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Patient Voices

1613 Treatment Adherence in Young Adults Receiving Kidney Replacement Therapy: A Patient Perspective

Amanda Grandinetti

See related Patient Voice editorial and article on pages 1615 and 1669, respectively.

1615 Treatment Adherence in Young Adults Receiving Kidney Replacement Therapy: A Caregiver Perspective

Pam Duquette and Kelly Helm

See related Patient Voice editorial and article on pages 1613 and 1669, respectively.

Editorials

1617 Kidney Biopsy in Hospitalized Patients with Acute Kidney Disease: Is There an Increased Risk?

Steven Darrow Weisbord

See related article on page 1633.

1619 Better Patient Ambulatory Care Experience: Does It Translate into Improved Outcomes among Patients with CKD?

Delphine S. Tuot

See related article on page 1659.

1621 ACCORDION: Ensuring That We Hear the Music Clearly

Muh Geot Wong, Hiddo J.L. Heerspink, and Vlado Perkovic

See related article on page 1693.

1624 Increasing Protection of Dialysis Patients against Influenza

Megan C. Lindley and David K. Kim

See related article on page 1703.

Original Articles

Acid/Base and Electrolyte Disorders

1627 Urea for the Treatment of Hyponatremia

Helbert Rondon-Berrios, Srijan Tandukar, Maria K. Mor, Evan C. Ray, Filitsa H. Bender, Thomas R. Kleyman, and Steven D. Weisbord

Acute Kidney Injury and ICU Nephrology

1633 Kidney Biopsy–Related Complications in Hospitalized Patients with Acute Kidney Disease

Dennis G. Moledina, Randy L. Luciano, Lidiya Kukova, Lili Chan, Aparna Saha, Girish Nadkarni, Sandra Alfano, F. Perry Wilson, Mark A. Perazella, and Chirag R. Parikh

See related editorial on page 1617.

Chronic Kidney Disease

1641 End-Stage Kidney Disease following Surgical Management of Kidney Cancer

Robert J. Ellis, Daniel P. Edey, Sharon J. Del Vecchio, Megan McStea, Scott B. Campbell, Carmel M. Hawley, David W. Johnson, Christudas Morais, Susan J. Jordan, Ross S. Francis, Simon T. Wood, Glenda C. Gobe, and Cancer Alliance Queensland

Chronic Kidney Disease (Continued)

1649 CKD in Patients with Bilateral Oophorectomy

Andrea G. Kattah, Carin Y. Smith, Liliana Gazzuola Rocca, Brandon R. Grossardt, Vesna D. Garovic, and Walter A. Rocca

1659 Patient Experience with Primary Care Physician and Risk for Hospitalization in Hispanics with CKD

Esteban A. Cedillo-Couvert, Jesse Y. Hsu, Ana C. Ricardo, Michael J. Fischer, Ben S. Gerber, Edward J. Horwitz, John W. Kusek, Eva Lustigova, Amada Renteria, Sylvia E. Rosas, Milda Saunders, Daohang Sha, Anne Slaven, and James P. Lash, on behalf of the CRIC Study Investigators
See related editorial on page 1619.

Clinical Nephrology

1669 Associations with Wellbeing and Medication Adherence in Young Adults Receiving Kidney Replacement Therapy

Alexander James Hamilton, Fergus J. Caskey, Anna Casula, Carol D. Inward, and Yoav Ben-Shlomo
See related Patient Voice editorials on pages 1613 and 1615.

Cystic Kidney Disease

1680 Kidney Function Reserve Capacity in Early and Later Stage Autosomal Dominant Polycystic Kidney Disease

A. Lianne Messchendorp, Marco van Londen, Jacob M. Taylor, Martin H. de Borst, Gerjan Navis, Niek F. Casteleijn, Carlo A.J.M. Gaillard, Stephan J.L. Bakker, and Ron T. Gansevoort, on behalf of the DIPAK Consortium

Diabetes and the Kidney

1693 Long-Term Effects of Intensive Glycemic and Blood Pressure Control and Fenofibrate Use on Kidney Outcomes

Amy K. Mottl, John B. Buse, Faramarz Ismail-Beigi, Ronald J. Sigal, Carolyn F. Pedley, Vasilios Papademetriou, Debra L. Simmons, Lois Katz, Josyf C. Mychaleckyj, and Timothy E. Craven
See related editorial on page 1621.

Maintenance Dialysis

1703 High-Dose Seasonal Influenza Vaccine in Patients Undergoing Dialysis

Dana C. Miskulin, Daniel E. Weiner, Hocine Tighiouart, Eduardo K. Lacson Jr., Klemens B. Meyer, Taimur Dad, and Harold J. Manley
See related editorial on page 1624.

