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Clinical and Laboratory Factors Associated With Atherosclerotic Disease in Patients Receiving Maintenance Hemodialysis: A Cross-Sectional Study

Qiu Li
Baoshan Second People's Hospital, Baoshan, Yunnan, China
Hongyong Yu
Baoshan Second People's Hospital, Baoshan, Yunnan, China
Chun Chen
Baoshan Second People's Hospital, Baoshan, Yunnan, China
Chunyan Yang
Baoshan Second People's Hospital, Baoshan, Yunnan, China
Jian Wang
Baoshan Second People's Hospital, Baoshan, Yunnan, China
Bidong Guo
Baoshan Second People's Hospital, Baoshan, Yunnan, China
Fang Jiang
Baoshan Second People's Hospital, Baoshan, Yunnan, China
Xuejiao Mei
Baoshan Second People's Hospital, Baoshan, Yunnan, China
Yingdie Xia
Baoshan Second People's Hospital, Baoshan, Yunnan, China
Zhihang Xiao
Baoshan Second People's Hospital, Baoshan, Yunnan, China
Ying Liang
Baoshan Second People's Hospital, Baoshan, Yunnan, China
Xiaojiao Zhao
Baoshan Second People's Hospital, Baoshan, Yunnan, China
Sining Chen
Baoshan Second People's Hospital, Baoshan, Yunnan, China
Shaojun Lu
784622173@qq.com

Baoshan Second People's Hospital, Baoshan, Yunnan, China

Article

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Qiu Li,Hongyong Yu,Chun Chen,Chunyan Yang,Jian Wang,Bidong Guo,Fang Jiang,Xuejiao Mei,Yingdie Xia,Zhihang Xiao,Ying Liang,Xiaojiao Zhao,Sining Chen,Shaojun Lu

Department of Nephrology, Baoshan Second People's Hospital, Baoshan, Yunnan, China

Corresponding author: Shaojun Lu, Bachelor of Medicine

E-mail: 784622173@qq.com

F-ORCID:0009-0007-7313-9969

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Abstract

Purpose: Patients receiving maintenance hemodialysis (MHD) have a substantial burden of atherosclerotic disease, yet the relative contributions of conventional and dialysis-related factors remain incompletely characterized. We investigated clinical and laboratory factors associated with atherosclerotic disease in an MHD cohort.

Methods: We conducted a cross-sectional study of 113 patients receiving MHD at Baoshan Second People's Hospital, Yunnan, China. Atherosclerotic disease was defined as documented coronary atherosclerotic disease, ischemic stroke/cerebral infarction, or lower-extremity arterial atherosclerotic plaque. Variables with P<0.10 in univariable logistic regression were entered into an initial multivariable model, from which a parsimonious six-variable model was derived by backward elimination. Multicollinearity, linearity in the logit, influential observations, calibration, discrimination, and bootstrap internal validation were assessed.

Results: Atherosclerotic disease was present in 74 of 113 patients (65.5%). In the parsimonious model, age (adjusted odds ratio [aOR] 2.41, 95% confidence interval [CI] 1.48–3.91 per 10 years), diabetes (aOR 5.56, 95% CI 1.04–29.61), smoking (aOR 5.03, 95% CI 1.41–18.00), interdialytic weight gain (IDWG; aOR 1.89, 95% CI 1.27–2.82 per percentage point), alkaline phosphatase (ALP; aOR 1.14, 95% CI 1.01–1.29 per 10 U/L), and monocyte count (aOR 1.56, 95% CI 1.10–2.22 per 0.1×10^9/L) were independently associated with atherosclerotic disease. The apparent AUC was 0.867 and the optimism-corrected AUC was approximately 0.832 after 1,000 planned bootstrap resamples (978 successful fits).

Conclusion: Older age, diabetes, smoking, greater IDWG, higher ALP, and higher monocyte count were independently associated with atherosclerotic disease in this MHD cohort. These findings are hypothesis-generating and do not establish causality or constitute an externally validated prediction model. Larger prospective multicenter studies with standardized vascular ascertainment are warranted.

