Editorials

1805 Change in Kidney Function over Time and Risk for Adverse Outcomes: Is an Increasing Estimated GFR Harmful?
Tanvir Chowdhury Turin and Brenda R. Hemmelgarn
See related article on page 1879.

1807 Cardiovascular Evaluation before Renal Transplantation: To Cath or Not to Cath?
Oscar C. Marroquin and Steven Weisbord
See related article on page 1912.

1810 GFR Estimation in Children: Questions and Answers (and Questions)
Jeffrey J. Fadrowski and Susan L. Furth
See related article on page 1956.

1813 Measured GFR as “Gold Standard”—All that Glitters Is Not Gold?
Chi-yuan Hsu and Nisha Bansal
See related article on page 1963.

Original Articles

Acute Renal Failure

1815 Urinary Biomarkers and Renal Recovery in Critically Ill Patients with Renal Support
Nattachai Srisawat, Xiaoyan Wen, MinJae Lee, Lan Kong, Michele Elder, Melinda Carter, Mark Unruh, Kevin Finkel, Anitha Vijayan, Mohan Ramkumar, Emil Paganini, Kai Singbartl, Paul M. Palevsky, and John A. Kellum

Chronic Kidney Disease

1824 Neurocognitive Functioning of Children and Adolescents with Mild-to-Moderate Chronic Kidney Disease
Stephen R. Hooper, Arlene C. Gerson, Robert W. Butler, Debbie S. Gipson, Susan R. Mendley, Marc B. Lande, Shlomo Shinnar, Alicia Wentz, Matthew Matheson, Christopher Cox, Susan L. Furth, and Bradley A. Warady

1831 Casual Blood Pressure and Neurocognitive Function in Children with Chronic Kidney Disease: A Report of the Children with Chronic Kidney Disease Cohort Study
Marc B. Lande, Arlene C. Gerson, Stephen R. Hooper, Christopher Cox, Matt Matheson, Susan R. Mendley, Debbie S. Gipson, Cynthia Wong, Bradley A. Warady, Susan L. Furth, and Joseph T. Flynn

1838 Chronic Kidney Disease Awareness Among Individuals with Clinical Markers of Kidney Dysfunction
Delphine S. Tuot, Laura C. Plantinga, Chi-yuan Hsu, Regina Jordan, Nilka Rios Burrows, Elizabeth Hedgeman, Jerry Yee, Rajiv Saran, and Neil R. Powe, for the Centers for Disease Control Chronic Kidney Disease Surveillance Team

1845 Effects of Antiproteinuric Intervention on Elevated Connective Tissue Growth Factor (CTGF/CCN-2) Plasma and Urine Levels in Nondiabetic Nephropathy
Maartje C.J. Slagman, Tri Q. Nguyen, Femke Waanders, Lilfert Vogt, Marc H. Hemmelder, Gozewijn D. Laverman, Roel Goldschmeding, and Gerjan Navis
Original Articles (Continued)

1851 Chronic Kidney Disease Stage Progression in Liver Transplant Recipients

1858 Racial and Ethnic Differences in Mortality among Individuals with Chronic Kidney Disease: Results from the Kidney Early Evaluation Program (KEEP)
Stacey E. Jolly, Nilka Ríos Burrows, Shu-Cheng Chen, Suying Li, Claudine T. Jurkovitz, Keith C. Norris, and Michael G. Shlipak

1866 Vision-Threatening Retinal Abnormalities in Chronic Kidney Disease Stages 3 to 5
Rajeev Deva, Mohamad Afzal Alias, Deb Colville, Foong Kien Newk-Fon Hey Tow, Qi Lun Ooi, Sky Chew, Nor Mohamad, Anastasia Hutchinson, Ignatios Koukouras, David A. Power, and Judith Savige

1872 The Microvasculature in Chronic Kidney Disease
Qi Lun Ooi, Foong Kien Newk-Fon Hey Tow, Raj Deva, Mohamad Afzal Alias, Ryo Kawasaki, Tien Y. Wong, Nor Mohamad, Deb Colville, Anastasia Hutchinson, and Judy Savige

1879 GFR Decline and Mortality Risk among Patients with Chronic Kidney Disease
Robert M. Perkins, Ion D. Bucaloiu, H. Lester Kirchner, Nasrin Ashouian, James E. Hartle, and Taher Yahya
See related editorial on page 1805.

