Abstract P42 Figure 1 Heatmap representation of cluster analysis of key iron mediators in SLE. Normalised data is presented as Z-score with -3.0

spite of immune activation. Hierarchical clustering is presented in figure 1. Cluster analysis demonstrates that the group with the highest mean SLEDAI-2K (10.7) had lower Hb, transferrin and LCN2 in addition to elevated IL1 β , IL6 and hepcidin compared with those with lower SLEDAI-2K. Elevated haptoglobin levels were seen in those in the higher disease activity groups, suggesting that haemolysis was an unlikely cause for anaemia.

Conclusions The findings of this study suggest increased lupus disease activity results in abnormal iron homeostasis through impaired cellular iron import (via reduced transferrin), a lack of stored iron release (under the actions of elevated hepcidin) and reduced iron sequestration by LCN2, which may represent a novel cause of non-haemolytic anaemia.

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P43 SERUM S100A8/A9 CONCENTRATIONS ARE ASSOCIATED WITH NEUROPSYCHIATRIC INVOLVEMENT AND FATIGUE IN SLE

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Background/Purpose Neuropsychiatric (NP) involvement and fatigue are both major problems in SLE. S100A8/A9 is a marker of inflammation, which responds to therapy in SLE patients. S100A8/A9 is expressed in the CNS. We investigated S100A8/A9 in relation to NPSLE and fatigue.

Methods In this cross-sectional study we used ELISA (Bhulmann MRP8/14 ELISA kit, Switzerland) to measure the concentration of S100A8/A9 in serum in 72 SLE patients and 26 healthy controls and in cerebrospinal fluid (CSF) in 33 SLE patients. NP involvement was determined according to ACR

case definitions for NPSLE. An MRI was performed in SLE patients and controls assessing white matter abnormalities and cerebral atrophy. Measurements of fatigue were performed using the Fatigue Severity Scale (FSS) and the Visual Analogue Scale (100 mm) (VAS). Statistical calculations were performed using non-parametric methods.

Results In all, 72 female SLE patients (median age 38, range 18–52) and 26 female healthy controls (median age 40, range 23–52) were included in this study. Forty-four (61%) patients had NP involvement. NPSLE patients had higher serum S100A8/A9 concentrations (median 1.40 μ g/ml) than the non-NPSLE patients (median 0.92 μ g/ml; $p=0.011$) and the control group (median 0.79 μ g/ml; $p=0.004$). Serum S100A8/A9 correlated with increased VAS fatigue in SLE patients ($r=0.311$; $p=0.008$), but not with FSS ($r=0.184$; $p=0.124$). Serum S100A8/A9 did not correlate with the extent of white matter lesions, atrophy of brain segments, or disease activity (SLEDAI-2K). S100A8/A9 was not detected in the CSF.

Conclusions Higher serum S100A8/A9 concentrations in NPSLE patients and patients with fatigue may indicate that S100A8/A9 is involved in the pathogenesis of these manifestations, although further investigation is needed.

P44 URTICARIAL VASCULITIS IN CHILDHOOD ONSET SYSTEMIC LUPUS ERYTHEMATOSUS

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Purpose Urticarial vasculitis (UV) is a rare type of vasculitis in children frequently, idiopathic, however, can be associated childhood onset SLE. The aim of the study is to describe the frequency of UV in cSLE and to compare the epidemiological, clinical and laboratory characteristics of cSLE stratified by the presence of UV in a cohort of patients of Arab ethnicity from Oman.

Methods We conducted a retrospective multicenter study among pediatric rheumatology centers in Oman over a 10year period.

Results A total of 148 cSLE patients, n=44 males (30%) and n=104 (70%) females, with male to female ratio of 1:2.4 were included in the study. The overall mean age at diagnosis was 7.6 ± 3.5 years and median disease duration was 9.5 years. A total of 36% (n=53) of children with SLE were diagnosed with UV. cSLE with UV were more likely to be males (57% *vs* 15%; $p < 0.001$), with a family history of SLE (53% *vs* 36%; $p = 0.044$), diagnosed at a younger age (5.9 *vs* 8.5 years; $p < 0.001$) and originating from the Al-Sharqiya region of Oman (62% *vs* 29%; $p < 0.001$). Compared to cSLE without UV, the clinical features of UV cohort was more frequently associated with conjunctivitis (32% *vs* 5.3%; $p < 0.001$). However, the UV cohort was less likely to be associated malar rash (5.7% *vs* 31%; $p < 0.001$), CNS involvement (20% *vs* 7.6% $p = 0.045$) and hematological manifestations such as leukopenia (9.4% *vs* 24%; $p = 0.028$), lymphopenia (28% *vs* 51%; $p = 0.009$) and thrombocytopenia (5.7% *vs* 18%; $p = 0.045$). When compared to cSLE without UV, the laboratory features of UV cohort was more likely to be associated with low C3 complement count (94% *vs* 66%; $p < 0.001$) and cytoplasmic antibody ANCA (11% *vs* 0%; $p = 0.022$). However, the UV cohort was less likely to be associated with ANA (65% *vs* 83%; $p = 0.016$), DsDNA (56% *vs* 72%; $p = 0.042$) and perinuclear anti-neutrophil cytoplasmic antibodies (33% *vs* 55%; $p = 0.047$).

