months of SLE onset were evaluated with yearly visits to update co-morbidities, pregnancy status, and medications. Study visits with a current pregnancy were assessed for aspirin use and preeclampsia risk factors. Aspirin use was compared over time and among those with and without traditional risk factors for preeclampsia (i.e. hypertension, renal disease, diabetes, nulliparity, BMI≥35, age >40), as well as known disease-specific risk factors (i.e. antiphospholipid antibodies [+aPL], nephritis).

Results: We identified 297 women who had 479 pregnancies over the study period. Mean age during pregnancy was 31 (SD 4.9) years and 30% were nulliparous. Half of the pregnancies experienced ≥1 traditional preeclampsia risk factors in addition to SLE, while a third had +aPL. We observed aspirin use in 121/475 25% of pregnancies (95% CI 22,29) versus 22% (95%CI 19,25) of visits before and after pregnancy among the same women. Aspirin use was similar among pregnancies with and without ≥1 traditional risk factor for preeclampsia [25% (95%CI 20,31) versus 26% (95%CI 21,32)], while we observed a higher prevalence of aspirin use in those with +aPL (38%, 95% CI 24,55) versus those without (23%, 95% CI 15,34). There was a significant difference in aspirin use based on maternal race/ethnicity, with 67/205 (33%, 95% CI 26,39) aspirin use in Caucasians versus 9/88 (10%, 95% CI 5,18) for black women. Prevalence of aspirin use in pregnancy varied across regions (12%>37%), and did not increase over time.

Abstract THU0334 – Table 1. Prevalence of preeclampsia (PE) risk factors among pregnant SLE visits and prevalence of ASA use among women with and without PE risk factors

Risk factor	Overall prevalence n=475 n (%)	Prevalence of ASA use (%, 95% CI)	
		With risk factor	Without risk factor
Age>40	14 (3)	2/14 (14%, 4–40)	119/461 (26%, 22–30)
BMI≥35	33/437[a]	8/33 (24%, 13–41)	113/404 (28%, 24–33)
Nulliparous	136/461[30]	37/136 (27%, 20–35)	80/325 (25%, 20–30)
Renal disease	83 (17)	17/83 (20%, 13–30)	104/392 (27%, 22–31)
Nephritis	53 (11)	11/53 (21%, 12–23)	109/422 (26%, 22–30)
Hypertension	79 (17)	24/79 (30%, 21–41)	97/396 (24%, 21–29)
Diabetes	2 (0)	0/2 (0%, 0–1)	121/473 (26%, 22–30)
≥1 traditional PE risk factor	234 (49)	58/234 (25%, 20–31)	63/241 (26%, 21–32)
aPL +	34/104[33]	13/34 (38%, 24–55)	16/70 (23%, 15–34)

Conclusions: In this cohort including 479 SLE pregnancies, most pregnant women were not on aspirin and half had preeclampsia risk factors in addition to SLE. It is possible that aspirin was introduced at/or following the study visit when the pregnancy was documented, highlighting the importance of the treating rheumatologist in reviewing aspirin use and initiating it in pregnant SLE women. Our findings suggest black SLE women as a potentially vulnerable group during pregnancy, having the lowest prevalence of aspirin use.

Disclosure of Interest: None declared

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THU0335 FACTORS ASSOCIATED WITH DEVELOPMENT AND MORTALITY OF PULMONARY HYPERTENSION IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS

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Background: Pulmonary hypertension (PH) is a major cause of death in patients with systemic lupus erythematosus (SLE). In recent years, SLE with PH has become more common in the past few decades, and novel therapies has been developed to improve the prognosis of PH in SLE patients. Therefore, it is necessary to investigate further to identify serological and clinical factors for the development and mortality of PH in SLE patients.

Objectives: This study aims to estimate the prevalence of PH in SLE patients and identify the factors associated with the development of and mortality from PH in SLE patients.

Methods: We conducted a prospective study of SLE patients with fulfilling the American College of Rheumatology criteria (ACR) in a single tertiary centre from February 1998 to December 2013. PH was defined as a systolic pulmonary arterial pressure (sPAP) ≥30 mmHg at rest on transthoracic echocardiography (TTE). We assessed potential associated factors contributing to the development and mortality of PH in SLE patients using univariate and multivariable logistic regression models.

