International Journal of Advanced Biochemistry Research

ISSN Print: 2617-4693
ISSN Online: 2617-4707
IJABR 2024; 8(9): 155-160
www.biochemjournal.com
Received: 14-06-2024
Accepted: 15-07-2024

Hanaa N Abdullah
Collage of Health and Medical Techniques, Middle Technical University, Baghdad, Iraq

Zainab S Muhana
Collage of Health and Medical Techniques, Middle Technical University, Baghdad, Iraq

Rana A Hikmet
Collage of Health and Medical Techniques, Middle Technical University, Baghdad, Iraq

Corresponding Author:
Hanaa N Abdullah
Collage of Health and Medical Techniques, Middle Technical University, Baghdad, Iraq

Estimation of biochemical parameters among COVID-19 patients

Hanaa N Abdullah, Zainab S Muhana and Rana A Hikmet

DOI: https://doi.org/10.33545/26174693.2024.v8.i9c.2106

Abstract
A SARS-CoV-2 infection is the source of the infectious illness COVID-19. The SARS CoV2 infection has remained severe after the World Health Organization (WHO) deemed it to be a global health crisis. This research aims to assess a few biochemical indicators in individuals with COVID-19. There are fifty COVID-19-infected patients in this study who received medical care at the Al-Najaf Al-Ashraf Teaching Hospital in Iraq. Levels of RBS, S. BUN, S. cre., D. dimer, and ferritin were analyzed by the Cobas c-111 and Hipro-TM devices. All patient measures were contrasted with those of fifty healthy people serving as a control group. The results of this study showed that, among the fifty COVID-19 patients, males had a higher infection rate than females. Out of 50 COVID-19 patients, 62% were men, and 38% were women. When contrasting the patient group with the control group, the mean of glucose is significantly different between the patient group and the control group, and the mean of blood glucose is (223.26±18.79) mg/dl. In addition, the mean blood sugar was (247.13±26.01) mg/dl in males, while the mean blood sugar was (184.31±23.57) mg/dl in females. There were no significant differences between the patient and the control group in the mean of urea and creatinine, in the patient group, the mean (BUN) was (63.02±15.08) mg/dl and (0.770±0.09) mg/dl, respectively. While in the control group, the mean (BUN) was (26.76±1.03) mg/dl and (0.672±0.03) mg/dl, respectively. D-dimer and ferritin levels in patients showed significant differences when compared with the control group; the mean was (3581.16±472.15) ng/mL and (691.50±60.07) ng/mL, respectively, in the patient group, while the mean was (165.96±6.72) ng/mL and (161.22±7.38) ng/mL, respectively, in the control group.

Keywords: COVID-19, RBS, S. BUN, ferritin, D. dimer

1. Introduction

The Chinese city of Wuhan was the core of mysterious pneumonia cases in December 2019, which were connected to a new coronavirus in January 2020 and swiftly escalated into a worldwide public health disaster [1]. This ailment, which is related to a beta coronavirus, can infect mammals as well as other animals. It is composed of single chains of positive RNA and is a member of the huge Coronaviridae subfamily [2]. The coronavirus 2 of the severe acute respiratory syndrome (SARS-CoV-2) [3] is the cause of the 2019 coronavirus illness (COVID-19). On March 11, 2020, the World Health Organization (WHO) formally declared it to be a worldwide crisis due to its rapid global spread [4].

A spectrum of (COVID-19) symptoms is wide, ranging from no symptoms to a potentially fatal illness. The virus is mostly spread through the air when people are close to one another. When an infected individual breathes, coughs, sneezes, or interacts with others, the virus can spread from them through their mouth, nose, or eyes. Additionally, it's probable that it will spread through contaminated surfaces because infected persons can carry the virus for up to two weeks after contracting it, even in the absence of symptoms [5, 6].

