Monaldi Archives for Chest Disease

eISSN 2532-5264 https://www.monaldi-archives.org/

Monaldi Arch Chest Dis 2026 [Online ahead of print]

To cite this Article:
Samet M, Aghaei-Meybodi FA, Hosseini S, et al. **Lung autopsy findings in 44 COVID-19 deceased patients: pathological and radiological insights**. *Monaldi Arch Chest Dis* doi: 10.4081/monaldi.2026.3412

Submitted: 6-02-2025
Accepted: 18-02-2026

Lung autopsy findings in 44 COVID-19 deceased patients: pathological and radiological insights

Mohammad Samet,[1] Fatemeh Alsadat Aghaei-Meybodi,[1]
Sina Hosseini,[2] Abbas Meidany,[3] Azadeh Fateh[3]

[1]Department of Internal Medicine, Shahid Sadoughi Hospital, School of Medicine, Shahid Sadoughi University of Medical Sciences, Yazd; [2]Department of Internal Medicine, Shahid Sadoughi University of Medical Sciences, Yazd; [3]Shahid Sadoughi University of Medical Sciences, Yazd, Iran

Correspondence: Azadeh Fateh, Shahid Sadoughi University of Medical Sciences, Yazd, Iran. Tel. +989134061205 -. Email: azadeh.f89@gmail.com

Contributions: all authors have contributed significantly and agree with the content of the manuscript. The authors have read and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Conflict of interest: the authors declare that they have no competing interests, and all authors confirm accuracy.

Ethics approval and consent to participate: ethical approval was granted by the ethical committee of Shahid Sadoughi University of Medical Sciences, Yazd , Iran (IRB No: IR.SSU.MEDICINE.REC.1399.134).

Informed consent: written Informed consent was obtained from the patients' families. The manuscript does not contain any individual person's data in any form.

Patient consent for publication: not applicable.

Availability of data and materials: the datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Acknowledgments: the authors wish to thank all the staffs of the Department of Pulmonology, and Departments of Pathology and Radiology for their support.

Abstract

This study presents a detailed histopathological analysis of lung tissue from 44 deceased COVID19 patients, aiming to elucidate the mechanisms driving severe disease progression. Postmortem biopsies were systematically examined, revealing diffuse alveolar damage in 95.5% of cases, predominantly in the acute/exudative phase. Characteristic features included extensive hyaline membrane formation, alveolar septal thickening, fibrin deposition, and red blood cell extravasation. Notably, advanced fibrosis, indicative of ongoing tissue remodeling, was observed in 86.4% of cases, highlighting the chronic pathological impact of the disease. Patient demographics showed a predominance of older males with comorbidities such as hypertension and diabetes, aligning with known high-risk profiles. The methodology involved meticulous autopsy procedures and standardized histopathological assessments to ensure the reliability of findings.

This study provides key insight into histological changes in the lungs of COVID-19 patients, helping to clarify the disease's progression. It provides valuable insights that may contribute to a better understanding of long COVID and the potential long-term pulmonary complications in these patients. These findings may ultimately support improved management approaches and therapeutic strategies for COVID-19 care.

Key words: COVID-19, pulmonary autopsy, diffuse alveolar damage, acute respiratory distress syndrome, microthrombosis, pulmonary fibrosis.

Introduction

COVID-19 first emerged as pneumonia of unknown origin in China in December 2019 and rapidly spread worldwide. In May 2023, the World Health Organization (WHO) declared that COVID-19 was no longer a Public Health Emergency of International Concern [1,2].

COVID-19 shares pathological and clinical characteristics with SARS and MERS, particularly regarding lung involvement and comorbidity-driven disease severity [3-6]. However, it demonstrates lower mortality but substantially higher transmissibility [7]. Moreover, ACE2-mediated endothelial injury and a higher prevalence of alveolar microthrombi distinguish COVID-19 from classical ARDS and other viral pneumonias [4,8,9].

Long-term pulmonary complications, particularly fibrotic interstitial lung disease, may result from persistent viral components and unresolved acute lung injury [10,11]. Although mortality has declined with widespread vaccination, long-COVID still poses substantial clinical challenges (2). Histopathological evidence remains limited and is mostly based on lung explants or conventional autopsies, with only a few studies specifically addressing COVID-19–associated interstitial lung disease [12,13].

Autopsy-based approaches, including core needle necropsy, are crucial for understanding COVID-19–related lung pathology, which may improve clinical management and contribute to reducing mortality [12,14]. Therefore, this study aims to characterize lung pathology in 44 deceased patients with COVID-19 examined by core needle necropsy at a single center and to evaluate associated analytical, clinical, and radiological findings.

Materials and Methods

This retrospective descriptive-analytical study was conducted at Shahid Sadoughi Hospital (Yazd, Iran) between March 2020 and March 2022. The study included deceased patients with confirmed COVID-19 who had been admitted to the Intensive Care Unit (ICU).

