Caspian Journal of Internal Medicine 2026 (Spring); 17(2): 328-333
DOI: 10.22088/cjim.17.2. 328

Original Article

Prognostic role of monocyte-to-high-density lipoprotein ratio in mortality of VTE patients at Ardabil (2023-2024)

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Received: 23 July 2025
Revised: 22 Aug 2025
Accepted: 31 Aug 2025
Published: 10 March 2026

Abstract

Background: Venous thromboembolism (VTE), encompassing deep vein thrombosis (DVT) and pulmonary embolism (PE), is a leading cause of cardiovascular mortality. The monocyte-to-high-density lipoprotein ratio (MHR) is a novel inflammatory biomarker with potential prognostic value in VTE.

Methods: Venous thromboembolism (VTE), encompassing deep vein thrombosis (DVT) and pulmonary embolism (PE), is a leading cause of cardiovascular mortality. The monocyte-to-high-density lipoprotein ratio (MHR) is a novel inflammatory biomarker with potential prognostic value in VTE.

Results: The mortality rate was 9%. Higher MHR was significantly associated with mortality (adjusted OR: 1.102, 95% CI: 1.013-1.199, P = 0.023), indicating a 10.2% increased mortality risk per unit increase in MHR. No significant associations were found with hospital stay duration (P = 0.238, R^2 = 0.007), CT-scan findings (P = 0.60), or echocardiography results (P = 0.40).

Conclusion: MHR may serve as a predictor of mortality in patients with VTE, offering a cost-effective tool for risk stratification. These findings support its potential use in identifying high-risk patients for targeted interventions. Prospective studies are needed to validate and extend these results.

Keywords: Venous thromboembolism, Monocyte-to-high-density lipoprotein ratio, Mortality, Deep vein thrombosis, Pulmonary embolism.

Citation:
Aghah R, Mirak SM, Javaheri N, Sedigh-Namin A, Aliasgharzadeh Sh. Prognostic role of monocyte-to-high-density lipoprotein ratio in mortality of VTE patients at Ardabil (2023-2024). Caspian J Intern Med 2026; 17(2): 328-333.

Venous thromboembolism (VTE), encompassing deep vein thrombosis (DVT) and pulmonary embolism (PE), is a major global health concern, with an annual incidence of 1-2 per 1,000 individuals and a significant contribution to cardiovascular mortality (1, 2). DVT, characterized by thrombus formation in deep veins, primarily affects the lower limbs and can lead to PE when clots dislodge and obstruct pulmonary arteries. PE is associated with severe complications, including right ventricular dysfunction, pulmonary hypertension, and a 30-day mortality rate of up to 10%, particularly in high-risk cases (3, 4). The substantial clinical and economic burden of VTE underscores the urgent need for accessible, reliable prognostic markers to guide risk stratification and optimize patient management in diverse healthcare settings. The monocyte-to-high-density lipoprotein ratio (MHR) has emerged as a novel biomarker that integrates systemic inflammation and lipid metabolism. Monocytes, key players in the inflammatory cascade, promote thrombus formation by releasing pro-inflammatory cytokines (e.g., IL-6, TNF-α) and interacting with platelets and endothelial cells (5). In contrast, high-density lipoprotein (HDL) exerts anti-inflammatory and anti-thrombotic effects by inhibiting macrophage activation, reducing oxidative stress, and stabilizing endothelial function (6).

Recent studies have highlighted MHR's prognostic utility in cardiovascular diseases, such as acute myocardial infarction and ischemic stroke, where elevated MHR correlates with adverse clinical outcomes (7, 8). However, its application in VTE remains limited, despite the critical role of inflammation in thrombus initiation and progression. Current VTE risk stratification relies heavily on biomarkers like D-dimer and clinical scores (e.g., Wells Score, Geneva Score), but these have notable limitations. D-dimer, while sensitive for diagnosis, lacks specificity for prognostic purposes, and the neutrophil-to-lymphocyte ratio (NLR), another inflammatory marker, does not account for lipid metabolism's role in thrombosis (9, 10). Moreover, few studies have explored MHR's association with clinical outcomes, such as mortality, hospital stay duration, or imaging findings (e.g., echocardiography, CT scans), in VTE patients. Existing research often examines biomarkers in isolation, without integrating them with imaging or clinical parameters, limiting their utility in comprehensive risk assessment (11).

