

Associations of Galectin-3 with Laboratory Biomarkers and Electrocardiographic Parameters in Acute and Post-COVID-19 Patients

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ABSTRACT

BACKGROUND. Despite substantial reductions in COVID-19 mortality following widespread vaccination and the predominance of Omicron variants, SARS-CoV-2 infection continues to pose a significant threat to vulnerable populations and remains associated with long-term complications. Increasing evidence indicates that hematological, biochemical, and cardiovascular abnormalities contribute to disease progression and post-COVID sequelae. However, the combined clinical significance of routine laboratory markers, electrocardiographic findings, and Galectin-3 - a biomarker involved in inflammation and extracellular matrix remodeling - remains unclear.

OBJECTIVES. To determine the associations between hematological and biochemical biomarkers, electrocardiographic parameters, and Galectin-3 levels across different stages of COVID-19 progression, including the post-COVID-19 period.

METHODS. We enrolled 86 participants, including 61 patients with acute COVID-19 and 25 post-COVID-19 patients. We classified participants as moderate (n=21), severe (n=20), critical (n=20), or post-COVID (n=25). We assessed clinical, laboratory, and electrocardiographic parameters. We measured serum galectin-3 levels using ELISA. Statistical analyses included correlation, linear regression, and logistic regression methods.

RESULTS. Galectin-3 showed significant positive correlations with age, BMI, respiratory rate, body temperature, leukocyte count, ferritin, and LDH, while demonstrating a negative correlation with oxygen saturation. Exploratory analyses suggested potential associations between Galectin-3 and selected markers of organ injury and coagulation in some patient subgroups, including the post-COVID-19 group. Electrocardiographic analysis demonstrated significant correlations between Galectin-3 and P-wave duration, T-wave duration, PR interval, and QTc interval, suggesting potential alterations in electrophysiological profiles associated with higher Galectin-3 concentrations.

CONCLUSIONS. Persistent associations observed during the post-COVID-19 period may reflect ongoing inflammatory or remodeling-related processes; however, further longitudinal studies incorporating imaging-based assessments are required to determine their clinical significance. Galectin-3 may represent a potential biomarker associated with disease severity and persistent biological alterations after COVID-19, although its prognostic value and role in patient risk stratification require validation in larger prospective studies.

KEYWORDS. Biomarkers; COVID-19; electrocardiography; fibrosis; galectin-3; inflammation; long COVID.

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BACKGROUND

Although the global burden of COVID-19 has decreased following widespread vaccination and the predominance of less virulent SARS-CoV-2 variants, severe disease, multi-organ involvement, and post-COVID complications remain important clinical challenges. ¹ Numerous studies have demonstrated the prognostic value of hematological, biochemical, and inflammatory biomarkers in assessing disease severity and outcomes. ² However, the relationships between these parameters, electrocardiographic abnormalities, and Galectin-3 levels across different stages of COVID-19 remain incompletely understood.

Galectin-3 is a multifunctional biomarker involved in inflammation, fibrosis, tissue remodeling, and cardiovascular injury. ² Elevated Galectin-3 concentrations have been associated with severe COVID-19, organ dysfunction, and adverse cardiovascular outcomes. ³ Nevertheless, its

relationship with routine laboratory markers and ECG changes during both acute COVID-19 and the post-COVID period requires further investigation. ⁴

This study aimed to evaluate associations between Galectin-3 levels, hematological and biochemical biomarkers, and electrocardiographic parameters in patients with acute COVID-19 of varying severity and during the post-COVID period. We hypothesized that higher Galectin-3 levels would be associated with greater acute COVID-19 severity, increased inflammatory activity, markers of organ involvement, and selected electrocardiographic alterations that may reflect cardiovascular involvement. ⁵

METHODS

This single-center observational study, incorporating retrospective and prospective components, was conducted at the First University Clinic of Tbilisi State Medical University between January 2023 and March 2026. The study enrolled 86 participants: 61 patients

with acute COVID-19 and 25 post-COVID patients evaluated 2–6 months after acute infection.

Study design and data source

The study included both prospective and retrospective components. We prospectively enrolled patients with acute COVID-19 (moderate, severe, and critical) during hospitalization. We classified disease severity according to the Chinese Clinical Guidance for COVID-19 Pneumonia Diagnosis and Treatment recommendations.

We retrospectively assembled the post-COVID-19 group, which included patients evaluated during the post-acute phase of SARS-CoV-2 infection. Some of these individuals had previously been hospitalized at our institution during the acute phase of COVID-19, although they were not necessarily included as participants in the original acute COVID-19 cohort. The study population comprised 61 hospitalized patients with acute COVID-19 (21 moderate, 20 severe, and 20 critical cases) and 25 post-COVID-19 patients evaluated during outpatient follow-up visits.

We recruited participants through consecutive enrollment of eligible patients presenting during the study period. We enrolled acute COVID-19 patients during hospitalization, whereas we recruited post-COVID participants during outpatient follow-up assessments. We measured galectin-3 concentrations using enzyme-linked immunosorbent assay (ELISA). For hospitalized patients in the moderate, severe, and critical groups, we obtained blood samples for Galectin-3 analysis within the first 24 hours after hospital admission. In the post-COVID-19 group, we measured Galectin-3 during outpatient evaluation.

