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Cardiovascular disease risk factors in newly diagnosed rheumatoid arthritis: A retrospective cohort study

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ABSTRACT

Background and objectives: Patients with rheumatoid arthritis (RA) face an increased risk of cardiovascular disease (CVD) that traditional risk factors alone cannot fully explain. Chronic inflammation may influence lipid profiles and contribute to this risk. This study evaluates predictors of incident CVD in RA and explores how erythrocyte sedimentation rate (ESR) modifies the relationship between lipid levels and CVD outcomes.

Design, settings, participants, and measurements: This retrospective cohort study included 1,802 RA patients aged 40-79 years, diagnosed between 2015 and 2022, and free of CVD at diagnosis. We evaluated the association between traditional cardiovascular risk factors—including current smoking, diabetes mellitus, systolic blood pressure, body mass index (BMI), HDL cholesterol, and LDL cholesterol—and RA-specific inflammatory markers, including ESR and C-reactive protein (CRP), with the incidence of CVD. Cox proportional hazards models adjusted for age, sex, race/ethnicity, antihypertensive medications, lipid-lowering medications, and antiplatelet medications.

Results: During a median follow-up of 3.5 years, 187 patients (10.4 %) developed CVD. The mean BMI was 32 kg/m^2 (standard deviation [SD] 10), HDL cholesterol was 53 mg/dL (SD 17), and LDL cholesterol was 104 mg/dL (SD 37). The median ESR was 21 mm/hr (interquartile range [IQR] 11–42) and CRP was 6 mg/L (IQR 3–12). Higher LDL cholesterol was inversely associated with CVD risk (HR 0.77 per SD increase, 95 % CI 0.63–0.94), with this association weakening with increasing ESR levels (interaction term HR 0.84, 95 % CI 0.71–0.99). Elevated HDL cholesterol also showed significantly decreased CVD risk (HR 0.82 per SD increase, 95 % CI 0.68–0.97). Smoking and diabetes were associated with increased risks (HR 1.52, 95 % CI 1.07–2.17 and HR 2.08, 95 % CI 1.39–3.10, respectively).

Conclusion: This study highlights the complex interplay between lipid levels and inflammation in RA, highlighting the nuances of CVD risk assessment in RA.

1. Introduction

Rheumatoid arthritis (RA) significantly increases the risk of cardiovascular disease (CVD), which is a significant contributor to mortality and morbidity in this patient population [1–3]. Traditional cardiovascular risk factors, such as dyslipidemia, do not fully account for the heightened cardiovascular risk observed in those with RA [4]. This discrepancy suggests that RA-related inflammatory processes may have a crucial role in cardiovascular disease pathogenesis beyond what is explained by conventional risk factors alone [5].

Although previous studies have explored the relationships between lipid levels, inflammatory markers, and cardiovascular outcomes in RA, the findings have been inconsistent [6–9]. This variability highlights the need for further investigation into how these factors may interact and lead to the increased CVD observed among those with RA. Further, current cardiovascular risk assessments are predominantly designed for the general population and thus may not adequately reflect the complex interplay of risk factors in RA, potentially leading to inadequate management and prevention strategies in this group [4]. In light of these inconsistencies and limitations, we conducted a retrospective cohort

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study in a diverse group of patients newly diagnosed with RA to examine the associations between traditional and RA-specific risk factors and the incidence of CVD. In addition, we explore the role of ESR in altering the association between lipid levels and CVD outcomes.

2. Methods

2.1. Study population

A retrospective cohort study was conducted at the University of Illinois Hospital and Health Sciences System (UI Health), which is defined by the United States Department of Education as a minority-serving institution [10]. All patients aged 40 to 79 years diagnosed with RA, as indicated by International Classification of Diseases (ICD-10) codes M05-M06, from October 1, 2015, to June 1, 2022, during an outpatient encounter were included. Only patients with at least 1 encounter in an outpatient rheumatology clinic during the study period were included. Patients with documented CVD prior to or within 30 days of the index date of RA diagnosis (initial ICD-10 diagnosis) were excluded to reduce the likelihood of reverse causality. Additionally, patients with a prior diagnosis of RA before or within 30 days of their first encounter within the UI Health system were excluded from the study. The study was approved by the University of Illinois Chicago Institutional Review Board (UIC IRB #2021-0311).

