

**Abstract 400 Table 2** Baseline, univariate and multivariate associations of autoantibody profiles of ANA-positive (presence of nuclear IIF pattern), ANA-negative (no IIF pattern), and pure cytoplasmic/mitotic (CMP) groups

	ANA+ Mean or % n=1049	ANA- Mean or % n=71	Pure CMP Mean or % n=17	ANA- or Pure CMP Mean or % n=88	Univariate model Odds ratio 95% CI	Primary multivariate model Odds ratio 95% CI	Secondary multivariate model Odds ratio 95% CI
ANA	100.0*	0.0*	100.0 <sup>†</sup>	19.3*			
DFS ANA by IIF	1.6	0	0	0			
Anti-dsDNA	40.5*	70.6*	26.7	21.7*	0.41 (0.24, 0.70)*		0.53 (0.3, 0.94)*
Autoantibodies							
Monospecific DFS70	0.8	5.6	5.9	5.7*	7.84 (2.51, 24.5)*	4.23 (1.32, 13.58)*	4.45 (1.37, 14.39)*
PCNA	7.3*	1.4*	11.8	3.4	0.45 (0.14, 1.44)		
Ribosomal-P	16.1*	5.6*	11.8	6.8*	0.38 (0.16, 0.89)*		
Ro52/TRIM21	35.9*	21.1*	23.5	21.6*	0.49 (0.29, 0.83)*		
SSA/Ro60	47.3*	22.5*	29.4	23.9*	0.35 (0.21, 0.58)*	0.46 (0.27, 0.77)*	0.51 (0.30, 0.87)*
SSB/La	15.9*	5.6*	11.8	6.8*	0.39 (0.17, 0.90)*		
Sm	24.7*	5.7*	11.8	6.9*	0.23 (0.10, 0.52)*		
U1-RNP	32.4 <sup>†</sup> *	11.3*	11.8 <sup>†</sup>	11.4*	0.27 (0.14, 0.52)*	0.36 (0.18, 0.73)*	0.35 (0.17, 0.70)*
Antiphospholipid Ab							
Lupus Anticoagulant	20.8*	20.6	6.7*	17.9	0.83 (0.46, 1.52)		
Anti-cardiolipin	12.6	11.1	12.5	11.4	0.89 (0.44, 1.83)		
Anti-β2glycoprotein I	15.0	15.9	12.5	15.2	1.01 (0.53, 1.92)		

†, \*, or in combination: values with the same superscript are significantly different from each other, i.e. \* is different from † and † and \* are not. Abbreviations: ANA, anti-nuclear antibody; DFS, dense fine speckled; dsDNA, double stranded DNA; IIF, indirect immunofluorescence; PCNA, proliferating cell nuclear antigen; RNP, ribonucleoprotein; Sm, Smith (U2-U6 RNP); SSA, Sjögren's syndrome antigen A; SSB, Sjögren's syndrome antigen B; TRIM21, tripartite motif 21.

#### 402 CLINICAL PICTURE OF LUPUS NEPHRITIS IN PERSIAN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS (SLE): RESULTS OF A LARGE SURVEY

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**Background and aims** Systemic lupus erythematosus (SLE) is a chronic multisystem disorder. Lupus nephritis (LN) is a common serious complication of SLE. LN needs prolonged care and complex therapeutic modalities.

**Objectives** The aim of this study was to assess the characteristics of Persian SLE patients with LN (LN subgroup) and SLE subpopulation without LN (non-LN subgroup). Furthermore we studied the association of LN with extrarenal manifestations of SLE.

**Methods** In this study we assessed 2355 SLE patients of the electronic database of Rheumatology Research Centre (RRC), Tehran University of Medical Sciences (TUMS). The clinical and laboratory data of enrolled patients were retrieved. Chi-square test was used to compare the extrarenal manifestations between LN and non-LN subgroups. Odds ratio (OR) was used to present the strength of associations.

