

## Role of Inflammatory Markers, Immune Response, and Recovery Rate in COVID-19 Patients

Saira Afzal <sup>a</sup>, Tariq Iqbal <sup>b</sup>

<sup>a</sup> Department of Immunology, Dow University of Health Sciences, Karachi, Pakistan

<sup>b</sup> Department of Immunology, Dow University of Health Sciences, Karachi, Pakistan [tariqiqbal8@gmail.com](mailto:tariqiqbal8@gmail.com)

Correspondence: Tariq Iqbal ([tariqiqbal8@gmail.com](mailto:tariqiqbal8@gmail.com))

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### ABSTRACT

From mild symptoms to Severe Acute Respiratory Syndrome and multi-organ dysfunction induced by Coronavirus-2, COVID-19's clinical outcomes vary. An important aspect in illness severity and outcome is host immunological response, especially the ratio of protective immunity to excessive inflammation. In severe situations, abnormal immune activation can lead to hyperinflammation and elevated levels of biomarkers such as IL-6, CRP, TNF- $\alpha$ , ferritin, and D-dimer. These parameters are linked to illness severity, ICU admission, and death risk. Blood markers including lymphopenia and the neutrophil-lymphocyte ratio (NLR) also predict immunological failure. Balanced immune activation speeds virus clearance and recovery, but an excessive inflammatory response causes acute respiratory distress symptoms (ARDS) and prolonged hospitalisation. Inflammatory markers, immunological dysregulation, and their clinical relevance in COVID-19 disease development and recovery are highlighted in this study.

**Keywords:** COVID-19, Inflammatory markers, Immune response, IL-6, CRP, Cytokine storm, Recovery rate, Biomarkers, SARS-CoV-2, Disease severity

### INTRODUCTION

SARS-CoV-2, or COVID-19, emerged in late 2019 and became a global public health hazard. Since initial breakout, the disease has evolved to cause asymptomatic coinfection, acute pneumonia, ARDS, multi-organ failure, and death. Host immune response and inflammatory processes are crucial to disease outcome after infection (Wang et al., 2020). Covid-19 infection involves two immune system functions. It's important to heal from the virus, but an overactive immune system can cause inflammation and tissue damage. High levels of pro-inflammatory cytokines IL-6, IL-1 $\beta$ , and TNF- $\alpha$  cause a cytokine storm in severe SARS-CoV-2 infections. Dysregulated immune response is connected to respiratory failure and death (Huang et al., 2020; Del Valle, 2020).

COVID-19 biology will involve IL-6 as a key inflammatory marker. Serum IL-6 levels significantly correlate with illness severity, ICU admission, and poor prognosis. IL-6 also increases liver expression of acute-phase proteins such C-reactive protein, a clinical indication of systemic inflammation. High CRP shows active inflammation and is associated

with poor COVID-19 clinical outcomes (Chen et al., 2020).

Haematological markers, cytokines, and acute phase proteins can also indicate immune system health.

Low lymphocyte count (lymphopenia) is a common laboratory abnormality in severe COVID-19 and indicates a lack of adaptive immunity. Neutrophilia indicates innate immune activity. Thus, the neutrophil-to-lymphocyte ratio (NLR) is now widely used to predict illness severity and mortality (Lagunas-Rangel, 2020).

In severe COVID-19, inflammatory (ferritin) and coagulation (D-dimer) indicators are high, another important blood marker. Ferritin levels indicate hyperinflammation, whereas D-dimer levels indicate coagulation problems and thrombotic risk. All these markers provide a systemic view of inflammation and disease progression (Gómez-Pastora et al., 2020).

COVID-19 immunological response is very variable depending on illness severity and host immune state. In mild illness, the immune system fights the pathogen without inflammation. In severe cases,

dysregulation causes cytokine overproduction, endothelial damage, and widespread inflammation. Pro-inflammatory and anti-inflammatory imbalances determine disease severity and resolution (Mehta et al., 2020).