2.2. Exposures and covariables

The electronic medical record (EMR) was utilized to obtain the first recorded measurement of demographic and clinical characteristics following the index date of diagnosis: age, sex, race and/or ethnicity, smoking status, antihypertensive medications, lipid-lowering medications, antiplatelet medications, systolic blood pressure, body mass index, LDL cholesterol, HDL cholesterol, diabetes mellitus, erythrocyte sedimentation rate (ESR), and C-reactive protein. If a variable was not recorded after the index date, the most recent measurement before the index date was obtained. The absence of a smoking history was assumed if a patient's smoking status was not reported in the EMR. A diagnosis of diabetes mellitus was defined by either an ICD-10 diagnosis of diabetes mellitus (E08) or a hemoglobin A1c laboratory value greater than 6.4 %.

2.3. Outcomes

The primary endpoint included incident CVD. CVD was defined by ICD-10 codes for ischemic heart disease, myocardial infarction, stroke, heart failure, angina, peripheral artery disease, transient ischemic attack, abdominal aortic aneurysm, or history of carotid intervention (Supplemental Table 1). The EMR was also manually reviewed to verify death certificates. In a sensitivity analysis, we excluded heart failure from the composite outcome to evaluate for atherosclerotic CVD (ASCVD).

2.4. Statistical analyses

Characteristics of patients were summarized using the 'gtsummary' package in R with mean ± standard deviation (SD) or median (interquartile range) for continuous variables and percentages for categorical variables. Cox proportional hazard models were used to estimate the hazard ratios of the studied predictor variables, which included systolic blood pressure, smoking status, diabetes mellitus, body mass index, LDL cholesterol, HDL cholesterol, ESR, and C-reactive protein. We fit hierarchically adjusted models based on the biological and clinical plausibility of covariables as possible confounders: model 1 adjusted for age, sex, and race/ethnicity; model 2 further adjusted for antihypertensive medications, lipid-lowering medications, and antiplatelet medications). The 'mice' package (version 3.15.0) was employed to perform multiple imputations using predictive mean matching to handle missing covariate data. This process was carried out across ten imputed datasets and involved 100 iterations. The results from the imputed datasets were aggregated using Rubin's rules [11]. All analyses were performed using R (version 4.1.2), "Funny-Looking Kid."

To evaluate the presence of multicollinearity among the predictor variables, we utilized two approaches: correlation matrices and variance inflation factor (VIF) analysis. The correlation matrix revealed no pairwise correlations exceeding 0.7. Furthermore, the VIF values for all variables were less than 5, indicating minimal multicollinearity within our dataset.

3. Results

3.1. Baseline characteristics of the study population

During the study period, 3,382 patients were diagnosed with RA. Among these patients, 1,802 individuals aged 40 to 79 years were free of CVD within 30 days following their RA diagnosis and included in the final study cohort. As shown in Table 1, the mean age of patients at the time of RA diagnosis was 57 years, with a majority (79 %) being female. The racial/ethnic composition was predominantly non-Hispanic Black (41 %), followed by Hispanic (29 %), and non-Hispanic White (23 %). Notable clinical characteristics included 18 % smokers, a mean systolic blood pressure of 134 mmHg, average HDL cholesterol of 53 mg/dL, and LDL cholesterol of 104 mg/dL. Few patients were on common risk-modifying medications, including antihypertensive (8.5 %), lipid-lowering (3.4 %), and antiplatelet (5.3 %) agents.

3.2. Risks of incident CVD

Over an average follow-up of 3.5 years, 187 patients (10.4 %) developed incident CVD, with ischemic heart disease (34 %) and myocardial infarction (30 %) being the most common causes (Supplement 2). There was a total of 4 deaths (0.22 %) in the final cohort, two of which were attributed to cardiac causes. In fully adjusted Cox proportional hazards models (Table 2), current smoking (HR 1.52, 95 % CI 1.07–2.17) and diabetes mellitus (HR 2.08, 95 % CI 1.39–3.10) demonstrated significant associations with an increased risk of future CVD. Interestingly, increased levels of LDL cholesterol were associated with a reduced risk of cardiovascular events (HR 0.77 per SD, 95 % CI 0.63–0.94). Similarly, each SD increase in HDL cholesterol conferred a significant reduction in risk (HR 0.82, 95 % CI 0.68–0.97). Among RA-

Table 1
Baseline characteristics of rheumatoid arthritis cohort.

