

Research Article

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The importance of LDH/Albumin, LDH/Lymphocyte, and LDH/Platelet ratios in the evaluation of COVID-19 B.1.1.7 variant



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Abstract

Objectives: The aim is to investigate the usefulness of lactate dehydrogenase (LDH)/Albumin, LDH/Lymphocyte and LDH/Platelet ratios on the prognosis of coronavirus disease (COVID-19) Alpha (B.1.1.7) variant pneumonia.

Methods: A total of 113 patients who were diagnosed with COVID-19 pneumonia and 60 healthy control group were included in this study. The cases were divided into 2 as classic COVID-19 group, and COVID-19 B.1.1.7 variant group. Complete blood count (CBC) and biochemical parameters of the patients were analyzed retrospectively. Patients with COVID-19 B.1.1.7 variant group were also grouped according to the length of stay in the hospital and the days of hospitalization.

Results: LDH/Albumin, LDH/Platelet, and LDH/Lymphocyte ratios were found to be higher in COVID-19 B.1.1.7 variant group when compared to the control group ($p < 0.001$). The ferritin, neutrophils/lymphocyte (NLR) ratio, procalcitonin (PCT) and LDH/Albumin had the highest area under the curve (AUC) values in the COVID-19 B.1.1.7 variant group (0.950, 0.802, 0.759, and 0.742, respectively). Albumin, Lymphocytes and hemoglobin values were significantly higher in the COVID-19 B.1.1.7 variant group than in the classic COVID-19 group ($p < 0.05$).

Conclusions: LDH/Albumin and LDH/Lymphocyte ratios may be useful for clinicians in predicting the risk of progression to pneumonia in COVID-19 B.1.1.7 variant patients.

Introduction

The COVID-19 pandemic started to appear in China/Wuhan at the end of 2019 and many people lost their lives [1]. Hospitalization and quarantine periods are prolonged because of the high transmission and severe symptoms of the COVID-19 virus. SARS-COV-2 still continues its disease-causing effects through various mutations. Numerous variants have been identified so far, which provided evidence for altered characteristics of the COVID-19 virus [2]. According to the World Health Organization (WHO) classification, a few variants e.g., Alpha (B.1.1.7), Beta (B.1.351), Gamma (P.1), Delta (B.1.617.2), and Delta Plus (AY.1 or B.1.617.2.1) emerged temporarily with spikes in infection rates in the countries where they first appeared [2–6].

It was shown in many previous studies that COVID-19 and its variants cause high morbidity and mortality [7–9]. For this reason, it is important that healthcare services are provided regularly and adequately. In this respect, to make early clinical intervention and treatment possible, it is important to find reliable predictors for disease severity and mortality.

It has great importance to determine the predictor parameters for the infection in COVID-19 cases. The LDH, particularly high levels of LDH, were associated with worse outcomes in COVID-19 cases [7]. It was speculated that there are significant differences in LDH levels in COVID-19 cases. Low levels of albumin and lymphocyte are associated with morbidity and mortality in COVID-19 [8, 10, 11]. There is a need for parameters to help to understand the clinical progression of COVID-19 pneumonia and contribute to elucidating the pathophysiology of the disease. This study was planned based on the idea that LDH/Albumin, LDH/Lymphocyte, and LDH/Platelet ratios may be useful in evaluating the severity of the disease in patients with the COVID-19 B.1.1.7 variant.

The aim of this study was to make a comprehensive analysis of the clinical, laboratory and demographic

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characteristics of COVID-19 patients admitted to Ankara City Hospital and to compare their data with the classic COVID-19, COVID-19 B.1.1.7 variant and healthy control groups. In addition, the role of LDH/Albumin, LDH/Lymphocyte, and LDH/Platelet ratios in the progression of patients in the COVID-19 B.1.1.7 variant group to COVID-19 pneumonia and in the pathogenesis of COVID-19 pneumonia was investigated.

