MISJ-International Journal of Medical Research and Allied Sciences
Online ISSN: 2584-1432
Available Online at http://ijmraas.misj.net Volume 02|Issue 01 (January to April)|2024 Page: 22-30

Original Research Paper

# Comparison of Hematological Profile and Biochemical Markers of COVID-19 among Survivors and Non-Survivors of COVID-19 Patients in Eastern Region of Libya

**Authors:**

**Azeza S Guma[1], Fadhila. M. A. Othman[2], Hamza. M. A. Othman[3]**

[1] *Omar Al-Mukhtar University, Faculty of Medical Technology, Department of Lab. Medicine, Box 919, Al-Bayda, Libya*

[2] *Higher Institute of Sciences and Medical Technology, El -Bayda-Libya, Department of Medical Laboratories*

[3] *Omar Al- Mukhtar University, Faculty of Veterinary Medicine, Department of Pathology and Clinical Pathology, Al-bayda, 919, Libya.*

**Corresponding Author:**

Azeza S Guma

Article Received: 22-November-2023 Revised: 12-December-2023 Accepted: 02-January-2024

**ABSTRACT**:

**Background and Objectives**: The primary objective of this investigation was to examine various parameters that have the potential to serve as predictors of mortality in patients who have received a diagnosis of COVID-19 while hospitalized. **Materials and Methods**: In this prospective study, individuals who had been diagnosed with COVID-19 while in the hospital were included as participants. These patients were subsequently categorized into two distinct groups. Group 1, referred to as survivors, consisted of those patients who managed to overcome the illness, whereas Group 2, known as non-survivors, encompassed those patients who unfortunately succumbed to the disease. A comparative analysis was then undertaken to assess the laboratory findings of both groups. **Results**: Out of the total 200 patients included in this study, a significant majority of 128 individuals (equating to 64%) were able to survive the ordeal, while a smaller proportion of 72 patients (36 %) were unable to overcome the disease. It was revealed that non-survivor patients exhibited elevated heart rates, along with diminished peripheral oxygen saturation (SpO2) levels and diastolic blood pressure (DBP). The levels of white blood cells (WBC), lactate, D-dimer, C - reactive protein (CRP), alanine aminotransferase (ALT), aspartate aminotransferase (AST), and serum potassium were observed to be elevated, while the levels of lymphocytes and platelets were observed to be reduced in patients who did not survive.

**Conclusion**: COVID-19 is a severe illness with a mortality risk that can be estimated by evaluating the clinical and laboratory tests of patients. The measurement of oxygen saturation (SpO2) prior to initiating oxygen therapy, the levels of lymphocytes, and the laboratory findings all serve as significant predictors of mortality risk.

***Keywords: COVID-19, Oxygen saturation, Respiratory rate, Blood pressure, Heart rate, haematological marker, survivor, non-survivor Age, and Mortality***

## INTRODUCTION:

The newly discovered severe acute respiratory syndrome coronavirus (SARS-CoV-2), which was initially detected in China towards the end of 2019, rapidly impacted the global population and led to a widespread pandemic. Termed as coronavirus 2019 (COVID-19), this illness has the potential to induce severe pneumonia as well as the fatal acute respiratory distress syndrome (ARDS) (1). The COVID-19 pandemic has precipitated five waves of disease and presented significant challenges to healthcare systems worldwide including Libya. The World Health Organization (WHO), in February 2023, has recorded an alarming fatality count of over 6.8 million attributable to the COVID-19 pandemic; however, this estimate may in fact be an underestimation, as the actual number of deaths may exceed twice the officially reported statistics (2). Even with the advent of vaccines, the COVID-19 virus has persistently propagated, and its evolving mutations have the potential to undermine vaccine efficacy and give rise to future surges. The emergence of novel sublineages, such as BA.5, exemplifies the enduring impact of COVID-19 on healthcare systems, thus necessitating a comprehensive investigation into this disease (3).

While the disease may exhibit no symptoms, it is believed that the occurrence of severe acute respiratory distress syndrome (ARDS) can be attributed to an excessive release of inflammatory cytokines during this phase (4). In fact, other studies have indicated that this particular pathogen has the potential to cause a severe respiratory disorder, necessitating specialized care in intensive care units (ICUs) and leading to fatalities in certain instances (5-7). The majority of individuals infected with SARS-CoV-2 experience mild symptoms, such as fever, dry cough, difficulty


© Azeza S Guma et al.

breathing, muscle pain, fatigue, among others. However, in severe cases, the illness can rapidly progress to ARDS, septic shock, hemorrhaging, coagulation abnormalities, metabolic acidosis, and a notable mortality rate (8).