Ethical approval was obtained from the Institutional Review Board of Shahid Sadoughi University of Medical Sciences (IRB No: IR.SSU.MEDICINE.REC.1399.134). Written informed consent was obtained from the patients' families.

Inclusion and exclusion criteria

Patients were included if they had a confirmed diagnosis of COVID-19 based on real-time polymerase chain reaction (RT-PCR) testing of nasopharyngeal or pulmonary samples and if death was attributed to COVID-19–related complications. Patients with concurrent bacterial or non–COVID-19 viral pneumonia or with inadequate tissue samples for pathological evaluation were excluded.

Data collection and sample preparation

Demographic data, medical history, comorbidities, and radiological findings were extracted from hospital records. Postmortem lung tissue samples were obtained through a small surgical incision in the anterior chest wall between the second and third intercostal spaces using sterile instruments. Samples were fixed in 10% formalin and processed for histopathological analysis.

Pathological and radiological analysis

Histopathological examinations were independently performed by two expert pathologists, focusing on fibrosis, hyaline membrane formation, necrosis, exudates, and microthrombosis. Discrepancies were resolved by consensus. Radiological findings were independently evaluated by two experienced radiologists using high-resolution computed tomography (HRCT) scans obtained during hospitalization.

Statistical analysis

Statistical analyses were performed using SPSS version 26. Continuous variables were expressed as mean ± SD and categorical variables as frequencies and percentages. Group comparisons were conducted using the Chi-square test, independent t-test, or ANOVA, as appropriate. A p-value <0.05 was considered statistically significant.

Results

Patient demographics and comorbidities

This descriptive-analytical study examined the pathology of lung biopsy specimens from 44 deceased COVID-19 patients. The patients comprised 29 males (65.9%) and 15 females (34.1%). Characteristics of the study sample are summarized in Table 1. Among these patients, 15 were smokers, and 14 had a history of opium use. The average age of the participants was 64.77 ± 14.29 years, with the majority being over 60 years old . The average duration of symptoms prior to death was 6 ± 1.6 days. Concerning underlying conditions, 25 patients (56.8%) had diabetes, 32 (72.7%) had hypertension, 9 (20.5%) had heart diseases, 3 (6.8%) had chronic respiratory diseases, and 6 (13.6%) had chronic kidney failure (Figures 1 and 2)

Radiological findings in COVID-19 patients

In terms of radiological findings, 18 cases (40.9%) exhibited consolidation, and 24 individuals (54.5%) presented with ground-glass lesions. All lesions were bilateral, and 95.5% of the cases showed widespread involvement. No significant gender differences were observed in radiological findings. Specifically, consolidation was present in 12 males and 6 females

(totaling 18 cases) (p-value = 0.93), and ground-glass lesions were observed in 15 males and 9 females (p-value = 0.6) (Table 2).

Pathological features of lung biopsy samples

Pathological examination revealed that out of the observed samples, 38 cases (86.4%) had fibrosis, granuloma formation was absent in all cases, and 24 cases (54.5%) had hyaline membrane formation. Other pathological findings included tissue necrosis (63.6%), alveolar exudate (95.5%), microthrombosis (27.3%), septal widening (75%), and extravasation of RBC (70.5%) (Figure 3) and some postmortem pathological findings of covid_19 patients are illustrated in Figure 4.

Gender differences in pathological findings

Two pathological findings, alveolar exudative and microthrombosis, showed significant differences between males and females (Figure 5). Alveolar exudative was more common in males (p-value = 0.044), and microthrombosis was more common in females (p-value = 0.038). No significant gender differences were observed for fibrosis, hyaline membrane formation, tissue necrosis, septal widening, or extravasation of RBCs.

Patterns of infiltration in lung tissue

Regarding the infiltration observed in the biopsy samples, predominantly lymphocytic and polymorphonuclear (PMN) infiltration was found in 26 cases (59.1%), while infiltration was absent in only 4 cases (9.1%).

Correlation between symptom duration and pathological changes

The duration of symptoms prior to hospitalization was analyzed based on pathological findings (Figure 6). Patients with tissue necrosis had an average symptom duration of 6.4 ± 1.38 days, and those with alveolar exudative changes had an average duration of 5.77 ± 1.3 days. Patients with microthrombosis had a longer average duration of 6.89 ± 1.9 days. Patients with septal widening had an average duration of 6 ± 1.72 days, and those with extravasation of RBCs had an average duration of 5.93 ± 1.59 days. Overall, cases with tissue necrosis and microthrombosis demonstrated a higher mean duration of symptoms before hospitalization.

Discussion

In this autopsy-based study of 44 fatal COVID-19 cases, we demonstrate that severe SARS-CoV-2 infection is characterized by a constellation of diffuse alveolar damage, vascular injury, mixed inflammatory infiltration, and early fibrotic remodeling. These findings highlight the

multifactorial nature of COVID-19 lung injury, integrating epithelial, endothelial, and immune-mediated mechanisms.

