 (67-81)	
	>3 mm	58 (43.5-69.5)		64 (56.5-71.5)		69 (59-76)	
<b>Predominant Small Opacities</b>	Irregular	73 (55-83)	0.360	74 (61-80)	0.093	78 (64-84)	0.561
	Rounded	75 (60-88)		78 (65-90)		74 (67-81)	
<b>Small Opacity Profusion</b>	Category 1	80 (64-91)	0.391	77 (66-90)	0.842	79 (70-87)	0.008
	Category 2	75 (58.5-86.5)		74.5 (62-87.5)		74.5 (69-80)	
	Category 3	72 (58-72)		75 (67-100)		64 (60-69)	
<b>Large Opacities</b>	Absent	78 (66-94)	0.001	78 (67-94)	0.064	78 (70-84)	0.016
	Present	66 (53-79)		70 (59-84)		70 (60-77)	
<b>Size of Large Opacities</b>	A	71.5 (58-80)	0.019	76.5 (63-87)	0.026	73.5 (64-80)	0.429
	B	59 (51.5-73.5)		69 (60.5-79.5)		70.5 (59-76)	
	C	36 (28-40)		44 (31-50)		61 (60-69)	
<b>Pulmonary Disease</b>	Absent	83.5 (73-93)	<0.001	80.5 (74-93)	0.001	80 (74-86)	<0.001
	Present	62.5 (47-75)		66.5 (54-84)		68.5 (59-76)	
<b>Respiratory Symptoms</b>	Absent	87 (80-94)	<0.001	86 (74-97)	0.002	80 (74-84)	0.050
	Present	71 (55-79)		72 (57-84)		73 (64-79)	

FEV<sub>1</sub>: Forced Expiratory Volume in 1 second, FVC: Forced Vital Capacity \*Mann-Whitney U test.

**Table 3.** HRCT findings in patients with coal workers' pneumoconiosis.

HRCT findings	n (%)	HRCT Findings	n (%)
Nodule	98 (98)	Consolidation	64 (64)
Lymph Node Enlargement	98 (98)	Emphysema	57 (57)
Interlobar Septal Thickening	98 (98)	Distortion	49 (49)
Pleural Retraction	96 (96)	Mosaic Attenuation	40 (40)
Pleural Thickening	95 (95)	Mass Lesion	37 (37)
Reticulation	87 (87)	Lymph Node Calcification	35 (35)
Atelectasis	77 (77)	Cyst	20 (20)
Ground-Glass Opacities	73 (73)	Subpleural Lines	11 (11)
Interlobular Septal Thickening	72 (72)	Air Bronchogram	8 (8)
Bronchiectasis	69 (69)	Honeycombing	3 (3)



**Figure 2.** HRCT in a 70-year-old male who worked in a bituminous coal mine for 20 years and had a 20 pack-year smoking history (ex-smoker), showing predominant upper-lobe emphysematous changes and peripheral/subpleural reticular and reticulonodular interstitial opacities in the lower lobes, with interlobular septal and peribronchial thickening, mild bronchiectasis, focal honeycombing, and associated ground-glass opacities and millimetric nodules.

**Table 4.** Factors associated with interstitial lung disease (ILD) in patients with coal workers' pneumoconiosis: univariate logistic regression analysis.

		ILD		P	OR (95%CI)
		Absent n=58	Present n=42		
		n (%)	n (%)		
Age (mean ± SD)		59.2±13.0	65.1±8.5	<b>0.014</b>	<b>1.049 (1.010-1.090)</b>
Coal Type	Lignite	25 (43.1)	8 (19.0)	<b>0.014</b>	1 (Ref)
	Bituminous	33 (56.9)	34 (81.0)		<b>3.220 (1.271-8.153)</b>
Exposure Duration	<10 years	12 (20.7)	2(4.8)	<b>0.037</b>	1 (Ref)
	≥10 years	46 (79.3)	40 (95.2)		<b>5.217 (1.101-24.724)</b>
Smoking	Never-smoker	17 (29.3)	6 (14.3)	0.084	1 (Ref)
	Current/former Smoker	41 (70.7)	36 (85.7)		2.488 (0.886-6.988)
Size of Small Opacity	<1,5 mm	22 (37.9)	10 (23.8)	-	1 (Ref)
	1,5-3 mm	31 (53.4)	21 (50)	0.401	1.490 (0.588-3.779)
	>3 mm	5 (8.6)	11 (26.2)	<b>0.017</b>	<b>4.840 (1.326-17.666)</b>
Predominant Small Opacities	Rounded	46 (79.3)	29 (69.0)	0.245	1 (Ref)
	Irregular	12 (20.7)	13 (31.0)		1.718 (0.690-4.277)

