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Galectin-3 and the Right Side of Heart Echocardiographic Changes in COVID-19 Patients

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ABSTRACT

Background: Existing literature on COVID-19 infection, however, has not thoroughly investigated the combined use of Galectin-3 with echocardiographic parameters to assess cardiovascular complications in COVID-19.

Objectives: To evaluate the relationship between right-heart echocardiographic changes and serum Galectin-3 (Gal-3) levels in hospitalized patients during different phases of COVID-19 infection.

Methods: A total of 86 hospitalized patients of both sexes were enrolled and divided into four groups according to disease severity and phase: (i) Group I: Patients with moderately expressed symptoms (within 1 week of symptom onset) — 21 patients; (ii) Group II: Patients with severe symptoms (weeks 2–3 after symptom onset) — 20 patients; (iii) Group III: Patients in the critical phase — 20 patients; (iv) Group IV: Post-COVID-19 patients — 25 patients. All patients underwent standard laboratory evaluation at admission, including measurement of Galectin-3 using an ELISA kit (Human Galectin-3 "SUNLONG"), and transthoracic echocardiography performed with a GE Healthcare Vivid S5 CV ultrasound system, in accordance with COVID-19 clinical guidelines.

Results: Serum Galectin-3 levels and echocardiographic parameters reflected the extent of cardiovascular involvement in relation to COVID-19 severity. COVID-19 was associated with vascular injury and right-heart remodeling, manifested by right ventricular (RV) dilation, right atrial (RA) enlargement, decreased TAPSE values, and increased pulmonary artery systolic pressure (PASP), indicating right-ventricular dysfunction and elevated pulmonary pressures.

In the early acute phase among moderately ill patients, Gal-3 demonstrated positive correlations with RV size, RA size, tricuspid regurgitation rate (TRR), and RV function parameters. In severely ill patients, Gal-3 lost its association with RV structural changes, while maintaining correlations with RA size, TRR, and RV functional indices, and showed a significant association with PASP elevation. Despite markedly elevated Gal-3 levels in critically ill patients—reflecting disease severity—no significant correlations were observed between Gal-3 and right-heart structural parameters. In post-COVID-19 patients, Gal-3 appeared to play a different role, acting as an indicator of residual right-heart structural and functional alterations, as demonstrated by its correlation with RA size, TRR, and PASP.

Conclusions: Changes in Galectin-3 levels and echocardiographic parameters, as well as their interrelationships, reflect right-heart involvement, disease severity, and prognosis in COVID-19 patients. The pattern of correlation between Gal-3 and right-heart echocardiographic parameters varies across different phases of COVID-19, suggesting a potential role of Gal-3 in cardiac structural remodeling.

Keywords: COVID-19; fibrosis; galectin-3.

BACKGROUND

Atherosclerotic diseases represent a major contributor to global mortality, and their significance has become even more pronounced during the COVID-19 pandemic. Evidence suggests that cardiovascular complications in COVID-19 may arise both through direct myocardial injury and secondary effects of pulmonary dysfunction. Mortality is particularly high in patients with pre-existing cardiovascular disease, although cardiac complications may also occur in individuals without prior cardiac history. The lungs and heart are the two principal organs involved in COVID-19-related morbidity and mortality. COVID-19 is now recognized as a multisystem disease characterized by systemic inflammation, endothelial dysfunction, thrombotic complications, and multiorgan involvement.

According to the WHO COVID-19 dashboard, from 26 January to 1 February 2026, a total of 60,072 SARS-CoV-2 samples were tested across 86 countries. World Health Organization (WHO) collects this data from a global network of sentinel and systematic virologic surveillance sites. Out of these, 2,368 (3.9%) samples tested positive for the virus. During this period, SARS-CoV-2 activity was stable globally, with variations observed across some WHO regions. Elevated activity or increases were reported in countries in Temperate South America, Northern and South West Europe, Western Africa, and Eastern Asia.[1]

In this setting, biomarkers reflecting endothelial injury and fibrosis—including Galectin-3—have gained increasing attention. Recent research has identified Galectin-3 as an important biomarker of inflammation and fibrosis. Galectin-3 (Gal-3), a β-galactoside-binding lectin, plays a significant role in immune activation, oxidative stress, fibrosis, and cardiovascular remodeling. According to the ARIC study, elevated plasma Galectin-3 levels are strongly associated with inflammatory activity and tissue remodeling processes.[2]

Galectin-3 (Gal-3) has emerged as a significant biomarker in COVID-19, particularly as a predictor of disease severity, ICU mortality, and the development of acute respiratory distress syndrome (ARDS). Studies have shown elevated Gal-3 levels in critically ill patients. Recent research indicates that Gal-3, a key marker of fibrosis and inflammation produced by macrophages, acts as an independent risk factor for in-hospital mortality.[3] Elevated serum Galectin-3 concentrations have been associated with severe disease progression, increased inflammatory burden, and endothelial dysfunction. Given its established role in cardiovascular pathology, Galectin-3 may represent a pathophysiological link between COVID-19-related inflammation and cardiac remodeling, making it a promising biomarker for risk stratification and post-COVID-19 cardiac monitoring.