Materials and methods

Study design and participants

The present study was designed retrospectively and was approved by the Ethics Committee of Ankara City Hospital (E2-21-857). In the control group, patients who applied to Ankara City Hospital for routine control and had no known health problems were included. Between February and April 2021, patients with a diagnosis of classic COVID-19 and patients with a diagnosis of COVID-19 B.1.1.7 variant hospitalized in Ankara City Hospital's COVID-19 unit were included in the study group. The diagnostic criteria of the WHO temporary guideline were used in the study [1]. The demographic, clinical, laboratory data and radiological findings of the patients were obtained from electronic medical records and case record forms. The albumin, glucose, urea, creatinine, ferritin, ALT, AST, GGT, LDH, CRP, procalcitonin (PCT), D-dimer, troponin-I and CBC parameters of Group B.1.1.7 and the classic COVID-19 group were analyzed. CBC was analyzed using the ADVIA 2120 Hematology System (Siemens Healthcare Diagnostics, Erlangen, Germany). An Atellica Solution Immunoassay & Clinical Chemistry Analyzer (Siemens Healthcare Diagnostics, Erlangen, Germany) was used for measuring biochemical parameters. PT, APTT and D-dimer were measured using the Sysmex CS-5100 System (Siemens Healthcare Diagnostics, Erlangen, Germany). COVID-19 B.1.1.7 variant and classic COVID-19 groups were compared according to demographic data, radiological characteristics, laboratory findings, and clinical characteristics.

Diagnostic criteria

Oro/nasopharyngeal swab samples for RT-PCR, routine blood tests, and chest computed tomography (CT) were requested from the patients for the diagnosis of COVID-19. Those with a fever and respiratory symptoms, signs of pneumonia on CT, or clinical signs based on positive SARS-CoV-2 PCR results according to the WHO temporary guideline for the diagnosis of COVID-19 [1]. Those with positive B.1.1.7 variant analysis results for SARS-CoV-2 PCR were included in the study. Among the COVID-19 patients, the classic COVID-19 group was named Group N, and the COVID-19 B.1.1.7 variant group was named Group V.

Exclusion criteria

Patients who underwent mechanical ventilation, deceased patients, patients hospitalized in the intensive care unit (ICU), pregnant women and individuals younger than 18 years were not included in the study.

Statistical analysis

The Statistical Package for Social Sciences for Windows, Version 22 (IBM, Armonk, NY, USA) was used for the statistical analyses. The normality of variables was evaluated with the Kolmogorov–Smirnov test. Descriptive analysis was indicated using mean $\pm$ standard deviation (SD) for the parameters normally distributed, and median interquartile range, (IQR) was used for non-normally distributed variables. Normally distributed parameters were compared using one way ANOVA test. Non-normally distributed variables were compared with the Kruskal-Wallis test. Comparisons for categorical variables were performed using the Chi-square test or the Fisher's exact test. The receiver operation characteristic (ROC) curve was used to determine the efficacy of various parameters to predict the prognosis in the COVID-19 B.1.1.7 group. The optimal cut-off values of the PCT, ferritin, troponin-I, LDH, LDH/Albumin, LDH/Platelet, LDH/Lymphocyte and NLR ratios were calculated by applying the ROC analysis. A value of $p < 0.05$ was considered to be statistically significant.

Results

Demographic, clinical, and treatment results

A total of 113 patients, 64 of whom were diagnosed with classic COVID-19 and 49 of whom were diagnosed with COVID-19 B.1.1.7 variant and 60 healthy subjects were included in the study. The median age of the COVID-19 B.1.1.7 group was found to be 48 (38.5–64.5), and that of the classic COVID-19 group was 73 (59–80), and 65 of the COVID-19 patients were male ($p < 0.001$). Ground-glass opacity was found in the CT results of 44 patients in total, and ground-glass opacity was more dominant in the classic COVID-19 group (67.2%) than in the COVID-19 B.1.1.7 variant group (6.1%) ($p < 0.001$). The median length of hospitalization was 10 days for both COVID-19 patient groups. The number of individuals who died was one and the patient who died was excluded from the study (Table 1).

RT-PCR results were positive (100%) in all of the COVID-19 B.1.1.7 variant group. RT-PCR test was positive in 67.2% ($n=21$) of the classic COVID-19 group. The most common complaints at admission to the hospital in both COVID-19 groups were fever (30.9%), dry cough (28.3%) and shortness of breath (19.5%), respectively.

The comorbidity was detected in 85 (75.2%) patients in total, and comorbidity was higher in patients in the classic COVID-19 group ($p=0.015$). The most common comorbidities were systemic hypertension (46, 30.9, 10.6, and 10.6%), diabetes, congestive heart disease, and ischemic heart disease, respectively. Favipiravir ($n=69$) was used predominantly in the

Table 1: Demographic characteristics of the COVID-19 groups.

