INTERNATIONAL JOURNAL OF
MEDICAL BIOCHEMISTRY

DOI: 10.14744/ajh.2025.94546
Int J Med Biochem 2025;8(3):192–198

Research Article

Immature granulocyte, immature granulocyte-lymphocyte ratio, and other hematological inflammatory parameters in Alzheimer disease

Dilek Yegin, Seniz Korkmaz

Central Laboratory, Bursa City Hospital, Bursa, Türkiye

Abstract

Objectives: Persistent overactivation of inflammatory responses has been associated with various neurodegenerative disorders, including Alzheimer disease. This study aimed to investigate whether parameters derived from complete blood count, such as white blood cell populations, platelet counts, and hemogram-derived parameters like platelet-lymphocyte ratio, immature granulocyte-lymphocyte ratio, systemic immune-inflammation index, and platelet-neutrophil ratio, which can be easily detected without additional cost, could have diagnostic value in the pathogenesis of Alzheimer disease. Additionally, the co-occurrence rates of Alzheimer disease with other diseases (such as Parkinson disease, anxiety, diabetes mellitus, cancer, osteoporosis and kidney disease) were analyzed.

Methods: Complete blood count data of 231 patients diagnosed with Alzheimer disease after pre-screening were retrospectively reviewed. Complete blood count parameters were generated using hemogram device data. Platelet-lymphocyte ratio, immature granulocyte-lymphocyte ratio, systemic immune-inflammation index, and platelet-neutrophil ratio were calculated using neutrophil, lymphocyte, and platelet counts. 593 patients diagnosed with Alzheimer disease during pre-screening were retrospectively screened again and Alzheimer disease comorbidities were analyzed.

Results: The immature granulocyte, immature granulocyte %, immature granulocyte-lymphocyte ratio values were found to be statistically significantly higher in Alzheimer's patients compared to the control group. However, the receiver operating characteristic analysis did not provide sufficient discrimination. The diseases accompanying Alzheimer disease were determined and the numbers found were expressed as percentages. The most common diseases were anxiety disorder, vitamin D deficiency and hypertension, respectively. The comorbidity with Parkinson disease was found to be 13.8%.

Conclusion: Since immature granulocyte, immature granulocyte %, immature granulocyte-lymphocyte ratio values, which do not require extra cost and can be easily detected with complete blood count, were determined to have discriminatory value in some diseases, we hope that our study will guide future research.

Keywords: Alzheimer disease, anxiety, immature granulocyte, immature granulocyte-lymphocyte ratio, parkinson disease

How to cite this article: Yegin D, Korkmaz S. Immature granulocyte, immature granulocyte-lymphocyte ratio, and other hematological inflammatory parameters in Alzheimer disease. Int J Med Biochem 2025;8(3):192–198.

Alzheimer disease (AD) is the most common form of dementia, accounting for 60–70% of dementia patients [1]. AD is defined by the buildup of abnormal protein aggregates, such as amyloid plaques made of beta-amyloid peptides and neurofibrillary tangles resulting from hyperphosphorylated tau protein [2]. The prolonged overactivation of pro-inflammatory responses has been linked to various neurodegenerative disorders, including AD [3]. Neuroinflammation plays a role in various neurological and behavioral disorders, including AD, Parkinson disease, and depression. The previously held view that the brain is an "immune-privileged" organ has shifted, based on more recent findings, towards an understanding that recognizes the significant connection between the immune response in the central nervous system and the rest of the body [4]. It is

Address for correspondence: Dilek Yegin, MD. Central Laboratory, Bursa City Hospital, Bursa, Türkiye
Phone: +90 533 657 61 57 **E-mail:** dyegin2@gmail.com **ORCID:** 0000-0001-9933-5297
Submitted: January 13, 2025 **Revised:** March 11, 2025 **Accepted:** April 02, 2025 **Available Online:** June 17, 2025