1712 MicroRNA-21 and Venous Neointimal Hyperplasia of Dialysis Vascular Access

Chih-Cheng Wu, Li-Jing Chen, Mu-Yang Hsieh, Chien-Ming Lo, Ming-Hsien Lin, Hsiao-En Tsai, Hsiang-Lin Song, and Jeng-Jiann Chiu

Transplantation

1721 Acute Kidney Injury in Children with Kidney Transplantation

Omar Alkandari, Lieuko Nguyen, Diane Hebert, Valerie Langlois, Natasha A. Jawa, Rulan S. Parekh, and Lisa A. Robinson

Research Letter

1730 Changes in Transfusion Coding Among Hospitalized Medicare Beneficiaries after Implementation of ICD-10

Eric D. Weinhandl and Kristine M. Kubisiak

Kidney Case Conference: How I Treat

1732 The Patient Receiving Automated Peritoneal Dialysis with Volume Overload

Emilie Trinh and Jeffrey Perl

1735 Hepatitis C Virus Infection in ESKD Patients

Marco Ladino and David Roth

Nephro pharmacology for the Clinician

1738 Medication Safety Principles and Practice in CKD

Chanel F. Whittaker, Margaret A. Miklich, Roshni S. Patel, and Jeffrey C. Fink

Perspectives

1747 The New HHS Kidney Innovation Accelerator: When Innovation Stalls, HHS Says Floor it!

Kevin John Fowler and Paul T. Conway

See related article on page 1750.

1750 The Kidney Accelerator: Innovation Wanted, Nephrologists Needed

Suzanne Watnick

See related article on page 1747.

1753 Extracorporeal Removal of Light Chains: New Data and Continued Controversies

Kevin W. Finkel and Maurizio Gallieni

1755 Women in Nephrology Today

Eleanor Lederer

1757 Persistent Bias: A Threat to Diversity among Health Care Leaders

Deidra C. Crews and Donald Everett Wesson

Reviews

1760 Regulatory T Cells and Kidney Transplantation

Paloma Leticia Martin-Moreno, Sudipta Tripathi, and Anil Chandraker

1765 Recent Advances in the Management of Autosomal Dominant Polycystic Kidney Disease

Fouad T. Chebib and Vicente E. Torres

On the Cover

What's the diagnosis?

A 24-year-old man was found to have kidney failure and hyperuricemia by a local doctor and was referred to have his kidney failure evaluated. He was neither obese nor hypertensive. The values of serum creatinine, estimated glomerular filtration rate (eGFR), and serum uric acid were 1.31 mg/dl, 58 ml/min/1.73 m², and 9.1 mg/dl, respectively. After one year of follow-up, a kidney biopsy was performed because there was no improvement in his kidney function. The kidney biopsy findings showed minor glomerular abnormalities, and 16 out of 46 glomeruli had global sclerosis accompanied by tubulointerstitial fibrosis. An electron microscopic study revealed a layered structure in the thick ascending limb cell at a low magnification. The layered structure was compatible with hyperplasia of the membranes of the endoplasmic reticulum at a high magnification. A genomic sequence analysis of the UMOD gene revealed a heterozygous mutation of c.707C>G (p.P236R), which is a known pathogenic variant. Febuxostat was increased to 30 mg per day, and his serum uric acid improved to 6.5 mg/dl after 1 year.

Image Descriptions:

Left panel: A layered structure was observed in the thick ascending limb cell at a low magnification.

Right panel: At a high magnification, the layered structure was compatible with hyperplasia of the membranes of the smooth endoplasmic reticulum.

Teaching points:

A heterozygous mutation in the UMOD gene causes autosomal dominant familial juvenile hyperuricemic nephropathy. In the present case, electron microscopy revealed that the transverse section of the thick ascending limb contained a transverse section of the hyperplastic endoplasmic reticulum. The hyperplastic lesion implied that the endoplasmic reticulum reacted to the accumulated mutated uromodulin and led to the definitive genetic diagnosis. Careful examination of electron microscopic images of the tubulointerstitial area might help to diagnose this disease.

(Images and text provided by Keiko Oda, MD (Department of Cardiology and Nephrology, Mie University Graduate School of Medicine, Tsu, Japan), Kan Katayama, MD (Department of Cardiology and Nephrology, Mie University Graduate School of Medicine, Tsu, Japan), Kensuke Joh, MD (Department of Pathology, Tohoku University Graduate School of Medicine, Sendai, Japan), Eiji Ishikawa, MD (Department of Cardiology and Nephrology, Mie University Graduate School of Medicine, Tsu, Japan), Ryuji Okamoto, MD (Department of Cardiology and Nephrology, Mie University Graduate School of Medicine, Tsu, Japan), and Masaaki Ito, MD (Department of Cardiology and Nephrology, Mie University Graduate School of Medicine, Tsu, Japan))