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ORIGINAL ARTICLE

Predictive risk factors for one-year mortality in idiopathic inflammatory myopathy patients with interstitial lung disease: A retrospective, single-center cohort study

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ABSTRACT

Objectives: This study aimed to analyze the risk factors for mortality of idiopathic inflammatory myopathy (IIM) patients admitted with interstitial lung disease (ILD) to guide rapid and accurate judgment of clinical prognosis.

Patients and methods: This retrospective, single-center cohort study was conducted with 135 participants (37 males, 98 females; mean age: 54.8±11.1 years; range, 24 to 85 years) between June 1, 2016, and June 30, 2021. The participants were categorized into the survival group (n=111) and nonsurvivors (n=24) according to whether they survived during the one-year follow-up. The independent risk factors for mortality in one year after discharge were analyzed. Receiver operating characteristic curve analysis was used to determine the accuracy of oxygenation index at baseline combined with pulmonary infection (PI) at follow-up to indicate death in IIM-ILD patients.

Results: Compared to the survival group, nonsurvivors were older (p=0.006) and had a higher proportion of anti-MDA5 (melanoma differentiation-associated protein 5) positivity (p<0.001). The ILD duration was shorter (p=0.006), the oxygenation index was lower (p<0.001), and the intensive care unit occupancy rate (p<0.001) and ventilator utilization rate (p<0.001) were elevated in nonsurvivors compared to the survival group. Oxygenation index at baseline (odds ratio [OR]=1.021, 95% confidence interval [CI]: 1.001-1.023, p=0.040) and PI (clinical judgment) at follow-up (OR=16.471, 95% CI: 1.565-173.365, p=0.020) were found as independent risk factors for death in the year after discharge in IIM inpatients with ILD. An oxygenation index ≤279 mmHg at baseline combined with PI at follow-up exhibited a promising predictive value for all-cause death in IIM-ILD patients within one year.

Conclusion: Oxygenation index at baseline and PI during follow-up were independent risk factors for death of IIM-ILD patients within one year after discharge. Patients with an oxygenation index ≤279 mmHg at baseline had an increased risk of death once they developed PI during the one-year follow-up.

Keywords: Idiopathic inflammatory myopathy, interstitial lung disease, pulmonary infection, risk factors.

Idiopathic inflammatory myopathy (IIM) is a group of heterogeneous autoimmune myopathy mainly characterized by chronic muscle inflammation, skin damage, and interstitial pneumonia. IIM can be classified into polymyositis (PM), dermatomyositis (DM), anti-synthetase syndrome, immune-mediated necrotizing myopathy, inclusion body myositis, and overlap syndrome.[1] Myositis-associated interstitial pneumonia is the main extramuscular manifestation of IIM, occurring in approximately 20 to 80% of patients.[2,3] The course and severity of myositis-associated interstitial lung disease (ILD) are highly heterogeneous, and some patients with mild ILD can remain relatively stable for long periods of time and respond well to treatment, whereas others may progress rapidly, respond poorly to treatment, and it may even lead to death.[4] Therefore, it is essential to investigate risk factors for progression and mortality of myositis-associated ILD.

Previous studies have reported lower respiratory tract infections as the most common cause of death in IIM, followed by malignancy and rapidly progressive ILD (RP-ILD). Multivariate analysis showed that age and anti-MDA5 (melanoma differentiation-associated protein 5) antibody positive were predictive factors of IIM mortality.[5] Other studies have reported that older age, fewer peripheral blood lymphocytes, and skin involvement are risk factors for rapid progression of IIM-associated ILD.[6] A large number of studies have suggested that RP-ILD is a risk factor for the increased mortality in IIM patients. However, the pathogenesis of lung injury in IIM patients is still elusive. The present study aimed to analyze the risk factors for mortality of IIM patients admitted with ILD to guide rapid and accurate judgment of clinical prognosis.