Keywords: maintenance hemodialysis; atherosclerotic disease; interdialytic weight gain; alkaline phosphatase; monocytes; diabetes mellitus

1 Introduction

Atherosclerotic cardiovascular disease is a major contributor to morbidity and mortality in chronic kidney disease, and the burden is particularly high among patients receiving hemodialysis. The vascular phenotype of kidney failure is shaped by the interaction of conventional cardiovascular risk factors with uremia-related inflammation, endothelial dysfunction, oxidative stress, hemodynamic stress, volume overload, and disordered mineral and bone metabolism [1–3].

Recent evidence from Chinese MHD populations supports important associations of age, diabetes, dialysis duration, mineral metabolism, and inflammatory markers with coronary artery calcification. A 2025 meta-analysis of 24 studies including 2,875 Chinese patients receiving MHD reported significant pooled associations for diabetes, intact

parathyroid hormone, age, dialysis duration, calcium, phosphorus, high-sensitivity C-reactive protein, and sclerostin, whereas the pooled association for alkaline phosphatase (ALP) was not statistically significant [4]. Longitudinal data also indicate that vascular calcification progresses during dialysis and that risk-factor patterns may differ across vascular beds [5].

Interdialytic weight gain (IDWG) is a clinically accessible marker of interdialytic salt and water accumulation. Recent systematic-review evidence indicates that low-salt dietary strategies and nutritional counseling can reduce IDWG [6]. Whether IDWG is independently associated with clinically defined atherosclerotic disease in routine MHD populations remains insufficiently characterized. Monocytes may contribute to vascular injury through endothelial adhesion, migration into the vascular wall, macrophage differentiation, and plaque development. Nutritional and inflammatory markers may additionally reflect the complex malnutrition-inflammation milieu of dialysis.

We therefore aimed to identify clinical and laboratory factors associated with atherosclerotic disease in patients receiving MHD and to assess the robustness of a parsimonious multivariable model through model diagnostics and internal validation.

2 Materials and Methods

2.1 Study design and participants

This was a cross-sectional observational study of patients receiving MHD at Baoshan Second People's Hospital, Baoshan, Yunnan, China. The study period was January 2025 to December 2025. Patients were identified from the hospital hemodialysis information system and medical records. Inclusion criteria were Age ≥ 18 years,Receiving maintenance hemodialysis (MHD) at the hemodialysis center of Baoshan Second People's Hospital during the study period,Dialysis vintage ≥ 3 months at enrollment (to ensure a stable maintenance state),Complete clinical and laboratory records allowing ascertainment of atherosclerotic disease status and measurement of the study variables; and exclusion criteria were Age < 18 years,Dialysis vintage < 3 months, or acute kidney injury requiring dialysis (i.e., not yet on maintenance HD),Receiving peritoneal dialysis or having a functioning kidney transplant at enrollment,Active malignancy or active infection at enrollment,Missing essential data for outcome or key-covariate assessment,Refusal or inability to provide informed consent. A total of 113 eligible patients were included.

The study protocol was approved by the Medical Ethics Committee of Baoshan Second People's Hospital, Yunnan Province, China (Ethics Approval No. 2024-041 [Zi-Zong]-01). Written informed consent was obtained from all participants in accordance with the approved protocol. The study was conducted in accordance with applicable institutional ethical standards and the principles of the Declaration of Helsinki.

2.2 Outcome definition and ascertainment

The primary outcome was atherosclerotic disease, defined as the presence of at least one of the following: (1) documented coronary atherosclerotic disease supported by Coronary CT Angiography or invasive coronary angiography; (2) documented ischemic stroke/cerebral infarction supported by cranial CT or brain MRI; or (3) lower-extremity arterial atherosclerotic plaque documented by color Doppler ultrasonography of the lower-limb arteries. Participants meeting none of these criteria were classified as not having atherosclerotic disease.

Because cerebral infarction is etiologically heterogeneous, it was not assumed to represent atherosclerotic stroke in every case. Stroke etiology was not systematically adjudicated in the source database; this potential outcome heterogeneity was therefore considered when interpreting the results and is acknowledged as a study limitation.

