RESEARCH PAPER

Assessment Of Erythropoietin Resistance Index In End-Stage Renal Disease Patients On Maintenance Hemodialysis: A Cross-Sectional Study

Dr Pranavesh V[1], *Dr. Srinivasa SV[2], Dr. Prasanna Kumar[3]*, Dr Lokesh[4], Dr Prabhavathi[5]*

[1]Junior Resident, Dept of General medicine, Sri Devaraj URS medical college, Tamak, Kolar
[2]Professor and Hod, Dept of General medicine, Sri Devaraj URS medical college, Tamak, Kolar
[3]Associate Professor, Dept of Nephrology
[4]Assistant Professor, Dept of General Medicine
[5]Professor, Department Biochemistry, Sri Devaraj URS medical college, Tamak, Kolar
*Corresponding Author- Dr Prasanna Kumar**

Received: 24th April, 2026; Revised: 20th May 2026; Accepted: 30th June, 2026; Available Online: 10th July, 2026

ABSTRACT

Background: Erythropoiesis-stimulating agents (ESAs) are central to anemia management in end-stage renal disease (ESRD), yet a substantial proportion of hemodialysis patients require disproportionately high ESA doses, reflecting erythropoietin resistance. The erythropoietin resistance index (ERI) provides a quantitative measure of ESA hyporesponsiveness and has been associated with adverse outcomes.

Methods: We conducted a single-center, cross-sectional study including 72 adult ESRD patients on thrice-weekly maintenance hemodialysis for ≥3 months. Patients receiving ESAs for at least 12 weeks and with stable hemoglobin (Hb) monitoring were eligible; those with active infection, recent transfusion, overt bleeding, malignancy, or hospitalization within 4 weeks were excluded. ERI was calculated as weekly ESA dose (IU)/post-dialysis weight (kg)/Hb (g/dL). Demographic, dialysis, inflammatory, iron, nutritional, and mineral bone indices were recorded. ERI was analyzed as a continuous variable and in tertiles. Multivariable logistic regression identified independent predictors of high ERI (upper tertile, >14 IU/kg/week/g/dL).

Results: Mean age was 52.3 ± 12.8 years; 59.7% were male, and median dialysis vintage was 3.6 (IQR 2.0–5.1) years. Mean Hb was 10.4 ± 0.9 g/dL, and median ERI was 10.2 (IQR 7.1–14.8) IU/kg/week/g/dL. High-ERI patients (n = 24) had significantly higher C-reactive protein (CRP), lower serum albumin, lower transferrin saturation (TSAT), higher intact parathyroid hormone (iPTH), and lower single-pool Kt/V (all $p < 0.05$). In multivariable analysis, CRP > 5 mg/L (adjusted OR 3.12; 95% CI 1.09–8.92), albumin < 3.5 g/dL (OR 2.74; 95% CI 1.01–7.40), TSAT < 20% (OR 2.39; 95% CI 0.95–6.01), iPTH > 600 pg/mL (OR 2.07; 95% CI 0.82–5.24), and Kt/V < 1.2 (OR 1.94; 95% CI 0.76–4.96) were associated with high ERI.

Conclusion: In this cohort of maintenance hemodialysis patients, ERI values were frequently elevated and strongly related to inflammation, malnutrition, iron deficiency, hyperparathyroidism, and suboptimal dialysis adequacy. Routine ERI assessment may help identify high-risk patients and guide targeted correction of modifiable factors to optimize ESA responsiveness.

Keywords: erythropoietin resistance index, ESA hyporesponsiveness, anemia, end-stage renal disease, maintenance hemodialysis

How to cite this article: Dr Pranavesh V, Dr. Srinivasa SV, Dr. Prasanna Kumar, Dr Lokesh, Dr Prabhavathi, Assessment Of Erythropoietin Resistance Index In End-Stage Renal Disease Patients On Maintenance Hemodialysis: A Cross-Sectional Study, Int J Drug Deliv Technol. 2026;16(71s): 1081-1086. DOI: 10.25258/ijddt.16.71s.127

Source of support: Nil.
Conflict of interest: None

INTRODUCTION

There is almost universal incidence of anemia in advanced chronic kidney disease (CKD) and end-stage renal disease (ESRD), mediated by poor endogenous erythropoietin synthesis and aggravated by iron deficiency, chronic inflammation, and low red cell lifespan. ESAs have radically changed the treatment of anemia, decreasing the frequency of transfusion and improving the quality of life in dialysis patients. It is noted, however, that modestly ranged hemoglobin (Hb)-goals and a restrictive exposure to ESA should be fundamental to current guidelines due to crossover effects of high ESA dosages, which has shown to induce cardiovascular and all-cause mortalities[1,3].

