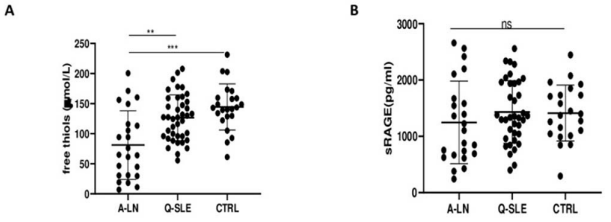
previously described.[2] sRAGE levels were measured by ELISA. In addition, the assessment of disease activity was performed using SLEDAI calculation for each patient. The changes in plasma-free thiols and sRAGE levels longitudinally were compared to clinical and biochemical markers using generalized estimating equations (GEE).

Results Thiols levels were significantly lower in active LN (at baseline) and Q-SLE patients compared to HC (Figure1 A). There was no significant difference in sRAGE levels between the groups (Figure 1 B). The median of changes in plasma-free thiols and sRAGE levels during the 36-month follow-up are shown in figure 2. In the univariate GEE model (table 1), changes of plasma-free thiols were negatively correlated with SLEDAI ($p < 0.001$). Changes of sRAGE were also correlated with SLEDAI ($p = 0.035$). In addition, changes of plasma-free thiols were also correlated with creatinine levels.

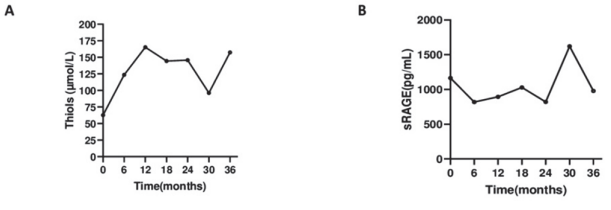
Conclusions Plasma-free thiols levels are significantly reduced in patients with SLE and LN at baseline compared to HC. In follow-up of LN patients, free thiols levels are significantly negatively correlated to SLEDAI. These results suggest that free-thiols could be a potential additional redox signaling marker to evaluate the activity of LN.

Abstract PO.5.111 Table 1 Association between oxidative stress biomarkers and clinical and laboratory parameters in GEE model

	Thiols		sRAGE	
	B (95% CI)	P-value	B (95% CI)	P-value
SLEDAI	-4.72 (-5.98—3.46)	**<0.001***	20.69 (1.47-39.91)	**0.035***
Hb	-0.23 (-11.64-11.17)	0.968	-54.45 (-207.14-98.24)	0.485
Leukocytes	-0.81 (-5.20-3.58)	0.718	-5.58 (-32.15-20.99)	0.680
Thrombocytes	-0.10 (-0.23-0.02)	0.113	0.09 (-1.74-1.92)	0.923
C3	-17.06 (-72.92-38.80)	0.550	256.06 (-258.55-770.68)	0.329
C4	-130.99 (-281.09-19.12)	0.087	1490.08 (-334.02-3314.19)	0.109
Creatinine	0.17 (0.00-0.33)	**0.048***	0.46 (-2.42-3.33)	0.756
urine protein/24h	-1.28 (-4.84-2.28)	0.481	-5.21 (-46.52-36.11)	0.805
Anti-dsDNA (pos vs. neg)	-12.64 (-40.14-14.85)	0.367	-184.13 (-459.76-91.50)	0.190



Abstract PO.5.111 Figure 1 Active LN is associated with lowered plasma-free thiols levels. A) Plasma-free thiols levels in active LN patient's at baseline are significantly decreased compared to quiescent SLE patients and HC. B) sRAGE levels are not different between the groups. A-LN: active lupus nephritis, Q-SLE: Quiescent systemic lupus erythematosus, CTRL: healthy control



Abstract PO.5.111 Figure 2 (A) Median plasma-free thiols levels during 36 months follow-up in LN patient's; (B) sRAGE levels during 36 montsh follow-up in LN patient's

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PO.5.112 DIFFUSE ALVEOLAR HEMORRHAGE IN LUPUS NEPHRITIS PATIENTS: A MULTICENTER RETROSPECTIVE STUDY

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10.1136/lupus-2022-elm2022.134

Purpose Diffuse alveolar hemorrhage (DAH) is a rare and potentially lethal complication of systemic lupus erythematosus (SLE) with a high mortality rate. It occurs more frequently in patients with lupus nephritis (LN). The aim of our study is to explore the characteristics of patients that develop DAH with lupus nephritis, risk factors that predispose DAH, treatment response and outcomes.

Methods Multicenter retrospective cohort study was undertaken including 6 centers in Saudi Arabia from 2002 to 2018. SLE patients meeting the SLICC criteria with lupus nephritis (biopsy proven or proteinuria or renal impairment due to lupus) presenting with diffuse alveolar hemorrhage (fulfilling a predefined criteria) were included in the study. An identical number of control group with lupus nephritis was also studied. Data was obtained from medical records by using a data sheet: demographics including age, gender, diagnosis, date of diagnosis of lupus, date of presentation of alveolar hemorrhage, clinical presentation, detection of alveolar hemorrhage proved by radiology, lavage or biopsy and laboratory parameters: including level of hemoglobin before and during DAH, sign of activity, treatment and outcome of DAH. Identification of risk factors predisposing to DAH in lupus nephritis patients was analyzed.

