Estimation of Liver and Kidney Biomarkers for People During Their Infection with SARA-COV-2 (COVID-19) Virus in Nineveh Governorate

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ABSTRACT

SARS-CoV-2 harms organs, kidneys, and liver. Most seriously ill COVID-19 patients had liver and renal failure. Our study examined liver and renal function in moderate and severe COVID-19 individuals. The study comprised 56 COVID-19 patients in Nineveh Governorate (32 men, 24 women). Their ages ranged between (30 and 65) years. These patients were divided into two groups according to the severity of the disease. The first group included 25 patients with mild disease, and the second included 31 patients with severe disease. Liver and kidney function and other biomarkers were measured. The results showed that all severe patients had a significant difference in alanine aminotransferase (ALT), aspartate aminotransferase (AST), bilirubin urea, and creatinine compared with mild patients (all $p<0.0001$ except creatinine, $p= 0.014$). Moreover, patients with severe disease were older than those with mild disease. Males were also more affected by Covid-19. We conclude that patients with COVID-19, especially those with severe disease, suffer from liver and kidney dysfunction. Therefore, the severity of this disease can be predicted by examining the levels of ALT, AST, bilirubin, urea, and creatinine. Moreover, the progression from mild to severe disease in COVID-19 patients can be predicted by the combination of ALT, AST, bilirubin, urea, and creatinine levels.

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1. Introduction

An international sanitary emergency has been sparked by the infectious disease COVID-19, brought on by the SARS-CoV-2 virus. COVID-19 can cause asymptomatic illness, mild-to-severe pneumonia, or none [1-3]. The World Health Organization (WHO) reported the discovery of a novel virus on 30 January 2020 and proclaimed a worldwide emergency. On 17 November 2021, the illness claimed the lives of more than 5 million people globally. Four of the seven coronavirus types that impact humans are quite prevalent. The infectious human coronaviruses SARS-CoV, MERS-CoV, and SARS-CoV-2 are linked to fatal respiratory infections and severe [2, 4, 5]. Earlier studies have shown that the SARS-CoV-2 virus enters the lungs through the angiotensin-converting enzyme 2 (ACE2) receptor [4, 6, 7], which, in severe cases, can result in severe lung fibrosis and consolidation, as well as a substantial fatality rate [4, 8]. Compared to mild patients, severe patients displayed high fever, cough, dyspnea, and anorexia rates [4]. Because COVID-19 can harm various organs, particularly the kidneys, the primary objective of this study was to evaluate the effect of various aspects of kidney damage that can be brought on by COVID-19 through a narrative review study [9]. Additional research has shown that this virus can harm the liver. [4, 10-13] and kidneys[4, 14-16]. because ACE2 is found in both organs [4]. Studies show that patients with severe critical disease had more lab evidence of cytokine storms linked to severe pneumonia, kidney and liver damage, and other abnormal lab data [17]. To better understand how COVID-19 patients transition from mild to severe disease, our study focuses on analyzing the liver and kidney biomarkers in those patients.

2. Experimental

2.1. population study:

The study involved 56 patients with COV D-19 virus in Nineveh Governorate of both sexes (32 males, 24 females); their ages ranged (from 30- 65) years. Patients were divided into two groups according to the severity of the condition. The first group included 25 patients with mild cases of both sexes (13 men and 12 females), and their ages ranged from (35- 55). The second group consisted of 31 individuals with severe illnesses affecting both sexes (18 males and 13 females), ranging from (45-65). disorders. The study subjects were the Nineveh Health Department-licensed outpatient medical clinics and the individuals intended for the clinics' specialized services. Additionally, the Ethics.

2.2. Samples Collection:

Collect serum samples carried out by 5 ml of venous blood was drawn, placed in a gel tube, and left at 37 ° C for 10 minutes. Afterward, the gel tube was centrifuged, and the serum was collected and stored at -20 ° C until the analysis was performed.

2.3. Variables Assay

2.3.1 Assay of ALT and AST serum:

Utilizing the Japanese-made Fuji automated analysis instrument and according to the guidelines provided by the creator.

2.3.2 Estimate of bilirubin level:

A total bilirubin test kit (Randox Laboratories Ltd., UK) was used to quantify the bilirubin level. A detergent-free albumin-bound bilirubin, which subsequently interacted with 2,4-dichloroaniline, created a colored substance whose absorbance was measured at 546 nm [18].

