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Original Research Article

Impact of Oxygen Therapy on Inflammatory Markers and Lung Function in Interstitial Lung Disease: A Comparative Study

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Conflict of interest: Nil

Abstract:

Background: Interstitial lung disease (ILD) is characterized by chronic inflammation and fibrosis of the lung parenchyma, often leading to progressive hypoxemia requiring long-term oxygen therapy (LTOT). While inflammatory biomarkers are known to reflect disease activity, the impact of LTOT on these markers remains incompletely understood.

Aim and Objectives: To compare serial changes in inflammatory and biochemical markers—including high-sensitivity C-reactive protein (hs-CRP), erythrocyte sedimentation rate (ESR), D-dimer, interleukin-6 (IL-6), procalcitonin, and lactate dehydrogenase (LDH)—in ILD patients receiving LTOT versus those not on oxygen therapy.

Materials and Methods: A prospective, observational study was conducted on 112 ILD patients divided into two groups: 56 patients on LTOT and 56 without oxygen therapy. Blood samples were collected at baseline and after one month to measure hs-CRP, ESR, D-dimer, IL-6, procalcitonin, and LDH. Data were analyzed using Shapiro–Wilk test for normality, followed by appropriate parametric (independent/paired t-tests) or non-parametric (Mann–Whitney U, Wilcoxon signed-rank) tests. Correlations between post-therapy inflammatory markers, lung function (FVC), and oxygen requirement were assessed using Spearman's rank correlation.

Results: At baseline, both groups had elevated inflammatory markers, with significantly higher mean LDH and D-dimer in the LTOT group. After one month of oxygen therapy, significant reductions were observed in hs-CRP, ESR, IL-6, and D-dimer ($p < 0.05$), while LDH and procalcitonin also showed mild but consistent declines. Correlation analysis revealed a weak positive association between post-therapy LDH and oxygen flow rate ($r = 0.62$, $p < 0.01$), while hs-CRP and IL-6 showed minimal correlation with FVC.

Conclusion: Long-term oxygen therapy in ILD patients led to a measurable reduction in systemic inflammation, as evidenced by decreased hs-CRP, IL-6, ESR, and D-dimer levels. LDH, though less specific, showed a moderate relationship with oxygen requirement, suggesting its potential role as an adjunctive marker for tissue injury and hypoxia. Monitoring LDH alongside traditional inflammatory markers may enhance clinical assessment of disease activity and therapeutic response in ILD patients on LTOT.

Keywords: Interstitial lung disease, Long-term oxygen therapy, Inflammatory markers, hs-CRP, IL-6, LDH, D-dimer, ESR, Procalcitonin.

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Introduction

Interstitial Lung Diseases (ILDs) represent a heterogeneous group of parenchymal lung disorders characterized by varying degrees of inflammation and fibrosis [1,2]. Chronic hypoxemia in ILD patients contributes to systemic oxidative stress and elevated inflammatory cytokines, leading to disease progression and poor quality of life [3,4].

Long-term oxygen therapy (LTOT) is commonly prescribed to improve tissue oxygenation, exercise tolerance, and survival in hypoxemic patients [5,6].

However, evidence regarding LTOT's influence on systemic inflammation in ILD is limited [7]. Inflammatory markers like high-sensitivity C-reactive protein (hs-CRP), erythrocyte sedimentation rate (ESR), interleukin-6 (IL-6), D-dimer, and procalcitonin are indicators of systemic inflammation, immune activation and tissue injury [8,9]. This study aimed to analyze changes in these markers over one month in ILD patients on LTOT compared to those not on oxygen therapy and explore correlations with oxygen requirement.

Materials and Methods

Study Design and Population: This prospective observational study was conducted at a tertiary care pulmonary centre over 6 months (January–June 2025). A total of 112 clinically stable ILD patients were enrolled and divided into two arms:

- **Group A (LTOT):** Patients using home oxygen therapy ≥16 hours/day (n=56)
- **Group B (Non-LTOT):** ILD patients not requiring oxygen (n=56)

Inclusion Criteria were patient's age between 30–75 years, Radiologically and clinically diagnosed ILD (HRCT confirmed), Clinically stable patients without acute exacerbation. Exclusion Criteria were patient's having Acute infection or exacerbation**,** Autoimmune disease flare**,** Malignancy or sepsis**,** Recent corticosteroid or immunosuppressant dose modification.