Results We identified 23 cases of DAH with lupus nephritis, all fulfilling the criteria. Mean age at presentation of DAH was 31.09 ± 12.6 years ranging from 14–57 years, of which 87% were females. 13 patients 56.5% had Class 4 LN and 21.7% had Class 4 and 5 LN on renal pathology. DAH occurred at a mean of 6.5 years ±3.8 in 13/23 patients with LN. Shortness of breath 95%, new chest x ray finding 95.7% and mean drop of haemoglobin of 2.72 gm/dl ±0.97 were more frequent at presentation of DAH with LN patients. High SLE disease activity - SELENA SLEDAI 2K was 38.56 ±19.3 was present at the onset of DAH. All were treated with methyprednisone,15/23 (65.2%) underwent mechanical ventilation and plasmapheresis was done in 21/23 patients (91.3%). Cyclophosphamide was given in 14/21 patients (60.9%), Intravenous immunoglobulins were given in 14/23 patients (65.2%) and dialysis was done in 12/23 patients (52.2%). Mortality occurred in 8 patients 34.8%. In comparison with the LN group, a mean haemoglobin of 7.56 ±

1.3, CNS involvement, vasculitis and fever>38 °C were of statistically significance P value: <0.001,0.02,0.03 and 0.03 respectively.

Conclusion In this multicenter cohort series with DAH in LN patients CNS involvement, vasculitis and fever>38 °C were associated in the occurrence of DAH. Mortality was low in our cohort in comparison to previous series which may be explained by early diagnosis and use of aggressive management.

PO.5.113 C3 AND C1Q DEPOSITION IN DIFFERENT KIDNEY COMPARTMENTS IS NOT ASSOCIATED WITH SERIOUS INFECTIONS IN LUPUS NEPHRITIS

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10.1136/lupus-2022-elm2022.135

Purpose Complement activation is an important step in the mechanism of tissue damage in lupus nephritis (LN). Complement deposition in kidney tissue might reflect different immunologic processes and higher disease severity and result in adverse outcomes, but very few studies explored these potentially important associations. Given these immunologic consequences, we have postulated that complement deposition in the kidney might be associated with higher risk for serious infections in LN.

Methods We have conducted a retrospective cohort study to evaluate the prognostic significance of C1q and C3 complement factors in renal tissue compartments for the occurrence of serious infections. We have collected data on demographics, clinical and laboratory parameters and histopathology (light, immunofluorescent and electron microscopy) at the time of biopsy and after long-term follow-up. Serious infections were defined as those that: 1. require intravenous therapy OR 2. lead to hospitalization OR 3. have resulted in death in 30 days from diagnosis. C1q and C3 expression graded in different kidney compartments (mesangium, glomerular basement membrane (GBM), tubular basement membrane (TBM) and peripheral capillary wall) as 0 to 3+ and another analysis was performed with dichotomized grading as 0 (absent) and 1+ to 3+ (present). SLE was diagnosed using the American College of Rheumatology criteria.

Results A total of 51 patients with biopsy-proven LN were followed up for 4.5±2.9 years (80% women, mean age at biopsy 38±14). Of these, 22 (43%) had at least one episode of serious infection with 4 patients having 2 episodes. Complement expression in different kidney compartments was as follows: mesangium (C1q 54%, C3 59%), GBM (C1q 34%, C3 41%), TBM (C1q 5%, C3 5%) and blood vessel wall (C1q 0%, C3 5%). There was no difference in the distributions of mesangial (present vs. absent, 80% vs. 78%, p>0.99), GBM (53% vs. 38%, p=0.53), TBM (20% vs. 5%, p=0.17) and peripheral capillary wall C3 deposition (25% vs. 35%, p=0.53) or mesangial (75% vs. 65%, p=0.53), GBM (47% vs. 33%, p=0.52), TBM (5% vs. 5%, p>0.99) and peripheral capillary wall C1q deposition (25% vs. 30%, p=0.75).

Conclusions Complement deposition in kidney tissue, while an underexplored and potentially important process, was not associated with serious infections in LN.

PO.5.114 ONLINE EDUCATION SIGNIFICANTLY IMPROVED RHEUMATOLOGISTS' KNOWLEDGE OF THE BURDEN OF LUPUS NEPHRITIS AND APPROPRIATE TREATMENT STRATEGIES FOR PATIENTS

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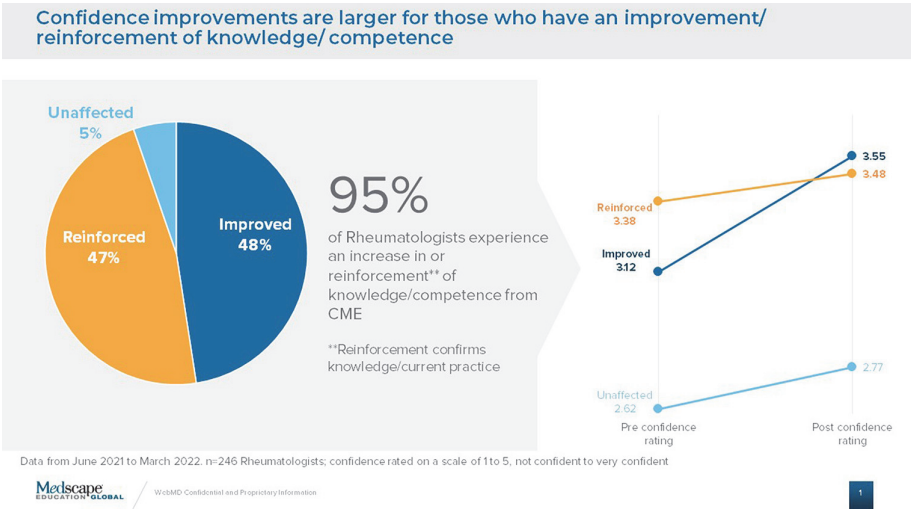
10.1136/lupus-2022-elm2022.136

Background/purpose Lupus nephritis (LN) is the most common severe manifestation of SLE and can progress to end stage



Abstract PO.5.114 Figure 1