PATIENTS AND METHODS

This single-center retrospective cohort study included 135 IIM inpatients (37 males, 98 females; mean age: 54.8±11.1 years; range, 24 to 85 years) complicated with ILD at the Department of Respiratory and Critical Care Medicine and the Department of Infectious Diseases and Clinical Microbiology between June 1, 2016, and June 30, 2021. The data of all cases included in the study were extracted from electronic medical records. The inclusion criteria were as follows: *(i)* DM/PM meeting diagnostic criteria suggested by Bohan and Peter's[7] DM/PM classification criteria or Sontheimer's[8] definitions; *(ii)* presence of a chest computed tomography (CT) completed at admission and the examination results independently reviewed by two radiologists to diagnose ILD; *(iii)* age ≥18 years. Anti-synthetase syndrome was diagnosed with definitive serology findings of one of anti-aminoacyl tRNA synthetase antibodies tested, along with at least one triad finding, including myositis, arthritis, and ILD.[9] The exclusion criteria were as follows: *(i)* the outpatient or inpatient follow-up data collected one year after discharge could not be consulted; *(ii)* no chest imaging was found at admission and one year after discharge. All cases were divided the into survival group (n=111) and nonsurvivors (n=24) according to whether they died within one year of follow-up (Table 1). A detailed flowchart is shown in Figure 1.

Baseline and follow-up data were collected for all subjects who were enrolled in the cohort. Baseline data included age, sex, duration of ILD and IIM, length of hospital stay, length of intensive care unit (ICU) stay, myositis antibodies, respiratory support conditions, past medical history, chest CT findings, primary treatment plan, whether anti-infective therapy was performed, and efficacy of anti-infective therapy. Chest CT findings included the presence of ILD, signs of infection, and pleural effusion. Chest CT was performed during follow-up to assess the progression of ILD. Primary treatment options included pulse therapy with glucocorticoids, high-dose therapy, the course of treatment with glucocorticoids, the decision to use immunosuppressants, and the type of immunosuppressant.

Pulse therapy with glucocorticoids was defined as more than 250 mg of prednisone per day, and high-dose therapy was defined as more than 30 mg and less than 250 mg of prednisone per day.[10] The duration of glucocorticoid treatment was measured in months. The type of immunosuppressive therapy was recorded and classified into antimetabolites, alkylating agents, calcineurin inhibitors, biologics.[11,12]

The baseline chest high-resolution CT was compared to the CT revisited after treatment, and the ILD progression was defined as >10% worsening in the area of the lesions of ILD.

The oxygenation index is defined as the arterial oxygen partial pressure/fraction of inspired oxygen ratio. It is the most commonly used index for assessment of oxygenation status in patients with respiratory failure. This study referred to the relevant classification criteria of acute respiratory distress syndrome,[13] and in the results section, grouping studies are conducted according to whether the oxygenation index is <300.

The primary outcome included death during follow-up. The progression of ILD was followed up for one year. The progression of ILD was assessed based on the comparison of interstitial lung lesions on chest CT with the baseline stage, and the results were determined by independent review performed by two radiologists.

Table 1. Characteristics of patients at study entry and follow-up according to groups

	1-year death (n=24)					1-year alive (n=111)					
	n	%	Mean±SD	Median	Min-Max	n	%	Mean±SD	Median	Min-Max	*p*
Demographics and condition of disease											
Age (year)			60.1±10.2					53.3±10.9			0.006
Sex Male	7	29.2				30	27.0				0.831
Course of IIM (month)				2.3	0.8-40.5				3.0	1.0-15.0	0.856
Course of ILD (month)				1.5	0.7-4.4				4.0	2.0-18.5	0.006
Anti-MDA5+	19	79.2				20	18.0				<0.001
ER therapy before admission	18	75.0				33	29.7				<0.001
ICU admission	10	41.7				3	2.7				<0.001
Length of stay (day)				9.0	7.0-25.8				11.0	9.0-15.0	0.926
Comorbidity											
Hypertension	6	25.0				32	28.8				0.705
Diabetes	2	8.3				9	8.1				1.000
Coronary heart disease	2	8.3				11	9.9				1.000
Cerebrovascular disease	1	4.2				3	2.7				1.000
Chronic airway disease	0	0				10	9.0				0.272
Radiological findings											
Consolidations	8	33.33				31	27.93				0.624
Ground-glass attenuation areas	11	45.83				68	61.26				0.178
Reticular opacities	10	41.67				56	50.45				0.503
Pleural effusion	10	41.67				14	12.61				0.002
Treatment and relevant data											
Mechanical ventilation	11	45.8				4	3.6				<0.001
Oxygenation index				254	124-340				371	319-418	<0.001
Glucocorticoids using											
Pulse therapy (>250 mg/d)	4	16.7				6	5.4				0.139
High-dose (>30 mg/d)	19	79.2				94	84.7				0.720
More than 1 month	6	25.0				31	27.9				0.771
Combination of two or more immunosuppressive agents	6	25.0				10	9.0				0.064
Follow-up data											
Pulmonary infection (clinical judgment) at follow-up*	6	85.7				20	18.0				<0.001
ILD progression at follow-up*	5	71.4				29	26.1				0.033