2.3.2 Assay of Urea and creatinine concentrations:

Urea concentrations were determined using a kit provided by Biosystems (Rifai, 2017), and creatinine concentrations were determined using equipment supplied by Biolabo Company.

2.3. Statistical analysis:

The statistical software package for social sciences (SPSS) version 25.0 was used to compile the results. The data is shown as a mean and standard deviation (SD). Statistical significance is deemed to exist when the p-value is less than 0.05.

3. Results and discussion

A comparison of the baseline characteristics of the patients in two different groups. We categorized the 56 patients into groups according to the severity of the disease; the first group included 25 mild patients and the second group (31) patients with severe cases (Table 1).

Table 1 Characteristics of the Patients.		
Characterise of patients	Mild, n=25	**Sever, n= 31**
Age (years)	47.4± 10.6	54.8 ± 10.9
Sex (32 M/ 24F)	13/12	18/13
IgG (U/ml)	12.7 ± 3.3	10.6 ± 2.4
IgM (U/ml)	1.3 ± 0.6	8.4 ± 1.2
IgG = Immunoglobulin G; IgM= Immunoglobulin M; M =Male; F= Female; n=Number.		

The age of patients who presented with severe symptoms was significantly higher than that of patients who presented with mild symptoms, as shown in Table (2) and Figure 1. Comparison of mean ages (57.42 ± 14.28 vs. 43.86 ± 15.71, $p \leq 0.001$). These findings are consistent with Qu et al. 2021 [4]. These findings are consistent with the previous study's findings, which indicated that elderly patients have a greater propensity to develop into severe patients and that a more significant percentage of severe patients exhibited high temperature, cough, and dyspnea. Additionally, inflammatory responses in severe patients were more intense than in less severe individuals [4].In addition, mild and severe patients were contrasted between liver and kidney functions. Except for creatinine ($p \leq .014$), we discovered that the AST, ALT, bilirubin, urea, and creatinine levels of the severe patients were significantly ($p \leq 0.0001$) higher than those of the mild patients. As illustrated in Figures 2, 3, 4, 5, and 6 and Table 2, we found that these results are consistent with the findings of Qu et al., 2021 [4]. For patients with COVID-19, we showed that ALT, AST, bilirubin, urea, and creatinine independently predicted the progression from mild to after-severe. The causes of liver and renal impairment caused by SARS-CoV-2 infection are currently unknown [4, 17].

Table 2: Compares the liver and renal functions of COVID-19 patients (mild and severe).

Cases	Mild	Sever	P- value
Mean ± SD			
Age	47.4±10.6	**54.8±10.9	0.014
AST (GOT) (U/L)	29.4±7.1	***50.3±4.5	0.0001
ALT (GPT) (U/L)	33.9 ± 3.7	***54.7± 5.7	0.0001
Bilirubin (µmol/L)	7.9±1.5	***11± 3.2	0.0001
Urea (mg/ dl)	32.8±7.6	***63.5±7.1	0.0001
Creatinine(mg/dl)	0.67±0.19	**2 ±0.4	0.014

AST stands for aspartate transaminase; GOT stands for glutamic-oxaloacetic transaminase; ALT stands for alanine aminotransferase; GPT stands for glutamate-pyruvic transaminase; mg stands for milligram; dl stands for deciliter; U stands for the unit; L stands for liter; SD stands for standard deviation; ** indicates a significant difference at the level of (p≤0.01), *** indicates a significant difference at the level of (p≤0.0001).

