Results: Out of 4,502 patients included in this study, 135 had a VTE event. After univariate analysis and Lasso regression, the following 11 variables were identified and included in the risk prediction model: male sex, age, BMI ≥ 25 kg/m², hyperlipidemia, hypoalbuminemia, hsCRP > 3 mg/L, renal involvement, nervous system involvement, anti- $\beta 2$ -glycoprotein I antibody positivity, lupus anticoagulant positivity, and no use of hydroxychloroquine. The AUC for the SLE-VTE score (Table 1) was 0.947 (95% CI, 0.9249-0.9694). The SLE-VTE score's sensitivity and specificity with the optimal cutoff value of 13 were 0.919 and 0.881, respectively. The SLE-VTE score was superior to the GAPSS system in predicting the risk of VTE in patients with SLE (AUC= 0.947 vs. 0.680, $P < 0.001$; integrated discrimination improvement (IDI)= 0.6652, $P < 0.001$; net reclassification improvement (NRI)= 0.6652, $P < 0.001$).

Table 1. Final multivariable analysis for venous thromboembolism risk in patients with SLE

	β coefficients	Odds ratio* (95% CI)	P-value	Points in scoring system
Male	0.621	1.86 (0.953-3.503)	0.061	2
Age at study entry(≥ 50)	0.837	2.308 (1.339-3.915)	0.002	3
BMI02(kg/m ²)	0.792	2.209 (1.333-3.627)	0.002	3
Hyperlipemia	0.838	2.313 (1.246-4.166)	0.006	3
Hypoalbuminemia	2.163	8.697 (5.185-14.794)	< 0.001	7
hsCRP > 3 mg/L	1.452	4.272 (2.618-6.968)	< 0.001	5
Anti $\beta 2$ GPI	1.013	2.754 (1.543-4.853)	0.001	3
LA	1.559	4.752 (2.799-8.072)	< 0.001	5
Nervous system	2.382	10.832 (6.163-18.998)	< 0.001	8
Lupus nephritis	0.835	2.305 (1.414-3.756)	0.001	3
No use of hydroxychloroquine	1.771	5.876 (3.722-9.401)	< 0.001	6

BMI: body mass index; hsCRP: Hypersensitive c-reactive protein; ACL: anticardiolipin, anti- $\beta 2$ GPI: anti- $\beta 2$ -glycoprotein I, LA: lupus anticoagulant; Values in bold are statistically significant at $p < 0.05$.

Conclusion: Various factors are related to the occurrence of VTE in patients with SLE. The proposed SLE-VTE risk score can accurately predict the risk of VTE and help identify SLE patients with a high risk of VTE who may benefit from thromboprophylaxis.

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POS0755

PREVALENCE AND RELEVANCE OF ANTIBODIES AGAINST CITRULLINATED ALPHA ENOLASE (ANTI-CEP1) IN CONNECTIVE TISSUE DISEASES

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Background: Anti-citrullinated alpha enolase antibodies have been investigated in rheumatoid arthritis and associated with bone erosion and interstitial lung disease but little is known about their prevalence and role in connective tissue diseases (CTDs).

Objectives: The aim of this study was to investigate the prevalence and relevance of anti-CEP1 antibodies in CTDs.

Methods: Serum samples from five independent patient cohorts were assessed: 1) established (est) primary Sjogren's syndrome (pSS) N=78, 2) est-systemic lupus erythematosus (SLE) N=52, 3) est-systemic sclerosis (SSc) N=71, 4) pSS at disease onset N=30, 5) SLE at disease onset N=46 (cohorts 4 and 5 had at least 3 years of follow-up). Samples from ninety sex and age matched healthy donors (HD) and 200 patients with est-RA (disease controls) were also tested. Anti-CEP1 IgG antibodies were measured with a commercially available ELISA kit (Euroimmun, Luebeck, Germany).

Results: Anti-CEP1 titer was significantly higher in est-pSS, est-SLE and est-SSc compared to HD, significantly lower in est-pSS and est-SSc compared to est-RA and comparable in est-SLE versus est-RA. We divided patients in every CTD group based on whether their anti-CEP1 titer was below or above the 25th, 50th and 75th percentile. In est-SLE anti-CEP1 values over the 25th percentile were associated with articular involvement (odds ratio, OR (95% confidence interval, CI)=11.5; 1.9-70.6, $p=0.008$). In est-pSS, no relationship between anti-CEP1 $> 25^{\text{th}}$ percentile and articular involvement was found but rather an association with rheumatoid factor positivity (OR (95% CI)=4.8, 1.6-14.1, $p=0.004$) and salivary gland swelling (OR (95% CI)=6.2, 1.3-29.1, $p=0.021$). In est-SSc no difference could be detected across the 3 groups. Anti-CEP1 titers in pSS and SLE at onset did not differ from each other, were comparable also to those of HD and significantly lower than those of est-pSS, est-SLE and est-RA patients (all $p < 0.0001$). Of interest, we could retrieve a serum sample collected at the time of diagnosis for 5 patients from the cohort of established pSS and we observed that anti-CEP1 titers were significantly lower at pSS onset than during follow up (at least 12 months after the diagnosis, $p=0.0024$). No difference was observed in the clinical presentation at disease onset according to different anti-CEP1 titer and they did not predict the development of new clinical manifestations during follow-up.

Conclusion: Anti-CEP-1 antibodies can be detected in CTDs at different title during the disease course and may increase overtime, at least in pSS. Although anti-CEP1 antibodies are associated with specific clinical manifestation in est-CTDs, such as articular involvement in est-SLE, they seem to lack a predictive value for future manifestations when measured at disease onset.

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POS0756

HOW HAS OUR MANAGEMENT OF SLE CHANGED OVER THE LAST THREE DECADES? REAL LIFE DATA FROM A MONOCENTRIC COHORT

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Background: Over the last decades new therapies have been approved and new treatment strategies have been proposed in the treatment of SLE. In particular, recently, EULAR recommendations¹ and the Treat to Target² approach established a new paradigm for the treatment of SLE based on the early introduction of immunosuppressive treatment and glucocorticoids (GC) minimization.