

The prognostic value of serological indices at diagnosis in antineutrophil cytoplasmic antibody-associated vasculitis: a single-centre experience

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Abstract

Introduction: Antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV) represents a heterogeneous group of small-vessel vasculitides characterised by systemic inflammation and multi-organ involvement. This study aimed to investigate the prognostic and clinical significance of haematological and biochemical indices – namely the neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio (PLR), haemoglobin–albumin–lymphocyte–platelet (HALP) score, neutrophil-to-albumin ratio, systemic immune-inflammation index (SII), C-reactive protein-to-albumin ratio, and albumin-to-globulin ratio (AGR) – measured at the time of diagnosis. We sought to determine their associations with disease activity, organ involvement, and overall prognosis in patients with AAV.

Material and methods: A retrospective cohort study was conducted on 56 patients diagnosed with AAV between January 2020 and August 2025, according to the 2022 American College of Rheumatology/European Alliance of Associations for Rheumatology classification criteria. Demographic, clinical, and laboratory data were extracted from medical records. Inflammatory and nutritional indices were calculated at diagnosis, and their correlations with disease activity (Birmingham Vasculitis Activity Score – BVAS), prognosis (Five-Factor Score – FFS), organ involvement, relapse, and mortality were analysed using appropriate statistical tests, including Spearman correlation and receiver operating characteristic analysis.

Results: The mean age was 54.2 ±15.7 years, and 73.2% of patients had granulomatosis with polyangiitis. Renal and pulmonary involvements were most common. The relapse rate was 32%, and mortality was 12.5%. None of the indices showed significant predictive ability for relapse (area under the curve < 0.6). A moderate negative correlation was found between FFS and AGR ($p = -0.31$, $p = 0.02$) and between BVAS and HALP ($p = -0.33$, $p = 0.01$). The HALP and AGR were significantly lower in renal involvement, while PLR and SII were higher and HALP lower in pulmonary involvement.

Conclusions: Inflammatory and nutritional indices at diagnosis may not predict relapse in AAV but may reflect disease activity and organ-specific involvement. These easily accessible parameters could serve as complementary biomarkers for early risk stratification, warranting validation in large prospective studies.

Key words: ANCA-associated vasculitis, granulomatosis with polyangiitis (GPA), microscopic polyangiitis (MPA), inflammatory and nutritional indices, disease activity, prognosis, organ-specific involvement.

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Introduction

Antineutrophil cytoplasmic antibody (ANCA)-associated vasculitides (AAV) comprise a group of necrotising vasculitides that predominantly affect small vessels and include the subtypes granulomatosis with polyangiitis (GPA), microscopic polyangiitis (MPA), and eosinophilic GPA (EGPA). Although rare, these disorders can present with life-threatening clinical manifestations and involve multiple organ systems [1]. The pathogenesis of the disease is multifactorial and results from the interaction of genetic susceptibility, environmental factors, and immune dysregulation. The clinical course is highly heterogeneous; major organ involvement, such as the kidneys, lungs, and nervous system, is associated with severe morbidity and mortality. In long-term follow-up, mortality, relapse, and permanent organ damage are among the most serious outcomes; therefore, reliable biomarkers and scoring systems to predict prognosis play a critical role in clinical management.

To date, indices such as the Five-Factor Score (FFS) [2] and Birmingham Vasculitis Activity Score (BVAS) [3] have been widely used to assess disease activity and prognosis [4, 5]. However, these scoring systems are mostly based on clinical parameters and incorporate laboratory data to a limited extent. In recent years, increasing attention has been directed toward the prognostic value of easily calculable haematological and biochemical indices that reflect the systemic manifestations of inflammation [4]. Parameters such as the neutrophil-to-lymphocyte ratio (NLR) [5], platelet-to-lymphocyte ratio (PLR) [6], haemoglobin–albumin–lymphocyte–platelet (HALP) score [7], neutrophil-to-albumin ratio (NPAR) [8], systemic immune-inflammation index (SII) [9], C-reactive protein-to-albumin ratio (CAR) [10], and albumin-to-globulin ratio (AGR) [11] have been shown to be associated with mortality and disease activity in various rheumatologic and inflammatory disorders. Although data on the use of these parameters in AAV are limited, their easy accessibility and low cost highlight their potential applicability in clinical practice.