Inflammatory response and immune repair affect COVID-19 patients' adaptive function and recovery rates. Patients with controlled cytokine levels and appropriate immune system balance heal rapidly, whereas those with rising levels have protracted hospital stays and sluggish recoveries. Studies show that greater levels of IL-6, CRP, and ferritin are linked to prolonged infection recovery and higher consequences (Zhou et al., 2020).

Recent investigations have linked inflammatory indicators to both the acute severity of COVID-19 and its long-term effects (post-acute sequelae—"long COVID"). Immune activation and persistent inflammation cause fatigue, dyspnea, and cognitive impairment in COVID-19 survivors (Nalbandian et al., 2021).

Combining various inflammatory indicators improves disease progression prediction. ELISION (IL-6, NLR, CRP, and D-dimer) improves ICU admission and death prognosis (Del Valle et al., 2020). The multi-marker approach emphasises systemic inflammation in COVID-19 aetiology.

Overall, this link is driven by inflammatory indicators, immunological response, and COVID-19 recovery rate. Dysregulated immune activation causes severe sickness, while well-balanced immune responses and faster recovery eliminate the infection. Inflammatory biomarkers are useful in diagnosis, clinical outcome prediction, therapeutic intervention design, and patient care.

#### LITERATURE REVIEW

##### COVID-19 Progression Inflammatory Markers

Recently, many study has shown that inflammatory indicators affect COVID-19 severity and outcome. SARS-CoV-2 infection produces systemic inflammation and raises blood indicators such IL-6, CRP, ferritin, and D-dimer. The prognostic significance of these biomarkers to identify high-risk individuals has been extensively studied (Zhang et al., 2021).

IL-6 is a key cytokine in severe COVID-19. Research found that elevated IL-6 levels are linked to respiratory failure, ICU admission, and mortality (Del Valle et al., 2020). CRP, another acute-phase reactant, positively and linearly correlates with disease severity and lung damage. CRP levels rise steadily during chronic infection, making it a good disease marker (Chen et al., 2020).

Ferritin also indicates hyperinflammatory disorders in COVID-19 individuals. High serum Ferritin levels indicate macrophage activation and cytokine storm, which increases death risk (Gómez-Pastora et al., 2020). Platelet counts were higher in severe patients, indicating aberrant coagulation and greater thrombotic consequences (Tang et al., 2020).

##### Immunity and Cytokine Storm

COVID-19 patients' immunity response protects them under the correct regulation but patientizes them under the wrong regulation. A well-coordinated immune response clears the virus, but an overactive immune reaction creates a cytokine storm, which causes organ damage and death in severe situations (Mehta et al., 2020).

Huang et al. (2020) discovered elevated levels of pro-inflammatory cytokines in very ill COVID-19 patients, including IL-2, IL-6, IL-7, TNF- $\alpha$ , and interferon-gamma. This hyper-inflammatory syndrome can damage endothelial cells, increase vascular permeability, and induce acute inadequate breathing. Wang et al. (2021) noted that immunological dysregulation caused lymphocyte depletion and neutrophil activation, which advanced the illnesses.

Cellular immunity studies link COVID-19 severity to poor adaptive immunity. Lymphocytes are low in severe patients, indicating T cell-mediated immune system failure (Zheng et al., 2020). Uncontrolled viral replication and inflammation result from this immunological imbalance.

##### Immunological and Haematological Biomarkers

Neutrophil to lymphocyte ratio (NLR), white blood cell (WBC) fluctuations, and platelet count have been intensively studied as COVID-19 severity measures. Lagunas-Rangel (2020) discovered that greater NLRs increase mortality and ICU admission.

Liu et al. (2021) found that severe COVID-19 patients had greater neutrophil and lower lymphocyte counts than moderate individuals. This imbalance indicates higher innate immune system activation and decreased adaptive immune system.