Variables	Total (n=113)	COVID-19 B.1.1.7 variant group (n=49)	Classic COVID-19 group (n=64)	p-Value
Age, years	45 (33–60)	48 (38.5–64.5)	73.0 (59–80)	<0.001
Gender, male/female	65/48	26/23	39/25	0.446
Abnormalities on chest CT, %	54 (47.8)	10 (20.4)	44 (72.1)	<0.001
Ground-glass opacity, %	44 (38.9)	3 (6.1)	41 (67.2)	<0.001
Viral pneumonia, %	10 (8.8)	7 (21.9)	3 (4.9)	0.090
Pulmonary parenchyma infiltration, %	2 (1.8)	2 (4.1)	0	N/A
Length of hospital stay, day, median	10 (7–18)	10 (6–15.5)	10 (7–21.5)	0.620
Death, %	1 (0.9)	1 (2.0)	0	N/A
Oxygen therapy, %	3 (2.7)	3 (6.1)	0	N/A
Comorbidities	85 (75.2)	31 (63.3)	54 (84.4)	0.015
Systemic hypertension, n (%)	52 (46.0)	17 (34.7)	35 (54.7)	0.038
Diabetes mellitus, n (%)	35 (30.9)	8 (16.3)	27 (42.2)	0.004
Congestive heart disease, n (%)	12 (10.6)	4 (8.2)	8 (12.5)	0.548
Ischemic heart disease, n (%)	12 (10.6)	1 (2.0)	11 (17.2)	0.012
Chronic renal disease, n (%)	2 (1.8)	1 (2.0)	1 (1.6)	0.681
Cancer, n (%)	8 (7.1)	3 (6.1)	5 (7.8)	0.515
Cerebrovascular events, n (%)	4 (3.6)	1 (2.0)	3 (4.7)	0.632
Chronic obstructive pulmonary disease, n (%)	5 (4.4)	1 (2.0)	4 (6.3)	0.386
signs and symptoms				
Fever, %	35 (30.9)	12 (24.5)	23 (35.9)	0.222
Dry cough, %	32 (28.3)	18 (36.7)	14 (21.9)	0.095
Myalgia, %	6 (5.3)	5 (10.2)	1 (1.6)	0.084
Dyspnea, %	22 (19.5)	6 (12.2)	16 (25)	0.100
Weakness, %	17 (15.0)	10 (20.4)	7 (10.9)	0.191
Treatment				
Chloroquine, %	17 (15.0)	7 (14.3)	10 (15.6)	0.530
Favipiravir, %	69 (61.1)	49 (100)	20 (31.3)	<0.001
Steroid, %	29 (25.7)	11 (22.5)	18 (28.1)	0.523
Colchicine, %	20 (17.7)	8 (16.3)	12 (18.8)	0.807

p-Values less than 0.05 were considered significant and highlighted in bold.

treatment and steroid (n=29) was used secondarily. In B.1.1.7 group patients, steroid treatment was used in addition to the Favipiravir treatment (Table 1).

Analysis of LDH/Albumin, LDH/Lymphocyte, and LDH/Platelet ratios in COVID-19 B.1.1.7 variant group patients

It was shown that the LDH, LDH/Albumin, LDH/Lymphocytes, and LDH/Platelet parameters were significant and higher in COVID-19 B.1.1.7 variant group patients when compared to the control group ($p<0.001$) (Table 2). There was a statistically significant difference between the COVID-19 B.1.1.7 variant group and the classic COVID-19 group in terms of LDH/Lymphocyte ratio ($p=0.037$) (Figure 1).

Laboratory findings of Group N and Group V

Significant differences were detected between all groups except for ALT levels ($p<0.05$) (Table 2). There were significant differences between the classic COVID-19 group and COVID-19 B.1.1.7 variant group in all parameters except AST, GGT, LDH, and creatinine ($p<0.05$). The WBC, Neutrophils, PLT, NLR, CRP, PCT, D-dimer, troponin-I, GGT, LDH/Lymphocyte, ferritin, glucose, creatinine and urea values were significantly higher in the classic COVID-19 group than in COVID-19 B.1.1.7 variant group ($p<0.05$). Albumin, hemoglobin and lymphocytes, values were significantly higher in the COVID-19 B.1.1.7 variant group than in the classic COVID-19 group ($p<0.05$) (Table 2). Albumin, WBC, neutrophils, lymphocytes, PLT, AST, ferritin, LDH, LDH/Albumin, LDH/Lymphocyte, and LDH/Platelet parameters were found to be statistically

Table 2: Blood routine parameters characteristics of patients group according to admission.

