positive) and in 34 – in CMA. IgM- α 2GP1 was combined with IgA- α 2GP1 with the same frequency in both methods (in 13 patients).

IgA- α 2GP1 was detected in 30 (48%) of 63 patients. They were combined with both IgG-aCL and IgG- α 2GP1 in all cases in both methods. IgM-aCL and IgM- α 2GP1 were detected in 14 and 11 of 30 patients with IgA- α 2GP1, respectively. The combination of IgA- α 2GP1 with IgG-aCL by ELISA was in 27 (in most cases highly positive – 20) and with IgM-aCL – in 10 (highly positive – 8). IgG- α 2GP1 was detected in 28 patients with IgA- α 2GP1 (high positive – 21) and in 11 patients with IgM- α 2GP1 (high positive – 7).

IgG- α 2GP1-D1 was revealed in 48 (76%) patients. It was combined with IgG-aCL – in 38, with IgM-aCL – in 15 patients by the ELISA. The combination of IgG- α 2GP1-D1 by CMA was as follows: with IgG-aCL – in 46, with IgM-aCL – in 17, and with IgA-aCL – in 33 patients. In most cases, IgG- α 2GP1-D1 was combined with highly positive aCL levels. IgG- α 2GP1-D1 positivity was associated with IgG- α 2GP1 positivity in 42 – by ELISA and 47 – by CMA, IgM- α 2GP1 – in 13 and 14 patients by ELISA and CMA, respectively, and IgA- α 2GP1 – in 29. Isolated IgG- α 2GP1-D1 positivity was not observed.

Conclusion: The frequency of IgA-aCL detection was 56% (35 patients out of 63), IgA- α 2GP1 – 48% (30 patients out of 63), IgG- α 2GP1-D1 – 76% (48 patients out of 63). There was not isolated positivity of this “extra” criterial antibodies. The presence of IgA-aCL, IgA- α 2GP1, IgG- α 2GP1-D1 was associated with highly positivity of IgG/IgM-aCL and IgG/IgM- α 2GP1.

Disclosure of Interests: None declared

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POS0713

PREDICTORS OF HOSPITALIZATION IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: A 10-YEAR COHORT STUDY OF 398 PATIENTS FROM A TERTIARY CENTRE

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Background: Patients with systemic lupus erythematosus (SLE) often require hospitalization. The cause of admission may vary, but active disease and infection are consistently reported as the main reasons for hospitalization and are associated with worse survival and damage accrual. Recent improvements in the standard of care, including minimization of glucocorticoid dose and more effective and safe immunosuppressive regimens, may have changed the incidence and risk factors for hospitalization due to these causes. Hence, it is useful to identify predictors of hospitalization to further reduce the risk of admission for disease activity and severe infection in patients with SLE.

Objectives: To identify predictors of hospitalization in patients with SLE, according to the underlying cause.

Methods: Patients with SLE fulfilling classification criteria (ACR'97 and/or SLICC), regularly followed at an academic lupus clinic from January 2009 to December 2020 and with at least two outpatient visits were included in this cohort study. Time to first hospitalization up to 120 months was identified separately for the following admission causes: (a) any cause; (b) active SLE; (c) infection. Predictors of hospitalization were sought through survival analysis, with distinct models for each of the major admission causes. Univariate analysis was performed using Kaplan-Meier curves and Log-Rank tests. Tested variables assessed at baseline included: gender; age at SLE onset; age; disease duration; SLE Disease Activity Index (SLEDAI-2K) score; ongoing antimalarial use; ongoing immunosuppressants; ongoing prednisolone daily dose; lupus nephritis up to baseline; SLICC Damage Index (SDI) score. Variables with $p < 0.1$ were further tested in multivariate Cox regression models. Hazard ratios (HR) were determined with 95% confidence intervals (95%CI).

Results: We included 398 patients (female: 86.2%, mean age: 41.2 ± 15.1 years, mean disease duration: 10.1 ± 9.2 years; previous lupus nephritis: 28.9%; mean SLEDAI-2K score: 3.4 ± 2.7 ; ongoing antimalarials: 78.9%; ongoing immunosuppressant: 29.9%; ongoing prednisolone > 7.5 mg/day: 17.1%; SDI score ≥ 1 : 28.4%). During the follow-up period, 50.5%, 23.6% and 17.3% were hospitalized at least once for any cause, active SLE or infection, respectively.

