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Original Research Article

Association Between Absolute Lymphocyte Count And Severity Of Covid-19: A Retrospective Cross-Sectional Study.

Name of Author:

Parvathy S[1]*, Neethu Showkath[1], Lizann Elizabeth Thomas[2], Arshad A[3].

Affiliation:

[1]Assistant Professor, Department of Physiology, Travancore Medical College, Kollam, Kerala, India
[2]Associate Professor, Department of Physiology, Travancore Medical College, Kollam, Kerala, India
[3]Assistant Professor, Department of Anaesthesiology, Travancore Medical College, Kollam, Kerala, India

Corresponding Author: Dr. Parvathy S.

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Abstract: ***Background:*** Lymphopenia is a frequently reported hematological abnormality in coronavirus disease 2019 (COVID-19) and has been associated with severe illness. Absolute lymphocyte count (ALC), being routinely available from a complete blood count, may provide an inexpensive marker for assessing disease severity, particularly in resource-constrained settings. ***Objectives:*** To estimate ALC among hospitalized patients with COVID-19 and determine its association with disease severity. Serum ferritin levels were additionally evaluated. ***Materials and Methods:*** This retrospective cross-sectional study included 200 adult patients with RT-PCR-confirmed COVID-19 admitted to Government Medical College, Thiruvananthapuram. Patients were classified into severity categories A, B, and C according to the prevailing state guidelines. Demographic characteristics, comorbidities, ALC, serum ferritin, and other hematological parameters were obtained from medical records. Quantitative variables were compared using one-way analysis of variance, followed by Tukey post-hoc testing where applicable. Categorical associations were evaluated using the chi-square test. A P value <0.05 was considered statistically significant. ***Results:*** Of 200 patients, 20 (10.0%) belonged to category A, 99 (49.5%) to category B, and 81 (40.5%) to category C. Mean ALC declined progressively from 1990.74 ± 620.78 cells/µL in category A to 1120.09 ± 624.37 cells/µL in category B and 780.45 ± 305.85 cells/µL in category C ($F_{[2,197]}=44.585$, $P<0.001$). Mean serum ferritin increased from 134.34 ± 224.90 ng/mL in category A to 539.84 ± 623.02 ng/mL in category C ($F=11.357$, $P<0.001$). ANC, NLR, and ESR also increased significantly with severity, whereas hemoglobin did not. ***Conclusion:*** ALC showed a significant inverse association with COVID-19 severity. Its routine availability, rapid turnaround, and low cost support its potential utility as a simple severity-associated laboratory marker in hospitalized COVID-19 patients.

Keywords: Absolute lymphocyte count; COVID-19; Lymphopenia; SARS-CoV-2; Disease severity.

INTRODUCTION

Coronavirus disease 2019 (COVID-19), caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), emerged as a major global infectious disease with a remarkably heterogeneous clinical spectrum. While many infected individuals remain asymptomatic or develop relatively mild respiratory illness, a proportion progress to severe pneumonia, acute respiratory distress syndrome (ARDS), multiorgan dysfunction, and death. Early clinical series demonstrated that severe disease was associated not only with respiratory compromise but also with characteristic alterations in routinely measured hematological and inflammatory parameters.[1,2]

Among the hematological abnormalities observed in COVID-19, lymphopenia emerged early as one of the most consistent findings. Guan et al., in a nationwide analysis of 1,099 laboratory-confirmed patients in China, demonstrated frequent lymphocytopenia, with

more pronounced laboratory abnormalities among patients with severe disease.[3] Similarly, Wang et al. reported lymphopenia in 70.3% of 138 hospitalized patients and observed important differences in clinical and laboratory characteristics between patients requiring intensive care and those managed outside the intensive care unit.[4] These observations suggested that the circulating lymphocyte count may reflect the intensity of systemic involvement in SARS-CoV-2 infection. Lymphocytes constitute a central component of antiviral immunity. SARS-CoV-2 infection is associated with quantitative and functional alterations of T lymphocytes, including reductions in CD4+ and CD8+ T-cell populations in patients with severe disease.[5] Proposed mechanisms for COVID-19-associated lymphopenia include lymphocyte apoptosis associated with inflammatory cytokines, redistribution of lymphocytes to affected tissues, impairment of lymphoid organs, metabolic abnormalities, and functional exhaustion of T cells. The resulting dysregulation of cellular immunity may contribute to inadequate viral control and exaggerated inflammatory responses.[5,6]