SD: Standard deviation; IIM: Idiopathic inflammatory myopathy; ILD: Interstitial lung disease; MDA5: Melanoma Differentiation-Associated protein 5; ER: Emergency room; ICU: Intensive care unit; * A total of 17 patients died during hospitalization or within 30 days of discharge, thus, these cases were not included in the analysis of disease assessment during follow-up.

Statistical analysis

Statistical analysis was performed using IBM SPSS version 23.0 software (IBM Corp., Armonk, NY, USA) and MedCalc version 19.6.4 (MedCalc Software, Ostend, Belgium). Data were presented as either median (interquartile range) (IQR) or mean ± standard deviation (SD) for numerical variables, and categorical variables were expressed as frequency and percentage. Continuous variables with normal distribution were compared using a t-test, while those with abnormal distribution were compared using the Mann-Whitney U test. Categorical variables were compared using the chi-square test. Variables that differed significantly between the survival group and nonsurvivors were considered potential risk

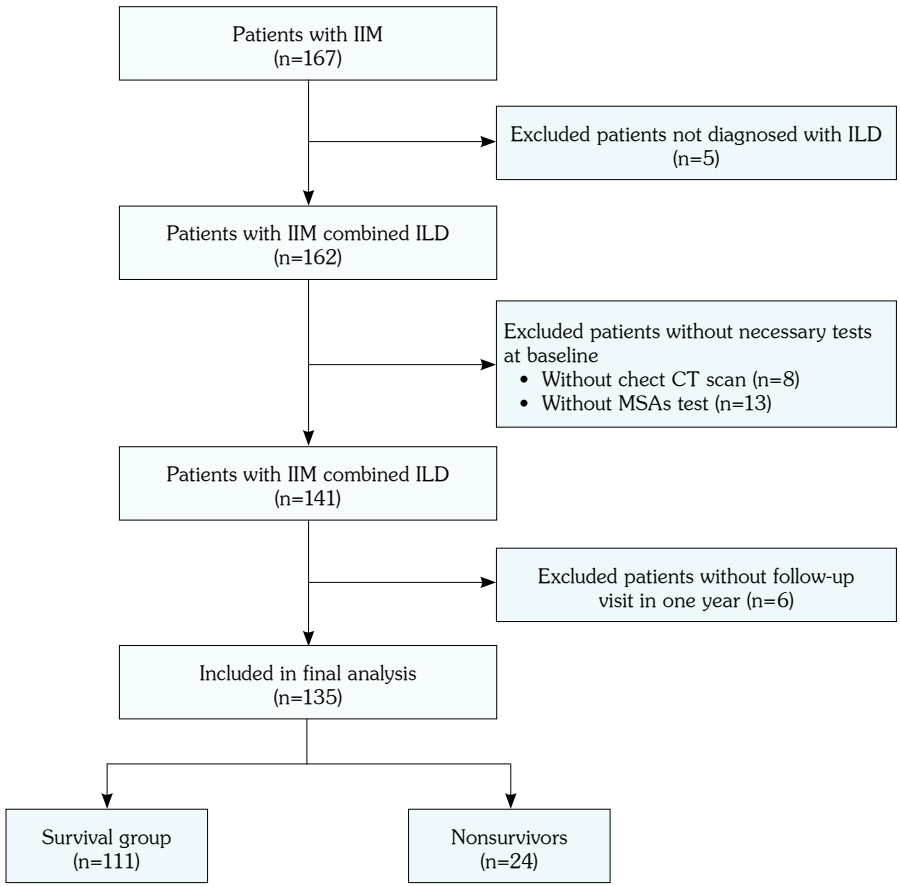


Figure 1. Flowchart of the study.

