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Editorials

1501 BP Targets in Older Adults with CKD: Additional Evidence, but Uncertainty Continues

Sankar D. Navaneethan and Adam Whaley-Connell

See related article on page 1553.

1504 Lifetime Risk of CKD: What Does It Really Mean?

Pierre Delanaye and Richard J. Glassock

See related article on page 1575.

1507 The Other Half of Informed Consent: Transplant Education Practices in Dialysis Centers

Lauren M. Kucirka and Dorry L. Segev

See related article on page 1617.

Original Articles

Acute Kidney Injury

1510 Epidemiology and Clinical Correlates of AKI in Chinese Hospitalized Adults

Xin Xu, Sheng Nie, Zhangsuo Liu, Chunbo Chen, Gang Xu, Yan Zha, Jing Qian, Bicheng Liu, Shuai Han, Anping Xu, Xing Xu, and Fan Fan Hou


1519 Randomized Trial of Bicarbonate or Saline Study for the Prevention of Contrast-Induced Nephropathy in Patients with CKD

Richard Solomon, Paul Gordon, Steven V. Manoukian, J. Dawn Abbott, Dean J. Kereiakes, Allen Jeremias, Michael Kim, Harold L. Dauerman, on behalf the BOSS Trial Investigators

Chronic Kidney Disease

1525 Associations of Soluble CD14 and Endotoxin with Mortality, Cardiovascular Disease, and Progression of Kidney Disease among Patients with CKD

Ruben Poesen, Ali Ramezani, Kathleen Claes, Patrick Augustijns, Dirk Kuypers, Ian R. Barrows, Jagadeesan Muralidharan, Pieter Evenepoel, Björn Meijers, and Dominic S. Raj

1534  Pravastatin Therapy and Biomarker Changes in Children and Young Adults with Autosomal Dominant Polycystic Kidney Disease

Jelena Klawitter, Kim McFann, Alexander T. Pennington, Wei Wang, Jost Klawitter, Uwe Christians, Robert W. Schrier, Berenice Gitomer, and Melissa A. Cadnapaphornchai

1542 Meta-Analysis of the Effect of Dietary Sodium Restriction with or without Concomitant Renin-Angiotensin-Aldosterone System-Inhibiting Treatment on Albuminuria

Lafranco D'Elia, Giovanni Rossi, Michele Schiano di Cola, Ivana Savino, Ferruccio Galletti, and Pasquale Strazzullo

1553 Systolic BP and Mortality in Older Adults with CKD

Jessica W. Weiss, Dawn Peters, Xiuhai Yang, Amanda Petrik, David H. Smith, Eric S. Johnson, Micah L. Thorp, Cynthia Morris, and Ann M. O'Hare

See related editorial on page 1501.

Clinical Nephrology

1560 Asymmetric and Symmetric Dimethylarginine and Sympathetic Nerve Traffic after Renal Denervation in Patients with Resistant Hypertension

Guido Grassi, Gino Seravalle, Fosca Quarti Trevano, Domenico Spaziani, Filippo Scalise, Carla Auguadro, Patrizia Pizzini, Giovanni Tripepi, Graziella D'Arrigo, Francesca Mallamaci, Giuseppe Mancina, and Carmine Zoccali

Original Articles (Continued)

Diabetes and the Kidney

1568 Predictors of Atrasentan-Associated Fluid Retention and Change in Albuminuria in Patients with Diabetic Nephropathy

Donald E. Kohan, Hiddo J. Lambers Heerspink, Blai Coll, Dennis Andress, John J. Brennan, Dalane W. Kitzman, Ricardo Correa-Rotter, Hirofumi Makino, Vlado Perkovic, Fan Fan Hou, Giuseppe Remuzzi, Sheldon W. Tobe, Robert Toto, Hans-Henrik Parving, and Dick de Zeeuw

Epidemiology and Outcomes

1575 Lifetime Risk of Stage 3–5 CKD in a Community-Based Sample in Iceland

Lesley A. Inker, Hocine Tighiouart, Thor Aspelund, Vilundur Gudnason, Tamara Harris, Olafur S. Indridason, Runolfur Palsson, Shani Shastri, Andrew S. Levey, and Mark J. Sarnak
See related editorial on page 1504.

1585 Risk of Stroke in Patients with ESRD

Philip Masson, Patrick J. Kelly, Jonathan C. Craig, Richard I. Lindley, and Angela C. Webster

1593 Nutritional Competence and Resilience among Hemodialysis Patients in the Setting of Dialysis Initiation and Hospitalization

Stephan Thijssen, Michelle M.Y. Wong, Len A. Usvyat, Qingqing Xiao, Peter Kotanko, and Franklin W. Maddux

ESRD and Chronic Dialysis

1602 The Safety of Eplerenone in Hemodialysis Patients: A Noninferiority Randomized Controlled Trial

Michael Walsh, Braden Manns, Amit X. Garg, Joe Bueti, Christian Rabbat, Andrew Smyth, Jessica Tyrwhitt, Jackie Bosch, Peggy Gao, P.J. Devereaux, and Ron Wald

Ethics

1609 Disclosing Health and Health Behavior Information between Living Donors and Their Recipients

Leslie Mataya, Jacqueline Meadow, J. Richard Thistlethwaite Jr, Didier A. Mandelbrot, James R. Rodrigue, and Lainie Friedman Ross

Health Services Research

1617 Assessing Transplant Education Practices in Dialysis Centers: Comparing Educator Reported and Medicare Data

Amy D. Waterman, John D. Peipert, Christina J. Goalby, Katrina M. Dinkel, Huiling Xiao, and Krista L. Lentine
See related editorial on page 1507.

