

Causes of Acute Respiratory Failure in Hospitalized Patients With Interstitial Lung Disease: A Prospective Cohort Study

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Abstract

Background and aim:

Acute respiratory failure (ARF) is a major cause of morbidity and mortality in patients with interstitial lung disease (ILD). Data regarding the epidemiology and causes of ARF in this population remain limited. This study aimed to identify the causes of ARF and the associated clinical characteristics in hospitalized patients with ILD.

Methods

This prospective study included patients with ILD hospitalized between December 2020 and December 2021. ARF was defined as a PaO₂/FiO₂ ratio $\leq$ 300 mmHg and/or the requirement for at least 10 L/min supplemental oxygen and/or the presence of increased respiratory effort (tachypnea and/or use of accessory respiratory muscles) at the time of admission. Demographic characteristics, underlying ILD subtypes, and causes of ARF were recorded and analyzed.

Results

A total of 109 patients with ILD were included, 45 had ARF at admission. The most common cause of ARF was pulmonary infection (48.9%), followed by acute exacerbation of the underlying ILD (24.4%), pulmonary thromboembolism (13.3%), and congestive heart failure (8.9%). Idiopathic pulmonary fibrosis was the most frequent underlying ILD subtype among patients with ARF (37.8%), followed by connective tissue disease–associated ILD (22.2%), hypersensitivity pneumonitis (15.5%), and drug-induced ILD (13.3%).

Conclusions

Pulmonary infection and acute exacerbation are the leading causes of ARF in patients with ILD. Identification of ARF etiologies may facilitate timely diagnosis and guide appropriate management strategies in this high-risk population.

Introduction

Interstitial lung diseases (ILDs) are a group of disorders that diffusely affect the lungs and cause varying degrees of inflammation, fibrosis, and structural distortion in the lung parenchyma, with an acute or chronic clinical course **(1)**.

During the course of interstitial lung disease (ILD), episodes of acute respiratory failure (ARF) may develop. These episodes can be precipitated by respiratory tract infections, acute heart failure, pulmonary embolism, or other identifiable causes. When no precipitating factor can be identified, the event is defined as an acute exacerbation of ILD. Acute exacerbations are thought to represent an acceleration of the underlying fibroproliferative process or to be associated with occult or known external triggers, such as microaspiration or mechanical stress related to lung biopsy procedures.

Idiopathic pulmonary fibrosis (IPF) is the interstitial lung disease with the poorest prognosis, with a mean survival of approximately 2 to 3 years from the time of diagnosis. A definition of acute exacerbation of IPF is available in the literature and is characterized by an acute (less than one month) and clinically significant deterioration in respiratory status in a patient with previously diagnosed IPF, the presence of new, diffuse alveolar infiltrates on high-resolution computed tomography (HRCT), and the exclusion of alternative etiologies (such as heart failure, pulmonary embolism, and less commonly pneumothorax, drug toxicity, and diffuse alveolar hemorrhage) **(2)**. Such definitions are considered to contribute to improved patient management.

Although acute exacerbations have been described in non–idiopathic pulmonary fibrosis (non-IPF) interstitial lung diseases—particularly idiopathic fibrotic nonspecific interstitial pneumonia, connective tissue disease–associated interstitial lung diseases, and chronic hypersensitivity pneumonitis—robust data remain insufficient to extend existing diagnostic criteria to these entities **(3)**. Episodes of ILD-associated acute respiratory failure (ILD-ARF) are associated with substantially increased morbidity and mortality **(4)**. However, epidemiological data characterizing patients who develop these episodes are limited. Therefore, the aim of the present study was to evaluate the clinical and demographic characteristics, comorbidities, laboratory parameters, and radiological features of patients experiencing ILD-ARF episodes, in order to better define factors associated with morbidity and mortality.

Materials and Methods

Study Design and Population

This prospective, single-center study was conducted in patients with a diagnosis or prediagnosis of interstitial lung disease (ILD) who were hospitalized between December 2020 and December 2021. After obtaining approval from the local institutional ethics committee (approval date: 14 December 2020; file number: 2020-34), the study was initiated.

A total of 109 patients with diagnosed or suspected ILD were included. Patients were evaluated based on their clinical status at the time of admission and were classified into two groups: those hospitalized with acute respiratory failure (ARF) and those hospitalized for non-ARF indications, including ILD investigation and follow-up.

