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Original Research

OXFORD

The association between erythrocyte sedimentation rate, C-reactive protein, other blood derived inflammatory markers, and age: a retrospective cross-sectional study

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ESR, CRP, Novel Indices, and Age.

Abstract

Background: For many years, it has been suggested that commonly used inflammatory markers such as erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) increase with age, and formulas have even been developed for this purpose. However, the validity of these formulas in modern, strictly healthy populations remains questionable. This study aimed to re-evaluate the relationship between aging and inflammatory markers ESR, CRP, and novel integrated indices—the Systemic Immune-Inflammation Index (SII), Systemic Inflammation Response Index (SIRI), and Pan-Immune-Inflammation Value (PIV)—in a large, healthy study sample.

Methods: A cross-sectional study was conducted on 5510 strictly screened healthy individuals (2984 females, 2526 males; age range 18–93). Participants with chronic diseases, medication use, or abnormal metabolic profiles were excluded. Hemoglobin (Hb) levels were restricted to 10–16 g/dL to control for viscosity. Integrated indices were calculated using standardized hematological formulas.

Results: The mean observed ESR of 15.3 mm/h was lower than the upper limits predicted by the Miller formula of 26.5 mm/h. In a multivariate regression, the change in ESR per g/dL increase in Hb was −2.05 (−3.06 to −1.04) and per decade of age older 1.00 (0.16–1.84). There was no evidence of an association between SII, SIRI, and PIV, and age.

Conclusions: New inflammation markers derived from hemogram (SII, SIRI and PIV), and CRP are not affected by age. ESR, however, increases slightly with age. Elevated ESR in elderly patients should be considered as significant as in younger patients.

Keywords aging, erythrocyte sedimentation rate, C-reactive protein, systemic immune-inflammation index, systemic inflammation response index, pan-immune-inflammation value

Key messages

- **What is already known on this topic** – Traditional clinical rules (such as Miller's formula) assume that erythrocyte sedimentation rate (ESR) substantially increases with physiological aging, though whether this and complete blood count–derived composite indices drift across healthy adult lifespans remains controversial.
- **What this study adds** – In a rigorously screened healthy cohort of 5510 individuals, Miller's formula overestimated upper normal ESR limits by 73.2%, with the modest observed ESR rise being primarily driven by physiological hemoglobin variations rather than intrinsic inflamm-aging. Novel cellular indices (SII, SIRI, and PIV) demonstrated complete age-independent stability across adulthood, showing virtually zero correlation with advancing age.
- **How this study might affect research, practice, or policy** – Outdated age-adjustment formulas for ESR should be abandoned to prevent missing true inflammatory pathology in older adults, while integrated indices (SII, SIRI, PIV) can be safely utilized without age adjustment.

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Introduction

The erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) remain the most frequently utilized biomarkers for detecting systemic inflammation, infection, and malignancy in clinical practice [1, 2]. Despite the development of more specific assays, the ESR and CRP continue to hold significant diagnostic value due to its cost-effectiveness and broad clinical utility [3, 4].

However, the interpretation of the ESR and CRP is notoriously complex, as it is influenced by a myriad of non-inflammatory factors including age, sex, obesity, smoking and blood cell concentration [5, 6]. For many years, it has been suggested that CRP and ESR increase with age, and formulas have even been developed for this purpose. For age-adjusting ESR formula has been guided by Miller, which suggests an upper limit of normal calculated as Age/2 for men and (Age+10)/2 for women [7]. The formula recommended by some researchers for CRP was Age/50 (mg/dL) for men and (Age/50) + 0.6 (mg/dL) for women [8]. In addition, it has been suggested that ESR does not change with age [3].

These formulas were based on the premise that aging is inherently associated with a chronic, low-grade inflammatory state, a phenomenon recently termed "inflamm-aging" [9, 10]. While historical studies suggested a linear rise in inflammatory markers with advancing age, modern evidence argues that these elevations in healthy seniors are often misinterpreted, potentially leading to diagnostic complacency and this leads to some inflammatory processes going undiagnosed [11–13].