Galectin-3 promotes the progression of intimal hyperplasia by affecting endothelial cell function through various biological pathways. Additionally, it plays a role in the enlargement of the medial layer and the thickening of the adventitia by stimulating vascular smooth muscle cells and adventitial fibroblasts. In pulmonary hypertension, galectin-3 contributes to right ventricular remodeling by activating cardiac fibroblasts and reducing cardiomyocyte contractile performance. Beyond structural changes, galectin-3 also regulates associated inflammatory responses. This review discusses the molecular pathways underlying galectin-3 activity in pulmonary hypertension and assesses the therapeutic potential of its inhibition. Although targeting galectin-3 offers a promising and innovative approach to treating pulmonary hypertension, important limitations remain, including concerns about drug selectivity and the lack of demonstrated clinical effectiveness.[4]

Right ventricular involvement has been observed more commonly than left ventricular involvement in patients with COVID-19, with approximately 40% of patients experiencing RV dilatation and RV dysfunction. RV damage is associated with a higher incidence of myocardial damage in COVID-19 and generally predicts a worse prognosis.[5] COVID-19 often causes right ventricular involvement, and this dysfunction is associated with worse clinical outcomes.

Galectin-3 has been found to reflect the echocardiographic quantification of right ventricular failure. Serum levels of Gal-3 correlate with key echocardiographic parameters, specifically right atrial area and tricuspid annular plane systolic excursion (TAPSE). Gal-3 acts as an indicator for right heart adaptations to increased pulmonary arterial pressure and volume and is thought to be released by RV myocardial macrophages during the fibrotic remodeling process. Patients with Gal-3 levels ≥17.35 ng/mL are 3.6-times more likely to develop right ventricular failure, making Gal-3 a potential tool for monitoring right heart strain in COVID-19 cases.[4]

Echocardiography remains a key diagnostic tool in this context, with both focused and point-of-care approaches recommended by international guidelines. Studies have demonstrated that elevated Galectin-3 levels correlate with structural and functional cardiac abnormalities, particularly right-ventricular dysfunction associated with increased pulmonary vascular resistance and systemic inflammation. These findings suggest that galectin-3 could be a valuable prognostic biomarker for severe COVID-19 outcomes and require further studies.[6]

METHODS

Study cohort

This study was conducted at the First University Clinic of Tbilisi State Medical University. A total of 86 hospitalized patients with confirmed COVID-19 infection were enrolled. All participants were evaluated within 24 hours of hospital admission and were followed during their hospital stay.

Inclusion criteria

Inclusion criteria comprised hospitalized patients of both sexes aged ≥18 years with COVID-19 confirmed by reverse transcription polymerase chain reaction (RT-PCR) from nasopharyngeal or oropharyngeal swabs.

Exclusion criteria

Age <18 years, poor echocardiographic image quality, type 1 or type 2 diabetes mellitus (except steroid-induced hyperglycemia), mechanical ventilation at the time of enrollment, pregnancy, ongoing dialysis therapy, and type 4 or type 5 myocardial infarction (procedure-related myocardial injury).

Classification of COVID-19 severity:

Disease severity was classified according to the Chinese COVID-19 clinical guideline, with an additional post-COVID-19 group added.[7] Patients were categorized into four groups as follows:

- Moderate disease group: Patients with mild or moderate pneumonia without severe clinical manifestations.
- Severe disease group: Patients presenting with at least one of the following: respiratory rate >30 breaths/min, oxygen saturation (SpO_2) ≤93% at rest, PaO_2/FiO_2 ≤300 mmHg, or lung infiltration >50% within 24–48 hours.
- Critical disease group: Patients with respiratory failure requiring mechanical ventilation, shock, or multiple organ dysfunction syndrome requiring intensive care.
- Post-COVID-19 group: Patients evaluated 3–6 months after acute infection.

