e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2024; 16(12); 2954-2961

Original Research Article

Evaluation of Hematological, Inflammatory, and Organ Function Markers in Hospitalized COVID-19 Patients: A Descriptive Cross-Sectional Study

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Received: 11-10-2024 / Revised: 16-11-2024 / Accepted: 06-12-2024
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Conflict of interest: Nil

Abstract

Background: The clinical trajectory of coronavirus disease 2019 (COVID-19) is highly variable, and early identification of prognostic biomarkers is essential for guiding therapeutic decisions. Routine blood investigations offer a practical and widely accessible approach to risk stratification in resource-limited settings.

Aims: This study sought to characterize changes in hematological indices, inflammatory biomarkers, and organ function parameters among hospitalized COVID-19 patients and to determine their association with disease severity, intensive care unit (ICU) admission, and in-hospital mortality.

Methods: A hospital-based descriptive cross-sectional study was conducted over six months at Government Medical College and District Hospital, Palakkad. One hundred and seventy RT-PCR-confirmed COVID-19 patients aged ≥13 years were enrolled. Baseline hematological parameters (hemoglobin, total leukocyte count, neutrophil-to-lymphocyte ratio, platelet count), inflammatory markers (C-reactive protein, erythrocyte sedimentation rate, D-dimer), and organ function tests (urea, creatinine, serum albumin) were recorded at admission. Disease severity was classified as mild, moderate, or severe. Descriptive statistics and appropriate tests of significance were applied.

Results: The mean patient age was 46.76 years, and males constituted 62% of the cohort. Anemia, leukocytosis, neutrophilia, and thrombocytopenia were observed in 30%, 25%, 40%, and 15% of patients, respectively. Elevated C-reactive protein, erythrocyte sedimentation rate, and D-dimer levels were found in 70%, 65%, and 45% of patients. Hypoalbuminemia was present in 55%. ICU admission was required in 35%, and overall mortality was 15%. Comorbidities including diabetes mellitus, hypertension, and chronic kidney disease were significantly associated with severe outcomes.

Conclusion: Routine blood investigations serve as valuable prognostic indicators in COVID-19. A combination of elevated inflammatory markers, neutrophilia, lymphopenia, and hypoalbuminemia at admission identifies patients at heightened risk for adverse outcomes, particularly those with underlying comorbidities.

Keywords: COVID-19, hematological parameters, inflammatory markers, neutrophil-to-lymphocyte ratio, D-dimer, comorbidities, ICU admission, mortality.

Introduction

The emergence of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) in late 2019 precipitated an unprecedented global health emergency [1]. COVID-19, the disease caused by this pathogen, demonstrated an extraordinarily wide clinical spectrum ranging from entirely asymptomatic carriage to fulminant multi-organ failure and death [2]. The pandemic placed immense strain on healthcare infrastructure worldwide, demanding rapid development of reliable tools for early risk stratification and clinical decision-making [3]. From the earliest descriptions of the disease emanating from Wuhan, China, it became apparent that host factors played a determining role in shaping clinical trajectories [2]. While the majority of infected individuals experienced self-limiting respiratory symptoms, a substantial minority progressed to severe pneumonia, acute respiratory distress syndrome, thromboembolic complications, and death [3]. The mechanisms underlying this clinical heterogeneity are multifactorial, involving a complex interplay between viral pathogenicity, host immune response, and pre-existing comorbid conditions [4]. Central to the pathophysiology of severe COVID-19 is a dysregulated immune response characterized by an exaggerated release of pro-inflammatory cytokines, often termed a "cytokine

storm" [4]. This hyperinflammatory state drives widespread endothelial injury, microvascular thrombosis, and progressive organ damage [5]. The hematological manifestations of this immune dysregulation are protean and include lymphopenia, neutrophilia, an elevated neutrophil-to-lymphocyte ratio (NLR), and derangements in coagulation parameters [4,6]. These laboratory abnormalities have been consistently observed across diverse patient populations and healthcare settings, suggesting their utility as accessible biomarkers of disease severity [5,6].

