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SCORE2 *versus* SCORE in patients with systemic lupus erythematosus

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Abstract

Introduction: Systemic lupus erythematosus (SLE) has been associated with an increased risk of cardiovascular (CV) disease. Recently, the Systematic Coronary Risk Assessment (SCORE), a well-known CV risk algorithm, has been updated to a new predictive model (SCORE2). This new algorithm improves the identification of individuals at high risk of developing CV disease across Europe. Since carotid atherosclerosis is a predictor of future CV events and CV death, our objective was to compare the predictive capacity of SCORE2 *versus* SCORE for the presence of subclinical carotid atherosclerosis in patients with SLE.

Methods: Two hundred and thirty-five individuals over 40 years of age diagnosed with SLE were consecutively recruited in this cross-sectional study. SCORE and SCORE2 were calculated. The relationship of SCORE and SCORE2 with each other, and with the presence of subclinical carotid atherosclerosis (both carotid plaque and carotid intima media thickness -cIMT-), was studied.

Results: SCORE2 and SCORE did not correlate with each other (Spearman's Rho = 0.125, $p = 0.065$). Although SCORE did not correlate with cIMT (Spearman's Rho = -0.022, $p = 0.75$), the correlation of SCORE2 with cIMT was statistically significant (Spearman's Rho = 0.367, $p < 0.001$). Similarly, SCORE did not show significant discrimination for the presence of carotid plaque [AUC = 0.521 (95% CI = 0.443–0.600)], while SCORE2 did [AUC = 0.720 (95% CI = 0.656–0.785)]. The difference between AUCs was found to be statistically significant ($p < 0.001$), thus showing that the prediction capacity of SCORE2 was significantly higher than that of SCORE.

Conclusion: In SLE patients, the ability of SCORE2 to predict the presence of subclinical atherosclerosis is higher than that of SCORE. According to our results, SCORE2, rather than SCORE, should be used in the CV risk stratification of patients with SLE. Prospective studies are needed to confirm these findings.

Keywords: cardiovascular risk assessment, systemic lupus erythematosus

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Introduction

Patients with systemic lupus erythematosus (SLE) are at high risk of premature cardiovascular disease (CVD).[1] Both traditional and nontraditional risk factors contribute to this complication. As seen in other inflammatory diseases, current cardiovascular (CV) risk calculation tools used in the general population underestimate the actual CV risk of patients with SLE.[2–4] For example, in a previous report of SLE patients without prior CVD or diabetes, five generic and three SLE-adapted clinical risk scores underestimated high CVD risk as defined by the presence of atherosclerotic plaque.[4] This was also the case with the Systematic Coronary Risk Assessment (SCORE) CV death risk calculator, which was developed in 2003 for use in European populations.[5]

The SCORE CV risk algorithm has been updated to a new predictive model (SCORE2), which was launched in 2021.[6] SCORE2 has been calibrated and validated to predict the 10-year risk of first-onset CV disease in European populations. SCORE2 differs from SCORE in several aspects.

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For example, SCORE2 provides risk estimates for the combined outcome of fatal and nonfatal CVD events, in contrast with SCORE's use of CVD mortality only. In addition, SCORE2 has been systematically recalibrated using the contemporary CVD rates available, whereas the original SCORE model was based on data collected before 1986. Moreover, SCORE2 accounts for the impact of competing risks by non-CVD deaths whereas SCORE does not. In fact, and SCORE2 is recalibrated to four distinct European regions rather than the two-level regional stratification provided by SCORE.[6]

The predictive value of SCORE2 in identifying SLE patients at high risk of CV disease is unknown. Since carotid atherosclerosis is a predictor of future CV events and CV death in SLE patients,[7] our objective was to compare the predictive capacity of SCORE2 *versus* SCORE calculators for the presence of subclinical carotid atherosclerosis in these patients.