Demographic characteristics and comorbidities

Fatal COVID-19 in our study predominantly affected elderly individuals, a demographic pattern consistently associated with severe pulmonary involvement and adverse outcomes in prior autopsy studies [11,15-17].

Advanced age is associated with impaired resolution of inflammation and reduced fibroblast apoptosis, predisposing to prolonged tissue injury and aberrant repair responses in severe viral pneumonia, ultimately favoring fibrotic remodeling [4,18]. Similar fibroproliferative and collagen deposition patterns have been reported in SARS, MERS, and COVID-19, supporting a shared vulnerability of older individuals to severe coronavirus-induced lung injury [18,19].

Beyond age and sex, comorbidities—particularly hypertension (72.7%) and diabetes mellitus (56.8%)—were highly prevalent in our study, consistent with prior autopsy studies [5,9,20-22]. These conditions are associated with dysregulation of the renin–angiotensin system, which may be exacerbated by SARS-CoV-2–mediated ACE2 internalization, thereby amplifying endothelial dysfunction, inflammation, and alveolar–capillary barrier injury and potentially contributing to the severity of diffuse alveolar damage and vascular alterations observed in fatal cases [21].

Radiologic findings

Radiologic patterns in our fatal COVID-19 cases were dominated by bilateral and diffuse ground-glass opacities and consolidation, features that have been consistently associated with extensive alveolar involvement and severe disease. Similar imaging patterns have been reported in prior radiologic studies of severe COVID-19 [15,16,21,23].

The transition from ground-glass opacities to consolidation on imaging is thought to reflect progressive alveolar epithelial injury, edema, and exudative changes characteristic of diffuse alveolar damage. Additionally, the bilateral and lower-lobe–predominant distribution of these imaging findings—typically observed during the early to intermediate phases of severe disease—parallels the spatial extent of alveolar injury and correlates with clinical severity [9,18]. Collectively, these imaging patterns likely reflect the extent of alveolar injury and early inflammatory responses.

Pathologic findings: diffuse alveolar damage (DAD)

Diffuse alveolar damage emerged as the dominant pathological pattern in our study, with the acute/exudative phase identified in 95.5% of cases. This high prevalence is consistent with

prior autopsy studies, including the systematic review by Caramaschi et al., which identified DAD as the main feature of fatal SARS-CoV-2 infection [3].

Based on prior experimental and clinical evidence, SARS-CoV-2–induced diffuse alveolar damage has been attributed to ACE2-mediated pneumocyte injury and activation of innate immune pathways. In line with this proposed mechanism, the predominance of exudative-phase DAD in our cases supports a model of early epithelial injury followed by macrophage-driven neutrophilic inflammation, leading to alveolar–capillary barrier disruption, plasma protein leakage, and interstitial and alveolar edema [5,24].

Consistent with the predominance of the exudative phase of DAD in our study, severe alveolar epithelial injury was associated with increased permeability, hyaline membrane formation, surfactant dysfunction, alveolar collapse, and impaired gas exchange [24].

Hyaline membrane formation, a defining feature of the exudative phase of DAD, was identified in 54.5% of our cases and reflects profound alveolar–capillary barrier disruption resulting from severe epithelial injury [25]. Supporting our findings, Nunes et al. reported a 50% higher prevalence

of hyaline membranes in COVID-19–positive decedents compared with controls, associating viral pneumocyte injury to hyaline membrane formation in SARS-COV-2 infection [20].

Frequent red blood cell extravasation, observed in 70.5% of our cases, reflects early alveolar–capillary barrier disruption, with intra-alveolar erythrocyte leakage and inflammatory cell infiltration contributing to impaired gas exchange [3,24].These findings are consistent with prior COVID-19 autopsy studies, including reports by Ackermann et al. demonstrating erythrocyte leakage and fibrin deposition in severe disease [3,4,15,19].

Furthermore, alveolar septal widening was observed in 75% of our patients, likely represents interstitial edema and inflammatory cell infiltration. This observation supports the presence of a substantial inflammatory process affecting the alveolar–interstitial compartment in severe coronavirus infection and aligns with histopathological findings previously reported in SARS-CoV-related lung injury, including edema, inflammatory infiltration, septal widening, alveolar organization, and necrosis [22].

Lung tissue samples demonstrated predominantly mixed lymphocytic and polymorphonuclear (PMN) infiltration in over half of cases, accompanied by pulmonary tissue necrosis in 63.6%. Similar patterns of concurrent lymphocytic and neutrophilic infiltration with extensive necrosis have been reported in COVID-19 and SARS, supporting the presence of severe immune-mediated lung injury [4,15,16,26]. Although neutrophilic infiltration is classically associated with bacterial or aspiration pneumonia [3], Borczuk et al. demonstrated that in COVID-19, elevated neutrophil levels correlated with rapid respiratory failure even in the absence of superinfection, suggesting virus-induced alveolitis or endotheliitis rather than secondary

infection [9]. Similarly, Wu et al. attributed widespread necrosis and septal destruction to granulocyte-mediated oxidative stress, heightened inflammatory responses, and ACE2-associated cellular injury [27]. Taken together, these findings indicate that mixed lymphocytic–neutrophilic infiltration accompanied by extensive alveolar necrosis characterizes severe COVID-19–related lung injury and reflects predominantly virus-induced and immune-mediated pathology rather than secondary bacterial infection.