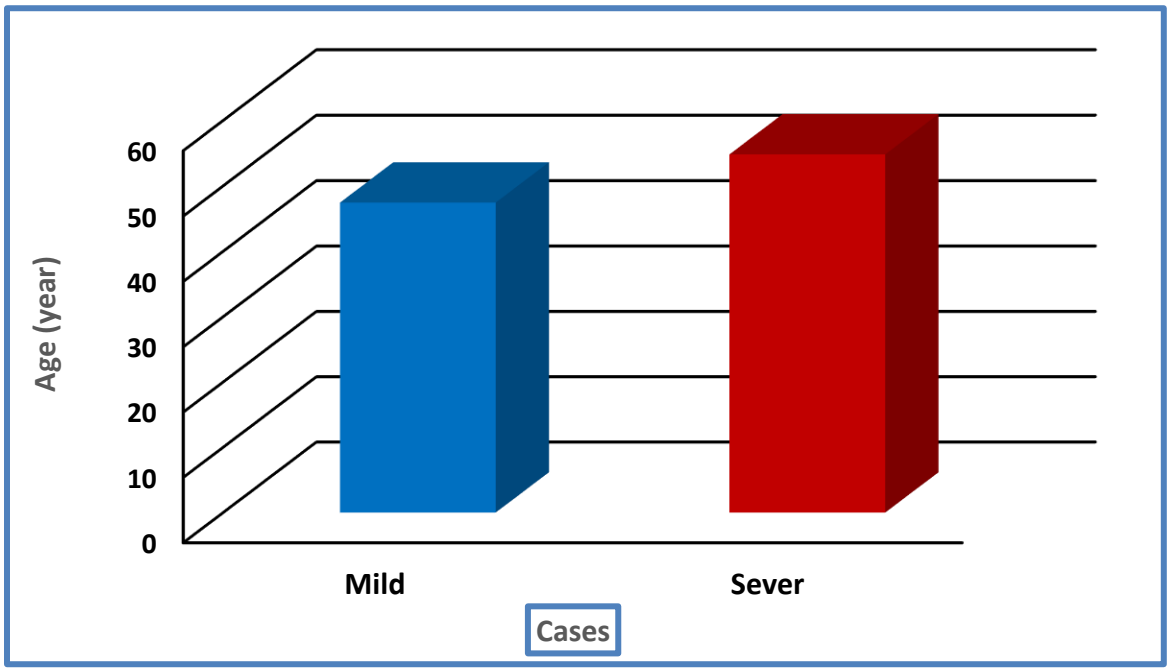

Figure 1: Comparison of the age factor in COVID-19 patients' various diseases (mild and severe).

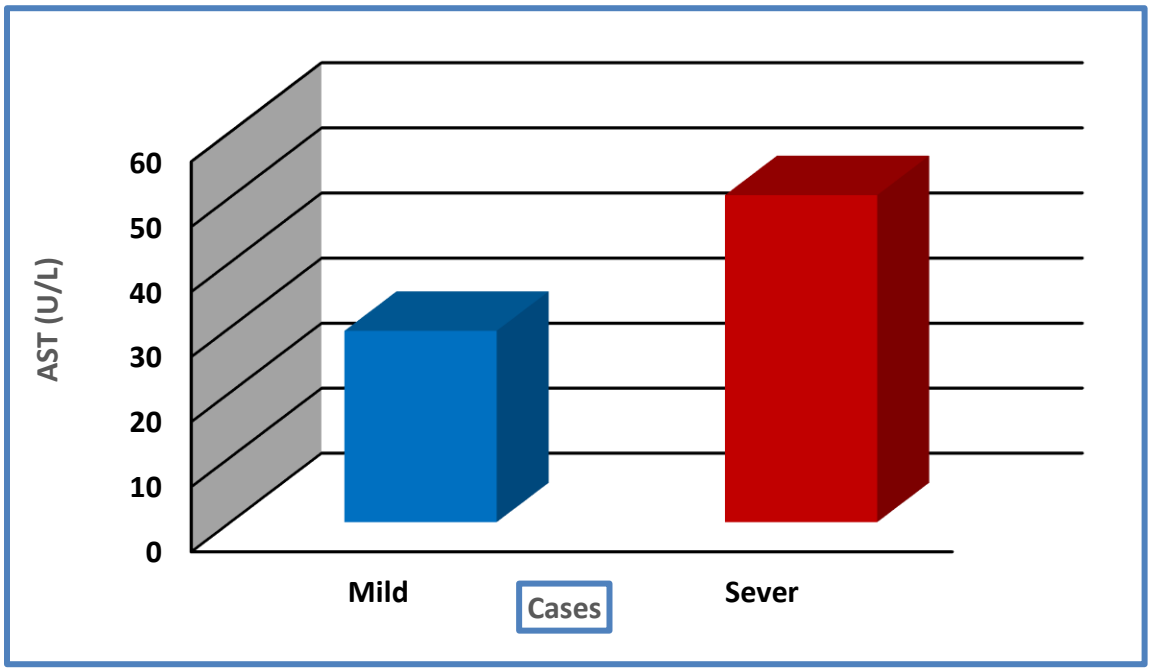

Figure 2: Comparison of AST levels in COVID-19 patients with various diseases (mild and severe).

