percentage of the positive stained cells, the semiquantitative analysis was made. The expressions of ERs, including ER α and ER β , of the two groups were compared, and the relationship between the expression of ERs and serum UA levels were analyzed with linear regression analysis.

Results: 1. The mean ages of the LN group and the IgA group were 27.39 $\pm$ 8.09 years and 33.08 $\pm$ 5.45 years, respectively, with significantly different ($t=2.128$, $p=0.042$). There was no renal failure subject in both groups (CRE level above 120 μ mol/l), and all the SLE patients were at stage of onset.

2. Mean UA level of the LN group was significantly higher than that of the IgA group ($t=-3.149$, $P=0.004$), and the CRE level of the two groups were similar ($t=-0.109$, $P=0.914$) (table1).

3. In each group, ER β was well expressed in the tubular epithelial cells, and no in kidney glomeruli. In contrast, ER α was not expressed in the renal tissues of both groups (figure1).

4. The median score of ER β expression in the LN group was remarkable lower than that of the IgA group ($z=-2.080$, $P=0.038$, table 1).

5. Linear regression analysis indicated that ER β expression scores of the total 30 patients had significantly negative correlation with serum UA levels ($t=-2.141$, $P=0.041$, table2).

Conclusion: 1. Low expression of ER β in renal tubular epithelial cell may contribute to hyperuricemia in premenopausal female SLE patients.

2. ER α showed no expression in LN and IgA nephropathy kidney tissues.

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Table 1. Comparative analysis of main clinical biomarkers in LN groups and IgA group

	LN group (n=18)	IgA group (n=12)	P value
Age (year)	27.39 $\pm$ 8.09	33.08 $\pm$ 5.45	$t=2.128$ $p=0.042$
UA (μ mol/L)	510.33 $\pm$ 141.50	383.25 $\pm$ 78.72	$t=-3.149$ $p=0.004$
CRE (μ mol/L)	92.22 $\pm$ 11.71	91.72 $\pm$ 12.64	$t=-0.109$ $p=0.914$
ER α	0	0	
ER β	0.08(0.02,0.23)	0.20(0.10, 0.30)	$z=-2.080$ $p=0.038$

UA: uric acid; CRE: creatine; ER: estrogen receptor.

Table 2. The correlation of ER β expression scores in the renal tubular epithelial cell with serum UA levels in premenopausal female SLE patients

Variable	Unstandardized Coefficients B	Standardized Coefficients SEM	t	P	95%CI
ER β	-317.004	148.040	-2.141	0.041	-620.251 -13.757
Constant	516.455	148.040	14.641	0.000	444.198 588.712

ER: estrogen receptor

Fig 1. ER β expressed in renal tubular epithelial cell of LN patients: a, ER β expressed in renal tubular epithelial cell of IgA nephropathy patients; c, ER α was not expressed in renal tissue of LN patients; d, ER α was not expressed in renal tissue of IgA nephropathy patients.

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FRI0227

PREGNANCY OUTCOMES IN A LONG-TERM FOLLOW-UP COHORT OF PEDIATRIC ONSET SYSTEMIC LUPUS ERYTHEMATOSUS (PSLE) AT A TERTIARY CARE CENTER IN NORTH-WEST INDIA

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Background: Pediatric-onset SLE (pSLE) represents 10–20% of all SLE cases, and is associated with more severe disease as compared to adult onset SLE. pSLE patients are known to have more frequent systemic involvement especially related to kidneys, central nervous system and musculoskeletal system(1). Pregnant women with SLE have increased risk of adverse outcomes including disease flare, spontaneous abortion, pre-eclampsia/eclampsia, premature birth and maternal death(2). There is paucity of literature in pregnancy outcomes among women with pediatric onset SLE, especially from developing countries

Objectives: To evaluate pregnancy outcomes in a long term follow up cohort of pediatric SLE patients from a tertiary care center in North India

Methods: We have reviewed our previously published cohort of 27 pSLE patients who were diagnosed as SLE between 1998 and 2003. They were being followed-up at Pediatric Rheumatology Clinic, Advanced Pediatrics Centre, Postgraduate Institute of Medical education and research, Chandigarh, India. We have analyzed disease related complications, anti-phospholipid antibody profile, pregnancy related complications and long-term outcomes in these patients.