Vascular damage in COVID-19

Microvascular thrombosis, identified in 27.3% of cases in our study, highlights the significant contribution of vascular injury to severe COVID-19 lung pathology.

Microvascular thrombosis is a recognized component of diffuse alveolar damage and has been reported with increased frequency in COVID-19–related lung injury [16].

COVID-19–associated vasculopathy is thought to result from direct viral infection of endothelial cells, dysregulation of local coagulation pathways, and activation of systemic thrombo-inflammatory responses [9,25].

Consistent with our observations, Mauad et al. reported frequent vascular involvement in severe COVID-19, detectable from early stages of DAD, and attributed endothelial injury to the presence of viral particles within endothelial cells [19].

Similarly, Ackermann et al. demonstrated marked endothelial disruption, including cell swelling, detachment from the basal membrane, and loss of intercellular junctions, indicating severe endothelial injury. The presence of SARS-CoV-2 within endothelial cells suggests both direct viral injury and perivascular inflammation contributing to endothelial damage [4].

Inflammatory activation of the endothelium in COVID-19 has been shown to induce a procoagulant phenotype, promoting microthrombosis [21].

The persistence of thrombotic events despite anticoagulation further supports the combined contribution of direct endothelial injury and systemic inflammation to COVID-19–associated coagulopathy [3,4,19,21].

Fibrosis and COVID-19

Histological evidence of fibrosis was identified in 86.4% of our cases, indicating a substantial burden of fibrotic remodeling. This finding is consistent with progression from the exudative to organizing phases of diffuse alveolar damage. During this phase, dysregulated repair—marked by fibroblast activation, myofibroblast differentiation, and type II pneumocyte hyperplasia—leads to excessive extracellular matrix deposition, resulting in parenchymal remodeling, pulmonary fibrosis, and persistent functional impairment [3,25]. These alterations disrupt alveolar architecture and drive structural remodeling detectable on chest CT,

supporting pulmonary fibrosis as a frequent pathological consequence of severe COVID-19, similar to other coronavirus infections [18].

Notably, Mauad et al. reported fibrotic changes in approximately 39% of cases, a lower prevalence than observed in our cohort, potentially reflecting differences in disease severity, duration, or patient selection [19].

Temporal progression of COVID-19-related lung pathology

In the present study, alveolar exudative changes and red blood cell extravasation were more frequently observed in patients with shorter symptom duration prior to hospitalization, indicating that epithelial injury and disruption of the alveolar–capillary barrier occur early in the course of COVID-19 (Figure 6) [11]

In contrast, tissue necrosis and microthrombosis were more commonly associated with longer symptom duration, suggesting progression toward more extensive parenchymal and vascular involvement [5].

Similar temporal trends have been reported in prior autopsy studies. Beigmohammadi et al. described acute diffuse alveolar damage with hyaline membrane formation and fibrin exudation in patients with shorter disease duration, whereas fibrotic changes were more frequent in prolonged hospitalization [15]. Larger autopsy series have likewise shown that exudative diffuse alveolar damage predominates early, while proliferative or organizing patterns emerge in longer clinical courses [5]. Importantly, epithelial, vascular, and fibrotic alterations often coexist rather than follow a strictly sequential pattern, underscoring the heterogeneous nature of COVID-19 lung injury [11,14].

Together, these findings support a dynamic model in which early epithelial and vascular injury may, in a subset of patients, rapidly evolve toward more severe histopathological patterns, progressing to necrotic, thrombotic, and fibrotic changes within a relatively short clinical timeframe.

A clear understanding of the predominant pathological patterns in fatal COVID-19 may help inform clinical decision-making, particularly regarding the interpretation of radiologic findings and the timing of supportive care in severe disease. Recognition of virus-driven inflammatory and vascular injury may also help reduce unnecessary antibiotic use in the absence of bacterial superinfection.

This study has several limitations inherent to autopsy-based analyses. Postmortem findings may not fully reflect the dynamic disease processes in living patients, and sampling constraints occasionally limited the availability of representative pulmonary tissue. In addition, systematic lesion-level CT–pathology correlation and comprehensive molecular analyses were not performed, which restricts direct inference between imaging patterns and specific histologic

changes. Future studies integrating longitudinal imaging, molecular profiling, and larger comparative cohorts are warranted to further clarify disease progression and therapeutic implications, including optimal anticoagulation strategies and prevention of severe thrombo-inflammatory responses.

Conclusions

In conclusion, diffuse alveolar damage represents the dominant histopathologic feature in fatal SARS-CoV-2 infection, accompanied by prominent vascular injury, mixed inflammatory infiltration, and fibrotic remodeling. The interplay between demographic factors, comorbidities, and lung pathology underscores the complex and multifactorial nature of severe COVID-19 and contributes to a more integrated understanding of its pulmonary manifestations.