Results: Of 1110 patients with SLE, 48 patients were identified to have PH. Multivariable analysis indicated that pleuritis or pericarditis (odds ratio (OR)=4.62, 95% confidence interval (CI)=2.46 to 8.70, p<0.01), anti-RNP antibody (OR=2.42, 95% CI=1.21 to 4.82, p=0.01), interstitial lung disease (ILD) (OR=8.34, 95% CI=2.21 to 31.54, p<0.01), and cerebro-cardiovascular disease (OR=13.37, 95% CI=3.56 to 50.21, p<0.01) were independently associated with the development of PH in SLE. Subgroup analysis among patients with PH demonstrated that there were no statistically significant factors associated with PH mortality in SLE.

Abstract THU0335 – Table 1. Factors associated with pulmonary hypertension development in systemic lupus erythematosus

	Univariate		*Multivariable*	
	OR (95% CI)	*p-value*	*OR (95% CI)*	*p-value*
Sex: female	1.92 (0.46 to 8.07)	0.37	4.44 (0.54 to36.76)	0.17
Age at diagnosis of SLE	0.99 (0.97 to 1.02)	0.59	0.99 (0.96 to 1.02)	0.49
Disease duration[a]	1.00 (1.00 to 1.00)	0.87	1.00 (0.99 to 1.00)	0.48
Ever smoker	0.52 (0.18 to 1.47)	0.22		
Pleuritis or pericarditis	4.60 (2.55 to 8.29)	<0.01	4.62 (2.46 to 8.70)	<0.01
Raynaud phenomenon	1.48 (0.79 to 2.77)	0.22		
Anti-Sm	1.65 (0.81 to 3.39)	0.17	0.89 (0.38 to 2.11)	0.75
Anti-RNP	2.48 (1.36 to 4.51)	<0.01	2.42 (1.21 to 4.82)	0.01
ACL	1.10 (0.55 to 2.19)	0.79		
LAC	0.83 (0.32 to 2.12)	0.69		
SLEDAI score at enrollment	1.03 (0.96 to 1.09)	0.42		
ILD (pulmonary, pleural fibrosis)	9.56 (2.89 to 31.69)	<0.01	8.34 (2.21 to 31.54)	<0.01
Cerebro-cardiovascular disease	7.34 (2.30 to 23.41)	<0.01	13.37 (3.56 to 50.21)	<0.01

OR: odds ratio; CI: confidence interval; SLE: systemic lupus erythematosus; Anti-Sm: Anti-Smith; Anti-RNP: Anti-ribonucleoprotein; ILD: interstitial lung disease.

[a]From diagnosis of SLE to last follow-up.

Conclusions: The prevalence of PH was 4.3% in our cohort. There were significant associations with pleuritis or pericarditis, ILD, cerebro-cardiovascular disease, and anti-RNP antibody in SLE, which may contribute to the development of PH. However, there were no statistically significant factors correlating PH mortality in SLE.

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THU0336 PREDICTORS OF SUBCLINICAL CAROTID ATHEROMATOSIS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: RELEVANCE OF ACTIVITY, DAMAGE AND SEVERITY INDEXES

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Background: The prevalence of subclinical atheromatosis in patients with systemic lupus erythematosus (SLE) is double that the observed in the general population. The mechanisms of this accelerated atherosclerosis are unknown, but they may include factors related to the disease, as well as the interaction of these with classic cardiovascular risk factors (CVRF).

Objectives: We analyse wich predictors of subclinical carotid atheromatosis exist in a large series of patients with SLE, with special emphasis on the role of the activity, damage and severity indexes.

Methods: Cross-sectional study that included 276 patients with SLE. Lipid serum levels, autoimmunity profile and the activity (SLEDAI), severity (Katz) and damage (SLICC) indexes were determined. The thickness of the carotid intima-media (cIMT) and the presence of plaques were determined by radiofrequency US. The cardiovascular risk was estimated through SCORE. Multivariate regression analysis was performed to assess the relationship of the indexes with CVRF, cIMT and

carotid plaque. Predictive capacity for the presence of plaque by comparing the AUC of the different models was performed through the DeLong method

