ABSTRACT: PUB609

Silent Hyperoxaluria Contributes to CKD Progression in a Patient with Short Bowel

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BACKGROUND

Hyperoxaluria is common in patients with short bowel syndromes due to enhanced colonic resorption of soluble oxalate, which can result in symptomatic calcium-oxalate nephrolithiasis and nephrocalcinosis. There are also reports of subtle calcium oxalate crystal deposition contributing to progressive CKD.

METHODS

A 75 year-old white female presented with elevated creatinine. She had a history of left nephrouretectomy and ileal resection secondary to urothelial carcinoma and carcinoid syndrome; HTN; and anxiety/depression. She reported frequent loose stools following meals for which she took diphenoxylate-atropine. She denied flank pain, dysuria, and kidney stones. She limited her liquid intake in order to reduce the frequency of bowel movements.

Creatinine was 1.4 mg/dl prior to nephrouretectomy and gradually rose to 2.5 mg/dl over 4 years. Renal ultrasound revealed a 9.6 cm right kidney with normal echotexture and no nephrolithiasis. The left renal fossa was replaced by scar tissue. Kidney biopsy had hypertensive glomerular changes with moderate interstitial fibrosis and tubular atrophy. Few tubular oxalate crystals were present with focal extravasation into the interstitium with granulomatous inflammation. No immune complexes were detected.

Discontinuation of a PPI, chlorthalidone, and fluoxetine and addition of a short course of steroids for the nephritis failed to improve the creatinine. A 24-hour urine assessment showed hypocalciuria; hyperoxaluria (63 mg/day; normal < 45); hypocitraturia; urine pH of 5.7; volume of 1.4 L.

CONCLUSION

Chronic calcium oxalate deposition in the tublointerstitium can lead to nephritis. While this patient's progressive CKD was multi-factorial, including hyperfiltration of a solitary kidney and HTN, the calcium oxalate crystal deposition was likely contributing. Despite a significantly reduced GFR, hyperoxaluria was evident on 24 hour urine collection. Due to the absence of symptomatic and radiologic nephrolithiasis, interstitial calcium oxalate deposition might otherwise be excluded from the differential diagnosis in this setting. This underscores that with ileal resection and CKD, hyperoxaluria should be considered. Early implementation of hyperoxaluric therapy might mitigate CKD progression

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