IIM: Idiopathic inflammatory myopathy; ILD: Interstitial lung disease; CT: computed tomography; MSAs: Myositis-specific antibodies.

factors. The association of these variables with different statuses was further assessed using univariate and multivariate logistic regression analyses. The results were presented as estimates of relative risk (RR) by odds ratio (OR) with 95% confidence interval (CI). Survival was estimated by the Kaplan-Meier method, and any differences in survival were evaluated with a stratified log-rank test. Receiver operating characteristic (ROC) curves were plotted to calculate the area under the curve (AUC) values for different diagnostic tools, and the logistic regression model for combined diagnoses was established based on the findings. The predictive probabilities of the combined diagnoses were calculated, the ROC curves were plotted according to the predictive probabilities, and the AUC values for different combined diagnoses were calculated. A p-value <0.05 was considered statistically significant.

RESULTS

One year mortality was affected by multiple factors at baseline and during follow-up.

Of the 24 nonsurvivors, eight patients died during the hospitalization period. Compared to the survival group, nonsurvivors were older (53.3±10.9 *vs.* 60.1±10.2 years, p=0.006). The proportion of anti-MDA5 antibody-positive patients was higher (79.2% *vs.* 18.0%, p<0.001), ILD duration was shorter (median: 1.5 *vs.* 4.0 months; p=0.006), oxygenation index was lower (median: 371 *vs.* 254; p<0.001), ICU occupancy rate (41.7% *vs.* 2.7%, p<0.001) and ventilator usage (45.8% *vs.* 3.6%, p<0.001) were higher and the follow-up pulmonary infection rate (85.7% *vs.* 18.0%, p<0.001) was higher in nonsurvivors compared to the survival group.

Table 2. Univariate and multivariate logistic regression analyses of potential risk factors for death

	Univariate logistic regression			Multivariate logistic regression		
	OR	95% CI	*p*	OR	95% CI	*p*
Age	0.938	0.894-0.984	0.008			
Anti-MDA5-positive	15.600	5.200-46.798	<0.001			
PI (clinical judgment) at follow-up	27.000	3.077-236.886	0.003	16.471	1.565-173.365	0.020
Oxygenation index	1.015	1.009-1.021	<0.001	1.012	1.001-1.023	0.040

PI: Pulmonary infection; OR: Odds ratios; CI: Confidence interval.

Compared to the survival group, nonsurvivors had a higher proportion of ILD progression during the one-year follow-up (26.1% *vs.* 71.4%, p=0.033).

For other radiological findings, the most common finding was ground-glass attenuation areas (n=79). Reticular opacities (n=66) and consolidations (n=39) were also frequent radiological changes. However, the above radiological changes were not statistically different between the survival group and nonsurvivors. Pleural effusion was more common in nonsurvivors, and the difference between the two groups was statistically significant.

Oxygenation index and PI at follow-up increased the risk of death of IIM-ILD patients during one-year follow-up.

The age, anti-MDA5-positive results, PI (clinical judgment) at follow-up, and oxygenation index were included in univariate logistic regression analysis with statistical significance. The meaningful indicators in the present study were respectively included in the multivariate logistics regression analysis. Oxygenation index at baseline (OR=1.021, 95% CI: 1.001-1.023, p=0.040) and PI (clinical judgment) at follow-up (OR=16.471, 95% CI: 1.565-173.365, p=0.020) were found as independent risk factors for death in one year after discharge in IIM inpatients with ILD (Table 2).

Low oxygenation index at baseline and PI at follow-up had influence on survival of patients.