now widely recognized that systemic inflammation can initiate or worsen the brain's inflammatory state, thereby contributing to chronic neuroinflammation and neurodegeneration [5]. Increased immature granulocyte (IG) counts in peripheral blood are a sign of improved bone marrow activity [6] Although IGs are typically absent in peripheral blood, their levels rise in conditions like bacterial sepsis, inflammation, trauma, cancer, steroid therapy, and myeloproliferative disorders [7]. Some studies have shown that IGs are more effective than markers like C-reactive protein and neutrophil-to-lymphocyte ratio (NLR) in determining the severity of infection [8]. Intensive research is being conducted to diagnose and prevent the disease before symptomatic period [9]. Early detection for the prevention and treatment of AD is highly clinically significant. Blood samples, in contrast to cerebrospinal fluid, are easier to collect and can be taken more frequently. As a result, identifying potential early diagnostic markers in blood is particularly crucial [10]. The platelet-lymphocyte ratio (PLR), platelet-neutrophil ratio (PNR), systemic immune-inflammation index (SII), and Immature granulocyte-lymphocyte ratio (IGLR) are values commonly used in clinical practice based on blood cell counts and are also recognized as novel inflammatory markers [11, 12]. The platelet-lymphocyte ratio (PLR) can be used to assess platelet activation induced by inflammatory-coagulation reactions, severe coagulation disorders, and systemic inflammatory responses. The systemic immune-inflammation index (SII) is a novel biomarker for malignancy and inflammatory diseases [13].

This study aimed to investigate the role of white blood cell (WBC) populations (IG%, IG count), lymphocyte percentage and count, platelet parameters (platelet count, mean platelet volume: MPV, platelet large ratio: P-LCR, platecrit: PCT) and derived hematological parameters such as PLR, PNR, IGLR and SII as potential biomarkers in the differential diagnosis of AD. Additionally, the co-occurrence rates of AD with other diseases (such as Parkinson disease, anxiety, diabetes mellitus, cancer, osteoporosis and kidney disease) were analyzed.

Materials and Methods

The study was approved by the Bursa City Hospital Clinical Research Ethics Committee (Date 01/02/2023, No: 2023-3/4) The study was designed in accordance with the Helsinki Declaration.

This current study was designed as a retrospective study. During the preliminary screening, patient data records of 593 patients diagnosed with AD between 01.01.2023 and 28.02.2022 were retrospectively reviewed. Patients diagnosed with "early-onset Alzheimer disease" were not included in the study. In addition, subjects with acute/chronic infectious diseases, systemic inflammatory disorders, diseases affecting hematological parameters, thyroid dysfunctions, cerebrovascular diseases, hematological malignancies, kidney failure, malignancies, and those with positive C-reactive protein were excluded. Complete blood count (CBC) data of 231 patients obtained after screening were examined. A total of 51 (45.1%) men and 62 (54.9%) women were included in the study. Characteristics such as age, sex, and disease diagnosis codes (ICD-10: International Statistical Classification of Diseases and Related Health Problems) were obtained from patient records. The number of healthy controls was determined as 113 using the G*Power statistical program version 3.1.9.4 (University of Düsseldorf, Germany). CBC parameters were measured using the Sysmex XN-10 Automated Hematology Analyzer (Kobe 651-0073 Japan) in the laboratory with EDTA-containing hemogram tubes. Parameters measured included platelet (PLT) count, platelet distribution width (PDW), mean platelet volume (MPV), plateletcrit (PCT) value, and white blood cell (WBC), neutrophil (NEUT), lymphocyte (LYMPH), monocyte (MONO), basophil (BASO), eosinophil (EO), and immature granulocyte (IG) counts from the CBC analysis. PLR (platelet-lymphocyte ratio), IGLR (immature granulocyte-lymphocyte ratio), SII (systemic immune-inflammation index), and PNR (platelet-neutrophil ratio) were calculated using neutrophil, lymphocyte, and platelet counts. The calculations were made as follows: PLR=Platelet count / Lymphocyte count, PNR=Platelet count / Neutrophil count, IGLR=Immature Granulocyte count/ Lymphocyte count, SII=Systemic Immune-Inflammation Index=(Platelet×Neutrophil) / Lymphocyte. The results were compared with those of the healthy control group.

During the pre-screening, 593 patients diagnosed with AD were retrospectively scanned again, and this time, the diseases accompanying AD were analyzed. Disease diagnosis codes (ICD-10: International Statistical Classification of Diseases and Related Health Problems) were obtained from the patient file data records. Thus, the diseases accompanying AD and their numbers were determined. In addition, this number was expressed as a percentage by comparing it to the total number of patients (total 593).

