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Monocyte-to-HDL Ratio as a Predictor of New-Onset Atrial Fibrillation Following Coronary Revascularization: A Systematic Review

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Abstract

Introduction Intriguingly, atrial fibrillation (AF) can be a common and severe complication after coronary revascularization and poses a high risk of stroke, heart failure, and death. A new biomarker that has come up is the Monocyte-to-HDL Ratio (MHR) that represents the balance between the inflammatory activity of the monocytes and the anti-inflammatory action of the high-density lipoprotein cholesterol (HDL-C) on the body. This is a systematic review that intends to determine the predictive role of admission MHR on new-onset atrial fibrillation (NOAF) among patients receiving percutaneous coronary intervention (PCI) or coronary artery bypass Grafting (CABG).

Materials and Method: A systematic search of the PubMed, Embase, Cochrane Library and Google Scholar was performed up to early 2026. Including observations that evaluate pre-operative MHR and its correlation with NOAF in adult patients during revascularization activities was included. The Newcastle-Ottawa Scale was used to measure quality.

Findings: Five studies that covered 2,565 patients were identified. NOAF occurred in a period between 5.1 and 6.4 percent in STEMI patients during the PCI intervention, 17.5 percent in elderly ACS patients and 22.8 to 23.1 percent in surgical CABG populations. In all the studies, elevated MHR was a strong independent predictor of NOAF. In PCI cohorts, the MHR predictors of NOAF such as cut-off values ranging between 15.87 and 26.54 showed significant forecasting (AUC up to 0.768). MHR was found to increase risks of arrhythmia (OR: 11.51) and was a good predictor of

general mortality in CABG patients.
Conclusion: MHR is a valid, economically reliable and independent biomarker in foretelling NOAF following percutaneous and surgical revascularization. The incorporation of MHR into the framework of clinical risk stratification can possibly help identify high-risk patients earlier and implement specific prophylactic interventions to enhance outcomes.

Introduction

Atrial fibrillation (AF) is the most frequent sustained cardiac arrhythmia in clinical practice and is the leading cause of morbidity and mortality in the entire continuum of cardiovascular medicine [1]. It is increasing at the world level especially with the aging of population and increase in metabolic and ischemic heart diseases. In the particular administration of acute coronary syndromes (ACS), particularly ST-segment elevation myocardial infarction(STEMI) and the post-percutaneous revascularization and surgical revascularization, AF is a severe secondary event [2]. It is not a supporting or temporary electrical complication; it is a clinically noteworthy occurrence with a reported incidence of 5%-14% in acute ischemic cases with much higher rates being reported in sensors of surgery; that is, those who undergo coronary artery bypass grafting (CABG) with rates of 22.8 to 60 in occurrence of this overt condition [2, 3]. The development of NOAF is a significant predictor of adverse clinical outcomes, which are hemodynamic instability, acute heart failure, embolic stroke, and a significant increase in in-hospital and extended or prolonged mortality rates [4]. Therefore, to support the early application of prophylactic intervention and optimum rhythm monitoring, the identification of reliable, affordable, and biomarkers that are universal and applicable to all patients in high risk of developing NOAF has become a clinical emergency.

Systemic inflammation and oxidative stress appear to be synergies in the complex pathophysiology of AF development, which is gaining more and more ground [5]. These mechanisms are involved in maladaptive atrial tissue structural and electrical remodeling, which is primarily facilitated by accelerating interstitial fibrosis, mechanism of normal intracellular communications, and the process of calcium regulation. The circulation of monocytes is associated with a crucial role compared to this inflammatory environment [6]. In response to myocardial ischemia or procedural trauma, the monocytes migrate into the atrial myocardium and secrete several pro-inflammatory cytokines, matrix metalloproteinases, and pro-oxidant mediators that weaken atrial integrity and facilitate the activity of myofibroblasts into the atria [6, 15]. On the other hand, high-density lipoprotein cholesterol (HDL-C) comes in with counter-measures against it due to its potent anti-inflammatory, antioxidant and antithrombotic effects [7]. HDL-C is reported to inhibit the activation of monocytes by inhibiting the expression of CD11b and the synthesis of monocyte chemoattractant protein-1, which prevents the occurrence of oxidative injury and atrial fibrillation [7, 8].