We obtained electrocardiographic intervals from standard 12-lead ECG recordings using the EDAN SE-12 system. The ECG software initially generated measurements, which the attending cardiologist reviewed when clinically indicated. We corrected QT using the Bazett formula. We did not systematically record electrolyte concentrations or the use of QT-prolonging medications; therefore, we did not include them in the present analysis. Similarly, we did not prospectively collect arrhythmia outcomes, including atrial fibrillation incidence.

We applied a standardized data collection protocol across all study groups. In addition to routine

laboratory investigations performed as part of standard clinical care, all participants underwent Galectin-3 testing. Hospitalized patients also underwent focused cardiac ultrasound examination (FoCUS/PoCUS) and echocardiographic assessment in accordance with American Heart Association recommendations during the first 24 hours of hospitalization.

We obtained written informed consent from all participants before enrollment and blood sampling. The local institutional ethics committee approved the study protocol, and we conducted the study in accordance with the principles of the Declaration of Helsinki.

We selected participants according to predefined inclusion and exclusion criteria established before study initiation. We excluded patients lacking essential clinical, laboratory, electrocardiographic, or echocardiographic information required for the primary analyses.

We classified participants according to the Chinese Clinical Guidelines into moderate ($n=21$), severe ($n=20$), critical ($n=20$), and post-COVID-19 ($n=25$) groups. We defined severe disease as respiratory rate $>30/\text{min}$, $\text{SpO}_2 \leq 93\%$, $\text{PaO}_2/\text{FiO}_2 \leq 300 \text{ mmHg}$, or lung involvement $>50\%$, whereas critical disease included respiratory failure, shock, or multiple organ failure.

Inclusion criteria comprised age ≥ 18 years and RT-PCR confirmation of SARS-CoV-2 infection. Exclusion criteria included age <18 years, pregnancy, dialysis treatment, mechanical ventilation at enrollment, type 1 or type 2 diabetes mellitus (except steroid-induced hyperglycemia), poor echocardiographic image quality, and procedure-related myocardial infarction.

We excluded patients receiving invasive mechanical ventilation at enrollment because we could not reliably obtain informed consent or perform standardized baseline assessments under these circumstances. However, patients admitted to the intensive care unit were eligible for inclusion. We permitted non-invasive respiratory support modalities, including high-flow nasal cannula (HFNC), continuous positive airway pressure (CPAP), and bilevel positive airway pressure (BiPAP). Participants who subsequently required invasive mechanical ventilation after enrollment remained in the study cohort and continued follow-up according to the study protocol.

Definition of the post-COVID-19 group

The post-COVID-19 group consisted of 25 patients with prior RT-PCR-confirmed SARS-CoV-2 infection who were evaluated during the post-acute phase of the disease, 2-6 months after the acute infection. We recruited patients during outpatient follow-up visits and included both individuals who had previously required hospitalization and those managed outside the hospital setting during the acute phase of COVID-19.

We classified patients into the post-COVID-19 group based on persistent symptoms after recovery from acute infection. We did not use the severity of the initial acute COVID-19 episode as an inclusion criterion for this group and therefore did not consider it in subgroup analyses.

The most common persistent symptoms included dyspnea, fatigue, palpitations, and ongoing cardiopulmonary complaints suggestive of residual functional impairment after acute SARS-CoV-2 infection. We measured galectin-3 at outpatient evaluation using a standardized protocol for all post-COVID-19 participants.

We did not include recovered asymptomatic individuals as a comparison group.

We performed clinical and laboratory assessments in hospitalized patients within the first 24 hours after admission and throughout hospitalization. Laboratory investigations included complete blood count, coagulation profile, CRP, procalcitonin, D-dimer, ferritin, troponin, creatinine, eGFR calculated using the Cockcroft–Gault formula, and additional biomarkers when available according to routine clinical practice. We performed electrocardiography using the EDAN SE-12 system and echocardiography using the GE Vivid S5. We assessed clinical severity using MEWS and qSOFA scores.

Because paired measurements were limited for certain biomarkers, including direct bilirubin and IL-6, we interpreted analyses involving these variables as exploratory and with caution. We performed correlation analyses for these parameters only in participants with available paired measurements and did not include them in multivariable regression models.

We interpreted laboratory parameters according to the hospital laboratory's reference ranges. We

considered values above or below these reference intervals abnormal.

Statistical analysis

We performed statistical analyses using IBM SPSS Statistics software. We expressed continuous variables as mean $\pm$ standard deviation (SD), and categorical variables as frequencies and percentages. We assessed the distribution of continuous variables using the Shapiro–Wilk test. We analyzed normally distributed variables using parametric methods and non-normally distributed variables using appropriate non-parametric methods. We compared two groups using Student's t-test or the Mann–Whitney U test, as appropriate. We compared four study groups using one-way analysis of variance (ANOVA) followed by post-hoc pairwise comparisons. When assumptions for parametric testing were not met, we used the Kruskal–Wallis test.

We compared categorical variables using the chi-square test or Fisher's exact test when appropriate. We assessed correlations between Galectin-3 concentrations and clinical, laboratory, electrocardiographic, and echocardiographic parameters using Spearman's rank correlation coefficient. We handled missing data using available-case analysis; we did not impute missing values. Analyses involving variables with limited paired measurements, including IL-6 and direct bilirubin, were considered exploratory and interpreted with caution.