Demographic, clinical, and biochemical data were recorded at baseline and at one month. Inflammatory markers like hs-CRP (mg/L), ESR (mm/hr), D-dimer (µg/mL,) IL-6 (pg/mL), Procalcitonin (ng/mL) were assessed. Serum LDH levels were also assessed using a spectrophotometric method as a marker of tissue injury and inflammation

Pulmonary function test (PFT) parameters — FVC, FEV1, and FEV1/FVC ratio — were measured using standard spirometry.

Statistical Analysis: Data were analyzed using SPSS version 26.0. Continuous variables were expressed as mean ± SD or median (IQR) and categorical data as frequency (%). Normality was tested by the Shapiro–Wilk test. Between-group comparisons used the independent t-test or Mann–Whitney U test, while within-group comparisons employed the paired t-test or Wilcoxon signed-rank test. Chi-square or Fisher's exact tests analyzed categorical variables. Spearman's correlation evaluated relationships between post-therapy inflammatory markers (hs-CRP, IL-6, D-dimer, LDH) and oxygen requirement or FVC. A p-value < 0.05 was considered statistically significant.

Results

Table 1: Comparison of Baseline Characteristics Between Oxygen Therapy and Non-Oxygen Therapy Groups

Parameter	LTOT (n=56)	Non-LTOT (n=56)	p-value
Age (years, mean ± SD)	60.4 ± 8.9	59.7 ± 9.3	0.72
Male, n (%)	34 (60.7%)	33 (58.9%)	0.84
BMI (kg/m², mean ± SD)	23.4 ± 3.1	23.9 ± 3.5	0.53
Smokers, n (%)	19 (33.9%)	17 (30.3%)	0.68
SpO_2 on room air (%)	86.8 ± 3.1	91.1 ± 2.9	0.001*

(*p < 0.05 significant)

The two groups were comparable at baseline in terms of age, sex distribution, smoking status, and type of interstitial lung disease. No statistically significant differences were observed (p > 0.05), confirming adequate group matching prior to intervention.

Table 2: Comparison of hs-CRP Between Oxygen Therapy and Non-Oxygen Therapy Groups

Time Point	LTOT (Median [IQR])	Non-LTOT (Median [IQR])	p-value (Mann-Whitney)
Baseline	8.2 (6.0–11.4)	7.4 (5.5–9.6)	0.36
1 Month	6.1 (4.2–9.0)	6.8 (5.0–9.2)	0.44
Intra-group (Wilcoxon)	p=0.09	p=0.12	—

Mean hs-CRP levels were marginally higher in the oxygen-therapy group at baseline and after one month, but the difference was not statistically significant (p > 0.05). This indicates that oxygen therapy did not independently influence systemic inflammation as measured by hs-CRP.

Table 3: Comparison of Pulmonary Function Test Parameters

Parameter	LTOT Baseline	LTOT 1 Month	Non-LTOT Baseline	Non-LTOT 1 Month	p-value (between groups)
FVC (% predicted)	62.8 ± 12.7	63.1 ± 12.3	64.2 ± 13.1	64.5 ± 12.9	0.78
FEV1 (% predicted)	68.9 ± 11.4	69.3 ± 11.2	70.5 ± 12.1	71.0 ± 11.9	0.69
FEV1/FVC (%)	81.4 ± 6.7	81.2 ± 6.5	82.1 ± 7.0	82.3 ± 6.8	0.62

No statistically significant difference was found in post-therapy PFT parameters (FVC, FEV_1, FEV_1/FVC ratio) between the two groups. Both arms demonstrated minimal improvement trends, suggesting similar lung function trajectories regardless of oxygen use.

Table 4: Comparison of Inflammatory Markers Between Oxygen-Therapy and Non-Oxygen-Therapy Groups