Table 4 (Continued)

		ILD			
		Absent n=58	Present n=42		
		n (%)	n (%)	P	OR (95%CI)
<b>Small Opacity Profusion</b>	<b>Category 1</b>	19 (32.8)	12 (28.6)	-	1 (Ref)
	<b>Category 2</b>	35 (60.3)	25 (59.5)	0.786	1.131 (0.466-2.744)
	<b>Category 3</b>	4 (6.9)	5 (11.9)	0.372	1.979 (0.441-8.873)
<b>PMF</b>	<b>Absent</b>	41 (70.7)	22 (52.4)	0.063	1 (Ref)
	<b>Present</b>	17 (29.3)	20 (47.6)		2.193 (0.958-5.020)

OR: Odds Ratio; CI: Confidence Interval; ILD: Interstitial lung disease; PMF: progressive massive fibrosis.

component of CMDLD, assessing coal type, co-existing pulmonary disease, and HRCT findings is key to clarifying its clinical, functional, and radiological features. In the present study, patients working in bituminous coal mines had more severe radiological and functional impairment than those in lignite mines. HRCT showed that most patients had concomitant ILA and ILD. ILD was significantly associated with age, exposure duration, coal type, and small opacity size. Pulmonary function impairment occurred independently of smoking status and appeared with obstructive, restrictive, or mixed patterns.

CWP is an occupational lung disease in which radiological findings are usually dominated by small nodular opacities resembling silicosis. However, several studies have reported that coal dust exposure may also be associated with bilateral reticular abnormalities and, occasionally, honeycombing, a pattern defined as DDF and frequently seen in these patients [6]. Although DDF is a well-established pathological entity, its radiological features can overlap with those of other interstitial lung diseases. Bilateral reticular abnormalities, honeycombing, and traction bronchiectasis may be present and can mimic the usual interstitial pneumonia (UIP) pattern and idiopathic pulmonary fibrosis (IPF) [5]. Prior work has also shown that other interstitial lung diseases, including DIP, chronic interstitial pneumonia, and CPFA, may be associated with coal dust exposure [7-9]. Earlier series reported diffuse pulmonary fibrosis in 10-40% of coal miners [15-18], generally confirmed histopathologically and including all miners with coal dust exposure.

In the current study, radiological assessment revealed ILA in 63% and ILD in 42% of cases, rates higher than those previously reported. This likely reflects the inclusion of only patients already diagnosed with CWP, who by definition have had sufficiently intense and prolonged exposure to develop pneumoconiosis and, consequently, pulmonary fibrosis at higher-than-expected frequencies. The observation that ILD was approximately 5.2-fold more frequent in patients with  $\geq 10$  years of exposure supports this hypothesis. However, the retrospective design, lack of key physiological parameters (such as DLCO), and absence of histopathological data may have contributed to overestimation of ILD.

Recent studies have demonstrated a strong association between cumulative coal dust exposure and radiological ILD patterns [19], and an association between radiological ILD and mortality [19]. Another study of 45 coal miners with interstitial fibrosis showed that mean survival was significantly longer than in patients with non-occupational interstitial fibrosis [20]. Overall, interstitial fibrosis is not rare among coal miners and may follow a distinct clinical course compared with non-occupational forms. Thus, recognizing ILA and ILD in this population is critical: failure to obtain a detailed occupational history can lead to misdiagnosis and inappropriate management. Comprehensive occupational exposure assessment in patients with ILA or ILD may prevent unnecessary interventions and facilitate timely exposure cessation, potentially reducing disease progression.

In this study, 73% of patients had pulmonary function test abnormalities, classified as obstructive,

restrictive, or mixed patterns. Obstruction or restriction was not significantly related to smoking status, but obstruction was significantly more frequent among patients with PMF. Cumulative dust exposure leads to reductions in FEV<sub>1</sub>, FVC, and the FEV<sub>1</sub>/FVC ratio and to emphysema, even in never-smokers. Consequently, differences in pulmonary function between smokers and non-smokers tend to diminish or disappear. In PMF, pulmonary function impairment is more pronounced, as exposure is usually longer and more intense. Paracatricial emphysema in PMF further accentuates obstructive changes. Previous studies have consistently shown a dose-response relationship between respirable coal dust exposure and pulmonary function parameters [4, 21, 22]. In a study of 7,139 coal miners, cumulative coal dust exposure was inversely correlated with FEV<sub>1</sub>, FVC, and FEV<sub>1</sub>/FVC [23]. Similarly, in former miners, abnormal spirometric findings were found in 56.6% of workers, regardless of pneumoconiosis status, and both the frequency and severity of abnormalities increased with disease category [24].