Recent evidence suggests that the perceived age-related increase in ESR may not be a primary inflammatory process but rather a secondary effect of age-associated physiological changes, particularly the subtle decline in hemoglobin (Hb) levels and subsequent alterations in plasma viscosity [14, 15]. Furthermore, the emergence of integrated hematological indices, such as the Systemic Immune-Inflammation Index (SII) [16], Systemic Inflammation Response Index (SIRI) [17], and Pan-Immune-Inflammation Value (PIV) [18], has provided a more robust framework for assessing systemic immune-inflammatory status. Unlike traditional protein markers, these indices offer potentially greater stability across the human lifespan in the absence of disease [19]. The necessity for redefining diagnostic thresholds is further emphasized by the limitations of single-protein markers in aging populations [20, 21].

The aim of this study was to explore the association between ESR, CRP, novel integrated indices; SII, SIRI, and PIV, and age in an administrative dataset of blood tests in patients undergoing routine health checks.

Materials and methods

Study sample and selection criteria

This study was conducted by reviewing the hospital data system files of individuals who underwent routine health checks in the last 5 years, between January 2021 and December 2025. Ethical approval for the study was obtained from the University Scientific Research Ethics Committee (26.01.2026, Decision No. 2026/08). Informed consent was not required due to the retrospective nature of the research. The study was conducted in accordance with the Declaration of Helsinki and with the principles of Research and Publication Ethics.

To ensure the integrity of the study sample, participants were selected based on a rigorous screening of electronic health records derived from the hospital central administrative database, which serves a wide catchment population of mixed urban and suburban residents in Ankara, Turkey. The individuals included in this study sample voluntarily applied to the hospital check-up center or preventive medicine outpatient clinics for routine non-symptomatic physical health checks, ensuring they did not enter the system due to active acute or chronic clinical complaints.

The inclusion was restricted to asymptomatic individuals who met the following strictly defined biochemical and hematological criteria:

- **Hematological Stability:** Hemoglobin (Hb) levels strictly between 10.0 and 16.0 g/dL to control for the impact of anemia or polycythemia on erythrocyte aggregation [14].
- **Metabolic Profile:** Fasting Blood Glucose <100 mg/dL and HbA1c <6.5% to exclude both undiagnosed diabetes and pre-diabetic inflammatory states [21, 22].
- **Lipid Panel:** Total Cholesterol <200 mg/dL and Triglycerides <200 mg/dL, hypercholesterolemia significantly accelerates ESR, especially in elderly men [23].
- **Organ Function:** Normal renal function (Serum Creatinine <1.2 mg/dL) and hepatic function (aspartate aminotransferase [AST]/alanine aminotransferase [ALT] levels within laboratory reference ranges)[24].
- **Thyroid Status:** Normal Thyroid Stimulating Hormone (TSH) levels (0.4–4.0 mIU/L), as thyroid dysfunctions are known to alter ESR and plasma protein concentrations [25].
- **Infectious Serology:** Only participants with confirmed negative results for HBsAg and anti-hepatitis C virus (anti-HCV) were included to eliminate potential chronic viral hepatitis-induced protein changes.

Beyond the biochemical thresholds, individuals were excluded if they had:

1) Any history of malignancy or chronic autoimmune/inflammatory disorders.
2) Evidence of active infection at the time of sampling (e.g. elevated white blood cell count or acute clinical symptoms).
3) Current use of medications known to affect inflammatory markers, including NSAIDs, corticosteroids, statins, or hormonal therapies.
4) Pregnancy [26, 27].
5) Hyperproteinemia [28].

Laboratory procedures

Venous blood was collected into EDTA tubes for complete blood count and ESR, and into gel-separator tubes for biochemical assays. ESR was determined by the Westergren method, and CRP was measured using high-sensitivity turbidimetry. All novel indices (SII, SIRI, PIV) were derived from the same standardized hematological run to ensure internal consistency.

Calculation of integrated inflammatory indices

The novel integrated immune-inflammation indices were calculated using the following standardized formulas:

1) Systemic Immune-Inflammation Index (SII): (Platelets × Neutrophils)/Lymphocytes.

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Table 1 Demographic and laboratory characteristics of the healthy study sample stratified by gender.

