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Comparison of Globorisk, SCORE2, and PREVENT in the Stratification of Cardiovascular Risk and its Relationship with End-Organ Damage Among Adults With Arterial Hypertension

Silvia Palomo-Piñón[1,2] | Luis Alcocer[1,3] | Humberto Álvarez-López[1,4] | Ernesto G. Cardona-Muñoz[1,5] | Adolfo Chávez-Mendoza[1,6] | Enrique Díaz-Díaz[1,6] | José Manuel Enciso-Muñoz[1,7,8] | Héctor Galván-Oseguera[1,6] | Martin Rosas-Peralta[1,9] | Luis Rey García-Cortés[1,10] | Moisés Moreno-Noguez[1,11] | Neftali Eduardo Antonio-Villa[1,12,13] | On behalf of the Mexican Group of Experts on Arterial Hypertension

[1]Grupo de Expertos en Hipertensión Arterial México (GREHTA), Ciudad de México, Mexico | [2]Grupo Colaborativo en Hipertensión Arterial (GCHTA), Ciudad de México, Mexico | [3]Hospital Ángeles del Pedregal, Ciudad de México, Mexico | [4]Hospital Puerta de Hierro Andares, Zapopan, Jalisco, Mexico | [5]Centro Universitario de Ciencias de la Salud, Universidad de Guadalajara, Guadalajara, Jalisco, Mexico | [6]Unidad Médica de Alta Especialidad de Cardiología, Centro Médico Nacional SXXI, Instituto Mexicano del Seguro Social, Ciudad de México, Mexico | [7]Coordinación de Cardiología, Hospital San Agustín, Zacatecas, Zacatecas, Mexico | [8]Asociación Nacional de Cardiólogos de México, Ciudad de México, Mexico | [9]Titular Academia Nacional de Medicina, Ciudad de México, Mexico | [10]Coordinación de Planeación y Enlace Institucional, Jefatura de Servicios de Prestaciones Médicas, Órgano de Operación Administrativa Desconcentrada Regional Estado de México Oriente, Instituto Mexicano del Seguro Social, Estado de México, Oriente, Mexico | [11]Coordinación Clínica de Educación e Investigación en Salud, Unidad de Medicina Familiar No. 55 Zumpango, Órgano de Operación Administrativa Desconcentrada Regional Estado de México Oriente, Estado de México, Mexico | [12]Departamento de Endocrinología, Instituto Nacional de Cardiología Ignacio Chávez, Ciudad de México, Mexico | [13]Department of Global Health and Population, Harvard T.H. Chan School of Public Health, Boston, USA

Correspondence: Silvia Palomo-Piñón (silvia-palomo@hotmail.com)

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ABSTRACT

Arterial hypertension often coexists with comorbidities that increase vascular damage. Although the primary goal is to reduce cardiovascular risk, the available risk scores can produce varying estimates. Here, we aim to compare the prevalence of cardiovascular risk categories using three equations (Globorisk, SCORE2, and PREVENT) in adults living with arterial hypertension and to assess their association as stratification tools for end-organ damage (EOD). To achieve this, we performed a cross-sectional sub-analysis of the RIHTA study, an electronic health record-based registry of adults with arterial hypertension in Mexican primary care centers. EOD was defined as aortic stiffness, reduced eGFR, hypertensive retinopathy, peripheral artery disease, or left ventricular hypertrophy. Inverse probability weighting (IPW) was used to evaluate the association between cardiovascular risk and EOD, adjusting for relevant confounders, and areas under the receiver operator curve (AUROC) were

Abbreviations: AUROC, area under the receiver-operating characteristic (curve); BMI, body mass index; CKD, chronic kidney disease; CVD, cardiovascular disease; DAGs, direct acyclic graphs; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; EOD, end-organ damage; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure.

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Neftali Eduardo Antonio-Villa is the last author.

calculated to assess detection capacity. Among 4512 participants (median age 64 years; 61% women), EOD was present in 33% (n = 1492). The PREVENT equation yielded the highest median 10-year risk (15%, IQR 8–24), followed by Globorisk laboratory-based (12%, 7–22), Globorisk office-based (11%, 7–19), and SCORE2 (5.06%, 3.86–7.18). In IPW models, each 1% increase in score was associated with higher odds of EOD (PREVENT OR 1.16, 95% CI 1.15–1.17; Globorisk-office 1.09, 1.08–1.10; Globorisk-lab 1.07, 1.06–1.08; SCORE2 1.04, 1.02–1.06). The PREVENT score demonstrated the strongest discrimination for detecting EOD (AUROC: 0.751, 0.735–0.750). These findings suggest that among adults with arterial hypertension, the PREVENT score identifies high-risk individuals and improves discrimination for EOD.

1 | Introduction

Arterial hypertension has consistently been recognized as the leading cardiovascular risk factor in several epidemiological and clinical studies [1–3]. However, due to its asymptomatic nature and inadequate detection programs in primary care, it often goes undiagnosed until significant vascular damage has already occurred [4, 5]. Consequently, the burden of cardiovascular mortality increases in low and middle-income countries (LMICs) [6]. Thus, effective cardiovascular risk stratification using validated risk equations is essential for guiding treatment decisions, especially for patients identified as being at intermediate risk.

Despite the clinical significance of cardiovascular risk stratification among patients living with arterial hypertension, most equations were initially developed and validated in Caucasian populations without considering the specific characteristics and epidemiological profiles of other ethnic groups [7, 8]. This issue is particularly pertinent given that the National Health and Nutrition Survey (*Spanish acronym—ENSANUT: Encuesta Nacional de Salud y Nutrición*) in Mexico reported that nearly half of the adult population suffers from arterial hypertension [9]. Furthermore, a previous report from the Registry of Arterial Hypertension in Mexico (*Spanish acronym—RIHTA: Registro de Hipertensión Arterial en México*) indicated that hypertensive patients often experience multiple concurrent cardiovascular conditions including overweight/obesity, dyslipidemia, type 2 diabetes mellitus and chronic kidney disease [10]. Although some guidelines have adapted these tools for the Mexican population, uncertainty remains about which cardiovascular risk equation most accurately stratifies this risk in patients with arterial hypertension [11, 12]. Although risk equations are used for incident cardiovascular events, in real-world clinical practice, clinicians also use them to identify patients who already had sub-clinical vascular damage and therefore, make intensified follow-up. Previous cross-sectional works has shown that higher predicted SCORE2 correlates with subclinical coronary atherosclerosis and arterial stiffness even in the absence of follow-up [13, 14].

