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1920 The Relationship between Epicardial Adipose Tissue and Malnutrition, Inflammation, Atherosclerosis/Calcification Syndrome in ESRD Patients
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1944 Geographic and Educational Factors and Risk of the First Peritonitis Episode in Brazilian Peritoneal Dialysis Study (BRAZPD) Patients

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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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1990 All-cause Mortality in Hemodialysis Patients with Heart Valve Calcification

Paolo Raggi, Antonio Bellasi, Christopher Gamboa, Emiliana Ferramosca, Carlo Ratti, Geoffrey A. Block,
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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,
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2003 Toward a Definition of Masked Hypertension and White-Coat Hypertension among Hemodialysis Patients

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2009 Flow-Mediated Vasodilation in End-Stage Renal Disease


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

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.)