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1870 25 (OH) Vitamin D Levels and Renal Disease Progression in Patients with Type 2 Diabetic Nephropathy and Blockade of the Renin-Angiotensin System
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1884 Alternative Complement Pathway Activation Products in Urine and Kidneys of Patients with ANCA-Associated GN
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1892 Short-Term Changes after a Weight Reduction Intervention in Advanced Diabetic Nephropathy
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1899 Effect of Renin-Angiotensin System Blockade on Soluble Klotho in Patients with Type 2 Diabetes, Systolic Hypertension, and Albuminuria
Janaka Karalliedde, Giuseppe Maltese, Benjamin Hill, Giancarlo Viberti, and Luigi Gnudi

1907 Albuminuria and Cognitive Decline in People with Diabetes and Normal Renal Function
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1915 Association of Cholesterol Levels with Mortality and Cardiovascular Events among Patients with CKD and Different Amounts of Proteinuria
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1927 Carbamylation of Serum Albumin and Erythropoietin Resistance in End Stage Kidney Disease
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1935 Bacteria-Derived DNA Fragment in Peritoneal Dialysis Effluent as a Predictor of Relapsing Peritonitis
Cheuk-Chun Szeto, Ka-Bik Lai, Bonnie Ching-Ha Kwan, Kai-Ming Chow, Chi-Bon Leung, Man-Ching Law, Vincent Yu, and Philip Kam-Tao Li

1942 Clinical Factors and the Decision to Transfuse Chronic Dialysis Patients
Cynthia B. Whitman, Sanatan Shreay, Matthew Gitlin, Martijn G. H. van Oijen, and Brennan M. R. Spiegel

1952 Dietary Sodium Restriction and Association with Urinary Marinobufagenin, Blood Pressure, and Aortic Stiffness
Kristen L. Jablonski, Olga V. Fedorova, Matthew L. Racine, Candace J. Geolfos, Phillip E. Gates, Michel Chonchol, Bradley S. Fleenor, Edward G. Lakatta, Alexei Y. Bagrov, and Douglas R. Seals

1960 Randomized Controlled Trial of Febuxostat Versus Allopurinol or Placebo in Individuals with Higher Urinary Uric Acid Excretion and Calcium Stones
David S. Goldfarb, Patricia A. MacDonald, Lhanoo Gunawardhana, Solomon Chefo, and Lachy McLean

1968 Fibroblast Growth Factor 23 and Cardiovascular Mortality after Kidney Transplantation
Leandro C. Baia, Jelmer K. Humalda, Marc G. Vervloet, Gerjan Navis, Stephan J.L. Bakker, and Martin H. de Borst, on behalf of the NIGRAM Consortium

1979 A Patient with Nephrotic-Range Proteinuria and Focal Global Glomerulosclerosis
Fernando C. Fervenza

1988 Medication Reconciliation and Therapy Management in Dialysis-Dependent Patients: Need for a Systematic Approach
Amy Barton Pai, Katie E. Cardone, Harold J. Manley, Wendy L. St. Peter, Rachel Shaffer, Michael Somers, and Rajnish Mehrotra, on behalf of the Dialysis Advisory Group of the American Society of Nephrology

Ann Rinehart
On the Cover

What’s the diagnosis? A young man with a renal carcinoma but no history of kidney stones or malabsorption underwent a nephrectomy and the non-neoplastic kidney parenchyma of the kidney was examined. The cortex was completely unremarkable. The image shows several large calcium phosphate deposits at the tip of papilla with associated medullary interstitial fibrosis. The calcium phosphate deposits are also present in the lumens of several tubules, suggesting a dystrophic type of calcifications that occur in the setting of cellular injury, as opposed to metastatic calcifications that are seen in the setting of hypercalcemic states and characterized by deposition along the tubular basement membranes. Interstitial calcium phosphate deposits, sometimes with associated interstitial fibrosis, have been identified in patients with kidney stones who have undergone biopsy during percutaneous stone removal. Some patients, typically those with an ileostomy or who have had a small bowel resection or gastric bypass surgery, may also have crystal deposits that plug the ducts of Bellini and inner medullary collecting ducts. The current hypothesis is that this interstitial calcium phosphate eventually erodes through the papillary epithelium to form a Randall’s plaque. Calcium oxalate and calcium phosphate then deposit on the plaque and grow into what is clinically recognized as a kidney stone. Thus, this interstitial process (which may also seen in individuals who have not formed a kidney stone), related to low urine volume and high urine calcium but for which the exact mechanism remains unknown, may be the initiating event for the common forms of calcium nephrolithiasis. (Images and text provided by Vanesa Bijol, MD, Brigham and Women’s Hospital)