2.3 Variables and measurements

The database included demographic, dialysis-related, hemodynamic, nutritional, inflammatory, mineral-bone, lipid, iron-status, and dialysis-adequacy variables. These included age, sex, vascular access, dialysis frequency, diabetes, smoking, hypertension, dialysis vintage, pre-dialysis blood pressure, IDWG, parathyroid hormone, hemoglobin, high-sensitivity C-reactive protein (hs-CRP), alanine aminotransferase, ALP, albumin, prealbumin, electrolytes, β2-microglobulin, urea, uric acid, creatinine, lipid indices, atherogenic index of plasma, ferritin, transferrin saturation, urea reduction ratio, Kt/V, lymphocyte count, monocyte count, and lymphocyte-to-monocyte ratio (LMR).

IDWG was expressed as the percentage increase in body weight between dialysis sessions according to the study database definition. Continuous variables were analyzed using their original measurement scales; effect estimates in the multivariable model were rescaled for clinical interpretability as specified in Table 2.

2.4 Statistical analysis

Continuous variables were assessed for distribution using the Shapiro–Wilk test within outcome groups. Variables approximately normally distributed in both groups are presented as mean±standard deviation (SD) and were compared using Welch's t-test. Non-normally distributed variables are presented as median (interquartile range [IQR]) and were

compared using the Mann–Whitney U test. Categorical variables are presented as n (%) and were compared using Fisher's exact test for 2×2 tables or Pearson's chi-square test for larger tables.

Univariable logistic regression was used to screen candidate variables. Variables with $P<0.10$ were entered into the initial multivariable logistic regression model. Ten variables met this criterion: age, diabetes, smoking, IDWG, hs-CRP, ALP, albumin, prealbumin, monocyte count, and LMR. Multicollinearity was assessed using variance inflation factors (VIFs); the maximum VIF was 2.21, indicating no important multicollinearity.

A parsimonious six-variable model comprising age, diabetes, smoking, IDWG, ALP, and monocyte count was obtained by backward elimination. Given the data-driven selection process and modest sample size, this model was regarded as an explanatory multivariable model rather than a validated clinical prediction tool. Adjusted odds ratios (aORs) with 95% confidence intervals (CIs) are reported using clinically interpretable scaling.

Linearity in the logit was explored using Box–Tidwell interaction terms. Influential observations were evaluated using leverage, Cook's distance, and standardized residuals. Calibration was assessed using the Hosmer–Lemeshow test, calibration intercept and slope, and a quintile-based calibration plot. Discrimination was assessed using the receiver-operating-characteristic (ROC) curve and area under the curve (AUC); the Brier score was also calculated.

Internal validation used 1,000 bootstrap resamples with a fixed random seed. Of these, 978 resamples converged successfully; mean AUC optimism was approximately 0.036, yielding an optimism-corrected AUC of approximately 0.832. Non-convergent resamples were reported rather than silently treated as successful. Sensitivity analyses included the initial 10-variable model, a log-transformed monocyte-count specification, and exclusion of the most influential observation. The primary six-variable model included all 113 participants because these six variables had no missing values. One value was missing for each of PTH, hs-CRP, and transferrin saturation; the 10-variable model therefore included 112 complete cases because of one missing hs-CRP value. All tests were two-sided and $P<0.05$ was considered statistically significant.

All analyses were performed using R software (version 4.3.2; R Foundation for Statistical Computing, Vienna, Austria). Logistic regression and model diagnostics were performed using the rms package, discrimination and calibration were assessed using the pROC and rms packages, and internal validation was performed using 1,000 bootstrap resamples with a fixed random seed.

2.5 Use of AI-assisted tools

During manuscript preparation, generative AI-assisted tools were used for language organization and editorial refinement. The authors critically reviewed and verified the scientific content, statistical results, and references, revised the manuscript as necessary, and take full responsibility for the final content.

3 Results

3.1 Study population

Among 113 patients receiving MHD, 74 (65.5%) had atherosclerotic disease and 39 (34.5%) did not. Compared with patients without atherosclerotic disease, those with disease were older (57.82±10.97 vs 47.31±13.37 years, P<0.001), more likely to have diabetes (25.7% vs 7.7%, P=0.025) and smoking exposure (40.5% vs 12.8%, P=0.003), and had greater IDWG (4.04±1.64% vs 3.34±1.69%, P=0.037). hs-CRP and ALP were higher, whereas albumin, prealbumin, and LMR were lower in the disease group (Table 1).