Definition of Acute Respiratory Failure

Acute respiratory failure was defined as a $\text{PaO}_2/\text{FiO}_2$ ratio ≤ 300 mmHg and/or the requirement for supplemental oxygen at a flow rate of ≥ 10 L/min, and/or the presence of increased respiratory effort (tachypnea and/or use of accessory respiratory muscles) at the time of admission.

Definition of Acute Exacerbation

Acute exacerbation was defined in patients with idiopathic pulmonary fibrosis as an acute worsening of dyspnea within the preceding 30 days, accompanied by newly developed bilateral ground-glass opacities and/or consolidation superimposed on a background of usual interstitial pneumonia on high-resolution computed tomography. Alternative causes, including pneumothorax, pleural effusion, pulmonary embolism, isolated heart failure, and fluid overload, were excluded (5).

Data Collection

Demographic characteristics, underlying ILD subtypes, comorbidities, and clinical features were recorded prospectively. In patients admitted with ARF, the etiology of respiratory failure was determined based on clinical evaluation, laboratory findings, and radiological imaging and classified as pulmonary infection, acute exacerbation of the underlying ILD, pulmonary thromboembolism, congestive heart failure, fibrotic progression, or other causes.

Statistical Analysis

Statistical analyses were performed using SPSS (Statistical Package for the Social Sciences), version 23.0 (IBM Corp., Armonk, NY, USA). Data were evaluated at a 95% confidence interval, and a p value < 0.05 was considered statistically significant. Descriptive statistics, including mean, standard deviation, median, frequency, percentage, minimum, and maximum values, were used to summarize the data.

The normality of continuous variables was assessed using the Shapiro–Wilk test. Comparisons between two groups were performed using the independent samples t-test or the Mann–Whitney U test for continuous variables and the chi-square test or Fisher’s exact test for categorical variables, as appropriate. For comparisons among more than two groups, the Kruskal–Wallis test was used.

Results

A total of 109 patients were included in the study, of whom 45% were female and 55% were male. The mean age of the patients was 65.80 ± 10.52 years. The mean body mass index (BMI) was 28.93 ± 5.31 kg/m². The demographic characteristics and comorbidities of the patients are presented in Table 1.

Table 1
Demographic characteristics of the patients

	N	%
Gender		
Female	49	45
Male	60	55
BMI		
Normal weight (18.50-24.99 kg/m ²)	25	22.9
Overweight (25.00-29.99 kg/m ²)	43	39.4
Obese (≥ 30.00 kg/m ²)	41	37.6
Tobacco exposure		
Never smoker	59	54.1
Current smoker	21	19.3
Former smoker	29	26.6
Total	109	100
Presence of comorbidities		
Any comorbidity	84	77.1
Hypertension	50	45.9
Diabetes mellitus	27	24.8
Coronary artery disease	19	17.4
Cancer	12	11
Concomitant chronic lung disease		
Any chronic lung disease	34	31.2
Chronic obstructive pulmonary disease (COPD)	13	11.9
Asthma	9	8.3
History of tuberculosis	5	4.6
History of pulmonary thromboembolism / venous thromboembolism (PTE/VTE)	4	3.7
Obstructive sleep apnea syndrome (OSAS)	2	1.8
Lung cancer	2	1.8
Other	2	1.8

Cough was present in 48.6% of the patients, sputum production in 15.6%, fever in 6.4%, and dyspnea in 82.6%. On auscultation, crackles were detected in 67.9% of the patients, wheezing in 7.3%, and pleural friction rub in 0.9%. The proportion of patients who had received the annual influenza vaccination was 20.2%, while 23.9% had received the pneumococcal vaccine. The mortality rate was 3.7%, with sudden cardiac arrest and sepsis identified as the causes of death (Table 2) .

Table 2
Clinical symptoms, medication use, vaccination
status, and mortality of the patients

Respiratory symptoms	N	%
Cough	53	48.6
Sputum production	17	15.6
Fever	7	6.4
Dyspnea	90	82.6
Other	17	15.5
Medications used		
Pirfenidone	5	4.6
Nintedanib	2	1.8
Corticosteroids	19	17.4
Azathioprine	3	2.8
Anti-TNF agents	1	0.9
Acetylsalicylic acid	22	20.2
Statins	22	20.2
Chemotherapeutic agents	5	4.6
Vaccination status		
Annual influenza vaccination	22	20.2
Pneumococcal vaccination	26	23.9
Mortality		
Death	4	3.7

Baseline laboratory parameters of all study groups are summarized in Table 3.