Variables	Control group (n=60)	Group 1 (n=49)	Group 2 (n=49)	Group 3 (n=49)	p-Value	Pairwise comparison
Age, years	40 (29–72)	48 (38.5–64.5)	48 (38.5–64.5)	48 (38.5–64.5)	0.004^a	C vs. G1, p=0.020 C vs. G3, p=0.020 G2 vs. G3, p=1.000
Gender, female/male	26/34	23/26	23/26	23/26	0.974 ^b	–
Albumin, g/L	46 (44–48)	44 (41–47)	40 (36–43)	39 (34–43)	<0.001^a	C vs. G1, p=0.007 C vs. G3, p<0.001 G2 vs. G3, p=1.000
Total protein, g/L	72 (68–72)	67 (64–69.5)	60 (58–64)	62 (58–66)	<0.001^a	C vs. G1, p=0.033 C vs. G3, p<0.001 G2 vs. G3, p=1.000
WBC, $\times 10^9/L$	7.2 (6.7–10.1)	5.8 (4.3–7.5)	6.8 (4.6–8.7)	7.4 (5.9–9.9)	<0.001^a	C vs. G1, p<0.001 C vs. G3, p=1.000 G2 vs. G3, p=0.508
Neutrophils, $\times 10^9/L$	4.7 (3.1–6.9)	3.4 (2.6–5.1)	4.3 (2.4–6.3)	4.4 (3.3–7.3)	0.029^a	C vs. G1, p=0.094 C vs. G3, p=1.000 G2 vs. G3, p=1.000
Lymphocytes, $\times 10^9/L$	2.0 (1.5–2.7)	1.3 (0.9–1.7)	1.3 (0.9–2.2)	1.9 (1.3–2.4)	<0.001^a	C vs. G1, p<0.001 C vs. G3, p=1.000 G2 vs. G3, p=0.076
RBC, $\times 10^{12}/L$	4.8 (4.5–5.2)	4.9 (4.4–5.1)	4.5 (4.4–4.8)	4.5 (4.1–4.7)	<0.001^a	C vs. G1, p=1.000 C vs. G3, p=0.002 G2 vs. G3, p=1.000
Hemoglobin, g/dL	13.8 (12.7–14.5)	14.1 (12.9–14.9)	13.4 (12.4–14.3)	13.2 (11.8–13.9)	0.025^a	C vs. G1, p=1.000 C vs. G3, p=0.292 G2 vs. G3, p=1.000
Hematocrit, %	43.9 (40.7–44.9)	41.6 (39.2–44.9)	39.6 (36.8–42.3)	38.2 (35.9–41.9)	0.001^a	C vs. G1, p=1.000 C vs. G3, p=0.004 G2 vs. G3, p=1.000
Platelet, $\times 10^9/L$	250 (100–304)	188 (155–220)	242 (157–305)	253 (199–344)	<0.001^a	C vs. G1 p<0.001 C vs. G3, p=1.000 G2 vs. G3, p=0.934
N/L	2.0 (1.4–4.9)	2.2 (1.7–4.2)	2.2 (1.4–8.1)	2.2 (1.5–5.4)	0.718 ^a	–
CRP, g/L	0.04 (0.02–0.07)	0.03 (0.01–0.04)	0.02 (0.01–0.05)	0.01 (0.01–0.03)	0.001^a	C vs. G1, p=0.283 C vs. G3, p=0.001 G2 vs. G3, p=1.000
Procalcitonin, $\mu g/L$	0.03 (0.03–0.07)	0.04 (0.03–0.08)	0.03 (0.03–0.06)	0.03 (0.03–0.04)	0.034^a	C vs. G1, p=0.674 C vs. G3, p=0.780 G2 vs. G3, p=0.624
D-dimer, mg/L	0.4 (0.2–0.7)	0.4 (0.3–0.9)	0.4 (0.3–0.9)	0.5 (0.3–0.6)	0.367 ^a	–
Troponin-I H, ng/L	2 (2–5)	2.5 (2.5–7)	2.5 (2.5–4)	2.5 (2.5–4.5)	0.068 ^a	–
Amylase, U/L	59 (51.3–82.8)	62.5 (48–78.5)	68 (56–79.5)	74 (55–72)	0.408 ^a	–
Lipase, U/L	30 (25–37)	42.5 (31–49.3)	46 (33.5–58.5)	48.5 (42–72)	<0.001^a	C vs. G1, p<0.001 C vs. G3, p<0.001 G2 vs. G3, p=1.000
CK, U/L	131 (102–154)	71 (49–127)	65 (41–90)	40 (29–65)	<0.001^a	C vs. G1, p=0.004 C vs. G3, p<0.001 G2 vs. G3, p=0.209
aPTT, s	24.5 (22.2–25.9)	24.6 (23.1–26.3)	24 (21.9–25.1)	23.5 (22.1–26.1)	0.350 ^a	–
PT, s	11.9 (10.9–12.8)	11.7 (11.3–12.8)	11.8 (11.4–12.6)	11.9 (11.5–13.1)	0.506 ^a	–
AST, U/L	17.5 (14–23.8)	30 (21–40.5)	36 (24–61)	31 (22.3–46)	<0.001^a	C vs. G1, p<0.001 C vs. G3 p<0.001 G2 vs. G3, p=1.000
ALT, U/L	23 (16–37.8)	26 (20–41.5)	38 (27–91)	48 (30–79)	<0.001^a	C vs. G1, p=1.000 C vs. G3 p<0.001 G2 vs. G3, p=1.000
ALP, U/L	65 (60–80)	67 (64–69.5)	60 (58–64)	62 (58–66)	0.926 ^a	–