Results: 106 (38%), 85 (31%), 47 (17%) and 21 (8%) patients showed respectively SLEDAI null, low, moderate and high, 197 (71%) had a SLICC >1 and 104 (38%) a Katz>3. In 36% (99) of the patients carotid plaques were detected, with an average cIMT of 0.631±0.108 mm. SLEDAI showed a positive relationship with hypertension; the Katz with hypertension and dyslipidemia; and the SLICC with these and also with age, body mass index and abdominal waist. The relationship of the latter with the CVRF was maintained after subtracting its items related to cardiovascular risk. SLICC was univariatelly related to plaque (OR 1.29 [95% CI 1.13–1.48], p=0.000) and a SLICC >1 showed a tendency to be associated with a higher cIMT (beta coefficient 0.03 [95% CI 0.00–0.06], p=0.053). No univariate relationships were found between Katz or SLEDAI with subclinical atheromatosis. The relationship of SLICC with plaque was maintained after adjusting for age, sex and CVRF (OR 1.19 [95% CI 1.00–1.42], p=0.047). Similarly, SLICC (even without its vascular damage items) (beta coefficient 0.26 [95% CI 0.12–0.41], p=0.000), but not Katz and SLEDAI, correlated significantly with SCORE. The predictive capacity of SCORE for the presence of plaque was AUC 0.788 (95% CI 0.735–0.842). Analogously SLICC showed an AUC 0.659 (95% CI 0.594–0.724) for plaque; the AUC of Katz and SLEDAI did not reach statistical significance. The AUC of the SCORE +SLICC versus SCORE did not show statistically significant differences (p=0.31). The statistical significance of the reclassification indexes were net reclasification index p=0.61, and integrated discrimination improvement p=0.01.

Conclusions: SLICC is independently related to the presence of plaque. SLE activity, severity and damage indexes are related to CVRF but they have little impact on the predictive capacity of SCORE for the presence of carotid plaque.

Disclosure of Interest: None declared

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THU0337 NONBACTERIAL THROMBOTIC ENDOCARDITIS (NBTE) IN SLE: PREVALENCE, CLINICAL CHARACTERISTICS AND SEROLOGICAL PROFILE

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Background: SLE is characterised by excessive production of various autoantibodies and correlation of these antibodies with organ involvement may help to evaluate disease severity and long term prognosis. NBTE is a rare cardiac manifestation of SLE with prevalence rate varying from 6%–11%. Many, but no all, studies have shown association of NBTE with anti phospholipid antibodies, but, except this association, data regarding clinical, laboratory and serological characteristics of NBTE is sketchy. We designed this study to evauate profile of patients having NBTE in SLE.

Objectives: 1. To study the prevalenence of NBTE in SLE patients.

2. To study association of NBTE with clinical and laboratory characteristics and serological profile.

Methods: All consecutive SLE inpatients and outpatients attending the department of Rheumatology from September 2015 to December 2017 were enrolled. Patients subjected to 2D Echo were included and their demographic, clinical, laboratory and serological profile were recorded. Serological profile was studied with Blue diver kit which is an immunodot blot assay measuring autoantibodies against 25 ENA. Anti cardiolipin and anti beta 2 glycoprotein antibody were tested by ELISA. Study was approved by an independent ethics committee [ECR/282].

Results: Total number of patients enrolled in study were **355** out of which **213** had undergone 2DEcho. NBTE was found in **33 (15.49%)** patients. Among all autoantibodies studied, we found that the presence of **anti-Nucleosome antibody, LAC, ACL and B2GP1** were significantly associated with NBTE (p<0.05). **Myocarditis, valvular** lesions and **Pulmonary Hypertension** were more common in NBTE group (p value: 0.012,<0.0001 and 0.013 respectively).

We also noticed that there is a statistically significant association between presence of NBTE with **APLA syndrome** and **Thrombotic events** (p value<0.0001 and 0.005 respectively).

Tab.1 Significant Serological association of SLE patients with NBTE.

Antibodies	SLE With NBTE-33 (15.49%)	SLE without NBTE-180	P value
Anti-nucleosome	27 (81.8)	32 (17.7)	<0.0001
LAC	16/30 (53.3)	31/149 (20.8)	0.0002
ACL (Ig M and IgG)	9/30[30]	13/128 (10.1)	0.004
B2GP-1(Ig M and IgG)	6/22[20]	9/103 (8.73)	0.033

APLA profile was available in 30 patients of NBTE and 147 patients not having NBTE. Out of this, positivity for APLA antibodies were seen in 17 (56.6%) and 36 (24.4%) patients respectively [p:0.005]. 82.3% patients with Anti phospholipid antibodies had APLA syndrome in NBTE group while in NBTE group 48.5% patients having Anti phospholipid antibodies had APLA syndrome. Thus, presence of NBTE increased the possibility of developing APLA syndrome in patients having positive serology for anti phospholipid antibodies

Tab.2 Significant Clinical and laboratory characteristic of SLE patients with NBTE.