This gap highlights the need for studies that evaluate MHR's prognostic potential across multiple dimensions of VTE management. This study investigates MHR's relationship with mortality, hospital stay duration, and imaging findings in 200 patients with DVT and/or PE at Imam Khomeini Hospital, Ardabil, Iran (2023-2024). We hypothesized that higher MHR is associated with increased mortality and adverse clinical outcomes, reflecting its potential as a cost-effective, accessible biomarker for VTE risk stratification. By addressing this research gap, our findings aimed to inform clinical decision-making and guide future studies toward integrating MHR with established biomarkers for improved VTE management.

Methods

Study design and population: This cross-sectional, analytical study was conducted at Imam Khomeini Hospital, Ardabil, Iran, from January 2023 to December 2024, enrolling 200 consecutive patients diagnosed with venous thromboembolism (VTE), encompassing deep vein thrombosis (DVT) and/or pulmonary embolism (PE). Inclusion criteria included age ≥18 years, confirmed DVT via Doppler ultrasound, and/or PE via computed tomography pulmonary angiography (CTPA), with complete medical records. DVT was diagnosed using a Siemens Acuson S2000 ultrasound system, confirming thrombus presence in deep veins (e.g., femoral, popliteal). PE was confirmed using a 64-slice Siemens SOMATOM Definition AS scanner, identifying pulmonary artery obstruction. Exclusion criteria comprised pregnancy, active infection (e.g., sepsis, pneumonia), malignancy requiring active chemotherapy, recent major surgery (within 1 week), severe renal or hepatic dysfunction (eGFR <30 mL/min/1.73 m² or Child-Pugh class C), incomplete laboratory or imaging data, or refusal to participate.

The study was approved by the Ethics Committee of Ardabil University of Medical Sciences (IR.ARUMS.MEDICINE.REC.1402.042, approved June 6, 2023) and adhered to the Declaration of Helsinki. Written informed consent was obtained from all participants, ensuring confidentiality of personal data.

Data collection: Demographic data (age, sex, comorbidities such as diabetes, hypertension, and prior VTE), clinical data (hospital stay duration, medications, and clinical outcomes), and laboratory parameters were extracted from electronic medical records using a standardized data collection form. Laboratory tests were performed within 24 hours of admission.

Monocyte count (cells/μL) was measured using a Sysmex XN-1000 automated hematology analyzer, with quality control per manufacturer protocols. High-density lipoprotein (HDL) cholesterol (mg/dL) was quantified using a Roche Cobas 6000 chemistry analyzer with enzymatic colorimetric assays. The monocyte-to-high-density lipoprotein ratio (MHR) was calculated as monocyte count divided by HDL level (MHR = monocytes/HDL). Additional laboratory parameters included white blood cell count, neutrophil count, lymphocyte count, platelet count, prothrombin time (PT), partial thromboplastin time (PTT), lactate dehydrogenase, and arterial oxygen saturation, measured using standardized protocols. All laboratory analyses were conducted in a certified hospital laboratory, with results validated by two independent technicians to ensure accuracy.

Imaging assessments : Doppler ultrasound assessed DVT location, classified as above-knee (proximal; e.g., femoral, iliac veins) or below-knee (distal; e.g., tibial, peroneal veins). CTPA, performed within 48 hours of admission, classified PE severity as mild (<50% pulmonary artery obstruction, affecting segmental arteries) or severe (≥50% obstruction, involving main or lobar arteries), based on the Qanadli score. Echocardiography, using a Philips Epiq 7 system, evaluated pulmonary artery pressure (mmHg, estimated via tricuspid regurgitation jet velocity) and right ventricular dysfunction (e.g., increased right ventricle size, McConnell sign). Imaging was conducted by experienced radiologists and cardiologists, with findings reviewed independently by two specialists; discrepancies were resolved through consensus to minimize bias. Inter-

observer agreement was assessed using Cohen's kappa coefficient, ensuring reliability (kappa >0.8).

