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# Elevated Red Cell Distribution Width (RDW) Identifies Elevated Cardiovascular Disease Risk in Patients with HIV infection

**Sadeer G. Al-Kindi**[1], **Chang H Kim**[1], **Stephen R. Morris**[1], **Michael L. Freeman**[2], **Nicholas T. Funderburg**[3], **Benigno Rodriguez**[2], **Grace A. McComsey**[4], **Jarrod E. Dalton**[5], **Daniel I. Simon**[1], **Michael M. Lederman**[2], **Chris T. Longenecker**[1,*], and **David A. Zidar**[1,*]

[1]Harrington Heart & Vascular Institute, University Hospitals Case Medical Center, Cleveland, OH

[2]Center for AIDS Research, Division of Infectious Diseases and HIV Medicine, Department of Medicine, Case Western Reserve University/University Hospitals Case Medical Center, Cleveland OH

[3]School of Health and Rehabilitation Sciences, Ohio State University, Columbus, OH

[4]School of Medicine, Case Western Reserve University, Cleveland, OH

[5]Section of Health Outcomes Research and Clinical Epidemiology, Lerner Research Institute, Cleveland Clinic, Cleveland, OH

## Abstract

Red cell distribution width (RDW) is linked to cardiovascular risk in the general population, an association that might be driven by inflammation. Whether this relationship holds for patients with Human Immunodeficiency Virus (HIV) infection has not been previously studied. Using a large clinical registry, we show that elevated RDW (>14.5%) is independently associated with increased risk of coronary artery disease (odds ratio (OR) 1.39 [1.25–1.55]), peripheral vascular disease (OR 1.41 [1.29–1.53]), myocardial infarction (1.43 [1.25–1.63]), heart failure (OR 2.23 [1.99–2.49]), and atrial fibrillation (OR 1.96 [1.64–2.33]). In conclusion, in the context of the inflammatory milieu that accompanies HIV infection, RDW remains a powerful marker of CV disease.

### Keywords

Cardiovascular disease; coronary; peripheral; heart failure; RDW

## Introduction

Red cell distribution width (RDW) is a measure of the variability of red blood cell size, also known as anisocytosis. Over the past decade, RDW has also been associated with incident myocardial infarction[1,2] and heart failure[3] in the general population. RDW has also emerged as one of the strongest predictors of poor survival in patients with established heart failure[4–6] and coronary artery disease[7–10]. Anisiocytosis is typically a result of impaired iron

Address for Correspondence: David A. Zidar, MD, PhD; 11100 Euclid Avenue, Mailstop LKS 5038; Cleveland, OH 44106, (216) 286-6564 phone; (216) 844-8954 fax; David.Zidar@UHhospitals.org.

*Equal Contribution

metabolism[11] and tends to correlate with low hemoglobin, reduced mean corpuscular volume, high iron binding capacity, and elevated erythropoietin levels despite normal ferritin levels. RDW has also been proposed as marker of immune activation, correlating with levels of tumor necrosis factor (TNF)-α[12] and interleukin (IL)- 6[11]. The processes of inflammation and dysregulated hematopoiesis may be linked, since IL-6 is crucial for the production of hepcidin in the liver, and thus may indirectly regulate iron metabolism.

Antiretroviral therapy has improved life expectancy in patients living with HIV, but the burden cardiovascular disease in this population is increasingly recognized[13]. Patients with HIV have a high prevalence of silent and clinically apparent cardiovascular diseases[14–16]. We and others have shown that cardiovascular disease in patients with HIV seems to be associated with inflammation, including IL-6[17–21] elevations, but as intra-individual variability in IL-6 levels is high, the identification of “at risk” patients remains problematic. The RDW is routinely reported as part of automated blood count from clinical laboratories, and therefore could be an attractive pragmatic biomarker of immune activation and cardiovascular risk. We therefore sought to test whether RDW is associated with cardiovascular disease prevalence specifically in patients with HIV.