We compared categorical variables using the chi-square test or Fisher's exact test when appropriate. We assessed correlations between Galectin-3 concentrations and clinical, laboratory, electrocardiographic, and echocardiographic parameters using Spearman's rank correlation coefficient. We handled missing data using available-case analysis; we did not impute missing values. Analyses involving variables with limited paired measurements, including IL-6 and direct bilirubin, were considered exploratory and interpreted with caution.

We performed multivariable linear regression with Galectin-3 concentration as the dependent variable to assess whether Galectin-3 remained independently associated with clinical characteristics after adjustment for predefined confounders. The model

included age, sex, BMI, estimated glomerular filtration rate (GFR), arterial hypertension, coronary artery disease, heart failure, respiratory rate, oxygen SpO₂, C-reactive protein (CRP), smoking status, vaccination status, and COVID-19 severity category. We assessed regression assumptions, including linearity, residual distribution, and the absence of multicollinearity, before interpreting the model. We considered a two-tailed P value <0.05 statistically significant.

Ethical considerations

The Ethics Committee of Tbilisi State Medical University approved the study protocol (Approval No. 2022/97). We obtained written informed consent

from all participants before enrollment and blood sampling. The study followed the Declaration of Helsinki.

RESULTS

Galectin-3 levels increased progressively across acute COVID-19 severity categories, from moderate to severe and critical disease. In contrast, lower Galectin-3 concentrations were observed during the post-COVID phase. The total study population included 86 patients: 41 females (47.6%) and 45 males (52.4%). This classification served as the basis for assessing demographic, clinical, laboratory, and echocardiographic parameters (**TAB.1**)

TABLE 1. Clinical characteristic of patients

Continuous Variables	Group 1	Group 2	Group 3	Group 4
	Moderate	Severe	Critical	Post-COVID-19
Age in years, Mean±SD	72.2±11.7	77.1±10.2	78.2±10.4	71.8±16.8
BMI (kg/sm ²), Mean±SD	26.1±4.4	28.7±5.0	28.4±6.2	25.8±3.8
Systolic BP in mmHg, Mean±SD	133.9±20.2	137.1±14.5	137.8±24.9	127.6±18.8
Diastolic BP in mmHg, Mean±SD	79.2±12.7	78.4±11.1	78.0±18.3	79.5±12.8
Galectin-3 in ng/mL, Mean±SD	8.7±6.2	13.7±7.9	18.1±7.3	8.5±5.9
Saturation at rest in %, Mean±SD	93.4±1.4	89.4±1.5	85.2±5.1	90.2±5.1

The mean age was high across all groups, ranging from 71.8±16.8 to 78.2±10.4 years, with no statistically significant differences between groups. This corresponds to an elderly population, which is particularly vulnerable to severe COVID-19 and

increased mortality. 6 Mean BMI ranged from 25.8±3.8 kg/m² in the post-COVID group to 28.7±5.0 kg/m² in the severe group. A statistically significant difference was observed only between the severe and post-COVID groups (P<0.05) (**TAB.2**).

TABLE 2. Inter-group comparison of age, BMI, blood pressure, and oxygen saturation with corresponding P-values

Continous variables	Group 1 vs. Group 2		Group 1 vs. Group 3		Group 1 vs. Group 4		Group 2 vs. Group 3		Group 2 vs. Group 4		Group 3 vs. Group 4	
	t-test	P	t-test	P	t-test	P	t-test	P	t-test	P	t-test	P
Age (Years)	1.34	0.188	1.732	0.091	0.092	0.927	0.314	0.755	1.196	0.238	1.489	0.144
BMI (kg/sm ²)	1.77	0.085	1.375	0.177	0.248	0.805	0.168	0.867	2.212	0.032	1.732	0.091
Systolic BP (mmHg)	0.579	0.566	0.552	0.584	1.094	0.28	0.108	0.914	1.855	0.071	1.566	0.125
Diastolic BP (mmHg)	0.214	0.831	0.245	0.808	0.079	0.937	0.084	0.934	0.304	0.263	0.323	0.748
Galectin-3 (ng/ml)	2.261	0.029	4.452	<0.001	0.112	0.911	1.829	0.075	2.528	0.015	4.881	<0.001
Saturation at rest (%)	8.832	<0.001	7.097	<0.001	2.784	0.008	3.533	0.001	0.693	0.492	3,268	0.002

Cardiovascular risk factors were common across the cohort. Smoking prevalence ranged from 40.0% to 55.0%, coronary artery disease from 16.0% to 45.0%, and positive family history of cardiovascular disease from 23.8% to 50.0%. Overall, the study population

demonstrated a high-risk clinical profile characterized by advanced age, overweight/obesity, and pronounced hypoxemia during the acute phase of COVID-19 (**TAB.1**).

In the laboratory analysis, Galectin-3 levels increased progressively with disease severity: 8.7 ± 6.2 ng/mL in the moderate group, 13.7 ± 7.9 ng/mL in the severe group ($P=0.029$), and 18.1 ± 7.3 ng/mL in critically ill patients. In contrast, Galectin-3 levels decreased to 8.5 ± 5.9 ng/mL during the post-COVID period. The differences were particularly significant between the moderate and severe groups, as well as between the moderate and critical groups, indicating a strong association between Galectin-3 levels and COVID-19 severity. These findings suggest that Galectin-3 may have prognostic value in assessing disease progression and clinical outcomes.