Statistical analysis

Continuous variables were expressed as mean±standard deviation, and categorical data as numbers and percentages. Normality analyses of continuous variables were performed using the Kolmogorov-Smirnov Goodness-of-Fit Test. For intergroup analyses of normally distributed data, the Student's T-test was used, and for non-normally distributed data, the Mann-Whitney U Test was applied. Categorical data comparisons were made using the chi-square test. Cutoff values for IG, IG%, and IGLR in Alzheimer patients were examined using Receiver Operating Characteristics (ROC) curve analysis. If significant threshold values were present, the sensitivity, specificity, positive, and negative predictive values of these thresholds were calculated. A Type 1 error level below 5% was considered to indicate that the test's diagnostic value was statistically significant. Analyses were conducted using IBM SPSS version 27.0 (IBM Corporation, Armonk, NY, USA), and a p-value of <0.05 was accepted as the threshold for statistical significance.

Results

There was no statistically significant difference in terms of age and gender between the patient and control groups (p=0.131 and p=0.118, respectively) (Table 1).

Table 1. Comparison of AD and control groups in terms of age and gender

	Control group (n=113)		Alzheimer group (n=231)		p
	n	%	n	%	
Age (years) (mean±SD)	70.33±8.71		71.39±4.31		0.131*
Gender					
Female	62	54.9	147	63.6	0.118**
Male	51	45.1	84	36.4	

*: Student's t test, **: Chi-square test

In AD, IG, IG%, and IGLR values were found to be statistically significantly higher compared to the control group (p=0.002, p=0.001, and p=0.016, respectively). No statistically significant differences were found between the groups in terms of WBC, NEUT, LYMPH, MONO, EO, BASO, MONO%, EO%, PLT, PDW, MPV, P-LCR, PCT, NEUT%, LYMPH%, BASO%, PLR, PNR, and SII values (p>0.05) (Table 2). IG, IG%, and IGLR values were found to be statistically significantly higher compared to the control group (Table 2).

However, the ROC analysis did not provide sufficient discrimination (Table 3) (Fig. 1).

The number of comorbidities of AD with other diseases (such as Parkinson disease, anxiety, diabetes mellitus, cancer,

Table 2. Comparison of some blood parameters between the groups

Parameters	Control group (n=113)	Alzheimer group (n=231)	p
WBC [median (min-max)]	6.5 (4.6–10.7)	6.9 (4.1–12.4)	0.054*
NEUT [median (min-max)]	3.8 (2.4–6.4)	4.1 (1.8–8.3)	0.112*
LYMPH (Mean±SD)	2.04±0.47	2.09±0.46	0.370**
MONO [median (min-max)]	0.53 (0.26–0.81)	0.54 (0.28–1.03)	0.376*
EO [median (min-max)]	0.14 (0.02–0.55)	0.14 (0.0–0.81)	0.816*
BASO [median (min-max)]	0.04 (0.01–0.14)	0.04 (0.0–0.11)	0.745*
IG (Mean±SD)	0.018±0.008	0.025±0.029	0.002**
MONO% [median (min-max)]	7.9 (4.7–12.8)	8.0 (4.5–13.9)	0.814*
EO% [median (min-max)]	2 (0.3–8.0)	2.1 (0.0–10.1)	0.989*
PLT [median (min-max)]	244 (152 - 381)	237 (127 - 474)	0.983*
PDW [median (min-max)]	12.1 (8.4–22.9)	11.9 (8.3–21.5)	0.736*
MPV [median (min-max)]	10.4 (8.7–13.9)	10.4 (8.4–13.8)	0.742*
P-LCR [median (min-max)]	28.4 (14.9–55.6)	29 (12.1–54.4)	0.607*
PCT [median (min-max)]	0.25 (0.16–0.39)	0.25 (0.15–0.47)	0.941*
NEUT% [median (min-max)]	57.7 (42.3–71.6)	59.9 (43.2–73.8)	0.501*
LYMPH% [median (min-max)]	30.9 (19.5–44.7)	29.1 (14.1–44.5)	0.449*
BASO% [median (min-max)]	0.6 (0.1–2)	0.6 (0.0–1.4)	0.202*
IG% (Mean±SD)	0.26±0.11	0.33±0.31	0.001**
PLR [median (min-max)]	122 (64.0–218.2)	113.1 (47.6–305.6)	0.458*
PNR [median (min-max)]	62.4 (30.2–106.1)	59.7 (26.1–136.3)	0.278*
IGLR [median (min-max)]	0.0089 (0.00–0.023)	0.0099 (0.00–0.23)	0.016**
SII [median (min-max)]	455.6 (168.7–1369.4)	493.2 (137.3–1511.8)	0.470*