The Pathogenesis of COVID-19 may have an impact on the upper and lower respiratory system [7]. Because COVID-19 gets into host cells via the enzyme Angiotensin-Converting Enzyme 2 (ACE2), which is most prevalent in type II alveolar cells of the lungs, the lungs are the organs most hardest hit by the virus [8]. The density of (ACE2) in each tissue corresponds with the severity of the illness in that tissue, therefore, reduced (ACE2) activity may be protective [9].

Numerous investigations are now being conducted with the goal of elucidating the specifics of the virological and clinical progression of SARS-CoV-2 infection.

Consequently, research indicates that comorbid conditions including diabetes and hypertension, together with advanced age, are associated with a greater risk of death and COVID-19 infection. Glycaemic control has received a lot more attention for people with both diabetes and COVID-19 as it has been shown to be linked to a poor prognosis for the virus. There is proof that improved glycaemic management is directly linked to better clinical outcomes for COVID-19 patients [10].

Renal complications are yet another frequent cause of death. According to preliminary research, as many as 30% of hospitalized patients in China and New York—including some who hadn't experienced kidney issues before—have some form of kidney injury [11]. The findings are consistent with the hypothesis that renal damage is prevalent in COVID-19 patients and may be a major factor in the disease's severity, ultimately leading to multiple organ failure and life loss. Thus, clinical management of infected individuals and beginning therapy early of renal failure with continuous therapy would both critically depend on kidney function monitoring [12].

The D-dimer is When fibrinolysis breaks down a blood clot, a small protein fragment known as the fibrin degradation product, or FDP, is released into the bloodstream. It is called upon by the fact that it is composed of D-fibrin protein fragments that are connected to one another via a cross-link [13]. D-dimer tests are widely used in medical practice to rule out the possibility of pulmonary embolism or deep vein thrombosis, and an elevated D-dimer indicates a higher risk of abnormal blood clotting. A higher mortality rate in community-acquired pneumonia has also been linked to elevated D-dimer ranges 14[].

The progression of COVID-19 illness may be associated with the protracted D-dimer stage. When (COVID-19) patients are hospitalized to the intensive care unit (ICU), their D-dimer stage is shown to be significantly better (15). When it comes to the risk of venous thromboembolism, patients with severe (COVID-19) who were frequently bedridden and had aberrant coagulation activity should receive special care [16].

An established intracellular protein called ferritin is responsible for controlling the release and storage of iron. It shields people from iron overload and shortage by functioning as a buffer [17]. It is present as a cytosolic protein in the majority of tissues. Nonetheless, small amounts are discharged into the serum, where it functions as a transporter of iron. Ferritin plays a major role in immunological dysregulation, especially when there is an extreme case of hyperferritinemia, which exacerbates the cytokine syndrome [18]. It has been noted that (COVID-19) associated deaths are linked to cytokine typhoon syndrome, which suggests that cytokine storms promote the severity of illness through direct immune-suppressive and pro-inflammatory actions [19, 20].

The analysis of the significance of biochemical biomarkers (RBS, S. urea S. cre, D-dimer, and ferritin) in COVID-19 patients is the main goal of this work.

2. Materials and Methods

2.1 Sample collection

The research study, which spanned from January 2021 to June 2021, involved 100 samples—50 patients who were receiving care at Al-Najaf Al-Ashraf Teaching Hospital and 50 samples from healthy, non-smoking people who do not suffer from chronic diseases that served as controls. Age range: 20–70 years old.

2.2 Methods

Both patients and controls had five milliliters of venous blood drawn and separated into two tubes (vacuum gel-plane tubes and vacuum sodium citrate tubes). The first part of the blood samples was centrifuged to obtain serum, which is used for the determination of serum concentrations of random blood sugar (RBS), serum blood urea (S. urea), and serum blood creatinine (S. cre.) by Cobas c 111 (Roche-USA). The second part of the blood samples (sodium citrate tubes) is applied to the Hipro™ device, manufactured by Hipro Biotechnology Corp. (USA) for measuring D. dimer and ferritin.

