J Nepal Health Res Counc 2026 Jan-March; 24 (70): 159-166

Association of Laboratory Parameters with Clinical Outcomes of SARS-CoV-2 Patients

Mamata Sharma Neupane,[1] Hafizah Che Hassan,[1] Surendra Kumar Uranw[2], Raj Kumar Mehta[3]

[1]Lincoln University College, Malaysia, [2]B.P.Koirala Institute of Health Sciences, Nepal, [3]Chitwan Medical College, Bharatpur, Nepal.

ABSTRACT

Background: The COVID-19 pandemic has imposed a significant burden on healthcare systems, particularly in resource-limited settings. Laboratory biomarkers may help predict disease outcomes and guide clinical decisions. However, there is limited evidence from Nepal regarding the prognostic value of these markers among hospitalized COVID-19 patients.

Methods: A hospital-based observational study was conducted among 348 RT-PCR–confirmed COVID-19 patients admitted to the COVID ICUs and wards of a tertiary care hospital in Nepal between March 2022 and June 2023. Data on sociodemographic characteristics, haematological parameters, liver and inflammatory markers, urine microscopy, and SARS-CoV-2 molecular gene markers were collected prospectively. Clinical outcomes were categorized as survived or died. Descriptive statistics, chi-square tests, and binary logistic regression were used to analyse associations and identify predictors of mortality.

Results: Out of the total 348 patients included in the study, 71 patients (20.4%) died, while 277 patients (79.6%) survived. Among 348 patients, significant associations with mortality were observed for lymphopenia ($p = .001$), neutrophilia ($p < .001$), elevated ALT ($p < .001$), elevated AST ($p = .001$), and positive D-dimer ($p < .001$). High viral load (Ct value ≤20) for E, N, and ORF1ab ab genes was also significantly associated with mortality in univariate analysis. Logistic regression identified D-dimer positivity (OR = 0.027, $p = .001$) and elevated ALT (OR = 0.356, $p = .016$) as independent predictors of mortality.

Conclusions: Laboratory parameters, especially lymphocyte count, D-dimer, and ALT levels, are valuable prognostic indicators of clinical outcomes in hospitalized COVID-19 patients. Integrating these markers into clinical assessment may enhance early risk stratification and resource allocation.

Keywords: Biomarkers; C-Reactive protein; COVID-19; critical care; d-dimer.

INTRODUCTION

Coronavirus disease 2019 (COVID-19), caused by SARS-CoV-2, has severely impacted global health systems. Laboratory biomarkers play a crucial role in predicting disease severity, guiding clinical management, and allocating intensive care resources in hospitalized patients.[1]

Previous studies have demonstrated that elevated levels of inflammatory markers such as C-reactive protein (CRP), D-dimer, and lactate dehydrogenase (LDH), along with lymphopenia, are associated with increased mortality in COVID-19 patients.[2,3] However, there is limited evidence from low- and middle-income countries like Nepal, where variations in healthcare delivery, comorbidities, and patient demographics may influence outcomes.

This study aims to explore the association between laboratory parameters and clinical outcomes among SARS-CoV-2 patients admitted to a tertiary care hospital in Nepal. This will help identify locally relevant prognostic indicators for improving patient triage and treatment strategies.

METHODS

This hospital-based observational study was conducted among RT-PCR-confirmed COVID-19 patients admitted to

Correspondence: Mamata Sharma Neupane, Lincoln University College, Malaysia. Email: sharma.mamata@cmc.edu.np, Phone:+9779855057357.

COVID ICU I, Respiratory ICU II, and designated COVID wards of a tertiary care hospital in central Nepal. The hospital, with a dedicated capacity of 26 ICU beds and 100 ward beds, served as a major referral center managing a substantial caseload during the COVID-19 pandemic. Ethical approval was obtained from the respective institutions (Ref: LUC/INT/113/2022 and NHRC/072/2022PhD), and permission for data collection was granted by the study site (Ref: CMC-IRC/078/079/149). Written informed consent was obtained from patients or their legally authorized representatives when direct consent was not feasible. Privacy and confidentiality were strictly maintained throughout the research process.

The study population included all adult patients (≥18 years) with laboratory-confirmed SARS-CoV-2 infection diagnosed by reverse transcription–polymerase chain reaction (RT-PCR) who were admitted to COVID-designated units of Chitwan Medical College Teaching Hospital (CMCTH) during the study period. Inclusion criteria comprised: (1) age ≥18 years; (2) confirmed SARS-CoV-2 infection; (3) admission to COVID ward, COVID ICU, or isolation units; and (4) availability of complete clinical and outcome data. Exclusion criteria included: (1) patients below 18 years of age; (2) incomplete or missing key clinical records; and (3) patients transferred to or from other institutions before definitive outcome determination.