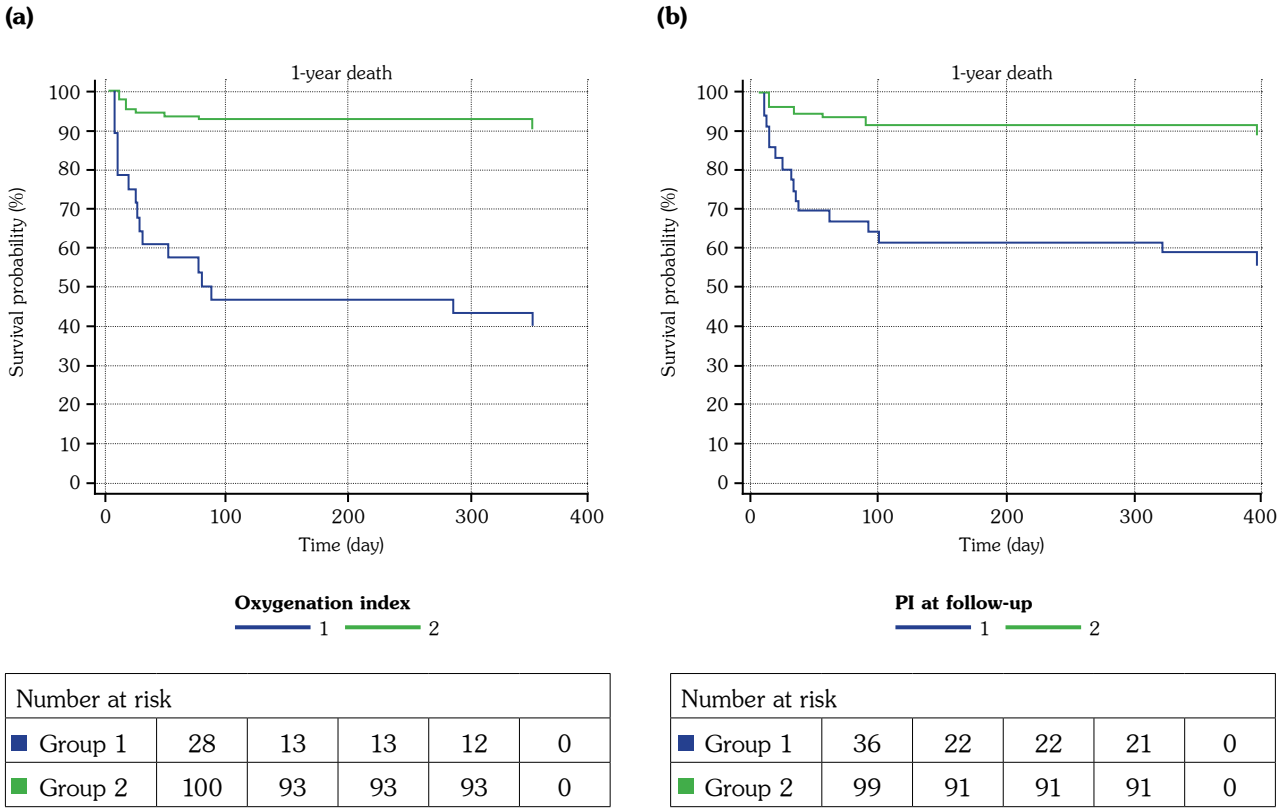
A total of 135 patients were followed up for one year, and 24 (17.78%) all-cause deaths occurred. There were 12 (33.33%) deaths with PI at follow-up and 12 (12.12%) deaths without PI. And there were 16 (57.14%) all-cause deaths with an oxygenation index ≤279 and eight (7.55%) deaths with an oxygenation index >279.

The Kaplan-Meier method and log-rank test were used in our study to investigate the relationship between oxygenation index, PI at follow-up, and IIM-ILD prognosis (Figures 2a, b). The results indicated that the oxygenation index had a significantly lower overall survival rate than other patients (hazard ratio [HR]=37.721, p<0.001), and PI at follow-up had a significantly lower overall survival rate than non-PI patients (HR=10.685, p<0.001).