2.3 Study design

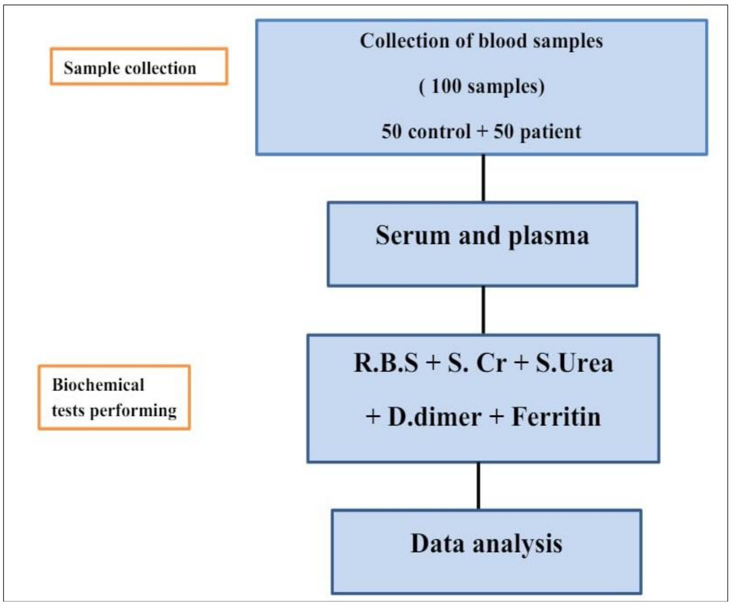

Fig 1: Research study design

2.3 Statistical Analysis
The Statistical Analysis System (SAS, 2018) software was employed to assess the impact of different groups (patients and controls) on the study parameters. A t-test was used to compare means for statistical significance, while a chi-square test was utilized to compare percentages for significance levels of 0.05 and 0.01 in this study [21].

3. Result and discussion
3.1 Analysis of sample study distribution among COVID patients and control groups based on gender
The outcome of this study based on collection of blood from patients that were mostly in ICU and it showed that the percentage of COVID-19 cases was higher in males compared to females, and this difference was statistically significant. In the control group, there was no significant difference between males and females. Additionally, no significant difference was observed between patients and controls, regardless of gender as shown in (Table 1).

Table 1: Distribution of the study sample by sex in COVID patients and control groups.

Factor		Patients (No=50)	Control (No = 50)	P-value.
Sex: No (%)	Male	31 (62.00%)	26 (52.00%)	0.507 NS.
	Female	19 (38.00%)	24 (48.00%)	0.581 NS.
	P-value.	0.0497 *	0.773 NS	---
* (p≤ 0.05), NS : Non- Significant.				

The gender disparity in the severity of COVID-19 illness may result from behavioral and sociocultural differences based on gender. In Iraqi society, men are more likely than women to visit crowded locations and leave the house. Because of the differences in access to medical facilities and treatment, infection rates may be biased toward men [22].
Because the X chromosome contains a large number of immune-related genes, women generally exhibit stronger innate and adaptive immune responses compared to men. The differences in immune responses between men and women may be related to sex-specific inflammatory reactions that are influenced by the X chromosome. Males are more susceptible to infections than females [23].
Sharma and colleagues [23] point to a number of important contributing factors to this phenomenon. Differential immune response activation in men and women these differences may be influenced by sex chromosomal genes and sex hormones like progesterone, estrogen, and androgens.
After viral infections, sex-specific disease outcomes are affected by several factors, including differences in the copy number of immune response genes linked to the X chromosome., sex-dependent steroid hormone synthesis, and virus susceptibility genes. Initial data from China indicated that the majority of hospitalized patients were male, with a median age of 56., 26% in need of an intensive care unit, and 28% dying [24]. This is evident from the results of the 50 patients in the current investigation, which showed that in all groups divided by the severity of the infection, women had a lower proportion than males.
Another aspect is the gender distribution in Iraqi society, which is somewhat greater than the percentage of females in the overall population, according to the website's Indexmundi statistics for 2020. The ratio of men to women in Iraq is 102.48 men for every 100 women [25]. Our findings agree with (Al-Rayahi.) who also discovered that the prevalence of COVID illness was greater in men than in women [26].