	Mean (Standard deviation)		
Patient characteristic	**Female (n = 2984)**	**Male (n = 2526)**	**Total (N = 5510)**
Age (Years)	46.2 (15.1)	45.8 (14.8)	46.0 (14.9)
Hemoglobin (g/dL)	13.1 (1.1)	14.9 (1.0)	13.9 (1.4)
ESR (mm/hour)	17.2 (9.8)	13.1 (8.2)	15.3 (9.3)
CRP (mg/L)	4.5 (5.9)	4.1 (5.2)	4.3 (5.6)
SII Index	482.4 (215.1)	445.6 (202.4)	465.5 (209.8)
SIRI Index	1.12 (0.65)	1.24 (0.72)	1.18 (0.68)
PIV Index	315.8 (185.4)	298.2 (174.6)	307.7 (180.5)

ESR: Erythrocyte Sedimentation Rate, CRP: C-Reactive Protein, SII: Systemic Immune-Inflammation Index, SIRI: Systemic Inflammation Response Index, PIV: Pan-Immune-Inflammation Value.

2) Systemic Inflammation Response Index (SIRI): (Neutrophils × Monocytes)/Lymphocytes.
3) Pan-Immune-Inflammation Value (PIV): (Neutrophils × Platelets × Monocytes)/Lymphocytes.

Statistical analysis

The normality of continuous variables was evaluated using the Kolmogorov–Smirnov test. Laboratory parameters were expressed as mean (standard deviation). Spearman's rank correlation coefficient (rho) was utilized to evaluate the relationships between age and each laboratory parameter. To satisfy the assumptions of multivariate linear regression models and manage potential skewness, C-reactive protein (CRP) values were natural logarithm-transformed (ln CRP). The near-normality of regression residuals and homoscedasticity assumptions were systematically verified. The performance of Miller's formula was evaluated by comparing the observed continuous ESR values against the formula-predicted upper limits of normal using a paired samples *t*-test. A direct comparison between male and female clinical subgroups was explicitly conducted using independent samples *t*-tests. All statistical analyses were performed using SPSS Statistics (version 26.0) and Python-based analytical libraries.

Results

Baseline characteristics of the study sample

The study included a total of 5510 healthy subjects with a mean age of 46.0 (14.9) years (range 18–93). The study sample consisted of 2984 (54.1%) females and 2526 (45.9%) males. The mean hemoglobin (Hb) level was 13.9 (1.4) g/dL, and the mean C-reactive protein (CRP) level was 4.3 (5.6) mg/L. Detailed demographic and laboratory characteristics, including the baseline values for integrated indices, are presented in Table 1.

Evaluation of the erythrocyte sedimentation rate and Miller's formula

The mean observed ESR for the entire study sample was 15.3 (9.3) mm/h, with females showing significantly higher values [17.2 (9.8) mm/h] compared to males [13.1 (8.2) mm/h; $P < .001$]. When observed continuous ESR values were directly compared against the individual upper limits predicted by Miller's formula (mean predicted limit: 26.5 mm/h), a statistically significant difference was identified via a paired samples t-test ($P < .001$), confirming that the traditional formula clinically overestimates normal thresholds by 73.2% in this strictly healthy sample. The distribution of ESR and natural logarithm-transformed CRP (ln CRP) values across age groups is visualized in Figs 1 and 2, respectively.

Correlation and regression analyses

Spearman's correlation analysis, summarized in Table 2, showed that age had a weak positive correlation with ESR (rho = 0.248, $P < .001$) and CRP (rho = 0.191, $P < .001$). In striking contrast, the integrated immune-inflammation indices exhibited remarkable stability across age groups. Specifically, the Pan-Immune-Inflammation Value (PIV) showed no significant correlation with age (rho = 0.001, $P = .964$), and the Systemic Immune-Inflammation Index (SII) remained entirely independent of chronological aging (rho = 0.000, $P = .971$). This age-independent stability of PIV, SII, and SIRI is clearly demonstrated in the combined regression plots in Fig. 3.

Multivariate linear regression analysis was performed to identify independent predictors of ESR. After adjusting for covariates, hemoglobin (Hb) emerged as a strong independent predictor of ESR, with an estimated slope coefficient of −2.05 (95% CI: −3.06 to −1.04, $P < .001$). Conversely, the independent effect of age per decade older remained controlled at 1.00 (95% CI: 0.16 to 1.84, $P = .021$).

Discussion

Our study, involving a large study sample of 5510 patients, confirms that age and gender have an effect on inflammatory marker levels. However, this effect is not as significant as claimed. In particular, the progressive increase in ESR levels can be explained not by the concept of "aging with inflammation," a chronic, low-grade inflammatory state associated with aging, as proposed by Franceschi *et al.* [9, 10], but by other age-related physiological changes, particularly the subtle decline in hemoglobin levels and subsequent alterations in plasma viscosity as proposed by Pawlotsky *et al.* and Narang *et al.* [14, 15].