The clinical relevance of lymphopenia extends beyond its frequency as a laboratory abnormality. Tan et al. demonstrated that lymphopenia was predictive of COVID-19 disease severity and proposed longitudinal lymphocyte percentage as a potentially useful marker of disease progression.[7] A systematic review and meta-analysis by Huang and Pranata subsequently showed that lymphopenia was significantly associated with severe COVID-19, with a pooled odds ratio of approximately 3.7.[8] Thus, absolute lymphocyte count (ALC), obtainable from a routine complete blood count, has potential value as an inexpensive and rapidly available indicator for early risk stratification. COVID-19 severity is also accompanied by activation of innate inflammatory pathways. Serum ferritin, an acute-phase reactant, frequently rises during systemic inflammation and hyperinflammatory states. Increased ferritin concentrations have been documented in severe COVID-19 and among patients with adverse clinical outcomes.[9] A systematic review and meta-analysis involving more than 10,000 patients demonstrated significantly higher ferritin concentrations in patients with severe disease and in non-survivors, supporting its potential role as an adjunctive marker of inflammatory burden.[10]

Although sophisticated biomarkers and imaging modalities may assist in prognostication, they may not always be immediately available, particularly in resource-constrained healthcare settings. ALC is routinely generated as part of the complete blood count and therefore represents a practical candidate for rapid clinical stratification. The present study was undertaken to determine ALC among hospitalized COVID-19 patients and evaluate its association with clinical severity. Serum ferritin and selected hematological parameters were also evaluated to characterize their relationship with worsening disease severity.

Aim

To determine the association between absolute lymphocyte count and severity of COVID-19 among patients admitted to Government Medical College, Thiruvananthapuram.

Objectives

Primary Objective

To estimate the absolute lymphocyte count in patients with COVID-19 admitted to Government Medical College, Thiruvananthapuram.

Secondary Objectives

1. To determine the association between absolute lymphocyte count and severity of COVID-19.
2. To estimate the mean serum ferritin level among patients with COVID-19.

MATERIALS AND METHODS

Study Design and Setting

This retrospective cross-sectional study was conducted at Government Medical College, Thiruvananthapuram, Kerala, India. The study included adult male and female patients admitted with laboratory-confirmed COVID-19. Data collection was performed during a one-month period following Institutional Ethics Committee clearance, during November–December 2020.

Study Population

The study population comprised adult patients admitted to Government Medical College, Thiruvananthapuram, with COVID-19 confirmed by real-time polymerase chain reaction (RT-PCR), irrespective of their presenting clinical signs and symptoms.

Inclusion Criteria

All adult male and female patients admitted with COVID-19 confirmed by RT-PCR were eligible for inclusion.

Exclusion Criteria

Patients with primary infections caused by other pathogens, including bacterial infections or other respiratory viral infections, were excluded. Patients with alternative causes of lymphopenia, including autoimmune disorders and malignancy receiving chemotherapy or radiotherapy, were also excluded.

Sample Size

The sample size was calculated using the formula:

$n = Z^2_{1-\alpha/2} \times S^2 / d^2$

where Z represented the standard normal deviate, S represented the standard deviation, and d represented the clinically expected variation. A standard deviation of 0.7 for absolute lymphocyte count was used based on a previous study, with a clinically expected variation of 0.1.

Accordingly:
$n = (1.96)^2 \times (0.7)^2 / (0.1)^2 = 188.23$
The final sample was rounded upward, and 200 patient records were included in the study.

Sampling and Data Collection
Case records of patients admitted with COVID-19 who fulfilled the predefined inclusion and exclusion criteria were reviewed until the required sample size was attained. Information regarding sociodemographic characteristics, relevant comorbidities, and laboratory parameters was extracted from the medical records using the study data-collection tool.

COVID-19 infection had been confirmed using nucleic acid amplification testing of nasal swab specimens by real-time PCR.