CRP and ESR are also good inflammatory indicators. ESR rises with lung inflammation and illness progression (Ali et al., 2022). Procalcitonin (PCT) was also connected to COVID-19 patients with bacterial co-infection or significant systemic inflammation (Lippi & Plebani, 2020).

##### Disease severity and inflammatory response

Several studies show that COVID severity is proportional to inflammation. Cytokine dysregulation, endothelial dysfunction, and multi-organ involvement occur in severe cases (Del Valle et al., 2020).

In hospitalised patients, Zhou et al. (2020) demonstrated that IL-6, CRP, and ferritin levels independently predict mortality. Chen et al. (2021) observed greater inflammatory markers were linked to longer hospital stays and respiratory failure.

Vascular problems result from inflammation. COVID-19-associated coagulopathy is strongly linked to high D-dimer levels, which increase the risk of thrombosis and stroke (Tang et al., 2020). COVID-19's multi-systemic inflammation will be highlighted.

### **Speed of immunological reaction and recuperation**

Recovery rate in COVID-19 patients depends on immune regulation and inflammatory control. Balanced immune responses heal faster, while overreactive responses delay recovery and cause problems (Nalbandian et al., 2021). New research shows that early IL-6 and CRP decrease improves recovery. Reducing hospital stays and ICU days (Zhang et al., 2022).

Long-term COVID—characterized by neurological and respiratory impairments, cognitive impairment, and fatigue—has been linked to persistent inflammation. Sudre et al. (2021) found that acute inflammatory scores increase the probability of long-term illnesses.

Also important for recovery are Immune Memory and Antibody Response. Studies showed that greater T-cell counts improved recovery and reduced reinfection risk (Wang et al., 2021).

### **H2.5 – Multi-Marker Approach in Clinical Prognosis**

Recent studies focusing on using more than one biomarker instead of just a single inflammatory biomarker. The simultaneous measurement of CRP, ferritin, NLR and IL-6 increases the accuracy in the prediction of severity and mortality (Del Valle et al., 2020).

Studies using machine learning approaches have shown that combined biomarker models have shown a significant improvement in clinical decision-making and patient stratification (Yan et al. 2020). These models enable early detection of high-risk patients and timely treatment, with optimistic outcomes.

Furthermore, multi-marker inflammatory profiles are more and more being incorporated into predictive scoring systems used to evaluate admission risk in an ICU and the probability of mortality (Liu et al., 2021).

### **Long COVID effects on the immune system**

Recently, attention has been drawn to alterations in long-term immune system dysfunction in COVID-19 survivors. Nalbandian et al. (2021) noted that a large percentage of recovered COVID patients are having complications involving their immune systems months after the infection.

Chronic inflammatory status has been associated with chronic fatigue syndrome (CFS), cardiovascular and neurological symptoms. It indicates that, in addition to being an acute infectious illness, COVID-19 also impacts immunity in the long-term (Sykes et al., 2022).

Also, research shows that the process of viral clearance is associated with the immune exhaustion and T-cell dysfunctions, leading to the delayed recovery and lowered quality of life (QOL) (Zheng et al., 2020).

These findings from the existing literature are generally in favor of the critical role of inflammatory

markers and immune response in the stratification of the severity of COVID-19 and the outcome of the recovery process. There is strong evidence showing that elevated levels of cytokines are strongly correlated with severe disease and poor prognosis, including levels of IL-6, CRP and TNF- $\alpha$ . Predictive accuracy is further supported by hematological parameters, like NLR. Furthermore, aberrations in immune function is a key mechanism that contributes to disease progression and late stage recovery. Markers and combined markers have been increasingly highlighted in recent studies for enhanced clinical prediction and management of the COVID-19 patient.

### **METHODOLOGY**

#### **Theoretical Framework**

The study was founded on two theories: Kytokine Storm Theory and the Immunopathogenesis Model of Viral Infection. The Cytokine Storm Theory suggests that the worsening of the effects of COVID-19 are due to the excessive and unchecked immune reactions which results in an uncontrolled overproduction of inflammatory cytokines such as interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF- $\alpha$ ), and other pro-inflammatory mediators. This hyperinflammatory status leads to tissue damage, acute respiratory distress syndrome (ARDS) and multi-organ damage (Mehta et al., 2020).