In 2023, the American Heart Association (AHA) reframed the concept of Cardio-Renal-Metabolic Syndrome (CRMS), emphasizing the pathophysiological interplay between metabolic abnormalities, chronic kidney disease (CKD), and cardiovascular dysfunction [15]. This integrated perspective highlights the need for comprehensive cardiovascular risk algorithms that incorporate diverse metrics to assess metabolic and renal factors. Nevertheless, direct comparisons of various cardiovascular risk assessment tools in our population and their capacity as stratification tools for relevant hypertension-related complications, such as end organ damage (EOD), have not yet been conducted.

Here, we aim to (1) estimate the prevalence of cardiovascular risk categories using three previously validated risk scores (Globorisk, SCORE2, and PREVENT), and (2) assess their association with EOD among patients living with arterial hypertension recruited for the RIHTA study.

2 | Methods

2.1 | Study Design and Participants

This study is a cross-sectional sub-analysis of the RIHTA study, which included patients registered between December 2021 and April 2023. The rationale, complete methodology, and initial findings of the RIHTA study have been published elsewhere [10]. Briefly, the RIHTA study was designed as a prospective electronic-based registry aimed at providing comprehensive insights into the clinical profile and treatment of arterial hypertension in Mexico. Primary care physicians affiliated with the Mexican Institute of Social Security across 26 states in Mexico were invited to participate. Registered physicians or their assistants entered data online using an electronic platform during each office visit to supplement the patient's medical records. During the clinical consultation, blood pressure measurements were taken using a standardized protocol given to all physicians. This protocol consisted of taking three separate readings after the patient had rested for 5 min in the quietest environment possible. Patients were verbally asked to avoid drinking caffeinated beverages and smoking for 24 h prior to their consultation. For this analysis, the final two readings were averaged. We provided a calibrated digital sphygmomanometer (OMRON Healthcare EMC Model HEM-9200T) to all clinicians in RIHTA for the BP measurements. All patients met the definition of arterial hypertension based on either of the following criteria: (1) systolic and/or diastolic blood pressure readings ≥140/90 mm Hg or (2) a prior clinical diagnosis of arterial hypertension with current antihypertensive treatment [16]. Participants with incomplete data necessary to estimate CVD risk were excluded from this sub-analysis. Additionally, individuals with self-reported history of myocardial infarction and stroke were also excluded. The RIHTA study was approved by the Medical Ethics Committee of the Mexican Institute of Social Security, and all patients provided verbal informed consent prior to registration. The ethics committee waived written consent because RIHTA collects routine clinical data and poses minimal risk to the patient's enrollment. Since this sub-analysis used previously collected data from the RIHTA study, additional ethics committee approval was waived. This study adheres to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for cross-sectional studies (Table S1) [17].

2.2 | Outcomes Definitions

2.2.1 | Our Analyses Focused on Two Primary Outcomes

a) *CVD risk categories*: CVD risk was estimated using three validated scores: the Globorisk office- and biochemical-based equations, the SCORE2 equation, and the PREVENT risk score (total CVD equation) [18–20]. A brief description of the components used to estimate each score, the population of origin, as well as strengths and limitation of these indexes are presented in Table S2. Results were estimated at 10-years predicted risk and standardized into three categories to facilitate comparisons across these scores: Low-to-Moderate, Intermediate, and High-to-Very High. Globorisk classifies Low-to-Moderate as a 10-year risk of less than 10%, Intermediate Risk as 10% to less than 20%, and High to Very High Risk as 20% or higher [18]. SCORE2 incorporates age-specific thresholds [20, 21]: for individuals under 50 years, Low to Moderate Risk is defined as a risk of less than 2.5%, Intermediate Risk as 2.5% to less than 7.5%, and High to Very High Risk as 7.5% or higher. For individuals aged 50–69, these thresholds are adjusted, with Low to Moderate Risk defined as a risk of less than 5%, Intermediate Risk as 5% to less than 10%, and High to Very High Risk as 10% or higher. PREVENT uses simplified thresholds, defining Low to Moderate Risk as a risk of less than 5%, Intermediate Risk as 5% to less than 20%, and High to Very High Risk as 20% or higher [19]. We defined ≥20% as high-to-very-high because the PREVENT developers propose 5%–20% as intermediate and ≥20% as high for total CVD; nevertheless, US guidelines treating ASCVD ≥10% as actionable were explored in a sensitivity analysis. Details of the variables used in each scoring system and their specific definitions are provided in Table S3.

b) *End-organ damage (EOD)*: For this outcome, we used Roland E. Schmieder's proposed definition, which was further adapted into Mexican clinical guidelines [22, 23]. An EOD event was defined as a participant with any of the following conditions: elevated albumin excretion, decreased estimated glomerular filtration rate (eGFR), aortic stiffness, hypertensive retinopathy, peripheral artery disease, or left ventricular hypertrophy. Table S4 provides the specific diagnostic criteria for each endpoint.

2.3 | Confounder Assessment

For this sub-analysis, we extracted key variables from the RIHTA study and categorized them into sociodemographic, clinical, anthropometric, and biochemical groups. The sociodemographic variables included age (grouped as <45, 45–65, and ≥65 years), years of education (classified as no education, elementary, high school, college, or higher), and ethnic group. Clinical variables encompassed diabetes (defined as fasting glucose ≥126 mg/dL or self-reported prior diagnosis), self-reported chronic kidney disease (CKD), smoking status, alcohol use, and exercise duration (categorized as no exercise, <150 min, and ≥150 min). Anthropometric variables included body mass index (BMI), waist circumference, heart rate, as well as systolic and diastolic blood pressure. Expanded methods for blood pressure assessment are detailed in Supporting Information. Biochemical variables recorded from the 6 months before the last clinical visit included fasting glucose, glycated hemoglobin (HbA1c), fasting triglycerides, total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL-C), and serum creatinine.

2.4 | Statistical Analyses

Continuous variables were presented in medians and interquartile range. Categorical variables were presented as frequencies and absolute proportions. To compare characteristics between men and women, we performed a chi-squared test for categorical variables and a Mann–Whitney test for continuous variables. For handling missing data, we assumed that it was completely at random when addressing missing biochemical values. Detailed results of imputed variables can be found in Figure S1. All statistical analyses were conducted in R Studio (Version 4.1.2), with the code publicly available in a GitHub repository accessible at https://github.com/neftalivilla/RIHTA_CVD_SCORES. Expanded statistical methods are included in Supporting Information. A p value < 0.05 was used as our statistically significant threshold.

2.5 | Prevalence Estimates

We used the Clopper–Pearson method to estimate the prevalence of CVD risk categories. This method was chosen because it is regarded as a straightforward and reliable way of estimating confidence intervals in binomial distributions, irrespective of sample size [24, 25]. We stratified the prevalence of CVD risk categories by sex and control of arterial hypertension (defined as a BP <130/80 mm Hg).