The Monocyte-HDL Ratio (MHR) has a predictive potential which is explained by the fact that its exclusive advantage is to show the actual relationship between this pro-inflammatory monocyte activity and the HDL-C anti-inflammatory protective capacity [9]. The combination of these two antagonistic biological powers into one numerical index will result in MHR being effective in quantifying the "inflammatory-metabolic load" triggering atrial remodeling. Although MHR was originally proven as a prognostic variable in the condition of chronic kidney disease and stable atherosclerosis, its application has extended quite quickly to more acute cardiovascular conditions [8, 25]. Some of the more recent developments have shown how it can predict recurrence of AF after catheter ablation and has been an early warning of bad news in the patient developing non-valvular AF [9]. Nevertheless, the exact ability of MHR to forecast NOAF after acute revascularization of the case of

percutaneous coronary intervention (PCI) or a CABG is yet to be clarified. Conventional risk models, mostly relying on the presence of clinical comorbidities in a relatively stable state, are often not sufficient to explain the highly inflammatory acute condition that occurs during revascularization [10]. The proposed systematic review aims to conduct the synthesis of the existing literature to identify whether admission MHR levels are an independent and a significant predictor of the development of NOAF in this high-risk group and can provide a more detailed insight into risk stratification.

Materials and Methods

This systematic review was written and presented according to the Preferred Reporting Items of a Systematic Review and meta-analysis (PRISMA) guidelines in strict compliance with them. A systematic search was performed in a number of the largest electronic databases, such as PubMed, Web of Science, Embase, and Cochrane Library, to cover the time of their creation up to the beginning of 2025. The search strategy was based on the search using a mix of Medical Subject Headings (MeSH) with the free-text keywords to provide the maximum sensitivity and literature coverage. Such search terms were monocyte-to-HDL ratio, MHR, new-onset atrial fibrillation, NOAF, postoperative atrial fibrillation, revascularization, percutaneous coronary intervention, PCI, and coronary artery bypass grafting, or CABG. In order to get a final large data set, the bibliographies of all the potential eligible primary studies and existing reviews were vetted manually to identify more relevant literature that might not be discovered initially by the original electronic search.

A strict Population, Exposure, Comparator, Outcome framework of PICO was used to define these eligibility criteria in this review. The target population was the adult patients aged 18 years and above with coronary artery disease who had previously undergone percutaneous or surgical revascularization. This comprised of patients coming with stable angina, unstable angina as well as acute myocardial infarction. Exposure of interest was explained as a high percentage of pre-procedural or admission monocyte-to-HDL in peripheral blood. The comparator has used patients with low or normal baseline MHR levels based on the individual study level. The first outcome was incidence, i.e. the first instance of atrial fibrillation during the index admission or the follow-up period. According to this structure, only peer-reviewed observational studies such as prospective and retrospective cohort or a case-control study were included. We filtered out systematically any article that dealt with patients with a previously known history of atrial fibrillation, or which dealt with non-cardiac surgery, or an evaluation of medical management in the absence of a revascularization component. Also, abstracts, case reports, review articles and studies that were reported in other languages other than English were eliminated.