## Methods

### Data source

Explorys (Explorys, Inc; Cleveland, Ohio) is a commercial cloud-based database that aggregates data from electronic health records of participating hospital systems. It currently encompasses 23 integrated health systems consisting of 360 hospitals, 315,000 providers, and about 50 million unique patients. It collects data through a healthcare gateway server behind the firewall of participating institutions. The data are collected from billing inquiries, electronic health records, and laboratory systems. These data aggregates are then de-identified and standardized into Unified Medical Language Systems (UMLS) ontologies to facilitate searching and indexing[22]. Diagnoses are mapped into systematized nomenclature of medicine-clinical terms (SNOMED-CT) hierarchy, prescriptions mapped to RxNorm, and laboratory test observations mapped to logistical observation identifier names and codes (LOINC). Data collection started in 1999 and are updated every 24–48 hours. The platform is compliant with the Health Insurance Portability and Accountability Act (HIPAA) and Health Information Technology for Economic and Clinical Health (HITECH) Act standards; hence, its use is exempted from Institutional Review Board review under a pre-specified policy. The database rounds number of patients to the nearest 10 for an added data protection. This platform has been used for research purposes and has been validated in fields of oncology[23], orthopedics[24], gastroenterology[25], gynecology[26] among others.

### Cohort selection and definitions

We selected all patients who are at least 18 years of age with a diagnosis of “Human Immunodeficiency Virus Infection” who had at least one measurement of RDW after documentation of HIV infection. High RDW (>14.5%) and low/normal RDW (</=14.5%) were defined in accordance with previously used cut-offs[27,28]. Cardiovascular diseases were identified by their umbrella terms: “disorder of coronary artery”, “peripheral vascular

disease", "heart failure", "myocardial infarction" and "atrial fibrillation". We calculated the prevalence of cardiovascular diseases separately in patients (18–65 years old) with or without HIV using their most recent RDW. This analysis was also replicated in patients without anemia (hemoglobin >12 g/dL) to test the effect of RDW in non-anemic patients, and in Caucasians to evade the possible interaction between RDW and sickle-cell trait that is prevalent in African Americans.

We also selected a cohort with HIV without CVD and then followed for incident cardiovascular diseases (myocardial infarction or heart failure) stratified by the first RDW after documentation of HIV infection. This analysis was adjusted for traditional cardiovascular risk factors: age, gender, hypertension, diabetes, and dyslipidemia.

### Statistical analysis

The data are presented as numbers and percentages. Pearson's chi-squared test was used to test for differences. Logistic regression models were used to identify the adjusted odds ratio of cardiovascular events. Social Package for Social Studies (SPSS) version 19.0 was used for all analyses, with significance level set at $p<0.05$.

## Results

Of the 30,590,990 adults (18–65 years) in the database at the time of the inquiry, 79,590 (0.26%) had HIV infection and 46,720 had at least one documented RDW. Of those who were HIV-infected, 16,570 patients (35% of the total HIV population) had an elevated RDW. Patients with HIV and a high RDW had several important clinical and demographic differences compared to those with a low or normal RDW (Table 1). High RDW patients were slightly older, more likely to be female (39% vs 26%, $p<0.001$), African American (62% vs 42%, $p<0.001$), and to have diabetes (22% vs 13%, $p<0.001$), hypertension (48% vs 36%, $p<0.001$), and to be actively smoking (45% vs 40%, $p<0.001$), compared to individuals with low or normal RDW. HIV-infected patients with elevated RDW were also less likely to be treated with antiretroviral therapy, compared to HIV-infected patients with a low/normal RDW (50% vs. 53%, $p<0.001$). Differences in subclasses of ART use are displayed in Table 1.