Table 2: (continued)

Variables	Control group (n=60)	Group 1 (n=49)	Group 2 (n=49)	Group 3 (n=49)	p-Value	Pairwise comparison
GGT, U/L	20 (15–32.8)	26 (17–50)	42 (18.5–66)	36 (22.3–95)	0.001^a	C vs. G1, p=0.765 C vs. G3, p=0.002 G2 vs. G3, p=1.000
LDH, U/L	198 (172–244)	271 (242–343)	301 (243–338)	278.5 (240–312)	<0.001^a	C vs. G1, p<0.001 C vs. G3, p<0.001 G2 vs. G3, p=0.737
LDH/Albumin	4.3 (3.7–5.2)	6.5 (5.2–7.7)	7.6 (5.6–9.2)	7.1 (5.8–8.4)	<0.001^a	C vs. G1, p<0.001 C vs. G3, p<0.001 G2 vs. G3, p=1.000
LDH/PLT	0.7 (0.6–1.0)	1.5 (1.2–2.1)	1.3 (0.9–1.9)	1.1 (0.8–1.5)	<0.001^a	C vs. G1, p<0.001 C vs. G3, p=0.005 G2 vs. G3, p=0.330
LDH/LYM	88.5 (67.6–146)	224 (135–314)	199 (115–396)	155 (107–233)	<0.001^a	C vs. G1, p<0.001 C vs. G3, p=0.003 G2 vs. G3, p=0.307
Ferritin, µg/L	58 (34–88)	146 (56–243)	212 (71–448)	158 (70–378)	<0.001^a	C vs. G1, p<0.001 C vs. G3, p<0.001 G2 vs. G3, p=1.000
Glucose, mg/dL	100 (85–113)	102 (81–117)	105 (80–151)	105 (84–143)	0.420 ^a	–
Urea, mg/dL	29 (22–39)	29.5 (24–36)	38 (27–45)	36 (26–45)	0.001^a	C vs. G1, p=1.000 C vs. G3, p=0.018 G2 vs. G3, p=1.000
Creatinine, mg/dL	0.7 (0.6–0.9)	0.8 (0.7–0.9)	0.8 (0.7–0.9)	0.8 (0.6–0.9)	0.245 ^a	–
Total bilirubin, mg/dL	0.8 (0.7–0.8)	0.4 (0.4–0.6)	0.4 (0.3–0.5)	0.4 (0.3–0.5)	<0.001^a	C vs. G1, p<0.001 C vs. G3, p<0.001 G2 vs. G3, p=1.000
Conjugated bilirubin, mg/dL	0.3 (0.2–0.4)	0.2 (0.1–0.2)	0.1 (0.1–0.2)	0.1 (0.1–0.2)	<0.001^a	C vs. G1, p<0.001 C vs. G3, p<0.001 G2 vs. G3, p=1.000

Descriptive statistics were presented as median (IQR). ^aKruskal–Wallis test. ^bChi square test. Bonferroni correction was used for pairwise comparisons. The significance level was set at p<0.05. LYM, lymphocyte; PLT, platelet. p-Values less than 0.05 were considered significant highlighted in bold.

significant between the control group and COVID-19 B.1.1.7 variant group (p<0.05) (Table 2).