Neutrophil-to-lymphocyte ratio has been identified as a practical indicator reflecting inflammatory burden and prognosis in various rheumatologic and vasculitic diseases [5]. Several studies in patients with AAV have demonstrated that the NLR measured at the time of diagnosis may have prognostic value in predicting vasculitis activity and the risk of relapse [12]. Similarly, the PLR has been shown to correlate with vasculitis activity and has been proposed as an independent marker for predicting severe disease [4]. However, data regarding the prognostic value of other indices such as HALP, NPAR, SII, CAR, and AGR in AAV remain limited, and further studies in this area may provide valuable contributions to clinical

practice. Although most previous studies have evaluated these indices individually, it is increasingly recognised that each parameter reflects a distinct biological dimension of systemic inflammation, such as neutrophil activation (NLR, NPAR), platelet-driven inflammatory response (PLR, SII), acute-phase activity (CAR), and nutritional or immune reserve (HALP, AGR). These indices have also been applied in other rheumatic and inflammatory diseases – such as rheumatoid arthritis [13], systemic lupus erythematosus [14], and Behçet’s disease [15] – as markers of inflammation, prognosis, or organ involvement, further supporting their biological relevance. Evaluating these indices together, even without establishing a formal composite score, may therefore provide a more integrated representation of the inflammatory and immunonutritional status at disease onset. Given the marked clinical and biological heterogeneity of AAV, the simultaneous assessment of multiple indices has the potential to capture prognostic patterns or organ-specific phenotypes that might not be identified when relying on single parameters alone. This multidimensional approach may offer incremental prognostic value beyond conventional clinical scoring systems and supports the relevance of studying these indices collectively.

Inflammatory indices in AAV have been reported to be associated not only with disease activity but also with specific organ involvements. In particular, NLR and PLR have been correlated not only with vasculitis activity but also with major organ involvement. In the literature, NLR and PLR levels have been evaluated in relation to both BVAS and organ involvement, showing significantly higher values in patients with pulmonary, cardiac, or renal manifestations. The prognostic potential of inflammatory indices is not limited to AAV alone. Their associations have also been investigated in other small-vessel vasculitides, such as immunoglobulin A (IgA) vasculitis. In a study conducted in adult patients with IgA vasculitis, the NLR was found to be significantly associated with the presence of systemic involvement (renal and/or gastrointestinal), and a cutoff value for this parameter was proposed [16]. Furthermore, a meta-analysis on Henoch-Schönlein purpura demonstrated that both NLR and PLR were significantly associated with disease complications and systemic involvement [17]. These findings suggest that inflammatory indices may serve as potential biomarkers reflecting not only overall disease activity but also organ involvement across different types of small-vessel vasculitides.

In this context, our study aims to evaluate the relationship between haematological and biochemical indices and prognosis, particularly organ involvement, in AAV. It also seeks to investigate the correlation

of these parameters with disease activity and prognostic scores (BVAS, FFS) at the time of diagnosis. In doing so, the study highlights the potential of easily accessible markers for early risk stratification and prediction of organ-specific phenotypes in clinical practice.

Material and methods

Study design and patient selection

This study was conducted through a retrospective analysis of patients who were followed in our clinic with a diagnosis of AAV between January 2020 and August 2025. During the specified period, a total of 87 patient records were reviewed, and 56 patients with complete laboratory data available at the time of diagnosis were included in the study cohort. Demographic characteristics, clinical features, organ involvement, and laboratory parameters of the patients were obtained from the electronic medical record system.

