







Liver Histopathology of Deceased Patients with COVID-19: A Case Series from Tehran, Iran

Mousa Ahmadi ¹, Shadi Mohammadi ², Mahtab Fotoohi ³, Morteza Aghajanoor ⁴, Ramin Yaghmayee ³, Jalil Rajabi ¹, Besharat Zarezadeh ⁵, Saeed Soleiman-Meigooni ^{6,*}

¹ Department of Infectious Diseases, Faculty of Medicine, AJA University of Medical Sciences, Tehran, Iran

² Department of Obstetrics and Gynecology, Khanevadeh University Hospital, AJA University of Medical Sciences, Tehran, Iran

³ Department of Pathology, Khanevadeh University Hospital, AJA University of Medical Sciences, Tehran, Iran

⁴ Department of Gastroenterology and Hepatology, Faculty of Medicine, AJA University of Medical Sciences, Tehran, Iran

⁵ Iranian Research Center for HIV/AIDS, Iranian Institute of High-risk Behaviors, Tehran University of Medical Sciences, Tehran, Iran

⁶ Infectious Diseases Research Center, AJA University of Medical Sciences, Tehran, Iran

*Corresponding Author: Infectious Diseases Research Center, AJA University of Medical Sciences, Tehran, Iran. Email: dr.saeed.meigooni@gmail.com

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Abstract

Background: SARS-CoV-2 receptors are distributed in several vital organs, and many of these organs are damaged in severe COVID-19. Liver inflammation in COVID-19 ranges from asymptomatic elevation of liver transaminase levels to fulminant hepatitis.

Objectives: The present study aimed to investigate the liver histopathology of deceased patients with COVID-19.

Methods: This case series study was conducted in Tehran, Iran, in 2021. The subjects were deceased patients with COVID-19. After obtaining informed consent from the first-degree relatives, at least two liver tissue samples were obtained through the right 10th to 12th intercostal spaces using a biopsy needle, following the Helsinki biopsy guidelines. Clinical information from the patients was extracted using a questionnaire. Fisher's exact test and Student's t-test were used to analyze the data.

Results: Liver sampling was performed in 12 deceased patients. The mean age of the patients was 73 years, with a male-to-female ratio of 9:3. The most common clinical cause of death was adult respiratory distress syndrome (ARDS), occurring in eight patients. An increase in liver transaminase levels was observed in four patients. Histopathological examination revealed normal results in three patients, moderate to severe inflammation according to the [non-alcoholic fatty liver disease (NAFLD) activity score (NAS score)] in eight patients, and mild NAS scores in four patients. Liver inflammation was not correlated with other parameters, including sex, diabetes mellitus (DM), chronic renal failure (CRF), transaminase level, Body Mass Index (BMI), C-reactive protein (CRP) level, or length of stay in hospital. Liver steatosis was observed in four of the 12 patients and was significantly associated with a higher BMI ($P = 0.014$).

Conclusions: In our study, most patients exhibited a moderate-to-severe NAS score that did not correlate with disease severity or underlying medical conditions. We recommend regular assessment of clinical and biochemical indicators of liver inflammation in individuals with severe COVID-19.

Keywords: NAS Score, SARS-CoV-2, COVID-19, Autopsy, Steatosis, Hepatitis, Iran

1. Background

Liver injury is a prevalent form of organ damage in patients with SARS-CoV-2. It is estimated that 14% to 53% of patients with COVID-19 develop abnormal liver function tests (1). The mechanisms of liver damage in COVID-19 are under investigation and appear to have

multifactorial etiologies. Liver injury may present with subclinical elevation of aminotransferase levels or fulminant hepatic failure (2, 3). Therefore, SARS-CoV-2-related liver injury has a spectrum of histopathological features. Autopsy studies have demonstrated steatosis, inflammation, vascular thrombosis, and hepatocyte necrosis in deceased patients with COVID-19 (4-7).

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According to recent pathology investigations, patients with COVID-19 had a greater rate of steatosis and lobular inflammation than those with other liver diseases. High-dose steroid administration contributes to the occurrence of steatosis (6). Liver injury is associated with prolonged length of stay (LOS) in hospital, higher rates of ICU admission, and increased mortality rates (8). Critically ill patients with COVID-19 exhibit increased hepatic failure, suggesting that liver damage is correlated with disease severity (9). Additionally, pre-existing liver disorders, including non-alcoholic fatty liver disease (NAFLD) and chronic viral hepatitis, may exacerbate the risk of liver damage (10). SARS-CoV-2-associated liver damage contributes to unfavorable outcomes in patients with COVID-19.

