Conclusions This study shows the pRAIL score (both absolute and standardized) distinguishes complete responders versus partial responders during induction therapy. Complete responders pRAIL score decrease by mean of 1 point, whereas partial responders had no change. Notably, standardized pRAIL scores yielded more pronounced differences during induction therapy among both partial and complete responders.

EVIDENCE IN SUPPORT OF THE HYPOTHESIS THAT BOLSTERING ENDOTHELIAL CELL SPHINGOSINE 1-PHOSPHATE RECEPTOR 1 SIGNALING IS A RATIONAL APPROACH FOR THE TREATMENT OF LUPUS NEPHRITIS

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Background Proliferative lupus nephritis (LN) is characterized by robust glomerular and tubulo-interstitial inflammation, sub-endothelial deposits of immunoglobulin, and increased endothelial cell permeability. Sphingosine 1-Phosphate Receptor 1 (S1PR1) has multiple protective effects on endothelial cells (ECs): it maintains barrier function thereby protecting against vascular leakage, it limits the number of leukocytes adhering to and transmigrating across ECs, and it protects ECs against apoptosis in response to inflammatory cytokines. Despite these important protective effects, the role of S1PR1 signaling in endothelial cells in lupus nephritis (LN) has yet to be elucidated. In prior work, we showed that S1PR1 modulators attenuated immune complex mediated vascular injury in skin and lung, leading to our hypothesis that EC S1PR1 signaling limits inflammatory injury in lupus nephritis. In current studies, we assessed whether patients with LN have decreased EC S1PR1 expression, and we performed in vitro mechanistic studies to determine whether S1PR1 maintains barrier function, at least in part, by restraining the metalloproteinase
Abstract 1116

SIPR1 antagonist NIBR-0213 exacerbates immune complex mediated arthritis. (A) Clinical scores of mice subjected to serum induced arthritis (SIA) days 0-7. (B) Representative H&E stained sample of ankle obtained on day 7 after SIA. (C) Histological scoring of mice on day 7 after SIA.

Abstracts

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SIPR1 antagonist NIBR-0213 exacerbates immune complex mediated arthritis. (A) Clinical scores of mice subjected to serum induced arthritis (SIA) days 0-7. (B) Representative H&E stained sample of ankle obtained on day 7 after SIA. (C) Histological scoring of mice on day 7 after SIA.

Lupus Nephritis

1117 NEUTROPHIL EXTRACELLULAR TRAPS AS A BIOMARKER TO PREDICT OUTCOMES IN LUPUS NEPHRITIS

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Background Neutrophil Extracellular Traps (NETs) have been implicated in Lupus Nephritis (LN) pathogenesis. SLE neutrophils release High Mobility Group Box-1 (HMGB1) protein, in turn, HMGB1 in NETs correlates with histologic findings of Active LN (ALN). The aim was to determine if the amount of NET complexes (Elastase-DNA and HMGB1-DNA) in serum at the time of a LN flare predicts renal outcomes in inactive SLE patients.

Methods

We performed a 2-staged approach. In an exploratory cohort composed of active SLE (clinical SLEDAI ≥ 1), inactive SLE and healthy controls (HC), we assessed the association between our in-house ELISA assays for Elastase-DNA and HMGB1-DNA against the diagnosis of LN flare for the following 24 months.