The required sample size was calculated based on logistic regression sample size estimation methods[4], assuming an event proportion (P = 0.20) and an odds ratio (r = 0.7) at one standard deviation above the mean of the covariate. The level of significance was set at α = 0.05 (one-tailed) with a statistical power of 80% (1−β = 0.80). According to these assumptions, the minimum required sample size was estimated to be ≥348 participants, ensuring sufficient statistical power and precision in evaluating predictor–outcome relationships. A consecutive sampling technique was used to recruit eligible patients who met the inclusion criteria during the study period.

Participants were recruited consecutively from March 2022 to June 2023, including adults (≥18 years) admitted to the COVID ICUs and wards. Data were collected prospectively using a standardized structured proforma, encompassing sociodemographic details, laboratory findings (hematological, liver function, inflammatory markers, urine microscopy, and SARS-CoV-2 molecular gene markers), and clinical outcomes. Standard infection prevention protocols were maintained during data collection. Patients were followed until discharge, with outcomes classified as either survived or deceased.

All collected data were reviewed for completeness, accuracy, and consistency prior to analysis. Descriptive statistics including frequencies, percentages, means with standard deviations (for normally distributed data), and medians with interquartile ranges (for skewed data) were used to summarize the variables. Inferential statistics, including chi-square tests and binary logistic regression, were applied to assess associations between laboratory parameters and patient outcomes (survived vs. died). Binary logistic regression analysis was used to identify independent predictors of mortality, and results were reported using odds ratios (Exp(B)), 95% confidence intervals, and p-values. All analyses were conducted using SPSS version 25, with a p-value <0.05 indicating statistical significance.

RESULTS

A total of 348 COVID-19 patients admitted in COVID ICUs and wards were enrolled. The age of the patients ranged between 18 to 97 years. The median age of the COVID-19 patient was 54.0 (IQR:68-40). Males comprised 188 (54.0%) and female comprised 160 (46.0%) of the total number of patients. Of the 348 patients, 71 (20.4%) died and 277 (79.6%) survived.

Table 1 presents the distribution of key laboratory parameters among 348 hospitalized COVID-19 patients. Among hematological parameters, anemia was found in 44.5% of patients, and abnormal (elevated) RBC counts were observed in 17.8%. Elevated total leukocyte counts were present in 31%, while leukopenia was observed in 8.3% of cases. Lymphopenia (<20%) was the most prevalent (59.2%), and a majority (67.8%) had neutrophilia (>70%). Thrombocytopenia (platelet count <150,000) was present in 35.9% of the cohort.

Regarding liver and inflammatory markers, elevated ALT and AST levels were noted in 34.5% and 49.4% of patients, respectively. Hypoalbuminemia (<3.4 g/dL) was common (60.6%), while elevated CRP levels (>100 mg/L) were found in 41.4%, indicating a significant inflammatory response. D-dimer positivity (≥0.5 ng/mL) was observed in 73% of patients. Urine microscopy revealed that 7.8% and 8.9% of patients had elevated epithelial and pus cell counts, respectively. Molecular testing showed high SARS-CoV-2 viral loads (Ct value 0–20) in 60.6% for E gene, 56.3% for N gene, and 57.2% for ORF1ab gene.

Table 2 outlines the bivariate associations between laboratory parameters and patient outcomes (survived vs. died). Hemoglobin levels showed a significant association with survival (p = .041), with anemia more common among non-survivors. Elevated total leukocyte counts (p = .003), lymphopenia (p = .001), and neutrophilia (p < .001) were significantly associated with mortality. While thrombocytopenia showed a trend, it did not reach statistical significance (p = .072).

Among biochemical markers, elevated ALT (p < .001) and AST (p = .001) levels were significantly associated with death. However, albumin levels did not show a significant association (p = .724). D-dimer positivity showed a strong association with mortality (p < .001), while CRP levels approached but did not achieve statistical significance (p = .089). For urine microscopy, epithelial cell counts (p = .014) were significantly associated with death, whereas pus cell counts were not. All three molecular markers (E gene, N gene, ORF1ab) showed significant associations with mortality (p-values = .028, .016, and .004, respectively), with higher viral loads linked to poorer outcomes.