Another bonus was the focus on the balance between innate and adaptive immune responses in the Immunopathogenesis Model. It offered the hypothesis that optimal immune response was important for the clearance of the virus and quick recovery, and failure to achieve optimal immune responses resulted in progression of the disease and in delayed recovery. Here, inflammatory markers that are quantifiable, including C-reactive protein (CRP), ferritin, and D-dimer were seen as reflecting immune system activation and disease severity.

Theories were the foundation of a conceptual framework for the study, as follows:

- Independent predictors were considered as the Inflammatory Markers (IL-6, CRP, Ferritin, D-dimer, NLR).
- Immune Response (lymphocyte count, neutrophil activity, cytokine regulation) was a mediating biological mechanism
- Duration of hospital stay, symptom resolution to clinical improvement was assumed as dependent outcome variables and is referred to as Recovery Rate

Higher inflammatory marker levels were assumed to have a negative impact on immune balance and thus on the number of healing patients in COVID-19 patients.

#### **Hypotheses Development**

Based on the theoretical foundation and literature review, the following hypotheses were developed:

H1: There was a significant positive relationship between inflammatory markers and COVID-19 disease severity.

H2: Elevated IL-6 levels were significantly associated with increased inflammatory response in COVID-19 patients.

H3: C-reactive protein (CRP) levels were significantly related to slower recovery rates in COVID-19 patients.

H4: There was a significant negative relationship between immune response efficiency and disease severity.

H5: Lymphopenia was significantly associated with prolonged recovery duration.

H6: Neutrophil-to-lymphocyte ratio (NLR) significantly predicted COVID-19 mortality and severity.

H7: Inflammatory markers significantly influenced recovery rate through immune response mediation.

H8: A combined inflammatory biomarker model significantly predicted recovery outcomes better than individual markers.

### Research Design

The type of the research in this study was quantitative correlational research. The design was chosen because it would enable relationships between inflammatory markers, immune response and recovery rate to be explored without changing factors.

The study was mainly conducted to explore the relationship and influence exerted between biological and clinical indicators of COVID-19.

### Population and Sample

The study population were confirmed COVID-19 cases who were hospitalized in tertiary care centers in the pandemic era. The study population was selected through a purposive sampling method as it involved only the laboratory confirmed COVID-19 cases with complete clinical and biochemical information.

Similar biomedical correlational studies were performed on samples of 250 patients or more, which was sufficient for the purpose of this study (Zhou et al., 2020).

### Data Collection Procedure

Secondary clinical data was obtained from hospital files, laboratory reports and published data sets of clinical information. The following data was collected:

- Interleukin-6 (IL-6, NLR), ferritin, D-dimer and CRP are inflammatory biomarkers.
- Coagulation Lab (PPT tests)
- Clinical outcomes including the recovery time, ICU admission and mortality status.

All the data was anonymized and kept confidential for ethical reasons.

### Measurement of Variables

- **Inflammatory Markers:** Measured using laboratory serum tests (pg/mL or mg/L depending on biomarker)

- **Immune Response:** Assessed through complete blood count (CBC) and cytokine profiles
- **Recovery Rate:** Measured as number of days from hospital admission to clinical recovery or discharge
- **Disease Severity:** Categorized as mild, moderate, and severe based on WHO COVID-19 clinical classification guidelines

### Data Analysis Techniques

Data collected were subjected to statistical analysis for the purpose of establishing the relationship between the variables. Techniques used:

- Patient characteristics and biomarkers within the patient population were summarized using descriptive statistics, including mean, standard deviation and frequency distribution.
- Correlation Analysis: Pearson correlation coefficient was used in investigating association between inflammatory markers with recovery rate.
- Regression Analysis: Multiple regression analysis was used to find the predictive role of inflammatory markers on recovery outcomes.
- Mediation Analysis: Immune response was explored as a mediator between inflammatory markers and recovery rate.
- Reliability of scale-based indicators has been used: Composite indices have been internally consistent by Cronbach's alpha.

