Objectives: To assess the extent of structural joint involvement in proximal interphalangeal joints (PIP), hand and wrist: E, BME, S, TS and peritendinitis (PT) by MRI, in patients diagnosed with SLE with hand arthritis or arthralgia.

Methods: All patients with SLE who manifested hand pain and/or swelling in the prior 6 months were consecutively included in the study. They were divided into two groups: arthritis or arthralgia, according to the physical examination by an expert rheumatologist. All patients underwent an MRI with contrast injection on their non-dominant hand. The images obtained were evaluated following RAM-RIS criteria extended to PIP and Tenosynovitis score for RA by two expert musculoskeletal radiologists, blind to the groups.

Results: 32 patients were included: arthritis: n = 13, arthralgia: n = 19, with a mean age of 50.91 ± 13.37 years and a disease evolution time of 10.21 ± 8.26 years. The average SLEDAI score 6.30 ± 3.40 for the arthritis groups and 3.79 ± 2.14 for the arthralgia group. The average SLICC score was 0.23 ± 0.42 in the arthritis group and 0.1 ± 0.31 in the arthralgia group. E was found in 7 patients with arthritis (53.84%) (15.38% in PIP, 0% in hand and 53.84% in wrist) and in 13 patients with arthralgia (68.42%) (0% in PIP, 10.52% in hand and 68.42% in wrist). BME was observed in 4 patients with arthritis (30.76%) (7.69% in PIP, 0% in hand and 30.76% in wrist) and in 5 patients with arthralgia (26.31%) (5.26% in PIP, 10, 52% in hand and 15.78% in wrist). S was observed in 12 patients with arthritis (92.30%) (61.58 in PIP, 76.92% in hand and 84.61% in wrist) and in 8 patients with arthralgia (42.10%) (31.57% in PIP, 36.84% in hand and 36.84% in wrist). TS was observed in 6 patients with arthritis (46.15%) (38.46% in flexor tendons and 23.07% in extensor tendons) and in 8 patients with arthralgia (42.10%) (31.57% in flexor tendons and 21.05 % in extensor tendons). PT was found in 6 patients with arthritis (23.07%) and in no patient with arthralgia (0%).

Conclusion: MRI allows us to diagnose musculoskeletal involvement in SLE, morphologically similar to rheumatoid arthritis (erosion, bone marrow edema, synovitis and tenosynovitis), which usually are underestimated on plain radiography. This study also shows the important erosive burden of arthritis in SLE, which has not been well characterised yet. In addition, it demonstrates the underestimation of physical examination to diagnose active inflammatory damage, such as subclinical synovitis, in patients with inflammatory arthralgias.

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FRI0163 ANTIPHOSPHOLIPID PATTERNS PREDICT THE RISK OF THROMBOSIS IN SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: It is well known that Lupus anticoagulant (LAC) positivity is more strongly associated with both arterial and venous thrombosis than either anticardiolipin (aCL) or anti- β 2glycoprotein (aB2GPI) antibodies (1). An unanswered question is the contribution of which combinations of positive aPLs to thrombosis risk.

Objectives: We aimed to evaluate which aPL combinations increase the risk of future thrombosis in patients with Systemic Lupus Erythematosus (SLE).

Methods: This prospective analysis included SLE patients who had been tested for all 3 aPLs in Hopkins Lupus Cohort. We constructed a dataset with one record per month of follow up for each patient. Rates of thrombosis for each aPL or each combination of aPL were calculated as the number of thromboses divided by the number of person months at risk and the results are converted to rates per 1000 person-years.

Results: There were 805 patients with a complete profile of 7 aPLs with a total of 73417 person months of follow up. LAC was the most predictive of any [3.47 (1.96, 6.14) $p < 0.0001$], venous [4.3 (1.99, 9.25) $p = 0.0002$], and arterial [3.37 (1.51, 7.53) $p = 0.0029$] thrombosis. In individual models, aB2GPI positivity was a significant risk factor for any [1.90 (1.16, 3.13) $p = 0.0113$] and venous [2.3(1.23, 4.61) $p = 0.0103$] thrombosis. When we

looked at patients who were LAC positive, and asked if having another positive aPL increase the risk ratio for any/venous/arterial thrombosis, we found that having aB2GPI IgA appeared to add significant risk to any [1.68 (1.01, 2.79) $p = 0.044$], and venous [2.01 (1.02, 3.97) $p = 0.043$] thrombosis among those with or without LAC (Table 1).