Diagnostic criteria, clinical assessment and laboratory parameters

The diagnosis of AAV was confirmed based on the 2022 American College of Rheumatology/European Alliance of Associations for Rheumatology classification criteria [18, 19] together with clinical, serological, and histopathological findings. Disease activity at the time of diagnosis was assessed using the BVAS for Wegener’s Granulomatosis and BVAS version 3 [3], while prognosis was evaluated using the revised 2009 FFS [2]; all assessments were performed by an experienced rheumatologist specialising in systemic vasculitis. At the time of diagnosis, complete blood count, biochemical parameters, and acute-phase reactants were used to calculate NLR, PLR, HALP, NPAR, SII, CAR, and AGR indices according to methods previously described in the literature (Suppl. Table I). All laboratory parameters were obtained at the time of initial presentation and before the initiation of any treatment; none of the patients had received corticosteroids prior to sample collection. The overall follow-up duration was substantial; however, one patient had a follow-up period of less than 6 months due to late enrolment.

Statistical analysis

All analyses were performed using SPSS software (IBM Corp., Armonk, NY, USA, Version 22.0). Visual (histograms and probability plots) and analytical (Kolmogorov-Smirnov and Shapiro-Wilk’s test) methods were utilised to assess whether the variables were normally distributed. Normally distributed variables were summarised as mean $\pm$ standard deviation, non-normally distributed

variables as median (interquartile range [IQR]), and categorical variables as number (*n*) and percentage (%). Student’s *t*-test was applied for normally distributed variables, while the Mann-Whitney *U* test was used for non-normally distributed variables. Categorical variables were compared using the χ^2 test. To minimise the risk of overfitting given the modest sample size, the number of variables included in multivariate analyses was restricted and statistical assumptions were carefully verified. The relationships between inflammatory and nutritional indices and prognostic scores were assessed using Spearman’s correlation analysis, and the correlation coefficient (ρ) was reported. A positive coefficient indicated a direct association, whereas a negative coefficient indicated an inverse association between variables. The strength of the correlation coefficients was classified as follows: 0.00–0.10 negligible, 0.10–0.30 weak, 0.30–0.50 moderate, 0.50–0.70 strong, 0.70–0.90 very strong, and 0.90–1.00 nearly perfect correlation. Predictive performance for relapse was evaluated using receiver operating characteristic (ROC) curve analysis, and the area under the curve (AUC) with a 95% confidence interval was calculated. The AUC values were interpreted as follows: ≤ 0.599 , no predictive ability; 0.60–0.70, poor; 0.70–0.80, acceptable; 0.80–0.90, excellent; and > 0.90 , outstanding predictive capacity. A *p*-value of < 0.05 was considered statistically significant.

Bioethical standards

The study protocol was designed and conducted in accordance with the ethical principles outlined in the Declaration of Helsinki, ensuring the confidentiality of patient data. The study was approved by the Ethics Committee of the University of Health Sciences, Gülhane Training and Research Hospital, Ankara, Türkiye (date 21.10.2025 and decision number 2025/518).

Results

A total of 56 patients with AAV were included in the study. The mean age was 54.2 ± 15.7 years, and the median follow-up duration was 43 months (IQR: 19–73). Of the cohort, 42.9% were female, and the most common subtype was GPA (73.2%). Renal, pulmonary, and ear, nose, and throat (ENT) involvements were the most frequently observed organ manifestations; renal involvement was present in approximately two-thirds of the patients, while pulmonary and ENT involvements were observed in more than half. Life-threatening organ involvement was identified in 76.8% of the patients, the relapse rate was 32%, and the mortality rate was 12.5% (Table I). Laboratory parameters and inflammatory/nutritional index values are presented in Table II.

Table I. Demographic, clinical, and treatment characteristics of patients with ANCA-associated vasculitis