The ROC analysis of routine blood parameters in predicting patients in the COVID-19 variant group B.1.1.7

The ROC Curve Analysis was used to determine the efficacy of various parameters to predict the prognosis in the COVID-19 B.1.1.7 variant group. The COVID-19 B.1.1.7 variant group was set as the positive group, and the classic COVID-19 group was set as the negative group. According to ROC Curve analysis, the ferritin, NLR ratio, PCT and LDH/Albumin had the highest AUC values in the COVID-19 B.1.1.7 variant group (0.950, 0.802, 0.759, and 0.742, respectively, as seen in Figure 2). The AUC, optimal cut-off, and sensitivity and specificity values of laboratory parameters are given in Table 3.

Discussion

The COVID-19 pandemic still continues to affect the whole world with its variants and might cause an extensive clinical spectrum from subclinical disease to respiratory failure. LDH is an intracellular enzyme in cells of almost all organ systems. LDH values may increase due to decreased oxygenation as a result of multiple organ damage and upregulation of the glycolytic pathway [12].

In this study, when the COVID-19 B.1.1.7 variant group was compared with the healthy control group, it was shown that especially the LDH/Albumin, LDH/Lymphocyte and LDH/Platelet ratios were very useful in predicting the disease. These evaluated rates, as well as the age, presence of comorbidity, and symptoms at presentation may contribute to elucidating the pathogenesis of the disease in the COVID-19 B.1.1.7 variant group.

Due to the increase in lactate as a result of infection and tissue damage, the extracellular pH becomes acidic,

