THE MEDICAL BULLETIN OF
SISLI ETFAL HOSPITAL

DOI: 10.14744/SEMB.2026.67764
Med Bull Sisli Etfal Hosp 2026;60(1):24-29

Original Research

The Relationship Between Neutrophil/Lymphocyte Ratio and the Progression and Clinical Features of Alzheimer's Disease

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Abstract

Objectives: This study aimed to investigate the relationship between the neutrophil-to-lymphocyte ratio (NLR), a peripheral inflammatory marker, and both baseline clinical features and short-term cognitive progression in patients with Alzheimer's disease (AD). Specifically, we sought to determine whether NLR is associated with disease stage, cognitive performance, vascular comorbidities, and 12-month cognitive decline assessed by the Mini-Mental State Examination (MMSE).
Methods: We conducted a retrospective observational study including 100 adults diagnosed with mild, moderate, or severe AD who were followed for at least 12 months at a tertiary memory clinic. Demographic characteristics, clinical data, and serial MMSE scores (baseline, 6 months, and 12 months) were extracted from medical records. Complete blood counts from the same time points were used to calculate NLR. Descriptive statistics summarized clinical variables. Correlations between NLR and MMSE scores were analyzed using Pearson and Spearman methods. Group comparisons across disease stage, sex, and vascular comorbidity were performed using t-tests or ANOVA as appropriate. Statistical significance was defined as p<0.05.
Results: The mean age of the cohort was 72.5±9.9 years, and 62% were women. Baseline disease severity was distributed as mild (28%), moderate (26%), and severe (46%). Mean baseline NLR was 2.41±1.17, increasing to 2.87±1.47 at 6 months and 3.75±3.54 at 12 months. Baseline NLR was significantly higher in patients with vascular comorbidities (p=0.008) but did not differ across AD severity categories. Higher baseline NLR was modestly associated with lower baseline MMSE scores (r=−0.24, p=0.021). Unexpectedly, higher baseline NLR correlated with a smaller decline in MMSE over 12 months (r=0.36, p=0.005). Patients with low NLR showed greater cognitive deterioration (−3.72±3.97 points) than those with high NLR (−1.38±4.38 points; p=0.037).
Conclusion: NLR was associated with worse cognitive performance at diagnosis and increased gradually over 12 months in AD patients, supporting its role as a marker of systemic inflammation. However, the counterintuitive finding that higher baseline NLR was linked to slower short-term cognitive decline highlights the complexity of inflammatory mechanisms in established AD. These results suggest that while NLR reflects clinically relevant inflammatory status, it should not be used as a standalone predictor of disease progression but rather as part of a broader multimodal biomarker framework.
Keywords: Alzheimer disease, biomarkers, cognition, inflammation, neutrophil-lymphocyte ratio, vascular comorbidity

Please cite this article as "Omercikoglu Ozden H, Ustuner FS, Ucar MB, Durmaz Celik FN. The Relationship Between Neutrophil/Lymphocyte Ratio and the Progression and Clinical Features of Alzheimer's Disease. Med Bull Sisli Etfal Hosp 2026;60(1):24-29".

Alzheimer's disease (AD), the most common cause of dementia worldwide, is characterized by progressive cognitive decline and complex neuropathological features, including amyloid-beta (Aβ) deposition and tau hyperphosphorylation. Despite significant advances in understanding its molecular underpinnings, the etiology and

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Submitted Date: November 20, 2025 **Revised Date:** December 11, 2025 **Accepted Date:** January 7, 2026 **Available Online Date:** March 23, 2026

progression of AD remain only partially explained by classical pathological hallmarks, suggesting that additional systemic and neuroinflammatory processes may contribute to disease development and progression.[1] Emerging evidence supports the role of immune dysregulation and systemic inflammation in the pathogenesis of AD. In particular, the neutrophil-to-lymphocyte ratio (NLR), a simple and cost-effective biomarker derived from peripheral blood counts, has gained attention as a potential indicator of inflammation-related neurodegenerative processes. Several studies have reported elevated NLR levels in patients with cognitive impairment and AD compared to cognitively normal individuals, proposing that heightened peripheral inflammation may reflect or contribute to central neuroinflammatory activity.[2,3]