Table 3 presents the results of binary logistic regression analysis identifying predictors of mortality among COVID-19 patients. Elevated ALT (SGPT) levels were independently associated with reduced odds of survival (OR = 0.356, p = .016). D-dimer positivity remained a strong predictor of mortality (OR = 0.027, p = .001), confirming its prognostic utility. Other predictors such as abnormal total leukocyte count, lymphocyte count, and ORF1ab gene expression showed trends but did not reach statistical significance in the multivariable model.

Notably, E gene (Ct >20) and N gene expression levels were not significantly associated with outcomes in the adjusted model, despite their significant bivariate associations. Similarly, high neutrophil or low albumin levels were not included in the final regression, suggesting potential confounding or interaction effects. Overall, the model underscores the importance of ALT and D-dimer as robust laboratory predictors of mortality in hospitalized COVID-19 patients.

Table 1. Laboratory parameters (Hematological Parameters, Liver and Inflammatory Markers, Urine Microscopy, COVID-19 Molecular Markers- Genes) of hospitalized COVID-19 patients. (n=348)

Variable	Category	Frequency	Percentage
Hematological Parameters			
Hb (g/dL)	Anemia	155	44.5
	Normal	193	55.5
RBC (million/µL)	Normal	286	82.2
	abnormal (excessive)	62	17.8
WBC / Total leukocyte count (cells/µL)	Below normal <4000	29	8.3
	Normal 4000-11000	211	60.6
	Above	108	31.0
Lymphocyte count (cells/µL)	<20	206	59.2
	20-40 Normal	123	35.3
	>40	19	5.5
Neutrophil count (cells/µL)	<40	10	2.9
	40-70 normal	102	29.3
	>70	236	67.8
Platelet count (cells/µL)	<150000	125	35.9
	150000-400000 Normal	223	64.1
Biochemical Parameters (Liver and Inflammatory Markers)			
ALT (SGPT) (U/L)	Normal	228	65.5
	More than normal >45	120	34.5
AST (SGOT) (U/L)	Normal <40	176	50.6
	Abnormal >40	172	49.4

Table 1. Laboratory parameters (Hematological Parameters, Liver and Inflammatory Markers, Urine Microscopy, COVID-19 Molecular Markers- Genes) of hospitalized COVID-19 patients. (n=348)

Variable	Category	Frequency	Percentage
Albumin (g/L)	Low (<3.4)	211	60.6
	Normal (3.4-5.4)	110	31.6
	High (>5.4)	27	7.8
CRP (C-reactive protein) (mg/L)	Normal (<10mg/L)	34	9.8
	Mild to moderate (10-100 mg/L)	170	48.9
	Severe (>100 mg/L)	144	41.4
D-dimer (ng/mL FEU)	Normal <0.5	94	27.0
	Positive ≥0.5	254	73.0
Urine Microscopy Parameters			
Epithelial cell in urine (HPF)	1-5	321	92.2
	6-16	27	7.8
Pus cell in urine (HPF)	0-5	317	91.1
	6-16	31	8.9
COVID-19 Molecular Markers (Genes)			
E gene (Ct Value)	High viral load (0-20)	211	60.6
	Low viral load (>20-40)	137	39.4
N gene (Ct Value)	High viral load (0-20)	196	56.3
	Low viral load (>20-40)	152	43.7
ORF1ab (Ct Value)	High viral load (0-20)	199	57.2
	Low viral load (>20-40)	149	42.8

Table 2. Association of Laboratory Parameters (Hematological Parameters, Liver and Inflammatory Markers, Urine Microscopy Parameters, COVID-19 Molecular Markers - Genes) with Clinical Outcomes in COVID-19 Patients. (n=348)

Variables	Outcome		χ^2	p-value
	Survived No. (%)	Died No. (%)		
Hematological Parameters				
Hb				
Anemia	131(84.5)	24(15.5)	4.163	.041*
Normal	146(75.6)	47(24.4)		
RBC				
Normal	225(78.7)	61(21.3)	.848	.357
abnormal (excessive)	52(83.9)	10(16.1)		
Total leukocyte count				
Below normal <4000	25(86.2)	4(13.8)	11.890	.003**
Normal 4000-11000	178(84.4)	33(15.6)		
Above	74(68.5)	34(31.5)		
Lymphocyte count				
<20	150(72.8)	56(27.2)	14.679	.001**
20-40 Normal	109(88.6)	14(11.4)		
>40	18(94.7)	1(5.3)		
Neutrophil count				
<40	10(100.0)	0(0.0)	15.989	.000**
40-70 normal	93(91.2)	9(8.8)		
>70	174(73.7)	62(26.3)		

