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Inflammatory Response and The Effect of Coronavirus on Some Body Organs in Hospitalized Patients

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ABSTRACT

Background. In the last three years, coronavirus was effectively introduced as leading cause of death worldwide. This virus actively damages the main parts of the respiratory system in the early infection. Immune response may limit the infection and rescue the patient somewhat. However, the chronic infection may have a serious systematic effect and reduce the function of other organs including kidney and liver. Investigation of inflammatory response and the main indicators of kidney and liver damage is the main purpose of this study.

Methods. Present study included a hundred and six patients with confirmed COVID-19 (52 females and 54 males). All patients were aged 25 or over. Twenty non-COVID-19 infected group were also used in this study. The sample collection take about eight weeks. Hematological parameters, inflammatory response and coagulation factor were determined with CBC, CD4, C-reactive protein (CRP) and D-dimer in Covid-19 infected patients and in non-COVID-19 infected groups. Serum concentrations of creatinine (Cre), Urea, Alanine transaminase (ALT), Aspartate aminotransferase (AST), Alkaline phosphatase (ALP), T.Bilirubin (TSB), D.Bilirubin (DB), and Albumin were also studied in Covid19 infected patients and in non-COVID-19 infected groups.

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Results. In Covid-19 infected patients, inflammatory markers, CRP and WBC are increased and immunological parameters, CD4 and LYM are decreased. Serum concentrations of Cre, urea, ALT, AST, ALP, TSB, and DB are all increased in Covid-19 infected patients, whereas the serum level of ALB was decreased. In Covid19 infected patients increased plasma level of D-dimer was observed.
Conclusion. It is concluded that in Covid-19 infected patients, renal and liver functions are significantly reduced with increased inflammatory response.

1.Introduction

The RNA viruses known as coronaviruses are covered with a protein sheath. It belongs to the Nidovirales order's family of Coronaviridae viruses. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) appears to share almost 80% of its genomic sequence with Severe acute respiratory syndrome coronavirus (SARS-CoV) and roughly 50% with Middle East respiratory syndrome coronavirus (MERS-CoV)(Tian & Ye, 2020).The betacoronavirus 2 (SARS-CoV-2) induces the coronavirus disease 2019 (COVID- 19)(Zhu et al., 2020). Coronavirus were found as zoonotic, which means they have ability to spread from animals to humans and cause a variety of viral infections (Al-Jumaili et al., 2020). Its infection manifest as a wide range of symptoms, from the ordinary cold to deadly diseases such as enteric and central nervous system diseases, as well as respiratory syndrome (Zhu et al., 2020). The protein-spike of coronavirus attached to angiotensin-converting enzyme 2 (ACE2) receptors, which are predominantly found on pulmonary epithelial cells but also on endothelial cells, lymphocytes, and other cell types, is a common route of cell entrance used by the virus (Napoli et al., 2021). Once the virus enter the cell, the cell's apparatus used by the virus for replication, due to this process the host cell destroy, and the virus spreads. (Clark et al., 2021). As ACE2 is broadly dispersed on lung cell surfaces, lungs become essential purpose of coronavirus. However, liver epithelial and bile duct cells also express ACE2, become basic for SARS-CoV-2 to tie to ACE2-positive

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cholangiocytes and disable the work of liver (Zhang et al., 2020). There is growing proof that the liver, heart, kidneys, central nervous system, and other organs are all affected by SARS-CoV-2 (Przekop et al., 2021). Patients with SARS CoV-2 have raised level of AST and ALT, which may be an indicator of liver destruction induced by the virus infection (Li et al., 2020).In the end, multiple organ failure was the decisive factor in the demise of COVID-19. (Hong et al., 2020) observed a large number of patients with acute renal impairment among patients with confirmed SARS-CoV-2 infection. Some of them even had renal failure and needed dialysis. The quick clinical deterioration that some patients experience is one of the key problems that are being seen in these patients. The body's ability to remove metabolites and poisons can be impaired by impaired renal function, which can negatively affect the body's electrolyte and acid-base balance. A serious renal injury can also lead to uremia, posing a life-threatening situation. In order to minimize complications and improve prognosis, it is imperative to recognize kidney damage symptoms early and to provide prompt, efficient treatment. Investigation of inflammatory response and the main indicators of kidney and liver damage is the main purpose of this study.