Lymphopenia, in particular, has emerged as a hallmark of severe COVID-19 infection [4]. The mechanisms responsible for this lymphocyte depletion are not entirely elucidated but are thought to involve direct viral cytopathic effects on lymphocytes, redistribution of circulating lymphocytes to sites of inflammation, and cytokine-mediated apoptosis [7]. Multiple studies have demonstrated a strong inverse relationship between absolute lymphocyte counts at admission and the likelihood of progression to critical illness [5,6]. Conversely, neutrophilia reflects the innate immune response to infection and, when excessive, contributes to tissue damage through the release of reactive oxygen species and proteolytic enzymes [4]. The NLR, which integrates information about both adaptive and innate immune activation, has consequently attracted considerable attention as a composite biomarker with superior predictive capacity compared to individual cell counts [5,8].

Beyond hematological indices, acute-phase reactants and coagulation markers have demonstrated significant prognostic value in COVID-19. C-reactive protein (CRP), an acute-phase protein synthesized by the liver in response to interleukin-6 stimulation, is markedly elevated in patients with moderate-to-severe disease and correlates well with the degree of systemic inflammation [7]. Similarly, the erythrocyte sedimentation rate (ESR), though a less specific marker, provides additional information about the inflammatory milieu [9]. D-dimer, a fibrin degradation product, has gained particular prominence as a predictor of thromboembolic risk and mortality in COVID-19 [7,8]. The coagulopathy associated with SARS-CoV-2 infection involves activation of both intrinsic and extrinsic coagulation cascades, leading to a prothrombotic state that is reflected by elevated D-dimer levels [8]. Meta-analytic evidence has established that patients with markedly elevated D-dimer concentrations face a substantially increased risk of adverse outcomes, including the need for mechanical ventilation and death [7].

The systemic nature of COVID-19 extends well beyond the respiratory tract. SARS-CoV-2 has demonstrated tropism for multiple organ systems, with the kidneys being particularly vulnerable [10]. Acute kidney injury (AKI) complicates a significant proportion of severe COVID-19 cases and is independently associated with increased mortality [10]. Renal involvement may result from direct viral invasion via angiotensin-converting enzyme 2 (ACE2) receptors expressed on renal tubular epithelial cells, hemodynamic instability, or cytokine-mediated injury [10]. Consequently, serial monitoring of renal function through serum urea and creatinine measurement constitutes an essential component of the laboratory evaluation in hospitalized COVID-19 patients.

Serum albumin, a negative acute-phase protein, has also attracted attention as a prognostic biomarker. Hypoalbuminemia in COVID-19 reflects a combination of hepatic reprioritization of protein synthesis toward acute-phase reactants, capillary leak secondary to systemic inflammation, and catabolic stress [11]. Evidence from observational studies and meta-analyses indicates that reduced serum albumin levels at admission independently predict progression to severe disease and increased mortality risk [11].

The influence of pre-existing comorbidities on COVID-19 outcomes has been extensively documented. Diabetes mellitus, hypertension, and chronic kidney disease have been consistently identified as independent risk factors for severe disease, ICU admission, and death [12,13]. Patients with diabetes mellitus exhibit impaired innate and adaptive immune responses, making them more susceptible to severe infections [12]. Hypertension, through its effects on endothelial function and the renin-angiotensin-aldosterone system, may further augment viral entry and the inflammatory cascade [13]. The presence of multiple comorbidities compounds these risks synergistically, necessitating heightened clinical vigilance in this patient population [12,13].

In the Indian healthcare context, where diagnostic resources may be limited in many settings, the ability to predict disease trajectory using readily available blood investigations assumes particular clinical importance. Routine hematological and biochemical tests are universally accessible, relatively inexpensive, and rapidly obtainable, making them ideal candidates for risk stratification tools in resource-constrained environments. Despite the wealth of global data, region-specific studies remain essential to account for potential differences in disease epidemiology, prevalence of comorbidities, and healthcare delivery patterns. Against this background, the present study was undertaken to systematically evaluate hematological parameters, inflammatory biomarkers, and organ function markers in COVID-19 patients admitted to a tertiary care center in Kerala, India, and to explore their

associations with disease severity, ICU admission, and mortality outcomes.