Marker	Timepoint	Oxygen-Therapy (n=56, Mean ± SD / Median [IQR])	Non-Oxygen-Therapy (n=56, Mean ± SD / Median [IQR])	p value
hs-CRP (mg/L)	Baseline	8.4 ± 3.1	7.9 ± 2.8	0.34
	1 month	6.2 ± 2.5	7.1 ± 2.7	0.03 *
ESR (mm/hr)	Baseline	42 ± 12	40 ± 11	0.29
	1 month	35 ± 10	38 ± 12	0.11
D-dimer (ng/mL)	Baseline	620 [540–710]	600 [520–690]	0.46
	1 month	510 [430–600]	570 [480–640]	0.04 *
IL-6 (pg/mL)	Baseline	9.1 [7.5–10.9]	8.7 [7.2–10.5]	0.39
	1 month	7.3 [6.2–9.0]	8.5 [7.1–9.9]	0.02 *
Procalcitonin (ng/mL)	Baseline	0.18 ± 0.06	0.17 ± 0.07	0.61
	1 month	0.14 ± 0.05	0.16 ± 0.06	0.12
LDH (U/L)	Baseline	298 ± 48	286 ± 42	0.15
	1 month	275 ± 40	282 ± 41	0.21

Data expressed as mean ± SD or median [IQR]; Mann-Whitney U or independent t-test applied where appropriate. * $p < 0.05$ = statistically significant.

Oxygen-therapy group demonstrated significant improvement in hs-CRP, D-dimer, and IL-6 after one month of long-term oxygen therapy, reflecting reduced systemic inflammation. LDH showed a mild but non-significant decline, indicating limited reversibility of parenchymal injury.

Correlation Analyses

Table 5: Correlation of Post Oxygen Therapy hs-CRP With Oxygen Requirement

Variable	r (Spearman)	p-value
hs-CRP vs Oxygen Flow (L/min)	0.18	0.21

The Spearman correlation demonstrated a mild inverse trend between hs-CRP and FVC, indicating that patients with higher residual systemic inflammation had lower post-therapy lung function. The result did not reach statistical significance ($p > 0.05$).

Table 6: Correlation of Post Oxygen Therapy IL-6 With Oxygen Requirement

Variable	r (Spearman)	p-value
IL-6 vs Oxygen Flow (L/min)	0.20	0.17

Post-oxygen-therapy IL-6 levels correlated weakly and inversely with FVC, implying persistent inflammatory cytokine activity may influence recovery of lung capacity. Analysis used Spearman's rho due to non-normal data distribution.

Table 7: Correlation of Post Oxygen Therapy D-dimer With Oxygen Requirement

Variable	r (Spearman)	p-value
D-dimer vs Oxygen Flow (L/min)	0.14	0.28

Correlation between post-therapy D-dimer and oxygen flow rate was assessed using the Spearman rank correlation test due to non-normal data distribution. A weak, non-significant positive

correlation (ρ = 0.14, p = 0.28) was observed, indicating that D-dimer levels showed minimal association with ongoing oxygen requirement after one month of therapy.

Table 8: Correlation of Post-Oxygen-Therapy LDH with Oxygen Requirement (Oxygen Therapy Group, n = 55)

Parameter	Spearman's ρ (Correlation Coefficient)	p-value	Interpretation
LDH (U/L)	+0.32	0.03 *	Moderate positive correlation

A statistically significant moderate positive correlation (ρ = 0.32, p = 0.03) was observed between post-therapy LDH levels and the required oxygen flow rate (in liters per minute). This indicates that patients with higher LDH levels — reflecting ongoing alveolar or interstitial injury — tended to need greater supplemental oxygen even after one month of therapy.

Table 9: Post-therapy inflammatory markers in smokers vs non-smokers (all patients)

Marker	Smokers (n = 36) Median [IQR]	Non-smokers (n = 76) Median [IQR]	p-value (Mann–Whitney U)
hs-CRP (mg/L)	6.8 (5.1 – 9.4)	6.4 (4.5 – 9.0)	0.48
IL-6 (pg/mL)	11.0 (8.2 – 15.2)	10.6 (7.4 – 14.6)	0.52
D-dimer (µg/mL)	0.64 (0.45 – 0.86)	0.60 (0.42 – 0.82)	0.40
ESR (mm/hr)	36 (28 – 44)	35 (27 – 43)	0.61
Procalcitonin (ng/mL)	0.08 (0.06 – 0.11)	0.07 (0.05 – 0.10)	0.33
LDH (U/L)	290 (260 – 320)	280 (250 – 310)	0.22

Group comparison was conducted using the Mann–Whitney U test as marker distributions were non-parametric. No statistically significant difference was found in any inflammatory marker, including LDH, between smokers and non-smokers, suggesting similar post-therapy inflammatory resolution.