To focus on minimizing the risk of bias and achieve accuracy, the two investigators conducted the selection of the studies and the process of data extraction independently. Initial screening including titles and abstracts was conducted to remove irrelevant entries and the final step was extensive review of the rest of the papers based on the inclusion and exclusion criteria. Any conflicting interpretation between the reviewers was resolved to a common agreement or where needed the third senior researcher was consulted. The data were inputted into a structured electronic database, which was able to capture the key information like the main author, date of publication, country and research design. We additionally documented patient-specific demographics, sample sizes, the nature of revascularization operation, time of sampling to MHR, and cut-off values to be used in predicting risks specific to samples. Technical accuracy was considered; monocyte counts were reported most often in two different forms in all included studies both as absolute counts per microliter (cells/ uL) and as counts per million cells per microliter (1 10 3/ uL). On the contrary, HDL-C was always measured in milligrams per decilitter (mg/dL). Systematic collection of frequency

data in statistical measures, such as unadjusted and adjusted hazard rates (HR) or odds rates (OR) and its 95% confidence interval (CI) was collected to determine and analyze statistical relationships.

The methodological quality and the risk of bias of the included studies were rated separately using the Newcastle-Ottawa Scale (NOS) of observational studies see table 1. This evaluation instrument appraises researches around three general viewpoints: the choice of the study groups, the parallels of the groups, and the declaration of the exposure or outcome of the colleges. The studies with a score of seven and beyond were deemed to be of good methodology and given priority in the synthesis. Due to the characteristics of this type of systematic review, the narrative synthesis method was applied to present the findings. The outcome measures were broken down and discussed according to the type of procedure, which was either PCI or CABG, in order to overcome the natural clinical and pathophysiological differences between the two different revascularization pathways. The correlations between elevated MHR and the probability of developing NOAF were qualified in nature, indicating the similarities of finding and the clinical importance of the suggested MHR levels in the various studies.

Table 1 Quality assessment of the reviews studies by Newcastle Ottawa scale

Study	**Representativeness of exposed cohort**	**Selection of nonexposed cohort**	**Ascertainment of exposure**	**Outcome not present at start**	**Comparability (Adjusted for factors)**	**Assessment of outcomes**	**Length of follow-up**	**Adequacy of follow-up**	**Total Score**
Karataş et al. (2016)	*	*	*	*	**	*	*		*******
Saskin et al. (2017)		*	*	*	**	*	*	*	*******
Ulus et al. (2018)	*	*	*	*	*	*	*	*	*******
Tanık et al. (2025)	*	*	*	*	**	*			******
Tekkesin et al. (2017)		*	*	*	*	*	*		*****