In the HIV negative population, an elevated RDW was associated with a higher prevalence of cardiovascular diseases, compared to those with a low/normal RDW (Figure 1). For instance, among the uninfected, patients with a high RDW had higher prevalence of coronary artery disease (8% vs 3%, $p<0.001$) and heart failure (7% vs 1%, $p<0.001$), compared with those having a normal or low RDW. HIV was associated with a higher prevalence of cardiovascular disease compared to HIV uninfected subjects, regardless of RDW status. For example, among patients with high RDW, CAD (12% vs 8%, $p<0.001$) and HF (14% vs 7%, $p<0.001$) were more prevalent in HIV+ than uninfected controls. Patients with HIV were more likely to have an elevated RDW compared to the HIV uninfected (35% vs. 17%, $p<0.001$).

Patients with HIV infection and an elevated RDW had a higher prevalence of cardiovascular disease compared to HIV+ subjects with a low/normal RDW. For instance, HIV+ patients

with high RDW had higher prevalence of coronary artery disease (12% vs 6%, p<0.001), prior myocardial infarction (8% vs 4%, p<0.001), and peripheral vascular disease (19% vs 10%, p<0.001). Patients with HIV and an elevated RDW also had a higher prevalence of heart failure compared to low/normal RDW HIV+ patients (14% vs 4%, p<0.001). Atrial fibrillation also tended to be more common in patients with elevated RDW among patients living with HIV (5% vs 2%, p<0.001).

To determine whether anemia or hemoglobinopathies might be confounding the association between RDW and CVD, two sensitivity analyses were performed. In HIV+ patients without anemia, the relationships between RDW and CVD remained unchanged: CAD (high RDW 10% vs low/normal 6%, p<0.001), PVD (high RDW 16% vs low/normal 10%, p<0.001), HF (high RDW 9% vs low/normal 3%, p<0.001), MI (high RDW 7% vs low/normal 4%, p<0.001) and AF (high RDW 4% vs low/normal 2%, p<0.001). Similar findings persisted in the Caucasian subgroup: CAD (high RDW 11% vs low/normal 7%, p<0.001), PVD (high RDW 18% vs low/normal 10%, p<0.001), HF (high RDW 11% vs low/normal 3%, p<0.001), MI (high RDW 9% vs low/normal 4%, p<0.001) and AF (high RDW 5% vs low/normal 2%, p<0.001).

An elevated RDW in HIV patients without CVD was associated with the development of future disease, even after adjustment for age, gender, hypertension, diabetes, and dyslipidemia. Compared to low/normal RDW HIV+ patients, those HIV+ patients with an elevated RDW had increased odds of incident coronary artery disease (unadjusted OR [95% CI] 1.42 [1.29–1.56], p<0.001); adjusted OR 1.39 [1.25–1.55], p<0.001), peripheral vascular disease (unadjusted OR 1.49 [1.38–1.61], p<0.001; adjusted OR 1.41 [1.29–1.53], p<0.001), myocardial infarction (unadjusted OR 1.43 [1.26–1.62], p<0.001; adjusted 1.43 [1.25–1.63], p<0.001), heart failure (unadjusted OR 2.39 [2.16–2.65], p<0.001; adjusted OR 2.23 [1.99–2.49], p<0.001), and atrial fibrillation (unadjusted OR 2.04 [1.72–2.41], p<0.001; adjusted OR 1.96 [1.64–2.33], p<0.001).

## Discussion

In this large electronic medical record cohort, we find that irrespective of HIV status, an elevated RDW is associated with a higher burden of atherosclerotic cardiovascular disease, heart failure, and atrial fibrillation. Thus, despite differences in the inflammatory milieu and medication exposures between those with and without HIV, the mechanisms that link RDW and cardiovascular disease appear to remain relevant. Given the availability of RDW during routine clinical practice and its strong relationship to CVD, these findings suggest additional study of this parameter as a possible immune and cardiovascular biomarker in HIV are warranted.