2.Materials and Methods:

2.1 Patients:

Patients with Covid19 were confirmed using the data from LALAV and West Erbil Emergency. A hundred and six patients were included in this study (52 females and 54 males). All patients were aged 25 or over. Twenty non-COVID-19 infected group were also used in this study.

2.2 Sample Collection:

Blood samples were collected from both Covid-19 and non-coronavirus patients for hematological, immunological, biochemical, and coagulation studies. Collected blood samples were centrifuged at 4000 rpm for 10 minutes to separate serum portion which were then aliquoted and stored at -20. Aliquoted samples were then used for immunological and biochemical determinations. For hematological parameters, blood samples were transferred into EDTA tubes for

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hematological parameters and assessed with fully automated CBC machine from Medonic M-series M32. Plasma concentrations of D-dimer were measured by using Indiko Plus.

2.3 ELISA:

The concentration of CD4 was determined in serum samples using Microelisa stripplate quantitative sandwich ELISA (ELISA kit, SL0465Hu, SUNLONG, China), as stated by manufactures instructions. Concisely, the standard is diluted in small tubes in the first step, standards (3.6, 2.4, 1.2, 0.6, 0.3 ng/ml), then pipette 50ul from each tube to each microplate well. In the wells of sample, 10µl sample are added and 40µl Sample dilution buffer (dilution factor is 5). To avoid touching the well wall, samples are loaded onto the bottom. Wells will gently shake. In the Microelisa stripplate, a well leaving empty for blank control. In the wells of sample, 40µl buffer of Sample dilution and 10µl sample are added (dilution factor is 5). To prevent touching the well wall, Samples loaded onto the bottom. Wells will gently mix. After wells sealed with closure plate membrane, incubate at 37℃ for 30 min. The concentrated washing buffer dilute with distilled water (30 times for 96T). The closure of plate membrane is carefully peel off, refill and aspirate the wells with the wash solution. After 30 seconds of resting wash solution, discard it. Repeat washing 5 times. Then 50µl of conjugated antibody-Horseradish Peroxidase (HRP) particular to CD4 is add to the wells except the blank control well. Incubate 30 min at 37℃. Refill and aspirate the wells with the wash solution. After 30 seconds of resting wash solution, discard it. Repeat washing 5 times. 50µl of Chromogen Solution A and 50µl Chromogen Solution B added to each well, mix is done by shaking gently then incubate for 15 min at 37℃. Must be stay away from light during coloring. The reaction was stopped by adding 50µl stop solution to each well, well colors changed from blue to yellow. The absorbance of optical density (OD) read by Microtiter Plate Reader, spectrophotometrically at 450nm. The OD value is set as zero of the blank control well. The concentration of CD4 is proportional to the OD value. The OD of the samples compared to the standard curve, the

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concentration of CD4 in the samples could be detected. After adding stop solution the assay carried out within 15 minutes.

Biochemical including renal function test (RFT) and liver function test (LFT) parameters and inflammatory biomarkers CRP and plasma concentration of D-dimer were measured using fully automatic clinical chemistry analyzer Indiko Plus.

2.4 Statistical Analysis:

The current study's data were represented as mean, standard error of mean (Mean ± SE), differences in mean values between 2 groups were analyzed by two samples independent students t-test, and the GraphPad Prism (version 9) was used as statistical software to analyze the data. The p value ($p<0.05$) level of significance was declared statistically significant. The area under the curve (AUC) for diagnostic accuracy in corona patients was determined using ROC curve (Receiver Operating Characteristic) analysis.

3.Results and Discussion:

The present study provide a clear data that the coronavirus, in addition to its primary effect on the respiratory system through increasing inflammatory indicators and coagulation factor, have a great effect on liver and kidney. The current study shows a significant increase in WBC count in the blood of COVID-19 patients (14.01 ± 0.4347 10^9/l) compared to healthy group (6.232 ± 0.2816 10^9/l), as demonstrated in (Table 1 and Figure 1). Similarly, the effect of coronavirus on LYM was clearly seen in infected patients (0.7932 ± 0.05356) in comparison to healthy group (2.162 ± 0.1626 10^9/l). Recent studies revealed that increased WBC and decreased LYM levels may reflect inflammatory response imbalances and consider a possible indicator of the severity rate of infectious diseases, such as bacteremia and sepsis. The previous study have shown that because of lymphocytes are effector cells of virus-fighting, most viruses cause lymphocytosis when they infect humans while the coronavirus family caused lymphocytic depletion in infected patients, and the illustrated mechanism presented that direct attack of coronavirus or by immune-mediated

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apoptosis of lymphocytes cause lymphocytopenia (Zheng et al., 2020)(Rabaan et al., 2020)(Chu et al., 2016).