Variables	Total patients (n = 56)
Sex, female [n (%)]	24 (42.9)
Age, mean ±SD	54.2 ±15.7
Age of diagnosis, mean ±SD	50.4 ±15.9
At least one comorbidity [n (%)]	30 (53.6)
Follow-up duration [months], median (IQR)	43.4 (19–73)
Diagnosis	
Microscopic polyangiitis	15 (26.8)
Granulomatosis with polyangiitis	41(73.2)
ANCA type at diagnosis [n (%)]	
MPO-ANCA	19 (33.9)
PR3-ANCA	30 (53.6)
ANCA negative	7 (12.5)
Clinical manifestations [n (%)]	
Constitutional symptoms	23 (41.1)
Articular involvement	10 (17.9)
Skin involvement	9 (16.1)
CNS involvement	3 (5.4)
Peripheral nervous system involvement	7 (12.5)
Eye involvement	10 (17.9)
Ear-nose-throat involvement	32 (57.1)
Pulmonary involvement	35 (62.5)
Diffuse alveolar haemorrhage	12 (21.4)
Renal involvement	35 (62.5)
Pauci-immune glomerulonephritis	24 (42.9)
GIS Involvement	2 (3.6)
Life-threatening organ involvement [n (%)]	43 (76.8)
Five factor score ≥ 1 [n (%)]	43 (76.8)
BVAS score, median (IQR) (at the time of diagnosis)	8.0 (5.0–12.5)
Vasculitis Damage Index (VDI) ≥ 1 [n (%)]	33 (58.9)
Death during follow-up [n (%)]	7 (12.5)
Relapse [n (%)]	18 (32.1)
Treatment agents, ever [n (%)]	
Pulse steroid therapy	47 (83.9)
Cyclophosphamide	36 (64.3)
Methotrexate	13 (23.2)
Mycophenolate mofetil	21 (37.5)
Azathioprine	20 (35.7)
Rituximab	35 (62.5)
Plasmapheresis	22 (39.3)
Haemodialysis	14 (25.0)

ANCA – antineutrophil cytoplasmic antibody, BVAS – Birmingham Vasculitis Activity Score, GIS – gastrointestinal system, IQR – interquartile range, MPO – myeloperoxidase, PR3 – proteinase 3, SD – standard deviation, VDI – Vasculitis Damage Index.

Table II. Laboratory parameters and inflammatory/nutritional indices

Parameter	Median (IQR)
Haemoglobin [g/dl]	10.3 (3.7)
RDW-CV [%]	14.20 (2.07)
Platelet [× 10 ³ /μl]	352.5 (112.0)
White blood cell [× 10 ³ /μl]	10.23 (5.2525)
Neutrophil count [× 10 ³ /μl]	7.45 (5.08)
Lymphocyte count [× 10 ³ /μl]	1.33 (0.997)
Eosinophil count [× 10 ³ /μl]	0.135 (0.200)
Albumin [g/dl]	3.4 (0.88)
Total protein [g/dl]	6.8 (1.45)
CRP [mg/l]	104.5 (123)
ESR [mm/hour]	76 (43.5)
CAR	3.26 (4.15)
NLR	5.91 (5.68)
PLR	258.35 (234.65)
AGR	0.96 (0.38)
SII	2,090.00 (2,397.29)
HALP score	11.18 (19.57)
NPAR	2.25 (1.65)

AGR – albumin-to-globulin ratio, CAR – C-reactive protein-to-albumin ratio, CRP – C-reactive protein, ESR – erythrocyte sedimentation rate, HALP – haemoglobin–albumin–lymphocyte–platelet score, IQR – interquartile range, NLR – neutrophil-to-lymphocyte ratio, NPAR – neutrophil-to-albumin ratio, PLR – platelet-to-lymphocyte ratio, RDW-CV – red cell distribution width, coefficient of variation, SII – systemic immune-inflammation index.

Table V. Comparison of indices according to major organ involvement

Index, median (IQR)	Renal (+)	Renal (–)	p-value	Pulmonary (+)	Pulmonary (–)	p-value
CAR	4.00 (4.70)	2.47 (3.07)	0.139	3.39 (3.88)	2.07 (4.86)	0.220
NLR	6.22 (5.19)	4.88 (5.49)	0.173	7.69 (6.86)	5.31 (4.07)	0.173
PLR	282.22 (472.07)	205.78 (259.80)	0.168	311.54 (314.24)	218.12 (137.67)	0.040
AGR	0.88 (0.30)	1.00 (0.53)	0.004	0.95 (0.29)	1.0 (0.55)	0.279
SII	3,733.66 (5,284.71)	2,075.80 (3,025.10)	0.571	3,060.00 (3,390.10)	1,801.35 (1,190.21)	0.022
HALP	11.08 (10.14)	25.99 (32.92)	0.010	8.58 (14.32)	18.65 (19.67)	0.005
NPAR	2.50 (2.22)	2.10 (1.41)	0.526	2.38(2.52)	1.92 (1.49)	0.117