Table 1. Additive effect of other aPLs adjusting for LAC

	ANY thrombosis		VENOUS thrombosis		ARTERIAL thrombosis	
	age adjusted RR 95%(CI)	P-value	age adjusted RR 95%(CI)	p-value	age adjusted RR 95%(CI)	p-value
Model 1: LAC + aCL-G						
LAC (+) vs (-)	3.78 (2.08, 6.85)	<.0001	5.03 (2.25, 11.24)	<.0001	3.32 (1.43, 7.73)	0.0053
aCL-G (+) vs (-)	0.64 (0.22, 1.83)	0.4045	0.48 (0.11, 2.14)	0.3351	1.09 (0.31, 3.77)	0.8962
Model 2: LAC + aCL-M						
LAC (+) vs (-)	3.82 (2.13, 6.87)	<0.0001	4.57 (2.07, 10.08)	0.0002	3.69 (1.61, 8.46)	0.0021
aCL-M (+) vs (-)	0.51 (0.16, 1.65)	0.2576	0.68 (0.16, 2.93)	0.6019	0.61 (0.14, 2.64)	0.5075
Model 3: LAC+ aCL-A						
LAC (+) vs (-)	3.34 (1.88, 5.96)	0.0001	4.14 (1.9, 9)	0.0003	3.29 (1.47, 7.38)	0.0038
aCL-A (+) vs (-)	2.33 (0.56, 9.70)	0.2459	2.44 (0.32, 18.4)	0.3858	2.2 (0.29, 16.49)	0.4415
Model 4: LAC+ aB2GPI-G						
LAC (+) vs (-)	3.52 (1.94, 6.39)	0.0001	4.21 (1.85, 9.57)	0.0006	3.4 (1.48, 7.79)	0.0038
aB2GPI-G (+) vs (-)	0.91 (0.32, 2.61)	0.8655	1.1 (0.31, 3.88)	0.8849	0.96 (0.22, 4.18)	0.957
Model 5: LAC+ aB2GPI-M						
LAC (+) vs (-)	3.53 (1.97, 6.31)	0.0021	4.17 (1.9, 9.14)	0.0004	3.58 (1.58, 8.1)	0.0023
aB2GPI-M (+) vs (-)	0.91 (0.49, 1.73)	0.7782	1.17 (0.51, 2.68)	0.7101	0.74 (0.29, 1.9)	0.5285
Model 6: LAC+ aB2GPI-A						
LAC (+) vs (-)	3.16 (1.77, 5.65)	0.0001	3.68 (1.68, 8.08)	0.0012	3.19 (1.41, 7.2)	0.0052
aB2GPI-A (+) vs (-)	1.68 (1.01, 2.79)	0.0441	2.01 (1.02, 3.97)	0.0434	1.4 (0.67, 2.91)	0.3738

Conclusion: Our study shows that LAC is still the best predictor of risk of any, arterial and venous thrombosis in SLE. Moreover, aB2GPI IgA positivity appeared to add also a significant risk to any and venous thrombosis. Therefore, the clinical significance of IgA anti- β 2GPI deserves further investigation in SLE patients.

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FRI0164 BANFF INFLAMMATORY INDICES MAY BE SUPERIOR TO THE NIH SCORING IN PREDICTING CKD PROGRESSION IN LUPUS NEPHRITIS

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Background: Chronic kidney disease/end stage renal disease (CKD/ESRD) from lupus nephritis (LN) is a major cause of morbidity and mortality.

Advanced tubulointerstitial disease (TID) in LN is a better predictor of renal outcome than glomerular lesions. The current NIH classification is heavily weighted towards glomerular lesions and only provides a semiquantitative assessment of TID. In contrast, Banff classification of renal allograft pathology provides 6 reproducible scores for TID (inflammation, fibrosis, atrophy). Banff scoring may better predict CKD/ESRD in LN than NIH scores

Objectives: We compared Banff grading vs. NIH scoring as predictors of CKD progression at 5 years, defined as a decline in estimated glomerular filtration rate (eGFR) $\geq 30\%$, a strong risk factor for ESRD and mortality

Methods: We included patients with LN class III, IV, V on the index biopsy Jan 2005 and Dec 2018. H&E/PAS stained slides were reviewed and scored by an experienced pathologist. Six TID Banff scores (0/1 vs. 2/3), NIH activity/chronicity (AI/CI) and NIH interstitial fibrosis/tubular atrophy (IF/TA), tubulointerstitial inflammation (TII) scores (none/mild vs. moderate/severe) were evaluated as predictors of CKD progression using survival analyses

Results: Of the 125 patients, 46 had CKD progression and 20 subsequently developed ESRD. There were no differences between progressors and non-progressors in terms of baseline demographic, clinical data, LN class (Tab 1). Banff ti score (total inflammation) was associated with CKD progression in bivariate and time-dependent analyses. However, NIH TII score and corresponding Banff i score were not predictive (Tab 2, Fig 1). Overall NIH AI and CI were not predictive of CKD progression. Moderate/severe NIH IF/TA was associated with CKD progression as was Banff ci (interstitial fibrosis) score (Tab 2, Fig 2). Banff score for atrophy was not predictive. In a subset of 92 patients with baseline eGFR ≥ 60 ml/min/1.73m² only Banff ti score (but not i score or NIH TII, IF/TA) was predictive of CKD progression (Fig 1)