Table 2. Association of Laboratory Parameters (Hematological Parameters, Liver and Inflammatory Markers, Urine Microscopy Parameters, COVID-19 Molecular Markers - Genes) with Clinical Outcomes in COVID-19 Patients. (n=348)

Variables	Outcome		χ^2	p-value
	Survived No. (%)	Died No. (%)		
Platelet				
<150000	93(74.4)	32(25.6)	3.245	.072
150000-400000 Normal	184(82.5)	39(17.5)		
Biochemical Parameters (Liver and Inflammatory Markers)				
ALT SGPT				
Normal	198(86.8)	30(13.2)	21.368	.000
More than normal >45	79(65.8)	41(34.2)		
AST SGOT				
Normal <40	153(86.9)	23(13.1)	11.794	.001
Abnormal >40	124(72.1)	48(27.9)		
Albumin				
Low (<3.4)	170(80.6)	41(19.4)	.647	.724
Normal (3.4-5.4)	87(79.1)	23(20.9)		
High (>5.4)	20(74.1)	7(25.9)		
CRP				
Normal (<10mg/L)	30(88.2)	4(11.8)	4.840	.089**
Mild to moderate (10-100 mg/L)	140(82.4)	30(17.6)		
Severe (>100 mg/L)	107(74.3)	37(25.7)		
D - dimer				
Normal <0.5	93(98.9)	1(1.1)	29.658	.000**
Positive >=0.5	184(72.4)	70(27.6)		
Urine Microscopy Parameters				
Epithelial cell in urine				
1-5	251(78.2)	70(21.8)	5.026	.014**
6-16	26(96.3)	1(3.7)		
Pus cell in urine				
0-5	255(80.4)	62(19.6)	1.561	.212
6-16	22(71.0)	9(29.0)		
Molecular parameters/ covid-19 molecular markers (genes)				
E gene				
High viral load (0-20)	176(83.4)	35(16.6)	4.802	.028*
Low viral load (>20-40)	101(73.7)	36(26.6)		
N gene				
High viral load (0-20)	165(84.2)	31(15.8)	5.811	.016*
Low viral load (>20-40)	112(73.7)	40(26.3)		
ORF1ab				
High viral load (0-20)	169(84.9)	30(15.1)	8.121	.004*
Low viral load (>20-40)	108(72.5)	41(27.5)		

** *denotes fisher exact test*

Table 3. Binary logistic regression analysis for Prediction of Clinical Outcomes Using Laboratory Parameters (Hematological Parameters, Liver and Inflammatory Markers, Urine Microscopy Parameters, COVID-19 Molecular Markers - Genes) in COVID-19 Patients.

Predictor Variable	B	Exp (B) Odds ratio	Significance p	95% CI for EXP(B)	
				Lower	Upper
Hb					
Anemia	.381	1.463	.319	.692	3.095
Normal	Ref	-	-	-	-
TLC					
Below normal <4000	-.007	.994	.993	.213	4.632
Normal 4000-11000	Ref	-	-	-	-
Above >11000	-.355	.701	.370	.323	1.524
Lymphocyte					
<20	-.879	.415	.054	.170	1.015
20-40 Normal	Ref	-	-	-	-
>40	.939	2.556	.423	.257	25.423
ALT SGPT					
Normal	Ref	-	-	-	-
Abnormal (High)	-1.034	.356	.016	.153	.825
AST/ SGOT					
Normal (<40)	Ref	-	-	-	-
High (>40)	-.455	.635	.293	.272	1.482
D Dimer					
Normal (<0.5)	Ref	-	-	-	-
Positive (>=0.5)	-3.624	.027	.001	.003	.216
Epithelial cell(urine)					
1-5	Ref	-	-	-	-
6-16	1.363	3.909	.244	.394	38.793
E gene					
High viral load (0-20)	Ref	-	-	-	-
Low viral load (>20-40)	.679	1.973	.336	.494	7.876
N gene					
High viral load (0-20)	Ref	-	-	-	-
Low viral load (>20-40)	-.250	.779	.720	.199	3.052
ORF1ab					
High viral load (0-20)	Ref	-	-	-	-
Low viral load (>20-40)	-1.371	.254	.066	.059	1.095

DISCUSSION

This study investigated the association of laboratory parameters with clinical outcomes among SARS-CoV-2 patients admitted to a tertiary care hospital in Nepal. Several hematological, biochemical, and molecular parameters showed significant associations with mortality, aligning with trends reported globally, while also revealing unique context-specific patterns.