Materials and methods

Study participants

This was a cross-sectional study that included 235 consecutively patients with SLE. Patients were recruited during 2018 and 2019. All SLE patients were 40 years old or older, had a clinical diagnosis of SLE, and fulfilled ⩾ 4 American College of Rheumatology (ACR) classification criteria for SLE.[8] Patients were excluded if they were diabetic or had a history of myocardial infarction, angina, stroke, a glomerular filtration rate <60 ml/min/1.73 m^2, a history of cancer, and/or any other chronic disease or evidence of active infection. We did not include diabetes patients as these individuals are generally considered at high risk of CVD (and, therefore, are automatically eligible for statin medications and other preventive interventions), and specific risk scores already exist for this population. Research was carried out in compliance with the Declaration of Helsinki. The study protocol was approved by the Institutional Review Committees at Hospital Universitario de Canarias and Hospital Doctor Negrín (both in Spain), and all individuals provided informed written consent (Approval Number 2015_84).

Data collection

RA patients recruited in this work completed a questionnaire on medication use and CV risk factors and underwent a physical examination. Body-mass index (the weight in kilograms divided by the square of the height in meters), abdominal circumference and systolic and diastolic blood pressure were assessed under standardized conditions. Obesity represents a body-mass index equal to or higher than 30 kg/m^2. Hypertension was defined as a systolic or a diastolic blood pressure higher than, respectively, 140 and 90 mmHg, in accordance with the 2018 ESC/ESH Guidelines for the management of arterial hypertension.[9] Smoking status (current smoker *versus* nonsmoker) was recorded. Dyslipidemia was defined if one of the following criterion was met: total cholesterol $>$ 200 mg/dl, triglycerides $>$ 150 mg/dl, HDL cholesterol $<$ 40 in men or $<$ 50 mg/dl in women, or LDL cholesterol $>$ 130 mg/dl.

SLE disease activity and damage were assessed using the Systemic Lupus Erythematosus Disease Activity Index—2000 (SLEDAI—2K)[10] and the SLICC/ACR Damage Index (SDI),[11] respectively. For the purpose of this study, the SLEDAI-2K index was broken down into none (0 points), mild (1–5 points), moderate (6–10 points), high (11–19), and very high activity ($>$20) as previously described.[12] Disease severity was measured as well, using the Katz Index.[13] The immunological data recorded represents the actual data at the time the study was performed.

The SCORE and SCORE2 were calculated as described elsewhere.[5,6] SCORE2 was calculated using age, smoking status, systolic blood pressure, and non-HDL-cholesterol. SCORE was assessed with age, smoking status, systolic blood pressure, and total cholesterol. For the first, only whole numbers are shown as it was calculated using the recently published charts.[6,14] In contrast, for SCORE, numbers with decimals were available as this was calculated using the exact algorithm described by Conroy *et al.*[5] SCORE has been classically categorized into low ($<$1%), moderate (1–4%), high (5–9%), or very high ($>$10%) risk categories. In contrast, the 2021 European Society of Cardiology Guidelines on CV disease prevention in clinical practice[14] proposed that the SCORE2 risk categories be reduced to three (low to moderate, high and very high) and that different numerical cutoff levels be used according to age groups ($<$50, 50–69, and ⩾70 years of age). In addition, SCORE estimated the 10-year risk of death from CV disease. However, since CV disease morbidity, combined with CV disease mortality, better reflects the total

burden of atherosclerotic CV disease, SCORE2 estimates an individual's 10-year risk of fatal and nonfatal CV disease events in individuals aged 40–69 years. For healthy people aged ⩾ 70 years, the SCORE2-OP (older persons) algorithm estimates 5-year and 10-year fatal and nonfatal CV disease events.

A carotid ultrasound examination was used to assess carotid intima-media wall thickness (cIMT) in the common carotid artery and to detect focal plaques in the extracranial carotid based on the Mannheim consensus.[15,16]

The reporting of this study conformed to the STROBE statement.[17] A checklist of these guidelines has been submitted (Supplementary Table 1).