3.2 Evaluation of R.B.S. control groups with COVID patients
The findings of this study, displayed in (Table 2), demonstrated that there was a highly significant difference between the Covid patients and control group and that the random blood sugar level became more severe in Covid patients as compared to control patients, especially in elderly individuals.

Table 2: Comparison between (COVID) patients and control groups in (R.B.S).

Group.	Mean±SE o RBS (mg/dl)
Patients	223.26±18.79
Control	93.66±2.05
T-test	37.527 **
P-value	0.0001
** (P≤0.01).	

In the current investigation, RBG levels were a separate risk factor for death. This result is consistent with research from a study conducted in Wuhan, China [27]. It was discovered that among COVID-19 patients, the capillary blood glucose level at admission constituted an independent risk factor for mortality [28].
There are a few reasons why RBG might be a separate risk factor for COVID-19 death; Increased glucose levels may encourage SARS-CoV-2 replication is influenced by the actions of hypoxia-inducible factor 1-alpha and mitochondrial functions. ROS [29]; High blood sugar may result in glucose toxicity, which raises the risk of ARDS (Serious lung condition that causes low blood oxygen) and death by causing interstitial lung damage [30]; Increased blood sugar levels may also result in endothelial damage, which raises the possibility of Thromboembolic events, such as pulmonary embolism [30], are therefore a concern., blood glucose control is essential when a patient is in the hospital, as indicated by a prior study [31]. In addition to in-hospital therapies, enhancing patients' self-efficacy, social support, and self-care practices may help achieve glycemic control [31]. These findings align with the study by Kocak *et al*., which observed elevated glucose levels in COVID-19 patients compared to the control group [33].

3.2 Comparison analysis of BUN and S. Creatinine levels between COVID patients and control groups
The results of the current research, as shown in (Table 3), demonstrated a substantial difference in urea levels between male and female patients, whereas there was no significant difference in S. Creatinine levels.

Table 3: Comparison between COVID patients and control groups in Urea and S. Creatinine

Group.	Mean ±SE	
	Urea (mg/dl)	S. Creatinine (mg/dl)
Patients	63.02±15.08	0.770±0.09
Control	26.76±1.03	0.672±0.03
T-test	30.01 *	0.194 N S
P- value	0.0184	0.318
* (P≤0.05), N S : Non-Significant.		

In this research it was found that certain COVID-19-infected individuals had (BUN) and creatinine levels that were greater than usual. Also, Sarhan *et al*., and Luther *et al*. found in their study on COVID-19-infected individuals elevated levels of creatinine and urea in comparison to normal ranges [34, 35].

A newly discovered respiratory infectious illness called COVID-19 has been linked to kidney damage in certain cases of its patients' clinical problems. Nonetheless, varying degrees of COVID-19 patient severity were linked to the dynamic variations in the two renal function indicators, creatinine and blood urea nitrogen. When it comes to severity categorization and triage, BUN has a strong correlation and a significant potential for predicting unfavorable outcomes in COVID-19 patients [36].

The exact mechanism behind the increase in BUN following SARS-CoV-2 infection remains unclear. Given that angiotensin-converting enzyme 2 (ACE2), the primary receptor for SARS-CoV-2, is abundantly expressed in renal epithelial cells, the virus may interact directly with this receptor in the kidneys, leading to reduced ACE2 expression. This interaction could result in abnormal activation of the renin-angiotensin-aldosterone system (RAAS). Elevated BUN levels might then occur due to the RAAS-induced increase in water absorption by renal tubules and enhanced urea reabsorption [37, 38].

While Acute renal failure has primarily been reported in critically ill individuals., it has also been shown that serum creatinine and cystatin C levels in both severe and less severe instances are normal (n = 178). Only 2.8% of the patients had elevated BUN levels, and none of them had a rise in serum creatinine, despite the fact that 23.6% of them had a decreased estimated glomerular filtration rate (eGFR). These findings show that, independent of their ICU admission, None of the patients experienced acute renal damage or acute renal insufficiency throughout their entire hospital stay [39].