Statistical analysis: Data were analyzed using SPSS Version 22.0 (IBM Inc., USA). Normality was assessed with the Kolmogorov-Smirnov test. Continuous variables were reported as mean±standard deviation (SD) or median (interquartile range) for non-normal data and compared using independent t-tests (two groups) or one-way ANOVA (multiple groups). Categorical variables were analyzed with chi-square or Fisher's exact tests. Multivariable logistic regression, adjusted for age, sex, and comorbidities (e.g., diabetes, hypertension, cancer), evaluated MHR's association with mortality, reporting odds ratios (ORs) and 95% confidence intervals (CIs). Linear regression assessed MHR's impact on hospital stay duration, with R^2 for model fit. A sample size of 200 was calculated to detect a 10% difference in MHR between outcome groups (survived vs. deceased) with 80% power and $\alpha = 0.05$, assuming a 9% mortality rate. Sensitivity analyses explored MHR's performance in subgroups (e.g., DVT-only vs. PE). Missing data (<5% of cases) were handled using listwise deletion. Statistical significance was set at $p < 0.05$.

Results

Among the 200 patients with venous thromboembolism (VTE), 50.5% (n=101) were females, with a mean age of 59.14±17.22 years (table 1). Diagnoses included deep vein thrombosis (DVT) alone in 79.5% (n=159), pulmonary embolism (PE) alone in 17.5% (n=35), and both DVT and PE in 3% (n=6). DVT was classified as above-knee in 64% (n=128) and below-knee in 36% (n=72). Common comorbidities were cancer (8.5%, n=17), prior VTE (8.5%, n=17), hypertension (7.5%, n=15), and diabetes (6%, n=12). The mean hospital stay was 5.17±2.73 days, with a mortality rate of 9% (n=18).

These characteristics indicate a diverse cohort with a significant comorbidity burden, consistent with clinical VTE populations. Laboratory parameters included a mean monocyte-to-high-density lipoprotein ratio (MHR) of 9.71±5.25, white blood cell count (7661.57±1897.18 cells/μL), HDL cholesterol (38.05±10.08 mg/dL), platelet count (248.37±73.03 $\times 10^3$/μL), and lactate dehydrogenase (147.77±25.03 U/L) (table 2). All variables except platelet count ($P = 0.200$) were non-normally distributed ($p < 0.05$, Kolmogorov-Smirnov test). Echocardiography revealed normal findings in 15.5% (n=31), increased right ventricle size in 46% (n=92), and McConnell sign in 38.5% (n=77). Among 41 PE patients, CT pulmonary angiography (CTPA) showed severe pulmonary involvement (≥50% obstruction, Qanadli score) in 61% (n=25) and mild involvement (<50%) in 39% (n=16).

Table 1. Patient characteristics and diagnoses

Variable	Value
Age (years, mean±SD)	59.14±17.22
Female, n (%)	101 (50.5%)
DVT alone, n (%)	159 (79.5%)
PE alone, n (%)	35 (17.5%)
DVT + PE, n (%)	6 (3%)
DVT location: Above-knee	128 (64%)
DVT location: Below-knee	72 (36%)
Cancer, n (%)	17 (8.5%)
Prior VTE, n (%)	17 (8.5%)
Hypertension, n (%)	15 (7.5%)
Diabetes, n (%)	12 (6%)
Hospital stay (days, mean±SD)	5.17±2.73
Mortality, n (%)	18 (9%)

Baseline characteristics, diagnoses, comorbidities, and clinical outcomes of 200 patients with venous thromboembolism (VTE). (SD: Standard Deviation, DVT: Deep Vein Thrombosis, Pulmonary Embolism)

Table 2. Laboratory parameters

Parameter	Mean±SD	P-value (Normality)
MHR	9.71±5.25	<0.05
WBC (cells/μL)	7661.57±1897.18	<0.05
HDL (mg/dL)	38.05±10.08	<0.05
Platelet count ($\times 10^3$/μL)	248.37±73.03	0.200
Lactate dehydrogenase (U/L)	147.77±25.03	<0.05

(SD: Standard Deviation, MHR: monocyte-to-high-density lipoprotein ratio, HDL: High-Density Lipoprotein)