The date here showed a significant decrease in PLT count in COVID-19 patients (110.3 ± 6.556) compared to non-coronavirus infected people (248.9 ± 12.38). In the COVID-19 patient's thrombocytopenia is a common occurrence and a low PLT count at the onset of infection can be one of the indicators of a poor prognosis and results in higher chance of mortality. This lowering in PLT may be associated with several factors including platelets hyperactivation consumption (X. Yang et al., 2020) (Liu et al., 2020) (Zhao et al., 2020) (Gowda et al., 2021) (Canzano et al., 2021).

Table (1): WBCs, LYM, and PLT values in COVID-19 infected patients and Non-COVID-19 infected group:

Parameters	Healthy group	Patient group	P value
WBC (10^9/l)	6.232 ± 0.2816	14.01 ± 0.4347	<0.0001
LYM (10^9/l)	2.162 ± 0.1626	0.7932 ± 0.05356	<0.0001
PLT (10^9/l)	248.9 ± 12.38	110.3 ± 6.556	<0.0001

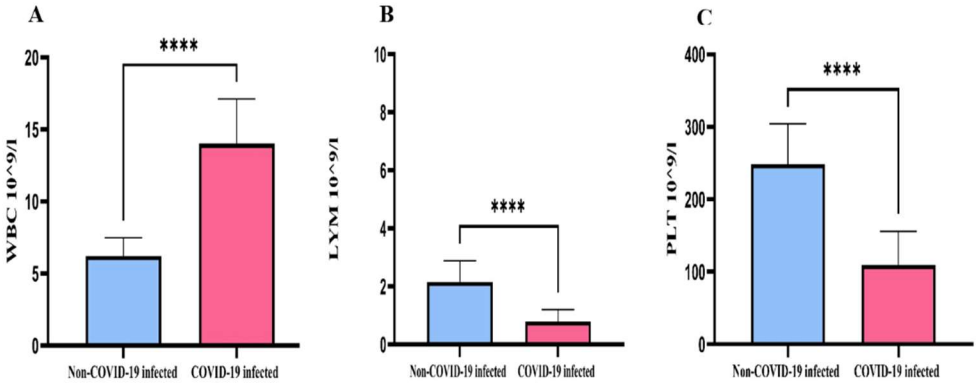

Figure (1): Blood parameters in COVID-19 infected and non- COVID-19 infected patients. (A) WBCs level in both groups. *P = 0.0001, n=106. (B) LMY level in both groups. *P= 0.0001, n=106. (C) PLT in both groups. *P= 0.0001, n=106.

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The concentration of inflammatory, immunological, renal, and liver parameters of the healthy and COVID-19 patient groups are displayed in (Table 2 and Figure 2). As shown, the serum levels of CRP are significantly higher in COVID-19 infected group (10.35 ± 1.987 mg/l) compared to non-COVID-19 infected group (3.957 ± 0.5918 mg/l). CRP levels are related to the level of inflammation, and its concentration is unaffected by age, gender, or physical condition. CRP levels can stimulate phagocytosis and activate the complement, ridding the body from pathogenic microorganisms. CRP levels can be used to detect pneumonia in its early stages, and CRP levels were also elevated in patients with severe pneumonia. It is a useful indicator for diagnosing and assessing severe pulmonary infection (Warusevitane et al., 2016)(Wang, 2020)(Chalmers et al., 2019). This study shown that as the disease advanced, CRP levels and the diameter of the lung lesion increased.