2. Objectives

In the present study, we aimed to evaluate liver histopathology and its clinical correlation in a series of patients who died of COVID-19.

3. Methods

We conducted a case series study of deceased patients with COVID-19 in Tehran, Iran, between January and May 2021. Subjects were selected through conventional sampling after obtaining written informed consent from their first-degree relatives. One of the authors, wearing protective equipment, including an N95 mask, gown, gloves, and face shield, performed the liver biopsy in a negative pressure room. The procedure was performed using a 16-gauge Jamshidi needle through the 12th right intercostal space. The samples were fixed in formalin and transferred to the pathology department. Subsequently, 10 μ m sections were prepared from the samples using a microtome, stained with hematoxylin-eosin, and mounted on slides. Each slide was independently examined by two pathologists, and the final report was compiled through collaboration between the attending physician and pathologists. Additionally, demographic characteristics, underlying conditions, clinical symptoms, and paraclinical findings, including blood cell counts, inflammatory biomarkers, liver function tests, and histological findings, were extracted from their registries. Fisher's exact test and Student's *t*-test were used for data analysis. This study is part of a multi-organ autopsy study in deceased patients with COVID-19, approved by the Ethics Committee of the AJA University of Medical Sciences (IR.AJUMS.REC.1399.030).

4. Results

During the study period, 12 deceased COVID-19 patients underwent liver biopsies. Of these, three were female and nine were male. The mean age of the patients was 73 ± 14.3 years, the mean Body Mass Index (BMI) was 26.6 ± 4 , and the mean LOS was 12.3 ± 3.3 days. The frequencies of underlying diseases included hypertension (HTN) in 11 patients (91.7%), ischemic heart disease (IHD) in eight patients (66.7%), diabetes mellitus (DM) in five patients (41.7%), and chronic renal failure (CRF) in three patients (25%). The most common symptoms on admission were dyspnea in 11 patients (91.7%), cough in nine patients (75%), fever in seven patients (58.3%), malaise, and decreased level of consciousness in six patients (50%). The most common clinical cause of death was adult respiratory distress syndrome (ARDS), which occurred in eight patients (66.7%).

Table 1 presents the demographic characteristics, clinical symptoms, and laboratory findings of patients on admission. Histopathological examination revealed portal and periportal inflammation as the predominant findings in nine patients (75%), followed by microvesicular steatosis in four patients (33.3%) and interstitial edema in three patients (25%) (Figure 1). Hepatic inflammation was classified as minimal to mild in four individuals and moderate to severe in eight patients according to the NAFLD activity score (NAS score). The Student's *t*-test (Table 2) did not reveal any correlation between the NAS score and age, BMI, LOS, C-reactive protein (CRP), lactate dehydrogenase (LDH), and alanine transaminase (ALT) levels. Additionally, Fisher's exact test did not reveal any correlation between NAS score and sex, DM, CRF, or etiology of mortality (Table 3). Patients with steatosis exhibited a higher BMI than those without steatosis (30.2 ± 3.6 vs. 24.7 ± 2.8 , respectively; $P = 0.014$).

5. Discussion

Our study is one of the few investigations focused on liver histopathology in patients with COVID-19. The most common histopathological finding was inflammation (75%), followed by steatosis (33.3%). Most patients were classified as having moderate or severe NAS scores, indicating the severity of liver injury in COVID-19. Our findings underscore the importance of liver involvement in patients with severe COVID-19. Furthermore, steatosis was more common in patients with a BMI > 30 . Similar studies have reported steatosis, inflammation, and necrosis as primary findings in liver pathology. The primary liver histological characteristics linked to SARS-CoV-2 infection include steatosis, portal and periportal inflammation, vascular thrombosis, and

Table 1. Demographic Characteristics and Laboratory Finding of the Study Population (N = 12)