Age-dependent variations in ESR and CRP

The most striking finding of our study is that the correlation between age and ESR is low-to-moderate (rho = 0.248), and the correlation between age and CRP is very weak (rho = 0.191). This indicates that ESR is more sensitive to cumulative physiological changes that naturally occur during the aging process. While CRP is a direct product of the innate immune response and acute phase reaction [12, 13],

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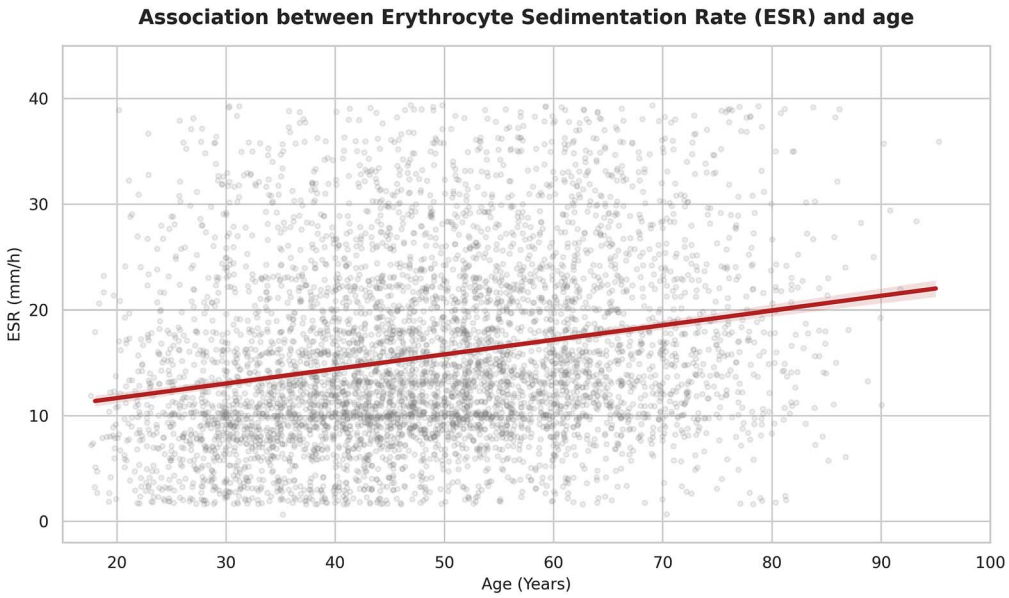


Figure 1 Scatter plot with a linear regression line illustrating the association between erythrocyte sedimentation rate (ESR) and age in the healthy study sample (n = 5510). The shaded area represents the 95% confidence interval, demonstrating a slight, progressive upward trend with advancing age

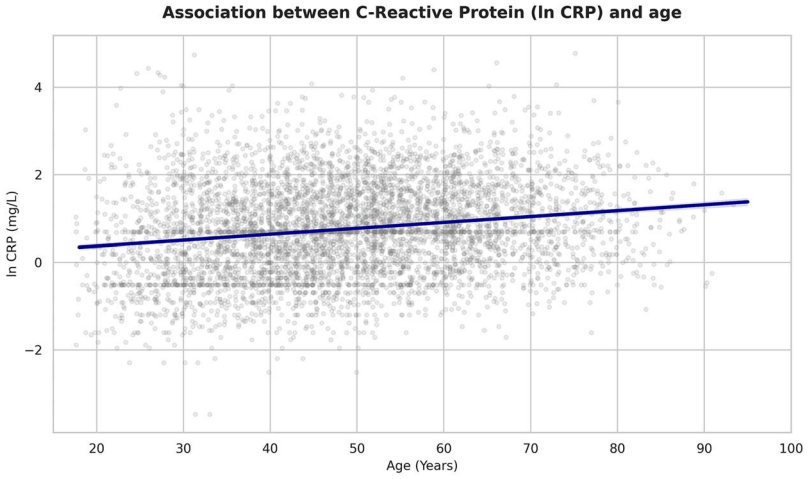


Figure 2 Scatter plot with a linear regression line illustrating the association between natural logarithm-transformed C-reactive protein (ln CRP) and age in the healthy study sample (n = 5510). The shaded area represents the 95% confidence interval, demonstrating a remarkably flat and independent chronological trajectory

Table 2 Spearman's rank correlation analysis between age and conventional inflammatory markers (ESR, CRP) and novel inflammatory indices (SII, SIRI, PIV).