MHR was significantly higher in deceased patients (12.40±5.12) compared to survivors (9.34±5.12, $P = 0.02$, independent t-test) (table 3). Multivariable logistic regression, adjusted for age, sex, and comorbidities (diabetes, hypertension, cancer), confirmed MHR as a predictor of mortality (adjusted OR: 1.102, 95% CI: 1.013-1.199, $P = 0.023$), indicating a 10.2% increased mortality risk per unit increase in MHR (table 4). Sensitivity analyses showed consistent associations in DVT-only (OR: 1.098, $P = 0.031$) and PE (OR: 1.115, $P = 0.029$) subgroups, suggesting robust prognostic value. Linear regression

revealed no significant association between MHR and hospital stay duration (β = 0.007, 95% CI: -0.004-0.018, P = 0.238, R^2 = 0.007), indicating limited utility for predicting short-term clinical course (table 4). MHR did not differ significantly by DVT location (below-knee: 9.41±5.74 vs. above-knee: 9.81±4.35, P = 0.50, t-test) or echocardiography findings (normal: 8.96±5.14; increased right ventricle size: 9.58±5.19; McConnell sign: 9.92±5.57, P = 0.40, ANOVA) (table 3). Similarly, MHR was comparable between severe (9.36±5.41) and mild (9.71±4.90) PE on CTPA (P = 0.60, t-test). These findings suggest MHR's prognostic value is primarily linked to mortality rather than imaging-based severity markers.

Table 3. MHR Comparisons across outcomes and imaging findings

Variable	Mean MHR±SD	P-value
Mortality		0.02
Survived (n=182)	9.34±5.12	
Deceased (n=18)	12.40±5.12	
DVT Location		0.50
Below-knee (n=72)	9.41±5.74	
Above-knee (n=128)	9.81±4.35	
Echocardiography		0.40
Normal (n=31)	8.96±5.14	
Increased RV size (n=92)	9.58±5.19	
McConnell sign (n=77)	9.92±5.57	

Laboratory parameters in 200 VTE patients, with normality assessed by Kolmogorov-Smirnov test. (SD: Standard Deviation, DVT: Deep Vein Thrombosis)

Table 4. Regression analyses for MHR associations

Outcome	OR/β (95% CI)	P-value
Mortality (logistic, adjusted)	1.102 (1.013-1.199)	0.023
DVT-only subgroup (logistic)	1.098 (1.009-1.195)	0.031
PE subgroup (logistic)	1.115 (1.011-1.230)	0.029
Hospital stay (linear, β)	0.007 (-0.004-0.018)	0.238

Comparison of monocyte-to-high-density lipoprotein ratio (MHR) by mortality (independent t-test), DVT location (t-test), and echocardiography findings (one-way ANOVA) in 200 VTE patients.

Discussion

This study delineates the prognostic significance of the monocyte-to-high-density lipoprotein ratio (MHR) in a meticulously characterized cohort of patients with venous thromboembolism (VTE) at Imam Khomeini Hospital, Ardabil, Iran. The calculated mean MHR in this population exhibits substantial heterogeneity in inflammatory and lipid profiles, reflecting a notable comorbidity burden involving malignancy, hypertension, and diabetes (1). The observed mortality rate aligns with global estimates for VTE-related lethality in tertiary care settings, where complex cases predominate (3). This underscores the need for accessible biomarkers in resource-limited contexts. Unlike D-dimer, which boasts high sensitivity but limited prognostic specificity (2), MHR amalgamates monocyte-driven inflammation mediated by cytokines like IL-6 and TNF-α with HDL's antithrombotic effects, offering a novel lens for risk stratification (12).

The positive correlation between elevated MHR and mortality, characterized by a statistically significant increase in the likelihood of death for every unit rise in the ratio, positions it as a compelling prognostic marker. This association is further substantiated by a markedly higher mean MHR observed in deceased patients compared to survivors, reflecting a more intense thrombo-inflammatory state in fatal cases. The consistent performance of this marker across both DVT-only and PE subgroups—even after rigorous adjustment for potential confounders such as age, sex, and underlying comorbidities—affirms the versatility and clinical reliability of MHR as a predictive tool (7). This mirrors cardiovascular evidence linking MHR to thrombus formation and endothelial dysfunction via pro-inflammatory pathways (13), with multivariate adjustment enhancing its validity over unadjusted analyses. The absence of significant associations between MHR and imaging or hospital stay outcomes warrants careful scrutiny. MHR showed no notable variation by DVT location (P = 0.50) or echocardiography findings (P = 0.40), and its correlation with hospital stay duration was negligible (P = 0.238, R^2 = 0.007). This specificity to mortality, rather than structural severity or short-term resource use, suggests MHR reflects systemic inflammation and lipid dysmetabolism rather than localized thrombus dynamics (14). The non-significant CT pulmonary angiography (CTPA) result in the PE subgroup (n=41) may stem from limited statistical power or indicate MHR's independence from pulmonary obstruction, diverging from neutrophil-to-lymphocyte ratio (NLR) studies that correlate with imaging severity (10). This distinction invites further mechanistic exploration. These findings both converge with and diverge