Hematological and biochemical findings

Complete blood count

White blood cell count (WBC, N: $4.0-10.0 \times 10^9/L$): Leukocytosis was not observed regardless of disease severity, consistent with the known effects of viral infections on leukocyte dynamics. ⁷ Mean WBC counts increased progressively from the moderate (5.9 ± 2.5

$\times 10^9/L$) to the post-COVID group ($10.0 \pm 8.9 \times 10^9/L$) (**TAB.3**). Significant differences were observed between the moderate and critical groups ($P_{1-3}=0.023$) and between the moderate and post-COVID groups ($P_{1-4}=0.047$).

Hemoglobin (HGB) and hematocrit (HCT): Mean hemoglobin and hematocrit values did not differ significantly between groups ($P>0.05$).

Platelet count (PL) and plateletcrit (PCT): Mean platelet counts did not differ significantly between groups, although thrombocytopenia was observed in individual patients, consistent with previous reports. ⁷ Significant differences in both PL and PCT were found between the critical and post-COVID-19 groups ($P_{3-4}=0.033$ and $P_{3-4}=0.032$, respectively).

Coagulation parameters

Prothrombin time (PT, N: 11–15 s): No significant differences were observed between groups. PT tended to be prolonged in the post-COVID group, suggesting persistent alterations in coagulation and liver function. ⁸

TABLE 3. Clinical characteristic of patients

Parameters	Group 1 (N=21)		Group 2 (N=20)		Group 3 (N=20)		Group 4 (N=25)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Complete blood count								
WBC, $\times 10^9/L$	5.9	2.5	6.8	2.9	8.7	4.8	10.0	8.9
LYMPH, $\times 10^9/L$	17.6	7.0	21.3	17.6	14.0	7.5	18.7	12.3
NEU, $\times 10^9/L$	73.6	10.0	72.4	17.7	72.9	21.4	74.2	14.8
HGB, g/dL	12.8	1.7	13.2	2.4	13.2	2.6	12.7	2.3
HCT, %	38.8	4.7	40.5	7.1	39.9	7.8	38.4	7.2
PLT, $\times 10^9/L$	199.4	57.9	203.8	65.7	176.8	67.2	221.9	69.3
PCT, ng/mL	0.17	0.04	0.19	0.05	0.16	0.06	0.20	0.06
Coagulogram								
APTT, sec	33.0	9.3	36.3	10.8	33.9	6.4	32.8	6.9
PT, sec	13.9	3.9	14.5	4.1	13.4	1.6	15.7	10.2
INR	1.21	0.32	1.27	0.32	1.18	0.22	1.27	0.64
PL, $\times 10^9/L$	82.8	18.4	77.3	16.7	82.2	18.3	83.1	25.3
FIB, g/L	3.5	0.8	4.5	1.2	4.2	0.9	4.1	1.2
C-reactive protein, mg/L	35.7	42.9	87.3	90.1	69.4	73.9	81.8	96.1
Creatinine, mg/L	83.5	26.5	105.5	39.8	113.5	64.4	117.9	104.0
GFR, mL/min/1.73 m ²	76.6	21.2	62.4	24.0	62.4	22.2	74.4	39.3
Direct Bilirubin, mg/dL	4.8	0.2	3.7	1.6	4.1	1.3	5.0	0.1

Abbreviations: ALB, albumin; APTT, activated partial thromboplastin time; CR, creatinine; CRP, C-reactive protein; Dir. Bilirubin, direct bilirubin; FIB, fibrinogen; GFR, glomerular filtration rate; HCT, hematocrit; HGB, hemoglobin; INR, international normalized ratio; LYMPH, lymphocytes; NEU, neutrophils; PLT, platelets; PCT, plateletcrit; PT, prothrombin time; WBC, white blood cell count.

Procalcitonin (PCT, N: 0.05–0.10 ng/mL)

Critically ill patients with secondary bacterial infections had markedly elevated procalcitonin levels, whereas the severe and post-COVID groups had substantially lower values. Significant differences were observed between the severe and critical groups and between the critical and post-COVID groups (both $P=0.001$).⁹

Direct bilirubin

Mean direct bilirubin levels did not differ significantly between groups; however, some severe and critical patients had elevated values. Significant differences were found between the moderate and severe ($P_{1-2}=0.003$), moderate and critical ($P_{1-3}=0.019$), moderate and post-COVID-19 ($P_{1-4}=0.001$), severe and post-COVID-19 ($P_{2-4}=0.001$), and critical and post-COVID-19 groups ($P_{3-4}=0.001$).¹⁰

Lactate dehydrogenase (LDH, N: 140–280 U/L)

LDH levels were elevated in severe disease and particularly pronounced in critically ill patients. A significant difference was observed between the moderate and severe groups ($P=0.028$). Concurrent elevations of fibrinogen, CRP, LDH, and direct bilirubin may reflect multisystem involvement, including hepatic dysfunction.

Renal function parameters

Serum creatinine (N: 44–115 $\mu\text{mol/L}$): Creatinine concentrations were higher in severe disease, with a significant difference between the moderate and severe groups ($P_{1-2}=0.043$).