Characteristics	N = 1,802
Age, years	57 ± 9
Male	375 (21 %)
Race/Ethnicity	
Hispanic	523 (29 %)
Non-Hispanic White	423 (23 %)
Non-Hispanic Black	745 (41 %)
Other	111 (6.2 %)
Current smoking	332 (18 %)
Diabetes mellitus	149 (8.3 %)
Antihypertensive medication	153 (8.5 %)
Lipid-lowering medication	61 (3.4 %)
Antiplatelet medication	96 (5.3 %)
Systolic blood pressure, mmHg	134 ± 21
Body mass index, kg/m^2	32 ± 10
HDL cholesterol, mg/dL	53 ± 17
LDL cholesterol, mg/dL	104 ±37
Erythrocyte sedimentation rate, mm/hr	21 (11 – 42)
C-reactive protein, mg/L	6 (3 – 12)

eGFR=estimated glomerular filtration rate, HDL=high density lipoprotein, LDL=low density lipoprotein.
Data are presented as mean ± SD, median (interquartile range), or count with frequencies (%).

Table 2
Association of traditional and non-traditional risk markers with incident cardiovascular disease.

Predictors	Model 1[a]		Model 2[b]	
	Hazard Ratio (95 % Confidence Interval)	P Value	Hazard Ratio (95 % Confidence Interval)	P Value
Current smoking	1.44 (1.01 – 2.06)	0.041	1.52 (1.07 – 2.17)	0.020
Diabetes mellitus	2.34 (1.58 – 3.46)	<0.001	2.08 (1.39 – 3.10)	<0.001
Systolic blood pressure	1.04 (0.90 – 1.20)	0.63	1.00 (0.86 – 1.15)	0.97
Body mass index	1.04 (0.89 – 1.20)	0.65	1.01 (0.86 – 1.20)	0.89
HDL cholesterol	0.82 (0.68 – 0.99)	0.038	0.82 (0.68 – 0.97)	0.025
LDL cholesterol	0.75 (0.61 – 0.92)	0.005	0.77 (0.63 – 0.94)	0.009
Erythrocyte sedimentation rate	1.26 (1.08 – 1.48)	0.004	1.27 (1.06 – 1.51)	0.005
C-reactive protein	0.97 (0.83 – 1.14)	0.74	0.97 (0.84 – 1.14)	0.77

eGFR=estimated glomerular filtration rate, HDL=high density lipoprotein, LDL=low density lipoprotein.
[a] Model 1: adjusted for age, sex, and race/ethnicity.
[b] Model 2: model 1 and further adjusted for antihypertensive medication, lipid-lowering medication, and antiplatelet medication.

specific risk markers, an elevated ESR consistently showed an increased risk of the composite outcome (HR 1.27 per SD, 95 % CI 1.06–1.51). Other factors, including systolic blood pressure, body mass index, and C-reactive protein, did not exhibit significant relationships with future CVD. In a further analysis of ASCVD risk that excluded heart failure in the composite outcome, the magnitudes of association were qualitatively unchanged (Supplemental Table 3).

3.3. Interaction between ESR and lipid profile markers

Given the inverse relationship between LDL cholesterol levels and cardiovascular risk, further analyses explored whether inflammatory markers, specifically ESR, might influence the association between lipoprotein cholesterol and CVD outcomes. We first examined the correlations between ESR and lipid profiles and then assessed whether ESR modifies the effect of HDL or LDL cholesterol. In the cohort, ESR levels were inversely correlated with lipid markers. As shown in Supplemental Figures 1 and 2, Pearson's correlation analysis revealed inverse correlations between ESR and lipid markers, with ESR negatively associated with LDL cholesterol ($r = -0.12$; $p = 0.006$) and HDL cholesterol ($r = -0.09$; $p = 0.040$). Subsequent analyses using Cox proportional hazards models, which adjusted for all covariates included in model 2, revealed that the decreased CVD risk associated with higher LDL levels was significantly modified by ESR levels (HR 0.84, 95 % CI 0.71–0.99). Similar analyses were conducted to evaluate the interaction between HDL cholesterol and ESR; however, the interaction term (HR 0.95, 95 % CI 0.78–1.15) was not significant.