Cases	Age	Sex	BMI (kg/m ²)	LOS	ALC	ANC	Hb	PLT (× 10 ³ U/L)	CRP (mg/L)	LDH (< 450 U/L)	ALT (< 40 IU/L)	PMH	Clinical Cause of Death	Hepatic Inflammation by NAS Score	Liver Pathology
1	78	F	24	7	873	8342	14.9	143	92	765	13	HTN, CRF	PTE	Moderate activity, 3 points	Portal and periportal mixed inflammation, ballooning degeneration, edema
2	79	F	29	12	640	6720	12.1	317	2	310	16	HTN, DM	ARDS	Mild activity, 2 points	Portal and periportal mixed inflammation, ballooning degeneration
3	70	F	27	17	666	2960	14.4	136	65	753	78	HTN	PTE	Moderate activity, 5 points	Portal and periportal mixed inflammation, microvesicular steatosis, focal necrosis, ballooning degeneration, edema
4	35	M	35	8	1910	16808	11.4	321	64	844	84	HTN, IHD, DM	ARDS	Moderate activity, 3 points	Mild inflammation, microvesicular steatosis
5	79	M	31	10	770	4510	14.8	115	43	1573	13	HTN, IHD	ARDS	Moderate activity, 4 points	Mild inflammation, microvesicular steatosis
6	78	M	21	12	1262	10500	12.7	212	54	280	24	HTN, CRF	ARDS	Severe activity, 7 points	Portal and periportal mixed inflammation, ballooning degeneration, edema
7	93	M	24	12	1221	15576	13.8	162	68	693	33	None	ARDS	Moderate activity, 4 points	Portal and periportal mixed inflammation, ballooning degeneration
8	69	M	28	10	3250	20750	9.2	397	66	476	66	HTN, IHD	ARDS	Moderate activity, 4 points	Portal and periportal mixed inflammation and hemorrhage, microvesicular steatosis
9	64	M	28	13	1137	4152	13.8	137	46	827	33	HTN, IHD, DM	PTE	Moderate activity, 4 points	Portal and periportal mixed inflammation, hemorrhage
10	75	M	24	17	567	5670	12.7	190	74	614	34	HTN, IHD	Sepsis and MOF	Normal	Normal
11	70	M	22	16	2250	12320	13.9	126	72	365	30	HTN, IHD, DM, CRF	Sepsis and MOF	Normal	Normal
12	86	M	26	14	336	2040	12.7	83	58	550	45	HTN, IHD, DM	PTE	Normal	Normal

Abbreviations: BMI, Body Mass Index; LOS, length of stay; ALC, absolute lymphocyte count; ANC, absolute neutrophil count; CRP, C-reactive protein; LDH, lactate dehydrogenase; ALT, alanine transaminase; F, female; M, male; HTN, hypertension; CRF, chronic renal failure; DM, diabetes mellitus; IHD, ischemic heart disease; NAS score, non-alcoholic fatty liver disease (NAFLD) activity score.

hepatocyte necrosis (4, 5, 9-14). However, we did not observe portal thrombosis in our subjects, which may be attributable to the small sample size or population differences. We also did not find fibrotic changes in our patients. Prolonged liver inflammation may result in hepatic fibrosis in patients with liver diseases (15). We suggest that since only a quarter of our patients exhibited increased transaminases and none had a history of chronic viral hepatitis, along with the acute nature of inflammation triggered by COVID-19, the development of fibrosis seemed unlikely. Although we did not find any correlation between the NAS score and other clinical or paraclinical parameters, this may be related to our limited sample size.

Angiotensin-converting enzyme 2 (ACE2) is a specific receptor for SARS-CoV-2 and is present in several vital organs (16). Autopsy studies have revealed severe inflammation and damage to the lungs, heart, brain,

kidney, and liver in a high percentage of patients with severe COVID-19 (17-20). The most common finding of liver injury during SARS-CoV-2 infection is elevated transaminase levels (1, 21, 22). Furthermore, some patients develop cholestasis and elevated alkaline phosphatase and bilirubin levels (23). Acute hepatic failure is a rare condition that develops in severe COVID-19 cases (3, 24). Several mechanisms have been proposed for liver injury during SARS-CoV-2 infection, including direct SARS-CoV-2 invasion (25), immune-mediated injury after cytokine storms (26), hypoxia-related liver injury (27), coagulopathy (28), and drug-induced hepatotoxicity, such as corticosteroids resulting in steatosis, or remdesivir resulting in hepatitis (14, 29). Pre-existing hepatic conditions, advanced age, and severe COVID-19 increase the likelihood of hepatic complications (10).

