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Source of Post-Transplant Care and Mortality among Kidney Transplant Recipients Dually Enrolled in VA and Medicare
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Impact of COVID-19 Pandemic in Children with CKD or Immunosuppression
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Outcomes of Patients on Maintenance Dialysis Hospitalized with COVID-19
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Correction: Infection-Related Acute Care Events among Patients with Glomerular Disease

GWAS-Based Discoveries in IgA Nephropathy, Membranous Nephropathy, and Steroid-Sensitive Nephrotic Syndrome
Elena Sanchez-Rodiguez, Christopher T. Southard, and Krzysztof Kiryluk

Resistant Hypertension in CKD
George Thomas and Mahboob Rahman

Children with CKD Are Not Little Adults with CKD: Pediatric Considerations for the Advancing American Kidney Health Initiative
Alexander J. Kula and Michael J.G. Somers, on behalf of the American Society of Pediatric Nephrology

Challenges with Providing Hospice Care for Patients Undergoing Long-Term Dialysis
Jane O. Schell and Douglas S. Johnson

Uric Acid and CKD Progression Matures with Lessons for CKD Risk Factor Discovery
Oluwaseun Oluwo and Julia J. Scialla

Preprint Servers in Kidney Disease Research: A Rapid Review
Caitlyn Vlasschaert, Cameron Giles, Swapnil Hiremath, and Matthew B. Lanktree
On the Cover

What is the diagnosis?

A 72-year-old patient with recent history of systemic lambda light-chain AL amyloidosis under chemotherapy, malabsorptive diarrhea, and normal baseline kidney function was admitted for acute kidney failure. The laboratory tests showed that serum creatinine raised from 1.13 mg/dl to 3.97 mg/dl, and proteinuria was at 0.18 g/24h with a small passage of albumin and lambda monoclonal light chains in urine and normal serum light chains.

Image Description:

With light microscopy (left image: hematoxylin staining, middle image and right image: Congo red staining and polarized light; ×300), histopathology showed birefringent crystals in several tubules (arrowheads). Amyloid deposits were observed in medium size vessels (arrows) and at the vascular pole of a glomerulus (asterisk) and in scarcity in glomerular and tubule-interstitial compartments. Tubular atrophy was associated with interstitial fibrosis (25% of total surface).

Teaching Points:

These findings suggest the importance of considering acute oxalate nephropathy in the diagnosis of nonresolving acute kidney failure induced by diarrhea even in patients with another active kidney disease. The gastrointestinal amyloid infiltrations lead to lymphatic congestion resulting in lipid-rich lymph leakage into the intestinal lumen (1) ensuring a saponification process due to calcium, thereby oxalate free for absorption in the colon. Chronic dehydration, low calcium levels, and diarrheal loss of crystallization inhibitors such as magnesium and citrate favor crystallogenesis. The prognosis is poor, and the treatment consists in a low oxalate and fatty diet and correction of ionic disorders (2). Resumption of chemotherapy is important in order to regress amyloid infiltration in the digestive tract. Glomerular deposits exist in almost 97% of cases, leading to proteinuria in the nephrotic ranges and mainly composed of light chains (3). Vascular-limited deposits lead to low-grade proteinuria and much worse kidney prognosis because the lack of proteinuria may delay the diagnosis and the treatment; therefore, kidney biopsy should be performed.

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