3.3 D-dimer and Ferritin analysis

The outcome of this analysis indicated a highly elevated and significant difference in D-dimer and ferritin levels between male and female patients, where the mean was 3581.16±472.15 ng/mL and 691.50±60.07 ng/mL, respectively, in comparison with the control group as shown in the (Table 4).

Table 4: Comparison between COVID patients and control groups in D-dimer and Ferritin

Group	Mean±SE	
	D-dimer (ng/mL)	Ferritin (ng/mL)
Patients	3581.16±472.15	691.50±60.07
Control	165.96±6.72	161.22±7.38
T-test	937.07	120.12 **
P-value	0.0001	0.0001
** (*p*≤0.01).		

D-dimer is an indicator that is sensitive to reflecting thrombosis and may specifically indicate secondary hyperfibrinolysis and a hypercoagulable state in the body. Due to its direct immunosuppressive properties and pro-inflammatory actions, ferritin plays a major role in immunological dysregulation, especially in cases of severe hyperferritinemia, which fuels the cytokine storm. Although ferritin is found in the intestinal mucosa, liver, kidney, spleen, and heart, it is unknown if serum ferritin contributes to or reflects inflammation or if it is a byproduct of inflammation [40, 41].

The vital intracellular iron store protein, or ferritin, is a positive acute phase reactant that increases under inflammatory circumstances. Furthermore, conditions including septic shock and macrophage activation syndrome have been linked to extremely high ferritin levels. As a biomarker for fiber production and breakdown, d-dimer is the primary fibrin disintegration fragment. Low amounts of D-dimer are seen in the blood of healthy individuals, but excessive levels are found in conditions linked to thrombosis [42, 43].

Our study's findings indicated that patients with COVID-19 disease had higher levels of D-dimer and ferritin, which is in line with studies done by Abdel Fattah *et al.* and (Kadhim and Abdullah.), who also discovered high levels of these two biochemical parameters in COVID-19 disease patients [44, 45].

3.5 Analysis the influence of gender in COVID-19 patient on biochemical tests

Numerous laboratory indicators facilitate the evaluation of the disease's severity and the forecasting of its potential progression towards more severe conditions, such as elevated BUN, creatinine, RBS, D-dimer, and ferritin, which are some of the characteristics for which an adverse course of the disease has been documented. In (Table 5), we summarise all biochemical parameters included in this study in relation to patient gender.

Table 5: Effect of Sex in parameters study of COVID patients group

Parameters.	Mean. ±SE		P-value.
	Male.	Female.	
RBS (mg/dl)	247.13±26.01	184.31±23.57	0.0074 **
S. Creatinine (mg/dl)	71.26±23.91	49.58±7.69	0.0719 NS
Urea (mg/dl)	0.725±0.04	0.842±0.23	0.894 NS
D-dimer (ng/mL)	3758.00±616.87	3292.63±744.84	0.251 NS
Ferritin (ng/mL)	743.54±72.58	606.57±104.31	0.167 NS
** (P≤0.01), NS: Non-Significant.			

In comparison with the other parameters in COVID patients, the analysis's outcome showed a considerably raised and significant difference in the random blood sugar level between male and female patients, with means of 247.13±26.01 mg/dl and 184.31±23.57 mg/dl, respectively. Our study also found that the level of creatinine in the blood increased at a slight rate in males compared to females, with means of 71.26±23.91 mg/dl and 49.58±7.69 mg/dl, respectively as shown in (Table 5). The elevated levels of RBS in our study agree with the study of Chen *et al*. In this meta-analysis, Three studies examining blood glucose levels in relation to COVID-19 severity were analyzed. The combined data showed a correlation between higher blood glucose levels and severe COVID-19 (WMD 2.21, 95% CI: 1.30–3.13, p< 0.001). Proper monitoring of blood glucose could improve the prognosis for COVID-19 patients, as elevated blood glucose is associated with severe disease [46]. Additionally, the slightly increased BUN and serum creatinine levels observed in our study align with findings from Zarębska-Michaluk *et al*., who reported elevated BUN levels in 23 cases, with 43% showing an increase within 2 to 10 days, and two-thirds of the non-surviving patients having significantly high levels before death. Among the 59 patients studied, 27% had elevated urea nitrogen levels, and 11 out of 59 patients (or 19% of the total) had elevated creatinine levels.