Organ involvement	SLE with NBTE-33	SLE without NBTE-180	p- value
Myocarditis	11	27	0.012
Valvulopathy	10	4	<0.0001
PAH	9	21	0.013
Thrombotic events	8	16	0.005
APLA syndrome	14	17	<0.0001

Conclusions: Presence of Anti nucleosome antibody, LAC, Anti cardiolipin and anti beta 2 glycoprotein antibodies may predict presence or future development of NBTE in SLE patients. Presence of NBTE increases probability of developing APLA syndrome in patients with anti phospholipid antibodies. We have found association of NBTE with myocarditis, valvulopathy and PAH and thus propose that such patients with NBTE should be treated early and aggressively.

Disclosure of Interest: None declared

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THU0338 OUTCOME OF STROKE IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS: A NESTED CASE-CONTROL STUDY

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Objectives: To evaluate the outcome of stroke in patients with systemic lupus erythematosus (SLE) in comparison with matched non-SLE patients.

Methods: Patients who fulfilled ≥4 ACR criteria for SLE and had a history of stroke were identified from our SLE database. The outcome of stroke in these patients was evaluated retrospectively and compared with a group of randomly selected age/gender-matched non-SLE patients (in a 1:3 ratio) admitted to our stroke unit within the same time period. The type and extent of stroke, atherosclerotic risk factors (hypertension, smoking, diabetes mellitus, dyslipidemia, atrial fibrillation, valvular lesions) and previous stroke were compared between the two groups of patients. The primary outcome of interest was the 90 day functional outcome as assessed by the modified Rankin scale (mRS) (score 0–2: functional independence; score 3–6: functional dependence). Secondary outcomes included all-cause mortality, 30 day stroke mortality, stroke recurrence and stroke complications. Factors independently associated with a poor functional outcome was studied by logistic regression.

Results: A total of 40 SLE patients (age 53.7±11.5, 88% women) with stroke were identified from our database (stroke prevalence 0.39/100 patient-year). A control group of 120 non-SLE patients (age 52.8±14.8, 87.5% women) with stroke were randomly selected from our stroke database. All were ethnic Chinese. The prevalence of atherosclerotic risk factors was similar between the two groups, except SLE patients had a higher atherogenic index (Log serum [triglyceride/HDL-cholesterol] (1.51±0.47 vs 1.32±0.31; p=0.005.) In SLE patients, the median time to stroke since diagnosis was 24 months. Ischaemic stroke was more common in SLE than non-SLE patients (90% vs 63%; p=0.001). Among patients with ischaemic stroke, SLE patients had more extensive infarction than controls on CAT scan (multiple infarct 65.7% vs 18.7%; p<0.001). The 90 day mRS score was significantly higher in SLE patients than controls (1.70±1.97 vs 0.88±1.36; p=0.004). Significantly more SLE patients had functional dependence (mRS score 3–6) at 90 days post-stroke than controls (32.5 vs 8.3%; p<0.001). Logistic regression showed that SLE was an independent risk factor for a poor stroke outcome after adjustment for age, sex, history of stroke, various atherosclerotic risk factors and the type of stroke (ischaemic vs haemorrhagic) (OR 12.2 [2.97–49.9]). Subgroup analysis of patients with ischaemic stroke showed that SLE was also independently associated with a poorer functional outcome after adjustment for the same confounding covariates and the extent of stroke (solitary vs multiple infarcts) (OR 12.4 [1.02–150]; p=0.048). There was no significant difference in the 30 day stroke mortality between SLE and non-SLE patients (5% vs 2.5%; p=0.43). However, SLE patients had a higher incidence of post-stroke epilepsy than controls (22.5% vs 3.3%; p=0.001). Upon a mean follow-up time of 7.5±5.2 years, SLE patients had a lower stroke recurrence free survival (59.5% vs 85.7%; p<0.001) and a higher rate of all-cause mortality (34.6% vs 15.1%;p<0.001).

Conclusions: Stroke in SLE patients is more likely to be ischaemic in origin and more extensive than matched controls. Short-term functional outcome of stroke is poorer in SLE patients. Over 7.5 years, stroke recurrence, post-stroke epilepsy and all-cause mortality is significantly more frequent in SLE than non-SLE patients.

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