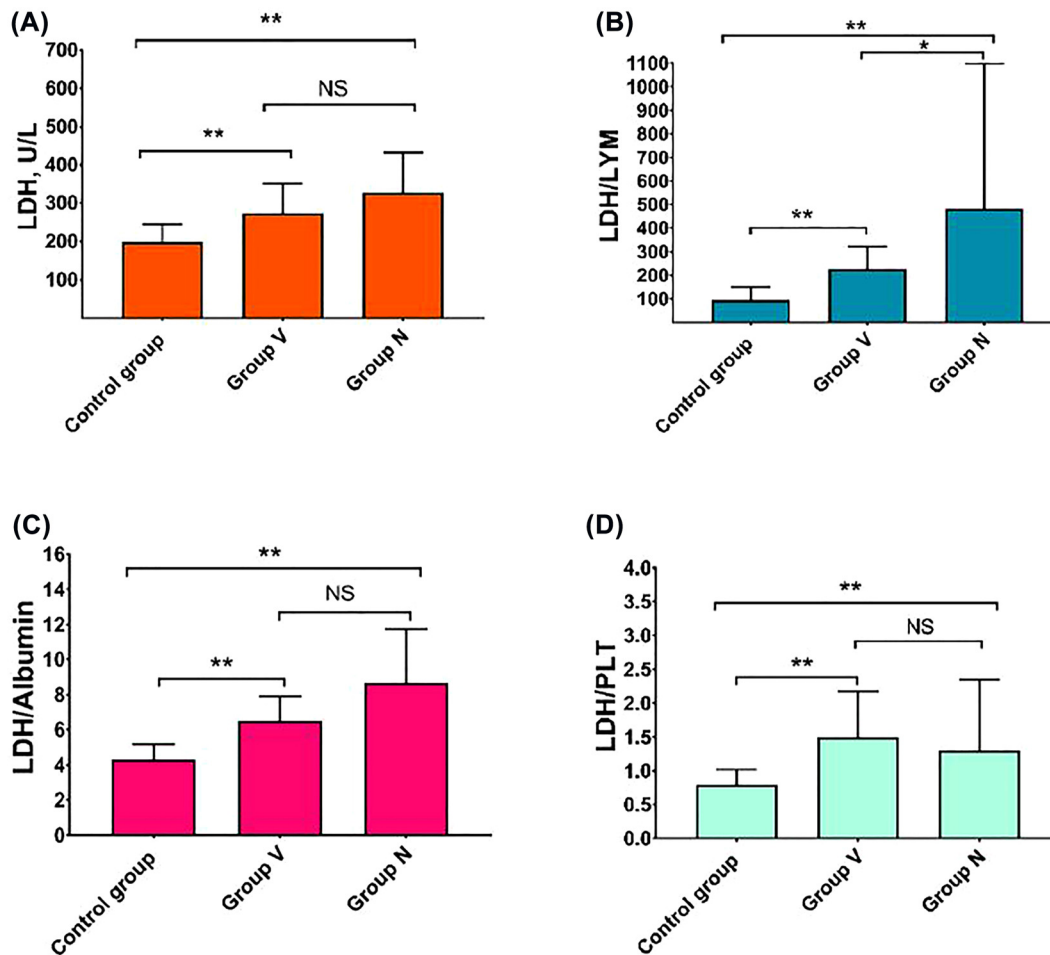


Figure 1: Comparison of LDH (A), LDH/LYM (B), LDH/Albumin (C), LDH/PLT (D) levels of control group, Group V and Group N groups. p-Values less than 0.001 were considered significant highlighted in double asterisk.

NS, not significant; PLT, platelet; and LYM, lymphocytes.

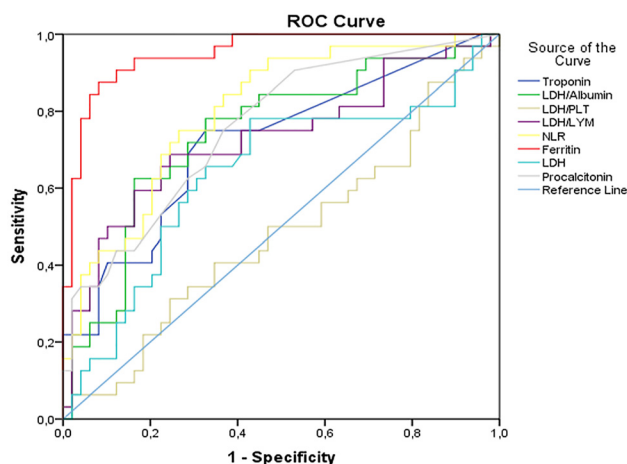


Figure 2: The ROC curves of LDH/Albumin, LDH/PLT, LDH/LYM, LDH, NLR, ferritin, procalcitonin, and troponin in predicting severe SARS-CoV-2 infection, on admission. PLT, platelet; LYM, lymphocytes; NLR, neutrophils-to-lymphocytes ratio.

triggering the activation of metalloproteases and increasing macrophage-mediated angiogenesis [13]. Severe infections might cause cytokine-mediated tissue damage and LDH release. Elevated LDH can be expected in patients with severe COVID-19 infection because LDH is present in lung tissue [7]. In this study, when the COVID-19 B.1.1.7 variant group and the healthy control group were compared, it was found that the LDH, LDH/Albumin, LDH/Lymphocytes, and LDH/Platelet parameters were significant and higher than the control group ($p < 0.001$).

The predictive value of LDH for severe COVID-19 patients was emphasized in some previous studies [14, 15]. In this study, the optimal cut-off was found to be 293 U/L for LDH (sensitivity and specificity 78.1 and 57.1%, respectively), which is consistent with the study by Poggialia et al. [15]. Moreover, our study found that LDH/Albumin and LDH/Lymphocyte, parameters can be used as predictive diagnostic tools to identify subjects with the COVID-19

Table 3: The value of blood routine parameters in diagnosis of patients with COVID-19 B.1.1.7 variant group on admission.

Variables	Cut-off value	AUC (95% CI)	Sensitivity, %	Specificity, %	p-Value
LDH, U/L	≥244	0.823 (0.738–0.908)	75.5	72.5	<0.001
NLR	≥2.03	0.548 (0.424–0.673)	61.2	50.0	0.436
Ferritin, µg/L	≥71.5	0.752 (0.648–0.856)	71.4	70.0	<0.001
Procalcitonin, µg/L	≥0.035	0.623 (0.505–0.740)	53.1	70.0	0.048
Troponin, ng/L	≥2.25	0.633 (0.507–0.759)	95.9	50.0	0.033
LDH/Albumin	≥5.22	0.854 (0.777–0.931)	77.1	75.0	<0.001
LDH/PLT	≥1.06	0.914 (0.858–0.971)	83.3	82.5	<0.001
LDH/LYM	≥142.5	0.844 (0.761–0.927)	75.0	75.0	<0.001

AUC, area under the curve; PLT, platelet; NLR, neutrophils-to-lymphocytes ratio; LYM, lymphocyte. Asymptotic significance less than 0.05 were considered significant. p-Values less than 0.05 were considered significant highlighted in bold.

