

# CJASN

Clinical Journal of the American Society of Nephrology

December 2020 • Vol. 15 • No. 12

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### 1749 Infection-Related Acute Care Events among Patients with Glomerular Disease

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### 1762 Multi-Autoantibody Signature and Clinical Outcome in Membranous Nephropathy

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Yuvaram N.V. Reddy and Mallika L. Mendu

## Reviews

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### 1838 **AKI!Now Initiative: Recommendations for Awareness, Recognition, and Management of AKI**

*Kathleen D. Liu, Stuart L. Goldstein, Anitha Vijayan, Chirag R. Parikh, Kianoush Kashani, Mark D. Okusa, Anupam Agarwal, and Jorge Cerdá, on behalf of the AKI!Now Initiative of the American Society of Nephrology*

### 1848 **Metabolic Alkalosis: A Brief Pathophysiologic Review**

*Michael Emmett*

#### On the Cover

##### *What is the diagnosis?*

A 63-year-old female was admitted to our hospital with kidney dysfunction without hematuria or proteinuria. Until then, her kidney function had been normal. She had a history of surgical resection of colorectal cancer. She had been taking diuretics for hypertension for 20 years. At admission, serum creatinine was 3.14 mg/dl; calcium, 9.5 mg/dl; and phosphate, 4.1 mg/dl. Her C-reactive protein was low. Urinalysis showed  $\beta_2$  microglobulin of 5510  $\mu\text{g/L}$  (reference range,  $<290 \mu\text{g/L}$ ) and N-acetyl- $\beta$ -D-glucosaminidase of 75 IU/gCr (reference range,  $<5.6 \text{ IU/gCr}$ ), which were measured to evaluate tubular damage.

##### **Image Description:**

Dominant findings of light microscopy were present in the tubulointerstitial compartment, which showed acute tubular injury and calcium phosphate crystals within the tubular lumens and the interstitium. The crystals are shown in purple by Hematoxylin and Eosin stain (left image). Electron microscopy showed an accumulation of prominent crystals with needle shaped margins (right image).

##### **Teaching Points:**

In accordance with the biopsy results, her drug record was revisited, and oral sodium phosphate was found to be provided as a colonic cleansing agent for colonoscopy before the onset of kidney injury. In Japan, sodium phosphate tablets (Visiclear) are still available for bowel preparation and used for patients who have difficulty drinking large amounts of liquid medications. Approximately 4.4 g of elemental phosphorus was administered in this case (usual dietary intake is 1 g/day). Acute phosphate nephropathy is caused by accumulation of calcium phosphate in the distal tubules and collecting ducts. The risks of acute phosphate nephropathy are age, female sex, CKD, renin-angiotensin system inhibitors, and diuretics (1). In general, reversibility of kidney function is limited, but this case showed decreased creatinine to 2.5 mg/dl after 6 months. A careful interview of medical history is important for approaching diagnosis of acute phosphate nephropathy.

##### **Reference:**

1. Markowitz GS, Perazella MA: Acute phosphate nephropathy. *Kidney Int* 76: 1027–1034, 2009

*(Image and text provided by Shinichi Mizuno, Noriyuki Kounoue, Satoru Sanada, and Toshinobu Sato, Japan Community Health Care Organization Sendai Hospital, Nephrology)*