UAPM
Journal of Respiratory Diseases
and Allied Sciences

Original Article

Pulmonary Sequelae of Moderate to Severe Atypical Pneumonia Caused by SARS - COV-2 Virus

Rakesh Kumar Serawat, Neeraj Gupta, Ramakant Dixit, Piyush Arora*, Ranjeet Meghwanshi, Siddarth Mathur

Department of Respiratory Medicine, Jawahar Lal Nehru Medical College, Ajmer, Rajasthan, India

Abstract

Background: Survivors of moderate to severe COVID-19 pneumonia frequently exhibit persistent pulmonary abnormalities, but the longitudinal radiological and biochemical evolution remains unclear.

Methods: This observational study included 50 patients with moderate to severe COVID-19 pneumonia. Clinical symptoms, inflammatory markers, and serial chest CT scans were evaluated from day 1 to 180. CT patterns and severity scores were analyzed to assess temporal changes and post-COVID pulmonary sequelae.

Results: The mean age was 52.4 ± 13.4 years, with male predominance (62%). Acute symptoms resolved within 30 days; however, breathlessness and fatigue persisted in 74% and 40% of patients at 6 months. CT imaging showed progressive resolution of ground-glass opacities and consolidation, while fibrotic-like changes, particularly parenchymal bands, reticulations, and traction bronchiectasis, increased over time, despite a significant decline in CT severity score (16.24–4.84). Inflammatory biomarkers normalized earlier than radiological findings, indicating persistent structural remodeling.

Conclusion: Moderate to severe COVID-19 pneumonia is associated with lasting fibrotic-like lung sequelae, highlighting the need for structured long-term follow-up.

Keywords: COVID-19 pneumonia, Post-COVID lung sequelae, Chest CT, Pulmonary fibrosis, Parenchymal bands, Traction bronchiectasis.

Introduction

Coronavirus disease 2019 (COVID-19) is a respiratory tract infection caused by the novel SARS-CoV-2 virus, first identified in Wuhan, China, in December 2019.[1] Genetic sequencing confirmed it as a β-coronavirus closely related to SARS-CoV. On January 30, 2020, the WHO declared COVID-19 the sixth Public Health Emergency of International Concern.

SARS-CoV-2 spreads mainly through respiratory droplets, aerosols, or direct contact. The virus binds to angiotensin-converting enzyme-2 (ACE-2) receptors, abundantly expressed on pulmonary and nasal epithelial cells, and enters via endocytosis or membrane fusion.[2] The infectious period extends up to 10 days in mild to moderate cases and up to 20 days in severe or immunocompromised individuals.

Typical symptoms include cough, fever, anosmia, and ageusia, with others such as myalgia, sore throat, diarrhea, or dyspnoea in moderate to severe disease.[3] Chest CT commonly shows bilateral, multilobar ground-glass opacities with or without consolidation, interlobular septal thickening, mosaic attenuation, and tractio bronchiectasis; cavitation, effusion, and lymphadenopathy are less frequent.[4]

While most cases are mild, around 14% develop severe illness requiring oxygen support and 5% need intensive care. Complications include ARDS, sepsis, multiorgan failure, and cardiac or renal injury. Advanced age, comorbidities,

Address for correspondence: Piyush Arora,
Department of Respiratory Medicine, Jawahar Lal Nehru Medical College, Ajmer, Rajasthan, India
E-mail: piyusharora23@gmail.com

Access this article online

Quick Response Code

Website: uapmjournal.in

DOI: 10.70192/v3.i2.02

How to cite this article: Serawat RK, Gupta N, Dixit R, Arora P, Meghwanshi R, Mathur S. Pulmonary Sequelae of Moderate to Severe Atypical Pneumonia Caused by SARS - COV-2 Virus. UAPM J. Respiratory Diseases Allied Sci. 2026;3(2):7-14.
Received: 30-03-2026, **Accepted:** 10-05-2026, **Published:** 15-06-2026

high SOFA score, and elevated D-dimer (>1 µg/L) are key mortality predictors.

Management includes supportive and pharmacologic therapies such as corticosteroids, antivirals, antibiotics, antifibrotics, anticoagulants, and oxygen or ventilatory support (HFNO, BiPAP, or invasive ventilation) tailored to disease severity.