Table 3
Baseline laboratory findings of the patients

Parameter	n	Value*
Leukocyte count (/mm ³)	109	8.43 ± 3.41
Eosinophils (%)	109	2.38 ± 1.83
Monocytes (%)	109	8.05 ± 6.51
Platelet count (/mm ³)	109	236128 ± 37428
Lymphocytes (%)	109	24.04 ± 10.49
C-reactive protein (CRP), mg/L	107	9.0 (3.3–21.3)
Procalcitonin (PCT), ng/ML	62	0.05 (0.03–0.16)
D-dimer, mg/L	84	0.73 (0.49–1.89)
Pro-B-type natriuretic peptide (proBNP), pg/mL	22	293.5 (72.8–987.3)
Erythrocyte sedimentation rate (ESR), mm/h	95	23.0 (11.0–41.0)
pH	79	7.37 ± 0.05
pCO ₂	80	45.00 ± 8.03
pO ₂	80	37.48 ± 17.94
SaO ₂	80	58.74 ± 24.19

***Data are presented as mean ± standard deviation for normally distributed variables and as median (interquartile range) for non-normally distributed variables.**

Microbiological growth was detected in 2.8% of blood cultures, 13.8% of sputum cultures, and 19.3% of bronchoalveolar lavage (BAL) cultures. Blood cultures were not obtained in 86.2% of patients, while sputum cultures and BAL samples were not collected in 78.9% and 54.1% of patients, respectively. Influenza PCR testing was performed in one patient and was negative. CMV PCR and *Pneumocystis jirovecii* (PCP) PCR tests were negative in all tested patients. Urinary *Legionella* antigen testing was performed in three patients and was negative in all cases. SARS-CoV-2 PCR testing was negative in 92.7% of patients, while 6.4% were not tested. A positive SARS-CoV-2 PCR result within the previous six months was documented in 25.7% of the patients.

On chest radiography, cardiomegaly was observed in 6.4% of patients, bilateral pleural effusion in 2.8%, Kerley B lines in 2.8%, consolidation in 11.1%, and increased bilateral reticulation in 72.5%.

Thoracic CT angiography was performed in 38.5% of the patients, and pulmonary thromboembolism was detected in 14.3% of those examined. According to acute-phase thoracic CT findings, bilateral ground-glass opacities were observed in 27.5% of patients, consolidation in 11.1%, and atelectasis in 1.9%.

Doppler ultrasonography was performed in 8.3% of the patients, and deep vein thrombosis was detected in 11.1% of those examined. On echocardiographic evaluation, right chamber dilatation was detected in 1% of 94 patients, while valvular heart disease was present in 69.1%. The mean ejection fraction was 58.15 ± 11.34%, and the mean systolic pulmonary artery pressure was 28.75 ± 10.30 mmHg.

In the evaluation of surgical history, 1.8% of the patients had undergone a non-thoracic surgical procedure within the previous year, and no postoperative exacerbation of the primary disease or development of acute respiratory failure was observed.

Diagnostic procedures were performed in 45% of patients once respiratory stability was achieved during hospitalization. Bronchoalveolar lavage was performed in 39.4% of patients, bronchoscopic transbronchial lung biopsy in 4.6%, transthoracic lung biopsy in 0.9%, and endobronchial ultrasound (EBUS) in 3.7% during hospitalization. After completion of the diagnostic workup, the final diagnosis was established by flexible bronchoscopy in 53.2% of patients, by clinical–radiological evaluation in 33.9%, by surgical procedures in 3.7%, by EBUS in 6.4%, and by transthoracic lung biopsy in 1.8%.

Overall, 53.2% of patients were hospitalized for interstitial lung disease (ILD) investigation, 11.0% for monitoring steroid-related adverse effects, 9.2% due to fibrotic progression, and 1.8% for treatment modification related to adverse drug effects. Acute respiratory failure was present in 45 patients, accounting for 41.3% of the study population. Among these patients, idiopathic pulmonary fibrosis was identified in 37.8%, connective tissue disease–associated ILD in 22.2%, hypersensitivity pneumonitis in 15.5%, and drug-related ILD in 13.3%.

Baseline demographic and clinical characteristics, including age, sex, smoking history, comorbidities, and the presence of chronic lung disease, were comparable among the primary disease exacerbation, fibrotic progression, and ILD investigation groups (all $p > 0.05$).

Primary disease exacerbation was identified in 12% of normal-weight patients, 14% of overweight patients, and 4.9% of obese patients. However, no statistically significant association was found between the presence of primary disease exacerbation and BMI categories ($p = 0.361$).