References

1. Zhu N, Zhang D, Wang W, et al. A novel coronavirus from patients with pneumonia in China, 2019. N Engl J Med 2020;382:727-33.
2. Huang G, Guo F, Liu L, et al. Changing impact of COVID-19 on life expectancy 2019-2023 and its decomposition: findings from 27 countries. SSM Popul Health 2024;25:101568.
3. Caramaschi S, Kapp ME, Miller SE, et al. Histopathological findings and clinicopathologic correlation in COVID-19: a systematic review. Mod Pathol 2021;34:1614-33.
4. Ackermann M, Verleden SE, Kuehnel M, et al. Pulmonary vascular endothelialitis, thrombosis, and angiogenesis in covid-19. N Engl J Med 2020;383:120-8.
5. Elsoukkary SS, Mostyka M, Dillard A, et al. Autopsy findings in 32 patients with COVID-19: a single-institution experience. Pathobiology 2021;88:56-68.
6. Xu Z, Shi L, Wang Y, et al. Pathological findings of COVID-19 associated with acute respiratory distress syndrome. Lancet Respir Med 2020;8:420-2.
7. Panahi Y, Gorabi AM, Talaei S, et al. An overview on the treatments and prevention against COVID-19. Virol J 2023;20:23.
8. To KF, Lo AW. Exploring the pathogenesis of severe acute respiratory syndrome (SARS): the tissue distribution of the coronavirus (SARS-CoV) and its putative receptor, angiotensin-converting enzyme 2 (ACE2). J Pathol 2004;203:740-3.
9. Borczuk AC, Salvatore SP, Seshan SV, et al. COVID-19 pulmonary pathology: a multi-institutional autopsy cohort from Italy and New York City. Mod Pathol 2020;33:2156-68.

10. Olteanu GE, Pezzuto F, Lunardi F, et al. Exploring the pathologist's role in understanding COVID-19: from pneumonia to long-COVID lung sequelae. Pathologica 2023;115:275-83.
11. Polak SB, Van Gool IC, Cohen D, et al. A systematic review of pathological findings in COVID-19: a pathophysiological timeline and possible mechanisms of disease progression. Mod Pathol 2020;33:2128-38.
12. Baldi BG, Fabro AT, Franco AC, et al. Clinical, radiological, and transbronchial biopsy findings in patients with long COVID-19: a case series. J Bras Pneumol 2022;48:e20210438.
13. Martín Sánchez FJ, Martínez-Sellés M, Molero García JM, et al. Insights for COVID-19 in 2023. Rev Esp Quimioter 2023;36:114-24.
14. Stoyanov GS, Yanulova N, Stoev L, et al. Temporal patterns of COVID-19-associated pulmonary pathology: an autopsy study. Cureus 2021;13:e20522.
15. Beigmohammadi MT, Jahanbin B, Safaei M, et al. Pathological findings of postmortem biopsies from lung, heart, and liver of 7 deceased COVID-19 patients. Int J Surg Pathol 2021;29:135-45.
16. Ramos-Rincon JM, Herrera-García C, Silva-Ortega S, et al. Pathological findings associated with SARS-CoV-2 on postmortem core biopsies: correlation with clinical presentation and disease course. Front Med 2022;9:874307.
17. Guan WJ, Ni ZY, Hu Y, et al. Clinical characteristics of coronavirus disease 2019 in China. N Engl J Med 2020;382:1708-20.
18. Ojo AS, Balogun SA, Williams OT, Ojo OS. Pulmonary fibrosis in COVID-19 survivors: predictive factors and risk reduction strategies. Pulm Med 2020;2020:6175964.
19. Mauad T, Duarte-Neto AN, da Silva LFF, et al. Tracking the time course of pathological patterns of lung injury in severe COVID-19. Respir Res 2021;22:32.
20. Nunes MC, Hale MJ, Mahtab S, et al. Clinical characteristics and histopathology of COVID-19 related deaths in South African adults. PLoS One 2022;17:e0262179.
21. Peiris S, Mesa H, Aysola A, et al. Pathological findings in organs and tissues of patients with COVID-19: a systematic review. PLoS One 2021;16:e0250708.
22. Li G, Fan Y, Lai Y, et al. Coronavirus infections and immune responses. J Med Virol 2020;92:424-32.
23. Ding Y, Wang H, Shen H, et al. The clinical pathology of severe acute respiratory syndrome (SARS): a report from China. J Pathol 2003;200:282-9.
24. Batah SS, Fabro AT. Pulmonary pathology of ARDS in COVID-19: a pathological review for clinicians. Respir Med 2021;176:106239.
25. Angeles Montero-Fernandez M, Pardo-Garcia R. Histopathology features of the lung in COVID-19 patients. Diagn Histopathol 2021;27:123-7.