2.6 | Association and Diagnostic Capacity of CVD Risk Equations With EOD

We first identified the minimal set of confounders necessary to block non-causal pathways in assessing the association between CVD risk scores and EOD. For this purpose, we created directed acyclic graphs (DAGs) based on recommendations by Digitale et al. [26]. Based on the DAGs shown in Figure S2, we estimated odds ratios (OR) using a binomial logistic regression model adjusted by an inverse probability-weighted score (IPW) for the following covariates: age, sex, smoking status, cumulative self-reported chronic comorbidities, arterial hypertension control, and self-reported use of ACEIs, ARBs, and statins. A Hosmer–Lemeshow test was performed to evaluate the model's calibration. We assessed the goodness of fit using the Bayesian Information Criteria (BIC); a lower BIC indicates a better fit. The variance explained by each model was evaluated using the Nagelkerke R^2; a higher Nagelkerke R^2 imply a better explanation of variance. Finally, to assess the diagnostic capacity of each CVD risk score to detect EOD, we estimated the area under the receiver operating characteristic curves (AUROC). Comparisons among AUROCs were carried out using the method proposed by Venkatraman et al. [27].

2.7 | Sensitivity Analyses

We performed several sensitivity analyses to confirm the robustness of our results. We repeated the unadjusted and adjusted binomial logistic models and AUROC calculations under four alternative scenarios: (1) restricting the sample to adults aged 40–70 years without diabetes, albuminuria, or chronic kidney disease to ensure equal applicability of all three scores; (2) removing albuminuria and reduced eGFR from both the predictor set and the EOD definition, excluding affected individuals to avoid circular reasoning; (3) excluding participants aged ≥60 years with pulse pressure ≥60 mm Hg because this surrogate for aortic stiffness is valid mainly in the elderly; and (4) excluding anyone with self-reported hypertensive retinopathy, peripheral artery disease, or left-ventricular hypertrophy to minimize outcome misclassification.

3 | Results

A total of 5590 participants were initially included in the RIHTA registry. Of these, 702 were excluded due to incomplete data necessary to estimate CVD risk, and 376 were removed based on history of myocardial infarction ($n = 315$) or stroke ($n = 61$). Thus, data from 4512 participants were analyzed (Figure 1). In Table 1, we summarize the descriptive characteristics of the study sample stratified by sex. Overall, the median age was 64 years (IQR: 55–72), with women representing 61% (n = 2734) of the sample. The most common comorbidity was diabetes (38%, n = 1716), while 16% (n = 743) had a history of smoking. More than half of the participants (54%, n = 2439) reported no exercise, and only 4.7% (n = 211) met the recommended physical activity of ≥150 min/week. In terms of anthropometric profile, the median BMI was 28.8 kg/m^2 (IQR: 25.9–32.2), with a median waist circumference of 95 cm (IQR: 87–101), and median SBP and DBP of 130 mm Hg (IQR: 121–139) and 80 mm Hg (IQR: 75–88), respectively. Regarding biochemical characteristics, the median glucose level was 102 mg/dL (IQR: 92–126), triglycerides were 153 mg/dL (IQR: 118–206), total cholesterol was 178 mg/dL (IQR: 154–200), and LDL-C was 106 mg/dL (IQR: 80–129). Concerning EOD, 33% ($n = 1492$) had at least one EOD component, with aortic stiffness (52%, $n = 776$) and decreased eGFR (51%, $n = 761$) being the most frequent. When comparing the characteristics by sex, men tended to be older (65 vs. 63 years, $p < 0.001$), had a higher proportion with more than 7 years of education (66% vs. 55%, $p < 0.001$), were more frequently ever-smokers (26% vs. 10%, $p < 0.001$), and had a higher prevalence of CKD (4.3% vs. 3%, $p = 0.029$). Conversely, women had a higher prevalence of diabetes (40% vs. 35%, $p = 0.005$), a slightly higher median BMI (29.3 kg/m^2 vs. 28.3 kg/m^2, $p < 0.001$), and total cholesterol (181 mg/dL vs. 174 mg/dL, $p < 0.001$). Regarding EOD, men tended to have a higher prevalence of EOD (35% vs. 32%, $p = 0.006$) with a higher proportion of decreased eGFR (55% vs. 48%, $p < 0.004$) compared to women

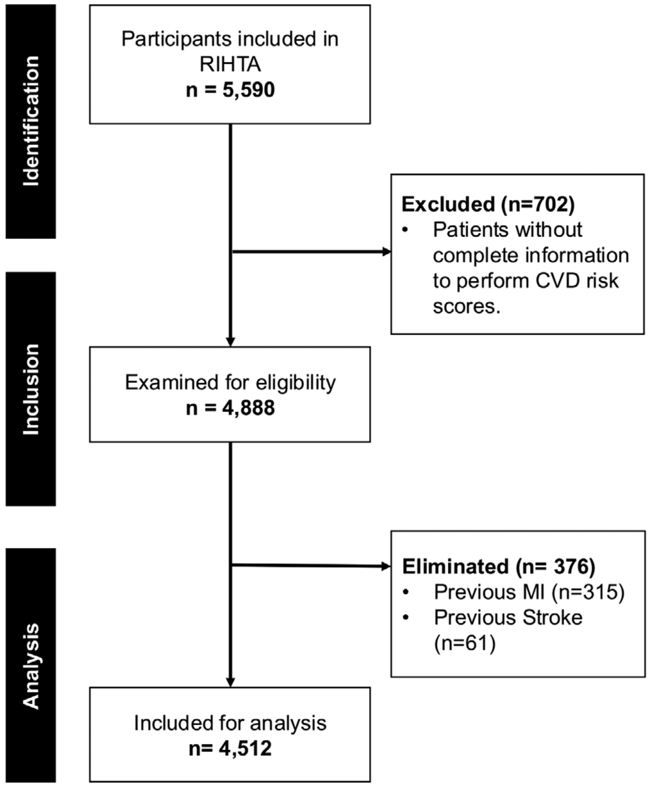

FIGURE 1 | STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) flowchart of our studied population.

