published.1 Extra-renal efficacy endpoints included: change from baseline in SLE Disease Activity Index-2000 (SLEDAI-S2K; excluding renal items); proportion of patients with SLEDAI-S2K organ system improvement (excluding renal involvement); proportion of patients with Systemic Lupus International Collaborating Clinics (SLICC)/American College of Rheumatology (ACR) Damage Index (SDI) worsening; and proportion of patients with prednisone-equivalent dose ≤7.5mg/day and ≤5 mg/day at Week 104.

Results Overall, 448 patients were randomised and received study treatment (safety population: n=224/treatment group; modified intention-to-treat [mITT] population: n=223/treatment group). In total, 91 (20.4%) patients withdrew from the study, mainly due to withdrawal of consent (10.1%). The mean (standard deviation [SD]) SLEDAI-S2K score at baseline was 12.3 (5.0) and mean (SD) SDI score was 0.4 (1.0). Most patients (86.1%) had immunologic SLEDAI-S2K involvement at baseline and 39.2% had mucocutaneous involvement (rash, n=119 [26.7%]; alopecia, n=101 [22.6%]; mucosal ulcers, n=29 [6.5%]). At Week 104, there was a significantly greater change from baseline in SLEDAI-S2K (excluding renal items) in the BEL group than in the PBO group (treatment difference vs PBO [95% confidence interval]: –0.4 [–0.8, 0.0]; p=0.0436; Table 1). Among patients with organ system involvement at baseline, at Week 104, greater proportions of BEL-treated patients versus PBO-treated patients had improvements from baseline in the immunologic, cardiovascular/respiratory and vascular SLEDAI-S2K domains, while improvements in mucocutaneous, musculoskeletal and hematologic domains favoured PBO (Table 1). At Week 104, numerically more PBO-treated patients (7.8%) than BEL-treated patients (6.5%) experienced SDI worsening while on treatment (p=0.7293; Table 1). At Week 104, significantly more BEL-treated patients versus PBO-treated patients were receiving average prednisone-equivalent doses of ≤7.5 and ≤5.0 mg/day (p=0.0139 and p=0.0444, respectively; Table 1).

Conclusions The 104-week BLISS-LN study demonstrated that some extra-renal outcomes and steroid dose reduction favoured BEL, compared with ST alone in patients with active LN.

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PO.5.107 REAL-WORLD CLINICAL RESPONSE OF TACROLIMUS TREATMENT IN PATIENTS WITH LUPUS NEPHRITIS

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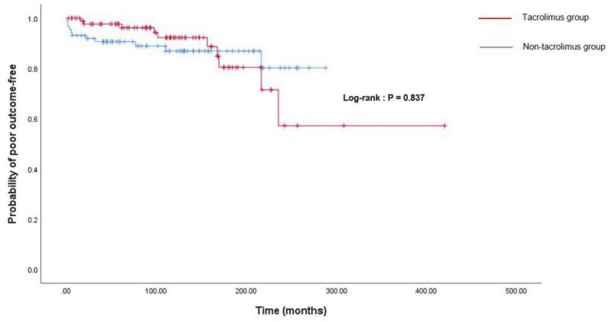
10.1136/lupus-2022-elm2022.130

Purpose Lupus nephritis (LN) is a severe manifestation of systemic lupus erythematosus (SLE), however, current treatment for LN is still associated with severe adverse effects, treatment failures, and relapse rates. Tacrolimus (TAC), a novel calcineurin inhibitor with immunosuppressive effects, has recently become increasingly interested in its role as a potential therapeutic agent in SLE. The aim of this study was to evaluate the efficacy of TAC as a treatment for LN.

Methods We retrospectively reviewed the medical records of patients with LN from January 1999 to December 2021.



Abstract PO.5.107 Figure 1 Kaplan-Meier plot of remaining free of poor-outcomes in patients with lupus nephritis

One-hundred seventy biopsy proven cases of LN were enrolled, with 92 in the TAC group and 87 in the non-TAC group. The clinical response of TAC treatment in patients with LN was evaluated by proteinuria, estimated glomerular filtration rate (eGFR), anti-double-stranded DNA (anti-dsDNA) antibody, complement 3 (C3), complement 4 (C4), and renal SLE disease activity index (SLEDAI). Complete renal response was defined as urine protein to creatinine ratio (UPCR) <0.5, normal serum creatinine or, if normal at baseline, not increased by ≥15%, and partial renal response was defined as a normal or near-normal GFR with a ≥ 50% reduction in proteinuria to sub-nephrotic levels. The poor outcomes were end stage renal disease or death.

