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Contrast-Induced Acute Renal Oxalosis with Sustained AKI in Enteric Hyperoxaluria

Session Information

- Publication Only

Category: Acute Kidney Injury

- 103 AKI: Mechanisms

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Introduction

Intratubular deposition of calcium oxalate crystals induces chronic renal disease in settings of primary hyperoxaluria (chronic renal oxalosis). Acute oxalosis is reported with extreme oxalate or Vitamin C intake and renal transplant settings.

Case Description

A 65-year-old W/M w creatinine 4.62 on chemotherapy screening (CS). Creatinine 1.3 six weeks prior to screening (PTS). CT scan w contrast 4 weeks PTS. PMH. adenocarcinoma of caecum (2013) treated w cytoreduction, HIPEC, right hemicolectomy + resection 2 feet terminal ileum. Chemo-Rx last 6 months was capecitabine-bevacizumab. ROS: No oliguria, flank pain, hematuria, dysuria, fever. Reports 2-4 loose stools/day since 2013 surgery. Serum HCO₃ normal, urine SG ≥ 1.020 x 12 months. Med: lisinopril, loperamide, citalopram, ferrous sulfate. Recently took amoxicillin\clavulanic acid x 7 days for "toe infection." No NSAIDS PE: BP 130/80 no orthostasis, no edema; H/L/A normal. Lab and imaging: BUN 43, creatinine 4.33, bicarbonate 20, uric acid 10.4. Lytes, Ca, Mg, LFTs, CBC normal. Noncontrast CT: normal-sized kidneys, no hydro. U/A: pH 5.5, SG 1.009, P/B/leuk. negative. Sediment: no casts, cells or crystals. UNa 64, Ucreat 82. Renal biopsy: Acute tubular injury with intratubular calcium oxalate deposits DCT c/w oxalate nephropathy. Treatment: 3 L fluid intake, low oxalate diet, Calcium + citrate supplements. Subsequent course: 14 d after peak Creat and prior to therapy, Creat \downarrow to 3.08, 24 hr U oxalate elevated (58 mg), U citrate undetectable. Plasma oxalate undetectable.

Discussion

Proposed mechanisms: PRE-EXISTING CONDITIONS: 1) Enteric mechanisms; \uparrow Ox permeability, \downarrow Ox secretion (intact ileum, proximal colon are net oxalate secretors), \uparrow Ox availability (Ca binding to FFA) . 2) chronic diarrhea; \uparrow U osm, \downarrow U citrate. CONTRAST INDUCED CONDITIONS: 1) Acute supersaturation: a) subclinical tubular injury \rightarrow systemic Ox accumulation \rightarrow acute \uparrow filtered Ox. b) early osmotic diuresis \downarrow washout/tubular stasis. 2) Contrast binding to Tamm-Horsfall protein \rightarrow \downarrow inhibitor crystallization. OTHER: eradication of oxalate degrading gut flora (Oxalobacter formigenes) by antibiotics.

Teaching point: Contrast loads in patients with Enteric hyperoxaluria and diarrhea following bowel resection may induce acute secondary oxalosis with sustained AKI. Appropriate screening, prophylaxis, imaging alternatives are advised.