3.1 | Prevalence of CVD Risk Categories

The PREVENT score had the highest median predicted 10-year cardiovascular risk (15%, IQR 8–24), followed by the Globorisk laboratory-based score (12%, 7–22), the office-based score (11%, 7–19) and the SCORE2 score (5.06%, 3.86–7.18). Overall, men displayed higher median values for the PREVENT (17 vs. 13, $p < 0.001$) and SCORE2 (7.71 vs. 4,37, $p < 0.001$) scores, whereas women showed slightly higher scores on the Globorisk laboratory-based (13 vs. 11, $p < 0.001$) and office-based (12 vs. 11, $p < 0.001$) assessments. When analyzing CVD risk categories, 20% (n = 903) of participants were categorized as Low-to-Moderate, 39.9% ($n = 1802$) as Intermediate, and 40.1% ($n = 1807$) as High-to-Very High according to the office-based Globorisk. In contrast, the laboratory-based Globorisk classified 40.9% ($n = 1848$) as Low-to-Moderate, 30.8% ($n = 1391$) as Intermediate, and 28.2% ($n = 1273$) as High-to-Very High. According to SCORE2, 43.8% (n = 1976) of the sample were Low-to-Moderate, 48.0% (n = 2165) were Intermediate, and 8.2% ($n = 371$) were High-to-Very High. Finally, the PREVENT score placed nearly one-quarter of participants (25%, n = 1145) in the Low-to-Moderate group, 39.5% (n = 1784) as Intermediate, and 35.1% (n = 1583) as High-to-Very High (Table S5). Stratifying the CVD risk by sex indicated that women had a higher prevalence of High-to-Very High risk using the Globorisk office-based equation (41.6% vs. 37.7%, $p < 0.001$), whereas men had a higher proportion in high-risk categories with both the PREVENT (41.3% vs. 31%, $p < 0.001$) and SCORE2 (20.5% vs. 0.2%, $p < 0.001$) scores (Figure 2). Stratification by <130/80 mm Hg BP goal did not substantially alter these distributions in the total sample (Table S6) or when evaluated by sex (Figure 3).

TABLE 1 | Descriptive characteristics of the studied sample stratified by sex.

Characteristics	Overall (n = 4512)	Women (n = 2734)	Men (n = 1778)	p value
Sociodemographic				
Age (years)	64 (55, 72)	63 (55, 71)	65 (55, 72)	0.6
Groups of age (%)				<0.001
<45	371 (8.2%)	175 (6.4%)	196 (11%)	
45–65	1988 (44%)	1300 (48%)	688 (39%)	
	2153 (48%)	1259 (46%)	894 (50%)	
Education in years (%)				<0.001
0 years	173 (3.8%)	118 (4.3%)	55 (3.1%)	
1–6 years	1652 (37%)	1102 (40%)	550 (31%)	
7–12 years	1999 (44%)	1130 (41%)	869 (49%)	
	688 (15%)	384 (14%)	304 (17%)	
Ethnic origin (%)				>0.9
Mestizo	4379 (97%)	2653 (97%)	1726 (97%)	
Other	133 (2.9%)	81 (3.0%)	52 (2.9%)	
Clinical				
Diabetes (%)	1716 (38%)	1088 (40%)	628 (35%)	0.005
CKD (%)	157 (3.5%)	81 (3.0%)	76 (4.3%)	0.029
Previous smoking (%)	743 (16%)	285 (10%)	458 (26%)	<0.001
Alcoholism (%)	14 (0.3%)	2 (<0.1%)	12 (0.7%)	<0.001
Time of exercise (%)				<0.001
No-exercise	2439 (54%)	1572 (58%)	867 (49%)	
<150 min	1860 (41%)	1046 (38%)	814 (46%)	
	211 (4.7%)	114 (4.2%)	97 (5.5%)	
Anthropometric				
BMI (kg/m^2)	28.8 (25.9, 32.2)	29.3 (26.3, 32.9)	28.3 (25.6, 31.3)	<0.001
Waist circumference (cm)	95 (87, 101)	95 (86, 101)	95 (89, 101)	0.004
HR (bpm)	75 (70, 82)	75 (70, 82)	76 (69, 82)	0.3
SBP (mm Hg)	130 (121, 139)	130 (120, 139)	130 (123, 139)	0.010
DBP (mm Hg)	80 (75, 88)	80 (75, 87)	81 (76, 88)	0.006
Biochemical				
Glucose (mg/dL)	102 (92, 126)	103 (92, 126)	101 (91, 125)	0.2
HbA1c (mg/dL)	7.10 (6.20, 8.96)	7.00 (6.23, 9.24)	7.40 (6.00, 8.54)	>0.9
Unknown	4288	2611	1677	
Triglycerides (mg/dL)	153 (118, 206)	154 (118, 205)	152 (118, 208)	0.8
Total cholesterol (mg/dL)	178 (154, 200)	181 (157, 201)	174 (151, 198)	<0.001
HDL-C (mg/dL)	46 (36, 57)	46 (36, 57)	46 (36, 55)	0.2
LDL-C (mg/dL)	106 (80, 129)	107 (80, 130)	105 (79, 128)	0.14
Serum creatinine (mg/dL)	0.83 (0.70, 1.00)	0.80 (0.70, 0.96)	0.90 (0.77, 1.10)	<0.001
CVD risk scores				
Office based – Globorisk score (%)	11 (7, 19)	12 (8, 19)	11 (5, 18)	<0.001
Laboratory based – Globorisk score (%)	12 (7, 22)	13 (7, 22)	11 (5, 22)	<0.001
SCORE2 score (%)	5.06 (3.86, 7.18)	4.37 (3.34, 5.07)	7.71 (6.46, 9.18)	<0.001
Prevent score (%)	15 (8, 24)	13 (7, 22)	17 (9, 27)	<0.001

(Continues)

TABLE 1 | (Continued)

Characteristics	Overall ($n = 4512$)	Women ($n = 2734$)	Men ($n = 1778$)	p value
End-organ damage				
Any end-organ damage component (%)	1492 (33%)	862 (32%)	630 (35%)	0.006
	776 (52%)	472 (55%)	304 (48%)	0.013
Decreased eGFR (%) *	761 (51%)	412 (48%)	349 (55%)	0.004
Left ventricular hypertrophy (%) *	69 (4.6%)	43 (5.0%)	26 (4.1%)	0.4
Hypertensive retinopathy (%) *	39 (2.6%)	23 (2.7%)	16 (2.5%)	0.9
Elevated albumin excretion (%) *	29 (1.9%)	7 (0.8%)	22 (3.5%)	<0.001
Peripheral artery disease (%) *	2 (0.1%)	1 (0.1%)	1 (0.2%)	>0.9
Unspecified end-organ damage (%) *	261 (5.8%)	134 (4.9%)	127 (7.1%)	0.002

Notes: Results are presented in n (%) or median (interquartile range), where appropriate. Annotations: * = Percentages are relative to the total cases of end-organ damage.

Abbreviations: BMI, body mass index; CKD, chronic kidney disease; CVD, cardiovascular disease; DBP, diastolic blood pressure; HbA1c, glycated hemoglobin; HDL-C, high-density lipoprotein cholesterol; HR, heart rate; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure.