from existing literature. Dogan et al. (2023) (15) reported no MHR-thrombus burden link in chronic DVT, possibly due to their focus on late-stage disease versus our acute cohort, or differing imaging modalities. Meng et al. (2023) (16) identified MHR's predictive role in ischemic stroke but not hemorrhagic transformation, suggesting context-specific effects extensible to VTE. The lack of hospital-stay association echoes Kundi et al. (2015) (10), who noted inflammatory markers' limited utility for short-term outcomes, advocating composite models. Compared to the Geneva Score, which relies on clinical variables (2) MHR provides a biochemical anchor, as supported by recent analyses of inflammatory indices in myocardial infarction (11). Clinically, MHR's derivation from routine hematology and lipid panels heralds a transformative approach to VTE risk assessment. An MHR exceeding 12.40 (the deceased cohort mean) may flag patients for intensified monitoring or tailored anticoagulation, leveraging its adjusted OR to potentially reduce mortality (7). However, its dissociation from hospital stay duration necessitates a multi-marker strategy, integrating MHR with D-dimer or the Wells Score for holistic evaluation. In low-resource settings, where advanced imaging is limited, MHR could enhance triage, though prospective trials are critical to establish cut-offs and therapeutic thresholds, ensuring its scalability (17). Future research must overcome current limitations to maximize impact. A larger, multicenter cohort particularly enriching the PE subgroup would bolster statistical power and generalizability (3). Longitudinal studies could elucidate MHR's prognostic trajectory beyond the acute phase, informing long-term care. The absence of a receiver operating characteristic (ROC) analysis, due to unavailable sensitivity-specificity data, is a key gap; future efforts will prioritize this to define optimal thresholds (11). Exploring MHR's synergy with NLR, C-reactive protein, or D-dimer, and its modulation by therapies like heparin, could refine its role. Collaborative, global initiatives leveraging VTE registries are vital to validate MHR's utility and address regional variations, cementing its role in precision medicine.

This study's focus is limited to a specific population of patients with similar characteristics (such as underlying diseases and advanced age), which may make it difficult to generalize the results to other populations. While we adjusted for cancer status and major comorbidities, unmeasured confounders (e.g., undiagnosed malignancies, thrombophilia profiles) may influence MHR's prognostic value of MHR. Additionally, this research is retrospective in design, and due to the constraints associated with this type of study, some vital information may not have been recorded, which could impact the final results. Due to the limited number of mortality events (n=18), we could not reliably determine the optimal prognostic cutoff for MHR via ROC analysis. Future larger-scale studies are necessary to define and validate a clinically applicable threshold. Furthermore, the study only addresses the immediate outcomes of the patients, and the long-term effects have not been examined. According to the findings of this study, it is suggested that MHR is recognized as a prognostic marker for mortality in patients associated with PE or/and DVT. The MHR may have a utility in identifying high-risk individuals who may require more aggressive clinical interventions. These findings highlight the crucial role of systemic inflammation and lipid metabolism in the development of DVT and PE.

Acknowledgments

The authors would like to thank the Medical Faculty of Ardabil University for their utmost support.

Funding: N/A.

Ethics approval: This project has been approved by the Research Ethics Committee of Babol University of Medical Sciences with code IR.MUBABOL.HRI.REC.1402.219.

Conflict of interests: The authors declare no conflicts of interest.

Authors' contribution: RA: Data curation, Investigation, Visualization, Writing – original draft; SMM: Methodology, Software, Formal analysis, Writing – review & editing; NJ: Validation, Resources, Writing – review & editing; ASN: Conceptualization, Project administration, Supervision; SA: Conceptualization, Supervision, Funding acquisition, Writing – review & editing, Final approval.

Institutional review board statement: The study was approved by the local Ethics Committee of the Ardabil University, Faculty of Medical Sciences (approval no.: 1402.042; date of approval: 6 June 2023). All of the experimental procedures were conducted by the principles embodied in the Declaration of Helsinki.

Informed consent statement: Informed consent was obtained from all subjects involved in the study.

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