Aims and Objectives

The primary objectives of this study were:

1. To characterize the spectrum of hematological, inflammatory, and organ function parameters in hospitalized COVID-19 patients.
2. To determine the association of these laboratory parameters with disease severity, ICU admission, and in-hospital mortality.
3. To evaluate the influence of pre-existing comorbidities on clinical outcomes in COVID-19 patients.

Materials and Methods

Study Design and Setting: This hospital-based descriptive cross-sectional study was conducted over a six-month period at two healthcare facilities: Government Medical College, Palakkad, and District Hospital, Palakkad, in Kerala, India. Both institutions served as designated COVID-19 treatment centers during the pandemic and catered to a diverse patient population from the Palakkad district and surrounding regions.

Study Population: The study enrolled 170 patients who were admitted to the above-mentioned facilities during the study period with a confirmed diagnosis of COVID-19.

Inclusion Criteria: Patients fulfilling all of the following criteria were included in the study: (a) age 13 years or above at the time of admission; (b) laboratory-confirmed SARS-CoV-2 infection by reverse transcription polymerase chain reaction (RT-PCR) performed on nasopharyngeal or oropharyngeal swab specimens; and (c) hospitalization at one of the designated study centers during the study period.

Exclusion Criteria: Patients meeting any of the following criteria were excluded: (a) incomplete medical records or missing laboratory data that precluded comprehensive analysis; (b) pregnant women, given the potential confounding influence of gestational physiological changes on hematological and biochemical parameters; and (c) patients with pre-existing hematological disorders, including chronic anemias, myeloproliferative neoplasms, or inherited coagulopathies, which could interfere with accurate interpretation of blood investigation results.

Data Collection: Relevant demographic and clinical information was extracted from hospital medical records in a standardized data collection format. The following categories of data were recorded:

Demographic Variables: Age, sex, and pre-existing comorbidities including diabetes mellitus, hypertension, and chronic kidney disease.

Hematological Parameters: Hemoglobin (Hb) concentration (g/dL), total leukocyte count (TLC; cells/mm^3), differential leukocyte count with calculation of the neutrophil-to-lymphocyte ratio (NLR), and platelet count (cells/mcL). Blood samples were obtained on the day of admission and analyzed using an automated hematologyanalyzer.

Inflammatory Markers: Serum C-reactive protein (CRP; mg/L) was measured using an immunoturbidimetric assay. Erythrocyte sedimentation rate (ESR; mm/hour) was determined by the Westergren method. D-dimer levels (ng/mL) were quantified using a latex-enhanced immunoturbidimetric assay.

Organ Function Markers: Serum urea (mg/dL) and creatinine (mg/dL) were measured using standard colorimetric methods to assess renal function. Serum albumin (g/dL) was determined by the bromocresol green method.

Severity Classification: Disease severity was categorized into three groups based on clinical presentation and treatment requirements as follows:

Mild Disease: Patients managed in general wards without supplemental oxygen therapy.

Moderate Disease: Patients requiring supplemental oxygen via nasal prongs or face mask but not necessitating intensive care.

Severe Disease: Patients requiring ICU admission, invasive or non-invasive mechanical ventilation, or those with fatal outcomes.

Statistical Analysis: Data were entered and analyzed using appropriate statistical software. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages.

The relationships between laboratory parameters and clinical outcomes (disease severity, ICU admission, mortality, and length of hospital stay) were evaluated using appropriate statistical tests. A two-tailed p-value of less than 0.05 was considered statistically significant.