4. Discussion

In this diverse cohort of over 1,500 patients newly diagnosed with RA, we report both traditional and non-traditional predictors of CVD. Elevated LDL cholesterol was associated with a reduced risk of CVD events, whereas increased HDL cholesterol led to a significant reduction in CVD risk, reinforcing its role in predicting better cardiovascular outcomes even in the context of chronic inflammation. Separately, we noted that ESR was inversely correlated with both LDL and HDL cholesterol levels, suggesting distinct effects of inflammation on lipid profiles. Notably, this is the first study to evaluate how inflammatory markers, specifically ESR, modify the association between LDL cholesterol and CVD outcomes in RA patients. These findings underscore the importance of a tailored approach to cardiovascular risk assessment in RA, integrating both traditional and inflammation-specific factors to effectively predict and manage CVD risk.

Exploring the nuanced relationship between lipids and CVD in patients with RA adds crucial insight into disease management strategies, as prior findings have been inconsistent. For example, a cohort study of 651 patients with RA found no significant relationship between LDL cholesterol and heightened CVD risk. Similar to our findings, though, an inverse relationship was noted in those with serum levels ≤4 mmol/L (~155 mg/dL) [12]. Further complicating the association between LDL cholesterol and heart disease risk, a large cohort of over 40,000 patients with RA found no linear relationship between LDL cholesterol and CVD [7]. In one of the only studies to demonstrate a statistically significant association between increasing LDL cholesterol levels and CVD outcomes in RA, Liao et al. suggested that high LDL cholesterol levels are linked to an increased risk of cardiac events compared to low levels [8]. These conflicting results highlight the need for a more refined approach to understanding lipid profiles in the context of RA. In an attempt to better utilize lipid profiles to understand CVD risk, some research suggests that total cholesterol-to-HDL cholesterol ratios may better predict CVD risk in patients with rheumatoid arthritis [9]. Building on this body of knowledge, our study adds further complexity by demonstrating that higher LDL cholesterol is associated with a reduced CVD risk in a uniquely diverse cohort. Furthermore, we also evaluate the relationship between LDL cholesterol and future CVD events within the context of inflammatory markers, allowing us to understand how these markers may modify the association between lipid profiles and CVD outcomes. By examining the complex interactions between lipid profiles, inflammatory markers, and cardiovascular risk, our research provides nuanced insights that may help refine risk assessment and management strategies for patients with RA.

In addition to the inverse relationship between LDL cholesterol and incident CVD, other findings in our study support the lipid paradox in RA—a phenomenon where heightened inflammation is linked to lower serum lipid levels but increased risk of heart disease [12,13]. Our analysis reveals a significant linear relationship between ESR and future CVD events, underscoring its potential as a chronic inflammation marker closely linked with CVD outcomes. Interestingly, while C-reactive protein, a marker responsive to acute inflammatory changes, did not exhibit a similar association, it highlights the different roles that acute and chronic inflammation markers play in cardiovascular health [14].

Additionally, our findings highlight the complexity of lipid interactions in RA. We observed that higher levels of ESR moderate the inverse relationship between elevated LDL cholesterol and CVD. Our findings indicate that the association between LDL cholesterol and reduced CVD risk is lessened with heightened inflammation. The nuanced interaction between LDL cholesterol and inflammatory markers, particularly ESR, supports the need to consider lipid levels and inflammatory states when assessing cardiovascular risk in RA. For example, in patients with elevated LDL cholesterol but low ESR, the reduced inflammation might suggest a different cardiovascular risk profile than those with both high LDL cholesterol and high ESR, necessitating tailored management strategies reflecting this complex interplay. In support of our findings, other studies have shown that RA patients treated with immunomodulatory agents, such as anti-tumor necrosis factor therapies, frequently experience an increase in apolipoprotein B and LDL cholesterol levels but fewer cardiovascular events [15]. These observations underscore the limitations of traditional lipid measures for assessing cardiovascular risk in RA, emphasizing the need for a tailored approach that accounts for the unique inflammatory responses and lipid interactions in this patient population.