Statistical analysis

Demographic and clinical characteristics in patients were described as mean (standard deviation) or percentages for categorical variables. For non-normally distributed continuous variables, data were expressed as median and interquartile range (IQR). Linear association between continuous variables was studied using Spearman Rho correlation coefficients. Relations of SCORE and SCORE2 to the presence of carotid plaque in SLE patients were analyzed through the relation of sensitivity *versus* false-positive frequency (1-specificity) using receiver-operating characteristic curves (ROC). A comparison of ROC curves, to test the statistical significance of the difference between the areas under two dependent ROC curves (AUC) (derived from the same cases), was conducted using the method of DeLong *et al.*[18] Missing data were handled through listwise deletion. All analyses used a 5% two-sided significance level and were performed using SPSS software, version 25 (IBM, Chicago, IL, USA). A p value < 0.05 was considered statistically significant.

Results

Demographic, laboratory, and disease-related data

A total of 235 patients with SLE were included in this study. Demographic and disease-related characteristics of the participants are shown in Table 1. Most of the patients were women (94%) and the mean age ± SD was 54 ± 9 years. Twenty-four of the patients were current smokers, 43% had hypertension, and 41% fulfilled the definition for dyslipidemia. Similarly, although patients who had had CV events were excluded, some were taking preventive drugs for CV disease. In this sense, 27% of the patients were taking statins, and 24% and 40% were, respectively, receiving aspirin or antihypertensive treatment (Table 1).

Disease duration was 18 (IQR 12–26) years. SLICC and Katz indexes were 1 (IQR 1–2) and 2 (IQR 1–4), respectively. Most SLE patients were in the no activity (43%) or mild activity (31%) categories as shown by the SLEDAI scores. Seventy-five percent of the patients had a SLICC/ACR DI score equal to or higher than 1, and 37% had a Katz index equal to or higher than 3. Almost half of the patients (47%) were taking prednisone (the median dose of those 111 patients on prednisone was 5 (IQR 5–7.5) mg/day at the time of the study). At the time of recruitment, 59% patients were positive for anti-DNA, and 23% were positive for ENA, with anti-Ro being the antibody most frequently found (33%). disease-modifying antirheumatic drug (DMARD) use was reported in 74% of the patients and 67% were taking hydroxychloroquine at the time of the study. Regarding subclinical carotid atherosclerosis, the mean cIMT was 650 ± 111 μm, and 41% of the patients had carotid plaques. Additional information on the SLE patients is shown in Table 1.

Relation between SCORE2 and SCORE and to carotid plaque and cIMT

Absolute values of SCORE and SCORE2 were, respectively, 0 (IQR 0–1) and 3 (IQR 2–4). Neither calculator correlated with the other (Spearman's Rho = 0.125, $p = 0.065$).

SLE patients were distributed into the following SCORE categories: 149 (63%) in the low CV risk category, and 59 (25%), 8 (3%), and 19 (8%) in the moderate, high, and very-high categories, respectively. When categories of SCORE2 were assessed, 165 (70%) of the patients were found to be in the low or moderate risk category, and 67 (29%) and 3 (1%) in the high and very-high categories. The distribution of patients according to categories was significantly different between the two scores ($p < 0.001$) (Figure 1).

Although SCORE was not correlated with cIMT (Spearman's Rho = -0.022, $p = 0.75$), the correlation of SCORE2 with cIMT was statistically significant (Spearman's Rho = 0.367, $p < 0.001$)

Table 1. Characteristics of SLE patients.