All data were analyzed with 95% confidence level ( $p < 0.05$ ).

### Ethical Considerations

Seems as though that local (institutional) ethics approval was granted before data collection was carried out. The patient information was kept anonymous and their identification data was not provided in the data set. Medical research ethics principles were followed in the study, such as anonymity, confidentiality of data and responsibility in reporting.

The study was overall a quantitative correlational design based on the cytokine storm and immunopathogenesis theoretical framework. All inflammatory markers were used independently as predictors, whereas immune response was used as a mediating biological mechanism, and recovery rate was used as a dependent variable. To get overall picture of COVID-19's disease progression and recovery relationship, statistical analyses were conducted to examine the strength and significance of relationships among the variables.

## DATA ANALYSIS AND RESULTS

### Demographic Characteristics of the Patients

Demographic profile of COVID-19 patients gave a grounding concept on the distribution of the sample and was useful in understanding variability in the

clinical profile in different groups. Analysis showed that there was a cross-section of patients of different ages, clinical severities, and genders. The majority of patients were located in the middle age group, indicating a higher number of hospital admissions owing to COVID-19 complications for this group of patients. The gender distribution was slightly more

males than females which is similar to the previously reported epidemiological result of more severe patients among males. Furthermore, there was a high percentage of patients in moderate and severe CLAD groups, meaning that inflammatory marker analysis was mainly pertinent to clinically relevant cases.

**Table 1: Demographic Profile of COVID-19 Patients (N = 250)**

Variable	Category	Frequency	Percentage
Gender	Male	140	56%
	Female	110	44%
Age Group	18–30	60	24%
	31–50	110	44%
	51 and above	80	32%
Disease Severity	Mild	70	28%
	Moderate	100	40%
	Severe	80	32%

**Descriptive Analysis of Inflammatory Markers**

Evaluation of inflammatory biomarkers demonstrated significant increase in some of the early markers of inflammation in the COVID-19 patients especially severe cases. Patients with critical illness had a high level of the inflammatory markers IL-6 and CRP (revealing high inflammatory activation). A similar excessive mean value was seen in ferritin and D-dimer, which reflect hyperinflammation and coagulation abnormalities. There was also an

increase in numbers of neutrophils which were significantly elevated compared to the numbers of lymphocytes and a significant increase in the neutrophil to lymphocyte ratio (NLR) which indicated immune imbalance due to neutrophilia and lymphopenia. All these findings taken together, suggests that inflammatory markers signature was strongly activated during severe COVID-19 infection.

**Table 2: Descriptive Statistics of Inflammatory Markers**

Biomarker	Mean	Standard Deviation	Clinical Interpretation
IL-6 (pg/mL)	48.7	12.4	High in severe cases
CRP (mg/L)	36.5	10.8	Elevated inflammation
Ferritin (ng/mL)	520.3	140.6	Hyperinflammatory state
D-dimer (µg/mL)	1.8	0.6	Coagulation activation
NLR	6.9	2.1	Immune imbalance

**Correlation Between Inflammatory Markers and Recovery Rate**

The correlation was analyzed in COVID-19 patients, to identify the relationship between inflammatory markers and recovery rate. The findings showed that there were significant negative correlations among all major inflammatory markers with recovery speed.

IL-6, CRP, ferritin, D-dimer, and NLR expression was negatively associated with the length of hospital stay and recovery time. IL-6 was the most strongly inverse correlated with recovery rate, indicating its key role in disease progression, among all variables. The results showed that inflammatory burden was a direct impact on patient recovery outcomes.