Materials And Methods

This study was conducted to evaluate the prevalence and severity of post–COVID-19 pulmonary sequelae. A total of fifty patients diagnosed with COVID-19 pneumonia were enrolled and followed up for a period of 180 days. Both hospitalized and domiciliary cases who developed respiratory symptoms and demonstrated computed tomographic abnormalities during the course of illness were included. Detailed demographic information, clinical presentation, radiological findings, hematological parameters, inflammatory markers, and treatment modalities were serially recorded and analyzed throughout the study duration.

High-resolution computed tomography (HRCT) of the chest served as the primary assessment tool for evaluating parenchymal involvement. The extent of lung involvement was graded using the semi-quantitative CT severity scoring system proposed by Pan et al.[5], in which each lobe was scored from 0 to 5 depending on the percentage of affected parenchyma (0 = no involvement; 1 = <5%; 2 = 5–25%; 3 = 26–50%; 4 = 51–75%; and 5 = >75%). The cumulative score represented the overall severity of pulmonary involvement.

Patients with pre-existing fibrotic lung diseases such as interstitial lung disease, previously treated or active tuberculosis, bronchiectasis, pleural pathology, or chest wall deformities were excluded from the study. Those lacking serial biochemical and inflammatory marker data at defined intervals or unwilling to provide informed consent were also excluded. The study protocol was approved by the Institutional Ethics Committee, and all participants provided written informed consent prior to inclusion.

Results

A total of fifty patients were included in the study, with a mean age of 52.4 ± 13.4 years (range 22–78 years), as shown in Table 1. The majority of participants (52%) belonged to the 51–70 year age group, and males constituted 62% (n = 31) of the study population. This demographic distribution reflects the typical pattern seen in moderate to severe cases of COVID-19 pneumonia, where middle-aged and elderly males are more frequently affected.

At baseline, the most common presenting symptoms were fever (90%), breathlessness (78%), and cough (74%), followed by fatigue (64%), sore throat (34%), and loss of smell or taste (20%). Over the 6-month follow-up period, acute symptoms such as fever, sore throat, and anosmia resolved completely by day 30, whereas breathlessness and fatigue persisted in 37% and 20% of patients, respectively, at day 180 (Table 2). The decline in fever and upper-respiratory symptoms was highly significant (p <0.001), while fatigue showed gradual improvement (p = 0.004). In contrast, the persistence of breathlessness did not show a statistically significant change (p = 1.000). These findings suggest that, although acute infectious symptoms subsided early, many patients experienced lingering post-viral functional limitations characterized by exertional dyspnoea and generalized fatigue.

Out of the total cohort, 86% were RT-PCR positive for SARS-CoV-2, and 56% required supplemental oxygen at the time of hospital discharge, while 44% were discharged on

Table 1: Baseline demographic characteristics and presenting clinical symptoms of patients with moderate to severe COVID-19 pneumonia (N = 50)

Parameter	*Category*	*Number (n)*	*Percentage (%)*
Age group (years)	<30	2	4
	30–40	10	20
	41–50	9	18
	51–60	12	24
	61–70	14	28
	>70	3	6
Mean age ± SD (years)	—	52.36 ± 13.44	—
Gender	Male	31	62
	Female	19	38
Presenting symptoms	Fever	45	90
	Breathlessness	39	78
	Cough	37	74
	Fatigue	32	64
	Sore throat	17	34
	Loss of smell/ taste	10	20

Table 2: Temporal evolution of clinical symptoms in patients with moderate to severe COVID-19 pneumonia over 180 days, showing the proportion of patients with persistent symptoms at each follow-up visit and corresponding statistical significance (p values)

Symptom	*Day 1 (%)*	*Day 30 (%)*	*Day 180 (%)*	*p-value*
Cough	74	32	26	<0.001
Fever	90	0	0	<0.001
Breathlessness	78	94	74	NS
Sore throat	34	6	0	<0.001
Fatigue	64	92	40	0.004
Loss of smell/ taste	20	6	0	0.002

Table 3: Serial evolution of CT chest findings and distribution of pulmonary opacities in patients with moderate-to-severe COVID-19 pneumonia followed up to 180 days. Early inflammatory patterns (GGO, consolidation, crazy-paving) showed significant regression, while fibrotic changes (parenchymal bands, reticulations, traction bronchiectasis) progressively increased. The mean CT severity score declined from 16.24 ± 3.17 on Day 1 to 4.84 ± 2.85 on Day 180, indicating radiologic improvement with residual fibrotic sequelae.