From a pathophysiological perspective, neutrophils have been implicated in promoting AD pathology through multiple pathways, including the disruption of cerebral blood flow, increased blood-brain barrier permeability, and activation of microglia and astrocytes. These effects may lead to exacerbation of Aβ aggregation, tau phosphorylation, and neuronal injury.[1,4] Conversely, lymphopenia, commonly observed in AD patients, may reflect a weakened adaptive immune response, further enhancing the dominance of pro-inflammatory innate immune activity.[4]

Clinical investigations, including large-scale population-based studies such as the Framingham Heart Study and community-based cohorts from China, have demonstrated that higher baseline NLR values are independently associated with an increased risk of incident dementia over time.[1,5] Moreover, longitudinal analyses suggest that elevated NLR may serve not only as a correlate of current disease status but also as a predictive marker of future cognitive decline, even in cognitively unimpaired older adults.[5,6]

Despite these promising findings, inconsistencies remain regarding the predictive utility of NLR across different stages of AD and its specificity relative to other dementia subtypes. This study aims to explore the relationship between the neutrophil-to-lymphocyte ratio and both the clinical features and progression of Alzheimer's disease. By integrating clinical, cognitive, and laboratory data, we seek to evaluate the potential of NLR as an accessible biomarker reflecting systemic inflammatory status and its association with the neurodegenerative trajectory in AD.

Methods

This study was designed as a retrospective observational analysis conducted at the Neurology Department of Marmara University School of Medicine Pendik Research and Training Hospital. The primary objective was to investigate the relationship between the peripheral neutrophil-to-lymphocyte ratio (NLR) and clinical progression in patients diagnosed with Alzheimer's disease (AD).

Study Population

The study included a minimum of 100 patients aged over 18 years who were followed at least twice over a 12-month period with a confirmed diagnosis of AD (mild, moderate, or severe stages). Patients were identified through electronic medical records and dementia clinic archives. Exclusion criteria comprised a diagnosis of non-Alzheimer's dementias or insufficient follow-up data.

Data Collection

Demographic characteristics (age, sex, education level), clinical information (disease onset date, stage of AD), cognitive assessments, and laboratory data were retrieved retrospectively. Cognitive status was evaluated using the Mini-Mental State Examination (MMSE), administered every six months. Educational background was considered when selecting the appropriate MMSE version (literate vs. illiterate form).

Routine hematological parameters were obtained from patients' medical records. These included white blood cell count (WBC), absolute neutrophil and lymphocyte counts, hemoglobin (Hb), hematocrit (Hct), and platelet count (PLT). NLR was calculated as the ratio of neutrophils to lymphocytes on the same test day as the MMSE evaluation when available.

Outcome Measures

The primary outcome was the progression of cognitive decline, determined by changes in MMSE scores and clinical documentation. The main exposure variable was the neutrophil-to-lymphocyte ratio. Secondary analyses explored correlations between NLR and disease stage (mild/moderate/severe), as well as associations with hemogram components.

Statistical Analysis

Descriptive statistics were used to summarize demographic and clinical characteristics. Continuous variables were presented as means±standard deviation or medians with interquartile ranges, depending on the normality of distribution. Categorical variables were expressed as frequencies and percentages. Correlations between NLR and MMSE scores were analyzed using Pearson or Spearman correlation coefficients. Comparative analyses between different disease stages were performed using ANOVA or Kruskal–Wallis tests. A $p<0.05$ was considered statistically significant. Statistical analyses were performed using

IBM SPSS Statistics, version 26.0.

Ethical Considerations

The study was approved by the Clinical Research Ethics Committee of Marmara University Faculty of Medicine (Protocol code: 09.2025.25-0636). As a retrospective and anonymized study, informed consent was waived in accordance with national regulations and the Declaration of Helsinki.