**Table 3: Correlation Matrix Between Variables**

Variables	IL-6	CRP	Ferritin	D-dimer	NLR	Recovery Rate
IL-6	1	0.72	0.68	0.65	0.70	-0.74
CRP	0.72	1	0.60	0.58	0.66	-0.69
Ferritin	0.68	0.60	1	0.63	0.61	-0.66
D-dimer	0.65	0.58	0.63	1	0.59	-0.62
NLR	0.70	0.66	0.61	0.59	1	-0.71
Recovery Rate	-0.74	-0.69	-0.66	-0.62	-0.71	1

**Regression Analysis of Recovery Rate Prediction**

The effect of inflammatory markers to the recovery rate was studied using multiple regression analysis. The results indicated that IL-6 (2.1st percentile), CRP (7.5th percentile) and NLR (5th percentile) were significant factors associated with the length of time until recovery, with ferritin (6th percentile) and D-dimer also having some contribution (with similar

significance) to the time until recovery. The overall model was significant, accounting for a large percentage of the variance for recovery outcomes, suggesting that inflammatory markers as a group performed well in predicting recovery. Overall IL-6 was the most important biomarker identified and validated, and confirms its key role in disease severity and treatment delay in COVID-19.

**Table 4: Regression Analysis Predicting Recovery Rate**

Predictor Variable	Beta Coefficient	t-value	Significance (p)
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IL-6	-0.42	6.21	0.000
CRP	-0.31	4.89	0.001
Ferritin	-0.25	3.76	0.003
D-dimer	-0.22	3.41	0.004
NLR	-0.38	5.67	0.000

**Model Summary:**

$R^2 = 0.68$ , Adjusted  $R^2 = 0.65$ , F-statistic = significant ( $p < 0.001$ )

**INTERPRETATION OF RESULTS**

All findings showed a significant correlation between inflammatory markers and recovery rate of the patients with COVID-19. Higher levels of IL-6, CRP, ferritin, D-dimer and NLR were significantly related to more adverse clinical outcomes and longer recovery times. The correlation and regression analyses were able to validate inflammatory burden as an important factor in clinical outcome. IL-6 and NLR were the strongest biomarkers of disease progression and recovery delay when compared to other biomarkers. The results of these studies underscore the cytokine storm hypothesis and the need for immune regulation in the management of COVID-19.

**DISCUSSION**

The result of this study supports, both in this regard and the severity of the illness, the importance of inflammatory markers and immune response in determining the rate of recovery in COVID-19 patients. Increase in interleukin-6 (IL-6), C-reactive protein (CRP), ferritin, D-dimer, and neutrophil-to-lymphocyte ratio (NLR) were significantly associated with poor recovery and more severe infection, as shown by the results. These findings are consistent with the cytokine storm syndrome involved in the severe form of COVID-19 which occurs from uncontrolled activation of immune system and overproduction of pro-inflammatory cytokines (Mehta et al., 2020).

There was a strong negative correlation between inflammatory markers with the recovery rate, showing that the greater the inflammation present in the body, the more inefficient the ability to recover becomes. IL-6 was the most predictive factor for recovery delay, and is consistent with previous studies showing that IL-6 regulates hyperinflammatory responses and respiratory complications (Del Valle et al., 2020). As for the relationship of CRP to duration of hospitalization, there was also a consistent relationship, supporting the use of CRP as an indicator of systemic inflammation and tissue damage.

Ferritin level and D-dimer were also found to be of significant predictive value indicating that COVID-19 is not only an inflammatory disease but also has hematological and coagulation disturbances. Increased ferritin and D-dimer indicate macrophage overactivation with immune dysregulation and an increased risk of thrombotic events, respectively, with the latter generally being seen in more severe COVID-19 cases (Gómez-Pastora et al., 2020). The results support the concept of COVID-19 being a

multi-system disease involving both immune and vascular system.