Parameter	*Day 1 (%)*	*Day 30 (%)*	*Day 180 (%)*	*p-value*
Lobar distribution				
Upper lobe involvement	82	36	14	—
Middle/lingula involvement	98	82	46	—
Lower lobe involvement	100	100	100	—
Bilateral disease	100	100	100	—
Peripheral predominance	34	64	88	—
CT opacities				
Ground-glass opacities	70	68	52	NS
GGO with consolidation	30	28	0	0.001
Crazy paving	32	18	0	<0.001
Septal thickening	82	70	82	NS
Parenchymal bands	16	76	100	<0.001
Reticulations	0	8	56	<0.001
Traction bronchiectasis	0	4	64	<0.001
Mosaic attenuation	0	8	12	0.014
CT severity score (Mean ± SD)	16.24 ± 3.17	10.74 ± 6.27	4.84 ± 2.85	—

room air. This oxygen dependency at discharge supports the inclusion of predominantly moderate-to-severe cases in the study population.

Serial high-resolution CT chest evaluations demonstrated a clear transition from acute exudative lesions to fibrotic-like sequelae over 180 days, as shown in Table 3. Ground-glass opacities and consolidation, which were observed in 70% and 30% of patients, respectively, at baseline, showed marked regression to 52% and 0% by Day 180 ($p = 0.001$). The crazy-paving pattern, initially present in 32% of cases, resolved completely ($p < 0.001$). In contrast, structural abnormalities consistent with fibrotic remodeling showed a progressive increase during follow-up. Parenchymal bands were observed in 16% of patients initially and became universally present (100%) by day 180 ($p < 0.001$). Similarly, reticulations increased from 0% to 56%, and traction bronchiectasis from 0% to 64% over the same period (both $p < 0.001$). Less common late findings included mosaic attenuation in 12% of patients, atelectasis in 6%, cavitation in 6%, bulla formation in 4%, and fungal ball in 2%. These radiological trends collectively indicate near-complete resolution of acute inflammatory lesions with subsequent development of post-inflammatory fibrotic-like changes, representing the healing phase of organizing pneumonia and post-COVID fibrosis.

Table 4: Temporal relationship between declining CT severity score and increasing prevalence of parenchymal bands on serial follow-up CT scans, demonstrating progressive fibrotic-like remodeling despite reduction in overall disease burden.

Follow-up day	*Mean CT severity score*	*Patients with parenchymal bands, n (%)*
Day 1	16.24 ± 3.17	8 (16%)
Day 7	15.82 ± 3.84	9 (18%)
Day 14	13.52 ± 5.01	13 (26%)
Day 30	10.74 ± 6.27	38 (76%)
Day 60	7.42 ± 4.69	45 (90%)
Day 180	4.84 ± 2.85	50 (100%)

An analysis of lobar and axial involvement revealed that lower lobe disease persisted in all patients throughout follow-up, while upper and middle lobe involvement showed progressive resolution. Bilateral involvement was universal at all stages. Peripheral distribution of opacities increased significantly from 34% on day 1 to 88% by day 180, in contrast to the stable bilateral pattern, which is consistent with the recognized basilar and subpleural predominance of post-COVID organizing pneumonia.

The mean CT severity score decreased steadily from 16.24 ± 3.17 on day 1 to 4.84 ± 2.85 on Day 180, indicating a substantial reduction in overall disease burden. Interestingly, this decline in severity score correlated inversely with the increase in parenchymal bands, suggesting that although the quantitative extent of inflammation diminished, residual structural remodeling continued to progress (Table 4).

Table 5: Association of major treatment modalities with CT chest findings at Day 180. Antibiotics, antivirals, corticosteroids, and supportive oxygen therapies (BiPAP, HFNO, NAC) were linked to resolution of inflammatory lesions (GGO, consolidation, crazy-paving), whereas fibrotic changes (parenchymal bands, reticulations, traction bronchiectasis) persisted across all groups. Antifibrotic therapy showed no clear preventive effect on residual fibrosis.