Previous research has demonstrated that alterations in routine blood parameters possess a distinct clinical significance in the prognostication of infectious diseases(9, 10). In fact, numerous irregularities have been observed in the peripheral blood of certain individuals afflicted with an infection(11) (4) (12), (13). Individuals with mild symptoms exhibit less severe manifestations and a favourable prognosis, while patients with severe or critical conditions pose challenges in treatment and are associated with a higher mortality rate (14). Certain investigations have even documented elevated mortality rates in severe COVID-19 patients, despite receiving intensive care intervention (15, 16). However, there is a lack of information regarding the early indicators that can predict severe and fatal cases, and further research is imperative (17, 18). Furthermore, the rapid transmission of the disease raises concerns about the necessity of intensive care, which has the potential to overload healthcare system resources (19).

Despite the growing number of cases observed on a daily basis, there exists limited knowledge pertaining to the hematological and laboratory findings associated with this disease (20)(4), (7), (21). It has been documented that routine laboratory data has demonstrated significant alterations in COVID-19, although a complete understanding of these changes, particularly in patients who have succumbed to the illness, is yet to be fully clarified (22-24).

The identification of prognostic indicators for mortality in patients with COVID-19 is imperative to facilitate informed treatment choices and assess the severity of the disease. Consequently, a significant amount of research endeavours have been directed towards establishing whether individuals who stand to gain from early intervention with supportive care are susceptible and the means to detect their susceptibility (25).

Furthermore, when analysing the alterations in routine blood values in relation to COVID-19, the identification of both severe and mild patients poses a significant challenge from a clinical perspective, considering the implications for morbidity and mortality. Previous research has undertaken comparisons of the routine blood values among individuals affected by COVID-19 based on factors such as the specific treatment units, levels of respiratory distress (as indicated by respiratory rate), resting oxygen saturation levels, and the ratios of arterial blood oxygen partial pressures to oxygen concentration (26), (27), (18).

Furthermore, it is important to mention that there is a lack of research on the dynamic changes in routine blood parameters, particularly in patients who experienced fatal outcomes. Only a few studies have looked at the Biochemical and haematological characteristics markers linked with mortality and survival following COVID-19 in Libya. Consequently, in this study we have conducted a concise comparative analysis involving 200 patients, in which we have examined differences between those who have successfully overcome the disease and those who have unfortunately died.

## MATERIALS AND METHODS:

### Study Design and Patients:

A retrospective observational study was conducted at Mansoura chest diseases hospital in the District of Jebel el-Akhdar, a region in north-eastern Libya. Data was collected from patients who were hospitalised with confirmed COVID-19 from August to December 2021. The confirmation of SARS-CoV-2 infection was done through an RT-PCR (Reverse Transcriptase Polymerase Chain Reaction) assay using either nasopharyngeal or oropharyngeal swabs. For this particular study, a total of 200 patients were recruited and then divided into two groups based on their outcomes: survivors (n = 178) and non-survivors (n = 72).

### Data collection:

The patients' clinical and laboratory data were collected from their medical records. The laboratory data, which comprised various laboratory parameters, such as haemoglobin, platelet counts, red blood cell counts, total leukocyte, lymphocyte counts, serum urea, serum creatinine, serum electrolytes (sodium, chloride, and potassium), Procalcitonin (PCT), total bilirubin (TB) (μmol/L), alanine aminotransferase (ALT), aspartate aminotransferase (AST) and alkaline phosphatase (ALP). Additionally, Erythrocyte Sedimentation Rate (ESR), C-reactive protein (CPR), lactate dehydrogenase (LDH), ferritin, serum D-dimer, and Oxygen saturation was monitored. The data obtained included demographic features such as age and gender at presentation as well as the outcomes. Supplementary Table 1, 2 and 3 provides a comprehensive list of the parameters that were evaluated

### Statistical Analysis:

Categorical variables were recorded as percentages. Continuous variables were reported by indicating their means and standard deviations (SD). Prior to conducting the statistical analysis, the data underwent a normality check. In the case of normally distributed continuous variables, the unpaired t-test was employed for comparison, while the Mann-Whitney U test was utilized for variables that did not exhibit normal distribution. The chi-square test was employed to analyze categorical variables. The level of significance was considered to be $p<0.05$. All statistical analyses

were performed using SPSS version 21.0 (IBM Corp., Armonk, NY, USA).

## RESULTS:

Patient demographics, clinical characteristics, and classification into survivors and non-survivors. This research included 200 patients with laboratory-confirmed COVID-19 who were admitted to the hospital between August and December 2020. 131 (65.5%) were males and 69 (34.5%) were females. Among the 200 participants, a total of 72 individuals deceased during the period of study. The subgroup of patients who did not survive is composed of 51 males, which accounts for 25.5% of the total, and 21 females, which represents 10.5% of the cohort. Conversely, the group of patients who managed to survive consists of 80 males, constituting 40% of the participants, and 48 females, amounting to 24% of the sample.Males were found to have a higher rate of infection than females. The average age of the study participants was found to be 62 years, whereas the average age for non surviver patients was 68years. The patients who did not survive were generally older adults in comparison to the survivors, with significant statistically meaningful variation in age observed between the two groups. Furthermore, Table 1 illustrates a comparison between individuals who survived and those who did not survive in terms of vital signs, including oxygen saturation, systolic and diastolic blood pressure, heart rate, temperature. The non-survivor group displayed a significantly lower level of oxygen saturation. Conversely, there was no significant distinction observed in heart rate, systolic blood pressure, and temperature between the survivor and non-survivor groups, as shown in Table 1.

