Editorials

1515 Urinary Angiotensinogen: A Promising Biomarker of AKI Progression in Acute Decompensated Heart Failure: What Does It Mean?
Jan Wysocki and Daniel Batlle
See related article on page 1536.

1518 What Is the Correct Approach for Comparing GFR by Different Methods across Levels of GFR?
Andrew D. Rule and Walter K. Kremers
See related article on page 1574.

1522 Assisted Peritoneal Dialysis as an Alternative to In-Center Hemodialysis
Edwina A. Brown and Martin Wilkie
See related article on page 1606.

1525 Pragmatic, Precision Medicine Approaches for Dialysis Vascular Access Dysfunction: Challenges and Opportunities
Prabir Roy-Chaudhury
See related article on page 1615.

Original Articles

Acute Kidney Injury

1527 Acute Kidney Injury Urine Biomarkers in Very Low-Birth-Weight Infants
David J. Askenazi, Rajesh Koralkar, Neha Patil, Brian Halloran, Namasivayam Ambalavanan, and Russell Griffin

1536 Urinary Biomarkers at the Time of AKI Diagnosis as Predictors of Progression of AKI among Patients with Acute Cardiorenal Syndrome
Chunbo Chen, Xiaobing Yang, Ying Lei, Yan Zha, Huafeng Liu, Changsheng Ma, Jianwei Tian, Pingyan Chen, Tiecheng Yang, and Fan Fan Hou
See related editorial on page 1515.

Chronic Kidney Disease

1546 Inflammation and Progression of CKD: The CRIC Study

1557 Silent Cerebral Microbleeds and Longitudinal Risk of Renal and Cardiovascular Events in Patients with CKD
Hideaki Shima, Tatsuhiko Mori, Masayuki Ooi, Mika Sonoda, Tetsuo Shoji, Eiji Ishimura, Mikio Okamura, Nobukazu Ishizaka, and Masaaki Inaba

1566 Variation in Patients’ Awareness of CKD according to How They Are Asked
Delphine S. Tuot, Yunnuo Zhu, Alexandra Velasquez, Juan Espinoza, C. Damaris Mendez, Tanushree Banerjee, Chi-yuan Hsu, and Neil R. Powe

1574 Measurement Error as Alternative Explanation for the Observation that CrCl/GFR Ratio is Higher at Lower GFR
Xuehan Zhang, Charles E. McCulloch, Feng Lin, Yen-chung Lin, Isabel Elaine Allen, Nisha Bansal, Alan S. Go, and Chi-yuan Hsu
See related editorial on page 1518.
Clinical Immunology and Pathology

1582 Clinical Significance of IgM and C3 Glomerular Deposition in Primary Focal Segmental Glomerulosclerosis
Yi-miao Zhang, Qiu-hua Gu, Jing Huang, Zhen Qu, Xin Wang, Li-qiang Meng, Fang Wang, Gang Liu, Zhao Cui, and Ming-hui Zhao

Epidemiology and Outcomes

1590 Risk Factors for and Outcomes of Catheter-Associated Peritonitis in Children: The SCOPE Collaborative
Christine B. Sethna, Kristina Bryant, Raj Munshi, Bradley A. Warady, Troy Richardson, John Lawlor, Jason G. Newland, and Alicia Neu on behalf of the SCOPE Investigators

ESRD and Chronic Dialysis

1597 Effect of Tenapanor on Interdialytic Weight Gain in Patients on Hemodialysis
Geoffrey A. Block, David P. Rosenbaum, Maria Leonsson-Zachrisson, Bergur V. Stefansson, Tina Rydén-Bergsten, Peter J. Greasley, Susanne A. Johansson, Mikael Knutsson, and Björn C. Carlsson

1606 Hospitalization Rates for Patients on Assisted Peritoneal Dialysis Compared with In-Center Hemodialysis
See related editorial on page 1522.