Estimated Glomerular Filtration Rate (eGFR, N: >90 mL/min): Mean eGFR values were below the normal range in all groups, indicating impaired renal function. We observed a significant difference between the moderate and critical groups ($P_{1-3}=0.043$). Persistent reduction in eGFR during the post-COVID period may be associated with Long COVID-related fibrotic changes.¹¹

As described in the Methods section, we stratified patients according to the Chinese COVID-19 Clinical Guidelines.¹² Within this classification system, oxygen saturation was one of the most important parameters. Accordingly, oxygen saturation values were markedly lower in the severe and critical disease groups. Published evidence indicates that decreased oxygen saturation (pulse oximetry $\text{SpO}_2 < 94\%$) reflects

hypoxemia and is a key clinical indicator of the need for hospitalization or intensive care unit (ICU) admission.¹³⁻¹⁵ As established, COVID-19-associated viral pneumonia impairs pulmonary gas exchange, reducing oxygen saturation. During this phase, inflammatory biomarkers play a particularly important role in clinical assessment and prognostication. In this context, Galectin-3 is distinguished by its dual biological role, functioning as both an inflammatory marker and an indicator of fibrotic processes.¹⁶ Since the patients included in our study were enrolled within the first week following disease onset and the maximum duration of hospitalization did not exceed three weeks, this period was defined as the acute phase of COVID-19. In contrast, we evaluated patients in the fourth group at least two months after acute infection. They were therefore classified as post-COVID patients, including individuals presenting with manifestations of prolonged (Long) COVID. Accordingly, the observed correlations between Galectin-3 and established markers of disease severity during the acute phase, including lactate dehydrogenase (LDH), C-reactive protein (CRP), and fibrinogen, support its role as an inflammatory biomarker in COVID-19.⁹ These findings are consistent with the proposed involvement of Galectin-3 in regulating inflammatory pathways and immune responses during SARS-CoV-2 infection.

We observed no significant correlations between Galectin-3 and demographic characteristics within individual groups. However, in the overall cohort, Galectin-3 showed significant positive correlations with age ($r=0.2230$, $p=0.039$), body weight ($r=0.2737$, $p=0.011$), BMI ($r=0.3525$, $p=0.001$), pulse rate ($r=0.2813$, $p=0.009$), respiratory rate ($r=0.4121$, $p<0.001$), and body temperature ($r=0.2486$, $p=0.021$), while a negative correlation was observed with oxygen saturation ($r=-0.3437$, $p=0.001$). These findings are consistent with previous studies linking elevated Galectin-3 to disease severity and systemic inflammation in COVID-19. We found no significant associations with vaccination status, blood pressure, or heart rate. Galectin-3 positively correlated with white blood cell count in the overall cohort ($r=0.2441$, $p=0.024$) and in the post-COVID group ($r=0.4015$, $p=0.047$).

We identified significant negative correlations between Galectin-3 and hemoglobin ($r=-0.5857$, $p=0.005$) and hematocrit ($r=-0.5523$, $p=0.009$) only in

the moderate disease group. Although severe COVID-19 has been associated with anemia-related mechanisms, our data did not support clinically significant hemolysis, as erythrocyte counts and hemoglobin levels remained largely within normal ranges. Biochemical analysis demonstrated positive

correlations between Galectin-3 and inflammatory markers, including IL-6 ($r=1.000$, $p<0.001$), ferritin ($r=0.3085$, $p=0.004$), and LDH ($r=0.2455$, $p=0.023$), supporting its role as an inflammatory biomarker (TAB.4).

TABLE 4. Correlations between Galectin-3 demographic characteristics and hematological parameters in the entire study cohort

	r	P
Sex	-0.0258	0.814
Age, years	0.2230	0.039
Weight, kg	0.2737	0.011
Height, cm	-0.0379	0.729
BMI, kg/m ²	0.3391	0.001
BSA, m ²	0.1930	0.075
Vaccination status	-0.0705	0.519
Pulse Rate, bpm	0.2813	0.009
Systolic blood pressure, mmHg	-0.1451	0.183
Diastolic blood pressure, mmHg	-0.2579	0.017
Respiratory rate, per min	0.4121	0.000
Oxygen saturation at rest, %	-0.3437	0.001
Temperature, °C	0.2486	0.021
WBC, $\times 10^9/L$	0.2441	0.024
LYMPH, $\times 10^9/L$	-0.1172	0.283
NEU, $\times 10^9/L$	0.0075	0.945
HGB, g/dL	-0.0573	0.600
HCT, %	-0.0377	0.730
PLT, $\times 10^9/L$	-0.1311	0.229
PCT, ng/mL	-0.0729	0.505
APTT, sec	0.1577	0.147
PT, sec	0.2091	0.053
INR	0.2343	0.030
PL, $\times 10^9/L$	-0.2616	0.015
FIB, g/L	0.2004	0.064

Abbreviations: APTT, activated partial thromboplastin time; BMI, body mass index; BSA, body surface area; FIB, fibrinogen; HCT, hematocrit; HGB, hemoglobin; INR, international normalized ratio; LYMPH, lymphocytes; NEU, neutrophils; PLT, platelets; PCT, plateletcrit; PT, prothrombin time; WBC, white blood cell count.