Study Variables
The principal laboratory variable was absolute lymphocyte count (ALC). The normal range adopted in the study was 800–4,000 cells/µL, and an ALC below 800 cells/µL was classified as lymphopenia. Serum ferritin was evaluated as an inflammatory biomarker. A reference range of 30–400 ng/mL was used, and values above 400 ng/mL were considered hyperferritinemia. Additional hematological variables evaluated included hemoglobin, absolute neutrophil count (ANC), neutrophil-to-lymphocyte ratio (NLR), and erythrocyte sedimentation rate (ESR).

Classification of COVID-19 Severity
Patients were categorized according to the prevailing state COVID-19 clinical guidelines into severity categories A, B, and C. For treatment-related clinical classification, mild disease was characterized by a respiratory rate below 24 breaths/min and oxygen saturation (SpO_2) above 94% on room air. Moderate disease was characterized by a respiratory rate of 24–29 breaths/min and SpO_2 of 91–94% on room air. Severe disease was characterized by a respiratory rate of ≥30 breaths/min and SpO_2 below 90% on room air. The corresponding symptom-based categories incorporated mild upper respiratory or gastrointestinal symptoms in category A; fever and/or more pronounced respiratory or gastrointestinal symptoms, or category A disease with specified risk factors and comorbidities, in category B; and red-flag manifestations such as breathlessness, chest pain, drowsiness, hypotension, hemoptysis, cyanosis, or worsening underlying chronic disease in category C.

Statistical Analysis
Data extracted from the case records were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 16.0. Categorical variables were summarized as frequencies and percentages, while quantitative variables were expressed as mean and standard deviation. One-way analysis of variance (ANOVA) was used to compare quantitative variables across COVID-19 severity categories. Where applicable, Tukey post-hoc analysis was performed to identify pairwise differences between severity categories. Associations between categorical variables were assessed using the chi-square test, and Cramer's V was used in the thesis to quantify the strength of association between nominal variables. A P value <0.05 was considered statistically significant.

Ethical Considerations
Institutional Ethics Committee approval was obtained before commencement of the study. Because the investigation involved retrospective review of existing case records, individual patient consent was waived by the Institutional Human Ethics Committee. Permission for use of medical records was obtained from the Medical College Superintendent and the DME COVID Cell. Patient confidentiality was maintained throughout data collection and analysis.

RESULTS

A total of **200 case records** of patients with RT-PCR-confirmed COVID-19 fulfilling the eligibility criteria were analyzed. Of these, 20 (10.0%) patients belonged to severity category A, 99 (49.5%) to category B, and 81 (40.5%) to category C. The overall mean age was **53.84 ± 18.19 years**. Mean age increased progressively across the severity categories, from 36.6 ± 11.97 years in category A to 49.1 ± 17.67 years in category B and 63.9 ± 14.22 years in category C. There were 85 (42.5%) males and 115 (57.5%) females.

Table 1. Baseline characteristics and distribution of study participants

Characteristic	Category A	Category B	Category C	Total
Number of patients, n	20	99	81	200
Distribution, %	10.0	49.5	40.5	100
Age, years, mean ± SD	36.6 ± 11.97	49.1 ± 17.67	63.9 ± 14.22	53.84 ± 18.19
Male, n (%)	—	—	—	85 (42.5)
Female, n (%)	—	—	—	115 (57.5)

The age profile demonstrated a progressive increase in mean age with increasing clinical severity. Category C patients were approximately 27 years older on average than patients in category A. The thesis reported that increasing age was significantly associated with worsening disease severity. Sex, however, was not reported to have a statistically significant relationship with severity.

Comorbidities and Disease Severity

Diabetes mellitus was documented in **79 (39.5%)** patients. None of the category A patients had diabetes, whereas 34 category B and 45 category C patients had diabetes. The thesis reported a Cramer's V of **0.338**, indicating an association between diabetes and severity.

Hypertension was present in **69 (34.5%)** patients, comprising one category A patient, 33 category B patients, and 35 category C patients. The reported Cramer's V was **0.229**. Coronary artery disease (CAD) was present in **30 (15.0%)** patients: 13 in category B and 17 in category C, with no CAD recorded in category A. The reported Cramer's V was **0.174**.