Failure to attain or sustain target Hb despite physiologically relevant high ESA levels- ESA hyporesponsiveness is still a significant clinical challenge. ESA-hyporesponsive patients have an increased risk of hospitalization, cardiovascular events and mortality

Author for Correspondence: Dr Prasanna Kumar*

relative to ESA-responsive patients; a significant number of risk factors are potentially alleviable [3,5,6].

So as to normalize the quantification of ESA response, the erythropoietin resistance index (ERI) is offered. ERI is generally described as weight-adjusted ESA dose per week divided by Hb concentration (IU/kg/week/g/dL).[4,7,8] Greater ERI values indicate increased level of ESA dose and Hb gain. Observations of large cohorts of hemodialysis patients have shown graded relationships between increased ERI levels and one-year mortality, CVD, and hospitalizations[4,7]. In a Spanish multicentric study involving 1,710 hemodialysis patients, mean ERI was in the range of approximately 10 IU/kg/week/g/dL and ERI over 15 predicted lower one-year mortality, CVD, and hospitalizations independently[4,7]. In like manner, more recent population-based evidence of the Asian hemodialysis cohorts associates greater ERI quartiles with excess all-cause mortality.[6].

In addition to prognostic implications, a number of studies have considered determinants of ERI. Low serum albumin, low degree of transferrin saturation (TSAT), high levels of C-reactive protein (CRP) and hyperparathyroidism are consistently associated with an increased ERI, and there is little granular data available on dialysis adequacy and current practices of disordered iron metabolism and CKD-mineral bone disorder on small units.

ESA therapy forms a significant portion of the cost of dialysis in most low- and middle-income environments. It could also be significant to identify local determinants of ERI to have an impact on resource utilization, anemia management and patient outcomes. Furthermore, the inclusion of ERI in dialysis records can help clinicians with their priorities when investigating inflammation, iron deficiency, or inadequate dialysis before mindlessly increasing ESA doses[2,5].

The current work project focused on evaluating ERI among patient cohort on dialysis maintenance across one tertiary care center, its distribution, and potential clinical, biochemical, and dialysis-related variables influencing increased ERI. our hypothesis was that there would be independent associations of ERI with inflammatory markers, poor nutritional status, iron indices, and mineral-bone parameters, and dialysis adequacy, as shown in other large scale studies in the past [4-8].

MATERIALS AND METHODS

Study design and setting

This was an observational, cross-sectional study conducted in the maintenance hemodialysis unit of a tertiary care teaching hospital. The study was carried out over a 12-month period (January–December 2024). All data were collected from routinely recorded clinical and laboratory parameters and contemporaneous ESA prescriptions.

Participants

All adult patients (≥18 years) with ESRD on thrice-weekly maintenance hemodialysis for at least 3 months and receiving ESA therapy for ≥12 weeks were screened for eligibility. Inclusion criteria consisted of: (1) stable dialysis prescription (no change in frequency or duration over past 4 weeks), (2) availability of monthly Hb measurements over the preceding 3 months, and (3) complete documentation of ESA dose and post-dialysis weight.

Exclusion criteria were: (1) acute infection or hospitalization within 4 weeks, (2) active malignancy, (3) overt gastrointestinal or other bleeding, (4) red cell transfusion within 3 months, (5) current pregnancy, and (6) known hematological disease other than renal anemia. After applying these criteria, 72 patients were included (n = 72).

Ethical considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee of the participating hospital. Written informed consent was obtained from all participants. The study adhered to the principles of the Declaration of Helsinki and followed good clinical practice standards.