	SLE patients	
	(*n* = 235)	Missing data
Age, years	54 ± 9	0 (0)
Women, *n* (%)	221 (94)	0 (0)
Body mass index, kg/m^2	28 ± 6	1 (0)
Abdominal circumference, cm	93 ± 13	4 (2)
Systolic blood pressure, mmHg	129 ± 19	0 (0)
Diastolic blood pressure, mmHg	85 ± 47	0 (0)
Cardiovascular comorbidity		
Current smoker, *n* (%)	57 (24)	0 (0)
Diabetes, *n* (%)	–	
Hypertension, *n* (%)	101 (43)	1 (0)
Obesity, *n* (%)	68 (29)	1 (0)
Dyslipidemia, *n* (%)	97 (41)	0 (0)
Statins, *n* (%)	64 (27)	0 (0)
Aspirin, *n* (%)	57 (24)	7 (3)
Antihypertensive treatment, *n* (%)	95 (40)	0 (0)
Analytical and lipid profile		
CRP, mg/dl	2 (1–4.9)	0 (0)
Cholesterol, mg/dl	198 ± 37	0 (0)
Cholesterol ⩾ 200 mg/dl, *n* (%)	160 (68)	0 (0)
Triglycerides, mg/dl	131 ± 81	0 (0)
HDL cholesterol, mg/dl	63 ± 20	0 (0)
LDL cholesterol, mg/dl	114 ± 29	0 (0)
LDL ⩽ 130 mg/dl, *n* (%)	123 (52)	0 (0)
Non-HDL cholesterol, mg/dl	136 ± 34	0 (0)
Atherogenic index	3.40 ± 1.04	0 (0)

(continued)

Table 1. (continued)

	SLE patients	
	(*n* = 235)	Missing data
SLE-related data		
Disease duration, years	18 (12–26)	1 (0)
SLICC	1 (1–2)	3 (1)
SLICC ⩾ 1, *n* (%)	175 (75)	3 (1)
Katz Index	2 (1–4)	7 (3)
Katz Index ⩾ 3, *n* (%)	87 (37)	7 (3)
SLEDAI	2 (0–4)	12 (5)
SLEDAI activity categories, *n* (%)		
No activity, *n* (%)	101 (43)	
Mild, *n* (%)	73 (31)	
Moderate, *n* (%)	31 (13)	
High and very high, *n* (%)	16 (7)	
Past renal involvement, *n* (%)	23 (10)	0 (0)
Auto-antibody profile		
Anti-DNA positive, *n* (%)	140 (59)	44 (19)
ENA positive, *n* (%)	55 (23)	16 (7)
Anti-Ro, *n* (%)	77 (33)	40 (17)
Anti-La, *n* (%)	34 (14)	41 (17)
Anti-RNP, *n* (%)	57 (24)	30 (13)
Anti-Sm, *n* (%)	28 (12)	16 (7)
Any antiphospholipid autoantibodies, *n* (%)		
Lupus anticoagulant, *n* (%)	57 (24)	34 (14)
ACA IgM, *n* (%)	24 (10)	32 (14)
ACA IgG, *n* (%)	44 (18)	32 (14)
Anti-beta2 glycoprotein IgM, *n* (%)	20 (8)	40 (17)
Anti-beta2 glycoprotein IgG, *n* (%)	31 (13)	40 (17)

(continued)

Table 1. (continued)

	SLE patients	
	(*n* = 235)	**Missing data**
C3, mg/dl	100 ± 27	38 (16)
C4, mg/dl	18 ± 8	38 (16)
Current prednisone, *n* (%)	111 (47)	4 (2)
Prednisone, mg/day	5 (5–7.5)	4 (2)
DMARDs, *n* (%)	175 (74)	3 (1)
Hydroxychloroquine, *n* (%)	157 (67)	3 (1)
Methotrexate, *n* (%)	29 (12)	0 (0)
Mycophenolate mofetil, *n* (%)	20 (8)	0 (0)
Azathioprine, *n* (%)	25 (11)	0 (0)
Rituximab, *n* (%)	6 (3)	0 (0)
Belimumab, *n* (%)	4 (2)	0 (0)
Cyclophosphamide, *n* (%)	1 (0)	0 (0)
Subclinical atherosclerosis		
Carotid IMT, microns	650 ± 111	0 (0)
Carotid plaques, *n* (%)	96 (41)	0 (0)