Treatment modality	*N*	*GGOs (%)*	*Septal thickening (%)*	*Parenchymal bands (%)*	*Traction bronchiectasis (%)*	*Other findings**
Antibiotics	50	52	82	100	64	Occasional reticulation, mosaic attenuation
Antivirals	50	52	82	100	64	Similar to antibiotic group
Corticosteroids	50	52	82	100	64	No protective effect on fibrosis
Anticoagulants	50	52	82	100	64	No structural modification
Antifibrotics	45	51	82	11	53	No significant reduction in fibrotic markers
BiPAP	22	50	81	100	54	Reflects severe disease subgroup
HFNO	11	45	72	100	72	Higher residual fibrosis
NAC	42	52	80	100	47	No impact on structural sequelae

Table 6: Temporal trends of hematological and inflammatory markers (CBC and N:L ratio) correlated with CT scan findings in COVID-19 pneumonia. CBC counts and N:L ratios showed a sharp rise from Day 1 to Day 7, corresponding to peak systemic inflammation, followed by gradual normalization by Day 180. Despite the decline in serum parameters, radiologic abnormalities—particularly parenchymal bands, reticulations, and traction bronchiectasis persisted, indicating ongoing structural lung remodeling even after biochemical recovery.

	CT pattern	*Day 1*	*Day 7*	*Day 14*	*Day 30*	*Day 60*	*Day 180*
CBC	GGOs	8.36 ± 5.61	15.01 ± 4.83	12.55 ± 4.08	9.49 ± 3.56	8.01 ± 2.92	8.68 ± 3.39
	GGO + consolidation	6.81 ± 4.21	13.13 ± 3.11	13.43 ± 4.51	9.99 ± 4.31	6.63 ± 1.20	—
	Septal thickening	8.97 ± 5.97	14.25 ± 4.54	13.01 ± 4.48	9.75 ± 3.88	7.73 ± 2.72	8.36 ± 2.67
	Crazy paving	9.08 ± 7.47	15.28 ± 4.03	12.74 ± 3.97	10.03 ± 1.62	9.65 ± 0.21	—
	Parenchymal bands	6.61 ± 3.49	12.82 ± 2.76	14.06 ± 4.21	9.67 ± 4.00	8.29 ± 3.12	8.47 ± 2.93
	Traction bronchiectasis	—	—	—	16.50 ± 6.36	8.17 ± 3.39	8.67 ± 3.27
	Reticulations	—	—	—	10.73 ± 6.54	9.00 ± 3.87	8.62 ± 2.82
N:L ratio	GGOs	7.77 ± 5.45	10.82 ± 7.07	8.31 ± 8.31	4.57 ± 4.47	2.90 ± 2.87	2.66 ± 2.49
	GGO + consolidation	6.89 ± 4.26	10.86 ± 6.14	4.58 ± 4.58	3.56 ± 2.16	1.12 ± 0.48	—
	Septal thickening	7.47 ± 5.67	10.65 ± 6.54	9.68 ± 9.68	4.27 ± 3.77	2.52 ± 2.48	2.02 ± 1.91
	Crazy paving	8.98 ± 7.32	9.20 ± 6.73	5.94 ± 5.94	3.48 ± 1.82	2.25 ± 0.07	—
	Parenchymal bands	9.73 ± 4.56	9.02 ± 4.45	8.83 ± 8.83	4.53 ± 4.16	2.71 ± 2.49	2.08 ± 1.94
	Traction bronchiectasis	—	—	8.52 ± 8.52	5.75 ± 3.18	3.32 ± 3.39	2.35 ± 2.35
	Reticulations	—	—	5.03 ± 5.03	8.43 ± 6.66	3.28 ± 3.44	2.55 ± 2.45

Initially, parenchymal bands were observed more frequently among hypertensive and diabetic patients; however, this association was not significant at the end of 6 months. Smoking and alcohol consumption were found to have a positive correlation with the persistence of parenchymal bands. Evaluation of therapeutic interventions showed that antibiotics, antivirals, and corticosteroids were significantly associated with faster resolution of ground-glass opacities, consolidation, and crazy-paving patterns ($p < 0.001$) (Table 5). However, antifibrotic agents such as nintedanib and pirfenidone did not prevent the development of parenchymal bands, and supportive therapies including BiPAP, HFNO, and N-acetylcysteine (NAC) were effective only in reducing inflammatory lesions without influencing long-term fibrotic outcomes.