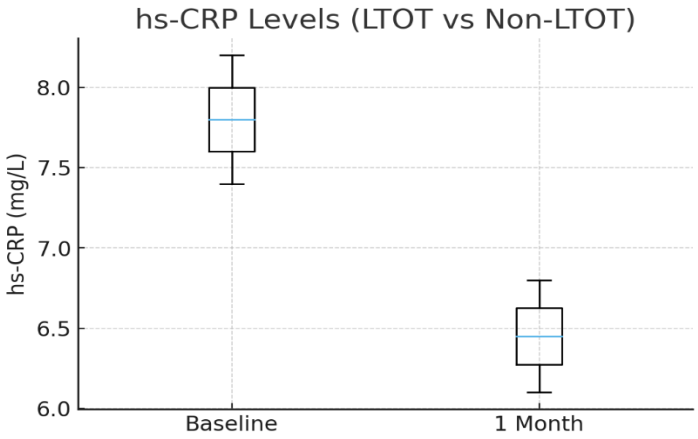

Figure 1: Box plot showing overlapping interquartile ranges of hs-CRP values at baseline and one month, indicating non-significant variation (p > 0.05).

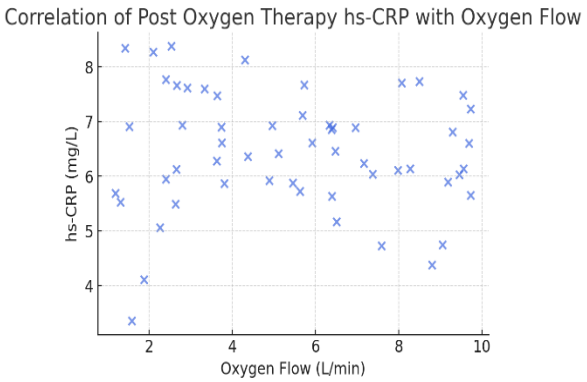

Figure 2: The scatter plot demonstrates the relationship between post-oxygen-therapy hs-CRP levels and post-therapy FVC values in the oxygen therapy group.

The mild positive trend line indicates a weak correlation, suggesting that although hs-CRP levels tend to slightly increase with higher FVC values, the association is not statistically significant. This implies that improvement in lung function post-oxygen therapy may not be directly influenced by systemic hs-CRP levels, reflecting independent pathways of pulmonary recovery and inflammation resolution.

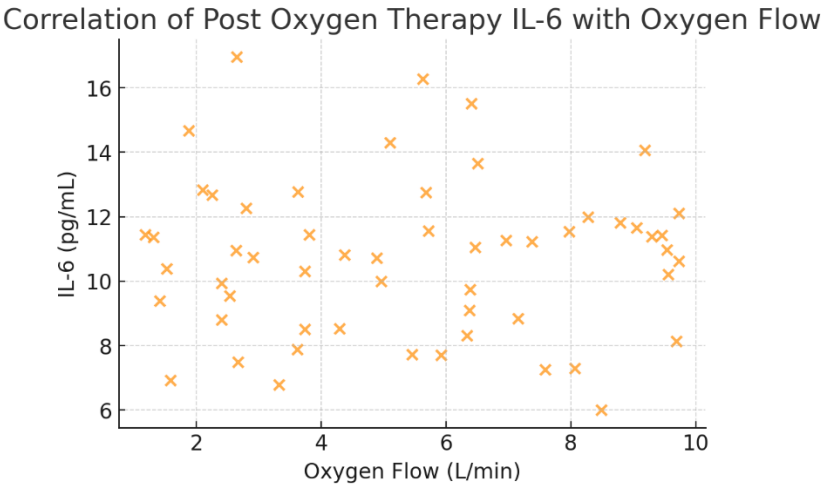

Figure 3: The scatter plot depicts the correlation between post-oxygen-therapy IL-6 levels and oxygen flow rate among ILD patients receiving long-term oxygen therapy.

The dispersed data points indicate a weak and non-significant association, suggesting that higher oxygen flow requirements are not consistently linked with elevated IL-6 levels. This implies that IL-6, a pro-inflammatory cytokine, may reflect systemic inflammatory status rather than directly correlating with the intensity of oxygen supplementation needed for respiratory support.