**Results** LN subgroup included 1604 cases (68.1%), with the mean age at SLE onset of 24.6±12.5 years and female to male ratio of 8.7/1. Class IV nephritis was the most common type of LN (53.1%). Comparison of the extrarenal manifestations revealed significant difference between LN and non-LN subgroups. Major organ involvements including cardiopulmonary, hematologic, musculoskeletal and neuropsychiatric features were significantly more common in LN patients. On the contrary, discoid rash was significantly more common in non-LN subgroup.

**Conclusions** This study revealed that LN was positively associated with musculoskeletal, mucocutaneous and neuropsychiatric features of SLE.

#### 403 STUDY OF FAMILIAL AGGREGATION OF AUTOIMMUNE DISEASES IN ASIAN INDIAN LUPUS PATIENTS (PROBANDS)

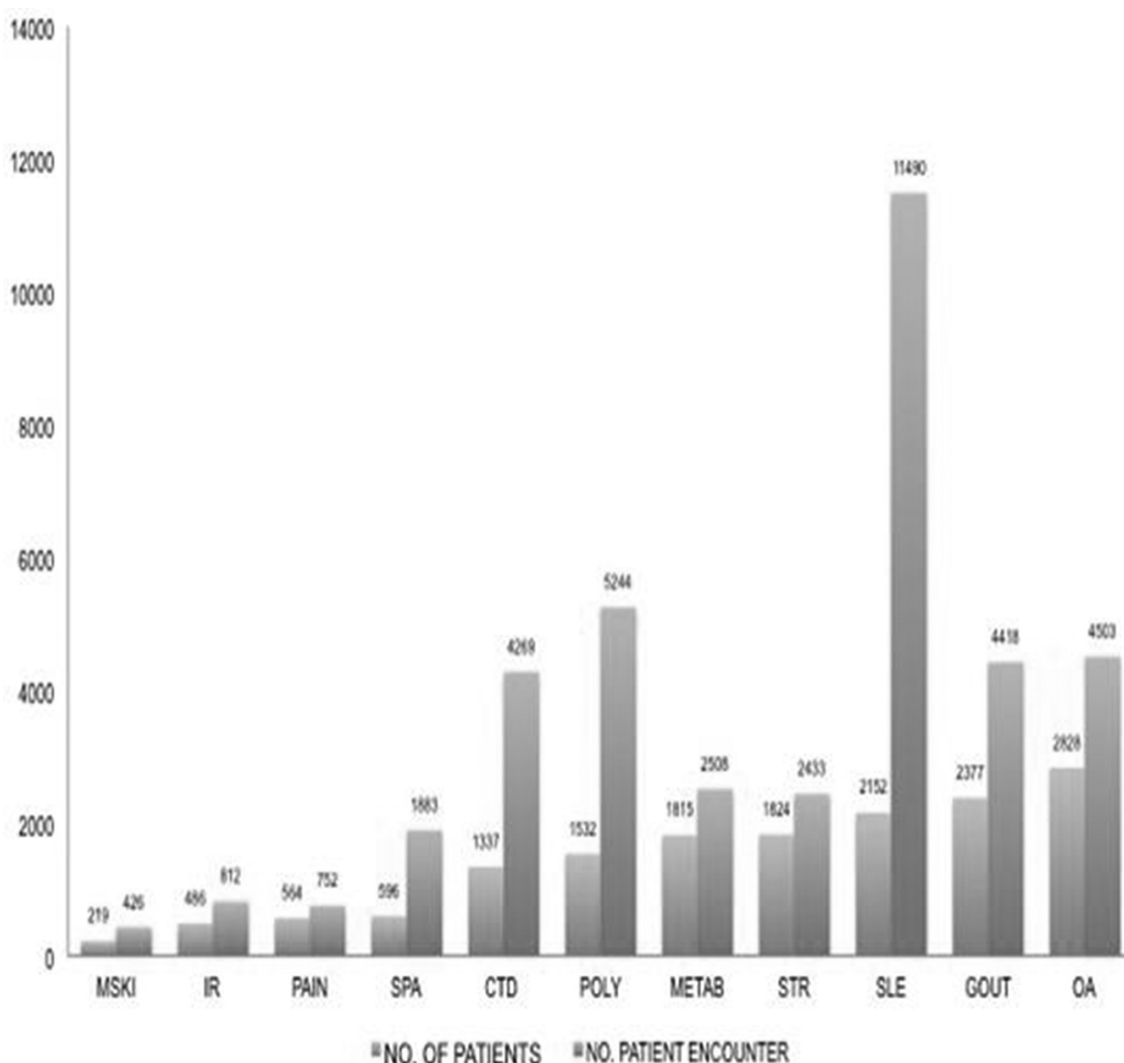
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**Background and aims** SLE and other auto-immune diseases (AID) tend to co-aggregate in families, making positive family history a risk factor for SLE. We aimed to calculate familial aggregation of rheumatic AIDs including SLE, in lupus probands and to compare familial and sporadic lupus pro-bands in our cohort.