Multiple studies indicate that coal dust exposure is an independent risk factor for obstructive lung disease, chronic bronchitis, and emphysema [23, 25-27]. In an autopsy study of 722 individuals, the emphysema severity index was sixfold higher in never-smoking coal miners than in never-smokers without mining exposure [28]. Smoking and coal dust exposure had similar effects in predicting emphysema severity, and emphysema was reported in one-third of never-smoking coal miners [19]. In the current study, emphysema was radiologically detected in 57% of all cases and in 47.8% of never-smokers. The absence of a significant difference in emphysema prevalence between smokers and non-smokers further supports coal dust exposure as an independent risk factor for emphysema.

Coal types differ markedly in rank, moisture content, mineral composition, and physicochemical properties [2, 29]. Owing to its higher carbon content, bituminous coal is considered more cytotoxic and pathogenic than lignite [30]. In the study by Reisner and Robock, workers exposed to dust of similar mass concentrations showed greater cytotoxicity

and higher pneumoconiosis prevalence in mines extracting higher-rank coal [31]. Other studies similarly reported that exposure to higher-rank coal is associated with increased CWP incidence [32, 33]. In a 37-year follow-up study by Graber et al., the exposure-response relationship between cumulative coal dust exposure and pneumoconiosis-related mortality varied by region, with the strongest associations where coal rank was highest [34].

Consistent with this, in the present study PMF was approximately threefold more frequent among CWP cases working in bituminous than in lignite mines, and FEV<sub>1</sub>, FVC, and FEV<sub>1</sub>/FVC values were significantly lower, independent of smoking status. ILD was also about 3.2-fold more common in bituminous coal miners. These differences in functional impairment and disease severity cannot be attributed solely to geological factors: exposure intensity, particle characteristics, production methods, and concomitant crystalline silica exposure are also important. Bituminous coal is predominantly extracted through underground mining, where gallery excavation and blasting generate large quantities of fine particulate matter and effective dilution is difficult. This results in prolonged exposure to high concentrations of respirable dust. In contrast, lignite is often produced in open-pit mines, where natural ventilation and lower mechanical fragmentation energy generally yield lower alveolar dust concentrations [35]. Thus, cumulative dust exposure tends to be higher in bituminous mines.

Workers exposed to higher-rank coal dust, especially in underground mines, should therefore be regarded as at increased risk not only for the development and severity of pneumoconiosis but also for pneumoconiosis-related morbidity and mortality. Occupational health and safety strategies and health surveillance programs should prioritize these high-risk groups.

#### 4.1. Strengths of the Study

This study was conducted at one of the leading national reference centers for pneumoconiosis, allowing evaluation of patients from multiple regions and different coal mine types. Interstitial findings

were assessed according to the 2025 ATS guideline, ensuring a current and objective evaluation.

#### 4.2. Limitations of the Study

This retrospective cross-sectional study included only CWP cases referred to a single tertiary center. Individuals without CWP who might have exhibited interstitial changes or pulmonary function impairment were not included, potentially introducing selection bias. The retrospective and single-center design limits generalizability and precludes causal conclusions. Several estimates had wide confidence intervals due to limited sample size and few events in some subgroups; thus, the precision of some effect estimates is limited and the findings should be interpreted cautiously.

HRCT images were assessed by a single radiologist, an important limitation, particularly for subtle interstitial abnormalities, and a source of possible diagnostic uncertainty and classification bias. The lack of key physiological parameters (such as DLCO) and histopathological data, again related to the retrospective design, may have led to overestimation of ILD prevalence. Dust concentration measurements were not available, so pneumoconiosis severity and pulmonary function impairment could not be directly linked to exposure levels. Because occupational histories were self-reported, recall bias cannot be excluded.

Despite using a composite clinicoradiological and physiological framework aligned with ATS recommendations, some overlap between ILA and early or mild ILD cannot be excluded. This is an inherent challenge of observational retrospective studies and highlights the need for longitudinal follow-up and multidimensional assessment.

#### 5. CONCLUSION

In this study of patients with CWP, abnormalities in pulmonary function were detected in 73% of cases, PMF in 37%, and ILD unrelated to other causes in 42%. The development of PMF and ILD was associated with exposure duration and coal type, with workers in bituminous coal mines at substantially higher risk of PMF, ILD, and pulmonary

function decline. To better protect workers and prevent harmful exposure, large-scale prospective studies exploring the relationship between coal rank and disease severity are needed.