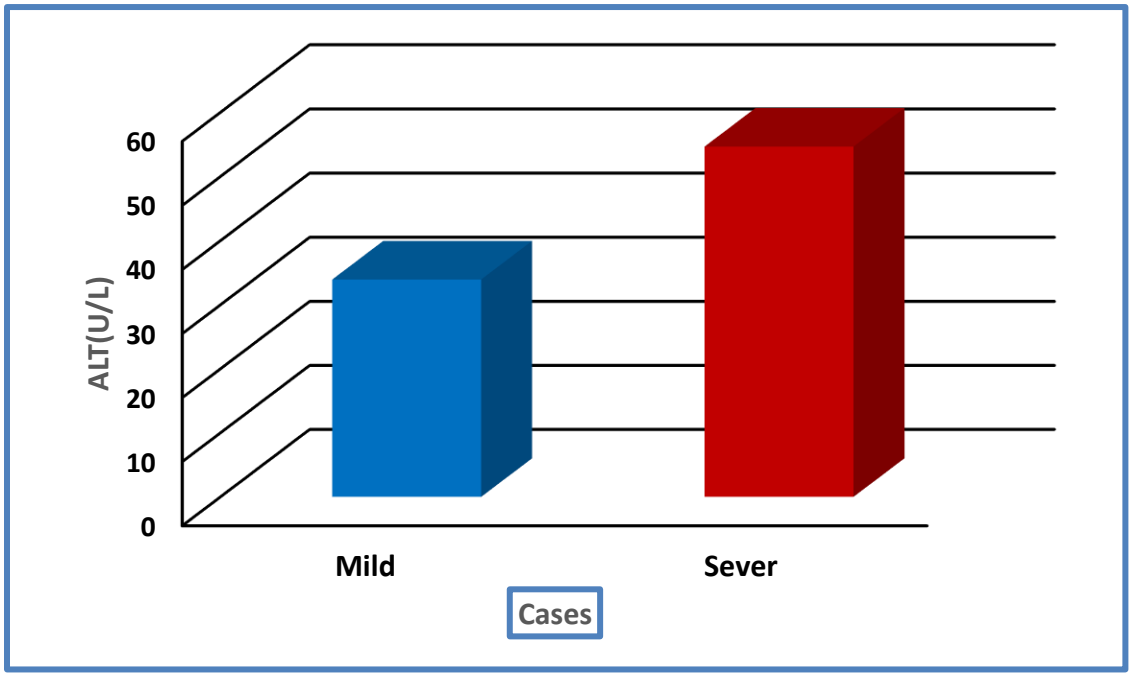

Figure 3: Comparison of ALT levels in COVID-19 patients with various diseases (mild and severe).