Acute respiratory failure developed due to pulmonary infection in 48.9% of patients, exacerbation of the primary disease in 24.4%, pulmonary thromboembolism in 13.3%, and congestive heart failure in 8.9% of cases (Table 4).

Table 4
Etiologies of acute respiratory failure

Pulmonary infection	N	%*
	22	48.9
Exacerbation of the primary disease	11	24.4
Pulmonary thromboembolism	6	13.3
Congestive heart failure	4	8.9
Fibrotic progression	2	4.4
Other	8	17.8

*Percentages exceed 100% because more than one etiology may have contributed to acute respiratory failure in some patients.

The treatments administered, respiratory support modalities, and clinical follow-up outcomes are summarized in Table 5.

Table 5
Treatments, respiratory support, and clinical follow-up

N	%
Treatment	
Systemic corticosteroid therapy	67 61.5
Anticoagulant prophylaxis	55 50.5
Antibiotic therapy	22 20.2
Diuretic therapy	8 7.3
Positive inotropic agents	5 4.6
Type of respiratory support	
Nasal cannula	27 24.8
Face mask	3 2.8
Reservoir mask	4 3.7
High-flow nasal cannula	2 1.8
Non-invasive ventilation (NIV)	3 2.8
Invasive mechanical ventilation (IMV)	1 0.9
Clinical follow-up	
Intensive care unit admission	6 5.6

When the distributions of laboratory parameters across five patient groups were compared, statistically significant differences were observed in leukocyte, eosinophil, monocyte, D-dimer, and CRP levels (Kruskal-Wallis test, $p < 0.05$). No significant difference was detected for total IgE levels among the groups ($p = 0.061$) (Table 6). Pairwise comparisons for the parameters found to be significant revealed the specific intergroup differences. Leukocyte levels were significantly higher in the PTE ($p = 0.0365$) and fibrotic progression groups ($p = 0.0312$) compared with the primary disease exacerbation group. Eosinophil levels were lower in the fibrotic progression group compared to the ILD investigation group ($p = 0.0265$). Monocyte levels were markedly lower in the primary disease exacerbation and fibrotic progression groups than in the ILD investigation, pneumonia, and PTE groups ($p < 0.05$). D-dimer levels were significantly higher in the pneumonia and PTE groups compared to the ILD

investigation and fibrotic progression groups (all $p < 0.05$). CRP levels were significantly higher in the primary disease exacerbation, fibrotic progression, pneumonia, and PTE groups compared to the ILD investigation group (all $p < 0.05$). The highest CRP levels were observed in the pneumonia group (median: 73.85), followed by the PTE group (median: 56.15), as shown in Table 6.

Table 6
Comparative Profiling of Laboratory Findings in Distinct Pulmonary Conditions

Parameter median (IQR)	PrimaryDiseaseExacerbation	FibroticProgression	ILD Investigation	Pneumonia	PulmonaryThromboembolism (PTE)	p-value
Leukocyte (cells/ μ l)	6250.0 (4610.0–8405.0)	7005.0 (5655.0–8957.5)	7730.0 (6110.0–9072.5)	10280.0 (8465.0–14225.0)	10285.0 (9165.0–12957.5)	0.005
Eosinophil (cells/ μ l)	110.0 (29.5–185.0)	85.0 (31.5–182.5)	170.0 (92.5–277.5)	70.0 (10.0–187.5)	100.0 (44.5–175.0)	0.016
Monocyte (cells/ μ l)	410.0 (370.0–510.0)	450.0 (340.0–570.0)	645.0 (510.0–737.5)	665.0 (460.0–822.5)	900.0 (702.5–1060.0)	0.001
Total IGE (kU/L)	15.4 (15.0–24.35)	32.75 (26.25–38.12)	79.5 (26.8–222.0)	133.05 (26.0–238.82)	87.95 (26.0–401.48)	0.061
D-dimer (μ g/mL)	1.1 (0.6–2.2)	0.5 (0.43–0.8)	0.65 (0.38–1.14)	1.87 (0.64–3.15)	2.25 (1.4–8.98)	0.007
C-reactive protein mg/L (CRP)	10.4 (5.45–38.4)	12.5 (7.25–20.2)	4.95 (2.1–9.15)	73.85 (21.72–140.0)	56.15 (21.65–78.12)	0.001
Note: Data are presented as median (25th–75th percentile). Comparisons between groups were performed using the Kruskal-Wallis test. A p-value of < 0.05 was considered statistically significant.						