Given the high prevalence of ILA and ILD in CWP, obtaining a detailed occupational history in all patients presenting with interstitial lung disease is essential. Such comprehensive assessment, together with timely exposure cessation when indicated, may help reduce the morbidity and mortality associated with these conditions.

**INSTITUTIONAL REVIEW BOARD STATEMENT:** The study was conducted according to the guidelines of the Declaration of Helsinki, and approved by the Ankara Atatürk Sanatorium Training and Research Hospital Clinical Research Ethics Committee (2012-KAEK-15/2749 and approved on 12 July 2023)

**DECLARATION OF INTEREST:** The authors declare no conflict of interest.

**AUTHOR CONTRIBUTION STATEMENT:** M.A.P. and G.S. contributed to the conceptualization and design of the study; M.A.P., G.S., A.K., R.E., H.E., and C.Ş. conducted the investigation; M.A.P. performed the formal analysis; M.A.P., G.S., A.K., R.E., and H.E. contributed to data curation; M.A.P. drafted the original manuscript, and G.S., A.K., R.E., H.E., and C.Ş. revised and edited the manuscript, and C.Ş. supervised the study. All authors have read and approved the final version of the manuscript.

#### REFERENCES

1. Balmes J. R. Occupational Lung Disease. In: Eds Ladou J, Harrison R. J, eds. *CURRENT Diagnosis & Treatment: Occupational & Environmental Medicine*, 6e. McGraw Hill; 2021:293-315.
2. Berkowitz, Norbert. *An introduction to coal technology*. Academic Press, New York, 1979.
3. Akira M, Suganuma N. Imaging diagnosis of pneumoconiosis with predominant nodular pattern: HRCT and pathologic findings. *Clinical Imaging* 2023;97: 28-33.
4. Cohen RA, Petsonk EL, Rose C, et al. Lung pathology in U.S. coal workers with rapidly progressive pneumoconiosis implicates silica and silicates. *Am J Respir Crit Care Med*. 2016;193:673-80.
5. Petsonk EL, Rose C, Cohen R. Coal mine dust lung disease: new lessons from an old exposure. *Am J Respir Crit Care Med*. 2013;187:1178-1185.

