162 HOMOCYSTEINE LEVEL IN PATIENTS WITH LUPUS NEPHRITIS

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Background In SLE, both disease-specific and traditional risk factors are important. Increased serum homocysteine levels are seen in approximately 15% of patients with systemic lupus erythematosus and are associated with an increased risk of atherothrombotic events in this population. The serum level of homocysteine in patients with lupus nephritis has not been well described.

Methods We performed a retrospective review of patients who had both, a biopsy proven lupus nephritis (class II-VI), and measured homocysteine levels for the first time during routine evaluation. Clinical and laboratory data were obtained from review of medical records.

Results Five patients with lupus nephritis had homocysteine level measured. The ages ranged from 29–47 years and were predominately African Americans. There were two patients with class III, one with class III-V, one with class IV, and one with class V lupus nephritis. All were female, with positive anti-dsDNA, low C4 and on hydroxychloroquine. Of the five patients, all had elevated homocysteine levels.

Conclusions This study demonstrates that patients with lupus nephritis are at a high risk for developing elevated homocysteine levels.

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163 BACTERIAL BIOFILM PRODUCT CURLI/EDNA INDUCES NETS AND SERUM ANTI- CURLI/EDNA LEVELS CORRELATE WITH BACTERIURIA AND LUPUS ACTIVITY

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Background Infections are a major contributor to lupus disease. We have previously demonstrated that bacterial amyloid curli, produced by E.coli, can accelerate disease in mouse models of lupus. Interestingly curli incorporates extracellular DNA, which in turn can be both adjuvant and a self-antigen in lupus. Finally, uropathogenic E. coli (UPEC) is responsible for the majority of urinary tract infections in SLE.

Methods Based on our previous results, we hypothesize that exposure to UPEC triggers anti- curli/eDNA antibodies and curli/eDNA complexes can trigger the innate immune system. We investigated 98 lupus patients who met at least 4 SLICC criteria. Results were compared to 54 age, sex and race matched healthy controls. We tested the production of anti-curli/DNA complex for both IgG and IgA subclasses. We also correlated the levels of anti-curli/DNA antibodies with clinical parameters. Finally, we treated human neutrophils with curli/eDNA complexes.

Results We found that curli/eDNA induces neutrophil extracellular traps in a ROS manner. Anti-curli/eDNA IgG levels were detected in lupus and controls plasma and the levels correlated with persistent bacteriuria ($p<0.05$) and disease flares in lupus patients. In addition, anti-curli/eDNA antibodies could bind to DNA demonstrating a potential molecular mimicry mechanism in lupus. Finally IgA anti-curli/eDNA levels were higher ($p<0.01$) in lupus donors compared to controls.

Conclusions We conclude curli/eDNA complexes can activate the innate and adaptive immune system and could be a mechanism to sustaining disease in lupus.

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164 URINARY ALCAM AS A NOVEL BIOMARKER FOR RENAL PATHOLOGY IN LUPUS NEPHRITIS

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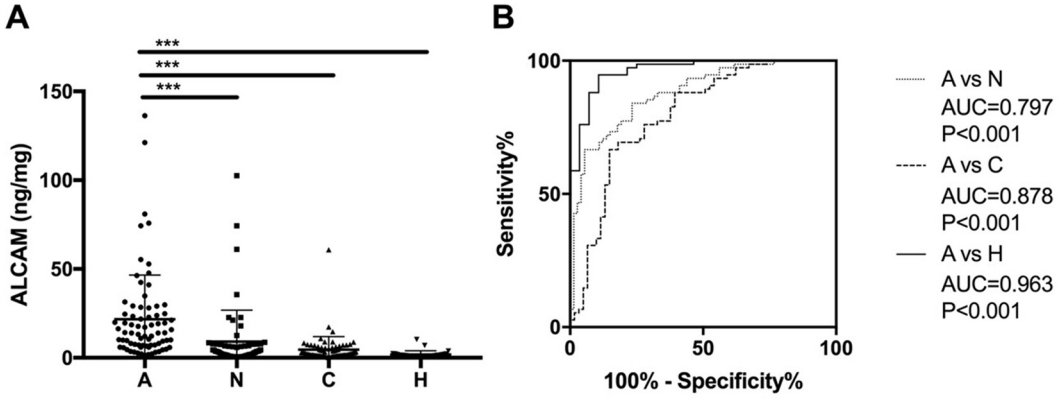
Background Lupus nephritis is one of the most common and severe complications of SLE affecting more than 60% patients. The management of lupus nephritis largely depends on renal histopathologic findings. Due to the invasive feature and complications of renal biopsy, it is significantly urgent to develop novel biomarkers to predict renal pathology.



Abstract 164 Figure 1 Urinay ALCAM levels were elevated in active LN patients. (A) Group comparison showed significant increase of urinary ALCAM in active LN patients (14 IQR (6.97-24.8) ng/mg) when compared to those in active SLE without renal involvement patients (3.54 IQR (1.31-7.58) ng/mg), inactive SLE patients (2.75 IQR (1.45-5.67) ng/mg), and healthy controls (1.29 IQR (0.665-1.89) ng/mg). (B) Receiver Operating Characteristic (ROC) curve analysis indicated a good performance of urinary ALCAM in discriminating active LN from active SLE without renal involvement, inactive SLE, and healthy controls. Group Description: A=Active LN group; N=Active SLE without renal involvement group; C=Inactive SLE group; H=healthy control group