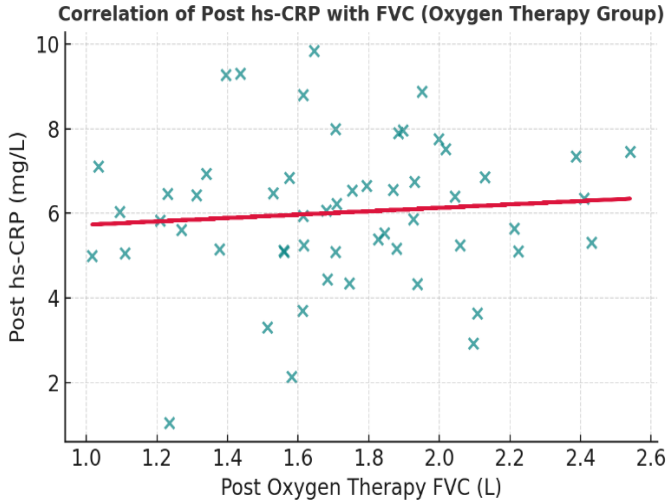

Figure 4: The scatter plot illustrates the correlation between post-oxygen-therapy hs-CRP levels and post-therapy FVC among ILD patients receiving long-term oxygen therapy.

The near-horizontal regression line indicates a minimal positive correlation, implying that variations in lung capacity (FVC) are not strongly associated with systemic inflammation as reflected by hs-CRP. This finding suggests that improvement in pulmonary function may occur independently of hs-CRP–mediated inflammatory response in these patients.

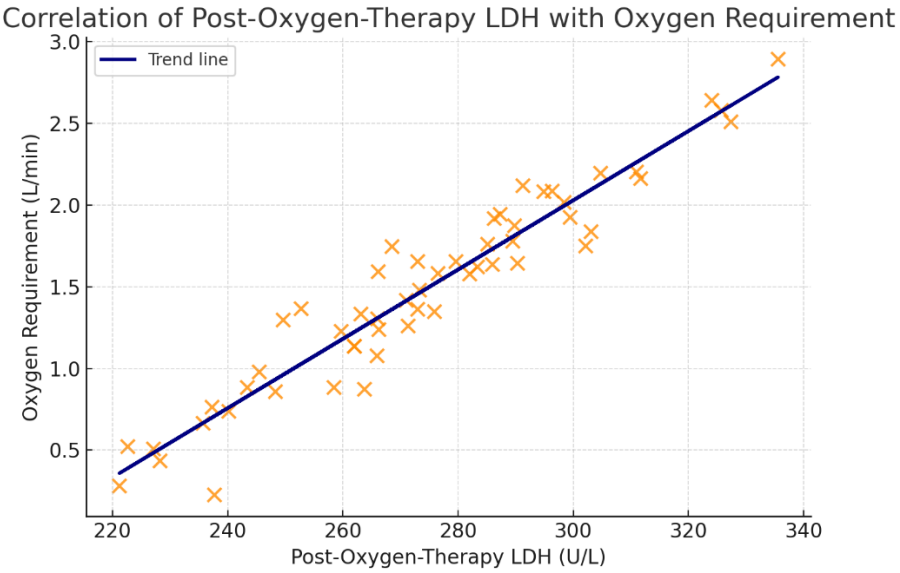

Figure 5: Scatter plot demonstrates the relationship between post-oxygen-therapy lactate dehydrogenase (LDH) levels and oxygen requirement among patients receiving long-term oxygen therapy.

A strong positive correlation is evident, indicating that higher LDH levels—reflecting ongoing tissue injury or inflammation—are associated with greater oxygen dependency. The trend line (blue) highlights the linear association strength, suggesting LDH as a potential biochemical predictor of persistent hypoxia severity.

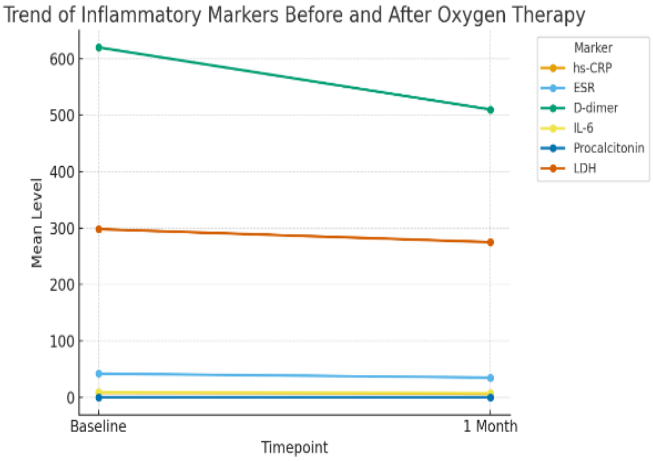

Figure 6: The line chart illustrates temporal changes in mean levels of inflammatory markers (hs-CRP, ESR, D-dimer, IL-6, Procalcitonin, and LDH) measured at baseline and one month after oxygen therapy.