Ethical Considerations: Approval for the study was obtained from the Institutional Ethics Committee prior to commencement. Given the retrospective observational nature of the data collection, the requirement for individual informed consent was waived. All data were handled in accordance with institutional guidelines to maintain patient confidentiality. The study adhered to the principles outlined in the Declaration of Helsinki.

Results

A total of 170 RT-PCR-confirmed COVID-19 patients admitted to the study centers during the six-month study period were included in the final analysis. The demographic, hematological, inflammatory, organ function, severity, and comorbidity profiles of the study cohort are detailed below.

Demographic Characteristics: The age of the study participants ranged from 15 to 87 years, with a mean age of 46.76 years. Males constituted 62% (n=105) of the cohort, while females accounted for 38% (n=65), yielding a male-to-female ratio of approximately 1.6:1 (Table 1).

Table 1: Demographic Profile of the Study Population (N=170)

Characteristic	Value
Age range (years)	15 – 87
Mean age (years)	46.76
Male, n (%)	105 (62%)
Female, n (%)	65 (38%)

Hematological Parameters: The mean hemoglobin level across the cohort was 12.75 g/dL, with individual values ranging from 8.6 to 15.2 g/dL. Anemia (hemoglobin below the gender-specific normal threshold) was identified in 30% (n=51) of patients. The mean total leukocyte count was 10,152 cells/mm³ (range: 3,600–16,700 cells/mm³). Leukocytosis, defined as a TLC exceeding 11,000 cells/mm³, was present in 25% (n=43) of patients, while leukopenia (TLC <4,000 cells/mm³) was documented in 10% (n=17). The mean NLR was 4.92 (range: 2.0–12.0), with markedly elevated ratios observed predominantly among patients classified as severe.

Neutrophilia (absolute neutrophil count >8,000/mcL) was present in 40% (n=68) of patients, and lymphopenia (absolute lymphocyte count <1,500/mcL) was noted in 35% (n=60). The mean platelet count was 185,571/mcL (range: 50,000–470,000/mcL), and thrombocytopenia (platelet count <150,000/mcL) was documented in 15% (n=26) of patients. Lower platelet counts were observed more frequently in patients who required ICU admission (Table 2).

Table 2: Hematological Parameters of the Study Population (N=170)

Parameter	Mean ± SD	Range	Abnormal, n (%)
Hemoglobin (g/dL)	12.75	8.6 – 15.2	51 (30%) – Anemia
Total Leukocyte Count (cells/mm³)	10,152	3,600 – 16,700	43 (25%) – Leukocytosis
Neutrophil-to-Lymphocyte Ratio	4.92	2.0 – 12.0	68 (40%) – Neutrophilia
Platelet Count (cells/mcL)	185,571	50,000 – 470,000	26 (15%) – Thrombocytopenia
Lymphopenia (<1,500/mcL)	–	–	60 (35%)

Inflammatory Markers: Serum CRP levels were elevated above 10 mg/L in 70% (n=119) of patients, with a mean CRP of 30 mg/L (range: 10–100 mg/L). The mean ESR was 38 mm/hour (range: 10–80 mm/hour), with elevations documented in 65% (n=111) of the cohort. D-dimer levels exceeded the upper threshold of 250 ng/mL in 45% (n=77) of patients, with a mean D-dimer value of 376.67 ng/mL (range: 100–1,000 ng/mL).

Higher values of all three inflammatory markers were associated with increased disease severity, prolonged hospitalization, and greater likelihood of ICU admission (Table 3).

Table 3: Inflammatory Markers in the Study Population (N=170)

Marker	Mean Value	Range	Elevated, n (%)
C-Reactive Protein (mg/L)	30	10 – 100	119 (70%)
ESR (mm/hour)	38	10 – 80	111 (65%)
D-dimer (ng/mL)	376.67	100 – 1,000	77 (45%)

Organ Function Markers: Renal function impairment, as evidenced by elevated serum urea and creatinine, was observed in 40% (n=68) of patients. The mean serum urea was 60.29 mg/dL (range: 25–140 mg/dL), and the mean serum creatinine was 1.66 mg/dL (range: 0.8–4.2 mg/dL). Hypoalbuminemia, defined as serum albumin below 3.5 g/dL, was present in 55% (n=94) of patients, with a mean serum albumin of 3.2 g/dL (range: 2.2–4.9 g/dL). Patients with lower serum albumin levels demonstrated a significantly higher requirement for mechanical ventilation and experienced prolonged ICU stays (Table 4).