1615 Preoperative Vascular Medial Fibrosis and Arteriovenous Fistula Development
Yan-Ting Shiu, Silvio H. Litovsky, Alfred K. Cheung, Daniel B. Pike, Jason Chieh Sheng Tey, Yingying Zhang, Carlton J. Young, Michelle Robbin, Kenneth Hoyt, and Michael Allon
See related editorial on page 1525.

Geriatric Nephrology

1624 Functional and Cognitive Impairment, Frailty, and Adverse Health Outcomes in Older Patients Reaching ESRD—A Systematic Review
Marije H. Kallenberg, Hilda A. Kleinveld, Friedo W. Dekker, Barbara C. van Munster, Ton J. Rabelink, Marjolijn van Buren, and Simon P. Mooijaart

Renal Transplantation

1640 Creatinine–Based and Cystatin C–Based GFR Estimating Equations and Their Non-GFR Determinants in Kidney Transplant Recipients
Mira T. Keddis, Hatem Amer, Nikolay Voskoboev, Walter K. Kremers, Andrew D. Rule, and John C. Lieske

1650 Acute Rejection Rates and Graft Outcomes According to Induction Regimen among Recipients of Kidneys from Deceased Donors Treated with Tacrolimus and Mycophenolate

Glomerular Disease Update for the Clinician

1662 Introduction: Glomerular Disease Update for the Clinician
Carla M. Nester and Ronald J. Falk

1664 The Players: Cells Involved in Glomerular Disease
A. Richard Kitching and Holly L. Hutton

Commentary

1675 Chemistry Testing on Plasma Versus Serum Samples in Dialysis Patients: Clinical and Quality Improvement Implications
Roger Neill Carey, Chinu Jani, Curtis Johnson, Jim Pearce, Patricia Hui-Ng, and Eduardo Lacson
On the Cover

What is the diagnosis? A 15-year-old with underlying Turner syndrome, hypothyroidism, and Guillain-Barre syndrome presented with symptoms concerning for meningitis and was treated with empiric antibiotics and intravenous acyclovir. Other medications included levothyroxine, ranitidine, furosemide and vitamin D. Kidney function remained stable; however, urinalysis revealed SG 1.020, pH 5.0, 1+ protein, 1+ glucose, and 3+ blood following acyclovir exposure. Urine sediment analysis demonstrated white blood cells, red blood cells, rare granular casts, and numerous needle shaped crystals (left panel), which were birefringent with polarization (right panel). These finding were consistent acyclovir-induced crystalluria. Acyclovir is one of a number of medications that is associated with crystal formation within the urine. Others drugs included on this list are sulfadiazine/sulfamethoxazole, indinavir, atazanavir, ciprofloxacin, methotrexate, ascorbic acid, ethylene glycol, triamterene, and amoxicillin. With these agents, either the drug or one of its metabolites may crystallize within the renal tubules. Crystal precipitation typically occurs due to one or more of the following factors: drug insolubility, high or low urine pH, sluggish urine flow rates, and underlying kidney disease. Acyclovir is unaffected by urine pH and crystal precipitation is generally driven by drug insolubility in urine when given in high, intravenous bolus doses to patients with underlying kidney disease or in the setting of volume depletion. Urinary pH plays a role for drugs such as ciprofloxacin, indinavir and atazanavir, which can precipitate in alkaline urine whereas sulfadiazine and methotrexate are insoluble in acid urine and more likely to precipitate at pH < 6.0. Clinically, patients may present clinically with asymptomatic crystalluria, crystalline-induced AKI, or sometimes nephrolithiasis. Prevention and therapy are directed at improving urinary flow rates, stopping or dose reducing the culprit drug, and when feasible, altering urine pH to enhance drug/metabolite solubility. Recovery of kidney function with drug removal is common; however, some patients develop CKD. (Images and text provided by José Antonio Tesser Poloni and Maria Solange Bressan Giordani, Irmandade da Santa Casa de Misericórdia de Porto Alegre, Porto Alegre, Brazil and Mark A. Perazella, Yale University, New Haven, Connecticut)