Results: Follow up records of 27 pSLE patients were retrieved from the clinical files. Data are available for 14 patients (i.e. 52% of the original cohort). Six (22%) of 14 patients expired due to disease related complications (2 had end stage renal disease, 1 had pulmonary thromboembolism, 2 had pulmonary hemorrhage, 2 had sepsis). Eight patients have been on follow-up till date with mean duration of follow-up of 20 years (range:16-25 years). Total patient years of follow up was 160 years. Of the 8 patients on follow up, renal manifestations were seen in 4(50%), neuropsychiatric in 4(50%), severe hematologic in 3(37.5%), musculoskeletal in 1(12.5%), anti-phospholipid antibody (APLA) positivity in 3(37.5%) patients. Disease and therapy related complications were seen in 3 patients (obesity in 2, osteopenia and avascular necrosis of femur in 1, hypertension in 1). Eight pregnancies were observed in 5 of these patients; 1 patient had primary infertility. Mean age at first pregnancy was 19 years (range: 21-25 years). All 5 patients received hydroxychloroquine throughout the pregnancy and disease activity was well controlled. APLA testing was done in 2 patients during pregnancy and both tested negative. Five live births and 3 fetal losses were observed. The fetal losses were spontaneous first trimester abortions. None had eclampsia/pre-eclampsia, ectopic pregnancy or adverse fetal outcomes (prematurity, intrauterine growth restriction, heart defects).

Conclusion: This is one of the longest follow-up studies in pSLE patients and probably the only one where pregnancy outcomes have been studied in a pediatric onset disease in context of a developing country. Our results show that in the presence of well controlled symptoms, pregnancy related outcomes are favorable with minimal fetal and maternal complications even in resource limited settings.

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FRI0228

KL-6 AS A BIOMARKER OF DEVELOPING INTERSTITIAL LUNG DISEASE IN PATIENTS WITH SJÖGREN SYNDROME

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Background: Sjögren syndrome (SS) is an autoimmune disease presented with exocrine gland inflammation and extra-glandular involvement. The

prevalence of pulmonary involvement, including interstitial lung disease (ILD), in patients with primary SS widely varies from 8% to 20%.¹ ²Patients with SS with ILD have high morbidity and a fourfold increase in mortality.³

Objectives: Krebs von den Lungen-6 (KL-6) is a mucin-like, high-molecular-weight glycoprotein; it is expressed in regenerating type II pneumocytes. Serum KL-6 is highly associated with the activity of ILD in patients with radiation pneumonitis, idiopathic pulmonary fibrosis, hypersensitivity pneumonitis, sarcoidosis, rheumatoid arthritis, polymyositis, dermatomyositis and systemic sclerosis.⁴⁻⁸ The increase in serum KL-6 level might reflect the increase in the number of regenerating type II pneumocytes secondary to pulmonary damage.⁸ We hypothesise that an early pulmonary damage occurs before clinical or radiological evidence of ILD in patients with SS.

Methods: In this retrospective case-control study, patients who were diagnosed with primary SS and fulfilled the American-European Consensus Group Criteria for Sjögren's Syndrome were included. Clinical information, laboratory results on inclusion, images and pulmonary functions test results were recorded via electronic medical records review. Pulmonary radiography including chest X-ray and chest computed tomography was reviewed by a chest physician.