*p-values were obtained using the Mann-Whitney U test.
AGR – albumin-to-globulin ratio, CAR – C-reactive protein-to-albumin ratio, HALP – haemoglobin–albumin–lymphocyte–platelet score, IQR – interquartile range, NLR – neutrophil-to-lymphocyte ratio, NPAR – neutrophil-to-albumin ratio, PLR – platelet-to-lymphocyte ratio, SII – systemic immune-inflammation index.

In the prediction of relapse, all inflammatory indices (CAR, NLR, PLR, AGR, SII, HALP, NPAR) showed AUC values below 0.599, indicating no significant predictive performance (Table III). In the correlation analysis, a moderate negative correlation was found between FFS and AGR (ρ = –0.310, p = 0.02), and between BVAS and HALP

Table III. Evaluation of the predictive ability of inflammatory indices for relapse using receiver operating characteristic analysis

Variables	AUC	95% CI	p-value
CAR	0.519	0.381–0.655	0.820
NLR	0.523	0.385–0.658	0.786
PLR	0.526	0.388–0.661	0.752
AGR	0.523	0.386–0.659	0.779
SII	0.528	0.390–0.663	0.739
HALP	0.550	0.389–0.711	0.551
NPAR	0.531	0.393–0.665	0.713

AGR – albumin-to-globulin ratio, AUC – area under the curve, CAR – C-reactive protein-to-albumin ratio, CI – confidence interval, HALP – haemoglobin–albumin–lymphocyte–platelet score, NLR, neutrophil-to-lymphocyte ratio, NPAR – neutrophil-to-albumin ratio, PLR – platelet-to-lymphocyte ratio, SII – systemic immune-inflammation index.

Table IV. Spearman correlation between inflammatory/nutritional indices and Five-Factor Score (FFS) and Birmingham Vasculitis Activity Score (BVAS) scores

Index	FFS ρ (p)	BVAS ρ (p)
AGR	–0.310 (p = 0.020)	–
HALP	–	–0.337 (p = 0.010)
Other indices	Not significant	Not significant

AGR – albumin-to-globulin ratio, HALP– haemoglobin–albumin–lymphocyte–platelet score, ρ – Spearman’s correlation coefficient.

(ρ = –0.337, p = 0.01). No significant correlations were observed with other indices (Table IV).

When compared according to organ involvement, AGR (p = 0.004) and HALP (p = 0.010) were significantly lower in patients with renal involvement. In patients with pulmonary involvement, PLR and SII were significantly

higher, whereas HALP was markedly lower ($p = 0.040$, $p = 0.022$, and $p = 0.005$, respectively) (Table V).

Discussion

Our study provides insight into the prognostic value of inflammatory and nutritional indices in AAV. Although these parameters did not demonstrate significant predictive power for relapse, their associations with organ involvement and prognostic scores are clinically meaningful. The negative correlation between FFS and AGR supports the link between poor nutritional status and increased mortality risk, whereas the negative correlation between BVAS and HALP suggests that higher disease activity may suppress nutritional reserves. In analyses based on organ involvement, lower AGR and HALP levels in patients with renal involvement, as well as higher PLR and SII but lower HALP levels in those with pulmonary involvement, indicate a relationship between these indices, systemic inflammatory burden, and organ damage. Overall, these findings suggest that inflammatory and nutritional parameters may reflect not only disease prognosis but also organ-specific phenotypic patterns in AAV.

In the multicentre cohort study by Liao et al. [20], age, ANCA type, renal function, C-reactive protein (CRP), and NLR were identified as independent predictors of long-term survival. In particular, elevated NLR and low albumin levels were shown to be associated with poor prognosis [20]. These findings suggest that, in addition to neutrophil- and albumin-based indices, other composite parameters reflecting the inflammatory burden may also provide potential contributions to risk stratification.