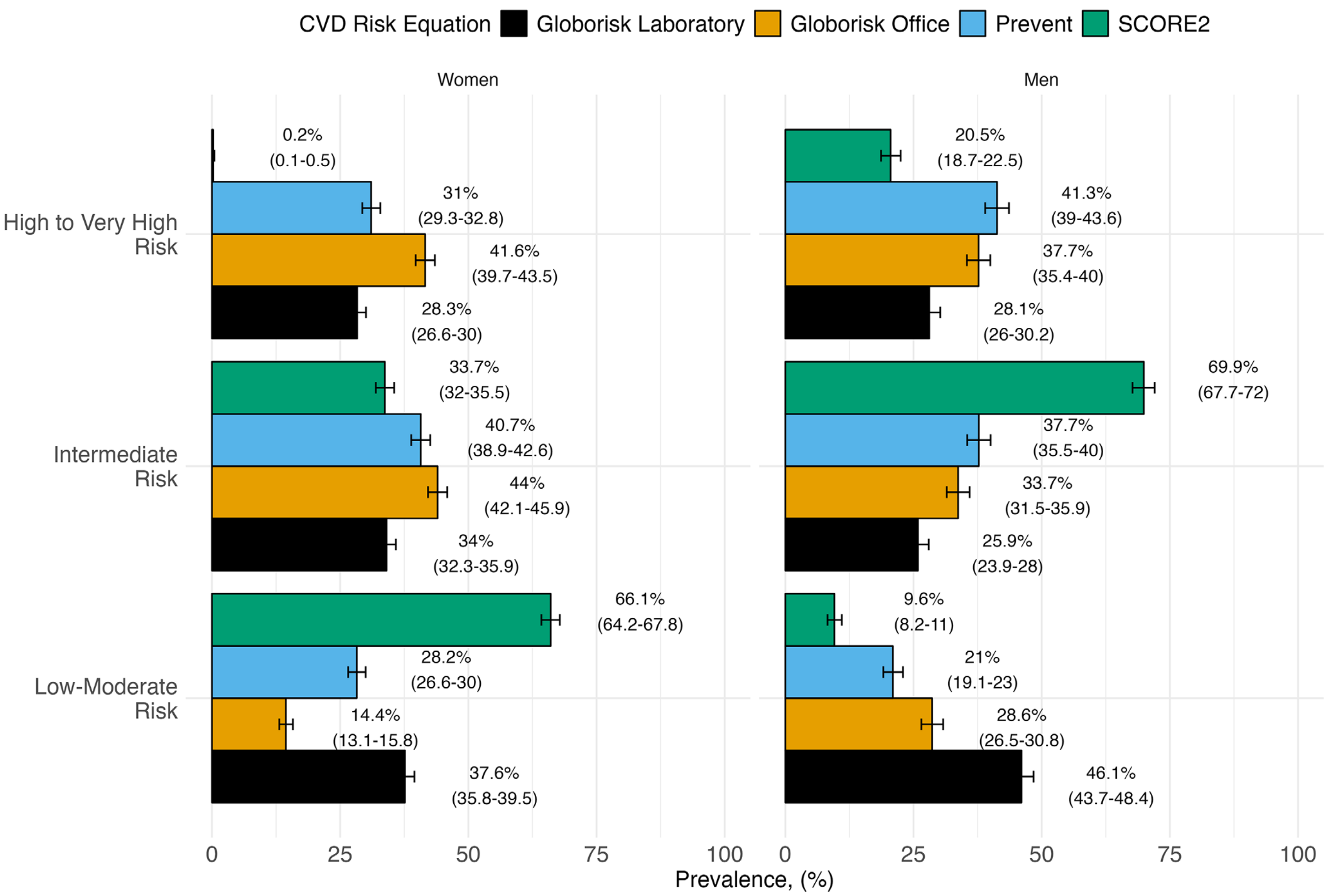

FIGURE 2 | Prevalence of CVD risk categories among women and men. Error bars represent 95% confidence intervals.

3.2 | Association of CVD Risk Scores and EOD

In our IPW models, a 1% increase in any of the CVD risk scores was positively associated with higher odds of EOD (Globorisk office-based: OR 1.09, [1.08–1.10]; Globorisk laboratory-based: OR 1.07, [1.06–1.08]; SCORE2: OR 1.04, [1.02–1.06]; PREVENT: OR 1.16, [1.15–1.17]). Similarly, classification as Intermediate (Globorisk office-based: OR 1.56, [1.39–1.76]; Globorisk laboratory-based: OR 1.58, [1.45–1.73]; SCORE2: OR 1.19, [1.09–1.30]; PREVENT: OR 3.40, [3.05–3.79]) or High-to-Very High (Globorisk office-based: OR 6.51, [5.8–7.32]; Globorisk laboratory-based: OR 3.41, [3.11–3.74]; PREVENT: OR 19.1, [17.1–21.4]) was linked to

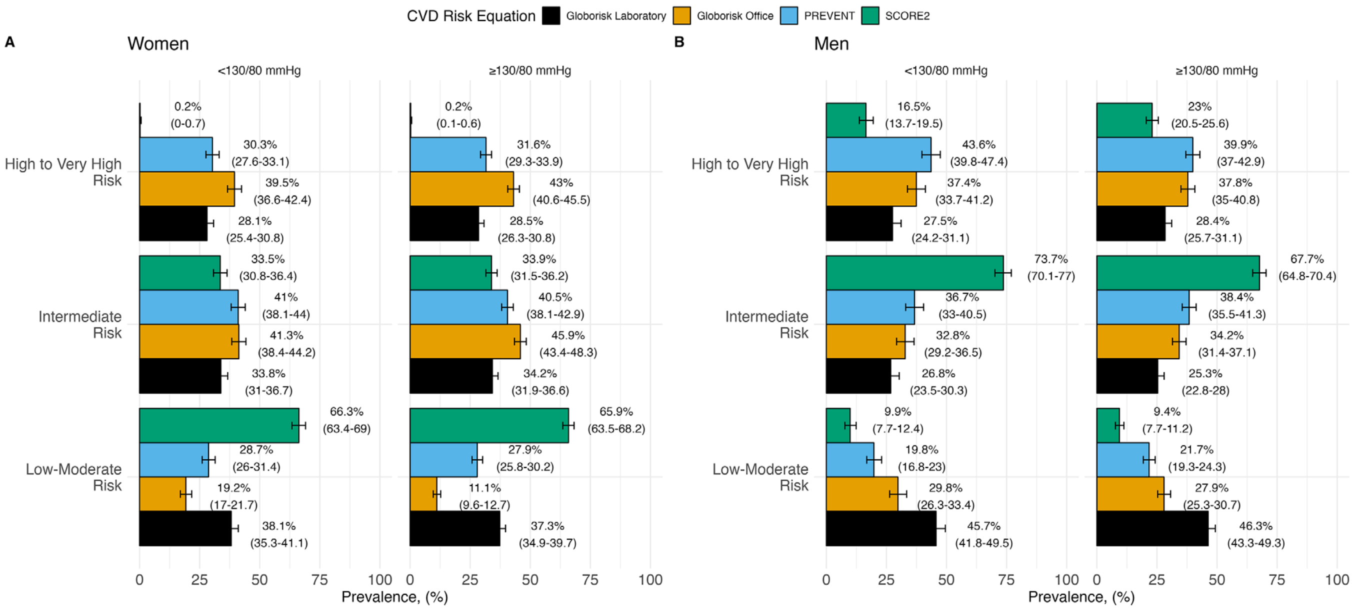

FIGURE 3 | Prevalence of CVD risk categories stratified by blood pressure control (<130/80 and ≥130/80 mm Hg) among women and men. Error bars represent 95% confidence intervals.