*: Mann Whitney U test, **: Student's t test. WBC: White blood cell, NEUT: Neutrophil, LYMPH: Lymphocyte, MONO: Monocyte, EO: Eosinophil, BASO: Basophil, IG: Immature granulocyte, PLT: Platelet, PDW: Platelet distribution width, MPV: Mean platelet volume, P-LCR: Platelet large cell ratio, PCT: Platecrit, PLR: Platelet-lymphocyte ratio, PNR: Platelet-neutrophil ratio, IGLR: Immature granulocyte-lymphocyte ratio, SII: Systemic immune-inflammation index, SD: Standard deviation.

Table 3. ROC analysis results and some cut-off values for IG, IG%, and IGLR in AD

	Diagnostic test					ROC curve		
	Cut-off	Sensitivity	Specificity	PPV	NPV	AUC	CI 95%	p
IG	≥0.020	70.4	36.3	69.2	37.6	**0.591**	0.530–0.652	**0.006***
IG %	≥0.25	64.3	46.0	70.8	38.8	**0.598**	0.537–0.659	**0.003***
IGLR	≥0.009	59.6	49.1	70.6	37.2	**0.580**	0.518–0.641	**0.016***

*: ROC analysis. ROC: Receiver operating characteristics; IG: Immature granulocyte; IGLR: Immature granulocyte/lymphocyte ratio; AD: Alzheimer disease; PPV: Positive predictive value; NPV: Negative predictive value; AUC: Area under the curve, CI: Confidence interval.

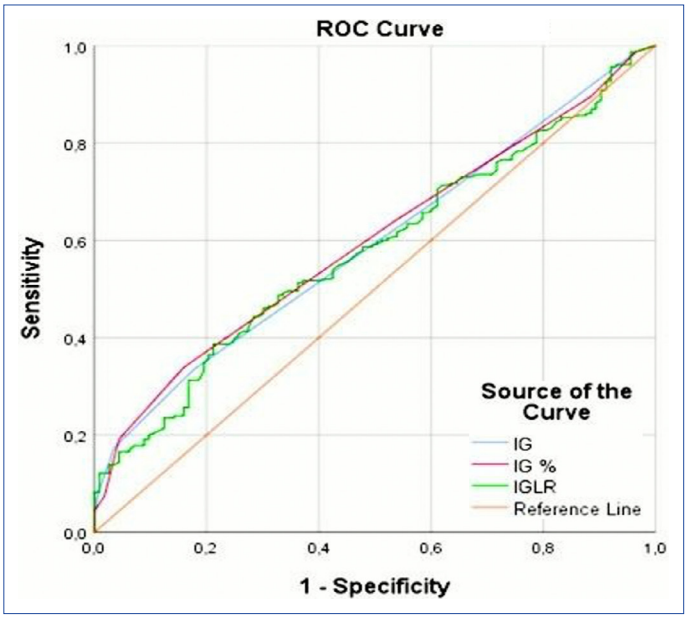

Figure 1. ROC Curve Graph For IG, IG%, and IGLR Values in AD.

ROC: Receiver operating characteristics; IG: Immature granulocyte; IGLR: Immature granulocyte/lymphocyte ratio; AD: Alzheimer disease.

osteoporosis and kidney disease) was determined. The numbers found were presented as a percentage by comparing to a total of 593 patients (Table 4).

Table 4. Numbers and rates of comorbidities in 593 patients diagnosed with AD

Diseases accompanying AD	Number of patients	% of the number of patients
Anxiety disorder	348	58.7
Vitamin D deficiency	321	54.1
Hypertension	299	50.4
Diabetes mellitus	180	30.4
Vitamin B-12 deficiency	161	27.2
Iron deficiency	86	14.5
Parkinson disease	82	13.8
Hyperlipidemia	72	12.1
Depressive episodes	70	11.8
Kidney failure	61	10.3
Thyroid disorders	55	9.3
Epilepsy	38	6.4
Delirium	35	5.9
Folic acid deficiency	33	5.6
Psychosis	30	5.1
Osteoporosis	28	4.7
Malignancy	24	4.0
Bipolar disorder	7	1.2
Migraines	5	0.8
Multiple sclerosis	2	0.3

AD: Alzheimer disease.