3.2 Univariable screening

Ten variables met the prespecified P<0.10 criterion for entry into the initial multivariable model: age, diabetes, smoking, IDWG, hs-CRP, ALP, albumin, prealbumin, monocyte count, and LMR. VIFs ranged from 1.08 to 2.21, indicating no meaningful multicollinearity.

3.3 Parsimonious multivariable model

In the parsimonious six-variable model, older age, diabetes, smoking, greater IDWG, higher ALP, and higher monocyte count were independently associated with atherosclerotic disease. The aORs were 2.41 (95% CI 1.48–3.91) per 10-year increase in age, 5.56 (95% CI 1.04–29.61) for diabetes, 5.03 (95% CI 1.41–18.00) for smoking, 1.89 (95% CI 1.27–2.82) per 1 percentage-point increase in IDWG, 1.14 (95% CI 1.01–1.29) per 10 U/L increase in ALP, and 1.56 (95% CI 1.10–2.22) per 0.1×10^9/L increase in monocyte count (Table 2).

3.4 Model diagnostics and internal validation

The apparent AUC of the six-variable model was 0.867. The Brier score was 0.141, and the Hosmer–Lemeshow test did not provide evidence of poor calibration (χ^2=7.55, P=0.479). The apparent calibration intercept and slope were

approximately 0 and 1, respectively. Maximum leverage was approximately 0.292, maximum Cook's distance was approximately 0.347, and the maximum absolute standardized residual was approximately 2.97.

Box–Tidwell testing did not show strong evidence of nonlinearity for age (P=0.491), IDWG (P=0.343), or ALP (P=0.837); monocyte count showed a borderline signal (P=0.077). A log-monocyte sensitivity specification produced a similar direction of association and did not provide strong evidence that the linear specification should be abandoned. Exclusion of the most influential observation did not reverse the direction of the principal associations, and the resulting AUC was approximately 0.885.

Across 1,000 planned bootstrap resamples, 978 fits converged. Mean AUC optimism was approximately 0.036, yielding an optimism-corrected AUC of approximately 0.832. The corresponding ROC curves, calibration plot, and adjusted-effect forest plot are shown in Figs. 1–3.

Table 1. Baseline characteristics according to atherosclerotic disease status

Variable	No disease (n=39)	Disease (n=74)	P value
Age, years	47.31 ± 13.37	57.82 ± 10.97	<0.001
Male sex, n (%)	21 (53.8)	47 (63.5)	0.419
Diabetes mellitus, n (%)	3 (7.7)	19 (25.7)	0.025
Smoking, n (%)	5 (12.8)	30 (40.5)	0.003
Dialysis vintage, months	34.00 (17.50, 55.50)	40.00 (19.25, 66.00)	0.371
IDWG, %	3.34 ± 1.69	4.04 ± 1.64	0.037
hs-CRP, mg/L	1.80 (1.80, 4.50)	3.00 (1.80, 11.30)	0.044
ALP, U/L	69.00 (59.50, 95.00)	86.50 (64.00, 122.50)	0.042
Albumin, g/L	38.90 (37.25, 40.90)	37.80 (35.60, 39.38)	0.037
Prealbumin, mg/L	271.31 ± 64.01	243.96 ± 68.57	0.038
Monocyte count, ×10^9/L	0.30 (0.25, 0.40)	0.44 (0.32, 0.54)	<0.001
LMR	3.40 (2.38, 3.95)	2.41 (1.85, 3.31)	0.005

Data are presented as mean±SD or median (IQR), as appropriate, unless otherwise indicated. IDWG, interdialytic weight gain; hs-CRP, high-sensitivity C-reactive protein; ALP, alkaline phosphatase; LMR, lymphocyte-to-monocyte ratio.

