inflammation and predict future GFR, they could be used to 1) monitor treatment response/failure, 2) allow early treatment changes, and 3) they could serve as surrogate endpoints in clinical trials.

307 THE QUEST FOR BIOMARKERS IN CUTANEOUS LUPUS ERYTHEMATOSUS

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Clinical challenges abound in cutaneous lupus erythematosus (CLE). Diagnosis of CLE is not straightforward because of its varied presentations and mimicry of other skin diseases. Up to half of CLE patients are recalcitrant to first-line treatments such as antimalarials. CLE patients who do not have SLE can later progress to SLE, but it is not evident which ones are at higher risk. Identification of key biomarkers in CLE can help address these conundrums. For instance, the anti-SS-A antibody has been associated with subacute CLE patients, which was first observed by Drs. James Gilliam and Richard Sontheimer in our institution. Type I interferon- inducible proteins such as MxA and guanylate binding protein-1 and the chemokines CXCL9 and CXCL10 have been proposed as biomarkers that may support diagnosis and track disease activity. Recently, biomarker candidates have emerged that may assist in determining treatment response to oral antimalarials. For example, an increased myeloid dendritic cell population with higher TNF-α expression may be predictive of poor treatment response to hydroxychloroquine in CLE patients. Finally, biomarkers can carry key prognostic information for CLE patients.

Autoantibodies against nuclear antigens (e.g. anti-double-stranded DNA and anti-Smith antibodies) and elevated erythrocyte sedimentation rate and low complement have been more often identified in CLE patients who develop SLE. Further studies are needed to confirm roles of these various candidate biomarkers and their involvement in CLE pathogenesis.

308 TRANSCRIPTIONAL PROFILES ASSOCIATED WITH PERICARDITIS IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS (SLE) IDENTIFY PURINE METABOLISM PATHWAY GENES AS CANDIDATE MEDIATORS

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Background Pericarditis is a frequent complication of systemic lupus erythematosus (SLE) with potential for important compromise of cardiac function. Pericarditis is characterized by chest pain, pericardial friction rub, and detectable changes on electrocardiography or transthoracic echocardiogram. Its prevalence in SLE patients ranges from 12% to 56%, and its underlying mechanisms remain poorly understood. Treatment with glucocorticoids is commonly used to resolve pericardial effusions.

Methods 263 SLE patients with a minimum of 5-years of follow-up were studied, identifying individuals with a history of pericarditis based on clinical presentation and/or documented moderate to severe effusion. Gene expression analysis was conducted on prospectively collected longitudinal blood samples from 15 SLE patients with pericarditis history and 51 SLE patients without such history as controls, totaling 329 blood samples (an average of 5 samples per patient in each group). Differentially expressed genes were identified using limma and lme4 models to account for random effects.

Results There were no prominent demographic differences between the patient groups. However, patients of African ancestry were more represented in the pericarditis group (40% vs. 32%). The pericarditis group showed a higher rate of class IV lupus nephritis (27% vs. 16%) and increased incidence of myocardial infarction (4% vs. 1%), cardiomyopathy (6% vs. 1%), and deforming or erosive arthritis (14% vs. 9%), but a lower incidence of diabetes (1% vs. 6%). Gene expression analysis identified 82 differentially expressed genes between the groups (FDR 5%), with the *COX7A1* mitochondrial gene being the most significantly downregulated gene in pericarditis patients. Notably, purine metabolism-related genes (*FHIT*, *PAPSS1*, *PDE9A*, *GUCY1A2*, *NT5C3B*) were affected among pericarditis patients according to KEGG pathway enrichment analysis. Laboratory data also indicated higher uric acid levels among pericarditis patients (OR 1.68 (0.54–2.81), p=0.006).

Discussion Pericarditis has known associations with uremia in gout disease and kidney failure, but a role for uric acid as a primary factor in predisposing SLE patients to pericarditis remains unreported. Interestingly, colchicine, recommended for acute and recurrent pericarditis in the general population, has shown success in treating SLE patients with pericarditis. Moreover, the downregulation of *COX7A1* in peripheral blood might reflect the inflamed pericardium and its impaired serosal tissue function among SLE patients with pericarditis.

309 SERUM MARKERS OF DISEASE ACTIVITY CORRELATE WITH PAIN PERCEPTION IN SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS

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Background/Purpose Systemic lupus erythematosus (SLE) is an autoimmune disease often characterized by multiorgan involvement with pain as a prevalent symptom. However, pain severity can differ greatly in each presentation.[1] While multifactorial, one mechanism is related to inflammatory processes which cause nociceptive receptor activation and central nervous system alteration.[2] Given the multitude of possible signaling pathways, there are still additional contributing mechanisms and factors that have yet to be fully determined. To add to the growing literature, we analyzed the inflammatory markers of SLE patients to explore their relation to varying degrees of pain.