Parameter	Female (rho)	*P*-value	Male (rho)	*P*-value	Total (rho)	*P*-value
ESR vs. Age	0.215	< .001	0.306	<.001	0.248	<.001
CRP vs. Age	0.215	< .001	0.145	<.001	0.191	<.001
SII vs. Age	−0.052	.003	0.071	.001	0.000	.971
SIRI vs. Age	−0.013	.436	0.113	<.001	0.029	.031
PIV vs. Age	−0.041	.017	0.072	.001	0.001	.964

ESR: Erythrocyte Sedimentation Rate, CRP: C-Reactive Protein, SII: Systemic Immune-Inflammation Index, SIRI: Systemic Inflammation Response Index, PIV: Pan-Immune-Inflammation Value

ESR is an indirect measure influenced by plasma protein composition and fibrinogen levels that naturally increase with age [20, 21]. This is further evidenced by our data showing that median ESR values of 9 mm/h in the 18–29 age group consistently rise to 26 mm/h in individuals over 80 years of age.

Sex-related disparities

We observed consistently higher ESR and CRP levels in females across almost all age groups. These sex-related differences have been widely documented, Alende-Castro *et al.* [5] and Wener *et al.* [8] noted that

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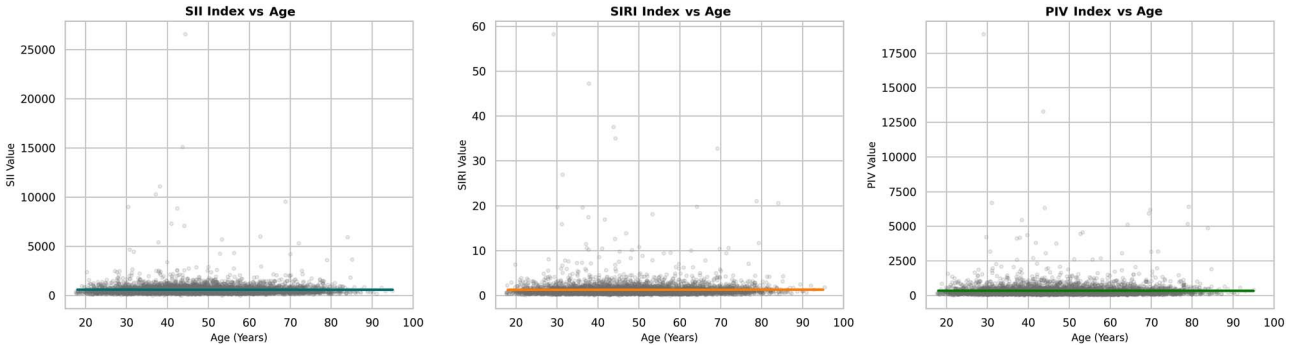


Figure 3 Multi-panel regression scatter plots demonstrating the chronological stability of novel integrated hematological indices across the human lifespan: (A) age versus systemic immune-inflammation index (SII), (B) age versus systemic inflammation response index (SIRI), and (C) age versus pan-immune-inflammation value (PIV). All three cell-derived cellular markers display complete horizontal stability across age groups

women tend to have higher baseline inflammatory markers, likely due to hormonal influences and lower average hemoglobin levels. Understanding these sex-specific variations is crucial to avoid the misinterpretation of laboratory results in clinical practice.

The clinical necessity of age-adjustment

Perhaps the most critical consequence of our findings is that ESR shows a slight increase with aging, in contrast to the "simple rule" proposed by Miller *et al.* [7], which suggests calculating the upper limit of normal ESR as (Age/2) for men and (Age + 10)/2 for women. Furthermore, contrary to Steinvil *et al.* [3] that claim that ESR levels do not change with aging, our study shows a slight increase in ESR levels with aging. Our data confirm that "normal" values in the elderly can often exceed the standard laboratory limits set for younger populations. However, as suggested by Cankurtaran *et al.* [11], it is very important that high ESRs in the elderly have similar pathological significance to those in younger people.