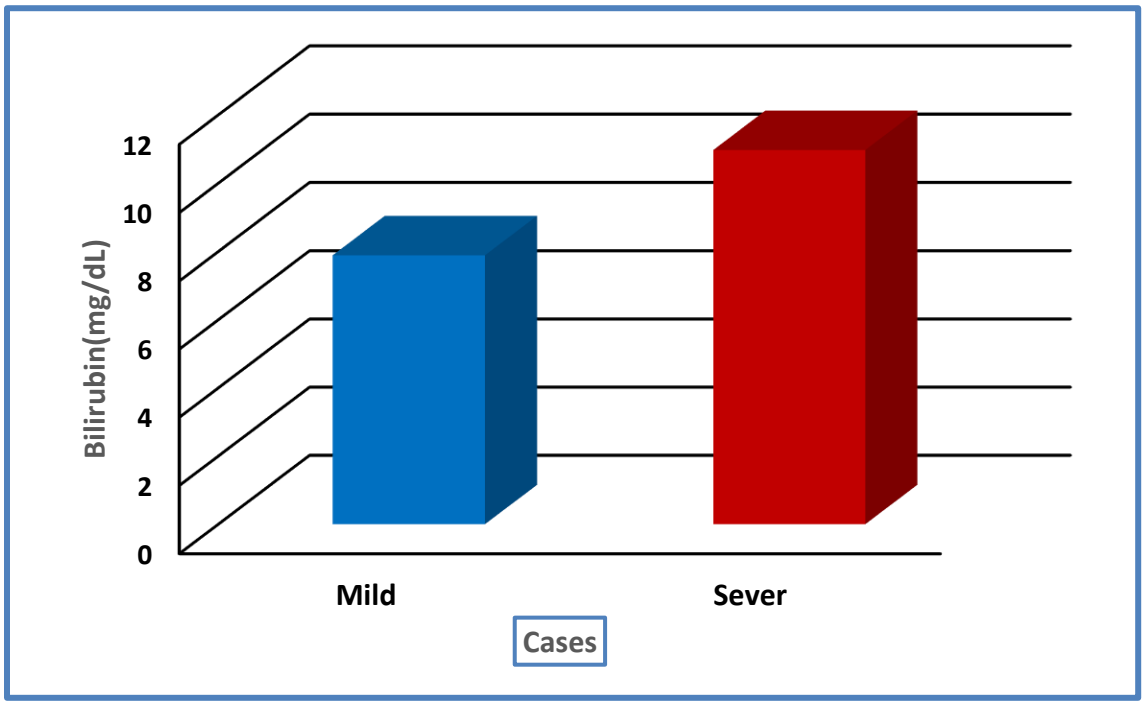

Figure 4: Comparison of COVID-19 patients' bilirubin levels under various conditions (mild and severe).

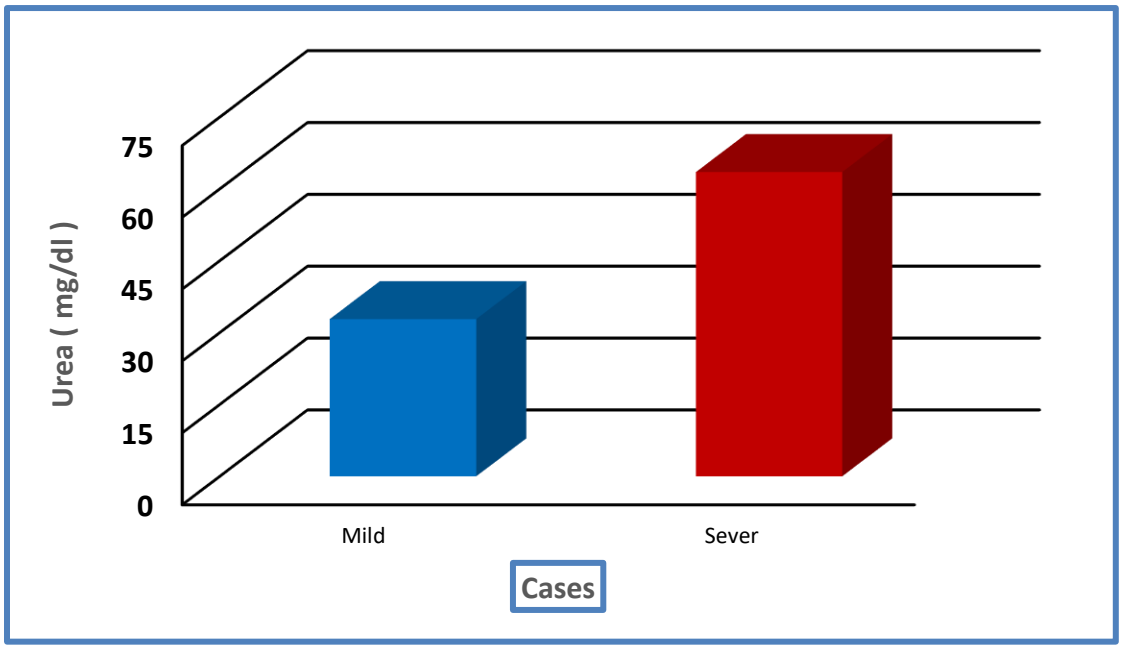

Figure 5: Comparison of COVID-19 patients' urea levels under various conditions (mild and severe).