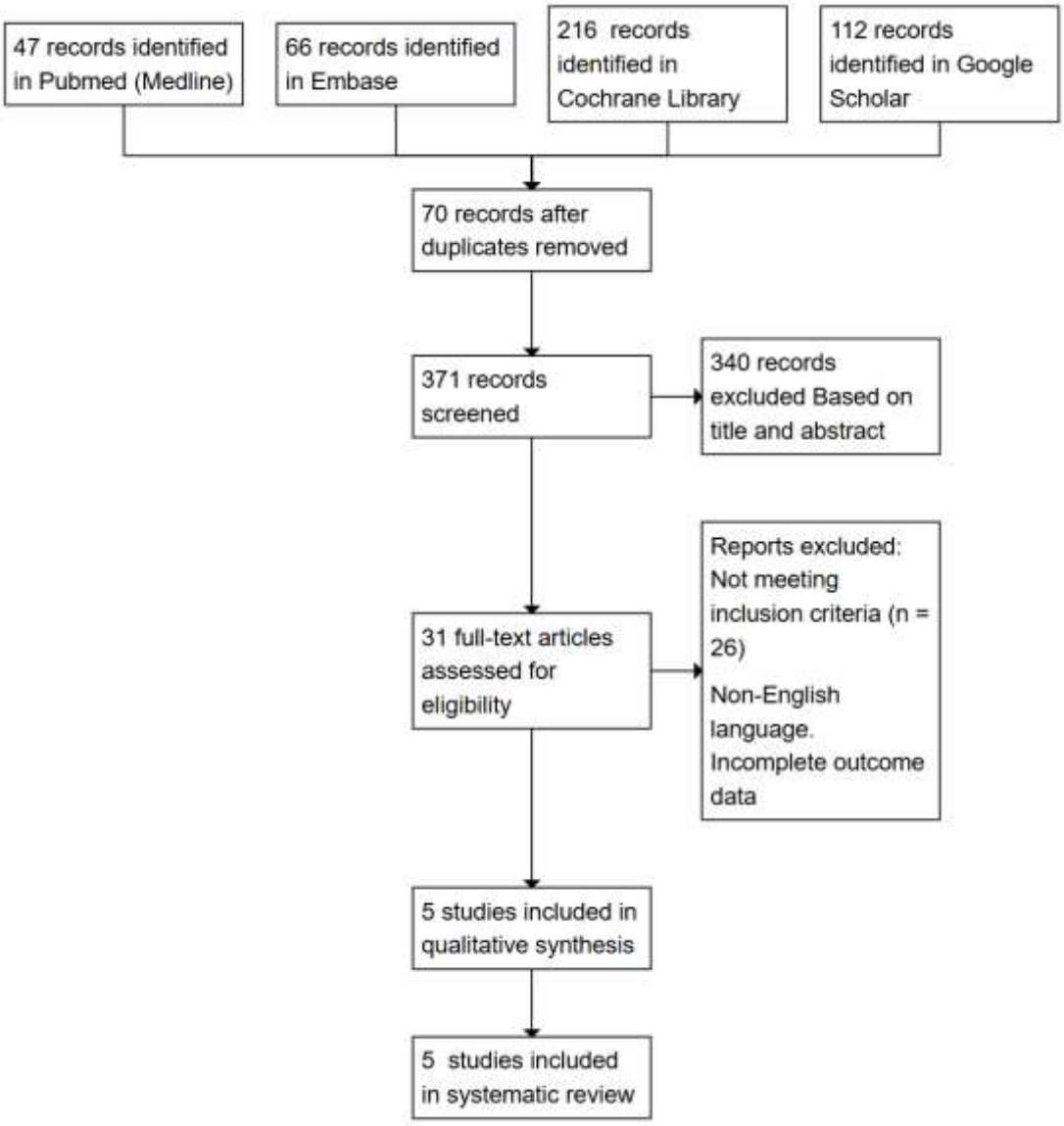

Table 2. Prisma Flow Diagram

Results

There were five main observational studies that included 2,565 patients in total that satisfied all the inclusion criteria, which were identified during the qualitative synthesis. Another important result of these studies is that the rates of NOAF differ considerably depending on the specifics of procedures and clinical situation. The rates of NOAF were comparatively low in patients primary percutaneous coronary intervention cases of STEMI of between 5.1 and 6.4% [11, 15]. Nevertheless, in the case of elderly patients (75 years and above) with acute coronary syndrome (ACS), the data prevalence was significantly greater being 17.5% indicating the high susceptibility of the geriatric population to the rhythm disturbances in the ischemic strain [14]. Arrhythmia occurred the most in the operating environment, and the largest percentage was found in studies on isolated coronary artery bypass grafting with postoperative atrial fibrillation (POAF) ranging between 22.8 and 23.1 percent [12, 13]. In all the covered studies, pre-procedural or admission MHR levels were always significantly and statistically different in patients who developed AF versus those that stayed in sinus rhythm indicating the same biological signal in various revascularization strategies.