CRP as a marker of subclinical processes

Although CRP showed a weaker correlation with age than ESR, its elevation in older populations remains clinically significant. As noted by Bray *et al.* [2], even minor "physiological" elevations in CRP might reflect underlying subclinical conditions, such as metabolic syndrome or atherosclerosis [29, 30]. Therefore, while ESR may be more reflective of the protein shifts of aging, CRP remains a more specific indicator for monitoring acute inflammatory responses [12, 13].

Study limitations

Despite the strength of our large-scale dataset (n = 5510), several limitations should be acknowledged. First, the study design is retrospective and unicentric, which may limit the generalizability of our findings to diverse ethnic populations or different geographic regions. Although our sample reflects a large segment of the sample, ethnic variations in CRP levels—as documented in the literature—could not be explored in this single-center study.

Second, while we implemented rigorous exclusion criteria to ensure a "strictly healthy" study sample, the retrospective nature of the data relies on existing medical records, and the presence of occult (subclinical) conditions cannot be entirely ruled out.

Third, the analysis did not account for certain lifestyle factors such as dietary habits, physical activity levels, and smoking status, all of which have been suggested to modulate low-grade systemic inflammation. Furthermore, Body Mass Index (BMI) data was unavailable; given the known association between adiposity and CRP, this remains a significant covariate that was not adjusted for in our correlation analysis. However, since all participants underwent rigorous screening to exclude abnormal metabolic profiles, glucose intolerance, and dyslipidemia, the potential confounding effect of obesity-induced subclinical inflammation was minimized.

Additionally, while we controlled for hemoglobin levels, other factors influencing plasma viscosity, such as hydration status and specific globulin fractions, were not available for all participants. Lastly, as a cross-sectional analysis of retrospective data, these findings demonstrate associations rather than longitudinal trajectories of inflammatory markers within the same individuals over time.

However, we believe that the unprecedented sample size of 5510 individuals and the stringent multi-stage screening process significantly mitigate these limitations, providing a robust baseline for redefining inflammatory reference intervals in the modern era.

Conclusion

In conclusion, this study, conducted on a strictly screened study sample of 5510 healthy individuals, provides robust evidence that the widely used Miller's formula (Age/2 rule) has lost its clinical validity in modern medical practice. Our analysis reveals that this outdated formula leads to a significant overestimation of normal erythrocyte sedimentation rate thresholds by 73.2%. Yes, ESR does increase somewhat with age, but not at the rates claimed. Therefore, it can lead to serious consequences by causing the early diagnosis of some diseases to be missed in the elderly.

These findings demonstrate that the perceived age-related rise in ESR is not an intrinsic inflammatory phenomenon but is primarily driven by physiological variations in hemoglobin (Hb) levels and subsequent changes in plasma viscosity. In contrast, the novel integrated immune-inflammation indices—specifically SII, SIRI, and PIV—exhibit remarkable stability across the human lifespan, remaining entirely independent of chronological aging in the absence of disease.

Based on these results, we propose the following clinical recommendations:

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1) The routine use of Miller's formula for age-adjusting ESR should be reconsidered and potentially phased out in favor of more contemporary, evidence-based reference intervals.
2) Clinicians should prioritize integrated cellular indices (SII, SIRI, PIV) over traditional protein-based markers (ESR, CRP) when assessing systemic inflammation in elderly patients, as these indices do not require complex age-adjustment.
3) Future diagnostic guidelines must account for the impact of hemoglobin levels to prevent the misinterpretation of ESR as a marker of pathology in healthy seniors.

By adopting these modern diagnostic frameworks, clinicians can achieve higher diagnostic accuracy, avoid misinterpretations of normal values in elderly patients, and contribute to the early diagnosis of disease states in elderly patients.

Author contributions

AOA conceived the study, participated in its design and coordination, performed the statistical analysis, and drafted the manuscript. All authors read and approved the final manuscript.

Conflicts of interest

The authors declare that they have no competing interests.

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Data availability

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki. Ethical approval for the study was obtained from the University Scientific Research Ethics Committee (Date: 26.01.2026, Decision No: 2026/08). Informed consent was waived by the Lokman Hekim University Scientific Research Ethics Committee due to the retrospective nature of the research.

Consent for publication

Not applicable.

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