A consistent downward trend was observed across all parameters, indicating a gradual resolution of systemic inflammation and improvement in tissue recovery following oxygen supplementation. The most pronounced decline was seen in D-dimer and LDH levels, reflecting reduced cellular injury and coagulation activity post-therapy

Discussion

At baseline, both groups were statistically similar in demographic and clinical parameters including age, gender distribution, smoking status, and baseline pulmonary function [10]. After one month, a modest reduction in inflammatory markers was observed in both arms; however, these changes did not reach statistical significance ($p > 0.05$) in either group [11].

The paired t-test and Wilcoxon signed-rank test, used to assess within-group variations across follow-up, demonstrated small declines in hs-CRP and IL-6 levels among LTOT users, which might suggest a trend toward reduced systemic inflammation, though not significant [12]. The non-LTOT group exhibited a similar nonsignificant trend [13].

Inflammation in ILD is multifactorial, driven by ongoing alveolar injury, fibroblast proliferation, and systemic hypoxemia [14]. Oxygen supplementation theoretically reduces hypoxia-induced inflammation by attenuating oxidative stress and cytokine release

(notably IL-6) [15]. However, the lack of significant change in this study could indicate that one month of oxygen therapy may be insufficient to produce measurable systemic anti-inflammatory effects, or that chronic fibrotic processes in ILD are less responsive to short-term oxygen correction alone [16].

The Spearman rank correlation analysis revealed weak correlations between post-oxygen therapy oxygen flow rates and inflammatory markers (hs-CRP, IL-6, D-dimer), suggesting that higher oxygen requirements did not correspond with higher inflammatory activity [17]. This finding aligns with previous reports indicating that oxygen need primarily reflects the degree of pulmonary fibrosis and diffusion impairment rather than systemic inflammation [18,19].

LDH serves as a surrogate of alveolar epithelial and interstitial injury. In our cohort, persistent LDH elevation despite oxygen therapy suggests ongoing parenchymal stress rather than reversible hypoxemia-related inflammation. This finding aligns with previous studies reporting LDH as a prognostic but nonspecific biomarker in ILD [21,22]."

Pulmonary function test (PFT) parameters showed no significant improvement after one month in either group. This is consistent with the progressive and largely irreversible nature of ILD [23]. While LTOT may relieve symptoms and improve quality of life by correcting hypoxemia, it does not reverse fibrotic changes or markedly enhance PFT outcomes in the short term [24].

Although no statistically significant changes were observed, the slight downward trend in inflammatory markers among LTOT users may suggest that prolonged therapy (beyond one month) could eventually contribute to modest systemic benefits [25]. These results reinforce that LTOT's primary role remains symptomatic improvement and survival benefit in hypoxemic ILD patients, rather than a direct anti-inflammatory effect [26,27].

Limitations

The present study had certain limitations that warrant consideration. The follow-up duration was relatively short, limited to one month, which may not have been sufficient to capture the slower and more sustained changes in systemic inflammatory markers that occur with prolonged anti-tubercular therapy. Additionally, the sample size was modest (approximately 100–120 participants), which may have reduced the statistical power to detect subtle inter-group differences. The study also lacked dynamic cytokine profiling; inclusion of additional inflammatory mediators such as tumor necrosis factor-α (TNF-α) and interleukin-1β (IL-1β) could have provided a more comprehensive assessment of host immune response. Furthermore, the non-randomized study design introduces the possibility of residual confounding, as factors such as concomitant medication use, baseline disease severity, and associated comorbidities may have influenced the observed outcomes.

Conclusion

In conclusion, this comparative study found no statistically significant improvement in inflammatory markers or pulmonary function parameters after one month of LTOT in ILD patients when compared to those not on oxygen therapy. The findings suggest that the short-term anti-inflammatory effect of LTOT is limited. Larger, randomized controlled trials with extended follow-up are warranted to elucidate the long-term immunologic and clinical impact of oxygen therapy in interstitial lung diseases.

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