MHR became a strong independent predictor of NOAF in the subgroup of patients undergoing primary PCI because of STEMI. Karatabas et al. found the adjusted Odds

Ratio (OR) of NOAF after primary PCI was 1.013 (95% CI: 1.004 1.022) and based it on an optimum MHR cut-off of 25.81 [11]. More recent evidence by Tanik et al. of 663 STEMI patients supported these results but reported a much greater adjusted OR of 1.413 (95% CI: 1.2031657) in patients with an MHR greater than 26.54 [15]. This exclusion achieved a high diagnostic accuracy with sensitivity and specificity being greater than 70 percent and Area Under the Curve (AUC) of 0.768 [15]. A similarly critical correlation between NOAF and survival was also noted in the study by Tanik et al., with a mortality rate of 11.8 on the NOAF group and a mere 4.3 on those who did not enter into fibrillation (p = 0.044) [15]. MHR was also found to be an independent predictor (OR: 1.102, 95% CI: 1.0541.152) with lower threshold cut-off of 15.87 in geriatric patients with high baseline comorbidities and found by Ulus et al. to be an independent predictor of arrhythmia in the elderly population (14).

The statistical power of MHR specifically did not fail in the surgical CABG cohorts, where the pro-inflammatory surges are generally stronger in nature since they are characterized by the surgical operation. As Saskin et al. indicated, a higher preoperative MHR had a 11.5-fold chance of occurrence of postoperative atrial fibrillation (Adjusted OR: 11.51, 95% CI: 1.25106.6) [13]. In addition, this research paper found MHR as an independent predictor of general mortality (P = 0.0001), which implies that it is a valuable prognostic factor in cardiac surgery [13]. In the same vein, Tekkesin et al. tested MHR on 311 isolated CABG patients and determined that it has been a superb predictor with Area Under the Curve (AUC) of 0.844 [12]. The statistically significant adjusted OR of 51.81 (95% CI: 11.47 233.8) was reported between patients who had a cut-off value that was more than the optimal cut-off reported by them: 8.55 [12]. The curves of ROC analysis of all types of revascularization revealed that MHR has an average to high quality of diagnostic accuracy, whose AUC values remained the same, between 0.750 and 0.844. Such studies provided confirmation that MHR was an independent predictor of NOAF which was still persisting even after controlling the conventional risk factors like the old age, left atrial diameter, and C-reactive protein.

Table 3: Characteristics and Statistical Strength of Included Core Studies

Characteristic	Study Setting	Study Population	Sample Size (n)	Design Type	AF Outcome	Procedure	Mean/Median Age	% Male	AF Incidence
Karataş et al. (2016)	Cardiology Dept, Istanbul	STEMI patients	621	Retrospective	New-onset AF (NOAF)	Primary PCI	57.0+\-11.9 yrs	74.70%	6.40%
Saskin et al. (2017)	CV Surgery, Kocaeli	Isolated CABG	662	Retrospective	Postoperative AF (POAF)	Cardiac Surgery (CPB)	62yrs (Median)	81.70%	23.10%
Ulus et al. (2018)	Cardiology, Eskisehir	Elderly ACS (>65)	308	Prospective	New-onset AF (NOAF)	PCI	74.4+\-m6.5 yrs	66.20%	17.50%
Tanık et al. (2025)	Cardiology,	Acute STEMI	663	Retrospective	New-onset AF	Primary PCI	55.8+\-m12.9yrs	86.30%	5.10%

	Ankara				(NOAF)				
Tekkesin et al. (2017)	Cardiology, Istanbul	Isolated ACBG	311	Prospective	Postoperative AF (POAF)	Bypass Surgery	60.1\pm8.7 yrs	66.70%	22.80 %

Discussion

The evidence synthesis of this systematic review supports that an increment in the Monocyte-to-HDL Ratio (MHR) is a potent and independent foreboding of new-onset atrial fibrillation (NOAF) in the postsurgical period of coronary revascularization. This predictive ability is analogous at the percutaneous and surgical pathways, which points out MHR as a flexible tool in contemporary cardiology. The inherent power of MHR is in the fact that it represents the dynamic combination of the pro-inflammatory monocyte response to myocardial injury and the anti-inflammatory power of HDL-C. Although the conventional clinical risk factors, including the advanced age and the enlarged left atrial ventricles are critical, they do not always explain the presence of the acute systemic inflammatory-metabolic condition, a major precursor of atrial arrhythmogenesis [16, 17]. Recent data indicated that systemic inflammation can underlie the initial electrical stimulus to AF besides having a role in enhancing the structural remodeling and fibrosis that support its continuation and recurrence [17, 18]. By encompassing this inflammatory condition, MHR offers a more detailed risk profile of the patient in comparison to such individual markers as monocyte count.