Analysis of the oxygenation index ROC curve that differentiated the survival group and the mortality group of patients with IIM-ILD showed that the AUC was 0.809 (95% CI: 0.673-0.946, p<0.006). Analysis of the PI at the follow-up ROC curve that differentiated the survival group and the mortality group of patients with IIM-ILD showed that the AUC was 0.831 (95% CI: 0.674-0.989, p<0.003).

Low oxygenation index at baseline combined with PI at follow-up could predict the potential risk of one-year death.

The prediction value of the two factors in combination for mortality was assessed. The findings showed that the AUC of ROC of oxygenation index + PI (clinical judgment) at follow-up was 0.895 (95% CI: 0.822-0.949, p<0.001). The sensitivity was 85.71%, and the specificity was 88.35%. Compared to the oxygenation index, oxygenation index + PI (clinical judgment) at follow-up improved the



Number at risk					
Group 1	28	13	13	12	0
Group 2	100	93	93	93	0

Number at risk					
Group 1	36	22	22	21	0
Group 2	99	91	91	91	0

Figure 2. **(a)** Oxygenation index K-M curve. **(b)** PI at follow-up K-M curve.
K-M: Kaplan–Meier; PI: Pulmonary infection.

prediction accuracy, and the difference was statistically significant (p=0.028).

Further analysis of the cut-off value for the oxygenation index with PI at follow-up demonstrated that it had a satisfactory value in determining patients' death during a one-year follow-up, with the presence of PI at follow-up and an oxygenation index ≤279 mmHg.

DISCUSSION

Interstitial lung disease is the most prominent extramuscular manifestation of IIM, which significantly affects the prognosis of the disease. In the present study, 135 patients with IIM-ILD who were followed up for one year were enrolled. The risk factors of death in IIM patients with ILD were analyzed and evaluated. The oxygenation index at baseline combined with PI at follow-up was used for the first time to predict the one-year death of patients.

In the present study, the risk of PI was significantly elevated in nonsurvivors, and PI during follow-up was an independent risk factor for death in IIM-ILD patients, which was consistent with previous findings that respiratory tract infection was the cause of progression and death of IIM-ILD.[14] Several reasons may cause infection, particularly PI, in IIM-ILD patients. Among hospitalized individuals with IIM, infection is not rare and is the leading cause of mortality.[15] In addition, several studies have found evidence of occult respiratory infections at autopsy in patients who died of acute exacerbation of idiopathic pulmonary fibrosis,[16] which would refer to a respiratory tract infection as a risk factor for death. Studies have found that the cumulative mortality at one year after diagnosis with IIM was 9%.[17] However, Chen et al.[18] demonstrated that after severe infection in IIM patients, survival declined to 84.7% at 30 days and 68.3% at one year. In our study, the mortality of hospitalized IIM-ILD patients in

the first year after discharge was 17.8%, and the leading cause of death was PI.

The mechanism by which PI leads to poor prognosis may be multifactorial. It is speculated that on the one hand, the pathogen may directly damage the lungs, leading to pulmonary fibrosis and aggravated ILD, or activate the immune system, causing macrophages, neutrophils, eosinophils, and T helper type 2 cells to accumulate in the injured site and release a large number of proinflammatory and profibrotic cells or factors. The combination of pathogens and these factors leads to persistent and substantial lung injury, promotes pulmonary fibrosis, and causes aggravation of ILD.[19] On the other hand, PI may change the microecological environment of pulmonary pathogens, promote pulmonary fibrosis, and aggravate ILD.[20]

Although infection was not found as an independent risk factor for death in IIM-ILD patients in a past study,[5] Bai et al.[21] demonstrated that infection was the main factor for death in IIM-ILD patients. At the same time, it was also found in the present study that the rates of baseline PI, all-cause infection, and follow-up PI were significantly higher in nonsurvivors, and the differences were statistically significant, suggesting that vigilant against infection is still essential throughout the treatment, particularly PI.