Discussion

Acute respiratory failure (ARF) is defined as a rapid deterioration in respiratory function over a period of several days (6). Although acute exacerbations may occur in non-IPF interstitial lung diseases (ILDs), current evidence is insufficient to extend the same diagnostic criteria to all ILD subtypes (3). In this study, the concept of acute exacerbation was adapted to other ILDs and classified as “primary disease exacerbation.”

While ILD typically progresses insidiously toward chronic respiratory failure, in some cases it may deteriorate acutely. A prospective study in IPF patients demonstrated that the development of chronic respiratory failure and the need for high-flow oxygen were associated with increased mortality, independent of pulmonary function test results (7). Among ILDs with a worse prognosis, such as IPF, earlier and more frequent onset of chronic respiratory failure is observed (8,9). In our study, three out of four patients who died during the one-year follow-up had already been diagnosed with chronic respiratory failure and were receiving oxygen therapy prior to hospital admission.

Several studies have attempted to identify unified risk factors for AE-ILD, although results have often been inconsistent (10). In IPF, factors such as reduced FVC, DLCO, lower six-minute walk distance, presence of pulmonary hypertension, baseline hypoxemia, and progressive decline in FVC have been associated with increased risk of exacerbation (2). For non-IPF ILDs, parameters including FVC, BMI, total lung capacity, DLCO, and partial oxygen pressure have been reported as independent predictors of AE (11–13). While Awano et al. found higher mortality in underweight and lower mortality in obese patients with AE-IPF (14), Qiu et al. reported no significant association with BMI(15). In our study, although no statistically significant relationship was found between BMI categories and the presence of primary disease exacerbation ($p = 0.361$), exacerbation rates differed across groups: 12% in normal-weight, 14% in overweight, and 4.9% in obese patients.

Pulmonary infections are a common cause of hospitalization in patients with IPF. In our study, 48.9% of patients admitted with respiratory failure had pulmonary infections. Similarly, previous studies have highlighted significant infection-related morbidity and mortality in this population (16,17).

Bronchoalveolar lavage (BAL) cultures yielded microbial growth in 19.3% of our patients. Prior research has reported various gram-negative bacteria and fungal organisms in BAL samples of IPF patients, with some suggesting an association between microbial burden and disease progression (18).

Regarding demographic characteristics, older age and male sex have consistently been associated with an increased risk of developing ILD (19–23). In our cohort, the majority of patients were male with a mean age of approximately 66 years, consistent with previous epidemiological observations.

Patients with ILD are at increased risk of venous thromboembolic disease due to immobility caused by dyspnea and musculoskeletal pain. A prothrombotic milieu has been suggested in both IPF and non-IPF ILDs, with studies showing a higher prevalence of this condition in IPF patients compared to healthy controls (24). The elevated thromboembolic risk is thought to be secondary to underlying inflammation, mediated by pro-inflammatory cytokines that promote endothelial dysfunction and coagulation cascade activation. Sarcoidosis has also been associated with an increased risk of venous thromboembolism in population-based studies (25). In our study, acute pulmonary embolism was detected in 13.3% of patients presenting with respiratory failure, while a prior history of VTE or PE was present in 3.7%. Sode et al. hypothesized that procoagulant states in individuals with undiagnosed microemboli may contribute to the development of progressive interstitial fibrosis and ILD over time (26).

It has been reported that activated intravascular coagulation may play a role in the pathophysiology of acute exacerbations and that d-dimer levels are elevated in IPF-AE (27). Measurement of d-dimer in patients with ILD may be useful in the differential diagnosis of pulmonary thromboembolism (28). In our study, d-dimer levels were highest in the PTE group and showed significant differences among the different etiologies of hospitalization.

Hospitalization and oxygen therapy are required in acute exacerbations of IPF. In the review by Zhao et al., high-flow nasal cannula was associated with lower intubation rates, while no differences were observed between HFNC and NIMV in terms of intubation or mortality (29). In our study, most patients were managed with conventional oxygen therapy, and only one patient required invasive mechanical ventilation. When mechanical ventilation is required, the risk of ventilator-induced lung injury should be taken into consideration (30).

Despite the overall decline in tobacco use, smoking remains one of the leading risk factors for disability related to chronic respiratory diseases among men worldwide (31). Several studies have demonstrated that a smoking history of more than 20 pack-years plays a role in the pathogenesis of IPF and that prognosis is worse in current or former smokers compared with never-smokers (32). In our study, 19.3% of the participants were current smokers, with a mean smoking exposure of 32.38 ± 22.1 pack-years. These findings suggest that smoking history remains an important risk factor in our patient population.