26. Luo W, Yu H, Gou J, et al. Clinical pathology of critical patient with novel coronavirus pneumonia (COVID-19). 2020. Available from: https://www.preprints.org/manuscript/202002.0407/v1.
27. Wu J, Yu J, Zhou S, et al. What can we learn from a COVID-19 lung biopsy? Int J Infect Dis 2020;99:410-3.

Table 1. Patient characterization.

	Patient characterization (n=44)	
Gender		
Female	15	34.1%
Male	29	65.9%
Age (average)		64.77±14.29 years
Average duration of symptoms		6±1.6 days
Smoking status		
Smoking cigarette	15	34.09%
Opium-user	14	31.81%
Underlying diseases		
Diabetes	25	56.8%
Hypertension	32	72.7%
Ischemic heart diseases	9	20.5%
Chronic obstructive pulmonary disease	3	6.8%
Asthma	0	0%
Cancer	1	2.3%
Chronic kidney failure	6	13.6%
Thyroid diseases	2	4.5%
Mixed connective tissue disease	0	0%

Table 2. Distribution of radiological findings and their patterns in 44 COVID-19 patients.

Radiological findings	Overall (%)	Male (% of 27)	Female (% of 17)	p
Lesions				
Consolidation	40.9% (18/44)	44.4% (12/27)	35.3% (6/17)	0.93
Ground-glass opacity	54.5% (24/44)	55.6% (15/27)	52.9% (9/17)	0.60
Pleural effusion	0%	0%	0%	N/A
Patterns				
Bilateral	100%	–	–	–
Widespread	95.5%	–	–	–

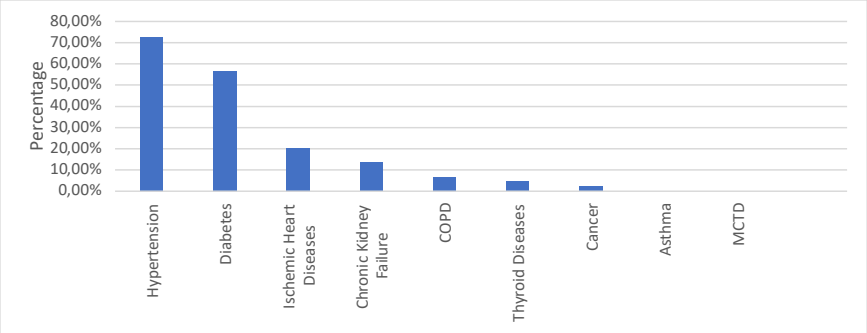

Figure 1. **Distribution of underlying diseases in COVID-19 patients. COPD, chronic obstructive pulmonary disease; MCTD, mixed connective tissue disease.**

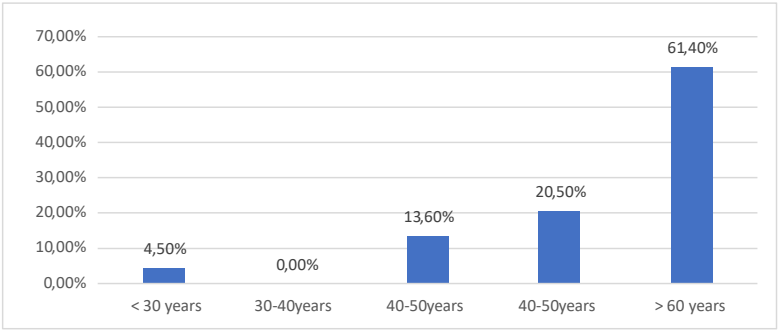

Figure 2. Distribution of COVID-19 patients by age.