B.1.1.7 variant ($p < 0.05$). In cases of COVID-19 with chest tomography findings, the diagnosis of COVID-19 is definitive. However, the RT-PCR test may be negative in COVID-19 B.1.1.7 variant cases that do not have tomographic findings, and evaluating blood parameters may help diagnosis. High LDH/Albumin, LDH/Platelet, and LDH/Lymphocyte ratios in COVID-19 cases may contribute to the diagnosis of the COVID-19 B.1.1.7 variant. Since there is no definitive cure for COVID-19 yet and the importance of early isolation is indisputable, these rates can help clinicians with their ease of application and early diagnosis and mortality estimation.

We think that the high LDH/Albumin and LDH/Lymphocyte ratios in the COVID-19 B.1.1.7 variant provide important information about the clinical course of the disease, as well as showing that there is recovery after treatment. These rates can give an idea for clinicians about the prognosis of the disease.

It was reported in various studies in the literature that serum albumin, lymphocytes, and platelet values were found to be low in COVID-19 patients [7, 12, 16, 17]. Lymphopenia, elevated ferritin, procalcitonin and NLR were reported to be predictors of severe prognosis [18]. In our study, lymphopenia, high NLR, ferritin, procalcitonin levels were detected in both COVID-19 groups, which is consistent with the results of these studies [18, 19]. Moreover, our study found that ferritin, NLR, and procalcitonin parameters can be used as predictive diagnostic tools to identify subjects with the COVID-19 B.1.1.7 variant (AUC values were 0.950, 0.802, and 0.759, respectively).

Changes were detected in some laboratory parameters in the COVID-19 B.1.1.7 variant group when the laboratory results were evaluated. When the laboratory parameters of the COVID-19 B.1.1.7 variant group were compared to the control group, the albumin, Lymphocytes, PLT parameters decreased, and the urea, creatinine, Troponin I, PCT, NLR,

AST, ALT, ferritin, LDH, LDH/Albumin, LDH/Lymphocytes, and LDH/Platelet parameters were elevated in COVID-19 B.1.1.7 variant group in line with the meta-analysis study [12, 19]. Although the NEU and CRP values were higher in the classic COVID-19 group in line with the meta-analysis when compared to the control group, they were found to be lower in the COVID-19 B.1.1.7 variant group [12]. In their meta-analysis, Ji et al. reported that inflammatory markers increased according to the severity of COVID-19. In this study, inflammatory markers WBC, CRP and PCT were found to be lower in the COVID-19 B.1.1.7 variant group compared to the classical COVID-19 group. Therefore, this study suggests that the prognosis of the COVID-19 B.1.1.7 variant is milder than that of the classic COVID-19 group, similar to the results of the meta-analysis by Ji et al. [20].

In the present study, the most commonly used drug was found to be Favipiravir in the treatment of COVID-19 patients. Hydroxychloroquine was used in limited cases. Although their use was controversial at the beginning of the pandemic, these drugs were used widely for treatment as the experience was gained in time, and were added to the treatment with steroids in some patients within the indication.

When the comorbid disease states of COVID-19 cases were examined in this study, the most common comorbid diseases were found to be systemic hypertension, Diabetes mellitus, congestive heart disease, ischemic heart disease, chronic renal disease, cancer, cerebrovascular events, and chronic obstructive pulmonary disease [1, 21–25]. Fever, cough, shortness of breath, and fatigue were the most common symptoms in the symptomatology. One of the hospitalized cases died of all these additional diseases and symptoms. One case that was recorded was an intubated patient who had bilateral lung involvement and died as a

result of cardiac ischemia and respiratory failure with ischemic heart disease comorbidity.

The limitation of this study was the small sampling size. We included all COVID-19 patients who met the diagnostic criteria and were hospitalized due to the variant. Could the COVID-19 B.1.1.7 variant group accompany by undiagnosed diseases cause the deterioration of pneumonia? Does it play a role in the pathogenesis of the disease? Comprehensive studies may be necessary in this regard.

Conclusions

The LDH/Albumin, LDH/Platelet and LDH/Lymphocyte ratios increased in COVID-19 B.1.1.7 variant patients. The LDH/Albumin, LDH/Lymphocyte ratios, ferritin and procalcitonin may be useful for clinicians in predicting the risk of progression to pneumonia in COVID-19 B.1.1.7 variant patients.

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