248 VALIDATION OF THE ADJUSTED GLOBAL ANTI-PHOSPHOLIPID SYNDROME SCORE IN THE ARGENTINE POPULATION

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10.1136/lupus-2019-lsm.248

Background Assessment of risk both for pregnancy morbidity and thrombosis in the presence of antiphospholipid antibodies (aPL) is still a challenge.

Objective to assess the performance of adjusted Global Anti-phospholipid Syndrome Score (aGAPSS) in predicting thrombosis in the setting of an external cohort study in patients with Systemic Lupus Erythematosus (SLE).

Methods Consecutive SLE patients from five rheumatology centers were included. Conventional cardiovascular risk factors were recorded as well as other underlying factors for thrombosis. Immunological tests were also recorded: ANA, anti-DNA, anti-SSA/SSB, anti-RNP, anti-Sm and aPL (Lupus Anticoagulant (LA), anti-cardiolipin (aCL) and 2 Glycoprotein I (2GPI). Medications received by patients were hydroxicloroquine, aspirin and anticoagulants. aGAPSS was calculated for each patient using a point system: 1 for arterial hypertension, 3 for dyslipidemia, 4 for LA and B2GPI and 5 for aCL. The score ranges from 0 to 17. The discriminative ability of aGAPSS was calculated by measuring the area under the receiver operating characteristic (ROC) curve (AUC). Multivariate survival analysis was performed using the proportional hazards model to identify the association of the aGAPSS cut-off value with thrombotic events adjusted to potential confounding factors. Multivariate logistic regression analysis was performed to examine the impact of multiple cardiovascular risk factors and laboratory parameters on the occurrence of thrombosis. A 95% confidence interval (CI) was selected and a p value<0.05 was considered significant.

Results Information was collected from 216 SLE patients (89.8% women, mean age at SLE diagnosis of 31 years (SD ±10.5). Ninety patients (41.6%) presented thrombotic and/or pregnancy complications. Forty-three patients (19.9%) presented at least one thrombotic episode (53 events; 28 arterial and 25 venous thromboses). Sixty women (30.9%) presented at least one pregnancy complication (81 events; 34 miscarriages, 28 fetal deaths and 19 premature deliveries). Median aGAPSS was significantly higher in patients who experienced a thrombotic event compared with those who had not [4 (IQR 1–9) versus 1 (IQR 0–5); p 0.001]. The AUC showed that aGAPSS 8 presented the best diagnostic accuracy [0.63 (CI95% 0.55–0.70) p 0.03] with 30% sensitivity and 82.5% specificity. Multivariate analysis indicated aGAPSS 8 was an independent predictor of thrombosis [OR: 2.1 (CI95% 1.03–4.12) p 0.04].

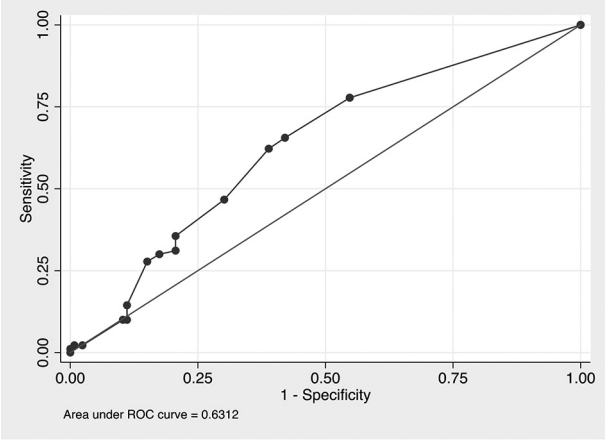
Conclusions This score is a simple tool to predict risk of thrombosis in SLE patients in daily practice. The use of aGAPSS could change the non-pharmacological and pharmacological treatment in higher risk patients to improve survival.

Funding Source(s): None

Abstract 248 Table 1 Detailed report of sensitivity and specificity

			Correctly		
Cut point	**Sensitivity**	**Specificity**	**Classified**	**LR+**	**LR-**
(≥0)	100.00%	0.00%	41.67%	1.0000	
(>1)	77.78%	45.24%	58.80%	1.4203	0.4912
(≥3)	65.56%	57.94%	61.11%	1.5585	0.5945
(≥4)	62.22%	61.11%	61.57%	1.6000	0.6182
(≥5)	46.67%	69.84%	60.19%	1.5474	0.7636
(≥6)	35.56%	79.37%	61.11%	1.7231	0.8120
(≥7)	31.11%	79.37%	59.26%	1.5077	0.8680
(≥8)	30.00%	82.54%	60.65%	1.7182	0.8481
(≥9)	27.78%	84.92%	61.11%	1.8421	0.8505
(≥10)	14.44%	88.89%	57.87%	1.3000	0.9625
(≥11)	10.00%	88.89%	56.02%	0.9000	1.0125
(≥13)	10.00%	89.68%	56.48%	0.9692	1.0035
(≥14)	2.22%	97.62%	57.87%	0.9333	1.0016
(≥16)	2.22%	99.21%	58.80%	2.8000	0.9856
(≥17)	1.11%	100.00%	58.80%		0.9889
(>17)	0.00%	100.00%	58.33%		1.0000

	ROC		Asymptotic	
Obs	Area	Std. Err.	[95% Conf. Interval]	
216	0.6312	0.0374	0.55792	0.70443



Abstract 248 Figure 1 ROC Curve

249 SPLENOMEGALY AND ANTI-NUCLEAR ANTIBODIES ARE DRIVEN BY INTERFERON- STIMULATION OF B CELLS IN LUPUS-LIKE DISEASE

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10.1136/lupus-2019-lsm.249

Background Systemic Lupus Erythematous (SLE) is an autoimmune disease of unknown etiology affecting 5 million people worldwide. It is known that 50%–70% of lupus patients present with an interferon-alpha (IFN-) gene signature, and it has been shown in multiple mouse models that lupus-like disease can be abolished by IFN- receptor (IFNAR) gene deficiency. Furthermore, disease can be halted by ablating the main producers of IFN-, the plasmacytoid dendritic cells. Thus, IFN- likely has a causative role in lupus-like disease.