a greater likelihood of EOD compared to Low-to-Moderate categories for Globorisk (both laboratory- and office-based) and PREVENT (Table 2). Model assumptions are presented in Table S7. In Table 3, we display the performance of the four cardiovascular risk equations in discriminating the presence of EOD. Overall, the PREVENT score (AUROC: 0.751, [0.735–0.750]) demonstrated the highest discriminatory capacity, followed by the Globorisk office-based model (AUROC: 0.671, [0.655–0.672]), the Globorisk laboratory-based model (AUROC: 0.664, [0.647–0.664]), and the SCORE2 model (AUROC: 0.536, [0.519–0.538]). When stratifying the models by sex, we found that the Globorisk office-based model improved its diagnostic performance (AUROC: 0.706, [0.686–0.706]) in women compared to men (AUROC: 0.642, [0.616–0.643], $p = 0.001$). A similar trend was observed with the Globorisk laboratory-based equation (AUROC 0.692, [0.672–0.693] in women and 0.644, [0.618–0.645] in men, $p < 0.001$) and the PREVENT model (AUROC 0.763, [0.742–0.762] in women and 0.731, [0.704–0.730] in men, $p < 0.001$). Finally, SCORE2 exhibited lower discrimination in both groups (AUROC 0.569, [0.549–0.572] in women, 0.508, [0.481–0.508] in men, $p < 0.001$) (Figure 4).

3.3 | Sensitivity Analyses

In our sensitivity analyses, we observed that the PREVENT score consistently showed the strongest association and the greatest capacity to detect EOD. In the subset where we limited to adults aged 40–70 years without diabetes, albuminuria, or chronic kidney disease, the PREVENT score achieved an AUROC of 0.775 (95% CI 0.747–0.804). Then, after removing albuminuria and reduced eGFR from the EOD definition, its AUROC remained the highest at 0.679 (0.651–0.707), whereas both Globorisk versions improved slightly and SCORE2 remained lowest. In the third and fourth sensitivity analyses, where we excluded participants ≥60 years with pulse pressure ≥60 mm Hg, and separately removing all self-reported EOD events (hypertensive retinopathy, peripheral artery disease or left-ventricular hypertrophy), produced AUROCs for PREVENT that were virtually identical to the primary analysis, again exceeding those of the Globorisk and SCORE2 scores (Tables S8–S11).

4 | Discussion

The findings from this sub-analysis of the RIHTA study indicate that both the prevalence of cardiovascular risk categories across three risk equations and their capacity to detect EOD vary depending on the equation used. Compared to SCORE2 and the Globorisk equations, the PREVENT score demonstrated the highest prevalence of intermediate and high-to-very high cardiovascular risk and the strongest association with EOD. These findings were consistently replicated thought sensitivity analyses. Notably, blood pressure control did not diminish the classification of cardiovascular risk across the three calculators. Overall, these results demonstrate that the PREVENT equation can provide a more accurate assessment of cardiovascular risk in hypertensive populations.

Our findings underscore the potential value of incorporating kidney- and adipose-related markers into cardiovascular risk stratification for patients with arterial hypertension, especially since we observed a persistently high cardiovascular risk regardless of whether participants achieved blood pressure control. This suggests that underlying endothelial and vascular alterations, driven by cardiometabolic comorbidities, may continue to elevate cardiovascular risk in these patients [28]. A major differentiating factor of PREVENT is its inclusion of renal function and adiposity parameters, along with the option to consider the urine albumin–creatinine ratio, HbA1c, and the social deprivation index in the US, which are features that are not commonly integrated into many existing risk assessment models. Recent studies support this comprehensive approach. For instance, Carrillo-Larco et al.

TABLE 2 | Logistic regression models assessing odds ratios for end-organ damage associated with three CVD risk scores before and after inverse probability weighting adjustment.

CVD risk score	Unadjusted			After inverse probability weighting		
Continuous score	**OR**	**95% CI**	***p* value**	**OR**	**95% CI**	***p* value**
Office based – Globorisk	1.07	1.06, 1.08	<0.001	1.09	1.08, 1.10	<0.001
Laboratory based – Globorisk	1.06	1.05, 1.06	<0.001	1.07	1.06, 1.08	<0.001
SCORE2	1.04	1.02, 1.07	<0.001	1.04	1.02, 1.06	<0.001
PREVENT	1.09	1.08, 1.10	<0.001	1.16	1.15, 1.17	<0.001
CVD risk categories						
Office based – Globorisk						
Low-Moderate Risk	—	—		—	—	
Intermediate Risk	1.65	1.35, 2.02	<0.001	1.56	1.39, 1.76	<0.001
High to Very High Risk	4.02	3.32, 4.89	<0.001	6.51	5.80, 7.32	<0.001
Laboratory based – Globorisk						
Low-Moderate Risk	—	—		—	—	
Intermediate Risk	1.71	1.46, 2.00	<0.001	1.58	1.45, 1.73	<0.001
High to Very High Risk	3.66	3.13, 4.28	<0.001	3.41	3.11, 3.74	<0.001
SCORE2						
Low-Moderate Risk	—	—		—	—	
Intermediate Risk	1.34	1.18, 1.53	<0.001	1.19	1.09, 1.30	<0.001
High to Very High Risk	0.94	0.74, 1.20	0.6	0.99	0.90, 1.09	0.9
PREVENT						
Low-Moderate Risk	—	—		—	—	
Intermediate Risk	2.47	2.02, 3.03	<0.001	3.40	3.05, 3.79	<0.001
High to Very High Risk	7.99	6.57, 9.78	<0.001	19.1	17.1, 21.4	<0.001

Notes: The minimum set of confounders for the inverse probability weighting adjustment were age, sex, smoking status, cumulative self-reported chronic comorbidities, hypertension control (office BP < 130/80 mm Hg), and self-report use of ACEIs, ARBs, and statins. Horizontal bars indicate the reference group.
Abbreviations: OR, odds ratio; CI, confidence Interval.

TABLE 3 | Receiver operator curves to evaluate the capacity of CVD risk equations to predict end-organ damage.

