

# CJASN

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## Original Articles

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*Kenji Ishikura, Norishige Yoshikawa, Hitoshi Nakazato, Satoshi Sasaki, Kazumoto Iijima, Koichi Nakanishi, Takeshi Matsuyama, Shuichi Ito, Nahoko Yata, Takashi Ando, and Masataka Honda, for the Japanese Study Group of Renal Disease in Children*

**1584 Pulmonary Embolism in Patients with CKD and ESRD**

*Gagan Kumar, Ankit Sakhuja, Amit Taneja, Tilottama Majumdar, Jayshil Patel, Jeff Whittle, and Rahul Nanchal, for the Milwaukee Initiative in Critical Care Outcomes Research (MICCOR) Group of Investigators*

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*Catherine Sullivan, Janeen B. Leon, Srilekha S. Sayre, Marquisha Marbury, Michael Ivers, Julie A. Pencak, Kenneth A. Bodziak, Donald E. Hricik, E. Janie Morrison, Jeffrey M. Albert, Sankar D. Navaneethan, Christina M. Delos Reyes, and Ashwini R. Sehgal*  
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### **1646 Association of Interleg BP Difference with Overall and Cardiovascular Mortality in Hemodialysis**

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#### Health Services Research

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## Special Features

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### **1664 Critical and Honest Conversations: The Evidence Behind the “Choosing Wisely” Campaign Recommendations by the American Society of Nephrology**

*Amy W. Williams, Amy C. Dwyer, Allison A. Eddy, Jeffrey C. Fink, Bertrand L. Jaber, Stuart L. Linas, Beckie Michael, Ann M. O’Hare, Heidi M. Schaefer, Rachel N. Shaffer, Howard Trachtman, Daniel E. Weiner, and Ronald J. Falk, on behalf of the American Society of Nephrology Quality, and Patient Safety Task Force*

### **1673 Monitoring Quality of Care at Dialysis Facilities: A Case for Regulatory Parsimony—and Beyond**

*John C. Stivelman*

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*Marc Ghannoum, Thomas D. Nolin, David S. Goldfarb, Darren M. Roberts, Robert Mactier, James B. Mowry, Paul I. Dargan, Robert MacLaren, Lotte C. Hoegberg, Martin Laliberté, Diane Calello, Jan T. Kielstein, Kurt Anseeuw, James F. Winchester, Emmanuel A. Burdmann, Timothy E. Bunchman, Yi Li, David N. Juurlink, Valery Lavergne, Bruno Megarbane, Sophie Gosselin, Kathleen D. Liu, and Robert S. Hoffman*

## Moving Points in Nephrology

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### **1691 Onco-Nephrology: What the Nephrologist Needs to Know about Cancer and the Kidney**

*Jeffrey S. Berns and Mitchell H. Rosner*

### **1692 Onco-Nephrology: AKI in the Cancer Patient**

*Albert Q. Lam and Benjamin D. Humphreys*

**1701 Onco-Nephrology: Glomerular Diseases with Cancer**

*Jean-François Cambier and Pierre Ronco*

**1713 Onco-Nephrology: Renal Toxicities of Chemotherapeutic Agents**

*Mark A. Perazella*

**1722 Onco-Nephrology: The Pathophysiology and Treatment of Malignancy-Associated Hypercalcemia**

*Mitchell H. Rosner and Alan C. Dalkin*

**1730 Onco-Nephrology: Tumor Lysis Syndrome**

*F. Perry Wilson and Jeffrey S. Berns*

## Erratum

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**1740 Correction**

### On the Cover

*What's the diagnosis?* Renal biopsy reveals many tubules which appear to be obstructed by abundant clear needle-shaped crystals, some of which have dissolved with tissue processing. The crystals are surrounded by intra-tubular macrophages and multinucleated giant cells. There is evidence of acute tubular injury, interstitial chronic inflammation, and tubulointerstitial scarring. The patient presented with sterile pyuria and acute kidney injury; the biopsy is diagnostic of a "crystal nephropathy". In this case, the etiology of crystal nephropathy is the protease inhibitor, indinavir. Crystal deposition within tubular lumina of the kidney can cause both acute and chronic kidney injury. While a number of endogenous processes are associated with crystal-induced kidney injury (tumor lysis syndrome with acute uric acid nephropathy, gastric bypass with acute oxalate nephropathy), several medications can cause this clinical renal syndrome. Common examples include agents such as acyclovir, sulfadiazine, ciprofloxacin, methotrexate, indinavir, atazanavir, sodium phosphate bowel purgatives, ascorbic acid, ethylene glycol, and triamterene. With these agents, either the drug or its metabolites crystallize within the renal tubules, often due to one or more factors such as inherent drug insolubility, urine pH (high or low), sluggish urine flow rates (increased urinary drug concentration), and underlying kidney disease. For example, ciprofloxacin, indinavir and atazanavir can precipitate in tubular lumens in alkaline urine. In contrast, drugs such as sulfadiazine and methotrexate (and their metabolites) are less soluble in acid urine and more likely to precipitate in the kidney at pH < 6.0. Drugs such as orlistat can induce malabsorption with enteric hyperoxaluria and acute oxalate nephropathy. Clinically, patients may develop asymptomatic crystalluria, crystal-induced AKI, or nephrolithiasis. Prevention and therapy are directed at improving urinary flow rates, stopping or dose reducing the culprit drug, and altering urine pH (when feasible) to enhance drug/metabolite solubility. Most patients recover kidney function with drug discontinuation; however, some patients develop chronic kidney disease. (Image and text provided by Mark A. Perazella, Yale University School of Medicine; and Glen S. Markowitz, Columbia University College of Physicians and Surgeons)