Background Systemic Lupus Erythematosus (SLE) patients are known to be prone to infections. In particular, differential diagnosis between an acute infection or a disease flare should be performed in any SLE patient with fever or recurrent fever and signs of acute disease. Differential diagnosis becomes even more difficult in an SLE patient with central nervous system (CNS) involvement. The aim was to describe an SLE patient with CNS involvement and acute West Nile virus infection.

Methods A female patient was diagnosed with SLE at the age of 32 with a light-sensitive face eruption, hair loss, fatigue, arthralgias, ANA 1/320 (+) and anti-dsDNA (+). At the age of 45 she developed CNS involvement with epileptic seizures, dysarthria, memory loss, concentration difficulties and an abnormal EEG. At the age of 53 she had an acute SLE flare and pulse cyclophosphamide iv was administered. Thereafter rituximab was given followed by hydroxychloroquine and prednisolone she continued photoprotection. She presented to the emergency department with deteriorating renal function, fever and confusion.

Results A brain MRI showed meningeal thickening and a lumbar puncture was performed. The diagnostic evaluation of the fluid aspirated showed a recent infection with the West Nile virus with IgM (+++) in serum and IgM (+) in the cerebrospinal fluid. Two weeks later the patient had improved, was oriented in place and time and had no focal neurological signs.

Conclusions Patients with SLE are prone to infection, especially if they are on long-standing treatment with steroids. Whenever they present with signs of acute disease they should be carefully evaluated for the presence of an acute infection, as infections demand a different therapeutic approach to a disease flare. A patient with CNS involvement demands even more careful and extensive evaluation. The presence of West Nile virus in Europe in recent years along with other mosquito-borne viruses have created new diagnostic and therapeutic challenges in the management of immunosuppressed patients, as was the case in the patient presented herein.

Background/Purpose This is a prospective study analysed the incidence of skin cancer (SC) (melanoma and non-melanoma SC) in 90 adult patients affected by Systemic Lupus Erythematosus (SLE), followed-up in one single Rheumatological Center, compared with 54 patients affected by Systemic Sclerosis (SSc) and 90 sex- and age-matched 90 control subject.

Methods In a period between February and July 2019, every patient underwent a complete dermatological evaluation and filled out a questionnaire regarding their personal or family history of SC, the presence of different risk factors of SC and the occurrence of photosensitivity.

Results 90 SLE patients (96.7%, female, mean age: 44 years; range: 18–78) showed photosensitivity in 60% 63% of patients avoided sun exposure at every hour of the day, 80% used photoprotection and 28% referred systemic worsening of SLE features after sun exposure. No new onset skin cancer was diagnosed.

Three SLE patients referred a history of SC (1 basaloma, 1 melanoma, 1 multiple actinic lesions) onset after the SLE diagnosis. Patients with skin cancer (SC+) didn’t show any differences compared with patients without skin cancer (SC-) except for more frequent photodermage features (p: 0.032) and less frequent photosensitivity (0.031).

SLE patients more frequently showed photosensitivity (p<0.0001), photoprotection (p<0.0001), disease worsening and skin worsening after sun exposure (p: 0.033 and 0.002, respectively) compared with SSc cases. No differences in past history of SC was evident between groups.

Comparing SLE with age-, sex- and phototype-matched control cohort, SLE patients showed a lower rate of past history of basaloma skin cancer (p: 0.013), lower rate of photodamage (0.027) and higher rate of photosensitivity (p<0.0001).

Conclusions SLE patients showed a significant lower rate of skin cancer, despite a higher rate of photosensitivity, compared with control cohort. This data could be due to a strict and continued photoprotection.

Influence of Dietary Fibre and Short-Chain Fatty Acids on the Pathogenesis of Systemic Lupus Erythematosus

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Background/Purpose In Westernized nations, the incidence of autoimmune and inflammatory diseases has dramatically risen over the past few decades. Environmental influences and their interplay with genetic risk factors have been suggested as important contributors to the rapid epidemic progress. Among environmental influences, Western diet – high in fat, sugar and salt – has been postulated as important risk, while beneficial effects were described for dietary fibre and their bacterial fermentation products, short chain fatty acids (SCFA). In SLE, their impact remains largely unexplored and was addressed in this study.

Methods Lupus prone NZB/WF1 mice were fed a low– (LFD) versus high-fibre diet (HFD) from the age of 3 weeks and over the course of the whole experiment. Determined were
development of clinical disease and associated changes in immune status, gut and energy homeostasis.

Results Animals fed a HFD showed lower autoantibody titres going along with an improved overall survival and a tenden-
tiously lower infiltration of the kidney by leukocytes. Benefici-
cial clinical effects were reflected in systemic immunologic changes, as the distribution and differentiation of main immune cell subsets in HFD animals more closely resembled that of yet healthy animals. We assume that most probably a complex interplay of different fiber-associated effects underlies these favorable effects. This may involve intestinal leakage and bacterial translocation that were increased in LFD animals. Further, LFD animals showed a significant increase in body weight and white adipose tissue expressing more leptin and inflammatory cytokines. We are currently testing, if the observed beneficial effects may also be attributed to an increased fermentation of dietary fibre into SCFA. SCFA inter-
sect in various ways and at different sites with the immune system and mostly have anti-inflammatory effects.

Conclusion Altogether, we think that intake of dietary fiber affects immune status, gut and energy homeostasis. These may be interlinked and affect each other, inflicting more or less systemic chronic inflammation promoting lupus pathology.

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P56 RHUPUS SYNDROME IN A TERTIARY HOSPITAL

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Background/Purpose Rheumatoid syndrome (RhS) is a rare combination of Rheumatoid Arthritis (RA) and Systemic Lupus Erythematosus (SLE). Different studies describe RhS cases that begin with erosive arthritis and the presence of rheumatoid factor (RF) and/or anti CCP and then the SLE symptoms.

Despite the fact that RhS shows a low prevalence, it would be useful to know clinical characteristics of RhS patients since their therapy and outcome differ from those having RA or SLE alone.

Methods Retrospective study with systematic revision of electronic clinical records of RhS patients was performed. Demo-

graphic, clinical and immunological data were collected.

Results Eight RhS patients were included (all fulfilled SLICC 2012 criteria for SLE and ACR 2010 for RA). Mean age was 67.3 (45–84) years (7 were female).

In 3 cases RA was the first diagnosis with a mean evolution of 4.5 years until SLE diagnosis. In contrast, in 5 cases SLE was the first diagnosis with a mean evolution of 7.2 years until RA diagnosis. Photosensitivity and arthritis were the predominant clinical manifestations. One patient presents pericar-
ditis and other case showed rheumatoid nodules in elbows. Renal, pulmonary or neurological affection was no reported.

4 patients were under biological/JAK inhibitors therapies (2 abacetapet, 1 rituximab and 1 baricitinib) with favorable response of treatment.

Conclusions In contrast to other series, only the 37.5% of our RhS cases begins with polyarticular seropositive arthritis. The 62.5% started with SLE symptoms as haematological alter-
tations, cutaneous and serological manifestation, and showed longer progression to have polyarticular affection. Thus, RhS diagnosis is earlier in patients that begin with RA symptoms. 4 RhS patients were refractory to DMARD treatments, where biological/JAK inhibitors therapies are needed.