Table 4: Organ Function Markers in the Study Population (N=170)

Marker	Mean Value	Range	Abnormal, n (%)
Serum Urea (mg/dL)	60.29	25 – 140	68 (40%) – Elevated
Serum Creatinine (mg/dL)	1.66	0.8 – 4.2	68 (40%) – Elevated
Serum Albumin (g/dL)	3.2	2.2 – 4.9	94 (55%) – Hypoalbuminemia

Disease Severity and Mortality: Among the 170 patients, 35% (n=60) required ICU admission, 25% (n=43) were managed in general wards, and the remaining 40% (n=67) received care in intermediate or step-down facilities. The overall in-hospital mortality rate was 15% (n=26). The mean duration of hospitalization was 14 days, with ICU patients experiencing considerably longer stays.

Mortality was highest among patients who exhibited concurrent elevation of CRP, D-dimer, and NLR alongside lymphopenia (Table 5).

Table 5: Disease Severity and Clinical Outcomes (N=170)

Outcome	n (%)
ICU Admission	60 (35%)
General Ward Management	43 (25%)
Moderate Care / Step-down	67 (40%)
In-hospital Mortality	26 (15%)
Mean Length of Stay (days)	14

Comorbidity Profile: Pre-existing comorbidities were documented in 60% (n=102) of patients. Diabetes mellitus was the most frequently observed comorbidity, affecting 40% (n=68) of the cohort. Hypertension was present in 30% (n=51), and chronic kidney disease was identified in 20% (n=34). Patients harboring multiple comorbidities demonstrated a statistically higher incidence of severe disease, ICU admission, and mortality compared to those without comorbidities (Table 6).

Table 6: Distribution of Comorbidities in the Study Population (N=170)

Comorbidity	n (%)
Diabetes Mellitus	68 (40%)
Hypertension	51 (30%)
Chronic Kidney Disease	34 (20%)
Any Comorbidity Present	102 (60%)

Blood Group and Disease Severity: A preliminary assessment of the relationship between ABO blood group and disease severity revealed a trend toward more severe clinical presentations among individuals with blood group O+. However, this association did not achieve statistical significance in the present study cohort and warrants further investigation in larger populations.

Discussion

The present study evaluated 170 hospitalized COVID-19 patients at a tertiary care center in Kerala, India, and identified significant derangements in hematological, inflammatory, and organ function markers that correlated with disease severity and clinical outcomes. These findings contribute to the growing body of literature supporting the role of routine blood investigations in prognostic assessment during SARS-CoV-2 infection.

Demographic Observations: The mean age of 46.76 years and the male preponderance (62%) observed in our cohort are broadly consistent with demographic patterns reported in large epidemiological studies. Richardson et al. [14], in their analysis of 5,700 hospitalized COVID-19 patients in New York City, reported a median age of 63 years with 60.3% male patients. The relatively younger mean age in our study may reflect the demographic structure of the source population and possibly lower thresholds for hospitalization in the Indian setting.

The male predominance aligns with observations across multiple geographies and may be attributable to hormonal, immunological, or behavioral differences between sexes [3,14].

Hematological Parameters: The hematological profile of our cohort revealed a spectrum of abnormalities consistent with established pathophysiological mechanisms of SARS-CoV-2 infection. Anemia was documented in 30% of patients, a finding that, while not specific to COVID-19, likely reflects the systemic inflammatory response and associated hepcidin-mediated iron sequestration characteristic of critical illness [4]. Terpos et al. [4] provided a comprehensive review of hematological derangements in COVID-19 and noted that anemia, though not a primary feature, contributes to tissue

hypoxia and may exacerbate disease severity when present.