Table 2. Parsimonious multivariable logistic regression model

Predictor	aOR	95% CI	P value	Scale
Age	2.41	1.48–3.91	<0.001	per 10 years
Diabetes mellitus	5.56	1.04–29.61	0.044	yes vs no

Smoking	5.03	1.41–18.00	0.013	yes vs no
IDWG	1.89	1.27–2.82	0.002	per 1 percentage point
ALP	1.14	1.01–1.29	0.038	per 10 U/L
Monocyte count	1.56	1.10–2.22	0.012	per 0.1×10^9/L

The six-variable model included age, diabetes, smoking, IDWG, ALP, and monocyte count. aOR, adjusted odds ratio; CI, confidence interval.

Table 3. Model diagnostics and internal validation

Model/diagnostic	**Result**
Participants in primary model	113
Atherosclerotic disease, n (%)	74 (65.5)
Apparent AUC	0.867
Optimism-corrected AUC	0.832
Brier score	0.141
Hosmer–Lemeshow χ^2 (P)	7.55 (0.479)
Maximum leverage	0.292
Maximum Cook's distance	0.347
Maximum |standardized residual|	2.97
Bootstrap resamples planned / successful	1000 / 978
Mean AUC optimism	0.036
AUC after excluding most influential observation	0.885

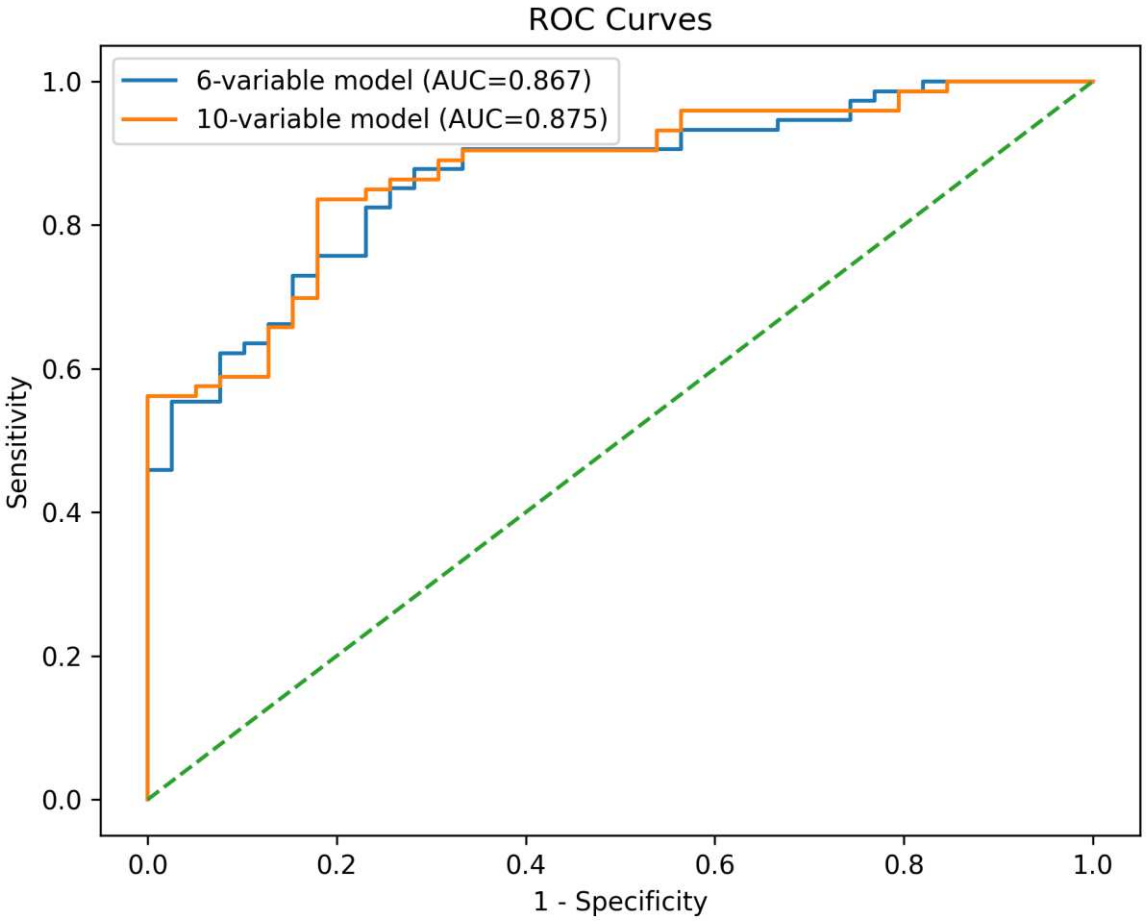


Fig. 1 ROC curves for the parsimonious six-variable model and the initial 10-variable model