The oxygenation index is an important predictor of the prognosis of patients in a critical condition. In patients with persistent pneumonia, the oxygenation index was shown to decrease, indicating that the body was in severe hypoxia. Through dynamic monitoring of the oxygenation index, treatment protocols can be effectively guided while evaluating the prognosis.[22] However, some patients with severe ILD and low oxygenation index cannot tolerate bronchoscopy, which limits the predictive role of bronchoalveolar lavage fluid in RP-ILD death in IIM patients. It is crucial to simply, quickly, and effectively determine whether IIM-ILD patients die within one year. The present study is the first to combine the oxygenation index during hospitalization with PI at follow-up, and it was found that an oxygenation index at baseline ≤279 mmHg combined with PI at one-year follow-up had a promising value in predicting the death of IIM patients combined with ILD within one year. Arterial blood gas analysis is a routine examination for IIM-ILD patients admitted to a hospital, and PI is also a routine monitoring content for these patients during follow-up. This combination has a strong clinical utility and provides an important evaluation tool for the follow-up of such patients.

Some limitations of the present study should be noted. First, this study included 135 hospitalized IIM patients with ILD from a single center. The clinical condition of these patients who necessitated hospitalization was relatively severe or complicated, making it difficult to generalize the findings, and there may be a potential selection bias. Additionally, the small sample size limited the statistical power. Second, due to the influence of other factors, such as the interference of COVID-19 (coronavirus disease 2019), the follow-up of this study was only one year, and thus the conclusions may be limited by the duration of follow-up. These limitations can be eliminated in future multicenter prospective studies.

In conclusion, it was found that oxygenation index at baseline and PI during follow-up were independent risk factors for the death of IIM-ILD patients within one year after discharge. Oxygenation index ≤279 mmHg at baseline combined with PI at follow-up exhibited a promising predictive value for all-cause death in IIM-ILD patients within one year.

Ethics Committee Approval: The study was approved by the Institutional Review Board of Beijing Chao-Yang Hospital (date: 25.11.2022; no: 2022-KE-611). All methods in this study were carried out in accordance with the Declaration of Helsinki.

Patient Consent for Publication: A written informed consent was obtained from each patient.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: Performed data collection, analyzed data, and wrote the manuscript: M.J.; Were responsible for data analysis and recruiting patients: X.W., S.X., Y.G.; Contributed as a primary investigator and was responsible for designing the study and writing the manuscript: Y.B.; All authors read and approved the final manuscript.

