CJASN
Clinical Journal of the American Society of Nephrology

September 2014 • Vol. 9 • No. 9

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On the Cover

What's the diagnosis? A 33-year-old woman with unremarkable past medical history except for a diagnosis of atypical migraine headaches 6 months ago recently noted the acute onset of mild ankle swelling that rapidly worsened and progressed to involve her upper thighs. In addition, she noted increasing fatigue and malaise, 25 pound weight gain, and loss of appetite. Her only medications included oral contraception pill, multi-vitamins, and occasional naproxen for headaches. On evaluation in an ambulatory care clinic, physical examination revealed 3+ pitting edema of her lower extremities including her thighs. There were no joint swelling, skin rash or other abnormal exam findings. Serum chemistries were notable for normal electrolytes, BUN 45 mg/dl, serum creatinine 4.3 mg/dl, and serum albumin 2.1 g/dl. Urinalysis revealed SG 1.014, pH 5.5, 3+ protein, negative blood, and trace leukocytes. Urine microscopy demonstrated 1-3 renal tubular epithelial cells/HPF, 0-2 white blood cells/HPF, 2-6 oval fat bodies /HPF, and 1-2 lipid casts/LPF. 24-hour urine collection contained 8.6 grams of protein. The patient underwent kidney biopsy for AKI and nephrotic syndrome, which revealed diffuse interstitial infiltrate containing lymphocytes, PMNs and eosinophils (left panel) consistent with drug-induced acute interstitial nephritis (AIN). The right panel shows globally effaced foot processes without deposits consistent with minimal change disease (MCD). The combination of eosinophil-dominant AIN and MCD pointed to naproxen as the underlying cause of the kidney lesion. Naproxen was discontinued and oral prednisone (60 mg/day) was administered for 4 weeks followed by taper with improvement in kidney function (serum creatinine 1.3 mg/dl) and resolution of proteinuria. NSAID-induced renal syndromes include a number of electrolyte/acid-base and other kidney lesions. In this case, naproxen caused 2 of those lesions—AIN and MCD. The mechanism by which these 2 lesions develop with NSAIDs is not known, but is speculated to be due to shunting of arachidonic acid pathway into the lipoxygenase pathway with production of proinflammatory and vasoactive leukotrienes. It is possible that leukotrienes promote interstitial inflammation and injure glomerular epithelial cells (podocytes). Therapy consists of NSAID discontinuation, and steroids if kidney function and nephrotic proteinuria do not improve. In general, patients recover from this renal lesion, but permanent chronic kidney disease may occur later. (Images and text provided by Gilbert Moeckel, MD and Mark A. Perazella, MD, Yale University School of Medicine, New Haven, Connecticut)