According to this classification, study participants were distributed as follows:

- Group I (moderate disease): 21 patients (10 females, 47.6%; 11 males, 52.4%), evaluated within the first week after symptom onset.
- Group II (severe disease): 20 patients (9 females, 45.0%; 11 males, 55.0%), evaluated during weeks 2–3 of symptom onset.
- Group III (critical disease): 20 patients (8 females, 40.0%; 12 males, 60.0%).
- Group IV (post-COVID-19): 25 patients (14 females, 56.0%; 11 males, 44.0%).

The presence of cardiovascular disease in study participants was determined according to established international guidelines and recommendations.[8]

Echocardiographic methods and Galectin-3: Heart failure in hospitalized COVID-19 patients was assessed using American Heart Association criteria, including echocardiographic indicators, and functional classification was determined according to New York Heart Association guidelines.[9]

All patients underwent standard transthoracic echocardiography (TTE) in accordance with the American Society of Echocardiography (ASE) recommendations.[10] Measurements included right ventricular diameter (RVd), right atrial diameter (RAd), pulmonary artery systolic pressure (PASP), tricuspid annular plane systolic excursion (TAPSE), and right ventricular fractional area change (RVFAC), obtained using M-mode, two-dimensional imaging, and color Doppler techniques.[11]

Serum Galectin-3 levels were measured using a commercially available ELISA kit (Human Galectin-3, SUNLONG).

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics software. Continuous variables with normal distribution are

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presented as mean ± standard deviation (SD), while categorical variables are expressed as frequencies and percentages. Associations between echocardiographic parameters and Galectin-3 levels were assessed using Spearman's rank correlation coefficient. Logistic regression analysis was also performed. A p-value <0.05 was considered statistically significant.

Ethical approval

The study protocol was approved by the Research Ethics Committee of Tbilisi State Medical University. Due to the study's retrospective nature, informed consent was waived.

RESULTS

In our study, the mean age of Group 1 patients was 72.2±11.7 years; in Group 2: 77.1±10.2 years; in Group 3: 78.2±10.4 years, and in Group 4: 71.8±16.8 years. No statistically significant age difference was observed (Tab.1).

TABLE 1. Clinical characteristics of patients by groups

	Group 1	Group 2	Group 3	Group 4
Disease severity	Moderate	Severe	Critical	Postcovid
Number of patients	21	20	20	25
Age (Years), Mean±SD	72.2±11.7	77.1±10.2	78.2±10.4	71.8±16.8
BMI (kg/cm^2), Mean±SD	26.1±4.4	28.7±5.0	28.4±6.2	25.8±3.8
Systolic BP (mmHg), Mean±SD	133.9±20.2	137.1±14.6	137,8±24.9	127.6±18.8
Diastolic BP (mmHg), Mean±SD	79.2±12.7	78.4±11.1	78.0±18.3	79.5±12.8
Galectin-3 (ng/ml), Mean±SD	8.7±6.2	13.7±7.9	18.1±7.3	8.5±5.9
Saturation at rest (%), Mean±SD	93.4±1.4	89.4±1.5	85.2±5.1	90.2±5.1

Abbreviations: BMI, body mass index; BP, blood pressure; SD, standard deviation.

No statistically significant differences were observed in systolic or diastolic blood pressure among the study groups (P > 0.05) (Tab.1). Based on the mean systolic and diastolic values, hypertension was not detected, which may be explained by the fact that all patients were hospitalized and receiving continuous monitoring and antihypertensive treatment.

BMI in Group 1 was 26.1±4.4 kg/m^2; in Group 2:28.7±5.0 kg/m^2, in Group 3:28.4±6.2 kg/m^2; and Group 4:25.8±3.8 kg/m^2. A statistically significant difference was observed between Group 2 and Group 4, P<0.05 (Tab.2). Oxygen saturation was significantly lower in Group II compared with Group I (P=0.001). No statistically significant differences were observed between the remaining intergroup comparisons (P>0.05) (Tab.2).

TABLE 2. Pairwise comparisons of clinical characteristics among study groups

	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 or Chi2-test	P value	t-test or Chi2-test	P value	t-test or Chi2-test	P value	t-test or Chi2-test	P value	t-test or Chi2-test	P value	t-test or Chi2-test	P value
Age, years	1.340	0.188	1.732	0.091	0.092	0.927	0.314	0.755	1.196	0.238	1.489	0.144
BMI, kg/cm^2	1.770	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	0.280	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	0.763	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

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Based on BMI, obesity was present in 3 patients (14.3%) of Group 1, 7 patients (35%) of Group 2, 5 patients (25%) of Group 3, and 2 patients (8%) of Group 4 (Tab.3).