Clinical Nephrology

1887 A Randomized Study of Allopurinol on Endothelial Function and Estimated Glomular Filtration Rate in Asymptomatic Hyperuricemic Subjects with Normal Renal Function
Mehmet Kanbay, Bulent Huddam, Alper Azak, Yalcin Solak, Gulay Kocak Kadioglu, Ismail Kirbas, Murat Duranay, Adrian Covic, and Richard J. Johnson

1895 Oxalate Nephropathy Associated with Chronic Pancreatitis
Claire Cartery, Stanislas Faguer, Alexandre Karras, Olivier Cointault, Louis Buscail, Anne Modesto, David Ribes, Lionel Rostaing, Dominique Chauveau, and Patrick Giraud

1903 Oxidative Stress and Galactose-Deficient IgA1 as Markers of Progression in IgA Nephropathy

1912 Cardiac Survival after Pre-emptive Coronary Angiography in Transplant Patients and Those Awaiting Transplantation
Nicola Kumar, Christopher S.R. Baker, Kakit Chan, Neill Duncan, Iqbal Malik, Andrew Frankel, Damien R. Ashby, Adam McLean, Andrew Palmer, Tom D. Caims, and David Taube
See related editorial on page 1807.

Dialysis

1920 The Relationship between Epicardial Adipose Tissue and Malnutrition, Inflammation, Atherosclerosis/Calcification Syndrome in ESRD Patients
Kultigin Turkmen, Hatice Kayikcioglu, Orhan Ozbek, Yalcin Solak, Mehmet Kayrak, Cigdem Samur, Melih Anil, and Halil Zeki Tonbul

1926 Cardiac Geometry in Children Receiving Chronic Peritoneal Dialysis: Findings from the International Pediatric Peritoneal Dialysis Network (IPPN) Registry
Sevcan A. Bakkaloglu, Dagma Ar Borych, Il Soo Ha, Erkin Serdaroglu, Rainer Büschler, Paulina Salas, Hiren Patel, Dorota Drozdz, Karel Vondrak, Andreia Watanabe, Jorge Villagra, Onder Yavascan, Maria Valenzuela, Deborah Gipson, K.H. Ng, Bradley A. Warady, and Franz Schaefer, for the International Pediatric Peritoneal Dialysis Network
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Dagmara Borzych, Sevcan A. Bakkaloglu, Joshua Zaritsky, Angela Suarez, William Wong, Bruno Ranchin, Cao Qi, Attila J. Szabo, Paula A. Coccia, Jérôme Harambat, Florin Mitu, Bradley A. Warady, and Franz Schaefer, for the International Pediatric Peritoneal Dialysis Network

1944 Geographic and Educational Factors and Risk of the First Peritonitis Episode in Brazilian Peritoneal Dialysis Study (BRAZPD) Patients
Luis C. Martin, Jacqueline C.T. Caramori, Natalia Fernandes, Jose C. Divino-Filho, Roberto Pecoits-Filho, and Pasqual Barretti, on behalf of the Brazilian Peritoneal Dialysis Multicenter Study BRAZPD Group

Epidemiology and Outcomes
1952 Systematic Shifts in Cystatin C Between 2006 and 2010
David M. Maahs, Diana Jalal, Kim McFann, Marian Rewers, and Janet K. Snell-Bergeon

Blanche M. Chavers, Michelle N. Rheault, and Robert N. Foley
See related editorial on page 1810.

1963 Relative Performance of the MDRD and CKD-EPI Equations for Estimating Glomerular Filtration Rate among Patients with Varied Clinical Presentations
Kazunori Murata, Nikola A. Baumann, Amy K. Saenger, Timothy S. Larson, Andrew D. Rule, and John C. Lieske
See related editorial on page 1813.