4. Conclusion

The laboratory's role involves evaluating the biochemical parameters of COVID-19 patients. Clinical laboratories are crucial to the diagnosis of the virus, patient follow-up (tracking their progress), and epidemiological surveillance by identifying serum-related indicators in their systems. Validated SARS-CoV-2 laboratory assays are essential for early COVID-19 management because they facilitate clinical decision-making regarding infection control and asymptomatic case detection. This facilitates prompt isolation and appropriate treatment, which in turn lowers the rate of contagion. Among all the biochemical markers assessed in our investigation, RBS, D-dimer, ferritin, blood urea nitrogen, and creatinine showed a significant difference, in that order. In fifty samples from COVID-19 patients, the infection incidence in men was higher than in women. In general, results and observations typically showed that males usually had higher levels of all biochemical markers than females.

5. Reference

1. Xiang HX, Fei J, Xiang Y, Xu Z, Zheng L, Li XY, *et al*. Renal dysfunction and prognosis of COVID-19 patients: a hospital-based retrospective cohort study. BMC Infectious Diseases. 2021;21:158. DOI: 10.1186/s12879-021-05861-x.
2. Sarhan AR, Flaih MH, Hussein TA, Hussein KR. Novel coronavirus (COVID-19) outbreak in Iraq: The first wave and future scenario. BMJ. 2020. DOI: 10.1101/2020.05.14.20091026. (Preprint from Cold Spring Harbor Laboratory).
3. Huang C, Wang Y, Li X, Ren L, Zhao J, Hu Y, *et al*. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. The Lancet. 2020;395:497-506. DOI: 10.1016/S0140-6736(20)30183-5.
4. Liu YM, Xie J, Chen MM, Zhang X, Cheng X, Li H, *et al*. Kidney function indicators predict adverse outcomes of COVID-19. Med. 2021;2(1):38-48. DOI: 10.1016/j.medj.2020.11.001.
5. World Health Organization. WHO Director-General’s opening remarks at the media briefing on COVID-19 - 11 March 2020. WHO Director General’s speeches. Geneva: World Health Organization; 2020. Available from: https://www.who.int/dg/speeches/detail/who-director-general-s-opening-remarks-at-the-media-briefing-on-COVID-19---11-march-2020.
6. Machhi J, Herskovitz J, Senan AM, Dutta D, Nath B, Oleynikov MD. The natural history, pathobiology, and clinical manifestations of SARS-CoV-2 infections. Journal of Neuroimmune Pharmacology; c2020 .p. 1-28. DOI: 10.1007/s11481-020-09944-0.
7. Harrison AG, Lin T, Wang P. Mechanisms of SARS-CoV-2 transmission and pathogenesis. Trends in Immunology. 2020;41(12):1100-1115. DOI: 10.1016/j.it.2020.10.004.
8. Verdecchia P, Cavallini C, Spanevello A, Angeli F. The pivotal link between ACE2 deficiency and SARS-CoV-2 infection. European Journal of Internal Medicine. 2020;76:14-20. DOI: 10.1016/j.ejim.2020.04.037.
