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Editorials

1107 Shades of Grey: The Conundrum of Implantable Defibrillators in Individuals with Advanced CKD

Aisha Khattak and David M. Charytan
See related article on page 1119.

1110 Treating Elderly Patients with ANCA-Associated Vasculitis

J. Ashley Jefferson
See related article on page 1128.

1114 Nonblood/Urine Biomarker of Progression of Kidney Disease in Diabetes Mellitus

Ramin Tolouian and Aaron I. Vinik
See related article on page 1136.

1117 ESRD Outcomes and GN Subtypes

Eric W. Young
See related article on page 1170.

Original Articles

Chronic Kidney Disease

1119 Implantable Cardioverter-Defibrillators in Patients with CKD: A Propensity-Matched Mortality Analysis

Georges N. Nakhoul, Jesse D. Schold, Susana Arrigain, Serge C. Harb, Stacey Jolly, Bruce L. Wilkoff, Joseph V. Nally Jr, and Sankar D. Navaneethan
See related editorial on page 1107.

Clinical Nephrology

1128 Outcome and Treatment of Elderly Patients with ANCA-Associated Vasculitis

Maria Weiner, Su Mein Goh, Aladdin J. Mohammad, Zdenka Hruskova, Anisha Tanna, Annette Bruchfeld, Daina Selga, Zdenka Chocova, Kerstin Westman, Per Eriksson, Charles D. Pusey, Vladimir Tesar, Alan D. Salama, and Mårten Segelmark
See related editorial on page 1110.

Diabetes and the Kidney

1136 Cardiac Autonomic Neuropathy and Early Progressive Renal Decline in Patients with Nonmacroalbuminuric Type 1 Diabetes

Steven Orlov, David Z.I. Cherney, Rodica Pop-Busui, Leif E. Lovblom, Linda H. Ficociello, Adam M. Smiles, James H. Warram, Andrzej S. Krolewski, and Bruce A. Perkins
See related editorial on page 1114.

Epidemiology and Outcomes

1145 Light-Intensity Physical Activities and Mortality in the United States General Population and CKD Subpopulation

Srinivasan Beddhu, Guo Wei, Robin L. Marcus, Michel Chonchol, and Tom Greene

1154 Rate of Change in Renal Function and Mortality in Elderly Treated Hypertensive Patients

Enayet K. Chowdhury, Robyn G. Langham, Zanfina Ademi, Alice Owen, Henry Krum, Lindon M.H. Wing, Mark R. Nelson, and Christopher M. Reid, on behalf of the Second Australian National Blood Pressure Study Management Committee

1162 Serum 25-Hydroxyvitamin D Level and Kidney Function Decline in a Swiss General Adult Population

Idris Guessous, William McClellan, David Kleinbaum, Viola Vaccarino, Henry Hugues, Olivier Boulat, Pedro Marques-Vidal, Fred Paccaud, Jean-Marc Theler, Jean-Michel Gaspoz, Michel Burnier, Gérard Waeber, Peter Vollenweider, and Murielle Bochud

1170 Patient Characteristics and Outcomes by GN Subtype in ESRD

Michelle M. O'Shaughnessy, Maria E. Montez-Rath, Richard A. Lafayette, and Wolfgang C. Winkelmayer
See related editorial on page 1117.

ESRD and Chronic Dialysis

1179 Changes in Pulse Pressure during Hemodialysis Treatment and Survival in Maintenance Dialysis Patients

Paungpaga Lertdumrongluk, Elani Streja, Connie M. Rhee, John J. Sim, Daniel Gillen, Csaba P. Kovesdy, and Kamyar Kalantar-Zadeh

1192 Body Composition and Survival in Dialysis Patients: Results from an International Cohort Study

Daniele Marcelli, Len A. Usvyat, Peter Kotanko, Inga Bayh, Bernard Canaud, Michael Etter, Emanuele Gatti, Aileen Grassmann, Yuedong Wang, Cristina Marelli, Laura Scatizzi, Andrea Stopper, Frank M. van der Sande, and Jeroen Kooman, on behalf of the MONitoring Dialysis Outcomes (MONDO) Consortium

1201 Patient and Health Care Professional Decision-Making to Commence and Withdraw from Renal Dialysis: A Systematic Review of Qualitative Research