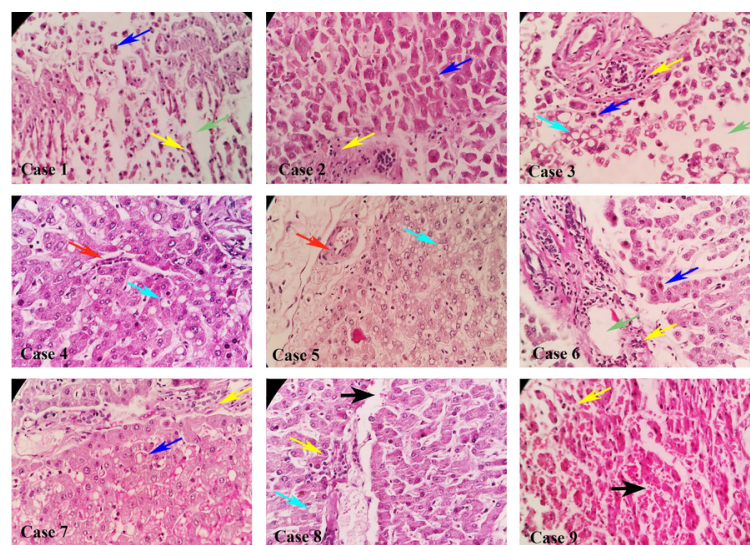


Figure 1. Liver histopathology of deceased patients with COVID-19. Yellow arrow, portal and periportal mixed inflammation; cyan arrow, microvesicular steatosis; red arrow, mild inflammation; black arrow, hemorrhage; blue arrow, hepatocyte injury, ballooning degeneration; pea green arrow, edema.

Table 2. Liver Histopathology in the Subgroups of the Study Population (N = 12)

Variables	NAS Score		P-Value (Fisher's Exact Test)
	Normal or Mild Score (n = 4)	Moderate or Severe Score (n = 8)	
Gender			1.000
Male	3	6	
Female	1	2	
DM			0.081
Yes	4	3	
No	0	5	
CRF			1.000
Yes	1	2	
No	3	6	
Death etiology			0.067
ARDS	1	7	
PTE	1	1	
Sepsis	2	0	

Abbreviations: NAS score, non-alcoholic fatty liver disease (NAFLD) activity score; DM, diabetes mellitus; CRF, chronic renal failure; ARDS, adult respiratory distress syndrome.

Previous studies have reported that liver injury in patients with COVID-19 is associated with prolonged LOS, higher rates of ICU admission, and increased mortality rates (8). Although studies have suggested a correlation between liver inflammation and disease severity, we did not find any correlation between the NAS score and CRP levels in our study (9, 14). While additional research is required to definitively ascertain the relationship between liver inflammation and COVID-19-related mortality, existing evidence indicates that hepatic inflammation significantly influences the prognosis and outcome of COVID-19. Despite the absence of an association between the NAS score and the

history of DM, CRF, CRP levels, or BMI, this inconsistency may be attributable to the limited sample size of our study.

5.1. Limitations

Our study has several limitations. The principal limitation was the absence of a control group to compare liver pathology in hypoxic-ischemic liver injury with that in viral hepatitis. Another challenge was the absence of prior liver function tests, which affected liver pathology before admission. Finally, our study was conducted with a small subject group, which may limit the generalizability of our findings.

Table 3. Liver Histopathology in the Subgroups of the Study Population (N = 12)

Variables	NAS Score		P-Value (t-Test)
	Normal or Mild Score (n = 4)	Moderate or Severe Score (n = 8)	
Age	73 ± 12.5	70.7 ± 16.9	0.820
BMI	27 ± 3.9	27.2 ± 4.4	0.926
LOS	16.7 ± 22.2	5.9 ± 2.1	0.180
CRP	42.5 ± 30.4	62.2 ± 15.3	0.156
LDH	416 ± 104	776 ± 375	0.095
ALT	26.5 ± 14.1	43 ± 28.7	0.312

Abbreviations: BMI, Body Mass Index; LOS, length of stay; CRP, C-reactive protein; LDH, lactate dehydrogenase; ALT, alanine transaminase; NAS score, non-alcoholic fatty liver disease (NAFLD) activity score.

5.2. Conclusions

Our study revealed a high percentage of liver involvement in deceased COVID-19 patients. Therefore, clinicians should monitor liver function in patients with COVID-19, particularly in those with preexisting liver conditions.