**Table 1. Demographic and Comorbidities in COVID-19 patients and their association with disease severity**

| Total (*N*= 200) | | Non-Survivors N =72 | Survivors N= 128 | *P value* |
|---|---|---|---|---|
| Gender N (%) | Male 131(65.5%) | 51 (25.5%) | 80 (40%) | |
| | Female 69 (34.5%) | 21 (10.5%) | 48 (24%) | |
| Age | | 68.19±12.41 | 57.97±15.93 | 0.0030* |
| Body temperature | | 37.026±0.066 | 37.204±0.081 | 0.091 |
| SpO2 (%) | | 80.9±1.5 | 86.51±0.85 | 0.001* |
| Heart rate (/min) | | 89.7±2.6 | 84.57±1.0 | 0.068 |
| Systolic pressure [mm hg] | | 131.4±2.6 | 126.9±1.7 | 0.149 |
| Diastolic pressure[mm hg] | | 73.3±1.3 | 75.1±0.94 | 0.247 |

**The P * value less than 0.05 indicates statistical significance. All values are expressed as either N (%) or mean (SD). SD represents the standard deviation. Additionally, SpO2 is used to refer to oxygen saturation**

## Haematological Markers:

The routine hematologic parameters, including white blood cells (WBC), lymphocytes, hemoglobin (Hb), red blood cells (RBC), and platelet count (PLT), were investigated among the comparative groups. A significant increase (p value = <0.001) in WBC was observed in the deceased group compared to the surviving individuals. Likewise, a significant difference (p value = <0.001) in white blood cells was found between the deceased patients ($11.51 \times 10^9$/L) and the surviving patients ($9.60\times 10^9$/L). Also, Laboratory examinations revealed a decrease in lymphocyte count among all patients affected by COVID-19, exhibiting a statistically noteworthy distinction between the individuals who survived ($3.87\times 10^9$/L) and those who did not survive ($1.55\times 10^9$/L ) ($p > 0.05$)., as showed in Table 2. Moreover, the counts of RBC and PLT and the Hb value did not differ significantly between the survivors and the cohort who died . A comparison of the values of hematological parameters for the groups that survived and did not survive can be observed in Table 2.

**Table 2: This table showed the hematology Laboratory findings of patients infected with COVID-19 on admission to the centre in the cases that have been studied, the mean, (SD Mean) and p-value**

| Hematological indicators | Normal range | Survivor patients | Deceased patients | P-value |
|---|---|---|---|---|
| **Haemoglobin mg/dl** | **11.5–15 mg/dl** | **12.55±1.60** | **11.99±1.74** | **0.076** |
| **White blood cell $\times 10^9$/L** | **3.5–4.5** | **9.60±3.91** | **11.51 ±5.37** | **0.0253*** |
| **Lymphocyte ($\times 10^9$/L)** | **1.1-3.2** | **3.87±5.64** | **1.55 ±1.50** | **0.0121*** |
| **Red Blood Cell($\times 10^{12}$ /L)** | **4.5-5.15** | **4.73±2.60** | **4.57±1.07** | **0.571** |
| **Platelets ($\times 10^9$/L)** | **125–350** | **277.46±105.81** | **250.63±97.97** | **0.1716** |

**Biochemical Profile, Coagulation Profile and Inflammatory Indicators**:

The biochemical data were categorized into subgroups such as and inflammatory markers, Renal function profile, Liver function test, Coagulation marker, Acid/base analysis, and others. Mean ± SD values were presented in Table 3

The comparison between the biochemical parameters of the groups that survived and those that did not survive was conducted (Table 3). Examination of biochemical data suggests that there were significant differences in LDH, ferritin, ALT, and AST levels between the group of survivors and the group of non-survivors. The non-survivor group exhibited a distinct increase in the values of these parameters (p=0.001). Similar differences were observed in the profiles of D-dimer and Procalcitonin (PCT) (μg/L) between the survivor group and the non-survivor group. Conversely, no significant alterations were observed in urea, creatinine and Total bilirubin (TB) (μmol/Las demonstrated in Table 3. Regarding the inflammatory parameters, significant changes were observed, as indicated by a significantly substantial impact on the erythrocyte sedimentation rate (ESR) (62.13±32.71 versus 95.57±40.34 mm/h) and the C-reactive protein (CRP) (71.5±62.5 versus 132±78 mg/L) ($p > 0.001$).