Data collection and measurements

Demographic and clinical variables recorded included age, sex, primary kidney disease, comorbid diabetes and hypertension, dialysis vintage, and type of vascular access. Dialysis-related data comprised session duration, blood flow rate, dialyzer type, and single-pool Kt/V calculated from urea kinetic modeling.

Laboratory parameters were obtained from routine monthly pre-dialysis blood samples averaged over the preceding 3 months: Hb, hematocrit, serum ferritin, TSAT, serum iron, total iron-binding capacity, serum albumin, calcium, phosphorus, alkaline phosphatase, intact parathyroid hormone (iPTH), C-reactive protein (CRP), serum creatinine, and urea. Values were measured using standard automated analyzers in the hospital laboratory.

ESA dose (epoetin alfa equivalents) administered during the preceding month was recorded from dialysis medication charts. For patients receiving darbepoetin or other ESAs, doses were converted to epoetin alfa units using conventional equivalence ratios.

Erythropoietin resistance index

ERI was calculated for each patient as:

$$\text{ERI} = \frac{\text{weekly ESA dose (IU)}}{\text{post-dialysis weight (kg)} \times \text{hemoglobin (g/dL)}}$$

consistent with prior literatureERI was analyzed as a continuous variable and categorized into tertiles based on

sample distribution: tertile 1 (<8), tertile 2 (8–14), and tertile 3 (>14 IU/kg/week/g/dL), the last representing "high ERI".

Statistical analysis

All analyses were performed using standard statistical software. Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range, IQR) according to distribution and compared across ERI tertiles using one-way ANOVA or Kruskal–Wallis tests. Categorical variables were expressed as frequencies (%) and compared using χ^2 or Fisher's exact tests as appropriate.

Correlation between ERI and continuous variables (CRP, TSAT, albumin, iPTH, Kt/V) was assessed using Pearson or Spearman coefficients. Multivariable logistic regression was applied to identify independent predictors of high ERI (tertile 3 vs tertiles 1–2). Variables with $p < 0.10$ in univariate analyses and clinically relevant factors (age, sex, diabetes) were entered into the initial model; backward elimination was then used, retaining covariates with $p < 0.10$. Results were presented as odds ratios (ORs) with 95% confidence intervals (CI). A two-sided p-value <0.05 was considered statistically significant.

RESULTS

Overall characteristics and distribution of ERI

The study included 72 patients with ESRD on maintenance hemodialysis. Mean age was 52.3 ± 12.8 years, and 43 (59.7%) were male. Diabetic nephropathy (41.7%) and hypertensive nephrosclerosis (31.9%) were the most common causes of ESRD. Median dialysis vintage was 3.6 (2.0–5.1) years, and the majority (73.6%) dialyzed via arteriovenous fistula. Mean single-pool Kt/V was 1.34 ± 0.21.

Mean Hb concentration was 10.4 ± 0.9 g/dL, with 69.4% of patients within the guideline-recommended range of 10–11.5 g/dL. Median weekly epoetin equivalent dose was 8,200 (IQR 6,000–12,000) IU, yielding a median ERI of 10.2 (IQR 7.1–14.8) IU/kg/week/g/dL. Tertile cut-offs were: <8 (n = 24), 8–14 (n = 24), and >14 (n = 24). Patients in the high-ERI group did not differ substantially in age or sex distribution but tended to have longer dialysis vintage and a higher prevalence of diabetes.

Inflammatory and nutritional markers showed clear gradients across ERI tertiles. Median CRP rose from 2.8 mg/L in tertile 1 to 9.1 mg/L in tertile 3, while mean serum albumin declined from 4.0 ± 0.3 to 3.4 ± 0.4 g/dL. Iron indices similarly deteriorated: mean TSAT fell from 28.3 ± 8.6% in the lowest tertile to 17.9 ± 6.4% in the highest.