Jamilla A. Hussain, Kate Flemming, Fliss E.M. Murtagh, and Miriam J. Johnson

Ethics

1217 Focus Group Study of Public Opinion About Paying Living Kidney Donors in Australia

Allison Tong, Angelique F. Ralph, Jeremy R. Chapman, Germaine Wong, John S. Gill, Michelle A. Josephson, and Jonathan C. Craig

Hypertension

1227 Effect of Dietary Sodium Restriction on Human Urinary Metabolomic Profiles

Kristen L. Jablonski, Jelena Klawitter, Michel Chonchol, Candace J. Bassett, Matthew L. Racine, and Douglas R. Seals

Nephrolithiasis

1235  Clinical and Genetic Analysis of Patients with Cystinuria in the United Kingdom

Hannah L. Rhodes, Laura Yarram-Smith, Sarah J. Rice, Ayla Tabakert, Noel Edwards, Alice Hartley, Mark N. Woodward, Sarah L. Smithson, Charles Tomson, Gavin I. Welsh, Margaret Williams, David T. Thwaites, John A. Sayer, and Richard J.M. Coward

Renal Transplantation

1246 The Association between Marine n-3 Polyunsaturated Fatty Acid Levels and Survival after Renal Transplantation

Ivar A. Eide, Trond Jenssen, Anders Hartmann, Lien M. Diep, Dag O. Dahle, Anna V. Reisæter, Kristian S. Bjerve, Jeppe H. Christensen, Erik B. Schmidt, and My Svensson

Renal Physiology

1257 Renal Control of Calcium, Phosphate, and Magnesium Homeostasis

Judith Blaine, Michel Chonchol, and Moshe Levi

Renal Immunology

1273 A New CJASN Series: Renal Immunology for the Clinician

Fadi G. Lakkis and Paul M. Palevsky

1274 A Brief Journey through the Immune System

Karim M. Yatim and Fadi G. Lakkis

Role of the Medical Director

1282 The Medical Director in Integrated Clinical Care Models

Thomas F. Parker III and George R. Aronoff

Moving Points in Nephrology

1287 Drug-Induced Glomerular Disease: Attention Required!


Jai Radhakrishnan and Mark A. Perazella

1291 Drug-Induced Glomerular Disease: Direct Cellular Injury

Glen S. Markowitz, Andrew S. Bomback, and Mark A. Perazella

1300 Drug-Induced Glomerular Disease: Immune-Mediated Injury

Jonathan J. Hogan, Glen S. Markowitz, and Jai Radhakrishnan

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On the Cover

What's the diagnosis? A 28-year-old man presented with normocytic normochromic anemia, thrombocytopenia, and acute kidney injury with a serum creatinine of 6.7mg/dl. A kidney biopsy was performed. Light microscopy of the biopsy revealed unremarkable glomeruli. The proximal tubular epithelial cells showed coarse golden-brown pigment (top image), which gave a positive reaction on Perls Prussian blue stain for iron (bottom image), consistent with deposition of hemosiderin. There were changes of acute tubular injury, including dilatation of tubules, cytoplasmic vacuoles, and cellular debris in the lumen. There was no significant interstitial inflammation or fibrosis or vascular abnormalities.

Renal hemosiderosis occurs as a complication of intravascular hemolysis. Hemolytic anemias, mismatched blood transfusions, paroxysmal nocturnal hemoglobinuria and mechanical hemolysis from prosthetic cardiac valves are the common causes. Intravascular haemolysis releases hemoglobin (Hb) into plasma, where it binds to plasma haptoglobin (Hp), forming Hb-Hp complex. This complex is not filtered by the glomerulus and is degraded by the liver. In chronic hemolytic states, plasma haptoglobin is consumed. Unbound (free) Hb accumulates in plasma and is filtered by the glomerulus. It is reabsorbed by the proximal tubule cells, resulting in the accumulation of ferric ions (hemosiderin) and subsequent cell injury.

Renal hemosiderosis seldom causes renal dysfunction and is usually an incidental radiologic or autopsy finding. In cases with clinical involvement, it causes reversible acute dysfunction to chronic irreversible damage. Treatment is usually directed to the underlying disease. Iron chelation therapy may be helpful.

(Image and text provided by Anila Kurien, Center for Renal and Urological Pathology Pvt Ltd Chennai, Tamil Nadu, India, and Edwin Fernando, Government Stanley Medical College, Nephrology Chennai, Tamil Nadu, India)