Results The baseline clinical manifestations between the two groups showed no significant differences. Most of TAC group received combination therapy with other immunosuppressants, and only 19 (20.7%) patients maintained TAC monotherapy. After 5 years, there were no statistically significant differences in proteinuria, eGFR levels, anti-dsDNA, serum C3/C4, and renal SLEDAI. The overall (complete and partial) renal response rate was not significantly different: 72.9% of patients receiving TAC and 85.5% of patients not receiving TAC (p = 0.1). The poor outcomes were similar in both groups.

Conclusions Our results indicate that TAC is potentially effective in treating LN, and may be a reasonable option for patients with LN. TAC can help patients with LN achieve a renal response and slow progression.

PO.5.108 COMBINED MODEL OF RENAL HISTOPATHOLOGY AND CLINICAL PARAMETERS BETTER PREDICTS ONE-YEAR RENAL OUTCOMES IN LUPUS NEPHRITIS: ANALYSIS OF 334 KIDNEY BIOPSIES

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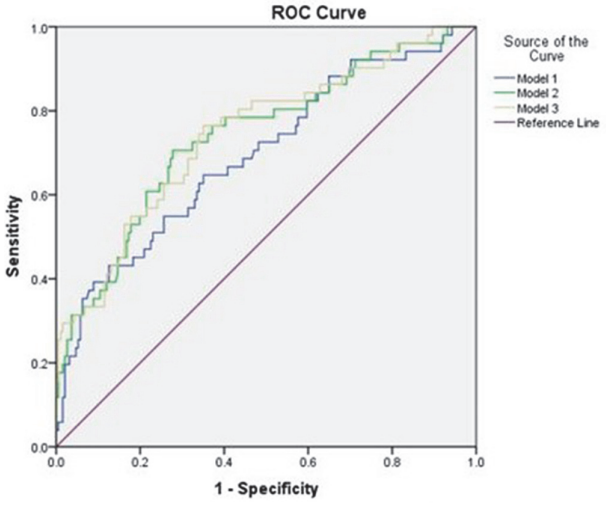
Background/Purpose Diagnosis of Lupus Nephritis (LN) is currently based on laboratory tests and renal histopathology. The role of histopathological features in determining long term outcomes is unclear. The objectives are to determine if clinical and biochemical parameters at baseline could identify renal histopathological class and to assess the clinico-histopathological predictors of renal response.

Methods This is a single centre retrospective study comprising 334 LN renal biopsies. Clinical and biochemical parameters at the time of biopsy were noted and their association with histopathological class, activity and chronicity scores (AS/CS) (ISN/RPS classification) were evaluated. Complete, partial or no response(CR, PR, NR) for the renal outcome (EULAR/EDTA) at 1 year were calculated for 293 patients. Binary logistic regression was done to look for the predictors of NR.

Results Class III/IV LN was seen in 240(71.8%). Hypertension was seen in (52.1%) of class III/IV and <25% each with class II, V and combined class(p<0.001). Class III/IV had lower eGFR [87.6(62.75–118.8)] (p<0.001) than the other classes. Nephrotic range proteinuria was seen in 32% of class V and 21% in class III/IV (p=0.004) Among class-III/IV AS had weak correlation with baseline UPCR (r=0.31) and eGFR (r=-0.172) (p<0.01). CS had weak negative correlation with eGFR (r=-0.212,p<0.01). NR at 1 year was higher in males (OR-4.6,95%CI-1.9–10.8,p<0.001), those with abnormal serum creatinine (OR-3.3,95%:CI1.6–7.02, p-0.001), higher renal SLEDAI (p<0.05), higher AS, CS (p<0.001), interstitial inflammation and tubular atrophy(p<0.005) (Table-1). On binary logistic regression a combined clinico-histopathological model comprising of serum creatinine, UPCR, male sex and CS performed best in predicting NR (figure 1).

Conclusion Clinical and biochemical parameters can predict the renal histological class to a fair extent but have limited value in predicting the activity and chronicity parameters. Since a combination of clinical and histopathology parameters are better in predicting renal outcomes, performing renal biopsies should be encouraged in LN.