Moreover, The analysis of the biochemical composition revealed that the group experiencing mortality demonstrated a significantly higher concentration of serum potassium ($P < 0.001$) compared to the group of individuals who survived. Nonetheless, there were no significant differences in chloride levels between the two groups mentioned above (all $P > 0.05$). (Table 3).

**Table 3. Comparison of Laboratory Data (Biochemical Profile, Coagulation Profile and Inflammatory Indicators) in Relation to the Outcome.**

| Parameter | Reference range | Survived Mean±SD | Died Mean±SD | P-value |
|---|---|---|---|---|
| Liver Functionality; | | | | |
| Total bilirubin (TB) (μmol/L) | **3.5-20.5** | **0.62±0.31** | **0.911±0.509** | **0.604** |
| Alanine aminotransferase (ALT) , U/L | **7–45** | **37.58±18.14** | **70.37±73.59** | **0.0103*** |
| Aspartate aminotransferase (AST) , U/L | **13–35** | **42.28±19.29** | **71.30±57.45** | **0.0063**** |
| Renal profile; | | | | |
| **Serum creatinine (mg/dL** | **0.74-1.35** | **1.17±0.58** | **1.5±1.1** | **0.1310** |
| **Blood Urea Nitrogen (mg/dL)** | **3.1-8.8** | **49.1±29.7** | **64.6±53** | **0.1005** |
| Serum sodium ( Na+), mmol/L | **135–145** | **135.720±6.47** | **141.19±11.83** | **0.0079**** |
| **Serum potassium ($K^+$ ), mmol/ L** | **3.5–4.5** | **4.06±0.66** | **5.00±1.06** | **0.0001***** |
| Serum chloride (Cl-), mmol/**L** | **95–105** | **94.86±8.32** | **93.92±12.87** | **0.6446** |
| Inflammatory Profile; | | | | |

| | | | | |
|---|---|---|---|---|
| Erythrocyte Sedimentation Rate (ESR) | **0 to 20 mm/h** | **62.13±32.71** | **95.57±40.34** | **0.0001*** ** |
| S ferrtiin ng/ml | **24 to 307** | **157±166.8** | **866.9±521** | **0.0001*** ** |
| Procalcitonin (PCT) (µg/L) | **<0.1** | **( 0.29 ±0.49)** | **( 0.49±0.70)** | **0.0490*** |
| C-reactive protein (CRP) (mg/L) | **0.068-8.2** | **71.5±62.5** | **132±78** | **0.0008*** ** |
| Lactate dehydrogenase (LDH) U/L | **109–245** | **325.26± 113.57** | **535.37±346.45** | **0.0091*** ** |
| **Coagulation Profile;** | | | | |
| D-dimer mg/ml | **0-0.55** | **1 ± 0.97** | **1.71±1.1** | **0.0008*** ** |

**DISCUSSION**:

With the current global spread of COVID-19 cases and the continuous emergence of various theories about its impact on the human body, our knowledge of several aspects of COVID-19 remains limited. However, there is still much to determine regarding the consequences of COVID-9 on various biochemical and haematological profiles in individuals who have either died or survived as result of the virus infection.

Our study presents comprehensive information regarding the demographic characteristics, biochemical and hematological parameters of patients who were admitted to Mansoura chest diseases hospital, located in the northeastern region of Libya, during an outbreak. Among the 200 individuals diagnosed with COVID-19, 131 (65.5%) were male and 69 (34.5%) were female. This distribution supports the previous data, where males accounted for 69.3% and females accounted for (30.7%) (29). This finding could potentially be connected to an enhanced production of IgG antibodies in females during the early phase of the COVID-19 infection. This phenomena might lead to a more favourable outcome for women (30). Furthermore, previous studies have indicated that males exhibit greater susceptibility to COVID-19 than females, possibly due to the lower expression of angiotensin-converting enzyme-2 receptors for coronavirus in females. In addation, Lifestyle factors, including increased levels of smoking and drinking among males (30).

The COVID-19 virus appears to have a higher mortality rate among males compared to females, as demonstrated in this particular study. Specifically, 51 males (25.5%) have succumbed to the virus in contrast to 21 females (10.5%). This phenomenon can also be observed in the two demographic groups most affected by the pandemic, namely Caucasians. In Italy, the majority of COVID-19 fatalities occurred among males, whereas in Spain, male deaths were nearly twice as high as female deaths (31, 32). Similar findings have been reported in other studies, where initial Chinese data revealed a gender disparity in mortality rates, with a higher proportion of male deaths compared to female deaths. Specifically, 41.9% of admitted patients were women, while the majority of deaths occurred among men (4, 33).