Our findings indicate that lymphopenia and neutrophilia were significantly associated with mortality. This aligns with prior studies, such as Tan et al., who reported that lymphopenia is a hallmark of severe COVID-19, likely reflecting virus-induced T-cell exhaustion or destruction.[5] Similarly, high neutrophil counts, indicative of an exaggerated inflammatory response, have been reported as predictors of poor prognosis.[6] A study by Liu et al. also found elevated neutrophil-to-lymphocyte ratios (NLR) to be strongly associated with disease severity.[7] Additional findings by Lagunas-Rangel emphasized that an elevated NLR serves as a cost-effective marker for early risk stratification in clinical settings.[8] The consistency of these hematological trends

across populations reinforces their prognostic reliability.

Elevated ALT and AST levels were significantly associated with death in our cohort. This is consistent with Zhang et al., who reported hepatic injury in severe COVID-19 cases due to either viral cytopathic effects or drug-induced hepatotoxicity.[9] A systematic review by Li et al. also found that liver enzyme elevations, especially in AST, correlated with ICU admissions and fatal outcomes.[10] Furthermore, high D-dimer levels were robustly predictive of mortality, supporting findings by Tang et al., where elevated D-dimer was associated with increased coagulation and thrombotic events in non-survivors.[11] The lack of a significant association between albumin levels and mortality in our study differs from some prior studies that reported hypoalbuminemia as a predictor of severe disease.[12] This discrepancy may reflect nutritional or hydration status differences in our setting, or timing of albumin measurements during the illness course.

Elevated epithelial cell counts in urine were significantly linked to mortality, possibly indicating renal tubular injury, consistent with findings from Pei et al. who reported that kidney involvement in COVID-19 correlates with disease severity.[13] However, pus cell counts were not significantly associated with outcomes, perhaps suggesting that bacterial coinfection was not a major contributor to mortality in this cohort. A multicenter study by Hirsch et al. also identified acute kidney injury as an independent predictor of mortality in hospitalized COVID-19 patients,[14] further supporting our findings.

Molecular markers, including E, N, and ORF1ab genes, showed significant associations between higher viral load (lower Ct values) and increased mortality in bivariate analysis. These results align with the findings of Magleby et al., who demonstrated that high viral load was associated with increased risk of intubation and death.[15] Similar conclusions were drawn by Fajnzylber et al., who found persistent high viral RNA levels to be significantly associated with worse clinical trajectories.[16] However, in multivariable analysis, only D-dimer and ALT remained significant, suggesting these markers may have more independent prognostic value than viral load in later stages of the disease.

CONCLUSIONS

This observational study highlights the significant association of various laboratory parameters with clinical outcomes among hospitalized COVID-19 patients in a tertiary care center in Nepal. Hematological markers such as lymphopenia and neutrophilia, elevated liver enzymes (ALT, AST), and raised D-dimer levels emerged as strong predictors of mortality. Additionally, lower Ct values for viral genes (E, N, ORF1ab) indicated higher viral loads and were associated with poorer outcomes. This study highlights that laboratory parameters, particularly lymphocyte count, D-dimer, and ALT levels serve as readily available, affordable, and valuable indicators for early risk stratification and outcome prediction in COVID-19 patients, especially within resource-constrained settings such as Nepal.

Clinicians should incorporate routine assessment of these biomarkers at the time of admission to guide triage decisions, prioritize intensive care monitoring, and potentially initiate early interventions for high-risk patients. Future multicentric and prospective studies are recommended to validate these findings and develop standardized clinical algorithms for COVID-19 management in similar healthcare contexts.

ACKNOWLEDGEMENTS

The authors would like to express their sincere gratitude to Prof. Dr. Harish Chandra Neupane and Prof. Dr. Gopendra Prasad Deo for their invaluable support in facilitating the data collection process. We also extend our appreciation to the COVID ICU and COVID Ward in-charges, as well as the dedicated staff involved in data collection.

CONFLICTING INTEREST

The authors declare no other conflicts of interest.