9. Rodríguez-Puertas R. ACE2 activators for the treatment of COVID-19 patients. Journal of Medical Virology. 2020;92(10):2102-2104. DOI: 10.1002/jmv.25946.
10. Zheng Z, Peng F, Xu B, Zhao J, Liu H, Peng J, *et al*. Risk factors of critical and mortal COVID-19 cases: A systematic literature review and meta-analysis. Journal of Infection. 2020, 81(2). DOI: 10.1016/j.jinf.2020.04.021.
11. Oudit GY, Pfeffer MA. Plasma angiotensin-converting enzyme 2: novel biomarker in heart failure with implications for COVID-19. European Heart Journal. 2020;41(19):1818-1820.
12. Cascella M, Rajnik M, Aleem A, Dulebohn SC, Di Napoli R. Features, evaluation, and treatment of coronavirus (COVID-19). StatPearls; c2023.
13. Adam SS, Key NS, Greenberg CS. D-dimer antigen: current concepts and future prospects. Blood, The Journal of the American Society of Hematology. 2009;113(13):2878-2887.
14. Snijders D, Schoorl M, Bartels PC, van der Werf TS, Boersma WG. D-dimer levels in assessing severity and clinical outcome in patients with community-acquired pneumonia: A secondary analysis of a randomised clinical trial. European Journal of Internal Medicine. 2012;23(5):436-441.
15. Huang C, Wang Y, Li X, Ren L, Zhao J, Hu Y, *et al*. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. The Lancet. 2020;395(10223):497-506.
16. Chen N, Zhou M, Dong X, Qu J, Gong F, Han Y, *et al*. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. The Lancet. 2020;395(10223):507-513.
17. Casiday R, Frey R. Iron use and storage in the body: ferritin and molecular representations. Department of Chemistry, Washington University, St. Louis. USA; c1998.
18. Huang C, Wang Y, Li X, Ren L, Zhao J, Hu Y, *et al*. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. The Lancet. 2020;395(10223):497-506.
19. Khalil UA, Seliem FO, Alnahal A, Awad M, Sadek AM, Fawzy MS. Association of serum ferritin with insulin resistance in offsprings of type 2 diabetics. The Egyptian Journal of Internal Medicine. 2018;30(1):13-17.
20. Son NE. Influence of ferritin levels and inflammatory markers on HbA1c in Type 2 Diabetes mellitus patients. Pakistan Journal of Medical Sciences. 2019;35(4):1030.
21. SAS. Statistical Analysis System, User's Guide. Statistical Version 9.6th ed. SAS. Inst. Inc. Cary. N.C. USA; c2018.
22. Judah G, Aunger R, Schmidt WP, Michie S, Granger S, Curtis V. Experimental pretesting of hand-washing interventions in a natural setting. American Journal of Public Health. 2009, 99(S2).
23. Sharma G, Volgman AS, Michos ED. Sex differences in mortality from COVID-19 pandemic: are men vulnerable and women protected? Case Reports. 2020;2(9):1407-1410.