Table 2. Distribution of comorbidities according to COVID-19 severity

Comorbidity	Category A, n	Category B, n	Category C, n	Total, n (%)	Cramer's V
Diabetes mellitus	0	34	45	79 (39.5)	0.338
Hypertension	1	33	35	69 (34.5)	0.229
Coronary artery disease	0	13	17	30 (15.0)	0.174

Comorbidities were concentrated in the moderate and severe disease categories. Diabetes showed the largest reported Cramer's V among the three comorbid conditions assessed. Importantly, the present manuscript does **not** assign additional P values or odds ratios to these comparisons because these values were not provided in the thesis results.

Absolute Lymphocyte Count and COVID-19 Severity

The mean absolute lymphocyte count (ALC) in the entire study population was **1069.60 ± 622.05 cells/µL**. A marked progressive reduction in ALC was observed with increasing COVID-19 severity.

Category A patients had a mean ALC of **1990.74 ± 620.78 cells/µL**, compared with **1120.09 ± 624.37 cells/µL** in category B and **780.45 ± 305.85 cells/µL** in category C.

Median ALC was 2001 cells/µL in category A, 928 cells/µL in category B, and 780 cells/µL in category C. The corresponding observed ranges were 850.50–3224, 350–3220, and 171–1552 cells/µL, respectively.

Table 3. Absolute lymphocyte count according to COVID-19 severity

Severity category	n	Mean ALC (cells/µL)	SD	Median	Minimum	Maximum
A	20	1990.74	620.78	2001	850.50	3224
B	99	1120.09	624.37	928	350	3220
C	81	780.45	305.85	780	171	1552
Total	**200**	**1069.60**	**622.05**	—	—	—

One-way ANOVA demonstrated a statistically significant difference in ALC across the three severity categories (**F[2,197] = 44.585, P < 0.001**). Tukey post-hoc analysis, as reported in the thesis, showed statistically significant reductions in ALC on pairwise comparison of the mild, moderate, and severe disease categories. Thus, increasing clinical severity was accompanied by a substantial decline in circulating absolute lymphocyte count.

Serum Ferritin and COVID-19 Severity

The overall mean serum ferritin concentration was **357.33 ± 481.49 ng/mL**. Ferritin increased progressively with worsening disease severity. Mean ferritin was **134.34 ± 224.90 ng/mL** in category A, **253.04 ± 308.91 ng/mL** in category B, and **539.84 ± 623.02 ng/mL** in category C.

Table 4. Serum ferritin levels according to COVID-19 severity

Severity category	n	Mean serum ferritin (ng/mL)	SD
A	20	134.34	224.90
B	99	253.04	308.91
C	81	539.84	623.02
Total	**200**	**357.33**	**481.49**

One-way ANOVA demonstrated a statistically significant difference in serum ferritin levels across severity categories (**F = 11.357, P < 0.001**). The approximately four-fold difference between mean ferritin levels in categories A and C demonstrated a clear increase in inflammatory burden across the clinical severity spectrum.

Other Hematological Parameters

The overall mean hemoglobin concentration was **12.416 ± 2.5**, mean absolute neutrophil count (ANC) was **7488.17 ±**

4894.80, mean neutrophil-to-lymphocyte ratio (NLR) was **8.96 ± 7.0**, and mean ESR was **37.94 ± 29.20**.

ANC, NLR, and ESR progressively increased across the severity categories. In contrast, hemoglobin showed no statistically significant association with severity.

Table 5. Other hematological parameters according to COVID-19 severity

Parameter	Category A, mean ± SD	Category B, mean ± SD	Category C, mean ± SD	Overall, mean ± SD
Hemoglobin	13.21 ± 2.3	12.60 ± 2.0	11.98 ± 3.0	12.416 ± 2.5
ANC	4879.55 ± 3183.89	6002.02 ± 2714.16	9948.67 ± 6132.07	7488.17 ± 4894.80
NLR	3.28 ± 4.03	6.29 ± 3.63	13.63 ± 7.93	8.96 ± 7.0
ESR	22.17 ± 13.99	30.60 ± 25.29	50.81 ± 31.55	37.94 ± 29.20

One-way ANOVA performed in the original thesis showed statistically significant relationships between severity and the quantitative parameters evaluated (**P < 0.01**), with the exception of hemoglobin (**P = 0.83**). The most pronounced hematological pattern was therefore characterized by declining ALC accompanied by increasing ANC, NLR, and ESR as disease severity worsened.