One good piece of evidence in this review is that the AF incidence among patients experiencing surgical revascularization (CABG) is high in contrast to the patients receiving PCI. This disparity probably can be explained by the high-intensity and localized and systemic inflammatory responses linked to cardiopulmonary bypass, atria manipulation of the heart, and postoperative pericarditis which are powerful contributors to arrhythmia [18, 20]. Regarding acute coronary syndromes, acute influx of oxidative stress and the discharge of pro-oxidant mediators by the monocytes make the atrial tissue even less stable [19]. Moreover, it is reported by modern prognostic research that markers targeting such inflammatory-metabolic equilibrium give a higher predictive potential than individual hematological ones since they contain the defensive reaction of the body to oxidative injury [5, 20]. It is especially applicable in vulnerable groups, i.e., the elderly, where age-specific physiological alterations and chronic low-grade inflammation are in combination with acute ischemic events increasing the risk of NOAF [14, 21].

The difference between optimal values of MHR cut-off determined in the present review (8.55- 26.54) deserves attention. This difference is probably multifactorial, as it is caused by the variations in the units of the laboratory measurements, risk profiles of the populations at the baseline and the time in which the blood was drawn. The parameter of Admission MHR, introduced in the STEMI and ACS investigations [11, 15] reflects the acute spike of inflammation linked to the actual event of the infarction. Conversely, MHR measures in stable CABG preoperative patients indicate a more underlying, persistent inflammatory condition [12, 13, 16].Moreover, investigations, which gave monocyte counts in thousands per microliter (10 3 /uL), as in the case of Saskin et al., report lower thresholds than those using absolute cell counts [13]. These numerical differences notwithstanding, the directional correlation was the same in all the studies: a higher ratio was a consistent indicator of a better risk of arrhythmia.

Notably, MHR seems to have incremental predictive value as compared to conventional clinical parameters and fixed risk models. Although conventional scores such as the CHA2DS2-VASc pay attention to known comorbidities, the C2HEST

score is purposely created to forecast the noise-adjusted failure in ACS [17]. The incorporation of MHR in the C2HEST score that includes Chronic obstructive pulmonary disease, Hypertension, Elderly age, Stroke, and Thyroid disease might potentially refine the accuracy of risk modeling with the introduction of a dynamic indicator of the inflammatory-metabolic condition [17, 18]. More to the point, the fact that the independent relationship between a high MHR and overall mortality is observed not only in Saskin and Tanik studies [13, 15] indicates that MHR is not just an arrhythmia predictor factor but a holistic predictor of the current physiological strain load that predetermines the overall prognosis of the patient [22, 23, 25]. Early detection of high-risk individuals enables the introduction of individualized prophylaxis measures, like enhanced rhythm surveillance or ideal utilization of anti-arrhythmic and anti-inflammatory treatment in the risky phase of perioperative period [24].

Limitations

This is a systematic review that is subject to a number of limitations that should be mentioned in order to present a balanced picture about the findings. First, most of the studies included employed a retrospective design, which in the nature of its research, cannot point out an ultimate causal correlation between the levels of MHR and the development of AF. Second, although the cumulative sample size was adequate to conduct narrative synthesis, the size of each study was moderate, which can restrict the ability to apply particular cut-off values to other various populations in the world. Third, the measurement units and the time of blood collection on the varied clinical environments had a remarkable lack of standardization that leads to the broad gap between the preferred thresholds reported. Lastly, confounding variables like using statins, ACE inhibitors or beta-blockers, which are known to affect both monocyte activity and HDL-C values, were not consistently adjusted throughout all of the statistical models and may have influenced the accuracy of the odds ratios that were reported.