CVD RISK SCORE	AUROC	Lower interval	Upper interval
Office based – Globorisk	0.671	0.655	0.672
Laboratory based – Globorisk	0.664	0.647	0.664
SCORE2	0.536	0.519	0.538
PREVENT	0.751	0.735	0.750

Abbreviations: AUROC, area under the receiver-operating characteristic (curve); CVC, cardiovascular disease.

found that, in a general US population drawn from the National Health and Nutrition Examination Survey, the PREVENT equation predicted a lower cardiovascular risk than the Pooled Cohort Equation [29]. Similarly, Zhou et al., in a 10-year follow-up of adults aged 40–75 from Kaiser Permanente Southern California, showed that the PREVENT equation offered a better 10-year ASCVD risk prediction [30]. Although both studies focused on the general population, our results from this sub-analysis of the RITHA study indicate that the PREVENT equation had the strongest odds ratio and the highest diagnostic performance for detecting EOD. Given the high prevalence of obesity and impaired renal function among hypertensive patients in Mexico, incorporating simple measures, such as eGFR and BMI, through the PREVENT equation may help facilitate earlier identification of subclinical organ damage related to both renal impairment and excessive adiposity. However, Cesaro et al. suggest including additional markers of ectopic fat distribution (e.g., visceral adiposity) to further enhance risk prediction of residual cardiovascular risk within the CRMS framework [31].

Hypertension is a silent condition that often goes undiagnosed and usually coexists with at least 5–6 cardiovascular risk factors [10]. Therefore, developing locally validated tools that incorporate these specific risk factors for Latino populations, within the broader CRMS framework, is a vital step toward a more comprehensive and personalized treatment strategies that would enhance cardiovascular risk assessment, particularly in patients with intermediate risk. The three risk scores assessed in our study differ markedly in their intended target populations, applicable age ranges, input variables, country-specific calibration options, cardiovascular outcomes, and output measures. Although they

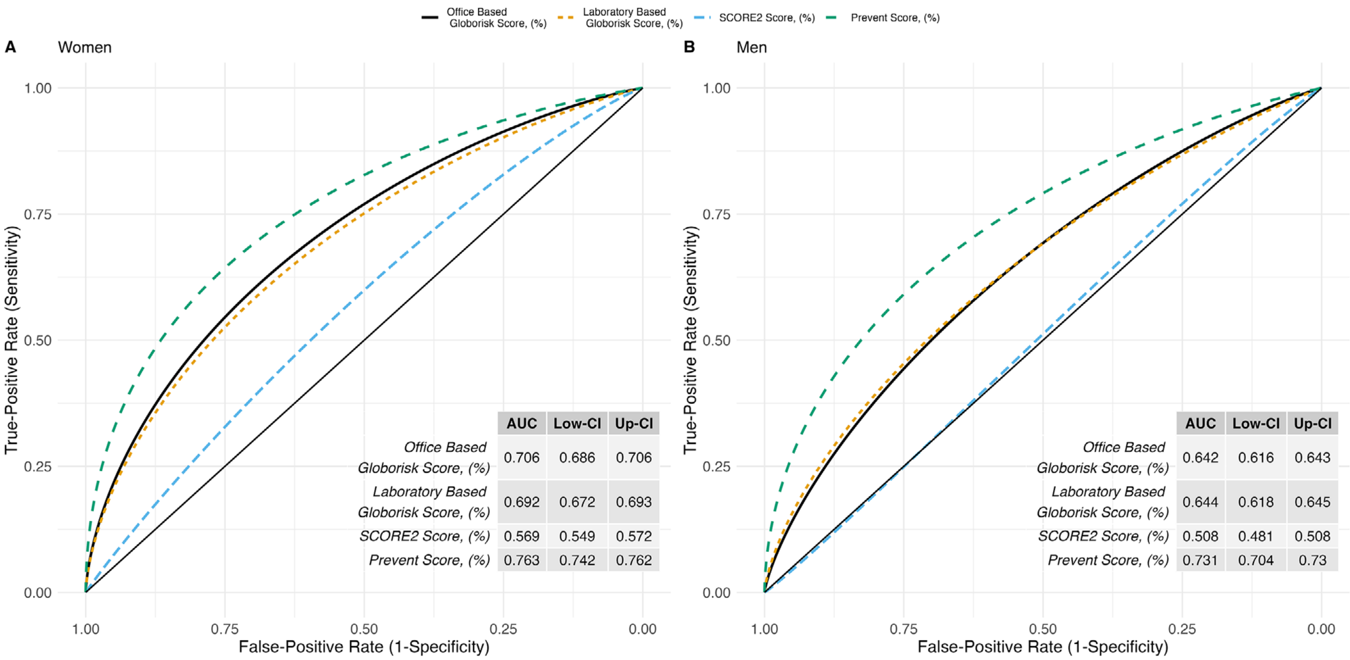

FIGURE 4 | ROC curves for CVD risk prediction models among women (A) and men (B). The area under the receiver operator curve (AUROC) and corresponding 95% confidence intervals are displayed in the table inset.

were not originally designed to detect established EOD, our findings suggest they can be used to screen for EOD within the CRMS framework and to identify patients who should undergo confirmatory imaging techniques. This is especially relevant because image-based diagnostic approaches to detect EOD are still scarce in Mexican primary-care settings. Although the AHA PREVENT calculator is not yet included in Mexican guidelines, it is a promising tool for thoroughly evaluating cardio-kidney-metabolic risk. Healthcare providers may find it relevant for Mexican patients with hypertension, as the results of our study can provide initial validation for this risk score and demonstrated improved accuracy to detect EOD compared to the Globo-Risk equation endorsed by the Mexican lipid guidelines and the SCORE 2 recommended in the Clinical Action Protocols of Mexico [11, 23]. Overall, our findings support the perspective that advocates for the use of the PREVENT equation as a holistic, patient-centered method of cardiovascular risk stratification [32].

4.1 | Strengths and Limitations

We must recognize both the strength and limitations of this study. One major strength is the data collection from RIHTA, with all participating clinics adhering to standardized protocols for blood pressure measurements and documenting clinical, treatment, and cardiovascular risk factors. Moreover, using EOD as a clinically significant outcome offers new insights that have not been assessed in hypertensive patients within our country before. However, several limitations merit consideration. First, the cross-sectional design of our study restricts our ability to draw causal inference, highlighting the necessity for replicating our findings using prospective cohorts among Mexican populations to explore the clinical screening utility of these cardiovascular risk scores. Second, some exclusion criteria and medical histories (e.g., previous cardiovascular disease) relied on self-reported information from patients, which introduces the possibility of misclassification bias. Thirdly, while we utilized a standardized definition for EOD, we could not verify three specific conditions, hypertensive retinopathy, peripheral artery disease, or left ventricular hypertrophy, leading to a potentially underestimated prevalence of these conditions. Finally, as the study population consisted of individuals from primary care centers, this could represent a selection bias and may restrict the applicability of our findings to other healthcare settings.