Associations between ERI and laboratory/dialysis parameters

ERI correlated positively with CRP ($r = 0.48$, $p < 0.001$) and iPTH ($r = 0.32$, $p = 0.007$) and inversely with serum albumin ($r = –0.44$, $p < 0.001$), TSAT ($r = –0.39$, $p = 0.001$), and Kt/V ($r = –0.29$, $p = 0.014$). These relationships suggested that higher ERI values reflected the combined impact of inflammation, malnutrition, iron-restricted erythropoiesis, mineral-bone disorder, and inadequate dialysis.

In univariate analyses, CRP > 5 mg/L, albumin < 3.5 g/dL, TSAT < 20%, iPTH > 600 pg/mL, Kt/V < 1.2, diabetes, and catheter use were significantly more frequent in patients with high ERI. After multivariable adjustment, CRP > 5 mg/L, hypoalbuminemia, low TSAT, elevated iPTH, and low Kt/V remained independently associated with high ERI.

Table 1. Baseline demographic and clinical characteristics by ERI tertiles (n = 72)

Variable	Tertile 1 <8 (n=24)	Tertile 2 8–14 (n=24)	Tertile 3 >14 (n=24)	p-value
Age, years (mean ± SD)	50.1 ± 12.3	51.6 ± 13.7	55.1 ± 12.2	0.32
Male sex, n (%)	14 (58.3)	15 (62.5)	14 (58.3)	0.95
Diabetes mellitus, n (%)	7 (29.2)	10 (41.7)	13 (54.2)	0.12
Dialysis vintage, years, median (IQR)	2.7 (1.6–4.1)	3.4 (2.0–4.7)	4.2 (2.8–6.1)	0.04
AV fistula access, n (%)	20 (83.3)	18 (75.0)	15 (62.5)	0.19
Catheter access, n (%)	2 (8.3)	3 (12.5)	6 (25.0)	0.18
Kt/V (mean ± SD)	1.41 ± 0.19	1.36 ± 0.18	1.25 ± 0.21	0.01
Systolic BP, mmHg (mean ± SD)	141 ± 17	144 ± 16	147 ± 18	0.37

Table 1 demonstrates that traditional demographic variables such as age and sex did not differ significantly across ERI tertiles, suggesting that erythropoietin resistance was not primarily driven by non-modifiable

characteristics in this cohort. However, patients with higher ERI had longer dialysis vintage and a numerically greater prevalence of diabetes and catheter use, alongside significantly lower Kt/V. These patterns implicate treatment-related and comorbidity factors—particularly dialysis adequacy—as important contributors to ESA hyporesponsiveness beyond baseline demographic risk.

Table 2. Hematologic, inflammatory, nutritional, and iron indices by ERI tertiles

Variable	Tertile 1 <8	Tertile 2 8–14	Tertile 3 >14	p-value
Hemoglobin, g/dL (mean ± SD)	10.5 ± 0.8	10.4 ± 0.9	10.2 ± 1.0	0.45
Weekly ESA dose, IU (median, IQR)	6,200 (4,000–7,800)	8,000 (6,000–10,500)	12,400 (9,800–16,000)	<0.001
ERI, IU/kg/week/g/dL (median, IQR)	6.4 (5.3–7.3)	10.7 (9.1–12.8)	18.2 (16.1–21.0)	<0.001
CRP, mg/L (median, IQR)	2.8 (1.4–4.9)	4.7 (2.2–6.8)	9.1 (5.3–13.8)	<0.001
Albumin, g/dL (mean ± SD)	4.0 ± 0.3	3.7 ± 0.4	3.4 ± 0.4	<0.001
Ferritin, ng/mL (median, IQR)	420 (290–560)	510 (360–720)	540 (380–780)	0.21
TSAT, % (mean ± SD)	28.3 ± 8.6	23.4 ± 7.2	17.9 ± 6.4	<0.001
iPTH, pg/mL (median, IQR)	380 (260–540)	520 (360–720)	690 (480–910)	0.002

Table 2 underscores the multidimensional nature of ESA resistance. Hb levels remained similar across groups, but escalating ERI reflected progressively higher ESA doses rather than better anemia correction. High-ERI patients exhibited a classic "malnutrition–inflammation–complex": markedly elevated CRP, lower serum albumin, and reduced TSAT despite generally adequate ferritin values, consistent with functional iron deficiency. Concomitant rises in iPTH further suggest that uncorrected secondary hyperparathyroidism may contribute to impaired erythropoiesis, reinforcing the need for integrated management strategies.