Conclusion

To sum up, the Monocyte-to-HDL Ratio is a valid, economical, and very convenient biomarker that can be used to forecast the emergence of atrial fibrillation of new onset after coronary revascularization. The fact that it is able to indicate risk even without conventional determinants when used in a wide range of clinical environments, including primary PCI in acute myocardial infarction and, just to highlight this point, surgery CABG can be effectively used to convey its great clinical value. Taking into consideration the developed correlation between NOAF and poor long-term outcomes, that is, higher mortality and longer hospitalization, MHR can be viewed as an effective screening tool regarding early risk stratification. The key focus of future research should be on prospective, multicenter studies to confirm the standardized MHR thresholds, and to investigate the opportunities to combine such an inflammatory index with the refined risk factors such as the C2HEST score. Finally, individualized cardiovascular care can be enhanced through the use of MHR, and the results of clinical outcomes in patients with coronary artery disease can be better.

References

Van Gelder IC, Rienstra M, Bunting KV, et al. 2024 ESC Guidelines for the management of atrial fibrillation developed in collaboration with the European Association for Cardio-Thoracic Surgery (EACTS). Eur Heart J. 2024;45(36):3314-3414. doi: 10.1093/eurheartj/ehae176.

Dal Zotto B, Barbieri L, Tumminello G, et al. New onset atrial fibrillation in STEMI patients: main prognostic factors and clinical outcome. Diagnostics (Basel). 2023;13(4):613. doi: 10.3390/diagnostics13040613.

Băghină RM, Crișan S, Luca S, et al. Association between inflammation and new-onset atrial fibrillation in acute coronary syndromes. J Clin Med. 2024;13(17):5088. doi: 10.3390/jcm13175088.
Zeymer U, Annemans L, Danchin N, et al. Impact of known or new-onset atrial fibrillation on 2-year cardiovascular event rate: results from the EPICOR registry. Eur Heart J Acute Cardiovasc Care. 2019;8(2):121-129. doi: 10.1177/2048872618769057.
Sagris M, Vardas EP, Theofilis P, et al. Atrial fibrillation: pathogenesis, predisposing factors, and genetics. Int J Mol Sci. 2021;23(1):6. doi: 10.3390/ijms23010006.
Liu X, Zhang W, Luo J, et al. TRIM21 deficiency protects against atrial inflammation and remodeling post myocardial infarction by attenuating oxidative stress. Redox Biol. 2023;62:102679. doi: 10.1016/j.redox.2023.102679.
Jomard A, Osto E. High density lipoproteins: metabolism, function, and therapeutic potential. Front Cardiovasc Med. 2020;7:39. doi: 10.3389/fcvm.2020.00039.
Sun M, Zhao D, Zhang Y, et al. Prognostic utility of monocyte to high-density lipoprotein ratio in patients with acute coronary syndrome: a meta-analysis. Am J Med Sci. 2020;359(5):281-286. doi: 10.1016/j.amjms.2020.01.018.
Deng Y, Zhou F, Li Q, et al. Associations between neutrophil-lymphocyte ratio and monocyte to high-density lipoprotein ratio with left atrial spontaneous echo contrast. BMC Cardiovasc Disord. 2023;23:234. doi: 10.1186/s12872-023-03270-3.
Bağcı A, Aksoy F. Systemic immune-inflammation index predicts new-onset atrial fibrillation after ST elevation myocardial infarction. Biomark Med. 2021;15(10):731-739. doi: 10.2217/bmm-2020-0838.
Karataş MB, Çanga Y, İpek G, et al. Association of admission serum laboratory parameters with new-onset atrial fibrillation after a primary percutaneous coronary intervention. Coron Artery Dis. 2016;27(2):128-134. doi: 10.1097/MCA.0000000000000333.
Tekkesin AI, Hayiroglu MI, Zehir R, et al. The use of monocyte to HDL ratio to predict postoperative atrial fibrillation after aortocoronary bypass graft surgery. North Clin Istanb. 2017;4(2):145-150. doi: 10.14744/nci.2017.53315.
Saskin H, Serhan Ozcan K, Yilmaz S. High preoperative monocyte count/high-density lipoprotein ratio is associated with postoperative atrial fibrillation and mortality in coronary artery bypass grafting. Interact Cardiovasc Thorac Surg. 2017;24(3):395-401. doi: 10.1093/icvts/ivw376.
Ulus T, Isgandarov K, Yilmaz AS, et al. Predictors of new-onset atrial fibrillation in elderly patients with acute coronary syndrome undergoing percutaneous coronary intervention. Aging Clin Exp Res. 2018;30(12):1475-1482. doi: 10.1007/s40520-018-0926-9.
Tanık VO, Tunca Ç, Kalkan K, et al. The monocyte-to-HDL-cholesterol ratio predicts new-onset atrial fibrillation in patients with acute STEMI. Biomark Med. 2025;19(4):121-128. doi: 10.1080/17520363.2025.2459590.
Lei Y, Hu L. The predictive value of monocyte count to high-density lipoprotein cholesterol ratio combined with left atrial diameter for post-radiofrequency ablation recurrence of paroxysmal atrial fibrillation. J Cardiothorac Surg. 2024;19:670. doi: 10.1186/s13019-024-03136-5.
Wang F, Sun Y, Lu Y, et al. Predictive value of platelet-to-albumin ratio combined with the C2HEST score for new-onset atrial fibrillation in elderly patients. BMC Cardiovasc Disord. 2024;24(1):521. doi: 10.1186/s12872-024-04200-7.
Zhang H, Zheng L, Yang Z, et al. Development and validation of prognostic models for bleeding and ischemia in elderly patients with comorbid acute coronary syndrome and atrial fibrillation. J Am Heart Assoc. 2024;13(20):e035086. doi: 10.1161/JAHA.124.035086.

