

# CJASN

Clinical Journal of the American Society of Nephrology

February 2015 • Vol. 10 • No. 2

## Editorials

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- 165 Can We Save the Kidneys by Protecting the Heart?**  
*Srisakul Chirakarnjanakorn and W.H. Wilson Tang*  
See related article on page 205.
- 167 Preventing Aristolochic Acid Nephropathy**  
*Nelomi Anandagoda and Graham M. Lord*  
See related article on page 215.
- 169 Four Plus Forty-Four: Hours to Modify, Theirs to Enjoy**  
*Sarbjit Vanita Jassal*  
See related article on page 260.
- 172 Alternative Strategies Needed To Improve Vascular Access Outcomes**  
*Alexander S. Yevzlin and Brad C. Astor*  
See related article on page 269.
- 174 Stones, Bones, and Cardiovascular Groans**  
*Eric N. Taylor*  
See related article on page 278.
- 177 Strategies To Facilitate Organ Donation among African Americans**  
*Dana H.Z. Robinson and Kimberly R. Jacob Arriola*  
See related article on page 286.

## Original Articles

---

- Acute Kidney Injury**
- 180 Renal Arcuate Vein Microthrombi-Associated AKI**  
*Andrew Redfern, Huda Mahmoud, Tom McCulloch, Adam Shardlow, Matthew Hall, Catherine Byrne, and Nicholas M. Selby*
- 187 AKI in Low-Risk versus High-Risk Patients in Intensive Care**  
*Florentina E. Sileanu, Raghavan Murugan, Nicole Lucko, Gilles Clermont, Sandra L. Kane-Gill, Steven M. Handler, and John A. Kellum*
- Chronic Kidney Disease**
- 197 Behavioral Stage of Change and Dialysis Decision-Making**  
*Suma Prakash, Anna McGrail, Steven A. Lewis, Jesse Schold, Mary Ellen Lawless, Ashwini R. Sehgal, and Adam T. Perzynski*
- Epidemiology and Outcomes**
- 205 NT-ProBNP and Troponin T and Risk of Rapid Kidney Function Decline and Incident CKD in Elderly Adults**  
*Nisha Bansal, Ronit Katz, Lorien Dalrymple, Ian de Boer, Christopher DeFilippi, Bryan Kestenbaum, Meyeon Park, Mark Sarnak, Stephen Seliger, and Michael Shlipak*  
See related editorial on page 165.

- 215 Chronic Dietary Exposure to Aristolochic Acid and Kidney Function in Native Farmers from a Croatian Endemic Area and Bosnian Immigrants**  
*Bojan Jelaković, Ivana Vuković Lela, Sandra Karanović, Živka Dika, Jelena Kos, Kathleen Dickman, Maja Šekoranja, Tamara Poljičanin, Maja Mišić, Vedran Premužić, Mirta Abramović, Vesna Matijević, Marica Miletić Medved, Ante Cvitković, Karen Edwards, Mirjana Fuček, Ninoslav Leko, Tomislav Teskera, Mario Laganović, Dubravka Čvorišćec, and Arthur P. Grollman*  
See related editorial on page 167.
- 224 Quantification of HDL Proteins, Cardiac Events, and Mortality in Patients with Type 2 Diabetes on Hemodialysis**  
*Chantal Kopecky, Bernd Genser, Christiane Drechsler, Vera Krane, Christopher C. Kaltenecker, Markus Hengstschläger, Winfried März, Christoph Wanner, Marcus D. Säemann, and Thomas Weichhart*
- 232  Association of IL-6 and a Functional Polymorphism in the IL-6 Gene with Cardiovascular Events in Patients with CKD**  
*Belinda Spoto, Francesco Mattace-Raso, Eric Sijbrands, Daniela Leonardis, Alessandra Testa, Anna Pisano, Patrizia Pizzini, Sebastiano Cutrupi, Rosa M. Parlongo, Graziella D'Arrigo, Giovanni Tripepi, Francesca Mallamaci, and Carmine Zoccali*
- 241 Adiposity Patterns and the Risk for ESRD in Postmenopausal Women**  
*Nora Franceschini, Natalia A. Gouskova, Alex P. Reiner, Andrew Bostom, Barbara V. Howard, Mary Pettinger, Jason G. Umans, M. Alan Brookhart, Wolfgang C. Winkelmayer, Charles B. Eaton, Gerardo Heiss, and Jason P. Fine*
- ESRD and Chronic Dialysis**
- 251 ESRD From Lupus Nephritis in the United States, 1995–2010**  
*Donal J. Sexton, Scott Reule, Craig Solid, Shu-Cheng Chen, Allan J. Collins, and Robert N. Foley*
- Geriatric Nephrology**
- 260 CKD in Elderly Patients Managed without Dialysis: Survival, Symptoms, and Quality of Life**  
*Mark A. Brown, Gemma K. Collett, Elizabeth A. Josland, Celine Foote, Qiang Li, and Frank P. Brennan*  
See related editorial on page 169.
- Health Services Research**
- 269 Provider Visit Frequency and Vascular Access Interventions in Hemodialysis**  
*Kevin F. Erickson, Matthew W. Mell, Wolfgang C. Winkelmayer, Glenn M. Chertow, and Jay Bhattacharya*  
See related editorial on page 172.
- Nephrolithiasis**
- 278 Vascular Calcification and Bone Mineral Density in Recurrent Kidney Stone Formers**  
*Linda Shavit, Daniela Girfoglio, Vivek Vijay, David Goldsmith, Pietro Manuel Ferraro, Shabbir H. Moochhala, and Robert Unwin*  
See related editorial on page 174.
- Renal Transplantation**
- 286 Identification of Strategies to Facilitate Organ Donation among African Americans using the Nominal Group Technique**  
*Jayne E. Locke, Haiyan Qu, Richard Shewchuk, Roslyn B. Mannon, Robert Gaston, Dorry L. Segev, Elinor C. Mannon, and Michelle Y. Martin*  
See related editorial on page 177.
- 294 Randomized Trial of Valganciclovir Versus Valacyclovir Prophylaxis for Prevention of Cytomegalovirus in Renal Transplantation**  
*Tomas Reischig, Martin Kacer, Pavel Jindra, Ondrej Hes, Daniel Lysak, and Mirko Bouda*