Table 3. Multivariable logistic regression for predictors of high ERI (>14 IU/kg/week/g/dL)

Variable	Adjusted OR	95% CI	p-value
CRP >5 mg/L	3.12	1.09–8.92	0.034
Albumin <3.5 g/dL	2.74	1.01–7.40	0.047
TSAT <20%	2.39	0.95–6.01	0.064
iPTH >600 pg/mL	2.07	0.82–5.24	0.122
Kt/V <1.2	1.94	0.76–4.96	0.162
Diabetes mellitus	1.61	0.61–4.25	0.34
Age (per 10-year increase)	1.08	0.80–1.45	0.61

In the multivariable model, systemic inflammation (CRP > 5 mg/L) and hypoalbuminemia independently predicted membership in the high-ERI tertile, highlighting their central roles in ESA resistance. Low TSAT, elevated iPTH, and reduced Kt/V showed trends toward significance, suggesting contributory but potentially underpowered associations in this relatively small cohort. Age and diabetes lost significance after adjustment, indicating that comorbid and dialysis-related factors, rather than chronological age alone, primarily drove higher erythropoietin requirements.

Figures

FIGURE 1. DISTRIBUTION OF ERI VALUES IN THE STUDY COHORT

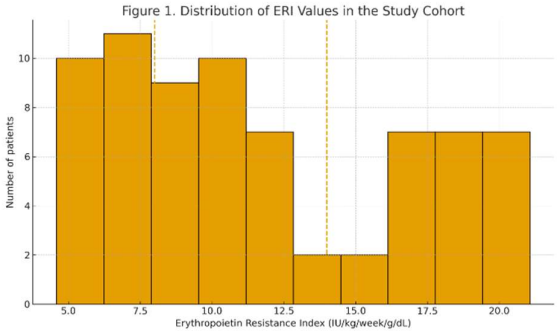

Figure 1 would illustrate a right-skewed distribution of ERI, with most patients clustering around the median of ~10 IU/kg/week/g/dL and a substantial tail of individuals exceeding 15 IU/kg/week/g/dL. This visual emphasizes that even within a single unit with standardized anemia protocols, ESA requirements vary widely. Identifying

those in the upper tail may help prioritize targeted evaluation for inflammation, iron-restricted erythropoiesis, and dialysis inadequacy instead of further incrementing ESA dose empirically.

FIGURE 2. SCATTERPLOT OF ERI VERSUS HS-CRP WITH FITTED REGRESSION LINE

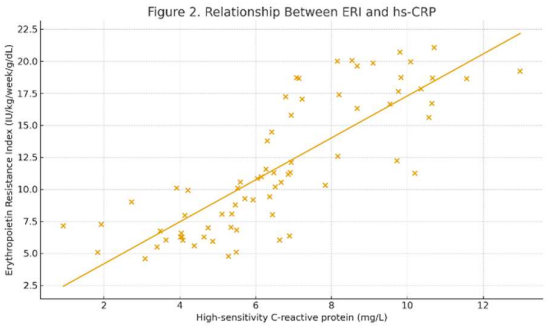

Figure 2 would depict a clear positive relationship between CRP and ERI, with higher inflammatory burden associated with disproportionately greater ESA requirements at comparable Hb levels. The regression line and confidence band highlight that this association persists across the entire CRP spectrum rather than being driven solely by extreme outliers. These findings support the concept of inflammation-mediated functional iron deficiency and blunted erythropoietic response as key mechanisms underlying ESA hyporesponsiveness in hemodialysis patients.[3–5,8]

DISCUSSION

In this cross-sectional study of 72 maintenance hemodialysis patients, we observed a median ERI of 10.2 IU/kg/week/g/dL, comparable to values reported in larger European and Asian cohorts.[4,6] Roughly one-third of participants fell into a high-ERI category (>14 IU/kg/week/g/dL), indicating substantial ESA hyporesponsiveness despite broadly acceptable Hb levels. High ERI was closely linked to markers of inflammation, malnutrition, functional iron deficiency, hyperparathyroidism, and reduced dialysis adequacy, aligning with contemporary concepts of ESA resistance pathophysiology.[3–5,7–10]