Discussion

AD, characterized by a progressive decline in memory and cognitive abilities, is the most common neurodegenerative disorder and significantly impairs the daily activities of elderly individuals [14]. Inflammation is believed to play a key role in the development of AD [15]. Given the role of inflammation in AD pathology, we proposed that routine blood parameters could hold diagnostic significance in AD. This study aimed to investigate the levels of total white blood cell (WBC) populations (such as IG, lymphocytes, etc.), platelet counts, and hemogram-derived parameters such as PLR, PNR, IGLR, and SII in the differential diagnosis of dementia. Human inflammatory markers (PLR, SII etc.) have been noted to potentially be associated with various health risks, including cardiovascular and cerebrovascular diseases in the elderly [16]. Many studies have reported a decrease in lymphocyte count in AD [16, 17]. However, when we compared the lymphocyte count and percentage values of the group with AD in this study to the control group, no significant difference was found between them (p=0.370, p=0.449, respectively) (Table 2). Fu et al. [18], demonstrated that P-LCR was significantly elevated in AD patients compared with controls. They found an increasing trend in the rate of change in P-LCR with disease progression. They showed that P-LCR may be a risk factor for AD after adjusting for age, sex, APOE4, and body mass index. In this study, no statistically significant difference was found between the groups in terms of P-LCR value (p>0.05) (Table 2). Systemic inflammation can be evaluated using various biochemical or hematological indicators, which are typically measured in routine blood tests or calculated from those results [19]. The systemic immune-inflammation index (SII) is new inflammatory biomarker calculated by (platelet count×neutrophil count) / lymphocyte count [20, 21]. In a study by Van der Willik et al. [22], higher levels of GLR, PLR, and SII ratios in the general population were found to be associated with an increased risk of dementia. In this study, when we compared the PLR and SII values of patients with AD to the control group, no statistically significant difference was found between them (p=0.458 and p=0.470, respectively) (Table 2). In a study by Wang et al. [23], MPV and PDW were found to be significantly lower in patients with AD compared to controls. In this study, no statistically significant difference was found between AD and the control group in terms of DW, MPV, and PNR values (p>0.05) (Table 2). Previous studies have used immature granulocyte (IG) count, a marker of heightened bone marrow activity, and IG percentage (IG%), the ratio of IGs to total WBC, in conditions such as acute necrotizing pancreatitis, pyelonephritis, sepsis, thyroid malignancies, and renal cell carcinoma [24]. However, there appears to be limited research in the literature on the relationship between IG, which stands out as an early marker of inflammation, and AD. Some other studies related to immature granulocytes are as follows: Gülten et al. [25], found immature granulocytes valuable in predicting short-term mortality in acute myocardial infarction. Another study [26], conducted on migraine patients found that IG count and NLR levels were significantly higher compared to the healthy

control group. In a different study [27], IG was found effective in predicting severe bacterial infection in the pediatric population. In a study by Sengul et al. [28], IG count and IG% were found not to be useful in determining the severity of H. pylori and inflammation. Another study [29], on subacute thyroiditis showed that IG significantly decreased after treatment, and IG and NLR could be used to evaluate treatment response. In another study by Ünal Y. [30], IG count has been shown to be important in the diagnosis of AA and the differential diagnosis of CAA with a cutoff value of >0.104 (10^3/uL), with 93% sensitivity and 93.8% specificity. When evaluating this study group, at the cut-off point of IG≥0.020 (10^3/uL), sensitivity was found to be 70.4% and specificity was 36.3% (AUC: 0.591, p=0.006) (Table 3). In the research conducted by Ünal et al. [31], an increased IG% was identified as an effective and reliable marker for the early detection of acute cholecystitis severity. They demonstrated that IG% had significantly greater predictive accuracy for stage 3 acute cholecystitis compared to other stages, with an AUC of 0.95, sensitivity of 92.5%, specificity of 84.2%, PPV of 32.5%, and NPV of 99.3%. In this study, when comparing AD with the control group, the difference in IG% was significant (p=0.012) (Table 2). However, despite this observed significance, when evaluated by ROC analysis, it was found that the ROC analysis, as shown in Table 3 and Figure 1, did not have sufficient effectiveness. (Area under the curve (AUC): 0.598, sensitivity: 64.3%, specificity: 46.0%, positive predictive value (PPV): 70.8%, and negative predictive value (NPV):38,8%). Kubat et al. [32], investigated the immature granulocyte-lymphocyte ratio (IGLR) in patients with acute appendicitis and concluded that it was useful to distinguish between complicated and uncomplicated acute appendicitis groups. For the immature granulocyte-lymphocyte ratio: AUC: 0.782, sensitivity: 71.8%, specificity: 74.3%, and NPV:95%. When we look at this study group, the difference between AD and the control group was significant (p=0.015) (Table 2). However, as shown in Table 3, for IGLR, the values were determined as (AUC: 0.580, sensitivity: 60.0%, specificity: 53.6%, PPV: 89.6%, NPV: 16.8). Although a significant difference was found between the groups, when evaluated by ROC analysis, it was determined that this distinction did not have sufficient effectiveness.