Abstract PO.5.108 Figure 1 ROC curve and AUC for the three different models
Model 1: Baseline serum creatinine, urine PCR, male sex; AUC −0.694 (0.609–0.779), p <0.001
Model 2: Baseline serum creatinine, urine PCR, male sex, chronicity score; AUC −0.740(0.660–0.820), p<0.001
Model 3: Baseline serum creatinine, urine PCR, male sex, chronicitv score, crescents, interstitial inflammation;
AUC −0.744(0.664–0.824), p<0.001
AUC, Area Under Curve; ROC, Receiver-Operating Characteristics

Abstract PO.5.108 Table 1 Comparison of baseline characteristics among those who attained any response (CR/PR) versus others at one year

Parameter	Any response Complete Response CR/PR (n=233)	Others (No response/rescue) (n=60)	OR (95% CI)	P value
Female/male, n (%)	221(94.8)/12(5.2)	48(80)/12(20)	**4.6(1.9-10.8)**	**0.001**
Median age at nephritis onset	28(11-65)	25(13-67)		0.079
Median SLE duration	12(0-232)	18(0-144)		0.770
Hypertension, n (%)	100(42.9)	34(56.7)		0.061
Creatinine>1.3mg/dL (median, IQR)	21(9.0)	15(25)	**3.3(1.6-7.02)**	**0.001**
eGFR categories, n (%)			**1.7(0.96-3.03)**	**0.003**
>90	137(58.8)	27(45)		
61-90	57(24.5)	15(25)		
30-60	34(14.6)	9(15)		
<30	4(1.7)	8(13.3)		
Active urinary sediments, n (%)	132(56.7)	44(73.3)		**0.019**
uPCR g/day (median with IQR)	1.38(0.8-2.67)	1.95(1.18-4.19)		0.098
Class III/IV, n (%)	167(71.7)	49(81.7)		0.117
Class V, n (%)	17(7.3)	5(8.3)		0.788
Combined class, n (%)	7(3.0)	3(5.0)		0.469
Activity score, median with IQR	3(1-6)	6(3-9)		**0.001**
Chronicity score, median with IQR	0(0-1)	1(0-2)	**5.06(1.49-17.21)**	**0.001**
Presence of Crescents, no (%)	43(18.5)	17(28.3)		0.104
Fibrinoid necrosis, n (%)	28(12.0)	7(11.7)		0.791
Interstitial inflammation, n (%)	86(36.9)	33(55)	**2.08(1.17-3.70)**	**0.003**
Interstitial fibrosis, n (%)	23(10.7)	9(15)		0.273
Tubular atrophy, n (%)	64(27.5)	27(45)		**0.003**
Blood vessel changes, n (%)				
Fibrinoid necrosis	2(0.9)	1(1.7)		0.606
Other changes*	206(88.4)	50(83.3)		0.339

PO.5.110 PLASMA LEVELS OF OSTEOPONTIN IN SLE

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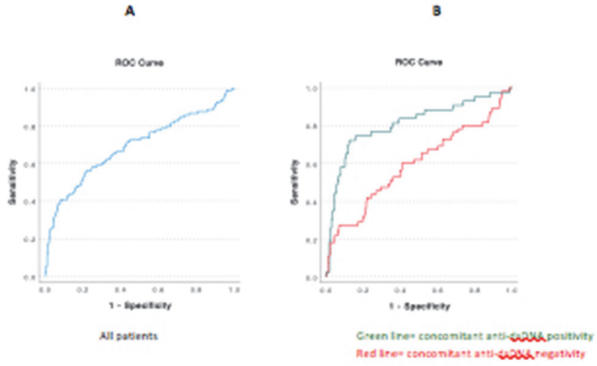
Purpose Predicting flares and disease progression, including the development of lupus nephritis (LN), a major and sometimes fatal clinical manifestation, remains challenging in patients with systemic lupus erythematosus (SLE). Several previous cross-sectional studies have suggested osteopontin (OPN) as a putative SLE biomarker, but longitudinal studies assessing its diagnostic accuracy are lacking. Therefore, we aimed to investigate the usefulness of measuring plasma levels of OPN (P-



Abstract PO.5.110 Figure 1