Serial assessment of biochemical and inflammatory markers, including complete blood counts, neutrophil-to-lymphocyte ratio (N:L), C-reactive protein (CRP), D-dimer, ferritin, interleukin-6 (IL-6), and lactate dehydrogenase (LDH), revealed a characteristic temporal pattern as shown in Tables 6 to 8. Most parameters exhibited an initial rise from Day 1 to Day 7, followed by a steady decline from Day 14 to Day 180, reflecting progressive resolution of systemic inflammation. Despite normalization of these serum biomarkers, radiological evaluation continued to demonstrate persistent fibrotic-like changes, highlighting a

Table 7: Trends in inflammatory biomarkers (CRP and D-dimer) correlated with CT findings over 180 days in moderate-to-severe COVID-19 pneumonia. Both CRP and D-dimer peaked around Day 7, mirroring maximal inflammatory and thrombotic activity during the acute phase. Progressive decline was observed by Day 30, with near-normalization by Day 180. Persistent structural changes—such as parenchymal bands, reticulations, and traction bronchiectasis—were noted despite biochemical recovery, reflecting incomplete resolution and possible post-inflammatory fibrosis.

Investigation	*CT pattern*	*Day 1*	*Day 7*	*Day 14*	*Day 30*	*Day 60*	*Day 180*
CRP	GGOs	53.58 ± 29.59	75.81 ± 21.57	31.01 ± 16.25	16.68 ± 11.90	15.37 ± 18.48	11.58 ± 6.79
	GGO + consolidation	60.47 ± 32.96	74.89 ± 28.46	36.30 ± 18.27	16.07 ± 8.07	20.67 ± 25.42	—
	Septal thickening	52.59 ± 30.94	74.69 ± 26.00	32.19 ± 16.22	15.11 ± 11.44	14.28 ± 16.69	10.93 ± 5.68
	Crazy paving	51.00 ± 20.68	75.50 ± 19.71	31.77 ± 14.38	15.33 ± 7.84	6.00 ± 0.00	—
	Parenchymal bands	60.38 ± 36.40	75.67 ± 25.60	36.31 ± 16.87	16.97 ± 11.74	14.03 ± 17.17	10.90 ± 5.47
	Mosaic attenuation	60.38 ± 36.40	—	—	10.75 ± 2.50	10.25 ± 1.26	9.17 ± 3.76
	Traction bronchiectasis	—	—	—	42.50 ± 38.89	16.11 ± 19.87	10.84 ± 6.38
	Reticulations	—	—	—	17.00 ± 11.37	15.08 ± 17.25	11.29 ± 6.78
D- dimer	GGOs	1.33 ± 1.33	2.42 ± 1.72	1.04 ± 0.72	0.61 ± 0.69	0.54 ± 0.99	0.27 ± 0.10
	GGO + consolidation	1.67 ± 1.98	2.53 ± 2.08	1.26 ± 1.30	1.19 ± 2.35	0.37 ± 0.16	—
	Septal thickening	1.36 ± 1.35	2.44 ± 1.77	1.08 ± 0.94	0.89 ± 1.61	0.50 ± 0.83	0.31 ± 0.16
	Crazy paving	0.93 ± 0.84	1.89 ± 1.48	0.95 ± 0.96	0.33 ± 0.22	0.44 ± 0.08	—
	Parenchymal bands	1.49 ± 1.60	2.58 ± 2.12	1.48 ± 1.41	0.89 ± 1.54	0.51 ± 0.81	0.31 ± 0.15
	Mosaic attenuation	1.49 ± 1.60	—	—	0.45 ± 0.13	0.36 ± 0.08	0.33 ± 0.12
	Traction bronchiectasis	—	—	—	5.05 ± 5.87	0.40 ± 0.19	0.30 ± 0.12
	Reticulations	—	—	—	0.82 ± 0.27	0.43 ± 0.20	0.27 ± 0.12

clear dissociation between systemic inflammatory recovery and ongoing pulmonary structural remodeling.

In summary, this study demonstrates that while biochemical and inflammatory markers normalize within weeks of recovery from acute infection, residual radiological abnormalities, particularly parenchymal bands, reticulations, and traction bronchiectasis, persist in a majority of patients for several months. Functional symptoms such as dyspnoea and fatigue tend to parallel these imaging findings. The persistence of fibrotic-like patterns despite resolution of inflammatory activity suggests that post-COVID-19 pulmonary sequelae represent a slowly resolving or potentially irreversible remodeling process that can continue to impact patient recovery and long-term lung function.

