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.