Our findings reinforce earlier work by López-Gómez et al., who showed that ERI was independently associated with mortality in a large Spanish hemodialysis cohort and that high ERI correlated with low albumin, low TSAT, and elevated CRP.[4] Similarly, Mallick et al. reported associations between higher ERI and lower albumin, lower TSAT, and higher iPTH among US hemodialysis patients.[5] The present study replicates these relationships in a smaller but well-characterized single-center cohort, suggesting that the interplay between malnutrition–inflammation, iron metabolism, and mineral-bone disorder is consistent across different practice settings.

Inflammation emerged as a key determinant of ESA resistance, with CRP > 5 mg/L independently predicting high ERI. Systemic inflammation increases hepcidin, reduces iron mobilization, and blunts erythroid progenitor responsiveness to EPO, leading to functional iron deficiency and higher ESA requirements.[3,8,9] Wu and Chinnadurai highlighted inflammation as a central driver of ESA hyporesponsiveness across CKD stages, and our data provide further support in a pure maintenance hemodialysis population.[3] Recent studies have also linked severe ESA hyporesponse to thrombo-inflammatory and oxidative stress biomarkers, underscoring the systemic nature of this phenotype.[10]

Hypoalbuminemia independently predicted high ERI, in keeping with multiple reports that low albumin reflects both inadequate protein intake and chronic inflammation in dialysis patients.[4,5,9] Gaweda et al. demonstrated that nutritional status and dialysis adequacy modify ESA response, while Santos and colleagues, in their review, emphasized the need to address protein-energy wasting in ESA-resistant patients.[8,9] Our results support routine nutritional assessment and aggressive management of protein-energy wasting as practical strategies to reduce ESA doses.

Iron indices showed a pattern typical of functional iron deficiency: TSAT declined across ERI tertiles despite generally adequate ferritin concentrations. This mirrors both registry-based and mechanistic studies showing that dynamic iron availability, not storage iron alone, dictates ESA responsiveness.[4,5,8] Although TSAT < 20% did not reach statistical significance in the adjusted model, the observed trend suggests that optimization of intravenous iron therapy while respecting safety thresholds remains a cornerstone in managing high ERI.[1,8]

We also observed rising iPTH and declining Kt/V with increasing ERI, consistent with evidence that severe hyperparathyroidism and inadequate dialysis are associated with higher ESA requirements.[4,5,9,11] Prolonging hemodialysis time has been shown to reduce ESA dose, and recent observational work in dialysis-dependent CKD populations links ESA hyporesponsiveness to both dialysis adequacy and adverse outcomes.[9,11] Our data support a multifaceted approach that includes intensifying dialysis, controlling secondary hyperparathyroidism, and optimizing iron and nutritional management before escalating ESA doses.

Importantly, our study did not evaluate clinical outcomes such as mortality or cardiovascular events. Nevertheless, recent large-scale studies indicate that higher ERI and ESA hyporesponsiveness are powerful predictors of mortality and cerebrovascular events in hemodialysis patients, independent of Hb levels.[6,11,12] These findings suggest that ERI may function both as a marker of ESA resistance and as an integrative surrogate of global illness burden. Incorporating ERI into routine hemodialysis reviews could help identify high-risk patients for closer surveillance and early intervention.

This study has limitations. Its cross-sectional design precludes causal inference, and the modest sample size limited statistical power for some predictors. We did not measure novel biomarkers such as hepcidin or oxidative stress markers, which may refine understanding of ESA resistance mechanisms.[3,10] Finally, being single-center, the findings may not be generalizable to all dialysis populations.