ACA, anticardiolipin; ANA, antinuclear antibodies; BMI, body mass index; C3 C4, complement; CRP, C-reactive protein; DMARD, disease-modifying antirheumatic drug; ENA, extractible nuclear antibodies; HDL, high-density lipoprotein; LDL, low-density lipoprotein; SLEDAI, Systemic Lupus Erythematosus Disease Activity Index; SLICC: Systemic Lupus International Collaborating Clinics/American Colleague of Rheumatology Damage Index.
Data represent mean ± SD or median (interquartile range) when data were not normally distributed.
SLEDAI categories were defined as: 0, no activity; 1–5 mild; 6–10 moderate; > 10 activity.
Dyslipidemia was defined if one of the following was present: total cholesterol > 200 mg/dl, triglyceride > 150 mg/dl, HDL cholesterol < 40 in men or < 50 mg/dl in women, or LDL cholesterol > 130 mg/dl.

(Figure 2). This was also the case for carotid plaque, since SCORE did not show significant discrimination for the presence of carotid plaque [AUC = 0.521 (95% CI = 0.443–0.600)], while SCORE2 did [AUC = 0.720 (95% CI = 0.656–0.785)] (Figure 2). In this regard, the difference between AUCs was found to be statistically significant ($p < 0.001$).

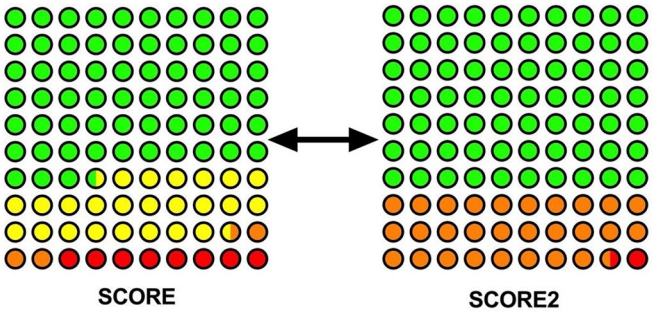
Moreover, 26% and 37% of the patients within the high or very-high CV risk categories per SCORE2 were taking, respectively, aspirin and statins (data not shown). This showed that most of the patients within these high and very-high categories were not taking preventive CV drugs.

Discussion

Calculating CV risk in patients with SLE is challenging. Most CV risk calculators used in the general population have been found to perform poorly in patients with SLE.[4] For example, in a recent single-center analysis involving 1887 patients with SLE followed prospectively, the authors sought to determine which among of the following methods best predicted CVD events: the QRESEARCH risk estimator versions 2 and 3, the Framingham Risk Score, the modified Framingham Risk Score or the SLE CV Risk Equation—SLECRE. It was concluded none of the scores achieve robust sensitivity, specificity, or accuracy in these population.[19] The chronic inflammation that accompanies the disease, the accelerated atherosclerosis process, the presence of inflammatory dyslipidemia,[20] and the alteration of glucose homeostasis metabolism[21] that these patients present are all responsible for this poor performance. According to our results, SCORE2, and not SCORE, is the better choice for CV risk assessment in patients with SLE.

In a recent work by our group, QRESEARCH risk estimator version 3 (QRISK3), which was developed in 2017, showed a discrimination for subclinical atherosclerosis higher than that of SCORE in patients with SLE.[3] SCORE was developed from cohorts recruited before 1986 and, to date, has not been systematically recalibrated to contemporary CV disease rates. We believe that those CV risk calculation systems developed in recent years, such as QRISK3 and SCORE2, may be more accurate at predicting CV events not only in the general European population, but also in patients with inflammatory diseases.

In our study, 11% of SLE patients were considered to be in the high or very-high CV risk category using SCORE. However, when the SCORE2 calculation was performed, the percentage of patients included in these categories rose to 30%.



		SCORE2 categories		
		Low to moderate	High	Very high
		165 (70%)	67 (29%)	3 (1%)
SCORE categories				
Low	149 (63%)	108	39	2
Moderate	59 (25%)	39	19	1
High	8 (3%)	4	4	0
Very High	19 (8%)	14	5	0

Figure 1. Differences in the distribution of CV risk categories between SCORE and SCORE2 calculators.