**Methods** We studied families of 157 consecutive lupus probands satisfying the 2012 SLICC Classification Criteria in a hospital-based, cross-sectional design, probing for 3 generation pedigree charting, clinical and investigational parameters.

**Results** Systemic AID was seen in 39 families with a point-prevalence of 24.8% (95% CI 18.1, 31.6) and aggregation relative risk (RR) of λ=2.48. Family history of SLE was seen in 19 families with a point-prevalence of 12.1% (95% CI 7.0, 17.2) and λ=2. Both AID as a whole and lupus alone were seen more commonly with parental consanguinity ( $p<0.05$ , Table 1) with no specific inheritance pattern. AID including lupus was seen commonly in 1<sup>st</sup> degree (64.1%–63.15% respectively) followed by 2<sup>nd</sup> degree relatives (43.5%–52.6%). Most prevalent co-existent organ-specific AID was auto-immune thyroid disease (AITD) seen in 42 (26.75%) families, which also co-existed in 27 (17.2%) lupus pro-bands. Familial aggregation in lupus pro-bands showed relatively higher percentage of affected males and lesser constitutional features ( $p<0.05$ ) than sporadic pro-bands (Table 2).



**Abstract 401 Figure 1** Number of patients and frequency of patient encounters per rheumatic disease at UST Hospital.

**Conclusions** In Asian Indian lupus pro-bands, familial aggregation of rheumatic AID and Lupus alone was noted in 24.8% (RR-2.48) and 12.1% (RR-2) respectively, more so in the background of parental consanguinity.

#### 404 PREDICTORS OF RENAL REMISSION, RENAL INSUFFICIENCY AND DAMAGE IN LUPUS NEPHRITIS

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**Background and aims** To explore predictors of renal remission, insufficiency and damage in lupus nephritis (LN).

**Methods** We retrospectively analysed our lupus cohort and studied LN patients with at least one renal biopsy since 1990 until 2016. Follow-up ended at last patient visit. Complete renal remission (CRR) was defined as proteinuria <0.5 g/day and normal serum creatinine (SCr); partial remission as proteinuria <3.5 g/day with normal SCr; renal failure as SCr

≥2 mg/dl; renal flare as an increase in proteinuria >0.5 g/day and/or requirement for treatment modifications. Damage was measured by SLICC damage index (SDI).

**Results** 81 patients were studied (Table) who underwent 110 biopsies. Forty patients (49.3%) went into CRR by the end of follow-up. Thirty-six (44.5%) had 2 renal flares while 21≥3 (26%). Six patients developed renal failure preceded by ≥3 flares in 5. One case of ESRD was reported. By the end of follow-up 30 patients (37%) had SDI≥2. At univariate analysis, increased proteinuria at 12 and 24 months from first biopsy and higher flare number were inversely associated with CRR, while long-lasting hypertension, abnormal SCr, decreased GFR and C4 at time of first biopsy were associated with renal failure. At multivariate analysis 24h-proteinuria at 24 months independently predicted lack of CRR (OR 3.7), while a higher number of renal flares (OR 5.27), higher SCr at 6 months from renal biopsy (OR 5.01) and a longer disease duration (OR 6.34) were independently associated with damage accrual. **Conclusions** Increased proteinuria and abnormal baseline renal function make CRR unlikely. Suboptimal control of LN activity and longer disease duration are associated with severe damage accrual.