Results

Patient Characteristics

A total of 100 patients with Alzheimer's disease were included in the analysis. The mean age at diagnosis was 72.5±9.9 years (range 44–93), and 62 patients (62.0%) were women and 38 patients (38.0%) were men. Most patients were uneducated (61.0%), whereas 15.0% had primary school, 8.0% secondary school, 11.0% high school, and 5.0% university education. At baseline, 28.0% of patients were classified as having mild, 26.0% as moderate, and 46.0% as severe Alzheimer's disease. The majority of cases presented with an amnestic phenotype (93.0%), while 7.0% were non-amnestic. Additional vascular comorbidities were documented in 49 patients (49.0%).

Baseline cognitive performance, assessed by the MMSE, was available for 91 patients and showed a mean score of 20.0±5.4 (median 20, interquartile range [IQR] 17–25; range 8–29). Follow-up MMSE scores at 6 and 12 months were available for 68 and 59 patients, respectively, with mean values of 19.5±5.3 and 18.7±5.2. Over the 12-month period, the mean change in MMSE (MMSE at 12 months minus MMSE at baseline) was −2.37±4.33 points (median −2, IQR −6 to −1; range −12 to +10), indicating an overall mild to moderate cognitive decline during follow-up.

Neutrophil-to-Lymphocyte Ratio in the Study Cohort

Baseline NLR values were available for all patients. The mean baseline NLR was 2.41±1.17 (median 2.16, IQR 1.51–3.02; range 0.77–6.71). At 6 and 12 months, NLR values remained available for the full cohort and showed a gradual increase over time, with a mean NLR of 2.87±1.47 at 6 months and 3.75±3.54 at 12 months (median 2.92, IQR 2.17–4.33; range 1.05–34.0). The longitudinal evolution of NLR over the 12-month follow-up is illustrated in Figure 1.

When stratified by sex, mean baseline NLR was 2.47±1.09 in men (n=38) and 2.37±1.23 in women (n=62); this difference was not statistically significant (p=0.69). In contrast, patients with additional vascular disease had significantly

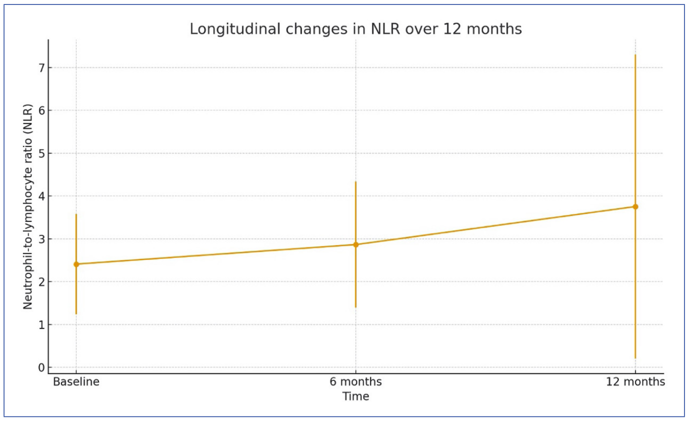

Figure 1. Longitudinal changes in nuetrophil-to-lymphocte ratio (NLR) over 12 months.

higher baseline NLR compared with those without vascular comorbidities (2.73±1.34 vs. 2.10±0.89, respectively; p=0.008). Baseline NLR did not differ significantly across clinical stages of Alzheimer's disease, with mean values of 2.33±0.91 in mild, 2.17±0.88 in moderate, and 2.59±1.42 in severe cases (F(2,97)=1.15, p=0.32). These comparisons are detailed in Table 1.

Relationship Between NLR and Cognitive Status

At baseline, higher NLR values were significantly associated with lower MMSE scores, indicating worse cognitive performance. In patients with available data (n=91), baseline NLR showed a modest but statistically significant negative correlation with MMSE (Pearson r=−0.24, p=0.021; Spearman ρ=−0.26, p=0.014). To further explore this relationship, patients were dichotomized according to the median baseline NLR. Those with lower NLR values (<median) had a mean baseline MMSE of 20.6±5.3, whereas those with higher NLR values (≥median) had a mean baseline MMSE of 19.5±5.5; this difference did not reach statistical significance (p=0.30), but was consistent with the overall trend toward poorer cognition in patients with a higher systemic inflammatory burden.