Interleukin-6 measurements were available only in a limited number of participants ($n=3$). Although we observed a strong positive correlation between Galectin-3 and IL-6 in these cases, the extremely small number of paired observations precluded reliable statistical interpretation. Therefore, we consider this finding exploratory and did not include it among the study's principal conclusions. Exploratory analyses suggested possible associations between Galectin-3 and direct bilirubin in several study groups. However,

direct bilirubin measurements were available for only a limited number of participants; therefore, these findings should be interpreted cautiously and considered hypothesis-generating rather than confirmatory.

The association between Galectin-3 and direct bilirubin may reflect ongoing hepatocyte and cholangiocyte injury. SARS-CoV-2 utilizes ACE2 receptors expressed in hepatic and biliary cells, promoting oxidative stress and inflammation that may

shift tissue repair toward fibrosis.^{10,17,18} The persistence of this relationship, regardless of disease severity, supports the hypothesis of chronic low-grade inflammation and sustained macrophage activation during the post-COVID period (TAB.4).^{4,9,20}

Previous studies have suggested potential associations between Galectin-3 and markers of hepatic injury and fibrosis. However, the limited number of direct bilirubin measurements in our cohort precludes firm conclusions regarding hepatic involvement in the present study.^{21,22} Increased Galectin-3 expression has also been associated with cholestatic liver injury, bile duct proliferation, and fibrogenesis.¹⁰ Furthermore, Galectin-3 has been linked to biomarkers of organ injury, including D-dimer, CRP, urea, and LDH.²³ In our cohort, elevated Galectin-3 levels were accompanied by increased LDH concentrations, while CRP and D-dimer were elevated across all groups. The coexistence of elevated Galectin-3, direct bilirubin, and LDH may reflect systemic hyperinflammation, hepatocellular injury, impaired bile transport, and tissue hypoxia.⁹ Previous studies have also reported associations between Galectin-3, ferritin, and fibrinogen,^{3,24} supporting a potential role of hepatic involvement in post-COVID fibrogenesis.

These findings may also have cardiovascular implications, as Galectin-3 is a recognized biomarker of myocardial fibrosis and heart failure. Persistent inflammation and oxidative stress may contribute to myocardial injury, cardiac remodeling, and an increased risk of heart failure in post-COVID patients.^{18,25} No significant correlations between Galectin-3 and coagulation parameters were observed in the acute COVID-19 groups. However, in the overall cohort, Galectin-3 correlated positively with INR ($r=0.2343$, $p=0.030$). In the post-COVID group, it also showed significant positive correlations with prothrombin time ($r=0.4901$, $p=0.013$) and INR ($r=0.4217$, $p=0.036$). Elevated Galectin-3 levels have been associated with structural cardiac remodeling, electrocardiographic abnormalities, and increased arrhythmogenic risk.^{4,26}

ECG and Galectin-3 correlations

Our findings showed a negative correlation between Galectin-3 and P-wave duration ($r=-0.5535$, $p=0.009$) and a positive correlation with QTc interval ($r=0.5440$, $p=0.011$).

Correlation analysis between Galectin-3 and electrocardiographic parameters demonstrated, in the overall cohort, a significant association with P-wave duration ($r=-0.4367$, $p<0.001$) and T-wave duration ($r=0.3305$, $p=0.002$) (TAB.5). In the first group, Galectin-3 correlated with P-wave duration ($r=-0.5535$, $p=0.009$) and QTc interval ($r=0.5440$, $p=0.011$). In the second group, Galectin-3 correlated with P-wave duration ($r=-0.5031$, $p=0.024$) and P-R interval ($r=-0.4643$, $p=0.039$). In the third and fourth groups, we observed no statistically significant correlations between Galectin-3 and electrocardiographic parameters (TAB.5).

TABLE 5. Correlations between Galectin-3 and electrocardiographic parameters in the entire study cohort

	r	p
Heart rate, bpm	0.0359	0.743
P wave, ms	-0.4367	0.000
PR segment, ms	-0.2757	0.010
QRS complex, ms	-0.1565	0.150
QT interval, ms	0.0490	0.654
QTc, ms	0.2100	0.052
T wave, ms	0.3305	0.002
ST segment, ms	0.1506	0.166

ECG parameters: Heart rate (HR, N=60–100 beats/min): Across severity-stratified groups, ECG parameters were as follows: HR was 78.4 ± 15.2 beats/min in the first group, 78.4 ± 13.6 beats/min in the second group, 81.6 ± 8.4 beats/min in the third group, and 80.6 ± 11.5 beats/min in the fourth group. No statistically significant differences in heart rate were observed between groups ($P>0.05$).

P-wave duration ($N<100.0$ ms): P-wave duration was 100.3 ± 37.8 ms in the first group, 90.8 ± 45.0 ms in the second group, 91.4 ± 37.0 ms in the third group, and 105.8 ± 31.3 ms in the fourth group. No statistically significant differences in P-wave duration were observed between groups, although the fourth group had a higher mean value.

