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1692 Depression: A Side Effect of CKD
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1728 Higher-Dose Sitagliptin and the Risk of Congestive Heart Failure in Older Adults with CKD
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**1740 Urinary Soluble CD163 and Disease Activity in Biopsy-Proven ANCA-Associated Glomerulonephritis**
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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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**1785 Depression Screening Tools for Patients with Kidney Failure: A Systematic Review**
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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 β2 microglobulin of 5510 μg/L (reference range, <290 μg/L) and N-acetyl-β-D-glucosaminidase of 75 IU/gCr (reference range, <5.6 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:
(Image and text provided by Shinichi Mizuno, Noriyuki Kounoue, Satoru Sanada, and Toshinobu Sato, Japan Community Health Care Organization Sendai Hospital, Nephrology)