Results in this study show that the plasma levels of D-dimer were greatly higher in COVID-19 infected group (1.332 ± 0.08978 µg/ml) compared to non-infected group (0.3050 ± 0.02881 µg/ml). Virus infection can progress to sepsis and cause coagulation dysfunction, which is common in advanced disease. Alternatively, an increase in D-dimers may be an indirect manifestation of the inflammatory response, because inflammatory cytokines can induce an imbalance in coagulation and alveolar fibrinolysis, which may trigger fibrinolytic system, and subsequently increases the level of D-dimers (Tan et al., 2020)(Yu et al., 2020).

The current study measured the serum level of CD4+T cells and the results showed that the concentration of this immunological marker was significantly reduced in COVID-19 patients (0.4706 ± 0.02325 ng/ml) in comparison to non-infected group (0.8833 ± 0.1351 ng/ml). Viral infections are controlled through complicated interactions between immune system cell types. Lymphocytes and subsets, such as CD4+T cells, play a crucial part in immune system function maintenance. Previous research suggested that the severity of the coronavirus infection is correlated with reduced CD4+T cells. Furthermore, COVID-19 patients in severe cases had a reduction in CD4+T cells. According to recent investigations, lymphocyte shortage in peripheral blood, which is mediated by

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antigenic stimulation, may be caused by lymphocyte migration from blood to the lung (Jiang et al., 2020) (Kazancioglu et al., 2021)(Wang et al., 2020). The role of lymphocytes might vary greatly depending on the stage of infection, were our patients in severe stage. This could explain why the lymphocyte function results in our study differ from prior published results.

In addition to the enormous effect of coronavirus on respiratory system, the infection seems to have a great effect on the other organs as well. This study shows that the serum concentration of Crea Enz and Urea were significantly increased in COVID-19 patients (1.811 ± 0.1396 and 69.34 ± 3.019 mg/dl respectively) compared to healthy group (0.8430 ± 0.05228 and 32.72 ± 1.345 mg/dl). Although the lung was widely assumed to be the primary target organ of coronavirus infection, as evidenced by early autopsy reports, we discovered that COVID-19 patients also had kidney dysfunction. ACE2, the cell entry receptor of coronavirus, has been found to be expressed nearly 100 times higher in the kidneys than in the lungs. The pathogenesis of kidney disease in COVID-19 patients is likely multifactorial, including direct cytotoxic effects on kidney tissue, endothelial damage, immune complex deposition, and virus-induced cytokines or mediators (Li et al., 2020)(Su et al., 2020)(Zhou et al., 2020)(Huang et al., 2020)(Sardu et al., 2020).

The mechanism underlying the rise in blood urea levels following coronavirus infection is still not completely understood. Because of ACE2 is highly expressed in the kidney epithelial cell as a primary cellular receptor of SARS CoV-2, direct interact of coronavirus to its receptor which is expressed in the renal reducing ACE2 expression, which would result in abnormal activation of the renin-angiotensin-aldosterone system (RAAS). The activated RAAS can greatly enhance water absorption by kidney tubules while increasing urea resorption, resulting in high blood Urea levels (Soleimani, 2020)(Q. Wang et al., 2020)(Liu et al., 2021). The increase in blood urea is not only a sign of kidney dysfunction, it can also be an indicator of other conditions, such as inflammation, nitrogen balance, catabolism, and renal hypoperfusion due to hypovolemia, sepsis, or decreased cardiac output, numerous of which have been linked to unfavorable outcomes

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in patients with COVID-19 (H. Li et al., 2020) (Oudit & Pfeffer, 2020) (Ronco et al., 2020).