Relationship Between NLR and Disease Progression

The association between baseline NLR and cognitive decline over 12 months was evaluated in the subset of patients with both baseline and 12-month MMSE measurements (n=59). In this group, higher baseline NLR values were significantly associated with a smaller decline in MMSE scores over 12 months. Baseline NLR correlated positively with MMSE change (ΔMMSE), both in parametric and non-parametric analyses (Pearson r=0.36, p=0.005; Spearman ρ=0.34, p=0.009).

Consistently, in the median-split analysis, patients in the lower NLR group experienced a more pronounced cognitive decline compared with those in the higher NLR group. The mean 12-month MMSE change was −3.72±3.97 points in the low-NLR group versus −1.38±4.38 points in the high-NLR group, and this difference was statistically significant (p=0.037). These associations between baseline NLR and cognitive outcomes are summarized in Table 1.

Table 1. Neutrophil-to-lymphocyte ratio and its associations with clinical variables and cognitive outcomes
1A. Baseline NLR according to categorical clinical variables

Variable / Group	n	NLR, mean±SD	p*
Gender			0.691
Women	62	2.37±1.23	
Men	38	2.47±1.09	
Disease stage			0.320†
Mild	28	2.33±0.91	
Moderate	26	2.17±0.88	
Severe	46	2.59±1.42	
Additional vascular disease			0.008
Absent	51	2.10±0.89	
Present	49	2.73±1.34	

*p-values obtained by independent-samples t-test (gender, vascular disease).
†p-value obtained by one-way ANOVA (comparison across disease stages).

1B. Associations between baseline NLR and cognitive measures

Outcome	n	Analysis	Coefficient (r)	p
Baseline MMSE	91	Pearson correlation	−0.24	0.021
Baseline MMSE	91	Spearman rank correlation	−0.26	0.014
MMSE change over 12 months (ΔMMSE)	59	Pearson correlation	0.36	0.005
MMSE change over 12 months (ΔMMSE)	59	Spearman rank correlation	0.34	0.009

1C. Cognitive outcomes according to baseline NLR (median split)

Group (baseline NLR)	n	Baseline MMSE, mean±SD	ΔMMSE 12 months, mean±SD	p (Baseline MMSE)*	p (ΔMMSE)**
Low NLR (< median)	45	20.6±5.3	−3.72±3.97	0.304	0.037
High NLR (≥ median)	46	19.5±5.5	−1.38±4.38		

*Independent-samples t-test comparing baseline MMSE between low and high NLR groups. **Independent-samples t-test comparing ΔMMSE between low and high NLR groups. Abbreviations: MMSE, Mini-Mental State Examination; NLR, neutrophil-to-lymphocyte ratio; SD, standard deviation.

Discussion

In this retrospective cohort of patients with clinically diagnosed Alzheimer's disease, we investigated whether the peripheral neutrophil-to-lymphocyte ratio (NLR), a marker of systemic inflammation, is related to cross-sectional clinical features and short-term cognitive decline. The main findings were: (i) baseline NLR was modestly but significantly associated with worse cognitive performance at diagnosis; (ii) NLR increased over the 12-month follow-up; (iii) baseline NLR was higher in patients with additional vascular comorbidities but did not differ across mild, moderate, and severe AD stages; and (iv) unexpectedly, higher baseline NLR was associated with a smaller decline in MMSE over 12 months, whereas patients with lower NLR values exhibited a more pronounced cognitive deterioration.