5 | Conclusions

In this sub-analysis of the RIHTA study, the PREVENT score indicated a greater prevalence of intermediate, high, and very-high cardiovascular risk, even among those managing blood pressure effectively. Additionally, it demonstrated a stronger positive correlation with EOD compared to Globorisk and SCORE2. These results highlight the necessity of incorporating kidney function and measures of adiposity into standard risk assessments using the CRMS framework, especially in Mexico, where arterial hypertension frequently occurs alongside other cardiometabolic disorders.

Author Contributions

Silvia Palomo-Piñón, Luis Alcocer, Humberto Álvarez-López, Ernesto G. Cardona-Muñoz, Adolfo Chávez-Mendoza, Enrique Díaz-Díaz, José Manuel Enciso-Muñoz, Héctor Galván-Oseguera, Martin Rosas-Peralta, Luis Rey García-Cortés, Moisés Moreno-Noguez, and Neftali Eduardo Antonio-Villa on behalf of the Mexican Group of Experts on Arterial Hypertension. Research idea and study design: Silvia Palomo-Piñón, Neftali Eduardo Antonio-Villa. Data acquisition: Silvia Palomo-Piñón, Humberto Álvarez-López, Ernesto G. Cardona-Muñoz, Adolfo Chávez-

Mendoza, Enrique Díaz-Díaz, José Manuel Enciso-Muñoz, Héctor Galván-Oseguera, Martin Rosas-Peralta, Luis Rey García-Cortés, Moisés Moreno-Noguez. Data analysis/interpretation: Neftali Eduardo Antonio-Villa. Statistical analysis: Neftali Eduardo Antonio-Villa. Manuscript drafting: Silvia Palomo-Piñón, Neftali Eduardo Antonio-Villa, Luis Alcocer. Supervision or mentorship: Silvia Palomo-Piñón, Luis Alcocer. Each author contributed significant intellectual content during the drafting or revision of the manuscript and accepted responsibility for the overall work by ensuring that any questions regarding the accuracy or integrity of any part of the work were thoroughly investigated and resolved.

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Ethics Statement

The RIHTA study received approval from the Medical Ethics Committee of the Mexican Institute of Social Security, and all patients provided verbal informed consent prior to registration. As this sub-analysis utilized previously collected data from the RIHTA study, additional ethics committee approval was waived.

Patients and Public Involvement

The individual patient data were anonymized and are not presented in this manuscript. Patients were involved during the application process. No patient was involved in the study planning nor the analysis nor dissemination of results.

Conflicts of Interest

The authors declare that they have no competing interests.

Data Availability Statement

The dataset presented in this article is owned by the Mexican Group of Experts on Arterial Hypertension. Requests to access the dataset should be directed to: silvia-palomo@hotmail.com. R code to reproduce the results can be found at: https://github.com/neftalivilla/RIHTA_CVD_SCORES.

Mexican Group of Experts on Arterial Hypertension

The Mexican Group of Experts on Arterial Hypertension is composed of (in alphabetical order): Luis Alcocer, Neftali Eduardo Antonio-Villa, Tabata Gabriela Anguiano-Velázquez, Humberto Álvarez-López, Diana Amaya-Mora, Ernesto G. Cardona-Muñoz, Adolfo Chávez-Mendoza, Ma. Adriana Cruz-Arce, Jairo Enoc Cruz-Toledo, María De Los Angeles Dichi-Romero, Enrique Díaz-Díaz, José Manuel Enciso-Muñoz, María Eugenia Figueroa-Suárez, Irma Fabiola García-Padilla, Luis Rey García-Cortés, Ana Lilia González-Ramírez, Vidal José González-Coronado, Héctor Galván-Oseguera, Ana Laura Guerrero-Morales, Imer Guillermo Herrera-Olvera, Oscar Jiménez-Jalpa, María Elisa López-Delgado, Gloria Mendoza-López, Moisés Moreno-Noguez, Flor Araceli Nava-Ayala, Silvia Palomo-Piñón, Isaac Pérez-Zamora, Olivia Reyes-Jiménez, Rubén Ríos-Morales, Martin Rosas-Peralta, Francisco Vargas-Hernández, Pedro Luis Vargas-Gutiérrez, and Alfonso Zempoalteca-Morales.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.

Table S1: STROBE cross-sectional guidelines report for study. This checklist was completed on March 17th, 2025, using https://www.goodreports.org/ , a tool made by the EQUATOR Network in collaboration with Penelope.ai (von Elm E, Altman DG, Egger M, Pocock SJ, GotzschePC, Vandenbroucke JP. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement: guidelines for reporting observational studies). **Table S2:** Components, Validation Populations, Strengths, and Limitations of CVD Risk Scores. **Table S3:** Definitions of Cardiovascular Disease Risk Categories Across Different Risk Scores. **Table S4:** Definition of individual EOD components based on the definition proposed by the definition proposed by Roland E Schmieder (7). **Table S5:** Prevalence of CVD Risk Within the Studied Sample. Abbreviations: CVD, Cardiovascular Disease; 95% CI, 95% Confidence Interval. **Table S7:** Models assumptions. **Table S8:** Sensitivity analysis 1 – Association between CVD risk scores and end-organ damage using logistic regression models before and after inverse probability weighting (IPW) in a harmonized subsample (n = 1,832) aged 4070 years without diabetes, microalbuminuria, or chronic kidney disease. **Table S9:** Sensitivity analysis 2 – Association between CVD risk scores and end-organ damage using logistic regression models before and after inverse probability weighting in a harmonized subsample (n = 3,737) of individuals without microalbuminuria or decreased eGFR (<60 mL/min/1.73 m^2). **Table S10:** Sensitivity analysis 3 – Association between CVD risk scores and end-organ damage using logistic regression models before and after inverse probability weighting in a harmonized subsample (n = 3,920) excluding individuals aged ≥60 years and with aortic stiffness (pulse pressure ≥60 mmHg). **Table S11:** Sensitivity analysis 4 – Association between CVD risk scores and end-organ damage using logistic regression models before and after inverse probability weighting in a harmonized subsample (n = 1,384) excluding self-reported history of hypertensive retinopathy, peripheral artery disease or left-ventricular hypertrophy. **Figure S1:** We used the mice R Package (Version 3.14.0) to impute continuous missing values, assuming that data were missing completely at random. The imputation was done using multiple chained equations, and we created five imputed datasets. We combined the imputed datasets using Rubin's rules for a maximum of five iterations. We report the percentage of missing values for continuous variables (A), as well as density histograms (B) and summary statistics (C) for both the original and imputed variables. We found no statistically significant differences between the imputed variables and the original distribution of the missing continuous variables. **Figure S2:** Direct acyclic graphs base on the recommendations by Digitale et al (9) to evaluate and exclude confounding effects between the relationship of CVD risk scores and end-organ damage.