This investigation demonstrated that the average age of the individuals who did not survive due to COVID-19 was significantly higher than the cohort that did survive. These results are consistent with previous studies conducted on individuals with COVID-19 (34, 35). The elderly population demonstrates a higher prevalence of comorbidities, restricted organ function, decreased lung capacity, compromised immune system, biological aging, and more severe complications. These factors have been commonly identified in earlier research on the elderly population with COVID-19, (36, 37) thus prompting clinicians to administer more attentive care to this high-risk group.

Reduced oxygen saturation upon admission was identified as an independent risk factor for mortality in individuals infected with COVID-19. A retrospective examination established a correlation between hypoxemia (SpO2 <90%) and fatality in patients with COVID-19 (20). We identified a noteworthy difference in SpO2 levels between patients who survived and patients who did not survive. Another study also indicated that reduced levels of blood oxygen saturation were observed in individuals who did not survive (38). Our findings are consistent with the above-mentioned investigation **Table 1.**

Regarding the hematologic parameters in this study, noteworthy alterations in the haematological parameters were observed in the COVID-19 patients who survived as opposed to those who did not survive. The current study's findings explained that viral infection has the potential to induce diverse haematological changes. The comparison of haematological findings reveals that the death cohort exhibited an elevated number of white blood cells. This discovery supports the results of a previous study conducted by (39, 40), which hypothesized that an escalated WBC level could exacerbate the infection's prognosis and heighten the risk of unfavourable outcomes. However, the WBC count did not exhibit significant changes in other studies (40).

Furthermore, the death cohort demonstrated a lower count of RBCs, lymphocytes and PLTs compared to the survivors' cases. The decrease in red blood cell count in our study can be attributed to the impact of inflammation. Inflammatory processes may influence the formation of red blood cells through various mechanisms, ultimately resulting in anaemia caused by inflammation (41). We observed a significant reduction in lymphocyte count in the group of non-survivors, when compared to the survivor groups, table 2 a finding that agree with previous investigations (42, 43). There are various mechanisms mentioned in the literature that may act simultaneously to induce lymphopenia in individuals affected by COVID-19. For instance, lymphopenia could be attributed to either the direct assault on the lymphocytes, leading to their destruction, or the sequestration of lymphocytes in the lung. Additionally, it could result from the suppression of hematopoietic stem cells or other indirect pathways, such as the cytokine storm, which triggers the apoptosis of lymphocytes (39). Based on our findings, the quantification of lymphocytes can serve as a rapid diagnostic tool to identify COVID-19 patients with severe manifestations.

In relation to inflammatory, it has been observed that non survivor patients show a significant increase in ferritin levels. This increase in ferritin concentration is closely associated with the severity of COVID-19 infection and is indicative of an increased risk of mortality. Furthermore, it is crucial to note that ferritin serves as a marker for both inflammatory processes and dysregulation of immune function (44, 45).

The C-reactive protein (CRP) functions as an inflammatory biomarker that is widely used in clinical investigations. Elevated levels of CRP can be regarded as a sign of inflammation resulting from various infections (46). A previous study elucidated that deceased COVID patients exhibited higher levels of CRP than infected patients who were still alive (11). In the current study, it was discovered that higher levels of CRP among patients who succumbed to the illness, compared to those who survived following their COVID-19 diagnosis. Zhang et al. (47) posited that CRP stands as a marker for cytokine storm development in COVID-19 patients, and it is closely associated with disease mortality. Within the confines of this investigation, it is plausible to assert that escalated levels of CRP can be attributed to increased acute respiratory distress and the onset of a cytokine storm, thereby serving as an underlying cause for mortality within the deceased group Table 3.

As seen in Table 3, In numerous investigations, it has been posited that the levels of serum AST, ALT, and LDH, which serve as liver function tests, exhibited higher values in individuals with severe COVID-19 as opposed to those with mild COVID-19 (1, 48). Onur et al.(49) Demonstrated that the activities of AST, ALT, and LDH were elevated in deceased COVID-19 patients compared to the living patients. Guan et al.(4) Observed that the levels of ALT and AST were higher in severe patients than in non-serious patients. Within this manuscript, it was determined that the levels of ALT, AST, and LDH were notably higher in the group of individuals who did not survive in comparison to the group of individuals who did survive. These findings indicate that severe liver dysfunction may have manifested in those who succumbed to COVID-19. Various investigations have been carried out concerning the magnitude of severity of COVID-19. In a specific investigation, it was recorded that the levels of ESR, D-dimer, and procalcitonin showed a significant increase in cases classified as severe(1, 48). In a separate study, it was determined that procalcitonin levels exhibited a substantial increase in COVID-19 patients classified as non-surviving and severe. In another investigation, it was explicitly stated that changes in laboratory parameters, such as an increased D-dimer, were significant predictors of admission to the intensive care unit (50). Within the present study, the measurements of D-dimer, ESR, and procalcitonin were found to be higher in individuals who succumbed to COVID-19 compared to those who survived (Table 3). Furthermore, the procalcitonin levels in deceased patients were notably elevated in comparison to the survivors, thereby serving as a distinct prognostic indicator of the disease (51).