Our findings are consistent with accumulating evidence linking peripheral inflammatory markers, including NLR, to dementia risk and related biomarkers. Large population cohorts such as the Framingham Heart Study and Chinese community samples have shown that higher NLR is independently associated with an increased risk of incident dementia and cognitive impairment, even after adjustment for vascular factors.[1,5] Studies in cognitively unimpaired older adults further demonstrate that elevated NLR correlates with amyloid-β deposition and other AD biomarkers, suggesting that systemic inflammation may precede clinical decline.[2,6] Cross-sectional studies and meta-analyses consistently report higher NLR in patients with AD or MCI compared with healthy controls, with values generally increasing alongside disease severity.[3,4,7] Our observation that higher baseline NLR is linked to lower MMSE scores aligns with these findings and supports the role of systemic immune dysregulation and low-grade inflammation in worse cognitive status. Recent reviews also indicate that NLR may be altered across the full AD continuum and interacts with neuroimaging, CSF biomarkers, and other immune indices.[4,8] Our cohort contributes to this literature by examining real-world patients with established AD and by evaluating short-term longitudinal changes in both NLR and cognition.

In line with the concept of chronic low-grade inflammation and accumulating comorbidities in older adults, we observed a gradual increase in mean NLR over 12 months, with the highest values at the 1-year follow-up. This temporal pattern is compatible with data from systematic reviews and population-based studies indicating that NLR tends to rise with age and is associated with adverse cardiometabolic outcomes that frequently coexist with AD.[4,7,8] The higher baseline NLR observed in patients with additional vascular disease in our cohort reinforces this interpretation and underscores the contribution of vascular pathology and systemic inflammation to the overall inflammatory burden.

The most intriguing and counterintuitive finding of our study is that higher baseline NLR was associated with a smaller decline in MMSE over 12 months, whereas the low-NLR group showed more pronounced cognitive worsening. On initial consideration, this seems to contradict prior work suggesting that elevated NLR predicts a higher risk of incident dementia and steeper cognitive decline in community-dwelling older adults and individuals with MCI.[1,3,5–8]

Several methodological and biological factors may explain this discrepancy. First, our cohort consisted only of patients with established AD, many already in moderate or severe stages, where the relationship between systemic inflammation and cognitive trajectory may become nonlinear. Prior meta-analyses also demonstrate heterogeneous NLR effects across disease stages, with weaker associations in advanced dementia compared with preclinical or MCI populations.[7,8] Thus, higher NLR in our cohort may reflect long-standing inflammatory exposure that influenced cognition before the 12-month follow-up window. Second, regression to the mean and selection bias may have affected our results: patients with the highest inflammatory burden or fastest progression may have been less likely to complete follow-up, enriching the sample with relatively stable high-NLR individuals. Survivor and attrition biases are well-recognized limitations in retrospective NLR studies.[1,4,7,8] Third, clinical management may have differed between NLR strata, with patients showing higher inflammatory markers potentially receiving closer monitoring and more frequent interventions, which could partially attenuate cognitive decline. Finally, MMSE is an insensitive measure in moderate-to-severe dementia and is prone to ceiling and floor effects. Many patients already had low MMSE scores at baseline, limiting the detectable range of decline in the high-NLR group. More detailed neuropsychological and functional measures would better capture disease progression in relation to NLR.

Experimental and clinical evidence highlights several mechanisms through which neutrophils and lymphocytes may influence AD pathophysiology. Neutrophils contribute to endothelial dysfunction, reduced cerebral blood flow, and increased blood–brain barrier permeability, enabling peripheral immune cell infiltration into the CNS.[1,4] They also release reactive oxygen species and neutrophil extracellular traps (NETs), which have been implicated in amyloid-β aggregation, tau phosphorylation, and neurovascular damage.[4,8] Conversely, lymphopenia frequently observed in older adults and some AD cohorts may reflect impaired adaptive immunity and reduced clearance of misfolded proteins.[4,7] As an integrated marker of innate and adaptive immune balance, NLR aligns with these mechanisms, consistent with our findings linking baseline NLR to cognitive status and vascular comorbidity and with prior reports correlating NLR with neurodegeneration, amyloid burden, and small-vessel disease.[2,4,8]

However, the paradoxical association between higher baseline NLR and slower short-term decline suggests that inflammatory–neurodegenerative interactions may not be linear, particularly in later disease stages. Higher NLR in some patients could signify a more preserved or reactive immune state, while very low NLR may reflect immunosenescence, which has been associated with more aggressive disease trajectories in other chronic conditions.[4,8] These interpretations remain speculative and warrant verification through longitudinal studies incorporating detailed immune phenotyping.