Recent research has uncovered the relationships between neurodegenerative disorders such as AD and Parkinson disease with other chronic diseases. Many common comorbid medical conditions, such as cancer, diabetes, cardiovascular disease, stroke, stress, seizures, osteoporosis, and kidney disease, have been associated with AD and Parkinson disease [33]. Although there are phenotypic and neuropathological differences among dementia, Parkinson disease, multiple sclerosis, and motor neuron diseases, research thus far has indicated that these four conditions are influenced by the interplay of genetic and environmental factors, which similarly result in central nervous system and immune dysfunctions [34]. The highly correlated prevalences of these diseases suggest that they share common etiological factors [35]. Considering that many common comorbid medical conditions have been associated with AD, the rates of co-occurrence of other diseases in AD patients were also determined in this study. Among the 593 patients diagnosed with AD, we found that 58.7% (348 patients) had anxiety disorder, 54.1% had vitamin D deficiency (321 patients), 50.4% had hypertension (299 patients), 30.4% had diabetes mellitus (180 patients), 27.2% had vitamin B-12 deficiency (161 patients), 14.5% had iron deficiency (86 patients), 13.8% had Parkinson disease (82 patients), 12.1% had hyperlipidemia (72 patients), 11.8% had depressive episodes (70 patients), 10.3% had kidney failure (61 patients), 9.3% had thyroid disorders (55 patients), 6.4% had epilepsy (38 patients), 5.9% had delirium (35 patients), 5.6% had folic acid deficiency (33 patients), 5.1% had psychosis (30 patients), 4.7% had osteoporosis (28 patients), 4% had malignancy (24 patients), 1.2% had bipolar disorder (7 patients), 0.8% had migraine (5 patients), and 0.3% had multiple sclerosis (2 patients) (Tablo 4). The most common diseases were anxiety disorder, vitamin D deficiency and hypertension, respectively. The comorbidity with Parkinson's disease was found to be 13.8%. It was thought that determining the accompanying diseases would be useful in terms of providing a horizon for new research.

In this study, IG, IG%, and IGLR values were found to be significantly higher in the AD group compared to the control group. However, ROC analysis did not provide sufficient discrimination. Since IG, IG%, and IGLR values are easily detected by complete blood count, do not require additional costs, and provide rapid results, their significant elevation in AD, along with their diagnostic differentiation in other diseases, highlights the importance of further studies investigating the usability of these parameters in AD. We believe this study is a pioneering one and hope it serves as a guide for future research into the usability of these parameters.

Conclusion

Since IG, IG%, and IGLR values, which do not require extra cost and can be easily detected with complete blood count, were determined to have discriminatory value in some diseases, we hope that this study will guide future research.

Ethics Committee Approval: The study was approved by the Bursa City Hospital Clinical Research Ethics Committee (no: 2023-3/4, date: 01/02/2023).

Informed Consent: Informed consent was obtained from all participants.

Conflict of Interest Statement: The authors have no conflicts of interest to declare.

Funding: The authors declared that this study received no financial support.

Use of AI for Writing Assistance: No AI technologies utilized.

Authorship Contributions: Concept – D.Y., S.K.; Design – D.Y., S.K.; Supervision – D.Y., S.K.; Funding – D.Y., S.K.; Materials – D.Y., S.K.; Data collection and/or processing – D.Y., S.K.; Data analysis and/or interpretation – D.Y., S.K.; Literature search – D.Y.; Writing – D.Y.; Critical review – D.Y., S.K.