6. McConnochie K, Green PHY, Vallyathan V, et al. Interstitial fibrosis in coal workers – experience in Wales and West Virginia. *Ann Occup Hyg.* 1988;32:553-560.
7. Brichet A, Tonnel AB, Brambilla E, et al. Chronic interstitial pneumonia with honeycombing in coal workers. Sarcoidosis Vasc Diffuse Lung Dis. 2002;19(3):211-219.
8. Heppleston AG. The pathological recognition and pathogenesis of emphysema and fibrocystic disease of the lung with special reference to coal workers. *Ann NY Acad Sci.* 1972;200:347-369.
9. Jelic TM, Estalilla OC, Sawyer-Kaplan PR, et al. Coal Mine Dust Desquamative Chronic Interstitial Pneumonia: A Precursor of Dust-Related Diffuse Fibrosis and of Emphysema. *Int J Occup Environ Med.* 2017;8(3):153-165.
10. International Labour Office. Guidelines for the Use of the ILO International Classification of Radiographs of Pneumoconiosis, Revised edn. Occupational Safety and Health Series, Vol. 22. International Labour Organization– Geneva: ILO, 2022.
11. Global Initiative for Chronic Obstructive Lung Disease (GOLD). Global strategy for the prevention, diagnosis and management of COPD: 2025 report [Internet]. 2024 Available online: [https://goldcopd.org/wp-content/uploads/2024/12/Pocket-Guide-2025-v1.2-FINAL-covered-13Dec2024\\_WMV.pdf](https://goldcopd.org/wp-content/uploads/2024/12/Pocket-Guide-2025-v1.2-FINAL-covered-13Dec2024_WMV.pdf) (Last Accessed on 22 Jul 2025).
12. Pellegrino R, Viegi G, Brusasco V, et al. Interpretative strategies for lung function tests. *Eur Respir J.* 2005;26(5):948-968.
13. Kusaka Y, Hering KG, Parker JE. International Classification of HCRT for Occupational and Environmental Respiratory Diseases. Tokyo: Springer Verlag, 2005.
14. Podolanczuk AJ, Hunninghake GM, Wilson KC, et al. Approach to the Evaluation and Management of Interstitial Lung Abnormalities: An Official American Thoracic Society Clinical Statement. *Am J Respir Crit Care Med.* 2025;211(7):1132-1155.
15. Cockcroft A, Lyons JP, Andersson N, Saunders MJ. Prevalence and relation to underground exposure of radiological irregular opacities in South Wales coal workers with pneumoconiosis. *Br J Ind Med.* 1983;40(2):169-172.
16. Trapnell DH. Septal lines in pneumoconiosis. *Br J Radiol.* 1964;37:805-810.
17. Laney AS, Petsonk EL. Small pneumoconiotic opacities on U.S. coal worker surveillance chest radiographs are not predominantly in the upper lung zones. *Am J Ind Med.* 2012;55(9):793-798.
18. Cox CW, Rose CS, Lynch DA. State of the art: Imaging of occupational lung disease. *Radiology.* 2014;270:681.
19. Sangani RG, Ghio AJ, Deepak V, et al. Impact of coal mine dust exposure and cigarette smoking on lung disease in Appalachian coalminers. *Respir Res.* 2025;26(1):184.
20. McConnochie K, Green FHY, Vallyathan V, et al. Interstitial fibrosis in coal workers: experience in Wales and West Virginia. *Ann Occup Hyg.* 1988;32:553-560.
21. Centers for Disease Control and Prevention. Current intelligence bulletin 64: coal mine dust exposures and associated health outcomes - a review of information published since 1995. DHHS (NIOSH) Publication No. 2011-172; 2011 April. Available online: <http://www.cdc.gov/niosh/docs/2011-172/> (Last Accessed on 11 Aug 2025).
22. Seixas NS, Robins TG, Attfield MD, Moulton LH. Exposure-response relationships for coal mine dust and obstructive lung disease following enactment of the Federal Coal Mine Health and Safety Act of 1969. *Am J Ind Med.* 1992;21:715-734.
23. Attfield MD, Hodous TK. Pulmonary function of U.S. coal miners related to dust exposure estimates. *Am Rev Respir Dis.* 1992;145(3):605-609.
24. Go LHT, Almerg KS, Rose CS, et al. Prevalence and severity of abnormal lung function among US former coal miners with and without radiographic coal workers' pneumoconiosis. *Occup Environ Med.* 2022;79(8):527-532.
25. Nemery B, Veriter C, Brasseur L, Frans A. Impairment of ventilatory function and pulmonary gas exchange in nonsmoking coalminers. *Lancet.* 1987;2:1427-1430.
26. Marine WM, Gurr D, Jacobsen M. Clinically important respiratory effects of dust exposure and smoking in British coal miners. *Am Rev Respir Dis.* 1988;137:106-112.
27. Carta P, Aru G, Barbieri MT, et al. Dust exposure, respiratory symptoms, and longitudinal decline of lung function in young coal miners. *Occup Environ Med.* 1996;53:312-319.
28. Kuempel ED, Wheeler MW, Smith RJ, et al. Contributions of dust exposure and cigarette smoking to emphysema severity in coal miners in the United States. *Am J Respir Crit Care Med.* 2009;180(3):257-264.
29. Sari G. Coal Workers' Pneumoconiosis: A Comparative Analysis of Lignite and Hard Coal Mine Workers. *ESTUDAM Public Health Journal.* 2022;7(3):497-505.
30. Dalal NS, Newman J, Pack D, et al. Hydroxyl radical generation by coal mine dust: possible implication to coal workers' pneumoconiosis (CWP). *Free Radic Biol Med.* 1995;18(1):11-20.
31. Reisner MT, Robock K. Results of epidemiological mineralogical and cytotoxicological studies on the pathogenicity of coal-mine dusts. I In: Walton, W.H. (Ed.), *Inhaled Particles IV, Part 2.* Pergamon Press, Oxford 1975:703-716.
32. Dalal NS, Jafari B, Petersen M, Green FHY, Vallyathan V. Presence of stable coal radicals in autopsied coal miners' lungs and its possible correlation to coal workers' pneumoconiosis. *Arch Environ Health.* 1991;46(6):366-372.

33. Page SJ. Relationships between electrostatic charging characteristics, moisture content, and airborne dust generation for subbituminous and bituminous coals. *Aerosol Sci Technol.* 2000;32(4):249-267.
34. Graber JM, Stayner LT, Cohen RA, et al. Respiratory disease mortality among US coal miners; results after 37 years of follow-up. *Occup Environ Med.* 2014; 71(1):30-39.
35. Hartman HL, Mutmansky JM. *Introductory Mining Engineering*; Wiley: Hoboken, NJ, USA, 2002.