TABLE 3. Distribution of cardiovascular disease risk factors by groups

Risk factors	Group 1	Group 2	Group 3	Group 4
Hypertension, n (%)	16 (76.2)	18 (90)	20 (100)	22 (88)
Obesity, n (%)	3 (14.3)	7 (35)	5 (25)	2 (8)
Smoking, n (%)	10 (47.6)	10 (50)	11 (55)	10 (40)
Coronary heart disease, n (%)	6 (28.6)	9 (45)	8 (40)	4 (16)
Family history, n (%)	5 (23.8)	10 (50)	8 (40)	9 (36)

Smoking was reported in 10 patients (47.6%) in Group I, 10 patients (50.0%) in Group II, 11 patients (55.0%) in Group III, and 10 patients (40.0%) in Group IV (Tab.3).

A history of coronary heart disease (previously confirmed myocardial infarction) at admission was identified in 6 patients (28.6%) in Group I, 9 patients (45.0%) in Group II, 8 patients (40.0%) in Group III, and 4 patients (16.0%) in Group IV (Tab.3).

A positive family history of cardiovascular disease was reported in 5 patients (23.8%) in Group I, 10 patients (50.0%) in Group II, 8 patients (40.0%) in Group III, and 9 patients (36.0%) in Group IV (Tab.3).

The mean serum Galectin-3 (Gal-3) level in group I was 8.7±6.2 ng/mL, increasing to 13.7±7.9 ng/mL in Group II. In critically ill patients (Group III), Gal-3 levels reached 18.1±7.3 ng/mL, whereas in post-COVID-19 patients (Group IV), this parameter decreased to 8.5±5.9 ng/mL.

Gal-3 levels in the moderately ill group (Group I) differed significantly from those in the severe group (Group II) ($P_{1\text{-}2}$= 0.029), as well as from the critical group (Group III) (Tab.2), indicating that higher Gal-3 concentrations are associated with increased disease severity and may have prognostic significance.

According to previous studies, Galectin-3 in coronary heart disease is primarily considered a marker of myocardial fibrosis, whereas in COVID-19, it appears to reflect phase-dependent pathological processes related to inflammation, tissue injury, and remodeling. Therefore, in the presence of elevated Gal-3 levels, a comprehensive assessment of cardiac structural and functional parameters using echocardiography is essential.[12]

Echocardiographic evaluation in this study was performed according to recommendations from the Copenhagen City Heart Study.[13]

Right-heart parameters in this study were evaluated according to the updated 2025 American Society of Echocardiography (ASE) guidelines for pulmonary hypertension, which support screening and monitoring of patients with suspected or established pulmonary hypertension.[14]

Right Atrial Size: Mean right atrial (RA) diameter was 3.6±0.4 cm in Group I and 3.9±0.7 cm in Group II, both within the normal reference range.[15] With worsening clinical status, RA size progressively increased, reaching 6.5±1.3 cm in critically ill patients (Group III), whereas in post-COVID patients, it returned to near-normal values.

Although RA size in moderately ill patients did not differ significantly from post-COVID patients, RA dimensions showed a severity-dependent increase. This was confirmed by statistically significant differences between Groups I and II ($P_{1–2}$=0.043) and between Groups II and IV ($P_{2\text{-}4}$=0.004).

Right Ventricular Diameter: The echocardiographic right ventricular (RV) end-diastolic diameter (normal range: 2.5-4.1 cm) measured 3.4±0.3 cm in Group I and 3.5±0.4 cm in Group II, without significant intergroup difference ($P_{1\text{-}2}$=0.660). In critically ill patients (Group III), the RV diameter was 3.5±0.2 cm, whereas in post-COVID patients (Group IV), it was 3.4±0.3 cm.

Right Ventricular Fractional Area Change (RVFAC): RVFAC, an indicator of RV systolic function particularly relevant in pulmonary hypertension and heart failure, remained within normal limits across groups. Values were 46.6±5.4% in Group I and 46.0±4.6% in Group II, with no significant difference ($P_{1\text{-}2}$=0.705). No significant differences were observed between Groups I-III ($P_{1\text{-}3}$=0.145), I–IV ($P_{1\text{-}4}$=0.193), or II–III ($P_{2\text{-}3}$=0.238).