Several studies have described the association between RDW and cardiovascular disease in the general population. Felker et al[29] were the first to demonstrate the association of RDW and heart failure outcomes in the Candesartan in Heart failure Assessment of Reduction in Mortality and morbidity (CHARM) program (n=2,679) and Duke Database (n=2,140). This association was independent of other laboratory and clinical predictors including age and was more powerful than functional class or ejection fraction. Other similar studies have

confirmed this relationship[30]. In a population-based analysis of 25,612 persons in Norway, there was a linear association between baseline RDW and risk of myocardial infarction, with 13% increase in MI risk for every 1% increment in RDW[31], which persisted after exclusion of patients with anemia[32]. Similarly, studies have associated RDW with the presence of coronary artery disease[33], peripheral vascular disease[34], and atrial fibrillation[35]. We are unaware of any prior report describing the relationship between RDW and cardiovascular disease in the context of HIV infection. Thus, our study suggests that the relationship between RDW and CVD holds in HIV infection. This is particularly interesting as both immunologic and erythropoetic perturbations are characteristic of HIV disease and its treatment.

There are a number of factors that may explain the RDW-CV disease association. Increased RDW can be observed in states of ineffective red blood cell production, including vitamin deficiency, malnutrition states, and anemia of chronic disease[36]. How such bone marrow dysfunction might promote cardiovascular disease is uncertain. Several groups have ascribed the relationship between RDW and cardiovascular disease to subclinical immune activation, which has been previously linked to cardiovascular disease. For example, RDW has been associated with elevated levels of C-reactive protein, tumor necrosis factor alpha[12], malondialdehyde[12], and interleukin-6[37], all of which have been associated with CV disease. Inflammatory cytokines can worsen red blood cell survival, lead to erythropoietin resistance, and stimulate the production of hepcidin. Each of these mechanisms may impair red blood cell production and increase RDW.

A previous study by Gallegio et al did not find an association between RDW and the aggregate of traditional cardiovascular risk factors (Framingham risk score) in patients with HIV[38]. In our current study, an elevated RDW was associated with modest differences in some, but not all, cardiovascular risk factors. We therefore sought to examine whether the association between RDW and CVD was linked to traditional risk factors. After adjustment for age, gender, dyslipidemia, diabetes, and hypertension, an elevated RDW independently predicts cardiovascular events. Thus, RDW may have added value to improve cardiovascular risk assessment in addition to traditional risk factors in patients with HIV. Future studies are needed to explore the extent to which RDW correlates with immune activation, which has also been linked to cardiovascular disease in HIV, and whether RDW might also be a marker of immunologic outcomes in HIV.

This large analysis is limited by its retrospective design, lack of patient-level data, and the potential variability of RDW measurements across different hospital systems. We were unable to explore in any detail the mechanisms of the observed associations. Because of these limitations, this study must be interpreted with caution as hypothesis generating. Nevertheless, the substantial risk differences and large sample size provide a broad observational overview of these relationships in a real-world population.

## Conclusion

In patients with HIV, an elevated RDW is associated with a higher risk of cardiovascular disease. The connections between bone marrow function, immune activation, and

thrombosis should be examined further. If validated prospectively, the RDW may be a convenient, pragmatic biomarker of risk in addition to that conferred by HIV and traditional risk factors alone.

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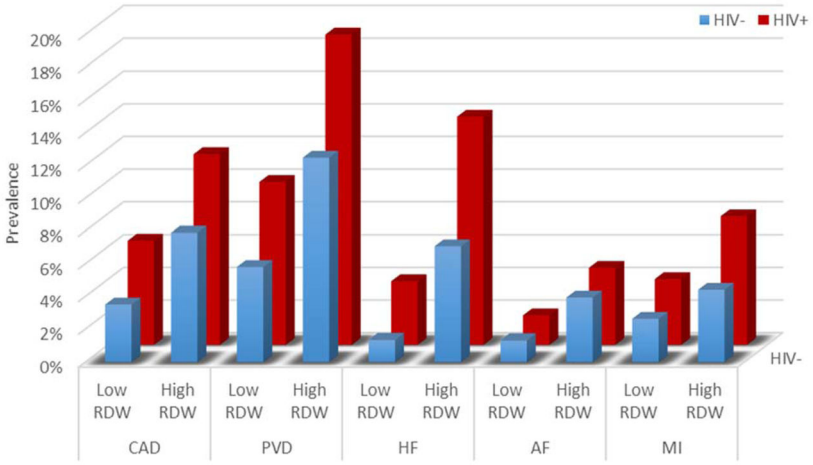


**Figure 1.**

Prevalence of CVD in patients with or without HIV stratified by RDW category. P-value <0.001 for all comparisons between high vs. low RDW categories for both HIV+ and HIV− patients.

