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Efficacy and Cardiovascular Safety of Roxadustat for Treatment of Anemia in Patients with Non–Dialysis-Dependent CKD: Pooled Results of Three Randomized Clinical Trials

Robert Provenzano, Lynda Szczech, Robert Leong, Khalil G. Saikali, Ming Zhong, Tyson T. Lee, Dustin J. Little, Mark T. Houser, Lars Frison, John Houghton, and Thomas B. Neff

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Effect of Vitamin D Supplementation on Kidney Function in Adults with Prediabetes: A Secondary Analysis of a Randomized Trial


Latency, Anti-Bacterial Resistance Pattern, and Bacterial Infection–Related Glomerulonephritis


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HLA Alleles and Prognosis of PLA2R-Related Membranous Nephropathy

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The Effects of Oral Energy-Dense Supplements on Nutritional Status in Nondiabetic Maintenance Hemodialysis Patients: A Randomized Controlled Trial

Yaya Yang, Xianhui Qin, Junzhi Chen, Qi Wang, Yaozhong Kong, Qijun Wan, Huiqin Tao, Aiqun Liu, Youbao Li, Zizhen Lin, Yan Huang, Yanhuan He, Zihan Lei, and Min Liang

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Risk of COVID-19 Disease, Dialysis Unit Attributes, and Infection Control Strategy among London In-Center Hemodialysis Patients

Ben Caplin, Damien Ashby, Kieran McCAfferty, Richard Hull, Elham Asgari, Martin L. Ford, Nicholas Cole, Marilina Antonelou, Sarah A. Blakey, Vinay Srinivasa, Dandisonba C.B. Braide-Azikwe, Tayeba Roper, Grace Clark, Helen Cronin, Nathan J. Hayes, Bethia Manson, Alexander Sarawkois, Richard Corbett, Kate Bramham, Eirini Lioudaki, Nicola Kumar, Andrew Frankel, David Makansjuola, Claire C. Sharpe, Debasish Banerjee, and Alan D. Salama, on behalf of the Pan-London COVID-19 Renal Audit Group

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Recurrence of IgA Nephropathy after Kidney Transplantation in Adults


Pharmacokinetics of Remdesivir and GS-441524 during PIRRT and Seraph 100 Therapy

Julius J. Schmidt, Stefanie M. Boden-Böger, Jens Martens-Lohenhofer, Marius M. Hoepner, and Jan T. Kielstein

Antibody Response to mRNA-1273 Sars-CoV-2 Vaccine in Hemodialysis Patients with and without Prior COVID-19

Lili Chan, Nicholas Fuca, Etti Zeldis, Kirk N. Campbell, and Aisha Shaikh

Correction

Introduction to Kidney Transplantation: Long-Term Management Challenges

Deirdre Sawinski and Emilio D. Poggio
Case Description:
What is the Diagnosis?
An 18-year-old female, diagnosed with seronegative polyarticular juvenile inflammatory arthritis at 4 years of age, presented with anasarca. She had been treated with methotrexate and nonsteroidal anti-inflammatory drugs (NSAIDs) for the past 9 years. On examination, she had an inflammatory, nondeforming symmetrical polyarthritis involving large joints, mainly the knees. Her serum creatinine was 2.1 mg/dl and 24-hour urine protein was 3.9 g. ANA was negative. C3 and C4 were normal.

Image Description:
Kidney biopsy showed the deposition of amorphous, acellular, eosinophilic material in the glomeruli (left) and on the basement membrane of some tubules and the walls of blood vessels. These deposits on Congo red staining gave an orange-red appearance under light microscopy, apple green birefringence under polarized light, and intense red fluorescence under fluorescence microscopy using a Texas red filter (middle). Immunofluorescent staining showed no deposition of immunoglobulins, complements, or light chains. The amyloid subtype was confirmed as AA amyloid by immunohistochemistry (right).

Teaching Points:
The fibrils in various types of amyloidosis have similar appearance under light and electron microscopy. They all generate birefringence under polarized light with Congo red dye. Immunohistochemistry can be used to determine amyloid subtype. Laser-capture microdissection followed by mass spectrometry is the new diagnostic tool for amyloid typing. Amyloidosis occurring in childhood is extremely rare and is usually of AA type, complicating chronic inflammatory diseases. Proteinuria in children with juvenile inflammatory arthritis could be due to the use of NSAIDs, antirheumatoïd drugs such as gold and penicillamine, and certain indigenous medicines. AA amyloidosis may develop in children who have long-standing, poorly controlled inflammatory arthritis. It is important to recognize the cause of proteinuria in these patients. AA amyloidosis occurs as a reaction to a chronic inflammatory condition. Infections and inflammation cause the liver to produce serum amyloid A (SAA) protein, an acute phase reactant protein, in high levels. When the inflammation persists, a portion of the SAA protein called the AA protein may be deposited in various tissues as insoluble AA amyloid fibrils. The kidney is the most affected organ in AA amyloidosis, leading to proteinuria, nephrotic syndrome, or kidney impairment. It is a progressive disease leading to kidney failure.

In addition to controlling the inflammation, treatment options for AA amyloidosis include colchicine and antiproinflammatory cytokines like IL1β, TNF, and IL-6. Since there was no active inflammatory process at present, antiproinflammatory treatment options were not considered. She is currently on diuretics, salt restriction, and angiotensin-converting enzyme inhibitors. Renal transplantation may be considered later.

(Text and images provided by Anila Abraham Kurien, Department of Pathology, Renopath Center for Renal and Urological Pathology, Chennai, India; Jerry Joseph, Department of Nephrology, Stanley Medical College, Chennai, India; and Edwin Fernando, Department of Nephrology, Stanley Medical College, Chennai, India.)