Neutrophil-to-lymphocyte ratio has been investigated in numerous studies as an easily accessible parameter reflecting inflammatory burden in AAV. Growing evidence from various cohorts has demonstrated that the NLR is associated with multiple dimensions of disease burden in AAV. A recent case-control study showed that routinely derived inflammatory indices – including NLR, PLR, SII, and systemic inflammation response index – not only distinguish AAV patients from healthy controls but also correlate with disease activity and prognosis, underscoring their potential utility in clinical assessment [4]. Fonseca et al. [5] reported that NLR was associated not only with vasculitis activity but also with severe infections and mortality. Similarly, Ahn et al. [12] demonstrated that a high NLR at the time of diagnosis was significantly associated with increased disease activity and poor prognosis. In a large retrospective cohort of myeloperoxidase (MPO)-AAV patients, Huang et al. [21] demonstrated that elevated NLR was independently associated with increased all-cause mortality, supporting its role as a meaningful prognostic marker in this AAV subtype. Collectively, these findings highlight NLR as a practical, inexpensive, and

clinically meaningful marker reflecting activity, organ involvement, prognosis, and complications in AAV. Nonetheless, despite the consistency of these reports, our cohort demonstrated only a weak and statistically non-significant correlation between NLR and BVAS ($p = 0.18$, $p = 0.19$). This divergence may reflect the modest sample size, heterogeneity in baseline clinical characteristics, or differences in inflammatory burden at the time of diagnosis. Some previous studies have reported significant associations even in smaller cohorts; however, variability in disease severity, population characteristics, and effect sizes across studies may account for the lack of statistical significance observed in our cohort despite similar trends.

In the literature, particularly in the retrospective analysis conducted by Park et al. [6], PLR was reported to show a significant correlation with disease activity at the time of diagnosis and to reflect the BVAS score. In contrast, in our cohort, only a weak positive correlation was observed between PLR and BVAS, which did not reach statistical significance ($p = 0.16$, $p = 0.22$). However, a moderate negative correlation was identified between BVAS and HALP. This finding suggests that as disease activity increases, nutritional reserves deteriorate, and the HALP score tends to decrease in an inverse relationship with disease activity. Low HALP scores may reflect chronic inflammation-induced nutritional depletion, because persistent systemic inflammation promotes catabolism and suppresses albumin synthesis [22]. This inflammation–malnutrition axis has been proposed as a driver of poor outcomes in other inflammatory diseases and may similarly contribute to adverse organ manifestations in AAV [23].

A similar pattern has been reported for CAR: its association with disease activity, organ damage, and mortality prediction has been demonstrated in several series. For instance, elevated CAR levels have been shown to correlate with disease activity and tissue damage [24] and to possess predictive value for mortality [10]. However, in the present study, CAR did not demonstrate a significant predictive value for relapse or mortality. This may be attributed to the limited sample size and heterogeneity in population characteristics, but it may also suggest that CAR primarily reflects concurrent disease activity and organ damage rather than serving as a long-term prognostic indicator in AAV.

Among the inflammatory composite indices, SII shows a significant association with the inflammatory response at diagnosis and may provide additional information regarding disease activity and clinical course in AAV. In a single-centre AAV cohort, higher SII levels were independently associated with severe disease at diagnosis, and patients with elevated SII values (cut-off $\approx 1,573$) demonstrated lower relapse-free and renal

survival rates, indicating a link with poor prognosis [9]. On the other hand, in a separate study conducted in China involving patients with MPO-AAV only, SII was found to be positively correlated with CRP and ESR. However, unexpectedly, a high SII value ($\geq 2,136$) was associated with a reduced risk of progression to end-stage renal disease (ESRD) and showed no significant association with mortality [25].