Discussion

SARS-CoV-2 primarily infects respiratory epithelial cells by binding to ACE-2 receptors, which are highly expressed on type II pneumocytes in the lung. Viral entry leads to an intense inflammatory response and immune activation that can cause diffuse alveolar damage (DAD), acute respiratory distress syndrome (ARDS), and organizing pneumonia. This cascade of pro-inflammatory cytokines (a "cytokine storm") contributes to acute lung injury and sets the stage for chronic structural changes in the lung parenchyma over time.[6,7]

In this observational cohort of 50 patients with moderate to severe COVID-19 pneumonia, the mean age was 52 years and the majority were male (62%), reflecting patterns seen in other post-COVID cohorts where severity and age predict outcomes. A significant proportion continued to require oxygen therapy at discharge, underlining the severity of pulmonary involvement during the acute stage. Although most acute symptoms such as fever, sore throat, and anosmia resolved within weeks, persistent symptoms like breathlessness (74%) and fatigue (40%) were still prevalent at six months, consistent with reported post-acute respiratory sequelae of COVID-19.[8]

Longitudinal chest CT imaging in our cohort showed a temporal evolution from acute inflammatory changes (ground-glass opacities, consolidation, and crazy-paving patterns) toward chronic structural abnormalities (parenchymal bands, reticulations, and traction bronchiectasis). The mean CT severity score improved significantly (from 16.2–4.8), yet many survivors retained persistent parenchymal abnormalities at six months. Similar long-term CT findings have been described in international follow-up studies, where a notable fraction of post-COVID patients exhibit persistent radiologic sequelae, including reticulation and traction bronchiectasis, months to years after infection.[9,10]

Risk factors such as diabetes, hypertension, smoking, and alcohol use have been implicated in more severe lung involvement and prolonged radiological changes following COVID-19, aligning with known factors that predispose to

Table 8: Trends in serum inflammatory and tissue injury biomarkers (Ferritin, IL-6, and LDH) correlated with CT findings over 180 days in moderate-to-severe COVID-19 pneumonia. All three markers showed peak elevation during the first week (Day 7), coinciding with maximal radiological disease activity (predominantly GGOs, consolidation, and septal thickening). A sharp decline followed by near-normalization was observed by Day 60–180. Despite biochemical recovery, imaging revealed persistence of fibrotic sequelae, parenchymal bands, reticulations, and traction bronchiectasis indicating structural remodeling and incomplete parenchymal restitution.

	CT pattern	*Day 1*	*Day 7*	*Day 14*	*Day 30*	*Day 60*	**Day 180**
Ferritin	GGOs	698.78 ± 526.75	817.42 ± 187.13	428.90 ± 77.19	77.14 ± 42.01	54.77 ± 22.95	62.86 ± 19.68
	GGO + consolidation	593.60 ± 367.96	1116.37 ± 723.40	430.35 ± 109.44	70.29 ± 11.34	44.67 ± 9.87	—
	Septal thickening	663.20 ± 512.34	956.54 ± 534.92	429.62 ± 99.11	75.45 ± 41.25	48.05 ± 18.02	56.71 ± 18.48
	Crazy paving	862.94 ± 750.22	834.00 ± 212.78	406.86 ± 100.38	73.00 ± 19.74	46.00 ± 16.97	—
	Parenchymal bands	846.63 ± 734.92	1072.33 ± 766.42	440.08 ± 54.56	78.81 ± 39.20	49.27 ± 20.76	57.15 ± 18.90
	Mosaic attenuation	846.63 ± 734.92	—	—	79.50 ± 17.54	49.75 ± 14.38	48.17 ± 21.93
	Traction bronchiectasis	—	—	—	60.50 ± 3.54	52.28 ± 23.05	59.51 ± 18.86
	Reticulations	—	—	—	73.73 ± 12.08	49.06 ± 23.50	58.58 ± 21.05
IL6	GGOs	69.22 ± 46.54	55.71 ± 13.99	21.28 ± 8.74	11.39 ± 5.78	12.44 ± 12.30	9.35 ± 5.57
	GGO + consolidation	86.46 ± 51.86	51.79 ± 21.87	21.83 ± 9.99	9.87 ± 6.79	9.00 ± 1.73	—
	Septal thickening	72.67 ± 46.51	55.62 ± 18.43	20.71 ± 10.13	10.61 ± 6.48	14.73 ± 16.36	10.54 ± 7.46
	Crazy paving	67.02 ± 40.67	52.96 ± 17.22	21.49 ± 9.19	11.22 ± 5.76	8.00 ± 2.83	—
	Parenchymal bands	85.13 ± 60.83	49.33 ± 27.60	20.00 ± 10.33	11.05 ± 6.20	14.89 ± 16.72	10.52 ± 7.25
	Mosaic attenuation	85.13 ± 60.83	—	—	5.43 ± 4.42	5.88 ± 2.25	6.33 ± 1.75
	Traction bronchiectasis	—	—	—	18.50 ± 13.44	13.15 ± 13.55	10.13 ± 6.68
	Reticulations	—	—	—	9.25 ± 5.50	18.63 ± 21.54	10.55 ± 6.24
LDH	GGOs	658.46 ± 286.47	791.06 ± 91.46	356.70 ± 41.56	245.62 ± 40.28	164.37 ± 38.05	162.54 ± 45.26
	GGO + consolidation	759.93 ± 298.81	745.42 ± 107.77	341.20 ± 34.80	258.21 ± 60.10	151.00 ± 14.93	—
	Septal thickening	659.54 ± 309.30	777.97 ± 93.45	355.88 ± 31.09	251.66 ± 46.57	160.51 ± 40.00	149.66 ± 37.16
	Crazy paving	670.13 ± 277.45	734.19 ± 112.99	340.86 ± 42.42	238.56 ± 44.64	182.50 ± 3.54	—
	Parenchymal bands	731.38 ± 274.18	850.00 ± 63.46	363.31 ± 31.14	251.82 ± 45.12	159.49 ± 40.01	151.30 ± 38.79
	Mosaic attenuation	731.38 ± 274.18	—	—	248.75 ± 30.65	176.00 ± 82.10	163.83 ± 63.20
	Traction bronchiectasis	—	—	—	327.50 ± 88.39	155.06 ± 45.88	150.19 ± 41.49
	Reticulations	—	—	—	243.00 ± 33.16	158.00 ± 43.49	151.21 ± 44.85