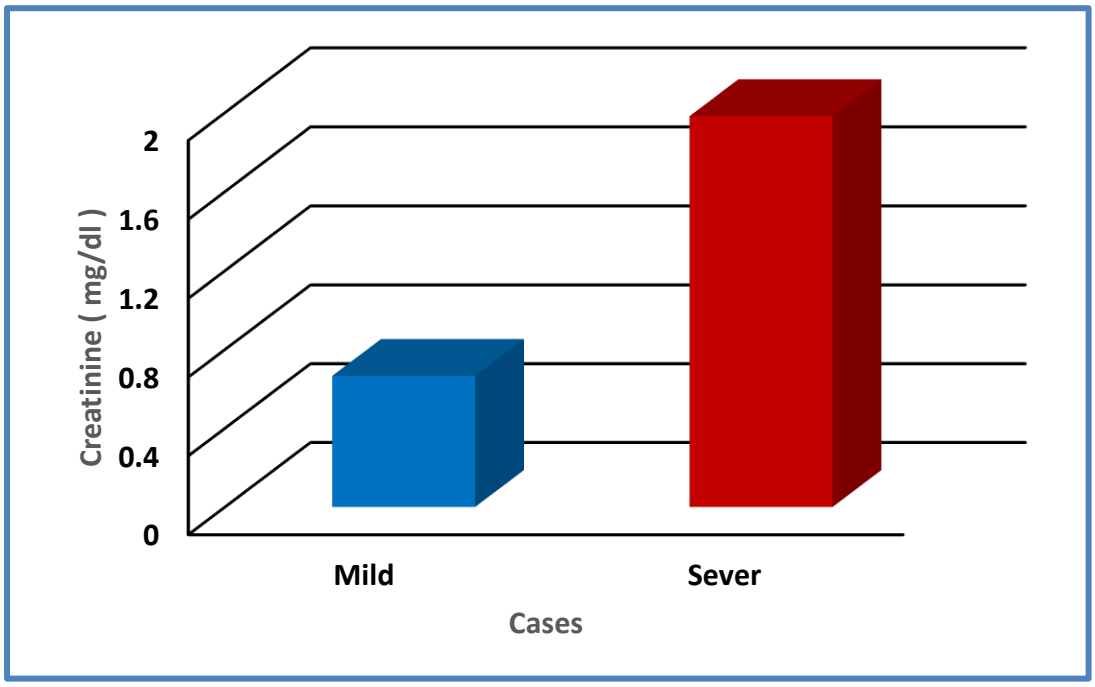

Figure 6: Comparison of COVID-19 patients' creatinine levels under various conditions (mild and severe).

Numerous studies have found severe COVID-19 individuals have a high mortality rate [19]. It is critical to identify early warning signs of illness aggravation. Previous research has demonstrated that the generation of interleukin and the duration of lung infection were strongly connected with disease severity and could be utilized as clinical indicators for determining COVID-19 severity [17, 20, 21]. The location and expression of ACE2 are significantly linked to the target organ of the SARS-CoV-2 infection[22]. Seow et al. observed the co-expression of ACE2 in the population of liver progenitors. They found a liver cell type that may be more susceptible to viral invasion[4, 17]. Additionally, the kidney may explain how SARS-CoV-2 can harm the kidney and liver and create high levels of renal biomarkers, including creatinine and blood urea nitrogen (BUN). Researchers found that patients with severe symptoms had more laboratory evidence that a cytokine storm was to blame for their severe pneumonia, organ damage, and abnormal laboratory results. According to certain research, the abnormal blood rheology and toxic effects of COVID-19 patients can make liver enzymes, bilirubin, urea, and creatinine serve as independent predictors for COVID-19 patients [17].

4. Conclusions

We conclude that patients with COVID-19, especially those with severe disease, suffer from liver and kidney dysfunction; therefore, the severity of this disease may be predicted by assaying the levels of ALT, AST, bilirubin, urea, and creatinine. Moreover, the progression of this disease from mild to severe in COVID-19 patients may be expected by combining the levels of ALT, AST, bilirubin, urea, and creatinine.

References

[1] P. Letelier, N. Encina, P. Morales, A. Riffo, H. Silva, I. Riquelme, N. Guzmán, Role of biochemical markers in the monitoring of COVID-19 patients, J. Med. Biochem. 40 (2021) 115, https://doi.org/10.5937/jomb0-29341

[2] S. Vahdat, Clinical profile, outcome and management of kidney disease in COVID-19 patients–a narrative review, Eur. Rev. Med. Pharmacol. Sci. 26 (2022) 2188-2195. https://doi.org/10.26355/eurrev_202203_28367

[3] C. Huang, Y. Wang, X. Li, L. Ren, J. Zhao, Y. Hu, L. Zhang, G. Fan, J. Xu, X. Gu, Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China, The lancet 395 (2020) 497-506. https://doi.org/10.1016/S0140-6736(20)30183-5.

[4] J. Qu, H.-H. Zhu, X.-J. Huang, G.-F. He, J.-Y. Liu, J.-J. Huang, Y. Chen, Q. Qu, Y.-L. Wu, X.-Y. Chen, Abnormal indexes of liver and kidney injury markers predict severity in COVID-19 patients, Infection and Drug Resistance 14 (2021) 3029, https://doi.org/10.2147%2FIDR.S321915.