1973 Association of Hemoglobin and Survival in Peritoneal Dialysis Patients
Miklos Z. Molnar, Rajnish Mehrotra, Uyen Duong, Csaba P. Kovesdy, and Kamyar Kalantar-Zadeh

Austin G. Stack, Amir Mohammed, Alan Hanley, Arif Mutwali, and Hoang Nguyen

ESRD and Chronic Dialysis
1990 All-cause Mortality in Hemodialysis Patients with Heart Valve Calcification
Paolo Raggi, Antonio Bellasi, Christopher Gamboa, Emiliana Ferramosca, Carlo Ratti, Geoffrey A. Block, and Paul Muntner

1996 Outcomes of Arteriovenous Fistula Creation after the Fistula First Initiative
Carrie A. Schinstock, Robert C. Albright, Amy W. Williams, John J. Dillon, Eric J. Bergstralh, Bernice M. Jenson, James T. McCarthy, and Karl A. Nath

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Rajiv Agarwal, Arjun D. Sinha, and Robert P. Light

2009 Flow-Mediated Vasodilation in End-Stage Renal Disease

Hypertension
2016 Intradialytic Hypertension and its Association with Endothelial Cell Dysfunction
Jula K. Inrig, Peter Van Buren, Catherine Kim, Wanpen Vongpatanasin, Thomas J. Povsic, and Robert D. Toto

Renal Transplantation
2025 Monitoring of CD4+CD25$$^{\text{high}}$$IL-7Rα$$^{\text{high}}$$ activated T Cells in Kidney Transplant Recipients
Laure Vallotton, Karine Hadaya, Jean-Pierre Venetz, Leo H. Buehler, Donatella Ciuffreda, Ghaleb Nseir, Laura Codarri, Jean Villard, Giuseppe Pantaleo, and Manuel Pascual

2034 Long-term Outcome of Renal Transplantation Patients with Henoch-Schönlein Purpura
Joyce P. Samuel, Cynthia S. Bell, Donald A. Molony, and Michael C. Braun
Original Articles (Continued)

2041 Incompatible Live-Donor Kidney Transplantation in the United States: Results of a National Survey
Jacqueline M. Garonzik Wang, Robert A. Montgomery, Lauren M. Kucirka, Jonathan C. Berger, Daniel S. Warren, and Dorry L. Segev

Mini-Reviews

2047 Insights from the Chronic Kidney Disease in Children (CKiD) Study
Lawrence Copelovitch, Bradley A. Warady, and Susan L. Furth

2054 Should Living Kidney Donor Candidates with Impaired Fasting Glucose Donate?
Christine Buchek Vigneault, William Stuart Asch, Neera Kanhouwa Dahl, and Margaret Johnson Bia

Moving Points in Nephrology

2060 Nephrolithiasis—New Directions in 2011
Howard Trachtman

2062 An Update on the Changing Epidemiology and Metabolic Risk Factors in Pediatric Kidney Stone Disease
David J. Sas

2069 Chronic Kidney Disease in Kidney Stone Formers
Andrew D. Rule, Amy E. Krambeck, and John C. Lieske

2076 Role of the Calcium-Sensing Receptor in Reducing the Risk for Calcium Stones
Kirsten Y. Renkema, René J.M. Bindels, and Joost G.J. Hoenderop

2083 Pathophysiology-Based Treatment of Idiopathic Calcium Kidney Stones
Fredric L. Coe, Andrew Evan, and Elaine Worcester

2093 Potential Pharmacologic Treatments for Cystinuria and for Calcium Stones Associated with Hyperuricosuria
David S. Goldfarb

On the Cover

What’s the diagnosis? Sickle cell anemia occurs due to a mutation in the globin gene that causes the replacement of one pair of amino-acid residues, resulting in drastically reduced solubility of deoxyhemoglobin S and its precipitation in the red blood cells. Affected red blood cells become elongated and rigid; this deformation and stiffness is the distinguishing feature of sickle cell anemia and is the primary cause of the symptoms. The pictured electronmicrograph shows a glomerular capillary loop filled with distorted red blood cells in a patient during sickle cell crisis. Normal red cells show dark and homogenous cytoplasm ultrastructurally, while sickled cells are of elongated and sometimes bizarre shapes, and reveal intracytoplasmic fibers organized in bundles; occasional vacuoles can also be seen in the cytoplasm of the abnormal red blood cells. These changes in the structure and shape of red blood cells result in injury to the endothelium and, over time, the glomerular capillary walls can show signs of remodeling, reduplication of the basement membranes, and a membranoproliferative pattern of glomerular injury, without electron-dense deposits - changes typically seen in patients with repetitive endothelial cell injury and chronic thrombotic microangiopathies. (Image and text provided by Dr. Vanesa Bijol, Brigham and Women’s Hospital.)