Peer-review: Externally peer-reviewed.

References

1. World Health Organization. Global action plan on the public health response to dementia 2017-2025. Available at: https://www.who.int/publications/i/item/global-action-plan-on-the-public-health-response-to-dementia-2017---2025. Accessed May 28, 2025.
2. Zhang Z, Yang X, Song YQ, Tu J. Autophagy in Alzheimer's disease pathogenesis: Therapeutic potential and future perspectives. Ageing Res Rev 2021;72:101464. [CrossRef]
3. Ozben T, Ozben S. Neuro-inflammation and anti-inflammatory treatment options for Alzheimer's disease. Clin Biochem 2019;72:87–9. [CrossRef]
4. Mukhara D, Oh U, Neigh GN. Neuroinflammation. Handb of Clin Neurol 2020;175:235–59. [CrossRef]
5. Butchart J, Holmes C. Systemic and central immunity in Alzheimer's disease: Therapeutic implications. CNS Neurosci Ther 2012;18(1):64–76. [CrossRef]
6. Georgakopoulou VE, Makrodimitri S, Triantafyllou M, Samara S, Voutsinas PM, Anastasopoulou A, et al. Immature granulocytes: Innovative biomarker for SARS CoV 2 infection. Mol Med Rep 2022;26(1):217. [CrossRef]
7. Jeon K, Lee N, Jeong S, Park MJ, Song W. Immature granulocyte percentage for prediction of sepsis in severe burn patients: A machine leaning-based approach. BMC Infect Dis 2021;21(1):1258. [CrossRef]
8. Ünal Y, Barlas AM. Role of increased immature granulocyte percentage in the early prediction of acute necrotizing pancreatitis. Ulus Travma Acil Cerrahi Derg 2019;25(2):177–182. [CrossRef]
9. Kumar A, Sidhu J, Lui F, Tsa JW. Alzheimer Disease. Treasure Island: StatPearls Publishing; 2024.
10. McDade E, Bateman RJ. Stop Alzheimer's before it starts. Nature 2017;547(7662):153–5. [CrossRef]
11. Liao M, Liu L, Bai L, Wang R, Liu Y, Zhang L. et al Correlation between novel inflammatory markers and carotid atherosclerosis: A retrospective case-control study. PLoS One 2024;19(5):e0303869. [CrossRef]
12. Alisik M, Erdogan UG, Ates M, Sert MA, Yis OM, Bugdayci G. Predictive value of immature granulocyte count and other inflammatory parameters for disease severity in COVID-19 patients. Int J Med Biochem 2021;4(3):143–9. [CrossRef]
13. Wang RH, Wen WX, Jiang ZP, Du ZP, Ma ZH, Lu AL, et al. The clinical value of neutrophil-to-lymphocyte ratio (NLR), systemic immune-inflammation index (SII), platelet-to-lymphocyte ratio (PLR) and systemic inflammation response index (SIRI) for predicting the occurrence and severity of pneumonia in patients with intracerebral hemorrhage. Front Immunol 2023;14:1115031. [CrossRef]
14. Morgan AR, Touchard S, Leckey C, O'Hagan C, Nevado-Holgado AJ; NIMA Consortium. Inflammatory biomarkers in Alzheimer's disease plasma. Alzheimer Dement 2019;15(6):776–87. [CrossRef]
15. Sayed A, Bahbah EI, Kamel S, Barreto GE, Ashraf GM, Elfil M. The neutrophil-to-lymphocyte ratio in Alzheimer's disease: Current understanding and potential applications. J Neuroimmunol 2020;349:577398. [CrossRef]
16. Li W, Li S, Shang Y, Zhuang W, Yan G, Chen Z, et al. Associations between dietary and blood inflammatory indices and their effects on cognitive function in elderly Americans. Front Neurosci 2023;17:1117056. [CrossRef]
17. Dong X, Nao J, Shi J, Zheng D. Predictive value of routine peripheral blood biomarkers in Alzheimer's disease. Front Aging Neurosci 2019;11:332. [CrossRef]
18. Fu J, Lai X, Zhang C, Wei Q, Chen X, Shang H. Correlation analysis of peripheral platelet markers and disease phenotypes in Alzheimer's disease. Alzheimer Dement 2024;20(6):4366–72. [CrossRef]
19. Sylman JL, Mitrugno A, Atallah M, Tormoen GW, Shatzel JJ, Tassi Yunga S. et al. The predictive value of inflammation-related peripheral blood measurements in cancer staging and prognosis. Front in Oncol 2018;8:78. [CrossRef]
20. Hu B, Yang XR, Xu Y, Sun YF, Sun C, Guo W. et al. Systemic immune-inflammation index predicts prognosis of patients after curative resection for hepatocellular carcinoma. Clin Cancer Res 2014;20(23):6212–22. [CrossRef]
21. Tong YS, Tan J, Zhou XL, Song YQ, Song YJ. Systemic immune-inflammation index predicting chemoradiation resistance and poor outcome in patients with stage III non-small cell lung cancer. J Transl Med 2017;15(1):221. [CrossRef]
22. Van der Willik KD, Fani L, Rizopoulos D, Licher S, Fest J, Schagen SB, et al. Balance between innate versus adaptive immune system and the risk of dementia: A population-based cohort study. J Neuroinflammation 2019;16(1):68. [CrossRef]
23. Wang RT, Jin D, Li Y, Liang QC. Decreased mean platelet volume and platelet distribution width are associated with mild cognitive impairment and Alzheimer's disease. J Psychiatr Res 2013;47(5):644–9. [CrossRef]
24. Barut O, Demirkol MK, Kucukdurmaz F, Sahinkanat T, Resim S. Pre-treatment delta neutrophil index as a predictive factor in renal cell carcinoma. J Coll Physicians Surg Pak 2021;31(2):156–61. [CrossRef]
25. Gülten S, Cihan Çalışgan N, Akyel S, Kukul Güven FM. Immature granulocyte in prediction of the short-term and long-term mortality of patients with acute myocardial infarction. Eur Res J 2023;9(2):338–47. [CrossRef]
26. Kocatürk İ, Gülten S. Immature granulocyte and other hematological inflammatory parameters in patients with migraine headache. Kastamonu Med J 2021;1(4):101–4. [CrossRef]
27. Pavare J, Grope I, Gardovska D. Assessment of immature granulocytes percentage to predict severe bacterial infection in Latvian children: An analysis of secondary data. Medicina (Kaunas) 2018;54(4):56. [CrossRef]
28. Sengul S, Kubat M, Guler Y, Calis H, Karabulut Z. Utility of immature granulocyte in severity of helicobacter pylori infection. Indian J Pathol Microbiol 2022;65(2):316–20. [CrossRef]
29. Topuz E, Tüzün D, Şahin M. Immature granulocytes and neutrophil-to-lymphocyte ratio as markers of treatment response in subacute thyroiditis. Arch Endocrinol Metab 2024;68:e23012. [CrossRef]
30. Ünal Y. A new and early marker in the diagnosis of acute complicated appendicitis: Immature granulocytes. Ulus Travma Acil Cerrahi Derg 2018;24(5):434–9. [CrossRef]