## Table 1

Patient characteristics stratified by HIV status and RDW category. P-value <0.001 for all comparisons between RDW categories.

| | HIV+ | | HIV− | |
|---|---|---|---|---|
| | **Low RDW** | **High RDW** | **Low RDW** | **High RDW** |
| Number of patients | 30,150 | 16,570 | 7,969,250 | 1,617,900 |
| **Age (years)** | | | | |
| 20–24 | 800 (3%) | 270 (2%) | 598,560 (8%) | 78,630 (5%) |
| 25–29 | 2,140 (7%) | 730 (4%) | 802,550 (10%) | 118,250 (7%) |
| 30–34 | 2,570 (9%) | 1,050 (6%) | 873,500 (11%) | 141,010 (9%) |
| 35–39 | 3,100 (10%) | 1500 (9%) | 864,190 (11%) | 153,850 (10%) |
| 40–44 | 3,390 (11%) | 1,990 (12%) | 810,060 (10%) | 159,670 (10%) |
| 45–49 | 4,760 (16%) | 2,830 (17%) | 865,350 (11%) | 184,790 (11%) |
| 50–54 | 5,720 (19%) | 3,200 (19%) | 925,350 (12%) | 213,800 (13%) |
| 55–59 | 4,340 (14%) | 2,740 (17%) | 984,120 (12%) | 242,890 (15%) |
| 60–64 | 2,860 (9%) | 1,930 (12%) | 910,420 (11%) | 255,090 (16%) |
| **Gender** | | | | |
| Male | 22,320 (74%) | 10,110 (61%) | 3,419,430 (43%) | 496,580 (31%) |
| Female | 7,830 (26%) | 6,450 (39%) | 4,547,640 (57%) | 1,120,910 (69%) |
| **Race** | | | | |
| African American | 12,740 (42%) | 10,200 (62%) | 911,050 (11%) | 427,450 (26%) |
| Caucasian | 14,450 (48%) | 5,090 (31%) | 5,859,390 (74%) | 966,950 (60%) |
| **Insurance** | | | | |
| Private | 13,670 (45%) | 5,750 (35%) | 4,516,050 (57%) | 811,620 (50%) |
| Medicaid | 7,420 (25%) | 5,010 (30%) | 910,970 (11%) | 279,160 (17%) |
| Medicare | 5,750 (19%) | 4,130 (25%) | 503,640 (6%) | 199,900 (12%) |
| **Antiretroviral therapy** | | | | |
| Any ART | 15,990 (53%) | 8,280 (50%) | N/A | N/A |
| Protease Inhibitor | 8,600 (29%) | 5,530 (33%) | N/A | N/A |
| Integrase Inhibitor | 2,460 (8%) | 1,550 (9%) | N/A | N/A |
| Reverse Transcriptase Inhibitor | 15,390 (51%) | 7,900 (48%) | N/A | N/A |
| Tenofovir | 12,600 (42%) | 5,700 (34%) | N/A | N/A |
| Thymidine analogue (Stavudine/Zidovudine) | 2,600 (9%) | 2,080 (13%) | N/A | N/A |
| **Risk factors** | | | | |
| Diabetes | 4,030 (13%) | 3,630 (22%) | 684,700 (9%) | 288,620 (18%) |
| Hypertension | 10,740 (36%) | 7,990 (48%) | 1,853,780 (23%) | 603,720 (37%) |
| Dyslipidemia | 9,180 (30%) | 4,540 (27%) | 1,747,490 (22%) | 413,160 (26%) |
| Smoking | 11,970 (40%) | 7,380 (45%) | 1,383,800 (17%) | 361,270 (22%) |