erythematosus (CLE). Aldara cream was applied topically to the back and ear of C57/BL6J mice directly following, or not, application of TLR7-inhibitory trimeric oligonucleotides formulated in F127 Pluronic gel. Mice were scored daily for the appearance and severity of skin inflammation. After four days, mice were humanely euthanised for multiplex ELISA and qPCR analysis of serum cytokines and inflammatory gene signatures in the skin, respectively.

Results Topical treatment with the TLR7-inhibitory trimeric oligonucleotides greatly ameliorated disease severity (measured by scaling and redness on the back and ear, as well as dermal thickening of the ear) and also led to a significant reduction in both NF-κB-dependent and Type I interferon-stimulated genes in the skin.

Conclusions Our novel TLR7-inhibitory trimeric oligonucleotides represent a promising new class of therapeutics for the treatment of TLR7-driven inflammation in skin manifestations of autoimmunity, for example, psoriasis and CLE.

REFERENCES

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LSO-098 DECREASED NATURAL KILLER T-LIKE CELLS CORRELATED TO DISEASE ACTIVITY IN SYSTEMIC LUPUS ERYTHEMATOSUS

[1]Sumei Tang, [2]Yushu Wang, [3]Xiangbo Ma, [1]Xiaohong Xiang, [4]Xinhua Zhou, [5]Yan Li, [1]Yuan Jia, [1]Fanlei Hu, [1]Yingni Li*. *[1]Rheumatology and Immunology, Peking University People's Hospital, China*; *[2]Inspection Center, Qingdao University Medical College Afliated Yantai Yuhuangding Hospital, China*; *[3]Department of Rheumatology and Immunology, Handan First Hospital, China*; *[4]Clinical Laboratory, Third Hospital of Nanchang, China*; *[5]Clinical Laboratory, First People's Hospital of Jinzhong, China*

10.1136/lupus-2023-KCR.50

Background The NKT-like cells were known to play a role of the suppression in many chronic inflammatory diseases. This study was designed to investigate both of the expression and the possible role of NKT-like cells in SLE patients.

Methods 79 patients with SLE together with 30 age- and sex-matched healthy controls were enrolled. Flow cytometric determination of peripheral NKT-like cells was carried out for all participants by detecting the absolute counts (Abs) and percentage (%) of CD3+CD16+CD56+ cells. Disease activity index, laboratory parameters and clinical manifestations were collected. The correlation between the cells and these parameters were analyzed.

Results SLE patients had, with respect to controls, considerably decreased values of NKT-like cells ($P < 0.001$ in both absolute number and percentage). The absolute number of NKT-like cells were found to have positive correlations with WBC, RBC, PLT, C3, C4, IgM and negative correlations with the disease duration, SLEDAI-2K, anti-dsDNA, anti-nucleosome, anti-ribosomal protein, and CRP, ESR. Meanwhile, it was found that the percentage values of NKT-like cells decreased in SLE patients with nephritis which was correlated with anti-ribosomal protein and CRP in comparison to SLE patients without nephritis. Moreover, an increase in the NKT-like cell counts was also observed in the patients with a clinical response to the treatment.

Conclusions The absolute counts and frequencies of NKT-like cells decreased in SLE patients significantly, which correlated to disease activities and could recover to normal after the treatment. The NKT-like cells may play an important role in the pathogenesis of SLE and could be a useful marker in the disease assessment.

Short oral presentation session 2: antiphospholipid syndrome

LSO-008 PRECIPITATING FACTORS OF CATASTROPHIC ANTIPHOSPHOLIPID SYNDROME: THE ROLE OF ANTICOAGULANT TREATMENT IN A SERIES OF 112 PATIENTS

Romain Stammler, Nathalie Costedoat-Chalumeau*. *Department of Internal Medicine, Cochin hospital, Paris, France, France*

10.1136/lupus-2023-KCR.51

Background The prevention of catastrophic antiphospholipid syndrome (CAPS), a rare complication of antiphospholipid syndrome (APS), is a major goal. The role of precipitating factors in CAPS development is well known. However, little is known about the specific role of anticoagulant treatment as a potential precipitating factor for CAPS. We analyzed precipitating factors of CAPS in a large series of patients, and we focused on anticoagulation immediately before CAPS episodes.

Methods We retrospectively analyzed patients in the French multicenter APS/systemic lupus erythematosus database with at least one CAPS episode. Then we compared each patient with known APS before CAPS with two non-CAPS APS patients matched for age, sex, center and APS phenotype.

Results We included 112 CAPS patients (70% female, mean age 43±15 years). At least one standard precipitating factor of CAPS was observed for 67 patients (64%), mainly infections (n=28, 27%), pregnancy (n=23, 22%), and surgery (n=16, 15%).

Before the CAPS episode, 67 (60%) patients already had a diagnosis of APS. Of the 61 treated with anticoagulants, 32 (48%) received vitamin K antagonists (VKA), 23 (34%) heparin, and 2 (3%) a direct oral anticoagulant. They were less likely than their matched APS patients without CAPS to receive VKA (48% vs 66%, P=0.001). Among those treated with VKA, 72% had a subtherapeutic INR < 2, versus 28% in APS patients without CAPS (P<0.001). Finally, if we excluded pregnant patients (n=14) for whom the effect of treatment versus pregnancy is impossible to differentiate, among the 47 remaining cases, 32 (68%) had either a recent introduction of DOAC (n=2), a planned bridging therapy (n=9) or a VKA treatment with INR <2 (n=21).

Conclusions These results strongly suggest that suboptimal anticoagulation management is a trigger of CAPS in patients with thrombotic APS.