A combination of three biomarkers may be useful for monitoring interstitial inflammation in patients with lupus nephritis, according to a report in Kidney International.

The researchers collected urine samples from 61 patients with lupus nephritis, at or around the time of renal biopsy. All patients met at least four American College of Rheumatology criteria for systemic lupus erythematosus, including immune complex glomerulonephritis. A renal pathologist graded interstitial inflammation and interstitial fibrosis in 64 biopsy specimens. Linear discriminant analysis was performed to evaluate various urinary biomarkers for inclusion in a “composite biomarker” of interstitial inflammation.

The composite biomarker of tubulointerstitial inflammation consisted of monocyte chemotactic protein-1; hepcidin, which reflects lupus nephritis flares; and liver fatty acid-binding protein.

Sensitivity was 100 percent, specificity 81 percent, positive predictive value 67 percent, and negative predictive value 100 percent. The composite biomarker had a misclassification rate of only 14 percent.

Renal biopsy is typically performed at diagnosis of lupus nephritis but for subsequent disease flares. Accurate, noninvasive indicator of kidney injury—particularly interstitial inflammation—would be helpful in planning and monitoring medical treatment.

The new composite biomarker shows promise for use in monitoring tubulointerstitial inflammation in lupus nephritis. Although further validation is needed, the authors believe that the biomarker could provide useful information about the renal interstitium in other kidney diseases as well [Zhang X, et al. A composite biomarker was developed in Transplantation.

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For kidney transplant patients going through rapid steroid withdrawal, switching from tacrolimus to sirolimus doesn’t reduce the rate of long-term changes on subsequent renal biopsy specimens, according to a report in Transplantation.

The randomized controlled trial included 122 kidney transplant recipients undergoing rapid steroid withdrawal. At 1 month, the patients were assigned to switch from tacrolimus to sirolimus or to remain taking tacrolimus. Protocol biopsy specimens were obtained at 1 month, 1 year, and 2 years for assessment of long-term changes, including interstitial fibrosis and tubular atrophy (IFTA) and the sum of Banff chronic changes (Total Score). The influence of previous rejection episodes on the long-term changes was assessed as well.

One-year biopsy specimens were obtained from 90 percent of patients in both groups. The two groups had similar and significant increases in long-term changes—i.e., proportion of biopsy specimens with IFTA scores of 2 or greater and Total Scores greater than 2. At 1 year, patients who had previous episodes of rejection and who continued to receive tacrolimus had higher IFTA scores and were more likely to have Total Scores greater than 2. Among those without previous rejection, both the IFTA and Total Score showed significant progression from 1 to 2 years.

Chronic calcineurin inhibitor nephrotoxicity contributes to the development of IFTA after kidney transplantation. In a previous report, the authors found no difference in 1-year kidney function among patients converted from tacrolimus to sirolimus 1 month after transplantation.

The new analysis showed no reduction in the progression of IFTA and other long-term changes through 2 years in kidney recipients who switched to sirolimus, compared long-term changes with those maintaining with tacrolimus. This was so even in patients with no previous episodes of rejection. Further study of the progression of long-term changes after early steroid withdrawal is needed [Heilman RL, et al. Impact of early conversion from tacrolimus to sirolimus on chronic allograft changes in kidney recipients on rapid steroid withdrawal. Transplantation 2012; 93:47–53].