The pathogenesis of the association between ILD and diabetes mellitus (DM) is multifactorial and complex (33). Although international guidelines do not list diabetes as a risk factor for ILD (34), diabetic autonomic neuropathy, increased oxidative stress, and microvascular changes in the alveolar capillary bed may adversely affect lung function. Excessive production of extracellular matrix components and non-enzymatic protein glycation induced by hyperglycemia lead to matrix stiffening and promote the progression of pulmonary fibrosis. In addition, hyperglycemia accelerates lung fibrosis through activation of STAT3, CTGF, and TGF- β (33). In a Japanese case-control study including 65 patients with IPF and 184 controls, the prevalence of DM was significantly higher in patients with IPF (32.7%) compared with controls (11.4%) (35). In our study, the prevalence of DM was 24.8%, which is consistent with the literature.

In our study, hospitalization was required in 11% of patients for initiation of steroid therapy and close monitoring related to treatment, and systemic corticosteroids were administered to 61.5% of the patients. It is well known that long-term use of systemic corticosteroids may lead to the development of steroid-induced diabetes mellitus. In a Korean study, steroid-induced diabetes mellitus was reported in 21.6% of 125 patients with idiopathic interstitial pneumonia treated with oral glucocorticoids (36).

The reported prevalence of obstructive sleep apnea syndrome (OSAS) in ILD ranges between 5.9% and 91% in the literature (37). Lee et al. reported advanced age, higher body weight, and diabetes mellitus as risk factors for OSAS in patients with ILD (38). In patients with ILD, OSAS is thought to occur more frequently due to an increased tendency for upper airway collapse during REM sleep, when intercostal muscle activity decreases and functional residual capacity is further reduced (39). In our study, the prevalence of OSAS was found to be 1.8%, which is markedly lower than the rates reported in the literature. This finding may be explained by the lack of systematic screening with polysomnography in all patients and by the inclusion of only previously diagnosed cases.

During the COVID-19 pandemic period covered by our study, the number of hospitalized patients decreased compared with previous years, likely due to reduced hospital admissions related to fear of infection and the potential radiological overlap between certain ILD subtypes and SARS-CoV-2 pneumonia. Nevertheless, we aimed to generate detailed epidemiological data on the causes of acute respiratory failure in patients with ILD.

In Turkey, there are limited data regarding acute respiratory failure in patients with interstitial lung diseases. In a study by Kaya et al. including 65 ILD patients admitted to the intensive care unit, disease activation (69%) and pulmonary infection (30.8%) were reported as the most common causes of acute respiratory failure (40). In our study, pulmonary infection was again the leading etiology, followed by primary disease exacerbation, pulmonary thromboembolism, and congestive heart failure. These findings expand the limited national epidemiological data and highlight the heterogeneity of ARF etiologies in ILD.

Limitations

This study has several limitations. First, although prospectively designed, it was conducted at a single tertiary center with a relatively limited sample size, which may restrict the generalizability of the findings. Second, microbiological investigations and polysomnographic screening for obstructive sleep apnea syndrome were not systematically performed in all patients, potentially leading to underestimation of certain etiologies and comorbid conditions. Third, the definition of acute respiratory failure included both objective oxygenation criteria and clinical assessment, which may have introduced some degree of heterogeneity. In addition, hospitalization patterns and healthcare-seeking behavior during the COVID-19 pandemic may have influenced patient characteristics and admission thresholds.

Conclusion

We believe that our findings provide valuable national epidemiological data on the etiologies of acute respiratory failure in patients with ILD and may facilitate earlier recognition and more appropriate management of this high-risk population.

Declarations

Ethics Approval

The study was approved by the Ethics Committee of Karadeniz Technical University Faculty of Medicine on December 14, 2020 (Approval No. 2020-34).

Consent to Participate

Written informed consent was obtained from all participants prior to their inclusion in the study.

Conflict of Interest

The authors declare that they have no competing interests.

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Author Contribution

Özge Argin and Funda Öztuna contributed to the conception and design of the study. Özge Argin collected and analyzed the data and drafted the manuscript. All authors reviewed and approved the final manuscript.

Data Availability

The datasets generated and/or analyzed during this study are not publicly available due to patient confidentiality but may be obtained from the corresponding author upon reasonable request, subject to institutional and ethical approval.

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