REFERENCES

1. Henry BM, de Oliveira MHS, Benoit S, Plebani M, Lippi G. Hematologic, biochemical and immune biomarker abnormalities associated with severe illness and mortality in coronavirus disease 2019 (COVID-19): a meta-analysis. Clin Chem Lab Med. 2020;58(7):1021–8. doi: https://doi.org/10.1515/cclm-2020-0369

2. Zhou F, Yu T, Du R, Fan G, Liu Y, Liu Z, et al. Clinical course and risk factors for mortality of adult inpatients with COVID-19 in Wuhan, China: a retrospective cohort study. Lancet. 2020;395(10229):1054–62. doi: https://doi.org/10.1016/S0140-6736(20)30566-3

3. Wynants L, Van Calster B, Collins GS, Riley RD,

Heinze G, Schuit E, et al. Prediction models for diagnosis and prognosis of COVID-19: systematic review and critical appraisal. BMJ. 2020;369:m1328. doi: https://doi.org/10.1136/bmj.m1328

4. Hsieh FY. Sample size tables for logistic regression. Stat Med. 1989;8(7):795-802. [Article]

5. Tan L, Wang Q, Zhang D, Ding J, Huang Q, Tang YQ, et al. Lymphopenia predicts disease severity of COVID-19: a descriptive and predictive study. Signal Transduct Target Ther. 2020;5(1):33. doi: https://doi.org/10.1038/s41392-020-0148-4

6. Qin C, Zhou L, Hu Z, Zhang S, Yang S, Tao Y, et al. Dysregulation of immune response in patients with COVID-19 in Wuhan, China. Clin Infect Dis. 2020;71(15):762-8. doi: https://doi.org/10.1093/cid/ciaa248

7. Liu Y, Du X, Chen J, Jin Y, Peng L, Wang HH, et al. Neutrophil-to-lymphocyte ratio as an independent risk factor for mortality in hospitalized patients with COVID-19. J Infect. 2020;81(1):e6-e12.doi: https://doi.org/10.1016/j.jinf.2020.04.002

8. Zhang C, Shi L, Wang FS. Liver injury in COVID-19: management and challenges. Lancet Gastroenterol Hepatol. 2020;5(5):428-30.doi: https://doi.org/10.1016/S2468-1253(20)30057-1

9. Tang N, Li D, Wang X, Sun Z. Abnormal coagulation parameters are associated with poor prognosis in patients with novel coronavirus pneumonia. J Thromb Haemost. 2020;18(4):844-7.doi: https://doi.org/10.1111/jth.14768

10. Huang J, Cheng A, Kumar R, Fang Y, Chen G, Zhu Y, et al. Hypoalbuminemia predicts the outcome of COVID-19 independent of age and comorbidity. J Med Virol. 2020;92(10):2152-8. doi: https://doi.org/10.1002/jmv.26003

11. Pei G, Zhang Z, Peng J, Liu L, Zhang C, Yu C, et al. Renal involvement and early prognosis in patients with COVID-19 pneumonia. J Am Soc Nephrol. 2020;31(6):1157-65.doi: https://doi.org/10.1681/ASN.2020030276

12. Magleby R, Westblade LF, Trzebucki A, Simon MS, Rajan M, Park J, et al. Impact of SARS-CoV-2 viral load on risk of intubation and mortality among hospitalized patients with coronavirus disease 2019. Clin Infect Dis. 2021;73(11):e4197-205. doi: https://doi.org/10.1093/cid/ciaa851

13. Lagunas-Rangel FA. Neutrophil-to-lymphocyte ratio and lymphocyte-to-C-reactive protein ratio in patients with severe coronavirus disease 2019 (COVID-19): A meta-analysis. J Med Virol. 2020;92(10):1733-4. doi: https://doi.org/10.1002/jmv.25819

14. Li J, Fan JG. Characteristics and mechanism of liver injury in 2019 coronavirus disease. J Clin Transl Hepatol. 2020;8(1):13-7. doi: https://doi.org/10.14218/JCTH.2020.00019

15. Hirsch JS, Ng JH, Ross DW, Sharma P, Shah HH, Barnett RL, et al. Acute kidney injury in patients hospitalized with COVID-19. Kidney Int. 2020;98(1):209-18. doi: https://doi.org/10.1016/j.kint.2020.05.006

16. Fajnzylber J, Regan J, Coxen K, Corry H, Wong C, Rosenthal A, et al. SARS-CoV-2 viral load is associated with increased disease severity and mortality. Nat Commun. 2020;11(1):5493.doi: https://doi.org/10.1038/s41467-020-19057-5