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1831 Casual Blood Pressure and Neurocognitive Function in Children with Chronic Kidney Disease: A Report of the Children with Chronic Kidney Disease Cohort Study
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1866 Vision-Threatening Retinal Abnormalities in Chronic Kidney Disease Stages 3 to 5
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1872 The Microvasculature in Chronic Kidney Disease
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1920 The Relationship between Epicardial Adipose Tissue and Malnutrition, Inflammation, Atherosclerosis/Calcification Syndrome in ESRD Patients
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1926 Cardiac Geometry in Children Receiving Chronic Peritoneal Dialysis: Findings from the International Pediatric Peritoneal Dialysis Network (IPPN) Registry
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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

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1963 Relative Performance of the MDRD and CKD-EPI Equations for Estimating Glomerular Filtration Rate among Patients with Varied Clinical Presentations
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1973 Association of Hemoglobin and Survival in Peritoneal Dialysis Patients
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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

2003 Toward a Definition of Masked Hypertension and White-Coat Hypertension among Hemodialysis Patients
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+CD25highIL-7Rαhigh 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
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.)