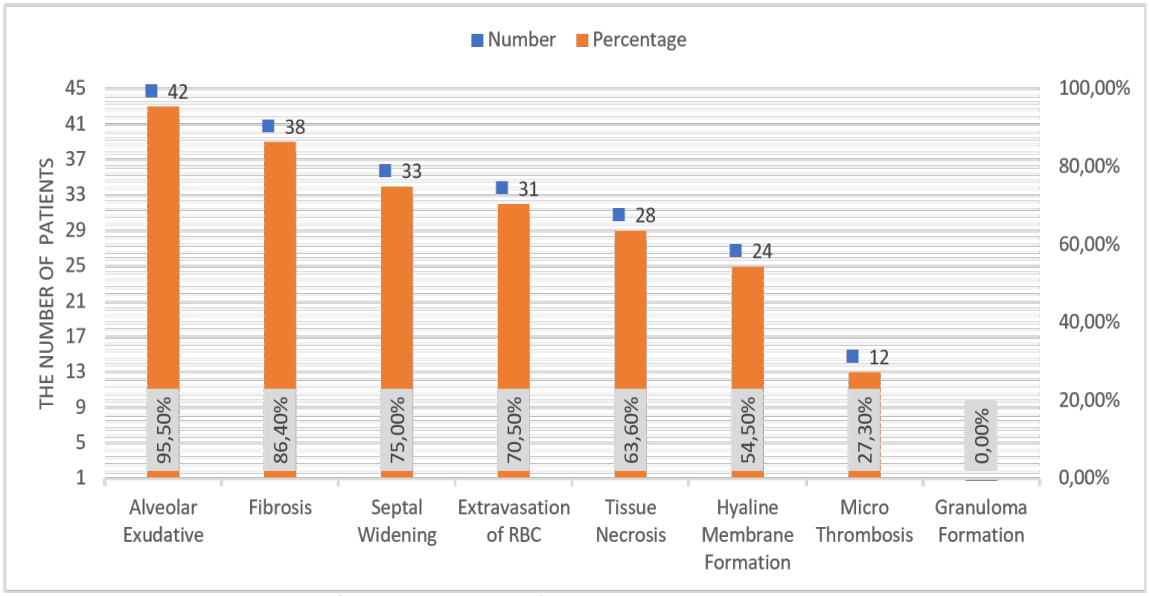

Figure 3. Distribution of pathological findings in 44 COVID-19 patients.

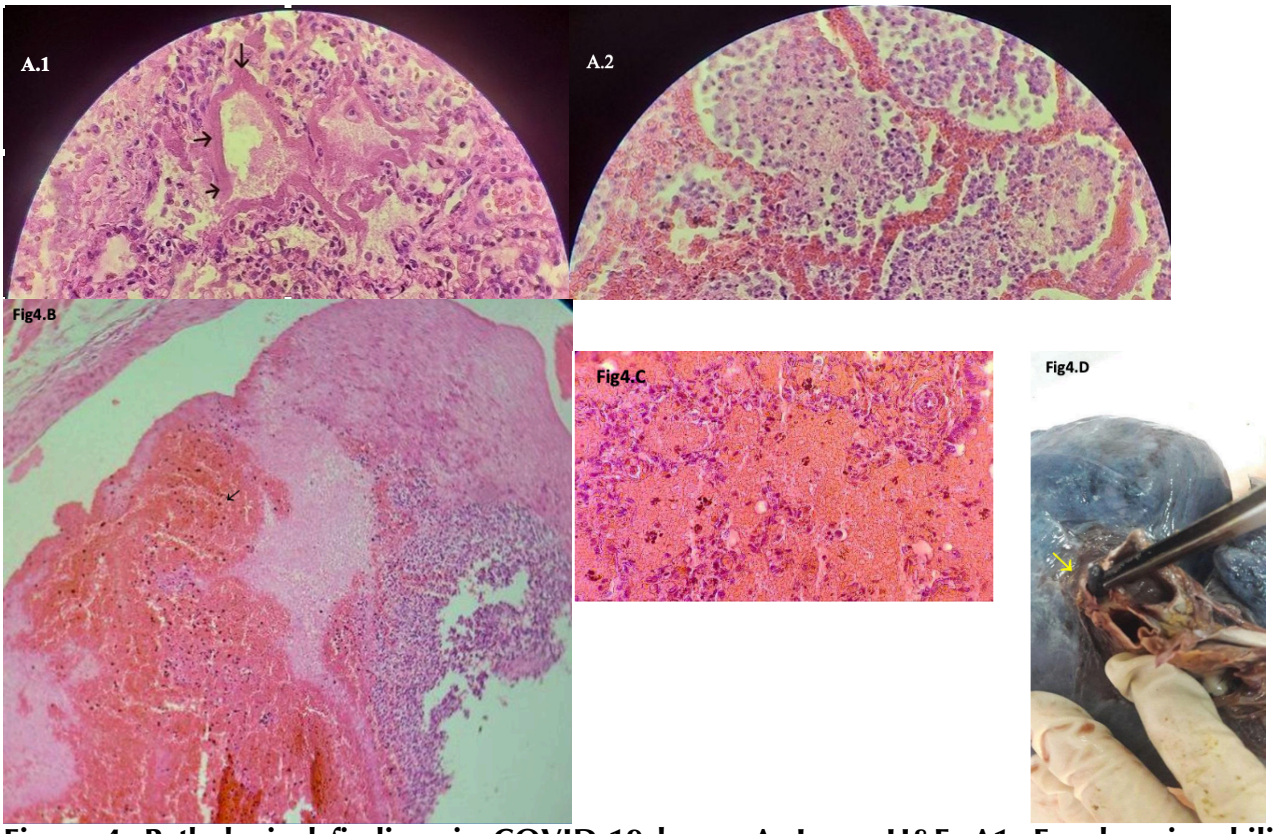

Figure 4. Pathological findings in COVID-19 lungs. A. Lung, H&E. A1. Focal eosinophilic hyaline membranes lining the alveolar septa (black arrows). A2. Prominent intra-alveolar neutrophilic infiltration in areas of diffuse alveolar damage (exudative phase). B. Lung, H&E. Organizing intra-alveolar exudate with fibroblastic plugs and septal thickening, compatible with the organizing (proliferative) phase of diffuse alveolar damage; collagen deposition (black arrow). C. Lung, H&E. Diffuse intra-alveolar red blood cells, consistent with alveolar hemorrhage. D. Gross examination of the lung hilum demonstrating an intraluminal thromboembolic mass adherent to the wall of the main pulmonary artery, showing a heterogeneous appearance (yellow arrow), consistent with antemortem pulmonary thrombosis in the prothrombotic state described in severe COVID-19.