24. Wayne MT, Weng W, O'Malley M, Bozyk P, Doshi MM, Flanders SA, *et al*. Variation in COVID-19 disease severity at hospital admission over time and across hospitals: A multi-institution cohort of Michigan hospitals. Medicine. 2021, 100(37). IndexMundi. https://www.indexmundi.com/
25. Al-Rayahi AM. A Study of Some Biochemical Parameters in COVID-19 Patients with Diabetes Mellitus: Comparative Study. Journal of Techniques. 2022, 4(4 Special Issue).
26. Shi Q, Zhang X, Jiang F, Zhang X, Hu N, Bimu C, *et al*. Clinical characteristics and risk factors for mortality of COVID-19 patients with diabetes in Wuhan, China: a two-center, retrospective study. Diabetes Care. 2020;43(7):1382-1391.
27. Chaudhuri SR, Majumder A, Sanyal D, Biswas A, Bhattacharjee K. 1098-P: Admission blood glucose but not HbA1c predicts mortality in people with diabetes hospitalized with COVID-19 infection. Diabetes. 2021;70(Supplement_1).
28. Lim S, Bae JH, Kwon HS, Nauck MA. COVID-19 and diabetes mellitus: from pathophysiology to clinical management. Nature Reviews Endocrinology. 2021;17(1):11-30.
29. Nor M, Nurrasyidah I, Mashuri M. The Correlations Between Clinical Characteristics and Inflammation Markers with Chest X-rays in COVID-19 Patients at Ulin Hospital. Jurnal Respirologi Indonesia. 2023;43(4):247-54.
30. Shi Q, Zhang X, Jiang F, Zhang X, Hu N, Bimu C, *et al*. Clinical characteristics and risk factors for mortality of COVID-19 patients with diabetes in Wuhan, China: A two-center, retrospective study. Diabetes Care. 2020;43(7):1382-91.
31. Paulsamy P, Ashraf R, Alshahrani SH, Periannan K, Qureshi AA, Venkatesan K, *et al*. Social support, self-care behaviour and self-efficacy in patients with Type 2 diabetes during the COVID-19 pandemic: A cross-sectional study. Healthcare. 2021;9(11):1607.
32. Kocak OF, Ozgeris FB, Parlak E, Kadioglu Y, Yuce N, Yaman ME, *et al*. Evaluation of serum trace element levels and biochemical parameters of COVID-19 patients according to disease severity. Biological Trace Element Research; c2022 .p.1-9.
33. Sarhan AR, Hussein TA, Flaih MH, Hussein KR. A biochemical analysis of patients with COVID-19 infection. Biochemistry Research International; c2021.
34. Luther T, Bülow-Anderberg S, Larsson A, Rubertsson S, Lipcsey M, Frithiof R, *et al*. COVID-19 patients in intensive care develop predominantly oliguric acute kidney injury. Acta Anaesthesiologica Scandinavica. 2021;65(3):364-372.
35. Liu YM, Xie J, Chen MM, Zhang X, Cheng X, Li H, *et al*. Kidney function indicators predict adverse outcomes of COVID-19. Med. 2021;2(1):38-48.
36. Deng YY, Zheng Y, Cai GY, Chen XM, Hong Q. Single-cell RNA sequencing data suggest a role for angiotensin-converting enzyme 2 in kidney impairment in patients infected with 2019-novel coronavirus. Chinese Medical Journal. 2020;133(9):1129-31.
37. Hirsch JS, Ng JH, Ross DW, Sharma P, Shah HH, Barnett RL, *et al*. Acute kidney injury in patients hospitalized with COVID-19. Kidney International. 2020;98(1):209-218.
38. Gharaibeh KA, Hamadah AM, El-Zoghby ZM, Lieske JC, Larson TS, Leung N. Cystatin C predicts renal recovery earlier than creatinine among patients with acute kidney injury. Kidney International Reports. 2018;3(2):337-342.
39. Kumar M, Bindu CM, Shyam AC, Reena R. Ferritin – The key model inflammatory marker in diabetic and non-diabetic COVID-19. Asian Journal of Medical Sciences. 2021;12(12):1-6.
40. Pangaribuan JP, Pase MA. Correlation between Hblc and D-dimer in Type 2 Diabetic with COVID-19 Patients. Journal of Endocrinology, Tropical Medicine, and Infectious Disease. 2021;3(4):145-151.
41. Huyut MT, Huyut Z. Effect of ferritin, INR, and D-dimer immunological parameters levels as predictors of COVID-19 mortality: A strong prediction with the decision trees. Heliyon. 2023, 9(3).
42. Farasani A. Biochemical role of serum ferratin and d-dimer parameters in COVID-19 diagnosis. Saudi Journal of Biological Sciences. 2021;28(12):7486-90.
43. AbdelFattah EB, Madkour AM, Amer SMI, Ahmed NO. Correlation between the serum level of ferritin and D-dimer and the severity of COVID-19 infection. The Egyptian Journal of Bronchology. 2023;17(1):1-11.
44. Kadhim AS, Abdullah YJ. Serum levels of interleukin-6, ferritin, C-reactive protein, lactate dehydrogenase, D-dimer, and count of lymphocytes and neutrophils in COVID-19 patients: Its correlation to the disease severity. Biomedical and Biotechnology Research Journal (BBRJ). 2021;5(1):72.
45. Jasim AA, Mohammed MJ. Correlation between D-dimer level and clinical outcomes of COVID-19 patients in AL-Nasiriya City, Iraq. Indian Journal of Forensic Medicine and Toxicology. 2022;16(1).