worse outcomes in acute viral pneumonia and fibrotic lung disease in general.[11] Importantly, although inflammatory biomarkers (e.g., CRP, IL-6, D-dimer, ferritin, neutrophil: lymphocyte ratio) peaked early in disease and normalized during recovery, CT abnormalities frequently persisted, pointing to a discordance between biochemical resolution and structural lung healing.[12]

From a therapeutic standpoint, acute management with corticosteroids, antivirals, and antibiotics appears to mitigate early inflammatory lung injury but does not reliably prevent chronic fibrotic-like changes. The role of antifibrotic agents such as pirfenidone and nintedanib in post-COVID pulmonary fibrosis remains under investigation, with current evidence not yet supportive of routine use in this context. Observational data and reviews suggest potential benefits of antifibrotic therapies based on analogies to idiopathic pulmonary fibrosis, but definitive trials are lacking.[13]

Supportive interventions, including high-flow nasal oxygen (HFNO), BiPAP, and mucolytic therapy (e.g., N-acetylcysteine), have shown value in improving oxygenation and symptom scores in the subacute period but have not been proven to alter long-term structural sequelae.

Overall, these data indicate that while inflammation subsides relatively quickly after acute COVID-19, parenchymal remodeling and fibrotic-like changes can persist or evolve independently of systemic inflammatory markers. Persistent CT abnormalities and functional limitations validate the need for structured post-discharge surveillance, including follow-up imaging, pulmonary function testing (e.g., spirometry and diffusion capacity), and enrollment in pulmonary rehabilitation programs. Early modification of risk factors (like smoking cessation and control of metabolic comorbidities) may also help mitigate the long-term impact of post-COVID lung disease.[14]

Strengths of this study include comprehensive serial imaging and correlation with biochemical markers; limitations include the small sample size, single-center design, and incomplete pulmonary function evaluation. Despite these constraints, the study contributes to the growing literature defining post-COVID lung sequelae as a distinct clinical entity requiring dedicated long-term management.

In summary, survivors of moderate to severe COVID-19 pneumonia demonstrate significant resolution of inflammatory lesions, yet persistent parenchymal bands, reticulations, and traction bronchiectasis suggest incomplete structural healing and early development of fibrotic patterns. Symptom burden often continues past biochemical recovery, underscoring the importance of multidisciplinary follow-up and research into targeted strategies to prevent chronic lung disease after COVID-19.