## Renal Physiology

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### 305 Collecting Duct Intercalated Cell Function and Regulation

Ankita Roy, Mohammad M. Al-bataineh, and Núria M. Pastor-Soler

## Role of the Medical Director

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### 325 Introduction: Role of the Medical Director Series

Robert Provenzano and Jeffrey L. Hymes

### 326 The Evolving Role of the Medical Director of a Dialysis Facility

Franklin W. Maddux and Allen R. Nissenson

## Commentary

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### 331 Interpreting Vitamin D Assay Results: Proceed with Caution


Glenville Jones

## Public Policy Series

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### 335 Integrated Renal Care: Are Nephrologists Ready for Change in Renal Care Delivery Models?

Edward R. Jones and Thomas H. Hostetter

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### On the Cover

*What's the diagnosis?* A 29-year-old man with ESRD underwent a deceased donor kidney transplant, which was complicated by delayed graft function. Despite excellent urine output, serum creatinine remained elevated and hemodialysis was initiated. Kidney biopsy was obtained on day 5 to assess the cause of delayed graft function. The glomeruli showed multiple intracapillary fibrin thrombi, mesangiolysis and numerous red blood cell fragments within the injured subendothelial and mesangial areas of the glomerular tuft. Fibrin stain on IF was strongly positive and electronmicroscopy studies showed intracapillary fibrin tactoids. The biopsy diagnosis was thrombotic microangiopathy (TMA). Thrombotic microangiopathy developing following kidney transplantation can be classified as either recurrent or de novo. Recurrent TMA is more common in adults than children following kidney transplantation. Recurrence is more likely to develop when the original cause was complement-associated atypical hemolytic uremic syndrome (HUS) as compared with infection-related TMA. In general risk of recurrent TMA depends on the mutation—more common with mutations in circulating factors such as complement factors I or H. Other factors associated with increased risk for recurrence in the allograft include older age, living related donor kidneys, calcineurin inhibitors, and short duration between disease onset and ESRD or transplantation. De novo TMA among kidney transplant recipients may be due to any of the causes that occur in the general population. However, immunosuppressive medications (calcineurin inhibitors, mTOR inhibitors), anti-platelet agents (ticlopidine, clopidogrel) and valacyclovir are notable causes in this group. Antibody-mediated rejection, which was thought to be the cause in the patient presented, is another etiology of TMA. Finally, infection with HIV, parvovirus B19, and cytomegalovirus have also been associated with TMA following kidney transplantation. Many but not all patients present with microangiopathic hemolytic anemia, thrombocytopenia, and schistocytes on peripheral smear. Urine studies reveal hematuria and low-grade proteinuria. Some may have TMA limited to the allograft without systemic abnormalities. Treatment is directed at the cause. Recurrent TMA from mutation in one the complement factors may benefit from eculizumab. Patients with de novo disease from either calcineurin or mTOR inhibitors should have the drug withdrawn and undergo plasmapheresis. Intravenous immunoglobulin and rituximab have also been utilized. Prognosis is better for de novo as compared with recurrent TMA; however, both suffer from allograft loss. (*Images and text provided by Mark A. Perazella, MD, and Gilbert Moeckel, MD, Yale University School of Medicine, New Haven, Connecticut*)