However, statistically significant differences were found between Groups II and IV ($P_{2\text{-}4}$=0.050) and between Groups III and IV ($P_{3\text{-}4}$=0.002), indicating improvements in RV functional parameters during the post-COVID period (Tab.4).

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TABLE 4. Clinical characteristics of patients by groups

	Group 2 vs. Group 3		Group 2 vs. Group 4		Group 3 vs. Group 4	
	t-test or Chi2-test	p value	t-test or Chi2-test	p value	t-test or Chi2-test	p value
Right ventricle, RV (cm)	0.691	0.494	0.700	0.487	1.801	0.079
Right atrium, RA (cm)	0.997	0.325	3.057	0.004	1.315	0.195
Pulmonary artery, PA (cm)	0.454	0.652	2.038	0.048	1.971	0.055
Ejection fraction, EF (%)	0.799	0.429	1.779	0.082	2.163	0.036
Fractional shortening, FS (%)	0,803	0.427	1.882	0.067	2.198	0.033
Pulmonary artery systolic pressure, PASP (mmHg)	0,242	0.810	2.159	0.037	1.886	0.066
Tricuspid annular plane systolic excursion, TAPSE	0.485	0.630	2.177	0.035	2.956	0.005
Right ventricular fractional area change, RVFAC	1.198	0.238	1.975	0.055	3.383	0.002

Pulmonary Artery Diameter: Pulmonary artery (PA) diameter (normal <2.5 cm) was 1.9±0.1 cm in Group I and 2.0±0.1 cm in Group II, with a statistically significant difference between them (P=0.028). In Group III, PA diameter remained 2.0±0.1 cm, while in Group IV it decreased to 1.9±0.1 cm.

Significant intergroup differences were observed between Groups I and III ($P_{1\text{-}3}$ = 0.039) and between Groups II and IV ($P_{2\text{-}4}$=0.048). These findings suggest progressive pulmonary vascular involvement that correlates with increasing disease severity.

Pulmonary Artery Systolic Pressure (PASP): PASP was elevated above normal values (<30 mmHg) in all study groups, including post-COVID patients. PASP measured 35.8±11.8 mmHg in Group I and 41.5±13.0 mmHg in Group II, without a significant difference (P=0.149). No statistically significant differences were observed between Groups I-III, I-IV, or II-III.

Tricuspid Annular Plane Systolic Excursion (TAPSE): Right ventricular contraction occurs predominantly in the longitudinal plane, and TAPSE (normal ≥1.7 cm) is a key indicator of RV systolic function and prognostic marker in pulmonary hypertension and heart failure.[16]

TAPSE values remained within normal limits across all groups: 2.10±0.22 cm in Group I, 2.04±0.21 cm in Group II, and 2.01±0.18 cm in Group III; the highest values were observed in post-COVID patients (2.16±0.16 cm).

Statistically significant differences were found between Groups III and IV ($P_{3\text{-}4}$ = 0.005) and between Groups II and IV ($P_{2\text{-}4}$=0.035), indicating recovery of RV longitudinal function in post-COVID patients.

DISCUSSION

According to current literature, COVID-19 significantly affects right-heart function due to severe pulmonary inflammation and lung injury, leading to RV strain, dysfunction, and occasionally RV failure.[17] These changes result in increased pulmonary artery pressure and RV overload, highlighting the crucial role of echocardiography in detecting RV dysfunction and elevated PASP for prognostic assessment.

Our findings indicate that PASP and RA size are the most sensitive right-heart parameters reflecting COVID-19 severity. Marked RA enlargement observed in critically ill patients is closely associated with arrhythmogenesis, particularly atrial fibrillation and supraventricular arrhythmias, as atrial dilation and structural remodeling disrupt normal electrical conduction pathways.[18]

A strong positive relationship between PASP and RA size has been reported in previous studies and was confirmed by our results.[19] PASP was elevated across all groups and may serve as an indicator of acute pulmonary inflammatory burden during active COVID-19 infection. In post-COVID patients, persistent PASP elevation may reflect residual pulmonary vascular remodeling or fibrosis.

Despite normal TAPSE values, this parameter was sensitive to disease severity, with significantly higher values in post-COVID patients than in the severe and critical groups. The TAPSE/PASP ratio, an established marker of RV-pulmonary arterial coupling, was reduced in acute COVID-19 groups and normalized only in post-COVID patients, further supporting its prognostic relevance.

Other right-heart parameters—including RV diameter, RVFAC, PA diameter, and TAPSE—remained within normal limits but demonstrated severity-dependent variations, indicating subclinical right-heart involvement during COVID-19 progression.