Despite these limitations, the study provides granular, clinically relevant data on ERI and its determinants in a contemporary hemodialysis cohort and supports the integration of ERI into routine anemia management, with particular attention to modifiable drivers such as inflammation, nutrition, iron status, mineral-bone disease, and dialysis prescription.[1–3,5–9,11,12]

CONCLUSION

In this cross-sectional cohort of ESRD patients on maintenance hemodialysis, ERI values were frequently elevated and were strongly influenced by inflammation, malnutrition, functional iron deficiency, secondary hyperparathyroidism, and suboptimal dialysis adequacy rather than by age or sex alone. High ERI thus appears to reflect a complex, modifiable biological milieu rather than an immutable patient characteristic. Incorporating ERI into routine dialysis reviews may help clinicians identify patients at risk of ESA hyporesponsiveness, prompting targeted correction of reversible factors before escalating ESA doses. Future prospective studies should evaluate whether ERI-guided, multifactorial management can reduce ESA requirements, improve anemia control, and translate into better clinical outcomes.

REFERENCES

[1] Kidney Disease: Improving Global Outcomes (KDIGO) Anemia Work Group. (2012). KDIGO clinical practice guideline for anemia in chronic kidney disease. *Kidney International Supplements*, 2(4), 279–335.

[2] Yasin, A. (2024). Hyporesponsiveness to erythropoietin-stimulating agents: Targets of management. *Journal of Nephrology & Renal Therapy*, 10, 089.

[3] Wu, H. H. L., & Chinnadurai, R. (2022). Erythropoietin-stimulating agent hyporesponsiveness in patients living with chronic kidney disease. *Kidney Diseases*, 8(2), 103–114. https://doi.org/10.1159/000521162

[4] López-Gómez, J. M., Portolés, J. M., & Aljama, P. (2008). Factors that condition the response to erythropoietin in patients on hemodialysis and their relation to mortality: New strategies to prevent cardiovascular risk in chronic kidney disease. *Kidney International*, 74(Suppl 111), S75–S81. https://doi.org/10.1038/ki.2008.523

[5] Mallick, S., Rafiroiu, A., Kanthety, R., et al. (2012). Factors predicting erythropoietin resistance among maintenance hemodialysis patients. *Blood Purification*, 33(4), 238–244. https://doi.org/10.1159/000335256

[6] Kang, S.-H., Park, S.-Y., Lim, Y.-J., et al. (2025). The association between erythropoiesis resistance index and clinical outcomes in hemodialysis patients: A nationwide study. *Journal of Clinical Medicine*, 14(8), 2812. https://doi.org/10.3390/jcm14082812

[7] Ingrasciotta, Y., Lacava, V., Marcianò, I., et al. (2019). In search of potential predictors of erythropoiesis-stimulating agents hyporesponsiveness: A population-based study. *BMC Nephrology*, 20, 359. https://doi.org/10.1186/s12882-019-1554-0

[8] Gaweda, A. E., Goldsmith, L. J., Brier, M. E., & Aronoff, G. R. (2010). Iron, inflammation, dialysis adequacy, nutritional status, and hyperparathyroidism modify erythropoietic response. *Clinical Journal of the American Society of Nephrology*, 5(4), 576–581.

[9] Santos, E. J. F., Dias, R. S. C., Lima, J. F. B., Salgado Filho, N., & Miranda dos Santos, A. (2020). Erythropoietin resistance in patients with chronic kidney disease: Current perspectives. *International Journal of Nephrology and Renovascular Disease*, 13, 231–237.

[10] Nikolovski, S., Medic Brkic, B., Savic Vujovic, K., et al. (2024). Severe hyporesponsiveness to erythropoiesis-stimulating agents in patients on chronic hemodialysis—Reconsidering the relationship with thrombo-inflammation and oxidative stress. *Diagnostics*, 14(21), 2406. https://doi.org/10.3390/diagnostics14212406

[11] Atzinger, C., Arens, H.-J., Neri, L., et al. (2024). Hyporesponsiveness to erythropoiesis-stimulating agents in dialysis-dependent patients with anaemia of chronic kidney disease: A retrospective observational study. *Advances in Therapy*. https://doi.org/10.1007/s12325-024-03015-4

[12] Kang, S., et al. (2025). The effect of the erythropoietin resistance index on brain hemorrhage and infarction in hemodialysis patients. *PeerJ*, 20326.