with resolution of AKI, we observed a persistence of macrophages and T cells (>10days).

Conclusions: The amount of renal epithelial cells in the urine seems to directly reflect tissue damage in AKI. Immune cells can be monitored in the urine of patients with AKI and persisted longer than the renal injury itself. This suggests that immune cells participate in mediating damage and tissue repair in human AKI.

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THE ROLE OF B7.1 AND suPAR IN PODOCYTE INJURY AND FOCAL SEGMENTAL GLOMERULOSCLEROSIS

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Introduction: The mechanism of injury to podocytes in focal segmental glomerulosclerosis (FSGS) remains unclear. Both B7.1 (CD80) and suPAR have been proposed to cause FSGS but these findings have not been validated. To survey these markers as potential mediators of podocyte injury and as an aid to histologic diagnosis of FSGS we evaluated the immunohistochemical expression of (1) B7-1 and suPAR in native and transplant kidney biopsies, and (2) suPAR in an experimental mouse model of FSGS.

Methods: Consecutive kidney biopsies from the surgical pathology files were selected for study groups of FSGS (n=10) and early post-transplant recurrent FSGS (n=10). Native kidney biopsies with membranous nephropathy (MN) (n=10) and minimal change disease (MCD (n=5), and 6 month post-transplant protocol biopsies with normal morphology [n=10] served as controls. Immunostains for B7.1 and suPAR were performed on formalin-fixed paraffin-embedded and frozen sections with immunoperoxidase (IHC) or immunofluorescent (IF) methodology and the localization of the positive signal was determined via co-stain with podocyte marker synaptopodin. Appropriate positive method controls were available. In addition, uPAR expression was assessed in the kidneys of wild type and suPAR deficient (suPAR -/-) mice infused with suPAR. The mouse kidneys were also evaluated by electron microscopy in conjunction with renal functional studies.

Results: B7.1 was not expressed in native kidneys with FSGS or MCD or in transplant kidneys with recurrent FSGS. In MN, B7.1 was localized only to the immune deposits. No apparent suPAR immunoreactivity was present in native kidneys or recurrent FSGS. suPAR infusion did not produce proteinuria or effacement of podocytes in wild type or suPAR deficient mice. uPAR was detected by IHC along the glomerular endothelial cells but not in podocytes in wild type mice while uPAR stains remained negative in suPAR deficient mice even after suPAR injection.

Conclusions: The data suggest that B7.1 and suPAR may not play a significant role in podocyte injury in native and transplant kidneys with FSGS. B7.1 and suPAR immunostains have a limited value as diagnostic immunophenotypical markers in routine kidney biopsies with FSGS and recurrent FSGS post-transplant.

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MARKERS OF OXIDATIVE STRESS IN PATIENTS OF SYSTEMIC LUPUS ERYTHEMATOSUS WITH OR WITHOUT LUPUS NEPHRITIS

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Introduction: The relevance of oxidative stress in systemic lupus erythematosus (SLE) in humans is not fully understood. Moreover, none of the previous studies have specifically studied the impact of oxidative stress in patients of lupus nephritis. We conducted this study to evaluate the markers of oxidative stress in patients of SLE with or without lupus nephritis and to correlate them with clinical presentation, disease activity, and type & severity of renal manifestations.

Methods: This was a prospective study including patients diagnosed to have SLE on the basis of ACR criteria. The study participants were divided into two groups depending on presence or absence of renal involvement, which was defined as either abnormalities in renal function test (serum creatinine ≥1.2mg/dl) or urine examination (urine protein excretion ≥500mg/day or presence of RBCs in urine) along with renal biopsy evidence of lupus nephritis. After inclusion, patient's particulars were noted including clinical manifestations, investigations, SLE Disease Activity Index (SLEDAI), details of renal involvement, renal biopsy, treatment received and the outcome. In both the groups, an additional 5 ml of venous blood sample was drawn after overnight (8-10 hours) fasting for analyzing the markers of oxidative stress. We studied Malondialdehyde (MDA) and Isoprostane as markers of lipid peroxidation; reactive nitrogen intermediates (RNI) as markers of oxidative protein damage; 8-hydroxy deoxy guanosine as marker of oxidative DNA damage and Superoxide dismutase (SOD) as a marker of antioxidant activity.

Results: During the study period a total of 120 cases were included (60 without lupus nephritis, 60 with lupus nephritis). The mean age of study population was 29 ± 9 years and 115 were females. SLEDAI was significantly higher in patients with nephritis as compared to non-nephritis group. The mean values of serum MDA, 8-Isoprostane, 8-Hydroxy 2-deoxyguanosine and RNI were significantly higher in nephritis group, while there was no difference in the levels of SOD. Significant positive correlations were observed between RNI and SLEDAI score ($r = 0.333$, $p<0.01$), and MDA and SLEDAI score ($r = 0.269$, $p<0.01$), while none of the markers showed significant correlation with serum creatinine. There was no significant correlation between these markers and severity of renal dysfunction, while 8-Hydroxy 2-deoxy guanosine levels showed significant negative correlation with degree of proteinuria ($r= -0.345$; p value 0.007). MDA levels were significantly higher in patients with proliferative lupus nephritis as compared to non proliferative nephritis. During six months follow-up, we noted 7 deaths in nephritis group as compared to none in non-nephritis group. The mean value of 8-Hydroxy 2-deoxy guanosine was significantly higher in those who died; however there was no significant difference in other oxidative markers.

Conclusions: SLE patients with nephritis have higher levels of oxidative stress. Increased oxidative stress in nephritis patients correlates more with SLEDAI than with type or severity of renal dysfunction.

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IL-2 THERAPY REDUCES RENAL INFLAMMATION AND CELLULAR ACTIVITY OF INTRARENAL CD4+ CONVENTIONAL T CELLS IN LUPUS