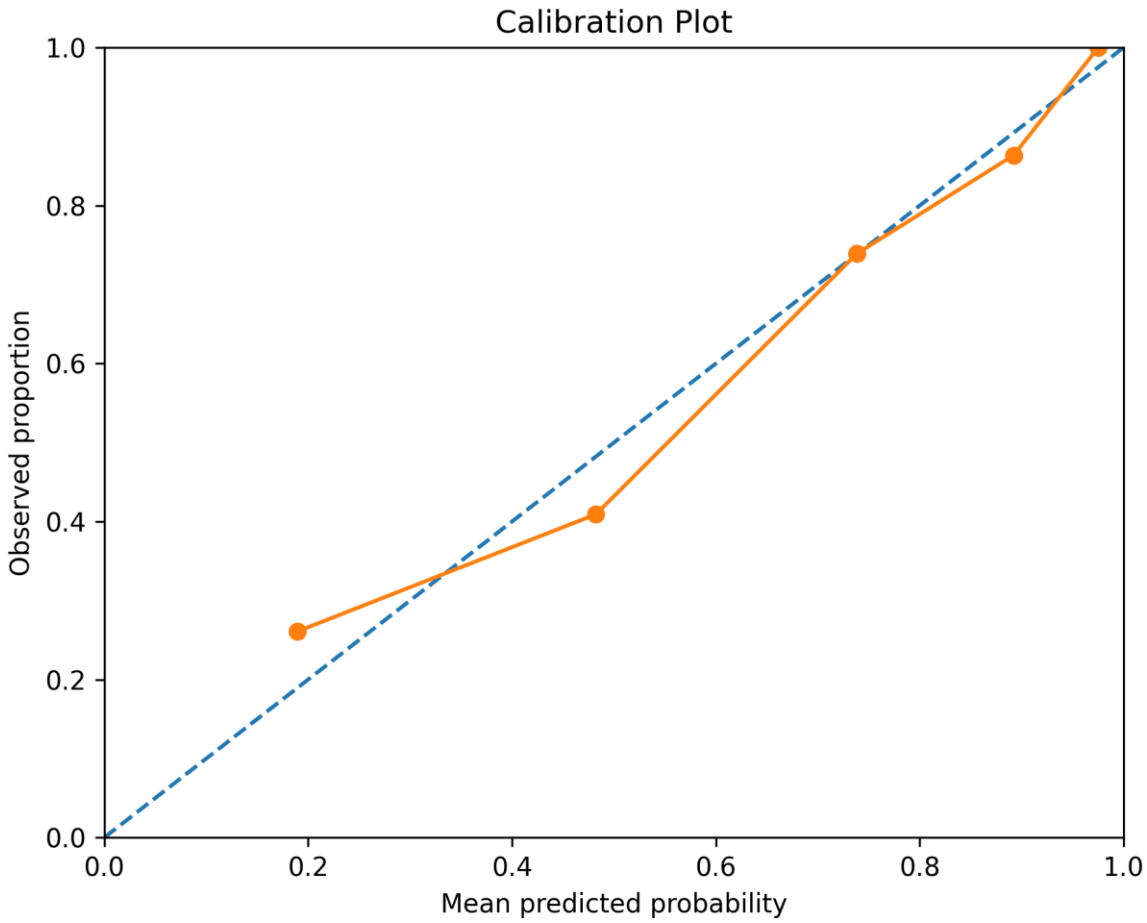


Fig. 2 Quintile-based calibration plot for the parsimonious six-variable model

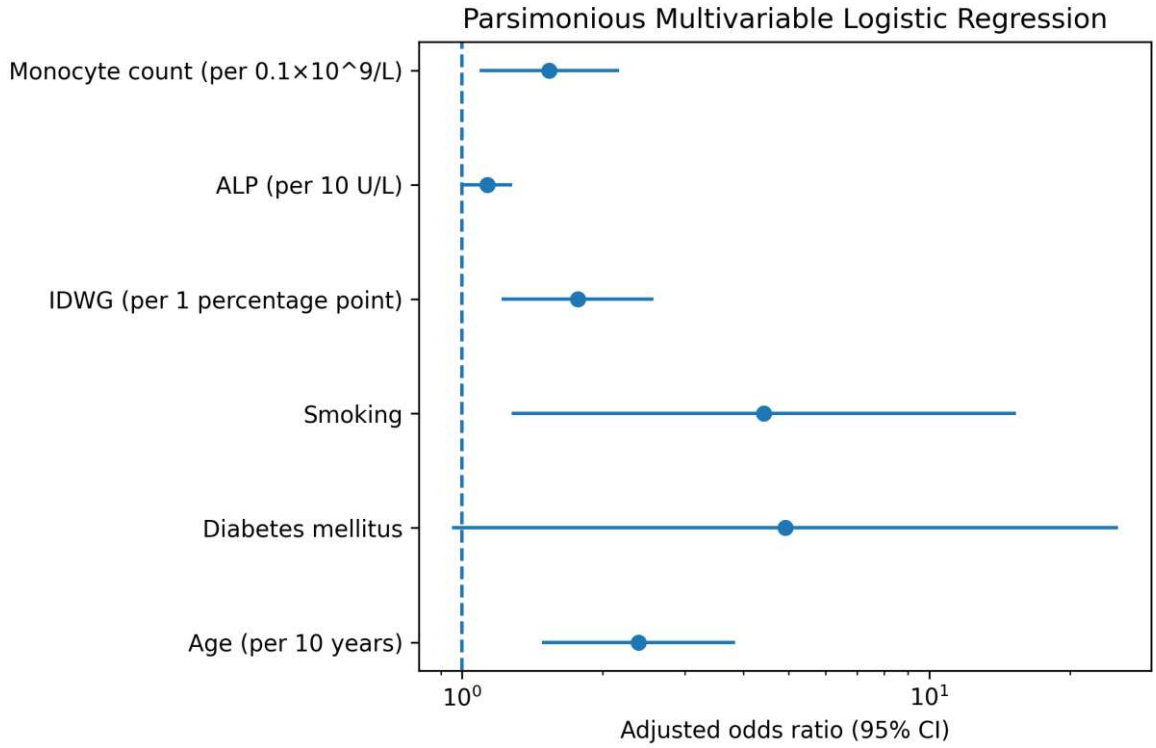


Fig. 3 Adjusted odds ratios for the parsimonious multivariable model

4 Discussion

4.1 Principal findings

In this cross-sectional study of 113 patients receiving MHD, atherosclerotic disease was identified in 65.5% of participants. Older age, diabetes, smoking, greater IDWG, higher ALP, and higher monocyte count were independently associated with the composite outcome in the parsimonious model. The direction of the principal associations remained stable across sensitivity analyses. Bootstrap correction reduced the apparent AUC from 0.867 to approximately 0.832, emphasizing the potential for optimism in a modest single-center cohort.

4.2 Age, diabetes, and smoking

Age was the strongest and most stable conventional correlate, with approximately 2.4-fold higher adjusted odds per 10-year increase. This is consistent with contemporary evidence that age is strongly associated with vascular calcification and atherosclerotic burden in MHD populations [4,5]. Diabetes was associated with a large effect estimate in the parsimonious model, although the confidence interval was wide, reflecting the relatively small number of patients

with diabetes without atherosclerotic disease. Smoking was associated with approximately fivefold higher odds, consistent with established endothelial, oxidative, and inflammatory effects of tobacco exposure.

4.3 IDWG and volume burden

IDWG was independently associated with atherosclerotic disease, with approximately 1.9-fold higher odds per 1 percentage-point increase. IDWG is a practical marker of interdialytic salt and water accumulation and is influenced by dietary sodium intake, residual kidney function, fluid management, and patient adherence. Recent evidence indicates that low-salt dietary strategies and nutritional counseling can reduce IDWG [6]. Nevertheless, the cross-sectional design prevents any conclusion that reducing IDWG would prevent or reverse atherosclerotic disease.