in a conducted study, it was determined that the serum levels of bilirubin, urea, and creatinine, which serve as renal function tests, were elevated in individuals afflicted with severe cases of COVID-19 and subsequently decreased in no survivor patients (28). Similarly, another study revealed that the bilirubin level was higher in severe instances of COVID-19 compared to less severe cases (4). In this particular investigation, the changes in the levels of bilirubin, urea, and creatinine were more prominent in the patients who did not survive, although not statistically significant. These findings suggest that the occurrence of kidney dysfunction may be attributed to the extensive involvement of the kidneys in those individuals who succumbed to COVID-19.

Studies have demonstrated the presence of electrolyte abnormalities in individuals infected with COVID-19 and have proposed the monitoring of patients' electrolyte levels (52). An investigation conducted by Liu S et al. involving 136 confirmed cases of COVID-19 revealed a noteworthy increase in mortality rates within 30 days among patients with K levels equal to or exceeding 5.0 mmol/L (53). Our findings provide strong support for the notion that electrolyte impairments are considerably more prevalent in severe cases of COVID-19. Although our analysis did not account for sodium (Na) and serum chloride impairments as predictors of mortality, our results align with the study conducted by Liu S et al., as they indicate a significant association between elevated K

levels and mortality in COVID-19 patients. Consequently, we recommend the accurate monitoring of K levels in COVID-19 patients to ensure they remain within the normal range.

## CONCLUSION:

This study has put forth the proposition of employing serial and admittance laboratory biomarkers for the purpose of assessing the outcome of COVID-19. A fresh and innovative method has been utilized to identify prognostic indicators through serial laboratory examinations. The careful observation and monitoring of laboratory findings of COVID-19 patients can prove instrumental in effectively managing the patients. On the basis of this extensive study, healthcare professionals can rely on the serial examination of PLT, WBC, CRP, LDH, D. Dimer, ferritin, AST, and ALT to guide their monitoring of patients. Furthermore, any disruption in the admittance levels of Complete blood count, CRP, Cr, potassium, PT, bilirubin, LDH, and D-dimer escalates the risk of mortality. Nevertheless, it is imperative that future studies be conducted to corroborate the findings of this retrospective study.

## REFERENCES:


1. Amgalan A, Othman M. Hemostatic laboratory derangements in COVID-19 with a focus on platelet count. Platelets. 2020;31(6):740-5.
2. World Health Organization. WHO Coronavirus (COVID-19) Dashboard. Geneva: World Health Organization; 2023.
3. Mohapatra RK, Kandi V, Sarangi AK, Verma S, Tuli HS, Chakraborty S, et al. The recently emerged BA. 4 and BA. 5 lineages of Omicron and their global health concerns amid the ongoing wave of COVID-19 pandemic–Correspondence. International Journal of Surgery (London, England). 2022;103:106698.
4. Guan W-j, Ni Z-y, Hu Y, Liang W-h, Ou C-q, He J-x, et al. Clinical characteristics of coronavirus disease 2019 in China. New England journal of medicine. 2020;382(18):1708-20.
5. Wu C, Chen X, Cai Y, Xia J, Zhou X, Xu S, et al. Risk Factors Associated With Acute Respiratory Distress Syndrome and Death in Patients With Coronavirus Disease 2019 Pneumonia in Wuhan, China. JAMA Intern Med. 2020;180(7):934-43.
6. Huyut MT, Ilkbahar F. The effectiveness of blood routine parameters and some biomarkers as a potential diagnostic tool in the diagnosis and prognosis of Covid-19 disease. International Immunopharmacology. 2021;98:107838.
7. 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.
8. Baj J, Karakuła-Juchnowicz H, Teresiński G, Buszewicz G, Ciesielka M, Sitarz R, et al. COVID-19: specific and non-specific clinical manifestations and symptoms: the current state of knowledge. Journal of clinical medicine. 2020;9(6):1753.
9. Khinda J, Janjua NZ, Cheng S, van den Heuvel ER, Bhatti P, Darvishian M. Association between markers of immune response at hospital admission and COVID-19 disease severity and mortality: a meta-analysis and meta-regression. Journal of medical virology. 2021;93(2):1078-98.
10. Huyut MT, Huyut Z. Forecasting of Oxidant/Antioxidant levels of COVID-19 patients by using Expert models with biomarkers used in the Diagnosis/Prognosis of COVID-19. International Immunopharmacology. 2021;100:108127.
11. Ardestani A, Azizi Z. Targeting glucose metabolism for treatment of COVID-19. Signal Transduction and Targeted Therapy. 2021;6(1):112.
12. Kratzer B, Trapin D, Ettel P, Körmöczi U, Rottal A, Tuppy F, et al. Immunological imprint of COVID-19 on human peripheral blood leukocyte populations. Allergy. 2021;76(3):751-65.
13. Elemam NM, Talaat IM, Bayoumi FA, Zein D, Georgy R, Altamimi A, et al. Peripheral blood cell anomalies in COVID-19 patients in the United Arab Emirates: A single-centered study. Frontiers in Medicine. 2022;9:1072427.
14. Liu Y, Yang Y, Zhang C, Huang F, Wang F, Yuan J, et al. Clinical and biochemical indexes from 2019-nCoV infected patients linked to viral loads and lung injury. Science China Life Sciences. 2020;63:364-74.
15. Azoulay E, Fartoukh M, Darmon M, Geri G, Voiriot G, Dupont T, et al. Increased mortality in patients with severe SARS-CoV-2 infection admitted within seven days of disease onset. Intensive care medicine. 2020;46:1714-22.
16. Hägglöf E, Bell M, Zettersten E, Engerström L, Larsson E. Long-term survival after intensive care for COVID-19: a nationwide cohort study of more than 8000 patients. Annals of Intensive Care. 2023;13(1):1-10.
17. Zheng Y, Zhang Y, Chi H, Chen S, Peng M, Luo L, et al. The hemocyte counts as a potential biomarker for predicting disease