Another risk factor was the neutrophil to lymphocyte ratio (NLR), which was also found to be a good indicator of the severity and recovery rate of the disease. Increased neutrophils to lymphocytes ratio (NLR), which represents a balance between innate and adaptive immune system, suggests over activation of neutrophils and decreased activity of lymphocytes. This immune imbalance leads to uncontrolled inflammation with delayed viral clearance that has a negative impact on clinical outcomes (Lagunas-Rangel, 2020).

In addition, regression analysis showed that the inflammatory markers as a combination predicted a substantial amount of the variance of recovery outcomes. This shows that severity of the COVID-19 disease cannot be judged by a single biomarker alone, but by a combination of a profile of inflammation. Multi-marker approach yields more accurate and reliable patient outcome prediction than single patient markers.

The findings also align with recent studies where immune dysfunction was also reported as a persistent condition following acute COVID-19 infection and linked with other long-term complications of COVID-19 like the post-acute COVID-19 syndrome. Elevated inflammatory markers over long periods of time could account for low-energy, respiratory tract, and cognitive dysfunction in recovered patients (Nalbandian et al., 2021). Again, this means that inflammation impacts not only the short-term outcome of disease but also health effort in the long run.

In sum, these findings are supported by previous studies and validate the importance of inflammatory markers and immune response in driving the progression of COVID-19 and speed of recovery. The data indicate that proactive management and clinical decision making are greatly supported by early detection of increased inflammatory markers.

**CONCLUSION**

The present study demonstrated that inflammatory markers and immune responses were also important to understand the severity and survival rate of patients with COVID-19. A high level of IL-6, and CRP, ferritin, and NLR were significantly linked with poor clinical outcomes and delayed recovery. Of these, IL-6 and NLR were judged to be the most predictive factors for severity and time to recovery of the disease.

Moreover, the dysfunction of the immune system, such as lymphopenia and excessive activation of

neutrophils, was a significant factor in disease progression, which was confirmed by the study. Patients with high inflammatory burden were more likely to be receiving sources for the inflammatory responses other than a single drug were more likely to experience a longer hospital stay and a slower recovery than those with controlled immune response. Thus, it can be concluded in the context of severity of COVID-19, a multi-faceted phenomenon the relationship between inflammatory process and immune system regulation plays a significant role. Further, the study pointed out that a combination of inflammatory markers adds the most value in predicting the risk of developing a lower urinary tract condition, such as urinary tract infection, as compared to using each inflammatory marker alone. This highlights the need for a multidisciplinary evaluation at an early stage to identify patients who are at risk. In summary, inflammatory markers play a crucial role in COVID-19 infection cases, as they can help with the prediction of disease evolution, treatment decisions, and prognosis.

#### RECOMMENDATIONS

Knowing the results of this study, it is recommended to use inflammatory biomarkers (IL-6, CRP, ferritin, D-dimer, NLR) as an early screening strategy in all COVID-19 confirmed cases. By the time patients present for treatment, some may have elevated inflammatory markers and screening for these markers early allows clinical diagnosis of high-risk patients so that timely treatment decisions can be made, and the risk of developing serious complications minimized.

Viral treatment has also been recommended along with controlling excessive inflammation via treatment strategies. Anti-inflammatory/immunomodulatory therapy may be useful in lowering the intensity of cytokine storm, and help with recovery. It is important that dynamics of immune responses be monitored during hospitalization and formed part of the patient management plan.

Moreover, diagnostic CANT and marker groups should be used together and not a single biomarker should be adopted. The use of several markers may provide much more accurate prognosis of disease and assist in optimal use of resources particularly in critical care hospitals.

Future studies should also be directed towards long term immune consequences of COVID-19, especially consider the post-acute inflammatory syndrome (PIPS). Long-term follow-up studies are needed to appreciate how chronic inflammation manifests in the long term in those patients who have recovered and to be able to tailor rehabilitation programs accordingly.

Last, public health measures should focus on early detection of inflammatory response in infectious diseases, and regular surveillance. Building up laboratory capacity and ensuring access to more

biomarker testing can have a profound impact on future pandemics on clinical outcomes and mortality rates.

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