[5] M. Wei, N. Yang, F. Wang, G. Zhao, H. Gao, Y. Li, Epidemiology of coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), Disaster Medicine and Public Health Preparedness, 14 (2020) 796-804, https://doi.org/10.1017/dmp.2020.155.

[6] J.S. Rico-Mesa, A. White, A.S. Anderson, Outcomes in patients with COVID-19 infection taking ACEI/ARB, Current Cardiology Reports, 22 (2020) 1-4, https://doi.org/10.1007/s11886-020-01291-4.

[7] T. Velavan, C. Meyer, La epidemia de COVID-19, Trop Med Int Health 25 (2020) 278-280, https://doi.org/10.1111/tmi.13383.

[8] X. Xu, P. Chen, J. Wang, J. Feng, H. Zhou, X. Li, W. Zhong, P. Hao, Evolution of the novel coronavirus from the ongoing Wuhan outbreak and modeling of its spike protein for risk of human transmission, Sci. China Life Sci., 63 (2020) 457-460, https://doi.org/10.1007/s11427-020-1637-5.

[9] P. Zhou, X.-L. Yang, X.-G. Wang, B. Hu, L. Zhang, W. Zhang, H.-R. Si, Y. Zhu, B. Li, C.-L. Huang, A pneumonia outbreak associated with a new coronavirus of probable bat origin, Nature, 579 (2020) 270-273. https://doi.org/10.1038/s41586-020-2012-7

[10] Y. Wang, S. Liu, H. Liu, W. Li, F. Lin, L. Jiang, X. Li, P. Xu, L. Zhang, L. Zhao, SARS-CoV-2 infection of the liver directly contributes to hepatic impairment in patients with COVID-19, J Hepatology 73 (2020) 807-816. https://doi.org/10.1016/j.jhep.2020.05.002

[11] F.S. Cardoso, R. Pereira, N. Germano, Liver injury in critically ill patients with COVID-19: a case series, Critical Care, 24 (2020) 1-2, https://doi.org/10.1186/s13054-020-02924-4.

[12] P. Chen, B. Zhou, Clinical characteristics of COVID-19 in patients with liver injury, Clinical Gastroenterology and Hepatology, 18 (2020) 2846-284 https://doi.org/10.1016/j.cgh.2020.04.043

[13] Z. Fan, L. Chen, J. Li, X. Cheng, J. Yang, C. Tian, Y. Zhang, S. Huang, Z. Liu, J. Cheng, Clinical features of COVID-19-related liver functional abnormality, Clinical Gastroenterology and Hepatology, 18 (2020) 1561-1566. https://doi.org/10.1016/j.cgh.2020.04.002

[14] G.-j. Zhao, C. Xu, J.-c. Ying, W.-b. Lü, G.-l. Hong, M.-f. Li, B. Wu, Y.-m. Yao, Z.-q. Lu, Association between furosemide administration and outcomes in critically ill patients with acute kidney injury, Critical Care, 24 (2020) 1-9. https://doi.org/10.1186/s13054-020-2798-6

[15] A. Post, E.S. den Deurwaarder, S.J. Bakker, R.J. de Haas, M. van Meurs, R.T. Gansevoort, S.P. Berger, Kidney infarction in patients with COVID-19, American J Kidney Dis., 76 (2020) 431-435. https://doi.org/10.1053/j.ajkd.2020.05.004

[16] K.-K. Lau, W.-C. Yu, C.-M. Chu, S.-T. Lau, B. Sheng, K.-Y. Yuen, Possible central nervous system infection by SARS coronavirus, Emerging Infectious Dis., 10 (2004) 342, https://doi.org/10.1159/000453066

[17] J. Qi, Y. Zhou, J. Hua, L. Zhang, J. Bian, B. Liu, Z. Zhao, S. Jin, The scRNA-seq expression profiling of the receptor ACE2 and the cellular protease TMPRSS2 reveals human organs

susceptible to SARS-CoV-2 infection, Int. J Env. Res Public Health, 18 (2021) 284, https://doi.org/10.3390/ijerph18010284

[18] R. Haque, F.B. Hafiz, M. Habib, K.R. Radeen, L.N. Islam, Role of complete blood count, antioxidants, and total antioxidant capacity in the pathophysiology of acute coronary syndrome, African J Bio Sci., 4 (2022) 37-47 https://doi.org/10.33472/AFJBS.4.1.2022.37-47