In the multivariate model, significant baseline predictors for hospitalization due to active disease were (table 1): SLEDAI-2K score > 5 ; disease duration ≤ 2 years; ongoing immunosuppressants; SDI score ≥ 1 . Baseline independent predictors of

hospitalization for infection included (table 1): male gender; SDI score ≥ 1 ; ongoing antimalarials were protective.

Table 1. Predictors of hospitalization in multivariate Cox regression according to the admission cause

Predictors	Hospitalization for active SLE	Hospitalization for infection
SLEDAI-2K score > 5	2.43 (1.53-3.88)	n.s.
SLE duration ≤ 2 years	1.70 (1.04-2.77)	n.s.
Ongoing immunosuppressant	1.91 (1.24-2.95)	n.s.
SDI score ≥ 1	1.82 (1.16-2.86)	2.14 (1.33-3.45)
Male gender	n.s.	2.19 (1.23-3.89)
No antimalarial treatment	n.s.	2.20 (1.34-3.60)

Risk for each predictor reported as Hazard Ratio (95% Confidence Interval); n.s.: non-significant

Conclusion: Tight control of disease activity, prevention of damage accrual, and treatment with antimalarials may contribute to minimize the risk of hospitalization for these two major causes of admission in patients with SLE.

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POS0714

WHITE MATTER HYPERINTENSITIES LEAD TO REDUCED PSYCHOMOTOR SPEED IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS AND NEUROPSYCHIATRIC SYMPTOMS

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Background: Cognitive impairment is common in patients with systemic lupus erythematosus (SLE) and neuropsychiatric (NP) symptoms, but the exact underlying structural brain correlates are unknown.

Objectives: We aimed to compare cognitive function between groups of patients with different phenotypes of (NP)SLE and assessed the association between brain volumes, white matter hyperintensity (WMH) volume and cognitive function.

Methods: Patients who visited the NPSLE clinic of the Leiden University Medical Center between 2007-2015 were included in this study ($n=151$; 42 ± 13 years, 91% female). In a multidisciplinary consensus meeting, phenotypes were established and neuropsychiatric symptoms were attributed to SLE (NPSLE, inflammatory ($n=24$) or ischemic ($n=12$)) or to minor involvement of SLE or other causes (minor/non-NPSLE ($n=115$)). All patients underwent standardized cognitive assessment of the four cognitive domains: global cognitive functioning (GCF), learning and memory (LM), executive functioning and complex attention (EFCA) and psychomotor speed (PS)). Cognitive dysfunction was defined as a T-score < 40 , with age, sex, gender and education matched individuals of the Dutch population as reference. In addition, automated volume measurements on brain MRI (white matter, grey matter, white matter hyperintensities (WMH) and total brain volume (TBV)) were performed. Patients with brain infarcts > 1.5 cm were excluded. Cognitive function (Z-score) was compared between different NPSLE phenotypes using multiple regression analyses corrected for age, sex and education. Associations between brain volumes, WMH and cognitive function were assessed per phenotype using multiple regression analyses corrected for age, sex and intracranial volume.

Results: Global cognitive functioning was impaired in 5%, learning & memory in 46%, and executive functioning & complex attention in 39% and psychomotor speed in 46% of all patients. Patients with inflammatory NPSLE showed the most cognitive impairment and reduced cognitive function compared to ischemic NPSLE (all domains) and minor/non-NPSLE (EFCA) ($p < 0.05$).

Lower total brain volume and grey matter volume were associated with lower cognitive functioning in all domains (β : 0.00/0.01 (0.00; 0.01)) and lower white matter volume associated with lower LM, EFCA and PS (β : 0.00/0.01 (0.00; 0.01)) in all patients. Higher WMH volume associated with lower psychomotor speed (β : -0.14 (-0.32; -0.02)), especially in patients with inflammatory NPSLE (β : -0.36 (-0.60; -0.12)).

Conclusion: Reduced brain volume leads to reduced cognitive function in multiple cognitive domains in all patients with SLE and neuropsychiatric symptoms. Increased WMH volume leads to reduced psychomotor speed, especially in patients with inflammatory NPSLE.