Leukocytosis was observed in 25% of patients, consistent with the findings of Huang et al. [2], who reported white blood cell elevations in a proportion of their Wuhan cohort. The coexistence of leukopenia in 10% of our patients highlights the heterogeneous immune response to SARS-CoV-2, with leukopenia typically observed in early viral illness and leukocytosis emerging as secondary bacterial infections or immune dysregulation supervene [4,6].

The elevated mean NLR of 4.92 in our study is of particular clinical significance. Lin et al. [5], in their study of 68 SARS-CoV-2-infected patients, identified NLR as the most powerful independent predictor of disease severity among all hematological parameters analyzed, with a higher area under the receiver operating characteristic curve compared to individual cell counts. Taj et al. [6] similarly demonstrated that NLR elevation correlated strongly with disease progression and mortality in their cohort of COVID-19 patients. The pathophysiological basis for the prognostic superiority of NLR lies in its simultaneous capture of neutrophil-driven innate immune activation and lymphocyte-mediated adaptive immune suppression, both of which are central features of severe COVID-19 [5].

Neutrophilia (40%) and lymphopenia (35%) in our cohort further underscore the immune dysregulation characteristic of moderate-to-severe COVID-19. Mina et al. [15] emphasized that lymphopenia represents one of the most consistent hematological findings in COVID-19 and carries significant prognostic implications. The selective depletion of T-lymphocyte subsets, particularly CD4+ and CD8+ cells, has been linked to impaired viral clearance and predisposition to secondary infections [4,15].

Thrombocytopenia was observed in 15% of our patients and was more prevalent among those requiring ICU care. This finding is consistent with the meta-analysis by Henry et al. [9], who reported that thrombocytopenia was associated with a five-fold increased risk of severe COVID-19. The mechanisms of thrombocytopenia in COVID-19 are multifactorial, involving direct viral effects on megakaryocytes, consumptive coagulopathy, and immune-mediated platelet destruction [4].

Inflammatory Markers: The inflammatory marker profile of our cohort demonstrated widespread immune activation, with CRP elevation in 70%, ESR elevation in 65%, and D-dimer elevation in 45% of patients. The mean CRP of 30 mg/L observed in our study indicates a moderate-to-marked inflammatory response across the cohort. Huang et al. [7], in their meta-analysis encompassing 5,350 patients from 25 studies, established that elevated CRP was associated with a risk ratio of 1.84 for composite poor outcomes, including mortality and the need for intensive care. Our findings reinforce the role of CRP as a readily available and sensitive marker of systemic inflammation in COVID-19.

D-dimer elevation in 45% of patients, with a mean value of 376.67 ng/mL, highlights the prothrombotic state that characterizes moderate-to-severe COVID-19. Tang et al. [8] were among the first to report that markedly abnormal coagulation parameters, including elevated D-dimer, were associated with poor prognosis in COVID-19 pneumonia. Subsequent meta-analytic evidence has confirmed that D-dimer elevation carries a risk ratio of approximately 2.93 for composite adverse outcomes and 4.15 specifically for mortality [7]. In our study, patients with higher D-dimer levels experienced more severe disease courses, longer hospital stays, and higher mortality rates, consistent with these global observations.

Organ Function Markers: Elevated urea and creatinine levels in 40% of patients indicate a substantial burden of renal impairment in our cohort. Puelles et al. [10] provided histological evidence of SARS-CoV-2 tropism for the kidney, demonstrating viral RNA and protein in glomerular and tubular epithelial cells. The high prevalence of renal dysfunction in our study is particularly notable given that 20% of the cohort had pre-existing chronic kidney disease, suggesting that both direct viral injury and pre-existing nephropathy contribute to the renal complications observed. Hypoalbuminemia was the most frequently observed biochemical abnormality in our study, affecting 55% of patients. Low serum albumin reflects a composite of hepatic reprioritization of protein synthesis, increased capillary permeability due to systemic inflammation, and catabolic stress [11]. The association between hypoalbuminemia and adverse outcomes in our cohort is supported by the meta-analytic findings of Violi et al. [11], who reported that decreased albumin levels independently predicted mortality in COVID-19 patients, with a hazard ratio of 0.38 for each unit increase in albumin. This suggests that serum albumin may serve as an integrative marker of both nutritional reserve and inflammatory burden in SARS-CoV-2 infection.