Results: Of the 39 patients with SS, 21 (53.85%) developed ILD at the end of follow-up. The follow-up period was 2.65 ± 1.88 years. The time to diagnosis of ILD was 2.72 ± 1.74 years in the ILD group. The serum KL-6 level was 1920.10 ± 1974.26 U/ml in the ILD group and 894.11 ± 788.53 U/ml in the non-ILD group ($p = 0.001$). The diffusing capacity of the lungs for carbon monoxide was 70.76 ± 19.63 mmHg and 91.88 ± 12.02 mmHg in the ILD group and non-ILD group, respectively ($p < 0.001$).

Conclusion: Serum KL-6 level is significantly higher in patients with primary SS with developing ILD and may represent an early non-radiographic pulmonary damage. Serum KL-6 can be a valuable biomarker in predicting the development of ILD in patients with primary SS.

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FRI0229

SOLUBLE CD163 IS A BIOMARKER FOR ACCELERATED ATHEROSCLEROSIS IN SLE PATIENTS AT APPARENT LOW RISK FOR CARDIOVASCULAR DISEASE

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Background: Cardiovascular disease (CVD) due to accelerated atherosclerosis is the leading cause of death in SLE. Probably because they do not incorporate important risk factors such as overweight, chronic kidney disease or systemic inflammation, models for predicting CVD established in the general population (such as Framingham model) underestimate the cardiovascular risk in SLE. Identification of biological markers for CVD may help to classify high-risk subjects and set up targeted prevention in SLE.

Objectives: Our study aimed to determine whether sCD163, a soluble macrophage marker upregulated in numerous inflammatory disorders, might be predictive of accelerated atherosclerosis associated with SLE

Methods: Carotid ultrasound was prospectively performed in 63 consecutive SLE patients asymptomatic for cardiovascular disease (CVD) and 18 volunteer health-workers. Ultrasound was performed at baseline and during follow up. Serum level of sCD163 was determined at baseline using ELISA. The primary outcome was the presence of a carotid plaque. Factors associated with carotid plaques were identified through multivariate analysis.

Results: Despite a low risk for cardiovascular events according to Framingham score in both groups ($2.1\% \pm 3.8$ in SLE vs $2.1\% \pm 2.9$ in controls; $p=0.416$), ultrasound study at baseline showed a carotid plaque in 23 (36.5%) SLE patients versus 2 (11.1%) controls ($p=0.039$). Multivariate analysis showed that SLE status increased the risk for carotid plaque by a factor of 9 ($p=0.017$). In SLE patients, sCD163 level was high ($483.7\text{ng/ml} \pm 260.8$ versus $282.1\text{ng/ml} \pm 97.5$ in controls; $p<0.001$) and independently associated with carotid plaques as assessed by stratification based on sCD163 quartile values ($p=0.009$), receiver operating characteristic (ROC) ($p=0.001$) and multivariate analysis ($p=0.015$). Eventually, sCD163 at baseline was associated with the onset of carotid plaque during follow up (3 ± 1.4 years) in SLE patients who had no carotid plaque at first evaluation ($p=0.041$)

Conclusion: sCD163 is associated with progressing carotid plaque in SLE and may be a useful biomarker for accelerated atherosclerosis in SLE patients at apparent low risk for CVD

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FRI0230

CLINICAL AND ECHOCARDIOGRAPHIC CHARACTERISTICS OF MYOCARDIAL INJURY IN SYSTEMIC LUPUS ERYTHEMATOSUS, CLASSIFIED ACCORDING TO CARDIAC MAGNETIC RESONANCE CRITERIA

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Background: Lupus myocarditis (LM) occurs in 5-10% of patients with systemic lupus erythematosus (SLE). Subclinical myocardial inflammation occurs in 37% at post mortem.¹ Echocardiographic strain (STE) supports subclinical myocardial dysfunction in SLE.² Tissue characterisation by cardiac magnetic resonance (CMR) identifies myocardial inflammation, necrosis and/or fibrosis, detecting clinical and subclinical myocardial injury (MIN) in SLE. It's the non-invasive gold standard for diagnosing myocarditis (all types) based on the Lake Louise criteria (LLC).³