In our study, traditional risk factors such as systolic blood pressure

and body mass index did not significantly predict CVD outcomes in those with RA. This observation contrasts with established cardiovascular risk equations, where systolic blood pressure directly influences risk, and body mass index indirectly effects outcomes through associated metabolic factors [16]. This finding, however, could be attributed to index event bias, where individuals with lower systolic blood pressure, having less frequent healthcare interactions, may receive a later RA diagnosis when the disease is potentially more severe [17]. Similarly, body mass index did not emerge as a significant predictor in our RA cohort, which echoes prior research suggesting that the relationship between obesity and CVD risk in RA is complex and may be influenced by factors such as muscle wasting or different fat distribution patterns, a condition previously referred to as rheumatoid cachexia [18,19]. Unlike systolic blood pressure and body mass index, other studied risk factors such as diabetes mellitus and smoking did align with known predictors of CVD in the general population. Both smoking and diabetes mellitus were significantly associated with increased CVD risk, reaffirming their strong and well-documented detrimental effects on cardiovascular health [20,21]. These findings underscore the notion that RA modifies traditional CVD risk pathways, necessitating a disease-specific approach to cardiovascular risk assessment and management [22].

The present study has numerous strengths. It features a comprehensive definition of CVD that captures the full spectrum of cardiovascular-related morbidity and mortality. Additionally, potential confounding variables such as age, sex, race and/or ethnicity, and use of lipid-lowering, antihypertensive, and antiplatelet agents are considered. This study is unique in that it includes both men and women from a broad range of racial and ethnic backgrounds, setting it apart from previous research examining the relationship between risk factors associated with heart disease in patients with RA [6,7,9,12,23]. Although White patients do not have a higher disease burden or disease activity, studies have historically overrepresented White participants. For example, in a 2016 study of over 35,000 patients with RA that examined the association between lipids and cardiovascular outcomes, the cohort was predominately male (90 %) and White (71 %) [6]. In another cohort study of similar size, 76 % of patients were female, but race and ethnic demographics were not provided [7]. A meta-analysis of 126 US-based RA randomized controlled trials found that racial and ethnic minority groups only comprised 16 % of enrolled individuals [24].

Limitations of the study include its retrospective nature and data collection from an EMR. Additionally, the study did not capture all potential confounding variables, such as family history of CVD or use of disease-modifying agents, which may have impacted the observed associations. The lack of data on the use of immunomodulatory agents at baseline is a notable limitation, as these medications may affect lipid levels and inflammation, as well as the association between inflammation and cardiovascular outcomes [25]. The study was also conducted at a single institution, limiting generalizability to other patient populations or healthcare settings. Furthermore, data on seropositivity of RA, which can influence the risk of CVD, were not available [26].

In conclusion, our research highlights the crucial role of the interaction between lipid levels and inflammation in determining cardiovascular risk in RA patients. This is the first study to evaluate how inflammation modifies the association between LDL cholesterol and CVD outcomes in RA patients. Additionally, our study uniquely studies a diverse cohort of patients, including men and women from many racial and ethnic backgrounds. The inverse relationship between LDL cholesterol and CVD risk challenges a simplistic approach to risk assessment and lipid-lowering therapies. The absence of significant associations with systolic blood pressure and body mass index further supports the need for RA-specific cardiovascular risk models incorporating traditional and RA-specific inflammatory markers. Given RA's association with substantial cardiovascular morbidity, our findings underscore the need for personalized cardiovascular risk management strategies. Future studies should validate these observations in larger, diverse cohorts and investigate the underlying mechanisms linking inflammation, lipid metabolism, and CVD outcomes in RA to inform more effective prevention and treatment approaches.

Author contributions

A.G.A and N.T.N were responsible for the concept and design of the study. A.G.A was responsible for statistical analyses. All authors interpreted the data. Each author contributed important intellectual content during manuscript drafting or revision and agrees to be personally accountable for the individual's own contributions and to ensure that questions pertaining to the accuracy or integrity of any portion of the work, even one in which the author was not directly involved, are appropriately investigated and resolved, including with documentation in the literature if appropriate.

CRediT authorship contribution statement

Avi G. Aronov: Writing – original draft, Investigation, Formal analysis, Data curation. **Yoo Jin Kim:** Writing – original draft, Investigation, Formal analysis, Data curation. **Salman Zahid:** Writing – review & editing, Formal analysis. **Erin D. Michos:** Writing – review & editing. **Noreen T. Nazir:** Writing – review & editing, Supervision, Project administration, Investigation, Conceptualization.

Declaration of competing interest

The authors declare that they have no relevant financial interests.

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.ajpc.2025.101002.

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