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REFERENCES

1. Callen JP. Dermatomyositis. Lancet 2000;355:53-7. doi: 10.1016/S0140-6736(99)05157-0.
2. Lundberg IE, Fujimoto M, Vencovsky J, Aggarwal R, Holmqvist M, Christopher-Stine L, et al. Idiopathic inflammatory myopathies. Nat Rev Dis Primers 2021;7:86. doi: 10.1038/s41572-021-00321-x.
3. Jablonski R, Bhorade S, Strek ME, Dematte J. Recognition and management of myositis-associated rapidly progressive interstitial lung disease. Chest 2020;158:252-63. doi: 10.1016/j.chest.2020.01.033.
4. Selva-O'Callaghan A, Pinal-Fernandez I, Trallero-Araguás E, Milisenda JC, Grau-Junyent JM, Mammen AL. Classification and management of adult inflammatory myopathies. Lancet Neurol 2018;17:816-28. doi: 10.1016/S1474-4422(18)30254-0.
5. Mehta P, Agarwal V, Gupta L. High early mortality in idiopathic inflammatory myopathies: Results from the inception cohort at a tertiary care centre in northern India. Rheumatology (Oxford) 2021;60:4281-90. doi: 10.1093/rheumatology/keab001.
6. Li Y, Gao X, Li Y, Jia X, Zhang X, Xu Y, et al. Predictors and mortality of rapidly progressive interstitial lung disease in patients with idiopathic inflammatory myopathy: A series of 474 patients. Front Med (Lausanne) 2020;7:363. doi: 10.3389/fmed.2020.00363.
7. Bohan A, Peter JB. Polymyositis and dermatomyositis (first of two parts). N Engl J Med 1975;292:344-7. doi: 10.1056/NEJM197502132920706.
8. Sontheimer RD. Would a new name hasten the acceptance of amyopathic dermatomyositis (dermatomyositis siné myositis) as a distinctive subset within the idiopathic inflammatory dermatomyopathies spectrum of clinical illness? J Am Acad Dermatol 2002;46:626-36. doi: 10.1067/mjd.2002.120621.
9. Cavagna L, Trallero-Araguás E, Meloni F, Cavazzana I, Rojas-Serrano J, Feist E, et al. Influence of antisynthetase antibodies specificities on antisynthetase syndrome clinical spectrum time course. J Clin Med 2019;8:2013. doi: 10.3390/jcm8112013.
10. Buttgereit F, da Silva JA, Boers M, Burmester GR, Cutolo M, Jacobs J, et al. Standardised nomenclature for glucocorticoid dosages and glucocorticoid treatment regimens: Current questions and tentative answers in rheumatology. Ann Rheum Dis 2002;61:718-22. doi: 10.1136/ard.61.8.718.
11. Aggarwal R, Oddis CV. Therapeutic advances in myositis. Curr Opin Rheumatol 2012;24:635-41. doi: 10.1097/BOR.0b013e328358ac72.
12. Moghadam-Kia S, Oddis CV. Current and new targets for treating myositis. Curr Opin Pharmacol 2022;65:102257. doi: 10.1016/j.coph.2022.102257.
13. Ranieri VM, Rubenfeld GD, Thompson BT, Ferguson ND, Caldwell E, Fan E, et al. Acute respiratory distress syndrome: the Berlin Definition. JAMA 2012;307:2526-33. doi: 10.1001/jama.2012.5669.
14. Shappley C, Paik JJ, Saketkoo LA. Myositis-related interstitial lung diseases: Diagnostic features, treatment, and complications. Curr Treatm Opt Rheumatol 2019;5:56-83. doi: 10.1007/s40674-018-0110-6.
15. Ge YP, Shu XM, He LR, Wang GC, Lu X. Infection is not rare in patients with idiopathic inflammatory myopathies. Clin Exp Rheumatol 2022;40:254-9. doi: 10.55563/clinexprheumatol/yps7ai.
16. Oda K, Ishimoto H, Yamada S, Kushima H, Ishii H, Imanaga T, et al. Autopsy analyses in acute exacerbation of idiopathic pulmonary fibrosis. Respir Res 2014;15:109. doi: 10.1186/s12931-014-0109-y.
17. Dobloug GC, Svensson J, Lundberg IE, Holmqvist M. Mortality in idiopathic inflammatory myopathy: Results from a Swedish nationwide population-based cohort study. Ann Rheum Dis 2018;77:40-7. doi: 10.1136/annrheumdis-2017-211402.
18. Chen IJ, Tsai WP, Wu YJ, Luo SF, Ho HH, Liou LB, et al. Infections in polymyositis and dermatomyositis: Analysis of 192 cases. Rheumatology (Oxford) 2010;49:2429-37. doi: 10.1093/rheumatology/keq279.
19. Huang WJ, Tang XX. Virus infection induced pulmonary fibrosis. J Transl Med 2021;19:496. doi: 10.1186/s12967-021-03159-9.
20. Invernizzi R, Wu BG, Barnett J, Ghai P, Kingston S, Hewitt RJ, et al. The respiratory microbiome in chronic hypersensitivity pneumonitis is distinct from that of idiopathic pulmonary fibrosis. Am J Respir Crit Care Med 2021;203:339-47. doi: 10.1164/rccm.202002-0460OC.
21. Bai Z, Shen G, Dong L. Analysis of risk factors of interstitial lung disease and mortality rates in Chinese patients with idiopathic inflammatory myopathy. Int J Rheum Dis 2021;24:815-27. doi: 10.1111/1756-185X.14128.
22. Liu W, Peng L, Hua S. Clinical significance of dynamic monitoring of blood lactic acid, oxygenation index and C-reactive protein levels in patients with severe pneumonia. Exp Ther Med 2015;10:1824-8. doi: 10.3892/etm.2015.2770.