Conclusion

This study highlights that moderate-to-severe COVID-19 pneumonia leaves behind significant and long-lasting pulmonary sequelae despite biochemical recovery and clinical improvement. While acute inflammatory lesions such as ground-glass opacities, consolidation, and crazy-paving patterns resolve within weeks, a large proportion of patients develop residual structural abnormalities, most notably parenchymal bands, reticulations, and traction bronchiectasis by six months of follow-up. These fibrotic-like changes were bilateral, basilar, and peripherally distributed, suggesting post-infectious organizing pneumonia or early fibrotic remodeling rather than complete restitution of lung architecture.

Persistent fatigue and breathlessness paralleled these imaging findings, emphasizing that symptomatic and functional recovery lag behind radiological improvement. Among pharmacologic therapies, corticosteroids, antivirals, antibiotics, and supportive oxygen therapies were associated with faster clearance of acute inflammatory opacities, yet none prevented fibrotic transformation. Antifibrotic agents showed no measurable benefit in halting parenchymal band progression during the study period.

The overall trajectory from inflammation to remodeling underscores the importance of structured post-COVID follow-up, including clinical, spirometry, and radiologic assessment. Early initiation of pulmonary rehabilitation and risk modification for smoking and comorbidities may improve long-term outcomes. Larger multicentre studies and randomized trials are warranted to determine whether targeted antifibrotic or immunomodulatory therapy can alter the course of post-COVID lung remodeling and prevent progression to chronic interstitial fibrosis.

References

1. Zhu N, Zhang D, Wang W, Li X, Yang B, Song J, et al. A novel coronavirus from patients with pneumonia in China, 2019. N Engl J Med. 2020;382(8):727-33.
2. Hamming I, Timens W, Bulthuis MLC, Lely AT, Navis G, van Goor H. Tissue distribution of ACE2 protein, the functional receptor for SARS coronavirus: a first step in understanding SARS pathogenesis. J Pathol. 2004;203(2):631-37.
3. Huang C, Wang Y, Li X, Ren L, Zhao J, Hu Y, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. Lancet. 2020;395(10223):497-506.
4. Pan F, Ye T, Sun P, Gui S, Liang B, Li L, et al. Time course of lung changes on chest CT during recovery from 2019 novel coronavirus (COVID-19) pneumonia. Radiology. 2020;295(3):715-21.
5. Pan F, Ye T, Sun P, Gui S, Liang B, Li L, Zheng D, Wang J, Hesketh RL, Yang L, Zheng C. Time Course of Lung Changes at Chest CT during Recovery from Coronavirus Disease 2019 (COVID-19). Radiology. 2020; 295(3): 715-21.
6. Lamers MM, Mykytyn AZ, Breugem TI, Wang Y, Groen N, Rosu ME, et al. SARS-CoV-2 infection of human airway organoids reveals persistent basal-cell replication and epithelial reprogramming. Nat Rev Microbiol. 2022; 20(7):615-29.
7. Wang F, Kream RM, Stefano GB. Long-term respiratory and neurological sequelae of COVID-19. Med Sci Monit. 2020; 26: e928996.
8. Esendağli D, Yilmaz A, Akçay Ş, Özlü T. Post-COVID syndrome: pulmonary complications. Turk J Med Sci. 2021;51(Suppl 1):3359-71.
9. Kanne JP, Little BP, Schulte JJ, Haramati A, Haramati LB. Long-term lung abnormalities associated with COVID-19 pneumonia. Radiology. 2023;306(2): e221806.

10. Singh SJ, Baldwin MM, Daynes E, et al. Respiratory sequelae of COVID-19: pulmonary and extrapulmonary origins, and approaches to clinical care and rehabilitation. Lancet Respir Med. 2023;11(8):709-25.
11. Shen H, Zhang N, Liu Y, Yang X, He Y, Li Q, et al. The interaction between pulmonary fibrosis and COVID-19 and the application of related antifibrotic drugs. Front Pharmacol. 2021; 12: 805535.
12. Muthiah D, Vaddadi K, Liu L. Breathless aftermath: post-COVID-19 pulmonary fibrosis. Viruses. 2025;17(8):1098.
13. George PM, Wells AU, Jenkins RG. Pulmonary fibrosis and COVID-19: the potential role for antifibrotic therapy. Lancet Respir Med. 2020;8(8):807-15.
14. Duong-Quy S, Vo-Pham-Minh T, Tran-Xuan Q, Huynh-Anh T, Vo-Van T, Vu-Tran-Thien Q, Nguyen-Nhu V. Post-COVID-19 pulmonary fibrosis: facts, challenges and futures—a narrative review. Pulm Ther. 2023; 9(3): 295-307.