DISCUSSION

The present study evaluated the relationship between absolute lymphocyte count and COVID-19 severity in 200 hospitalized patients. The principal finding was a strong inverse relationship between ALC and clinical severity. Mean ALC declined from 1990.74 ± 620.78 cells/µL in category A to 1120.09 ± 624.37 cells/µL in category B and 780.45 ± 305.85 cells/µL in category C. The difference was highly significant ($F[2,197] = 44.585$, $P < 0.001$), and post-hoc analysis demonstrated significant pairwise differences across the severity categories. Serum ferritin, ANC, NLR, and ESR showed the opposite pattern and increased with disease severity. Increasing age was also associated with greater disease severity. Mean age increased from 36.6 years in category A to 63.9 years in category C. This finding is consistent with the extensive clinical evidence identifying advanced age as an important determinant of severe COVID-19. Zhou et al. demonstrated that increasing age was associated with mortality among hospitalized patients with COVID-19.[11] The relationship is likely multifactorial and may reflect immunosenescence, reduced physiological reserve, increased burden of comorbid disease, endothelial dysfunction, and age-associated dysregulation of innate and adaptive immune responses.

The prevalence of diabetes mellitus, hypertension, and coronary artery disease was greater in the more severe categories in the present study. Diabetes was present in 39.5% of the overall population, hypertension in 34.5%, and coronary artery disease in 15%. None of the category A patients had diabetes or coronary artery disease. Parveen et al., in a systematic review and meta-analysis, demonstrated an association of diabetes and hypertension with COVID-19 severity.[12] A larger meta-analysis similarly found hypertension, cardiovascular disease, and diabetes to be associated with severe or fatal COVID-19.[13] These findings support the pattern observed in the present cohort, although the retrospective cross-sectional design does not permit determination of an independent causal contribution of individual comorbidities. The most important observation of this study was the progressive decline in ALC. The mean ALC among category C patients was approximately 61% lower than that of category A patients. This pattern is biologically plausible because lymphocytes, particularly T lymphocytes, have a central role in antiviral cellular immunity. Qin et al. demonstrated substantial immune dysregulation in severe COVID-19, characterized by lower lymphocyte counts and reductions in helper, suppressor, and regulatory T-cell populations.[14] Such alterations may impair effective antiviral immunity while simultaneously contributing to dysregulated inflammatory responses.

Tan et al. specifically evaluated lymphopenia as a predictor of COVID-19 severity and demonstrated that lymphocyte dynamics were closely associated with clinical progression.[15] Huang and Pranata, in a systematic review and meta-analysis, subsequently reported that lymphopenia was associated with approximately a threefold increased risk of severe COVID-19.[16] The present findings are consistent with this body of evidence and are particularly relevant because ALC can be obtained rapidly from a routine complete blood count without requiring specialized assays. Several mechanisms may explain lymphopenia during severe SARS-CoV-2 infection. These include destruction or dysfunction of lymphoid tissues, cytokine-mediated apoptosis, metabolic inhibition of lymphocyte proliferation, redistribution of circulating lymphocytes into infected tissues, and functional exhaustion of T cells. Diao et al. demonstrated significant reductions in circulating T cells in patients with COVID-19, particularly among patients requiring intensive care, and observed evidence of T-cell exhaustion associated with disease progression.[17] Therefore, a falling circulating ALC may represent both the intensity of systemic inflammation and deterioration of adaptive antiviral immune competence.

An important accompanying finding was the progressive increase in NLR. Mean NLR increased from 3.28 ± 4.03 in category A to 6.29 ± 3.63 in category B and 13.63 ± 7.93 in category C. This represents the combined effect of increasing neutrophilia and decreasing lymphocyte counts. Elevated NLR has repeatedly been associated with severe COVID-19 and adverse outcomes. Liu et al. demonstrated that NLR could assist in early identification of patients at risk of developing critical illness.[18] From a clinical perspective, the simultaneous presence of neutrophilia and lymphopenia may provide a broader representation of the balance between systemic innate inflammation and adaptive immune suppression than either parameter considered individually.