progression in COVID-19: a retrospective study. Clinical Chemistry and Laboratory Medicine (CCLM). 2020;58(7):1106-15.
18. Jiang S-Q. The association between severe COVID-19 and low platelet count: evidence from 31 observational studies involving 7613 participants. 2020.
19. Carter C, Notter J. COVID-19 disease: a critical care perspective. Clinics in Integrated Care. 2020;1:100003.
20. Chen R, Sang L, Jiang M, Yang Z, Jia N, Fu W, et al. Longitudinal hematologic and immunologic variations associated with the progression of COVID-19 patients in China. Journal of Allergy and Clinical Immunology. 2020;146(1):89-100.
21. Mertoglu C, Huyut MT, Olmez H, Tosun M, Kantarci M, Coban TA. COVID-19 is more dangerous for older people and its severity is increasing: a case-control study. Medical gas research. 2022;12(2):51.
22. Chen T, Wu DI, Chen H, Yan W, Yang D, Chen G, et al. Clinical characteristics of 113 deceased patients with coronavirus disease 2019: retrospective study. bmj. 2020;368.
23. Doğanay F, Elkonca F, Seyhan AU, Yılmaz E, Batırel A, Ak R. Shock index as a predictor of mortality among the Covid-19 patients. The American journal of emergency medicine. 2021;40:106-9.
24. Huyut MT, Üstündağ H. Prediction of diagnosis and prognosis of COVID-19 disease by blood gas parameters using decision trees machine learning model: a retrospective observational study. Medical gas research. 2022;12(2):60.
25. Li X, Wang L, Yan S, Yang F, Xiang L, Zhu J, et al. Clinical characteristics of 25 death cases with COVID-19: A retrospective review of medical records in a single medical center, Wuhan, China. International Journal of Infectious Diseases. 2020;94:128-32.
26. Cao B, Jing X, Liu Y, Wen R, Wang C. Comparison of laboratory parameters in mild vs. severe cases and died vs. survived patients with COVID-19: systematic review and meta-analysis. Journal of Thoracic Disease. 2022;14(5):1478.
27. Al Harbi M, Al Kaabi N, Al Nuaimi A, Abdalla J, Khan T, Gasmelseed H, et al. Clinical and laboratory characteristics of patients hospitalised with COVID-19: clinical outcomes in Abu Dhabi, United Arab Emirates. BMC Infectious Diseases. 2022;22(1):1-9.
28. Mertoglu C, Huyut MT, Arslan Y, Ceylan Y, Coban TA. How do routine laboratory tests change in coronavirus disease 2019? Scandinavian Journal of Clinical and Laboratory Investigation. 2021;81(1):24-33.
29. Usul E, Şan İ, Bekgöz B, Şahin A. Role of hematological parameters in COVID-19 patients in the emergency room. Biomarkers in Medicine. 2020;14(13):1207-15.
30. Zeng F, Dai C, Cai P, Wang J, Xu L, Li J, et al. A comparison study of SARS-CoV-2 IgG antibody between male and female COVID-19 patients: a possible reason underlying different outcome between sex. Journal of medical virology. 2020;92(10):2050-4.
31. Onder G, Rezza G, Brusaferro S. Case-fatality rate and characteristics of patients dying in relation to COVID-19 in Italy. JAMA. Advance online publication. 2020.
32. Capuano A, Rossi F, Paolisso G. Covid-19 kills more men than women: an overview of possible reasons. Frontiers in cardiovascular medicine. 2020;7:131.
33. Fu L, Wang B, Yuan T, Chen X, Ao Y, Fitzpatrick T, et al. Clinical characteristics of coronavirus disease 2019 (COVID-19) in China: a systematic review and meta-analysis. Journal of Infection. 2020;80(6):656-65.
34. Kashtanova DA, Erema VV, Gusakova MS, Sutulova ER, Yakovchik AY, Ivanov MV, et al. Mortality and survival in nonagenarians during the COVID-19 pandemic: Unstable equilibrium of aging. Frontiers in medicine. 2023;10:1132476.
35. Qin C, Zhou L, Hu Z, Zhang S, Yang S, Tao Y, et al. Dysregulation of immune response in patients with coronavirus 2019 (COVID-19) in Wuhan, China. Clinical infectious diseases. 2020;71(15):762-8.
36. Shahid Z, Kalayanamitra R, McClafferty B, Kepko D, Ramgobin D, Patel R, et al. COVID-19 and older adults: what we know. Journal of the American Geriatrics Society. 2020;68(5):926-9.
37. Kang S-J, Jung SI. Age-related morbidity and mortality among patients with COVID-19. Infection & chemotherapy. 2020;52(2):154.
38. Shi Q, Zhao K-L, Yu J, Feng J-R, Zhao K-P, Zhang X-Y, et al. Clinical characteristics of 101 non-surviving hospitalized patients with COVID-19—A single center, retrospective study. MedRxiv. 2020:2020-03.
39. 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.