In the present study, the levels of serum ALT, AST, ALP, TSB, DB, and ALB were all increased significantly in COVID-19 patients (56.07 ± 2.568 U/L, 56.85 ± 2.160 U/L, 100.1 ± 4.376 U/L, 1.471 ± 0.07488 mg/dL, 0.4198 ± 0.02455 mg/dL respectively) compared to non-coronavirus infected patients (20.05 ± 1.334 U/L, 20.83 ± 0.9321 U/L, 64.82 ± 3.186 U/L, 0.6242 ± 0.06667 mg/dL, 0.2200 ± 0.02128 mg/dL respectively). On the other hand, the serum levels of ALB was significantly decreased in COVID-19 patients (2.698 ± 0.04247 g/dL) compared to non-coronavirus group (4.121 ± 0.09050 g/dL). The pathogenic mechanisms for this abnormal increase in liver function tests in COVID-19 group are not fully understood. Many factors may be involved in these changes including, microthrombotic endothelialitis, immunological dysregulation, drug-induced liver injury, and hepatic ischemia related to hypoxia. The viral infection may directly induce liver damage and few investigations have shown that ACE2 is the primary receptor for coronavirus entrance into cells, and direct binding of it to ACE2 receptors in cholangiocytes may result in liver injury (Bertolini et al., 2020) (Y. Yang et al., 2020)(Yan et al., 2020)(Zhang et al., 2020). Numerous medication, including acetaminophen, antivirals, antibiotics, corticosteroids, and immune-modulators, that have the potential hepatotoxic effect are used to treat the symptoms or manage COVID-19 patients. (Przekop et al., 2021) found that using lopinavir/ritonavir to treat a severe COVID-19 infection resulted in an almost four-fold increase in liver damage. The liver produces albumin, which is essential for sustaining bodily nourishment and osmotic pressure, a drop in albumin levels is probably attributable to damage in liver and is most likely as a result of reverse medication responses as well as systemic inflammation in severely unwell COVID-19 patients. A number of studies have found that albumin levels in COVID-19 patients may predict disease severity. Low albumin levels also indicate that the patient's nutrition is inadequate and that the body's immunity is impaired. However, dietary deficiencies frequently reduce the host's immune response to RNA virus infection, which is often overlooked during clinical

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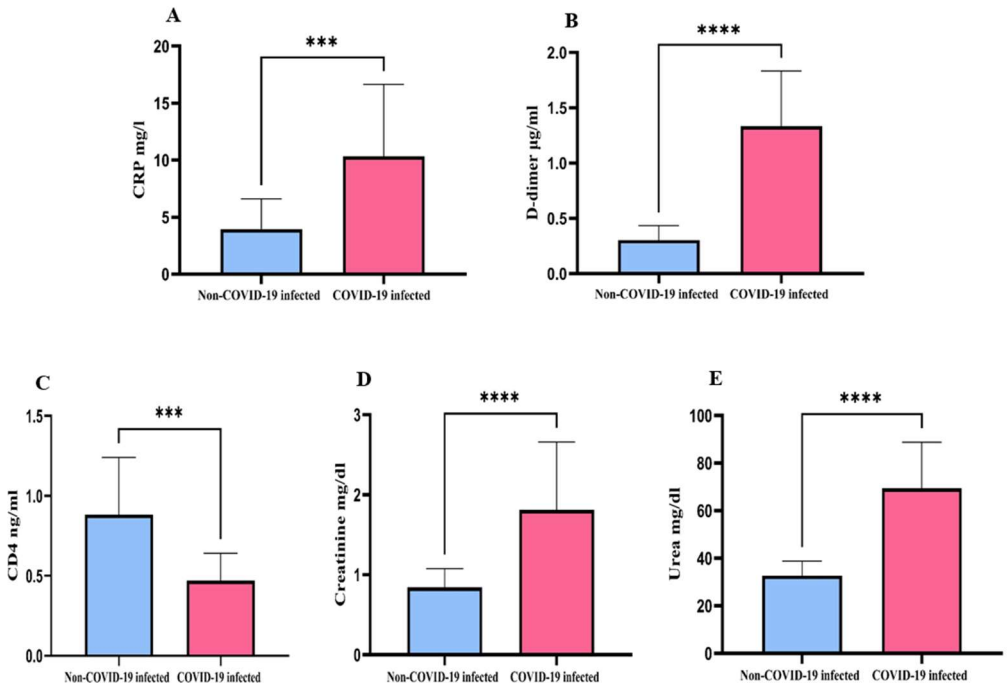
diagnosis and treatment. (Fan et al., 2020)(Bernardi et al., 2020) (W. Liu & Tao, 2020) (Liu et al., 2020). As a results, (J. Li et al., 2020) suggest verifying the nutritional state in patients infected with COVID-19 before administering general therapy.