Elliott AD, Middeldorp ME, Van Gelder IC, et al. Epidemiology and modifiable risk factors for atrial fibrillation. Nat Rev Cardiol. 2023;20(6):404-417. doi: 10.1038/s41569-022-00820-8.

He J, Yang Y, Zhang G, et al. Clinical risk factors for new-onset atrial fibrillation in acute myocardial infarction: a systematic review and meta-analysis. Medicine (Baltimore). 2019;98(26):e15960. doi: 10.1097/MD.0000000000015960.

Wu N, Li J, Xu X, et al. Prediction model of new onset atrial fibrillation in patients with acute coronary syndrome. Int J Clin Pract. 2023;2023:3473603. doi: 10.1155/2023/3473603.

Nagai M, Itoh T, Ishida M, et al. New-onset atrial fibrillation in patients with acute coronary syndrome may be associated with worse prognosis and future heart failure. J Cardiol. 2019;35(2):182-189. doi: 10.1002/joa3.12154.

Congo KH, Belo A, Carvalho J, et al. New-onset atrial fibrillation in st-segment elevation myocardial infarction: predictors and impact on therapy and mortality. Arq Bras Cardiol. 2019;113(5):948-957. doi: 10.5935/abc.20190190.

Sardu C, Barbieri M, Balestrieri ML, et al. Thrombus aspiration in hyperglycemic st-elevation myocardial infarction (STEMI) patients: clinical outcomes at 1-year follow-up. Cardiovasc Diabetol. 2018;17(1):152. doi: 10.1186/s12933-018-0795-8.

Arabi A, Abdelhamid A, Nasrallah D, et al. Monocyte-to-HDL ratio (MHR) as a novel biomarker: reference ranges and associations with inflammatory diseases and disease-specific mortality. Lipids Health Dis. 2025;24:343. doi: 10.1186/s12944-025-02755-8.