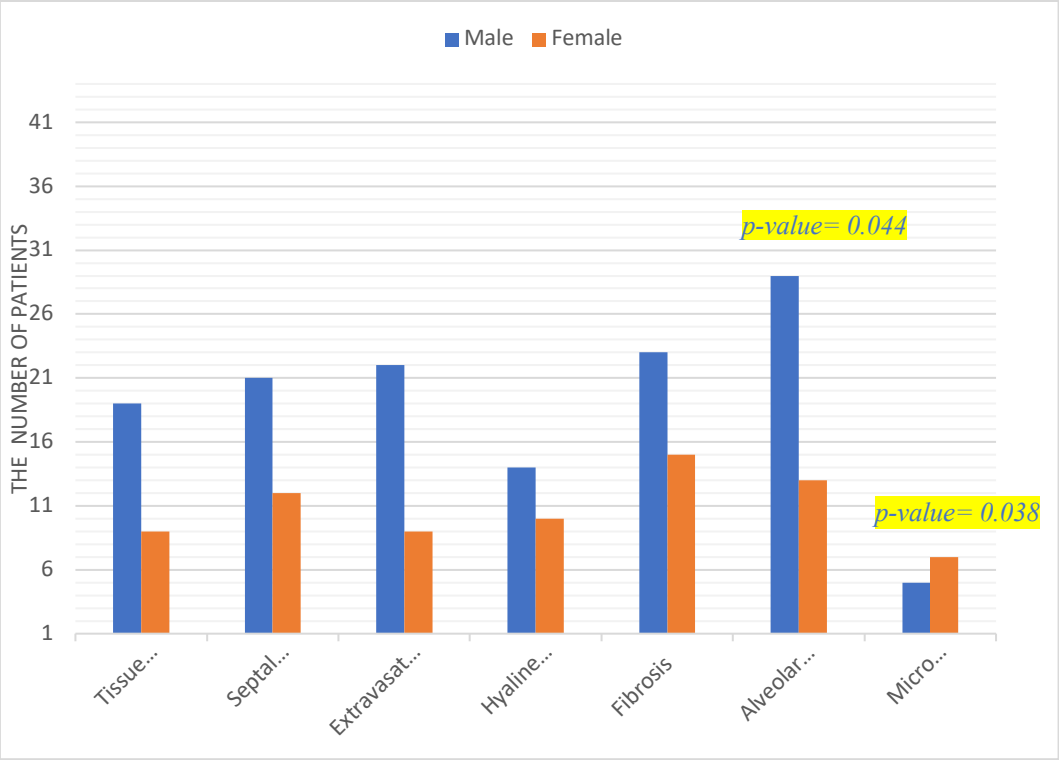

Figure 5. Distribution of pathological findings by gender among 44 COVID-19 patients. Alveolar exudative changes were more frequent in males (p = 0.044), while microthrombosis was more common in females (p = 0.038). No other significant gender-related difference.

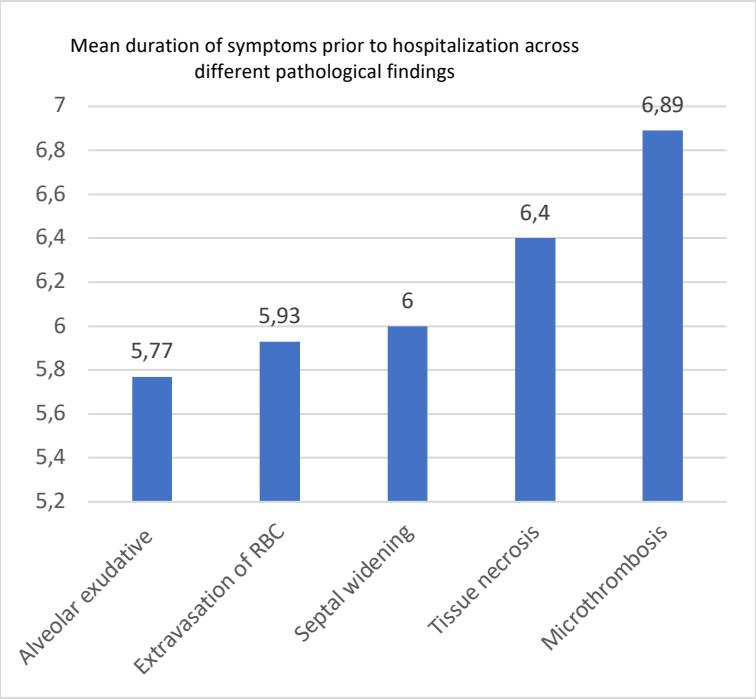

Figure 6. Mean duration from symptom onset to hospitalization across different pathological findings. Bars represent mean values for each pathological category.