4.4 ALP and mineral-bone biology

Higher ALP was independently associated with atherosclerotic disease. ALP may reflect bone turnover and aspects of CKD-mineral and bone disorder and may be biologically linked to vascular calcification. However, the 2025 meta-analysis of Chinese MHD populations did not identify a statistically significant pooled association between ALP and coronary artery calcification [4]. The present association should therefore be regarded as hypothesis-generating rather than confirmatory.

4.5 Monocytes and inflammatory pathways

Higher monocyte count was independently associated with the outcome. Monocytes can adhere to activated endothelium, migrate into the vascular wall, differentiate into macrophages, and participate in lipid uptake and plaque development. The lower LMR observed in the disease group did not remain independently associated after adjustment. The borderline Box–Tidwell signal for monocyte count also suggests that future studies should examine potential nonlinear relationships using prespecified flexible functions rather than assuming strict linearity.

4.6 Attenuation of hs-CRP, albumin, prealbumin, and LMR

Several variables associated with disease status in unadjusted analyses were not retained in the parsimonious model. This may reflect overlapping inflammatory and nutritional pathways, confounding, limited statistical power, and the limitations of single-time-point measurements. The absence of an independent association in this dataset should not be interpreted as evidence that these biological pathways are clinically irrelevant.

4.7 Clinical implications

These findings support an integrated view of vascular risk in MHD that considers conventional cardiovascular factors together with interdialytic volume burden and routinely available laboratory markers. The identified factors may help generate hypotheses for future risk-stratification research. However, because this study was observational and cross-sectional, the results do not establish causality, treatment effects, or the clinical utility of any individual marker.

4.8 Strengths and limitations

Strengths include assessment of a broad range of routine clinical and laboratory variables, explicit multicollinearity assessment, clinically scaled effect estimates, model diagnostics, ROC analysis, and bootstrap internal validation. Several limitations should be emphasized. First, the cross-sectional design precludes temporal inference and causal interpretation. Second, the sample size was modest and the number of outcome events limited the complexity of multivariable modeling. Third, variable selection was data-driven, and the parsimonious model has not undergone external validation. Fourth, the composite outcome combines coronary disease, cerebral infarction/ischemic stroke, and lower-extremity arterial plaque, which represent different vascular phenotypes. Fifth, exact diagnostic modalities and thresholds must be verified from the original source records before submission, particularly for stroke etiology. Finally, residual confounding and measurement error cannot be excluded. Bootstrap internal validation improves internal optimism assessment but does not substitute for external validation.

5 Conclusions

In this cross-sectional MHD cohort, older age, diabetes, smoking, greater IDWG, higher ALP, and higher monocyte count were independently associated with atherosclerotic disease. The findings support a multifactorial framework involving conventional cardiovascular risk, interdialytic volume burden, mineral-bone biology, and inflammatory pathways. These associations are hypothesis-generating and require confirmation in larger prospective multicenter studies with standardized vascular outcome ascertainment.

Statements and Declarations

Ethics approval

The study protocol (Version 1.0, dated 20 December 2024) was approved by the Medical Ethics Committee of Baoshan Second People's Hospital, Yunnan Province, China (Ethics Approval No. 2024-041 [Zi-Zong]-01). The approval was valid from 8 January 2025 to 7 January 2026.

Consent to participate

Written informed consent was obtained from all individual participants included in the study.

Consent for publication

Not applicable; no individually identifiable information is presented.

Competing interests

The authors declare that they have no competing interests.

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Author contributions

Qiu Li: conceptualization, data curation, formal analysis, investigation, methodology, writing—original draft, and writing—review and editing. Shaojun Lu: conceptualization, methodology, supervision, and writing—review and editing. Hongyong Yu,Chun Chen,Chunyan Yang,Jian Wang,Bidong Guo,Fang Jiang,Xuejiao Mei,Yingdie Xia,Zhihang Xiao,Ying Liang,Xiaojiao Zhao,Sining Chen: data curation, investigation, and writing—review and editing. All authors read and approved the final manuscript.

Data availability

The datasets generated and/or analyzed during the current study are not publicly available because they contain clinical information that may compromise participant privacy, but may be available from the corresponding author on reasonable request and subject to institutional and ethical restrictions.

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