Footnotes

Authors' Contribution: M. A., S. M., M. A., J. R., and B. Z. contributing to treatment, reviewing the literature, and writing the primary draft. R. Y. and M. F. were pathologists, contributing to pathological diagnosis, reviewing the literature and writing the primary draft. S. S. M. was the main author, contributing to the study design, treatment of the patients, reviewing the literature, analyzing of the data and writing the primary and final draft.

Conflict of Interests Statement: The authors declare no conflict of interest.

Data Availability: The dataset presented in the study is available on request from the corresponding author during submission or after publication. The data are not publicly available due to confidentiality and the corresponding author commitment not to disclose the identity of patients.

Ethical Approval: The present study was approved by the Ethics Committee of the AJA University of Medical Sciences ([IR.AJAUMS.REC.1399.030](#)).

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Informed Consent: Written informed consent was obtained from first-degree relatives for postmortem sampling.

References

- Vishwajeet V, Purohit A, Kumar D, Parag V, Tripathi S, Kanchan T, et al. Evaluation of Pathological Findings of COVID-19 by Minimally Invasive Autopsies: A Single Tertiary Care Center Experience from India. *J Lab Physicians*. 2021;**13**(2):97-106. [PubMed ID: [34483552](#)]. [PubMed Central ID: [PMC8409114](#)]. <https://doi.org/10.1055/s-0041-1730750>.
- Wijarnpreecha K, Ungprasert P, Panjawatanan P, Harnois DM, Zaver HB, Ahmed A, et al. COVID-19 and liver injury: a meta-analysis. *Eur J Gastroenterol Hepatol*. 2021;**33**(7):990-5. [PubMed ID: [32639420](#)]. [PubMed Central ID: [PMC8162043](#)]. <https://doi.org/10.1097/MEG.0000000000001817>.
- Melquist S, Estepp K, Aleksandrovich Y, Lee A, Beiseker A, Hamedani FS, et al. COVID-19 presenting as fulminant hepatic failure: A case report. *Medicine (Baltimore)*. 2020;**99**(43). e22818. [PubMed ID: [33120805](#)]. [PubMed Central ID: [PMC7581048](#)]. <https://doi.org/10.1097/MD.00000000000022818>.
- Sonzogni A, Previtali G, Seghezzi M, Grazia Alessio M, Gianatti A, Licini L, et al. Liver histopathology in severe COVID 19 respiratory failure is suggestive of vascular alterations. *Liver Int*. 2020;**40**(9):2110-6. [PubMed ID: [32654359](#)]. [PubMed Central ID: [PMC7404964](#)]. <https://doi.org/10.1111/liv.14601>.
- Chornenkyy Y, Mejia-Bautista M, Brucal M, Blanke T, Dittmann D, Yeldandi A, et al. Liver Pathology and SARS-CoV-2 Detection in Formalin-Fixed Tissue of Patients With COVID-19. *Am J Clin Pathol*. 2021;**155**(6):802-14. [PubMed ID: [33914058](#)]. [PubMed Central ID: [PMC8135761](#)]. <https://doi.org/10.1093/ajcp/aaqb009>.
- Righi FA, Vander Heide RS, Graham RP, Aubry MC, Trejo-Lopez JA, Bois MC, et al. A case-control autopsy series of liver pathology associated with novel coronavirus disease (COVID-19). *Ann Diagn Pathol*. 2024;**68**:152240. [PubMed ID: [37995413](#)]. <https://doi.org/10.1016/j.anndiagpath.2023.152240>.
- Bidari Zerehpooosh F, Sabeti S, Bahrani-Motlagh H, Mokhtari M, Naghibi Irvani SS, Torabinaid P, et al. Post-mortem Histopathologic Findings of Vital Organs in Critically Ill Patients with COVID-19. *Arch Iran Med*. 2021;**24**(2):144-51. [PubMed ID: [33636984](#)]. <https://doi.org/10.34172/aim.2021.23>.
- Kondo R, Kawaguchi N, McConnell MJ, Sonzogni A, Licini L, Valle C, et al. Pathological characteristics of liver sinusoidal thrombosis in COVID-19 patients: A series of 43 cases. *Hepatol Res*. 2021;**51**(9):1000-6. [PubMed ID: [34260803](#)]. [PubMed Central ID: [PMC8444906](#)]. <https://doi.org/10.1111/hepr.13696>.
- Chu H, Peng L, Hu L, Zhu Y, Zhao J, Su H, et al. Liver Histopathological Analysis of 24 Postmortem Findings of Patients With COVID-19 in China. *Front Med (Lausanne)*. 2021;**8**:749318. [PubMed ID: [34708059](#)]. [PubMed Central ID: [PMC8543004](#)]. <https://doi.org/10.3389/fmed.2021.749318>.
- Fassan M, Mescoli C, Sbaraglia M, Guzzardo V, Russo FP, Fabris R, et al. Liver histopathology in COVID-19 patients: A mono-Institutional