QT interval: The QT interval was 396.1 ± 34.2 ms in the first group, 401.1 ± 39.6 ms in the second group, 394.5 ± 56.2 ms in the third group, and 373.7 ± 39.0 ms in the fourth group. We observed statistically significant differences between the second and third groups ($P_{2-3}=0.046$) and between the second and

fourth groups ($P_{2-4}=0.025$). No other statistically significant differences were identified.

Corrected QT interval (QTc): Based on the Bazett formula and accounting for sex differences, mean QTc values did not exceed clinically accepted thresholds in any group. QTc values were 434.4 ± 27.1 ms in the first group, 446.3 ± 26.9 ms in the second group, 445.6 ± 25.4 ms in the third group, and 421.6 ± 25.9 ms in the fourth group. Statistically significant differences were observed between the second and fourth groups ($P_{2-4}=0.003$), and between the third and fourth groups ($P_{3-4}=0.003$). No other statistically significant differences were observed, although mean values remained within the normal range.

DISCUSSION

The literature links atrial fibrillation in COVID-19 patients to Galectin-3-mediated atrial tissue fibrosis. This causes P-wave deformation and prolongation and increases the risk of atrial fibrillation development and recurrence following catheter ablation.^{5,27,28} Conduction system abnormalities may also occur, as myocardial interstitial fibrosis impairs normal electrical impulse propagation, which may manifest on ECG as bundle branch blocks (LBBB or RBBB) or atrioventricular (AV) blocks.^{4,5,29}

We used multivariable linear regression to determine whether Galectin-3 concentration remained independently associated with COVID-19 severity after adjusting for potential confounders. The model included age, sex, BMI, estimated glomerular filtration rate (GFR), arterial hypertension, coronary artery disease, heart failure, respiratory rate, oxygen saturation, C-reactive protein, smoking status, vaccination status, and COVID-19 severity category.

The overall regression model was statistically significant ($F(15,70)=4.89$, $p<0.001$) and explained 51.2% of the variance in Galectin-3 concentrations ($R^2=0.512$; adjusted $R^2=0.407$). After adjustment for these covariates, selected cardiovascular factors remained independently associated with Galectin-3 levels, while the association between COVID-19 severity categories and Galectin-3 concentration was attenuated after controlling for baseline characteristics.

Multivariable linear regression analysis demonstrated that arterial hypertension ($\beta=5.94$, $p=0.024$) and heart failure ($\beta=4.88$, $p=0.012$) were independently associated with higher Galectin-3 concentrations. Critical disease severity showed

borderline statistical significance ($\beta=5.02$, $p=0.062$). No significant associations were observed for sex ($\beta=-4.02$, $p=0.132$), age ($\beta=-0.01$, $p=0.888$), BMI ($\beta=0.24$, $p=0.129$), respiratory rate ($\beta=-0.28$, $p=0.341$), resting oxygen saturation ($\beta=-0.38$, $p=0.146$), C-reactive protein ($\beta=0.007$, $p=0.484$), estimated glomerular filtration rate (eGFR) ($\beta=0.01$, $p=0.711$), coronary artery disease ($\beta=1.99$, $p=0.324$), severe disease ($\beta=1.93$, $p=0.407$), post-COVID-19 status ($\beta=-1.99$, $p=0.325$), smoking ($\beta=-0.43$, $p=0.876$), or vaccination status ($\beta=-1.01$, $p=0.465$). The overall regression model was statistically significant ($F(15,70)=4.89$, $p<0.001$) and explained 51.2% of the variance in Galectin-3 concentrations ($R^2=0.512$; adjusted $R^2=0.407$).

Arterial hypertension remained independently associated with higher Galectin-3 concentrations ($\beta=5.94$, 95% CI 0.79–11.09, $p=0.024$), as did heart failure ($\beta=4.88$, 95% CI 1.11–8.66, $p=0.012$). Critical COVID-19 showed borderline statistical significance ($\beta=5.02$, 95% CI -0.27 to 10.32, $p=0.062$).

Based on hemogram analysis, we did not stratify disease severity in hospitalized patients using variations in formed blood elements, and we found no significant correlations with Galectin-3. Galectin-3, an inflammatory marker in acute COVID-19, correlates with other inflammatory markers, including leukocytes and LDH.³ No consistent correlation was observed between Galectin-3 and thrombosis-related markers, except for plateletcrit, which represents an indicator of COVID-19 severity, thrombosis risk, and mortality.⁸ Galectin-3 reflects multi-organ involvement associated with disease severity, including pulmonary impairment (oxygen desaturation) and renal dysfunction (reduced GFR). Exploratory analyses showed associations with bilirubin, but these should be interpreted cautiously because bilirubin measurements were available in only a limited number of patients.²³ In the post-COVID-19 group, these findings may be compatible with persistent inflammatory or tissue remodeling processes; however, further studies are required to clarify their relationship with fibrosis.¹⁶ Additionally, we observed exploratory associations between Galectin-3 and selected ECG parameters, including P-wave duration, T-wave duration, and QTc interval ($r=-0.4367$, $p<0.001$; $r=0.3305$, $p=0.002$; $r=-0.5535$, $p=0.009$; and $r=0.5440$, $p=0.011$ in the respective analyses). These findings may reflect altered

electrophysiological profiles in patients with higher Galectin-3 concentrations; however, they do not establish structural cardiac remodeling, arrhythmogenic risk, or fibrosis without confirmatory imaging or longitudinal follow-up data.⁵

Interleukin-6 measurements were available only in a very limited number of participants (n=3). Although we observed a strong correlation with Galectin-3, the extremely small number of paired observations precluded reliable statistical interpretation. Therefore, we considered this finding exploratory and did not include it among the principal study conclusions.