From a clinical standpoint, our findings support the association between NLR and cognitive status in AD while underscoring the limitations of using NLR as a standalone prognostic biomarker. The modest effect sizes and the unexpected direction of its relationship with 12-month MMSE decline indicate that NLR should be interpreted together with vascular comorbidities, disease stage, and additional inflammatory markers or composite scores. Recent reviews emphasize that NLR is best integrated into a multimodal risk profile that includes neuroimaging, CSF or plasma biomarkers, and genetic or lifestyle factors.[4,7,8] Future studies should involve larger, prospective cohorts with longer follow-up and repeated measures of both NLR and detailed cognitive or functional outcomes across the AD continuum. Evaluating dynamic NLR changes in response to infections, vascular events, or targeted therapies (e.g., anti-amyloid or anti-inflammatory treatments) may clarify whether NLR acts merely as a passive marker or a modifiable component of AD pathophysiology.[4,8] Moreover, combining NLR with other blood-based markers of inflammation, evndothelial dysfunction, and neurodegeneration, as suggested by recent multi-omic studies, may further improve risk prediction and patient stratification.[2,6,8]

A key strength of our study is the use of real-world data from a specialized memory clinic with repeated NLR and MMSE measurements over 12 months and explicit consideration of vascular comorbidities. In contrast, the retrospective single-center design, modest sample size, and reliance on MMSE alone as the measure of cognitive progression—acknowledging that MMSE is primarily a screening tool with limited sensitivity to subtle or domain-specific cognitive changes—represent important limitations that should be addressed in future prospective studies, ideally incorporating more comprehensive neuropsychological assessments.

Conclusion

In summary, our study supports an association between higher NLR and worse cognitive status at diagnosis in patients with Alzheimer's disease and confirms that NLR increases over time in this population. At the same time, the unexpected finding that higher baseline NLR was linked to a smaller short-term decline in MMSE underscores the complexity of the relationship between systemic inflammation and neurodegenerative progression in established AD. Together with previous epidemiological, biomarker, and mechanistic studies, our results suggest that NLR reflects clinically relevant aspects of immune dysregulation in AD but is unlikely to serve as a simple, unidirectional predictor of disease progression. Instead, NLR should be considered as one component within a broader, multimodal biomarker framework aimed at capturing the interplay between systemic inflammation, vascular pathology, and neurodegeneration across the Alzheimer's disease continuum.

Disclosures

Ethics Committee Approval: The study was approved by Marmara University Faculty of Medicine Ethics Committee (No: 09.2025.25-0636, Date: 18.07.2025).

Informed Consent: As a retrospective and anonymized study, informed consent was waived in accordance with national regulations.

Conflict of Interest: The authors declare that there is no conflict of interest.

Financial Disclosure: This study received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Use of AI for Writing Assistance: Not declared.

Authorship Contributions: Concept – H.O.O., F.S.U., M.B.U., F.N.D.C.; Design – H.O.O., F.S.U., M.B.U., F.N.D.C.; Supervision – H.O.O., F.S.U., M.B.U., F.N.D.C.; Fundings – H.O.O., F.S.U., M.B.U., F.N.D.C.; Materials – H.O.O., F.S.U., M.B.U.; Data collection &/or processing – F.S.U., M.B.U.; Analysis and/or interpretation – H.O.O., F.N.D.C.; Literature search – H.O.O., F.N.D.C., F.S.U.; Writing – H.O.O., F.N.D.C.; Critical review – H.O.O., F.N.D.C.

Peer-review: Externally peer-reviewed.

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