31. Ünal Y, Tuncal S, Küçük B, Barlas AM, Altıner S, Balık R, et al. An effective and reliable marker in grading the severity of acute cholecystitis: Increased immature granulocyte percentage. Ulus Travma Acil Cerrahi Derg 2022;28(12):1716–22. [CrossRef]
32. Kubat M, Şengül S. Value of neutrophil-to-platelet ratio, immature granulocyte-to-lymphocyte ratio, red blood cell distribution width-to-lymphocyte ratio in differentiating complicated acute appendicitis. Ulus Travma Acil Cerrahi Derg 2022;28(5):607–14. [CrossRef]
33. Jabir NR, Firoz CK, Baeesa SS, Ashraf GM, Akhtar S, Kamal W, et al. Summary of the link between Alzheimer's and Parkinson's disease and chronic diseases. CNS Neurosci Ther 2015;21(1):1–7. [CrossRef]
34. Tysnes OB, Storstein A. Epidemiology of Parkinson's disease. J Neural Transm (Vienna) 2017;124(8):901–5. [CrossRef]
35. James LM, Georgopoulos AP. High correlations among worldwide prevalences of dementias, Parkinson's disease, multiple sclerosis, and motor neuron diseases ındicate common causative factors. Neuroscience Insights 2022;17:26331055221117598. [CrossRef]