In our cohort, significant differences were observed between certain inflammatory/nutritional indices and the presence of renal and pulmonary involvement. Specifically, lower AGR and HALP scores in patients with renal involvement suggest that impaired nutritional status may be associated with renal manifestations. In contrast, higher PLR and SII values and lower HALP scores in patients with pulmonary involvement support the potential utility of inflammatory indices as biomarkers of organ involvement. However, heterogeneity exists in the literature regarding the direction of the PLR association. While some AAV cohorts have reported increased PLR levels in relation to higher disease activity, other studies – particularly those involving MPO-AAV patients – have found that elevated PLR was paradoxically associated with a reduced risk of progression to ESRD [25]. This highlights the importance of subtype differences and comorbid factors and places our finding of increased PLR in pulmonary involvement within the broader perspective of organ-specific inflammation. Because multiple comparisons were performed in the analysis of organ involvement without p -value adjustment, the possibility of false-positive findings cannot be excluded, and these results should be interpreted with caution. However, it should be noted that organ involvement in our study was assessed only at baseline to describe cross-sectional subphenotypic characteristics. Organ involvement was not evaluated longitudinally and was not used as a prognostic outcome (e.g. new organ damage or organ failure). Therefore, the observed associations reflect baseline phenotypic patterns rather than prognostic implications, and no causal or predictive interpretation should be inferred.

From a nutritional standpoint, our findings are consistent with the limited data available in the literature. Evidence regarding the prognostic significance of nutritional indicators such as HALP and AGR in AAV remains scarce, and their clinical implications have not yet been fully clarified. In this context, our study provides one of the few available data sets suggesting that these parameters may be associated not only with overall prognosis but also with specific patterns of organ involvement.

An important advantage of inflammatory and nutritional indices is that they can be easily calculated from routine haematological and biochemical tests used in daily clinical practice, without incurring any additional

cost. This makes them particularly attractive in settings with limited resources or in clinical situations requiring the follow-up of large patient populations. However, because the ROC analyses yielded AUC values below 0.6, the discriminative capacity of these indices for predicting relapse appears to be limited. Although our findings show limited predictive value for relapse, the observed associations with organ involvement and prognostic scores suggest that these indices may serve as useful adjunct biomarkers for clinical risk stratification. Consequently, the use of simple and accessible parameters may facilitate the development of earlier and more targeted management strategies in AAV.

Antineutrophil cytoplasmic antibody-associated vasculitis represents a heterogeneous group of diseases, with marked clinical and immunological differences among its subtypes, i.e. GPA, MPA, and EGPA. These differences are likely to be reflected in inflammatory and nutritional indices as well. In particular, the predominance of eosinophilia and type 2 T helper cell-mediated immune responses in EGPA may lead to distinct patterns in neutrophil- and platelet-based parameters such as NLR, PLR, and SII [26]. Since the number of EGPA cases followed in our clinic was limited, this subtype was not included in our study, and a separate analysis by subtype could not be performed. However, future large-scale studies evaluating GPA, MPA, and EGPA individually may help to reveal the subtype-specific biomarker potential of these parameters.

Study limitations

This study has several limitations. First, its single-centre and retrospective design increases the potential for selection bias. The relatively limited sample size restricted the statistical power, particularly for the analysis of infrequent clinical outcomes such as mortality. Inflammatory and nutritional indices were calculated only from baseline data at diagnosis, and dynamic changes during follow-up were not evaluated. Furthermore, these parameters may be influenced by concurrent infections, comorbidities, and treatment modalities. Additionally, severe infection and end-stage kidney disease were not evaluated as outcomes because the number of events in our cohort was too low to permit reliable statistical analysis. Therefore, prospective multicentre studies with larger patient populations are needed to validate these findings and enhance their generalisability. Given these limitations, prospective multicentre studies with serial measurements of inflammatory and nutritional indices are essential to validate their prognostic relevance and to determine whether these markers can be incorporated into future risk stratification models in AAV.

Conclusions

When our findings are considered together with the existing literature, the studied easily accessible indices may not be sufficient on their own for relapse prediction but could hold clinical relevance in reflecting organ involvement phenotypes and concurrent disease activity or damage. Therefore, validation through larger prospective studies incorporating stratified analyses by subtype (GPA/MPA/EGPA) and combined assessment with clinical scores such as BVAS and FFS is warranted. Future large-scale prospective studies will further clarify the potential integration of these parameters into routine clinical practice.

Disclosures

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Data availability: The data that support the findings of this study are available on request from the corresponding author (P.A.D.).

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