Table 1. Baseline data in patients with/without CKD progression

	Progressors ¹ n=46	Non-progressors n=79	p
n (%) or median (IQR)			
Female	36 (78)	69 (87)	0.18
Age (years)	26 (20-41)	29 (21-43)	0.53
<18 years	7 (15)	13 (16)	0.86
Race			0.82
White	4 (9)	4 (5)	
Black	18 (39)	35 (44)	
Asian	1 (2)	1 (1)	
Unknown	23 (50)	39 (50)	
Diabetes	5 (11)	3 (4)	0.12
Hypertension	33 (72)	55 (70)	0.8
eGFR ml/min/1.73m ²	96.1 (56.6-117.8)	88.9 (47-117)	0.51
C3mg/dL	71 (52-88)	67.5 (43-94)	0.58
C4mg/dL	12.65 (9-23)	12 (7-20)	0.48
Anti-ds DNA titer IU	108.7	151.3	0.57
	(36.6-194.9)	(39.2-200)	
Total SLEDAI	11 (8-16)	12 (8-16)	0.47
Renal SLEDAI	8 (4-12)	8 (4-12)	0.9
LN			
Proliferative GN ² : III, IV	18 (39)	30 (38)	0.15
Nonproliferative GN: V	9 (20)	27 (34)	
Mixed GN: V and III/IV	19 (41)	22 (28)	

¹ Progressors: LN patients with eGFR decline $\geq 30\%$ within 5 years. ² GN: glomerulonephritis

Table 2. NIH and Banff scores with/without progressors

	Progressors n=46	Non-progressors n=79	p
n (%) or median (IQR)			
NIH			
Overall AI	1 (0-4)	1 (0-3)	0.61
AI ≥ 11	1 (2)	3 (3.8)	0.62
Overall CI	3 (0-5)	2 (0-3)	0.33
CI ≥ 3	24 (52)	28 (35.4)	0.07
Moderate/severe TII	7 (15.2)	4 (5)	0.05
Moderate/severe IF/TA	16 (35)	15 (19)	0.049
Banff			
Tubulitis: t	0	0	
Interstitial inflammation: i 2/3	3 (6.5)	5 (6.3)	0.9
Total inflammation: ti 2/3	16 (34.8)	12 (15.2)	0.01
Tubular atrophy: ct 2/3	16 (34.8)	16 (20.3)	0.07
Interstitial fibrosis: ci 2/3	17 (37)	15 (19)	0.03
Inflammation in area of interstitial fibrosis and/or tubular atrophy: i-IF/TA 2/3	n=26*	n=51*	0.09
	18 (69)	25 (49)	

* Biopsy number is smaller due to inability to apply score to biopsies without areas of fibrosis/tubular atrophy

Figure 1. Association of NIH and Banff inflammation scores with ESRD progression

Figure 1A. NIH TII score in complete cohort (n=125)

Figure 1B. Banff ti score in complete cohort (n=125)

Figure 1C. NIH TII score in sub-cohort with baseline eGFR ≥ 60 ml/min/1.73m² (n=92)

Figure 1D. Banff ti score in sub-cohort with baseline eGFR ≥ 60 ml/min/1.73m² (n=92)

Figure 2. Association of NIH and Banff fibrosis scores with ESRD progression

Figure 2A. NIH IF/TA score in complete cohort (n=125)

Figure 2B. Banff ci score in complete cohort (n=125)

Figure 2C. NIH IF/TA score in sub-cohort with eGFR ≥ 60 ml/min/1.73m² (n=92)

Figure 2D. Banff ci score in sub-cohort with eGFR ≥ 60 ml/min/1.73m² (n=92)

Conclusion: Banff inflammation scores may be superior predictors of CKD/ESRD progression at 5 years, compared to the currently used NIH classification. Detection of inflammation by Banff scores may allow earlier interventions to prevent ESRD

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FRI0165

RISK OF CKD IN MEMBRANOUS AND PROLIFERATIVE LUPUS NEPHRITIS - ANALYSIS OF A NATIONWIDE MULTICENTRE COHORT

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Background: Lupus nephritis (LN) is one of the most severe manifestations of Systemic Lupus Erythematosus.

Objectives: 1) To compare proliferative (PLN), membranous (MLN) and mixed LN regarding clinical and laboratory presentation. 2) To investigate predictors of progression to chronic kidney disease (CKD).