Galectin-3 plays a dual role in inflammation and fibrosis.[20] According to our findings, during the early stage of COVID-19, Gal-3 appears to be primarily involved in inflammatory activation and demonstrates strong correlations with right-heart echocardiographic parameters in moderately severe acute patients. In Group I, Gal-3 showed a strong positive correlation with RA diameter (r=0.7219, P=0.000), as well as moderate correlations with RV diameter (r=0.5111, P=0.018) and tricuspid regurgitation severity (r=0.5861, P=0.005) (Tab.5).

In patients with severe disease (Group II), this association persisted only for tricuspid regurgitation (r=0.4476, P=0.048), while correlations with other right-heart parameters were no longer observed.

In critically ill patients (Group III), right-heart echocardiographic parameters showed only weak, non-significant relationships with Gal-3. Notably, although Gal-3 reached its highest mean concentration in this group (18.1±7.3 ng/mL), no significant correlations were identified, suggesting that Gal-3 may have limited value as a marker of right-heart involvement during the advanced cytokine-storm phase of disease.

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In post-COVID patients (Group IV), a significant correlation between Gal-3 and RA diameter re-emerged (r=0.4543, P=0.023), suggesting ongoing fibrotic remodeling, during which Gal-3 appears to regain its role as a marker of fibrosis (Tab.5).

TABLE 5. Correlation indices between Galectin-3 and echocardiographic parameters

Echocardiographic parameters	Correlation index (r)				Significance coefficient (p)			
	Group 1	Group 2	Group 3	Group 4	Group 1	Group 2	Group 3	Group 4
Right ventricle, RV (cm)	0.5111	0.3500	0.0993	0.3154	0.018	0.130	0.677	0.125
Right atrium, RA (cm)	0.7219	0.5311	0.1759	0.4543	0.000	0.016	0.458	0.023
Pulmonary artery, PA (cm)	0.3528	0.3098	0.1906	0.1790	0.117	0.184	0.421	0.392
Ejection fraction, EF (%)	0.5861	0.4476	0.1558	0.3488	0.005	0.048	0.512	0.088
Fractional shortening, FS (%)	0.1762	0.3216	0.2544	0.3091	0.445	0.167	0.279	0.133
Pulmonary artery systolic pressure, PASP (mmHg)	-0.3021	-0.5008	-0.3024	-0.3931	0.183	0.025	0.195	0.052
Tricuspid annular plane systolic excursion, TAPSE	-0.1317	-0.6138	-0.1789	-0.3132	0.569	0.004	0.450	0.127

In the early acute phase of COVID-19, particularly in moderately ill patients, Galectin-3 demonstrates significant positive correlations with right-heart structural and functional parameters, including RV and RA size, tricuspid regurgitation, and RV functional indices. In patients with severe disease, Gal-3 loses its association with RV dimensions while maintaining correlations with RA size, tricuspid regurgitation, and RV function, and becomes significantly related to elevated pulmonary artery systolic pressure.

Despite markedly elevated Gal-3 levels in critically ill patients—reflecting overall disease severity—no significant correlations with right-heart structural changes were observed, suggesting reduced specificity during the hyperinflammatory phase.

In the post-COVID period, Gal-3 appears to assume a different pathophysiological role, acting as an indicator of residual right-heart structural and functional remodeling, as evidenced by its correlations with RA size, tricuspid regurgitation, and PASP.

CONCLUSIONS

The relationship between Galectin-3 and echocardiographic parameters was found to be phase-dependent. In the early acute stage of COVID-19, Galectin-3 showed strong correlations with right-heart structural and functional indices, reflecting active inflammatory involvement. In severe disease, these associations became more limited, primarily related to parameters of right-atrial remodeling and pulmonary hypertension. In critically ill patients, despite markedly elevated Galectin-3 levels, no significant correlations with right-heart parameters were observed, suggesting reduced specificity of this biomarker during the hyperinflammatory phase of the disease.

In the post-COVID period, Galectin-3 regained its clinical relevance as a marker of persistent right-heart remodeling, demonstrating significant associations with right-atrial size and pulmonary pressure indices.

Overall, the findings indicate that the combined analysis of Galectin-3 and echocardiographic parameters enhances the understanding of right-heart involvement, reflects the clinical severity of COVID-19, and may help identify patients at risk of persistent cardiovascular alterations. In this context, Galectin-3 emerges as a phase-dependent biomarker of inflammation-mediated cardiac remodeling during and after COVID-19 infection.

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