Conclusion We describe relatively high occurrence of UV (36%) in a cohort of cSLE of Arab ethnicity with unique clinical and laboratory features. Further studies are needed to evaluate the relationship of UV with SLE and to study the role of genetic, ethnic and environmental factors in disease expression.

P45 ANA POSITIVITY IN IGA NEPHROPATHY: IS SYSTEMIC LUPUS ERYTHEMATOSUS COMING UP?

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Background IgA nephropathy is the most common chronic primary glomerulonephritis, leading to progressive renal failure in at least one third of patients. Although IgA nephropathy is a limited non-systemic renal disease, many systemic diseases are sporadically associated with mesangial IgA deposition. Henoch-Schönlein purpura, systemic illness, has been closely linked to IgA nephropathy. Other systemic diseases in which mesangial deposits of IgA are regularly observed include SLE, hepatitis, dermatitis herpetiformis, and ankylosing spondylitis. The occurrence of non-lupus glomerulopathies has been rarely reported in patients with SLE. We report of 11 patients with biopsy-proven IgA nephropathy and ANA positivity.

Methods Of all patients with biopsy-proven IgA nephropathy followed in our Unit we selected patients with ANA positivity.

Results Of 11 patients with IgA nephropathy and ANA positivity six were females, mean age was 32 ± 11 years. The median follow-up was 7 years (range: 3–38 years). Mean serum creatinine was 1.2 ± 0.75 mg/dl; mean 24h-proteinuria was 1.6 ± 2.3 g/day. In all patients there was low C3. In table 1 are summarized patient characteristics. All patients were on Ace-i or ARB therapy, of these three were treated with steroids, four received steroids+ immunosuppressive drug. Mean follow-up serum creatinine was 1.1 ± 0.7 mg/dl; mean follow-up 24h-proteinuria was 0.4 ± 0.3 g/day. One patient developed SLE.

Conclusions Our cohort suggests the hypothesis that IgA nephropathy could be a special subtype of Lupus Nephritis; however it needs more clinical observation and research.

P46 CASE REPORT: A PATIENT WITH HUMAN IMMUNE DEFICIENCY VIRUS MIMICKING SYSTEMIC LUPUS ERYTHEMATOSUS

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Background Systemic lupus erythematosus (SLE) is an autoimmune disease characterized by various clinical presentations. Human immune deficiency virus (HIV) causes acquired immune deficiency syndrome (AIDS) in case of progression of the infection. Patients with HIV can present different rheumatologic findings including arthralgia, arthritis, myalgia,

Abstract P45 Table 1 Patient characteristics and follow-up

N.	SEX	AGE (years)	sCreat at onset (mg/dl)	Uprot at onset (g/day)	Therapy	Follow-up (years)	sCreat (mg/dl) at follow-up	Uprot (g/day) at follow-up
1	F	32	1.6	0.9	steroids+MMF	7	1.9	0.3
	F	17	0.7	0.4	steroids	38	0.6	0.1
3	M	37	1.8	6	steroids+MMF	4	0.8	0.5
4	F	48	1.1	1	steroids+AZA	12	0.8	0.3
5	F	47	0.7	0.3	ARB	3	0.7	0.8
6	M	29	0.9	0.3	ACE-I	3	0.8	0.4
7	M	39	3	1	steroids+MMF	3	2.7	0.3
8	F	23	0.8	0.2	ACE-I	15	0.8	0.1
9	M	16	0.8	0.3	steroids	5	1	0.7
10	F	22	0.8	0.4	ARB	19	0.7	0.1
11	M	39	0.9	6	steroids	10	1	0.1