Comorbidities and Outcomes: The high prevalence of comorbidities in our study (60%) and their significant association with adverse outcomes mirrors findings from large international cohorts. Diabetes mellitus was the most common comorbidity (40%), followed by hypertension (30%) and chronic kidney disease (20%). de Almeida-Pititto et al. [12], in their meta-analysis of

18,012 COVID-19 patients, demonstrated that diabetes mellitus was associated with an odds ratio of 2.35 for severe disease and 2.50 for mortality. Hypertension carried odds ratios of 2.98 and 2.88 for severity and mortality, respectively [12]. Parveen et al. [13] further confirmed the adverse prognostic impact of these comorbidities, reporting that hypertension was significantly associated with severity (OR: 2.69; 95% CI: 1.27–5.73; p=0.01).

The synergistic effect of multiple comorbidities observed in our cohort is of considerable clinical relevance. Richardson et al. [14] reported that among their cohort of 5,700 hospitalized COVID-19 patients, the majority had at least one comorbid condition, with hypertension (56.6%), obesity (41.7%), and diabetes (33.8%) being the most common. Patients with multiple comorbidities in our study demonstrated a disproportionately higher rate of ICU admission and mortality, reinforcing the importance of aggressive monitoring and early intervention in this high-risk subgroup.

Blood Group and COVID-19 Severity: The observation of a trend toward more severe disease in patients with blood group O+ is an intriguing finding, though the lack of statistical significance precludes definitive conclusions. This finding partially contrasts with some previous reports suggesting that blood group A may confer increased susceptibility to SARS-CoV-2 infection [3].

The discordance may reflect differences in sample size, population genetics, or the specific outcome measures evaluated. Further research with adequately powered study designs is warranted to clarify the relationship between ABO blood group and COVID-19 outcomes.

Limitations: Several limitations of the present study merit acknowledgment. First, the single-center design limits the generalizability of our findings to broader populations with potentially differing demographic and clinical characteristics. Second, the sample size of 170 patients, while adequate for descriptive analysis, may lack sufficient power to detect statistically significant associations for less common outcomes or risk factors. Third, the retrospective nature of data collection introduces the possibility of information bias and missing data. Fourth, the absence of standardized treatment protocols during the evolving pandemic may have influenced patient outcomes independent of the laboratory parameters assessed. Fifth, serial monitoring of biomarker trajectories was not systematically performed, precluding assessment of the dynamic changes in laboratory parameters over the disease course. Future prospective, multicenter studies with larger sample sizes and serial biomarker measurement would strengthen the evidence base for the prognostic utility of routine blood investigations in COVID-19.

Conclusion

This descriptive cross-sectional study of 170 hospitalized COVID-19 patients demonstrates that routine blood investigations provide clinically meaningful prognostic information. Elevated NLR, present in 40% of patients, and lymphopenia, observed in 35%, were associated with severe disease trajectories and increased ICU admissions. Elevated inflammatory markers, particularly CRP (70%) and D-dimer (45%), correlated with prolonged hospitalization and increased mortality. Thrombocytopenia and hypoalbuminemia were independently associated with severe outcomes and poorer prognosis. Comorbidities, most notably diabetes mellitus, hypertension, and chronic kidney disease, were present in 60% of patients and significantly amplified the risk of adverse outcomes. These findings underscore the value of integrating readily accessible laboratory parameters into clinical risk stratification frameworks, particularly in resource-constrained healthcare settings, to facilitate early identification and targeted management of high-risk COVID-19 patients.

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