Serum ferritin also showed a marked relationship with severity. Mean ferritin increased from 134.34 ± 224.90 ng/mL in category A to 539.84 ± 623.02 ng/mL in category C, and the overall difference was statistically significant ($F = 11.357$, $P < 0.001$). Ferritin is not simply a marker of iron storage; it behaves as an acute-phase reactant and increases during systemic inflammatory responses. Cheng et al., in a systematic review and meta-analysis involving more than 10,000 COVID-19 patients, demonstrated substantially higher ferritin concentrations in patients with severe disease and adverse outcomes.[19] The parallel increase in ferritin and reduction in ALC observed in this study may therefore represent different components of the same pathophysiological process: increasing systemic inflammation accompanied by progressive impairment of adaptive cellular immunity. Nevertheless, ferritin is nonspecific and may be influenced by hepatic disease, chronic inflammation, metabolic disorders, infection, and other conditions. It should therefore be interpreted as an adjunctive marker rather than a disease-specific indicator.

The study also demonstrated increasing ANC and ESR across severity categories. Mean ANC almost doubled between category A and category C, while ESR increased from 22.17 ± 13.99 to 50.81 ± 31.55. These findings further support an increasing systemic inflammatory response with worsening COVID-19. By contrast, hemoglobin did not demonstrate a statistically significant relationship with severity ($P = 0.83$), suggesting that the leukocyte-derived parameters were more closely related to acute disease severity in this cohort. The clinical significance of these findings lies in the accessibility of ALC. During periods of high patient volume and restricted healthcare resources, biomarkers that are inexpensive, rapidly available, and routinely measured are particularly valuable. ALC requires no additional specialized investigation beyond a routine complete blood count. In combination with clinical assessment, oxygen saturation and other laboratory indicators, a markedly reduced ALC may therefore assist clinicians in identifying patients requiring closer monitoring. However, because the present study was cross-sectional, the findings demonstrate an association with concurrent disease severity rather than proving that ALC independently predicts subsequent deterioration. This distinction is important when translating these results into clinical practice.

Overall, the present findings reinforce evidence that lymphopenia is an important hematological feature of severe COVID-19. The progressive and statistically significant reduction in ALC across all three clinical categories provides support for its use as a simple marker of disease severity. The concurrent elevation of ferritin, ANC, NLR, and ESR further indicates that severe disease is characterized by a combination of heightened systemic inflammation and depletion of circulating lymphocytes.

Strengths and Limitations

A major strength of this study was the evaluation of a readily available and inexpensive hematological parameter in 200 laboratory-confirmed COVID-19 patients across three predefined clinical severity categories. ALC was evaluated quantitatively rather than solely as the presence or absence of lymphopenia, enabling demonstration of a clear graded decline across increasing disease severity. Evaluation of ferritin, ANC, NLR, ESR, and clinically relevant comorbidities provided additional context for interpreting the relationship between ALC and systemic disease severity.

Several limitations must be acknowledged. First, the retrospective cross-sectional design establishes association but cannot establish temporality or determine whether reduced ALC independently predicts subsequent deterioration. Serial lymphocyte measurements were not evaluated; therefore, changes in ALC during progression and recovery could not be examined. Second, reliance on retrospective medical records introduces the possibility of incomplete or inaccurate documentation. Third, this was a single-center tertiary-care study, creating the potential for referral and selection bias and limiting generalizability to community-managed or asymptomatic patients. Fourth, potentially important confounding variables were not controlled through multivariable regression. Finally, although 200 patients were included, larger prospective multicenter cohorts would be required to validate clinically useful ALC thresholds and determine independent prognostic performance.

CONCLUSION

Absolute lymphocyte count demonstrated a strong inverse association with COVID-19 severity in this retrospective study of 200 hospitalized patients. Mean ALC declined progressively from 1990.74 ± 620.78 cells/µL in category A to 780.45 ± 305.85 cells/µL in category C, with a statistically significant difference across severity categories. Increasing severity was

additionally associated with higher serum ferritin, absolute neutrophil count, NLR, and ESR, while hemoglobin showed no significant association.

Diabetes mellitus, hypertension, coronary artery disease, and increasing age were also more prominent among patients with greater disease severity. These findings support ALC as a simple, inexpensive, and rapidly obtainable marker associated with the clinical severity of COVID-19. However, because of the retrospective cross-sectional design, ALC should be considered a severity-associated marker rather than an independently validated prognostic predictor. Prospective multicenter studies incorporating serial ALC measurements and multivariable adjustment are warranted.

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