- series of liver biopsies and autopsy specimens. *Pathol Res Pract.* 2021;**221**:153451. [PubMed ID: 33932720]. [PubMed Central ID: PMC8054534]. <https://doi.org/10.1016/j.prp.2021.153451>.
11. Lax SF, Skok K, Zechner P, Kessler HH, Kaufmann N, Koelblinger C, et al. Pulmonary Arterial Thrombosis in COVID-19 With Fatal Outcome: Results From a Prospective, Single-Center, Clinicopathologic Case Series. *Ann Intern Med.* 2020;**173**(5):350-61. [PubMed ID: 32422076]. [PubMed Central ID: PMC7249507]. <https://doi.org/10.7326/M20-2566>.
 12. Falasca L, Nardacci R, Colombo D, Lalle E, Di Caro A, Nicastri E, et al. Postmortem Findings in Italian Patients With COVID-19: A Descriptive Full Autopsy Study of Cases With and Without Comorbidities. *J Infect Dis.* 2020;**222**(11):1807-15. [PubMed ID: 32914853]. [PubMed Central ID: PMC7543426]. <https://doi.org/10.1093/infdis/jiaa578>.
 13. Hanley B, Naresh KN, Roufousse C, Nicholson AG, Weir J, Cooke GS, et al. Postmortem Findings in Italian Patients With COVID-19: A Descriptive Full Autopsy Study of Cases With and Without Comorbidities. *J Infect Dis.* 2020;**222**(11):1807-15. [PubMed ID: 32914853]. [PubMed Central ID: PMC7543426]. <https://doi.org/10.1093/infdis/jiaa578>.
 14. Zhao SW, Li YM, Li YL, Su C. Liver injury in COVID-19: Clinical features, potential mechanisms, risk factors and clinical treatments. *World J Gastroenterol.* 2023;**29**(2):241-56. [PubMed ID: 36687127]. [PubMed Central ID: PMC9846943]. <https://doi.org/10.3748/wjg.v29.i2.241>.
 15. Kolesova O, Vanaga I, Laivacuma S, Derovs A, Kolesovs A, Radzina M, et al. Intriguing findings of liver fibrosis following COVID-19. *BMC Gastroenterol.* 2021;**21**(1):370. [PubMed ID: 34635073]. [PubMed Central ID: PMC8503733]. <https://doi.org/10.1186/s12876-021-01939-7>.
 16. Li R, Qin C. Expression pattern and function of SARS-CoV-2 receptor ACE2. *Biosaf Health.* 2021;**3**(6):312-8. [PubMed ID: 34466800]. [PubMed Central ID: PMC8393493]. <https://doi.org/10.1016/j.bsheal.2021.08.003>.
 17. Nouri M, Soleiman-Meigooni S, Mohammadi S, Sakhabakhsh M, Yaghmayee R, Fotoohi M. Neuropathology of 30 deceased patients with COVID-19: A case series in Tehran, Iran. *Ann Med Surg (Lond).* 2023;**85**(2):271-5. [PubMed ID: 36845816]. [PubMed Central ID: PMC9949774]. <https://doi.org/10.1097/MS9.0000000000000203>.
 18. Soleiman-Meigooni S, Yaghmayee R, Mohammadi S, Ahmadi M, Sakhabakhsh M, Hamidi-Farahani R, et al. Cardio-Pulmonary Histopathology with Clinical Correlations of Deceased Patients with COVID-19: A Case Series in Tehran, Iran. *Arch Iran Med.* 2023;**26**(5):252-60. [PubMed ID: 38301088]. [PubMed Central ID: PMC10685862]. <https://doi.org/10.34172/aim.2023.39>.
 19. Nava-Santana C, Rodríguez-Armida M, Jimenez JV, Vargas-Parra N, Leon DEA, Campos-Murguía A, et al. Clinicopathologic characteristics of severe COVID-19 patients in Mexico City: A post-mortem analysis using a minimally invasive autopsy approach. *PLoS One.* 2022;**17**(3):e0262783. [PubMed ID: 35239660]. [PubMed Central ID: PMC8893646]. <https://doi.org/10.1371/journal.pone.0262783>.