Table (2): comparison of inflammatory, immunological, renal, and liver variables between non-Coronavirus infected and COVID-19 Coronavirus infected Patient:



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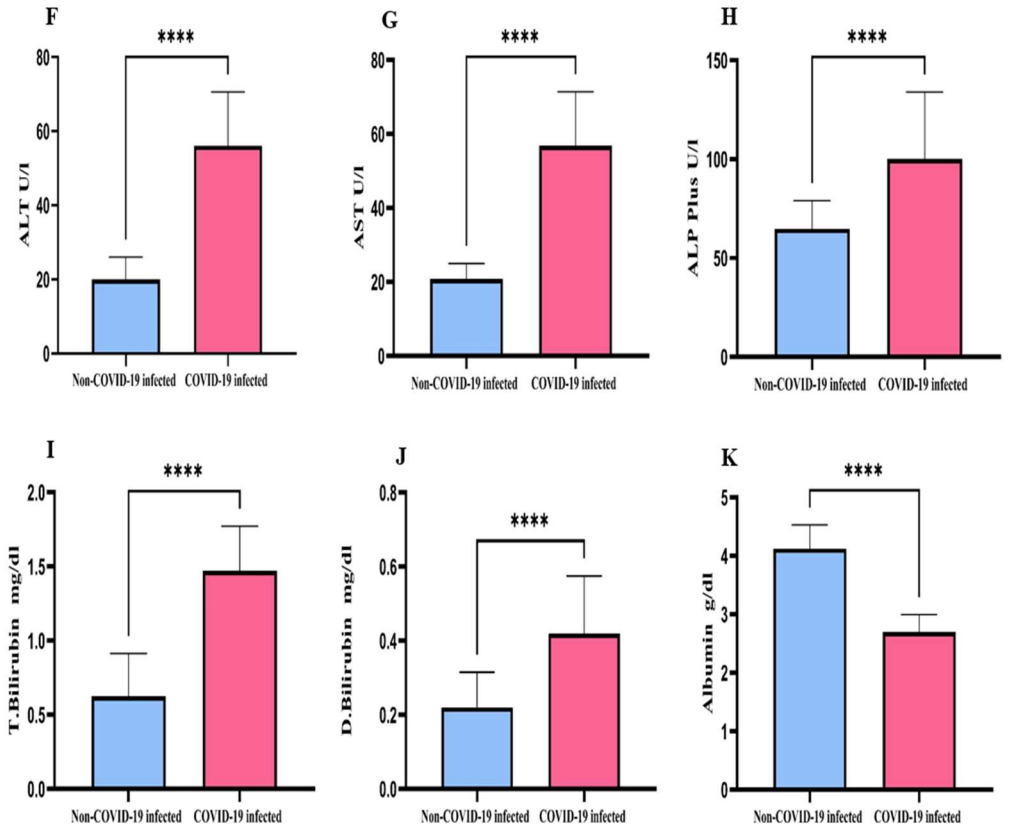

Figure (2): Inflammatory, Immunological, and biochemical parameters in COVID-19 infected and non- COVID-19 infected patients. (A) CRP level in both groups. *P = 0.0005, n=106. (B) D-dimer level in both groups. *P = <0.0001, n=106. (C) CD4 level in both groups. *P = 0.0002, n=106. (D) Creatinine level in both groups. *P = <0.0001, n=106. (E) Urea level in both groups. *P = <0.0001, n=106. (F) ALT level in both groups. *P = <0.0001, n=106. (G) AST level in both groups. *P = <0.0001, n=106. (H) ALP Plus level in both groups. *P = <0.0001, n=106. (I) T.Bilirubin level in both groups. *P = <0.0001, n=106. (J) D.Bilirubin level in both groups. *P = <0.0001, n=106. (K) Albumin level in both groups. *P = <0.0001, n=106.

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For determining the diagnostic accuracy of WBC, LYM, PLT, HGB, CRP, D-dimer, CD4, Urea, ALT, AST, ALP Plus, T.Bilirubin, D.Bilirubin, and Albumin, the ROC curve suggested. The AUC value in blood of WBC is 0.8241. The S.E value is 0.04828 and the 95% CI value is 0.7294 to 0.9187, ($p<0.0001$). While the AUC value of LYM is 0.9555. The S.E value is 0.02161 and the 95% CI value is 0.9132 to 0.9979, ($p<0.0001$). The AUC value in blood of PLT is 0.7089. The S.E value is 0.05593 and the 95% CI value is 0.5992 to 0.8185, ($p= 0.0040$). The AUC value of blood HGB is 0.8599. The S.E value is 0.03756 and the 95% CI value is 0.7862 to 0.9335, ($p<0.0001$), they exhibit that hematological changes are potential biomarkers for COVID-19.