[19] F. Ottosson, E. Baco, P.M. Lauritzen, E. Rud, The prevalence and locations of bone metastases using whole-body MRI in treatment-naïve intermediate-and high-risk prostate cancer, European Radiology 31 (2021) 2747-2753. https://doi.org/10.1007/s00330-020-07363-x

[20] J. Hadjadj, N. Yatim, L. Barnabei, A. Corneau, J. Boussier, N. Smith, H. Péré, B. Charbit, V. Bondet, C. Chenevier-Gobeaux, Impaired type I interferon activity and inflammatory responses in severe COVID-19 patients, Science 369 (2020) 718-724. https://doi.org/10.1126/science.abc6027

[21] S. Felsensteina, J.A. Herbertb, P.S. McNamarab, C.M. Hedrichb, COVID-19: Immunology and treatment options, Clinical Immunology, 215 (2020) 108448, https://doi.org/10.1016/j.clim.2020.108448

[22] A. Kumar, M.A. Faiq, V. Pareek, K. Raza, R.K. Narayan, P. Prasoon, P. Kumar, M. Kulandhasamy, C. Kumari, K. Kant, Relevance of SARS-CoV-2 related factors ACE2 and TMPRSS2 expressions in gastrointestinal tissue with pathogenesis of digestive symptoms, diabetes-associated mortality, and disease recurrence in COVID-19 patients, Medical hypotheses 144 (2020) 110271. https://doi.org/10.1016%2Fj.mehy.2020.110271

[23] L.Y. Chen, H.K. Chu, T. Bai, S.J. Tu, Y. Wei, Z.L. Li, L.L. Hu, R. Zhu, L. Zhang, C.Q. Han, Liver damage at admission is an independent prognostic factor for COVID-19, J Digestive Dis., 21 (2020) 512-518. https://doi.org/10.1111/1751-2980.12925

تقدير المؤشرات الحيوية للكبد والكلى للأشخاص أثناء إصابتهم بفيروس SARA-COV-2 (COVID-19) في محافظة نينوى

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المستخلص

لا يضر فيروس SARS-CoV-2 بالأعضاء فحسب، بل بالكلى والكبد أيضًا. يعاني معظم الأفراد المصابين بأمراض خطيرة والذين يعانون من عدوى مرض فيروس كورونا 2019 (كوفيد-19) من فشل الكبد والكلى. هدفت دراستنا إلى دراسة مستويات وظائف الكبد والكلى لدى مرضى كوفيد-19 في الحالات المرضية المختلفة، بما في ذلك المتوسطة والشديدة. شملت الدراسة 56 مريضاً بفيروس كورونا في محافظة نينوى من الجنسين (32 ذكراً، 24 أنثى). وتراوحت أعمارهم بين (30 و65) عاماً. وتم تقسيم هؤلاء المرضى إلى مجموعتين حسب شدة المرض. ضمت المجموعة الأولى 25 مريضا يعانون من مرض خفيف، والثانية ضمت 31 مريضا يعانون من مرض شديد. تم قياس وظائف الكبد والكلى والمؤشرات الحيوية الأخرى. أظهرت النتائج أن جميع المرضى ذوي الحالات الشديدة لديهم اختلاف كبير في ناقلة أمين الألانين (ALT)، ناقلة أمين الأسبارتات (AST)، يوريا البيليروبين، والكرياتينين مقارنة بالمرضى الخفيفين (جميعهم $P < 0.0001$ باستثناء الكرياتينين، $P = 0.014$). علاوة على ذلك، فإن المرضى الذين يعانون من مرض شديد كانوا أكبر سنا من أولئك الذين يعانون من مرض خفيف. وكان الذكور أيضًا أكثر تأثراً بكوفيد-19. نستنتج أن المرضى المصابين بـCOVID-19، وخاصة أولئك الذين يعانون من مرض شديد، يعانون من خلل في الكبد والكلى. لذلك، يمكن التنبؤ بخطورة هذا المرض من خلال فحص مستويات ALT، AST، البيليروبين، اليوريا، والكرياتينين. علاوة على ذلك، يمكن التنبؤ بتطور المرض من الخفيف إلى الشديد لدى مرضى كوفيد-19 من خلال الجمع بين مستويات ALT وAST والبيليروبين واليوريا والكرياتينين.