Renal Physiology

1626 Chemical and Physical Sensors in the Regulation of Renal Function

Jennifer L. Pluznick and Michael J. Caplan

Renal Immunology

1636 Molecules Great and Small: The Complement System

Douglas R. Mathern and Peter S. Heeger

Role of the Medical Director

1651 Legal Issues for the Medical Director

William G. Trulove

Moving Points in Nephrology

1656 Living Donor Kidney Transplantation: Best Practices in Live Kidney Donation—Recommendations from a Consensus Conference

James R. Rodrigue, Dianne LaPointe Rudow, and Rebecca Hays

1659 Living Donor Kidney Transplantation: Improving Education Outside of Transplant Centers about Live Donor Transplantation—Recommendations from a Consensus Conference

Amy D. Waterman, Marie Morgievlch, David J. Cohen, Zeeshan Butt, Harini A. Chakkerla, Carrie Lindower, Rebecca E. Hays, Janet M. Hiller, Krista L. Lentine, Arthur J. Matas, Emilio D. Poggio, Michael A. Rees, James R. Rodrigue, and Dianne LaPointe Rudow

1670 Living Donor Kidney Transplantation: Facilitating Education about Live Kidney Donation—Recommendations from a Consensus Conference

Jane C. Tan, Elisa J. Gordon, Mary Amanda Dew, Dianne LaPointe Rudow, Robert W. Steiner, E. Steve Woodle, Rebecca Hays, James R. Rodrigue, and Dorry L. Segev

1678 Living Donor Kidney Transplantation: Improving Efficiencies in Live Kidney Donor Evaluation—Recommendations from a Consensus Conference

Deonna R. Moore, David Serur, Dianne LaPointe Rudow, James R. Rodrigue, Rebecca Hays, and Matthew Cooper

1687 Living Donor Kidney Transplantation: Overcoming Disparities in Live Kidney Donation in the US—Recommendations from a Consensus Conference


James R. Rodrigue, Abby Swanson Kazley, Didier A. Mandelbrot, Rebecca Hays, Dianne LaPointe Rudow, and Prabhakar Baliga

1696 Living-Donor Kidney Transplantation: Reducing Financial Barriers to Live Kidney Donation—Recommendations from a Consensus Conference

Lara Tushla, Dianne LaPointe Rudow, Jennifer Milton, James R. Rodrigue, Jesse D. Schold, and Rebecca Hays

Erratum

1703 Correction

 eJournal club provides a timely and interactive electronic journal club experience by offering a forum in which CJASN readers have the opportunity to converse with the featured study authors. Visit ejc.cjasn.org to learn more.

On the Cover

What's the diagnosis? A previously healthy 18 year old male presented with leg swelling for three months, progressing to anasarca and hypertension. There was no family history of renal disease. Urinalysis revealed hemoglobin +2/+4 and the 24 hour urine collection contained 6.1g of protein. His serum creatinine was 0.80 mg/dl, cholesterol 268 mg/dl, high-density lipoprotein 18 mg/dl, and albumin 2.7 g/dl. Serum complement levels were normal and evaluation of secondary causes of the nephrotic syndrome was negative. Renal biopsy was performed. Light microscopy showed global endocapillary hypercellularity, mainly due to mesangial cells and eosinophilic deposits in subendothelial and subepithelial (granular pattern) locations. With silver stain, these deposits were negative and double contour of the basement membrane and spikes were seen (cover image). There were a few foci of interstitial fibrosis with rare atrophic tubules and no vascular lesions. Direct immunofluorescence was negative except for C3, which had strong mesangial staining and granular pattern in capillary loops, both subendothelial and subepithelial, global and diffuse in the glomeruli, and in vascular walls. Electron microscopy showed glomerular basement membrane vacuoles containing electron-lucent and electron-dense deposits (image below) compatible with Lecithin Cholesterol Acyltransferase Deficiency. The patient was treated with furosemide, captopril, prednisone and simvastatin with improvement of the edema and reduction of daily proteinuria to 2g. LCAT deficiency is a rare genetic disease with estimated prevalence of 1:100000 population. There are 88 genes mutations associated with the disease. This is an autosomal recessive disorder characterized by abnormalities in lipoprotein metabolism, leading to diminished formation of high-density lipoproteins and production of lipoprotein-X. These lipoproteins are deposited in the cornea and kidney, leading to progressive dysfunction. Other clinical findings include severe anemia and dyslipidemia. In the kidney, the deposits may be seen in glomeruli, arteries and arterioles. The ideal treatment would be enzyme replacement or LCAT gene replacement therapy however these options are not yet available in clinical practice. The current treatment is based in the management of nephrotic syndrome with statins, angiotensin II receptor blockers or angiotensin converting enzyme inhibitors and corticosteroids. The recurrence of the renal abnormalities after kidney transplantation is inevitable. (Images and text provided by Precil Diego Miranda de Menezes Neves, Federal University of Triângulo Mineiro - Nephropathology Service Uberaba, Minas Gerais, Brazil and colleagues) A complete list of authors is available as Supplemental Information.