The data exhibit that CRP, D-dimer, and CD4 could be a potential inflammatory biomarker for COVID-19 with AUC value 0.8150, 0.9605, 0.8995 respectively. The S.E of each one of them 0.09977, 0.02788, 0.04836 and the 95% CI value are 0.6195 to 1.000, 0.9058 to 1.000, 0.8047 to 0.9943, ($p= 0.0056$, $p<0.0001$, $p= 0.0006$) respectively.

The AUC of serum ALT, AST, ALP Plus, T.Bilirubin, D.Bilirubin, and Alb BCG are 0.7844, 0.9042, 0.7352, 0.6901, 0.8775, 0.9776 respectively. The S.E value of each one of them respectively are 0.05944, 0.03196, 0.05710, 0.07022, 0.05012, 0.01283 and the 95% CI value of them are 0.6679 to 0.9009, 0.8416 to 0.9669, 0.6233 to 0.8472, 0.5525 to 0.8277, 0.7793 to 0.9757, and 0.9524 to 1.000, ($p= 0.0002$, $p<0.0001$, $p= 0.0017$, $p= 0.0251$, $p<0.0001$, $p<0.0001$) respectively. According to obtained values, LFT parameters could be regarded as potential biomarker of COVID-19.

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Table (3): ROC curve analysis was used to determine the diagnostic accuracy of WBC, LYM, PLT, CD4, Alb BCG, Crea Enz, Urea, ALT, AST, ALP Plus, T. Bilirubin, D. Bilirubin and Albumin in COVID-19 patients:

Parameters	AUC	S.E.	95% CI	P value
WBC (10^9/l)	0.9912	0.009260	0.9730 to 1.000	<0.0001
LYM (10^9/l)	0.9555	0.02161	0.9132 to 0.9979	<0.0001
PLT (10^9/l)	0.9893	0.008744	0.9721 to 1.000	<0.0001
CRP (mg/l)	0.8150	0.09977	0.6195 to 1.000	0.0056
D-dimer (µg/ml)	0.9605	0.02788	0.9058 to 1.000	<0.0001
CD4 (ng/ml)	0.8995	0.04836	0.8047 to 0.9943	0.0006
Crea. Enz (mg/dl)	0.9601	0.02289	0.9153 to 1.000	<0.0001
Urea (mg/dl)	1.000	0.000	1.000 to 1.000	<0.0001
ALT (U/l)	1.000	0.000	1.000 to 1.000	<0.0001
AST (U/l)	1.000	0.000	1.000 to 1.000	<0.0001
ALP Plus (U/l)	0.8504	0.04605	0.7602 to 0.9407	<0.0001
T.Bilirubin (mg/dl)	1.000	0.000	1.000 to 1.000	<0.0001
D.Bilirubin (mg/dl)	0.8775	0.05012	0.7793 to 0.9757	<0.0001
Albumin (g/dl)	0.9906	0.009833	0.9714 to 1.000	<0.0001

4.Conclusion

It is concluded that in Covid-19 infected patients, renal and liver functions are significantly reduced with increased inflammatory response. And COVID-19 causes multi organic failure in the last stage of infection of severe cases.

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Zhao, X., Wang, K., Zuo, P., Liu, Y., Zhang, M., Xie, S., ... & Liu, C. (2020). Early decrease in blood platelet count is associated with poor prognosis in COVID-19 patients—indications for predictive, preventive, and personalized medical approach. EPMA Journal, 11(2), 139-145.

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وەڵامدانەوەی هەوکردن و کاریگەری ڤایرۆسی کۆرۆنا لەسەر بەشێک لە ئەندامەکانی لەش لە نەخۆشانی نەخۆشخانە

پـوختـه:

لە سێ ساڵی ڕابردوودا، ڤایرۆسی کۆرۆنا بە شێوەیەکی کاریگەر ناسێندرا بە هۆکاری سەرەکی مردن لە سەرانسەری جیهاندا. ئەم ڤایرۆسە لە سەرەتای هەوکردندا چالاکانە زیان بە بەشە سەرەکییەکانی کۆئەندامی هەناسە دەگەیەنێت. وەڵامدانەوەی بەرگری لەوانەیە هەوکردنەکە سنووردار بکات و نەخۆشەکە تا ڕادەیەک ڕزگار بکات. هەرچەندە هەوکردنی درێژخایەن لەوانەیە کاریگەرییەکی سیستماتیکی جددی هەبێت و کارکردنی ئەندامەکانی تر کەم بکاتەوە لەوانە گورچیلە و جگەر. لێکۆڵینەوە لە وەڵامی هەوکردن و نیشاندەری سەرەکی تێکچوونی گورچیلە و جگەر ئامانجی سەرەکی ئەم توێژینەوەیە.

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پارامێتەرەکانی خوێن، وەڵامی هەوکردن و فاکتەری کۆئەگولاسیۆن بە CBC و 4CD و CRP و D-dimer لە نەخۆشانی تووشبوو بە 19-Covid و لە نەخۆشانی غەیرە کۆرۆنادا دیاریکران. هەروەها چڕیی سیرۆمی Crea و Urea و ALT و AST و ALP و TSB و DB و Albumin لە نەخۆشانی تووشبوو بە 19Covid و لە نەخۆشانی غەیرە ڤایرۆسی کۆرۆنا لێکۆڵینەوەی لەسەر کرا.

لە نەخۆشانی تووشبوو بە 19-Covid، نیشانەکانی هەوکردن، CRP و WBC زیاد دەبن و پارامێتەرەکانی بەرگری، 4CD و LYM کەم دەبنەوە. چڕیی سیرۆمی Crea و Urea و ALT و AST و ALP و TSB و DB هەموویان لە نەخۆشانی تووشبوو بە 19-Covid زیاد دەبن، لە کاتێکدا ئاستی سیرۆمی Albumin کەم بووەتەوە. لە نەخۆشە تووشبووەکانی 19Covid بەرزبوونەوەی ئاستی پلازمای D-dimer بەدیکرا.

بەو ئەنجامە دەگەین کە لە نەخۆشانی تووشبوو بە 19-Covid، کارکردنی گورچیلە و جگەر بە شێوەیەکی بەرچاو کەم دەبێتەوە لەگەڵ زیادبوونی وەڵامی هەوکردن.

الاستجابة الالتهابية وتأثير الفيروس كورونا على بعض أعضاء الجسم في المرضى داخل المستشفى

الملخص:

في السنوات الثلاث الماضية، تم تقديم فيروس كورونا بشكل فعال إلى السبب الرئيسي للوفاة في جميع أنحاء العالم. يضر هذا الفيروس بنشاط الأجزاء الرئيسية من الجهاز التنفسي في الإصابة المبكرة. الاستجابة المناعية قد تحد من العدوى وتنقذ المريض إلى حد ما. ومع ذلك، قد يكون للعدوى المزمنة تأثير منهجي خطير وتقلل من وظائف الأعضاء الأخرى بما في ذلك الكلى والكبد. الغرض الرئيسي من هذه الدراسة هو التحقيق في الاستجابة الالتهابية والمؤشرات الرئيسية لتلف الكلى والكبد.

تم تحديد المعلمات الدموية والاستجابة الالتهابية وعامل التخثر باستخدام CBC و 4CD و CRP و D-dimer في المرضى المصابين بـ 19-Covid وفي المرضى غير المصابين بفيروس كورونا. كما تمت دراسة تركيزات مصل الكرياتينين (Crea) واليوريا و ALT و AST و ALP و TSB و DB و Albumin في المرضى المصابين بـ 19Covid وفي المرضى غير المصابين بفيروس كورونا.

في المرضى المصابين بـ 19-Covid ، تزداد علامات الالتهاب و CRP و WBC وتقل المعلمات المناعية و 4CD و LYM. تزداد تركيزات المصل من Crea و urea و ALT و AST و ALP و

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TSB و DB في المرضى المصابين بفيروس 19-Covid ، بينما انخفض مستوى Albumin في المصل. في المرضى المصابين بـ 19Covid لوحظ زيادة في مستوى D-dimer في البلازما.
وخلص إلى أنه في المرضى المصابين بـ 19-Covid ، تنخفض وظائف الكلى والكبد بشكل كبير مع زيادة الاستجابة الالتهابية.