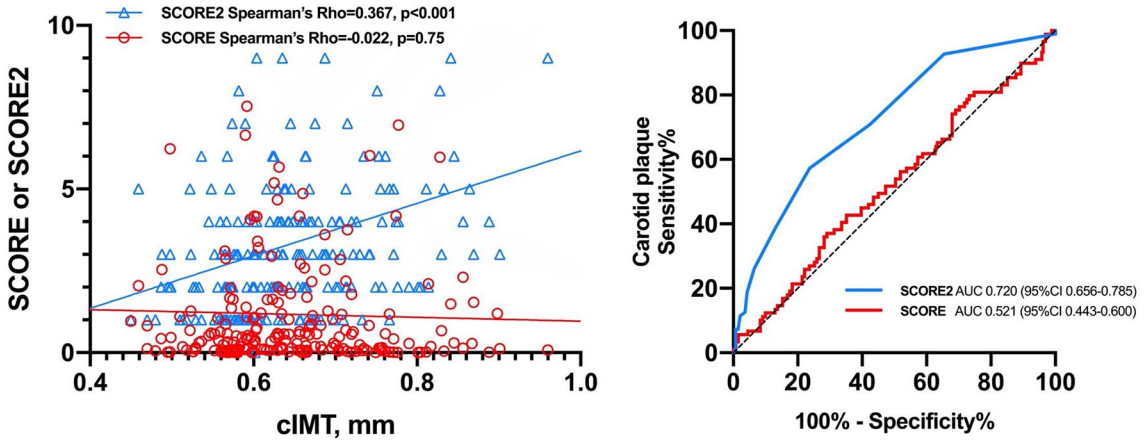


Figure 2. Relationship of SCORE and SCORE2 to cIMT and carotid plaque.

Moreover, according to our data, 63% and 74% of patients within these high or very-high SCORE2 CV risk categories were not taking, respectively, statins or aspirin preventive CV risk drugs. This is very relevant because with the new SCORE2 tool, the percentage of patients with SLE who would have an indication for lipid-lowering therapy or who would have required a lower LDL-cholesterol goal would be higher. For this reason, the use of the SCORE2, *versus* SCORE, would not only have implications in terms of a more precise CV risk calculation, but would also experience therapeutic repercussions.

We recognize certain limitations; that is, the number of patients recruited may be considered small and that SCORE2 was developed for predicting CV events and not subclinical arteriosclerosis. Regarding the first concern, we contend that future studies using a prospective design should be carried out to confirm these results. In regards to the latter, it must be taken into account that subclinical carotid arteriosclerosis has been shown to be strongly related to future CV events not only in the general population,[22] but also in other inflammatory diseases.[7,23] In addition, only Caucasians patients were included in our study. For this reason, we acknowledge that our findings cannot be extrapolated to other races. Moreover, disease duration in our series was found to be long, which may have affected our results. As previously mentioned, larger series of SLE patients under a prospective design study are needed to better analyze the effects that disease duration may have on CV risk calculators. Finally, because diabetes mellitus is equivalent to a very-high CV risk category, SLE patients with diabetes were not included in our study. We, therefore, acknowledge the limitation that our findings would not apply to those SLE patients who are diabetic.

In conclusion, according to our results, the updated SCORE2, a new version of SCORE, should be used for CV risk assessment in SLE

patients. Our findings will have to be confirmed in studies using a prospective design that set CV events as the outcome.

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Author contribution(s)

Juan Carlos Quevedo-Abeledo: Conceptualization; Data curation; Formal analysis; Methodology.

Miguel Á. González-Gay: Conceptualization; Funding acquisition; Writing – original draft; Writing – review & editing.

Iván Ferraz-Amaro: Conceptualization; Data curation; Formal analysis; Funding acquisition; Methodology; Writing – original draft; Writing – review & editing.

Conflict of interest statement

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Data availability

The data underlying this article will be shared upon reasonable request to the corresponding authors.

Supplemental material

Supplemental material for this article is available online.

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