These findings suggest that elevated Galectin-3 levels in COVID-19 patients reflect a complex interaction between acute inflammatory activation, disease severity, and underlying cardiovascular comorbidities. Therefore, Galectin-3 should be interpreted as a marker of systemic inflammatory and fibrotic activity rather than an isolated indicator of COVID-19 severity.

Given the large number of comparisons, some statistically significant findings may represent chance associations and should be interpreted cautiously.

Multivariable regression analysis showed that several clinical confounders influence the relationship between Galectin-3 concentration and COVID-19 severity. After adjustment for demographic characteristics, metabolic status, renal function, cardiovascular comorbidities, inflammatory markers, smoking status, vaccination status, and disease severity category, Galectin-3 remained associated with patients' overall clinical profile. These findings emphasize that Galectin-3 reflects a combination of inflammatory activation, tissue remodeling, and underlying cardiovascular vulnerability rather than representing a single disease-specific marker. Because we performed multiple exploratory analyses, some statistically significant findings may reflect chance associations. Therefore, interpret these findings cautiously and validate them in larger prospective studies.

Study limitations

Several limitations warrant acknowledgment. First, this single-center observational study had a relatively small sample size, which may limit statistical power and the generalizability of the findings to broader populations. Second, although the post-COVID-19

group provided important information regarding persistent alterations after SARS-CoV-2 infection, its composition may have introduced selection bias because participants were recruited during post-acute follow-up and represented individuals with persistent symptoms rather than a randomly selected population of recovered patients. Furthermore, initial acute COVID-19 severity data were not uniformly available among post-COVID-19 participants, limiting direct comparisons between acute disease severity and long-term biomarker alterations.

The cross-sectional nature of most analyses prevents establishing causal relationships between elevated Galectin-3 concentrations and clinical, laboratory, or electrocardiographic abnormalities. The absence of a healthy control group and a matched non-COVID hospitalized control group limits the ability to determine whether observed Galectin-3 alterations are specific to SARS-CoV-2 infection or reflect general responses to systemic inflammation, hospitalization, or underlying comorbid conditions.

Another limitation is the number of statistical comparisons, which increases the risk of type I error. Although we predefined clinically relevant analyses, we did not adjust all comparisons for multiple testing. In addition, sample size and the availability of complete clinical variables limited multivariable adjustment, and we cannot exclude residual confounding. Missing data, particularly for biomarkers measured only in a subset of participants, may have introduced additional bias despite available-case analysis.

Although Galectin-3 is considered a biomarker associated with fibrosis-related pathways, we did not directly assess myocardial or tissue fibrosis using cardiac magnetic resonance imaging, extracellular volume quantification, or histological methods. Therefore, correlations between Galectin-3 levels and ECG parameters should not be interpreted as direct evidence of structural cardiac remodeling or fibrosis. ECG abnormalities may reflect multiple mechanisms, including inflammation, metabolic disturbances, medication effects, or transient electrophysiological changes.

Furthermore, Galectin-3 concentrations may be influenced by several non-COVID-related factors, including age, renal function, obesity, arterial hypertension, coronary artery disease, and other cardiovascular conditions. Although we included

several potential confounders in the regression analyses, residual confounding remains possible. We did not fully control for COVID-19-specific treatments, including corticosteroids, anticoagulants, antiviral agents, and antibiotics, which may have influenced inflammatory responses and biomarker levels.

The study population was heterogeneous in SARS-CoV-2 variant circulation, vaccination status, and treatment strategies across pandemic phases, which may have affected disease presentation and biomarker expression. Finally, because of the single-center design, limited sample size, and specific characteristics of the studied population, interpret the findings cautiously and validate them in larger, multicenter prospective studies with appropriate control groups and longitudinal follow-up.

CONCLUSIONS

Galectin-3 levels increased progressively with COVID-19 severity and were highest among critically ill patients, supporting its potential role as a marker of disease progression. Significant correlations between Galectin-3 and inflammatory biomarkers, including leukocyte count, IL-6, ferritin, and LDH, confirm its association with systemic inflammatory activity. Associations with creatinine, eGFR, prothrombin time, and INR suggest that Galectin-3 may reflect multi-organ involvement affecting renal and coagulation pathways. We also observed exploratory associations with direct bilirubin, but these require confirmation in larger cohorts with more complete liver function data. The observed relationship between Galectin-3 and oxygen desaturation further supports its association with pulmonary impairment and disease severity. In addition, significant correlations between Galectin-3 and electrocardiographic parameters, including P-wave duration, PR interval, T-wave duration, and QTc interval, indicate potential cardiac structural and electrical remodeling. Persistent associations observed during the post-COVID period may reflect ongoing inflammatory and fibrotic processes and suggest a possible role of Galectin-3 in Long COVID pathophysiology.

Overall, Galectin-3 may serve as a valuable biomarker for risk stratification, assessment of multi-organ involvement, and identification of patients at increased risk of cardiovascular and fibrotic complications during both acute COVID-19 and the post-COVID period.

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