 20. Gheban-Rosca IA, Gheban BA, Pop B, Mironescu DC, Siserman VC, Jianu EM, et al. A histopathological analysis of extrapulmonary lesions in fatal coronavirus disease (COVID-19). *Pathol Res Pract.* 2024;**260**:155373. [PubMed ID: 38901140]. <https://doi.org/10.1016/j.prp.2024.155373>.
 21. Azizmohammadi S, Azizmohammadi S, Dahmardeh S, Hossein A, Shokouh SJH, Mohajeri-Iravani M, et al. Analysis of 239 ordinary and severe cases of COVID-19: Clinical features and treatment. *Eur J Transl Myol.* 2021;**31**(3). [PubMed ID: 34579515]. [PubMed Central ID: PMC8495366]. <https://doi.org/10.4081/ejtm.2021.9579>.
 22. Hamidi Farahani R, Gholami M, Hazrati E, Hosseini Rouzbahani N, Hejripour Z, Soleiman-Meigooni S, et al. Clinical Features of ICU Admitted and Intubated Novel Corona Virus-infected Patients in Iran. *Arch Clin Infect Dis.* 2020;**15**(2). e103295. <https://doi.org/10.5812/archcid.103295>.
 23. Diaz LA, Idalsoaga F, Cannistra M, Candia R, Cabrera D, Barrera F, et al. High prevalence of hepatic steatosis and vascular thrombosis in COVID-19: A systematic review and meta-analysis of autopsy data. *World J Gastroenterol.* 2020;**26**(48):7693-706. [PubMed ID: 33505145]. [PubMed Central ID: PMC7789052]. <https://doi.org/10.3748/wjg.v26.i48.7693>.
 24. Haji Esmaeil Memar E, Mamishi S, Sharifzadeh Ekbatani M, Alimadadi H, Yaghmaei B, Chegini V, et al. Fulminant hepatic failure: A rare and devastating manifestation of Coronavirus disease 2019 in an 11-year-old boy. *Arch Pediatr.* 2020;**27**(8):502-5. [PubMed ID: 33069564]. [PubMed Central ID: PMC7522641]. <https://doi.org/10.1016/j.arcped.2020.09.009>.
 25. Barnes E. Infection of liver hepatocytes with SARS-CoV-2. *Nat Metab.* 2022;**4**(3):301-2. [PubMed ID: 35347317]. <https://doi.org/10.1038/s42255-022-00554-4>.
 26. Ali FEM, Mohammedsahleh ZM, Ali MM, Ghogor OM. Impact of cytokine storm and systemic inflammation on liver impairment patients infected by SARS-CoV-2: Prospective therapeutic challenges. *World J Gastroenterol.* 2021;**27**(15):1531-52. [PubMed ID: 33958841]. [PubMed Central ID: PMC8058655]. <https://doi.org/10.3748/wjg.v27.i15.1531>.
 27. Nunes MC, Hale MJ, Mahtab S, Mabena FC, Dlodlu N, Baillie VL, et al. Clinical characteristics and histopathology of COVID-19 related deaths in South African adults. *PLoS One.* 2022;**17**(1). e0262179. [PubMed ID: 35051205]. [PubMed Central ID: PMC8775212]. <https://doi.org/10.1371/journal.pone.0262179>.
 28. Yadav J, Goel G, Purwar S, Saigal S, Tandon A, Joshi A, et al. Clinical, Virological, and Pathological Profile of Patients Who Died of COVID-19: An Autopsy-Based Study From India. *Cureus.* 2022;**14**(3). e23538. <https://doi.org/10.7759/cureus.23538>.
 29. Elsoukkary SS, Mostyka M, Dillard A, Berman DR, Ma LX, Chadburn A, et al. Autopsy Findings in 32 Patients with COVID-19: A Single-Institution Experience. *Pathobiology.* 2021;**88**(1):56-68. [PubMed ID: 32942274]. [PubMed Central ID: PMC7573917]. <https://doi.org/10.1159/000511325>.