Clinical Chemistry and Laboratory Medicine (CCLM). 2020;58(7):1021-8.
40. Zhou B, She J, Wang Y, Ma X. Utility of ferritin, procalcitonin, and C-reactive protein in severe patients with 2019 novel coronavirus disease. 2020.
41. Ali ET, Jabbar AS, Mohammed AN. A comparative study of interleukin 6, inflammatory markers, ferritin, and hematological profile in rheumatoid arthritis patients with anemia of chronic disease and iron deficiency anemia. Anemia. 2019;2019.
42. Bobcakova A, Petriskova J, Vysehradsky R, Kocan I, Kapustova L, Barnova M, et al. Immune profile in patients with COVID-19: lymphocytes exhaustion markers in relationship to clinical outcome. Frontiers in cellular and infection microbiology. 2021;11:646688.
43. 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):507-13.
44. Kell DB, Pretorius E. Serum ferritin is an important inflammatory disease marker, as it is mainly a leakage product from damaged cells. Metallomics. 2014;6(4):748-73.
45. Kernan KF, Carcillo JA. Hyperferritinemia and inflammation. International immunology. 2017;29(9):401-9.
46. Mo P, Xing Y, Xiao YU, Deng L, Zhao Q, Wang H, et al. Clinical characteristics of refractory COVID-19 pneumonia in Wuhan, China. Clin infect dis. 2020;10.
47. Zhang Jj, Cao Yy, Tan G, Dong X, Wang Bc, Lin J, et al. Clinical, radiological, and laboratory characteristics and risk factors for severity and mortality of 289 hospitalized COVID-19 patients. Allergy. 2021;76(2):533-50.
48. Erol AT, Aşar S, Sabaz MS, Bilgin BÖ, Çukurova Z. Risk Factors for 28-day Mortality Among COVID-19 Patients in an Intensive Care Unit of a Tertiary Care Center in Istanbul. Medical Journal of Bakirkoy. 2021;17(1).
49. Tural Onur S, Altın S, Sokucu SN, Fikri BI, Barça T, Bolat E, et al. Could ferritin level be an indicator of COVID-19 disease mortality? Journal of medical virology. 2021;93(3):1672-7.
50. Tang N, Li D, Wang X, Sun Z. Abnormal coagulation parameters are associated with poor prognosis in patients with novel coronavirus pneumonia. Journal of thrombosis and haemostasis. 2020;18(4):844-7.
51. Sun S, Cai X, Wang H, He G, Lin Y, Lu B, et al. Abnormalities of peripheral blood system in patients with COVID-19 in Wenzhou, China. Clinica chimica acta. 2020;507:174-80.
52. Sarvazad H, Cahngaripour SH, Roozbahani NE, Izadi B. Evaluation of electrolyte status of sodium, potassium and magnesium, and fasting blood sugar at the initial admission of individuals with COVID-19 without underlying disease in Golestan Hospital, Kermanshah. New microbes and new infections. 2020;38:100807.
53. Liu S, Zhang L, Weng H, Yang F, Jin H, Fan F, et al. Association between average plasma potassium levels and 30-day mortality during hospitalization in patients with COVID-19 in Wuhan, China. International journal of medical sciences. 2021;18(3):736.