

Introduction

Oxalate is a renally excreted end-product of metabolism. Excess urinary oxalate is associated with renal inflammation, fibrosis, and progressive renal failure [1]. Underlying mechanisms for increase in urinary oxalate in post-bariatric surgery patients are not completely understood, however, they may be accounted for by dietary factors, intestinal fat malabsorption, alterations in gut microbiota, and changes in intestinal oxalate transport [2]. We report a case of gastric bypass-induced oxalate nephropathy.

Case Presentation

A 55-year-old female with a history of gastric bypass, diabetes, and hypertension presented to hospital following routine lab evaluation which revealed an elevated creatinine of 4.05 from her baseline of 0.84. Her urinalysis was notable for hematuria and albuminuria, and her 24-hour urine revealed non-nephrotic range proteinuria. Ultrasound of her kidneys showed normal sized echogenic kidneys, and her BUN/Creatinine was not suggestive of a pre-renal etiology.

A broad differential of ATN/AIN/GN/oxalate nephropathy was considered. Renal serologies, including ANA, ANCA, SPEP/UPEP, complement, Hepatitis B/C, and PLA2R Ab, were all negative. The patient refused renal biopsy, and was discharged after seven days with only slight improvement of creatinine to 3.54. Unfortunately, she did not follow-up. Approximately two months later, she presented to ED with uremic encephalopathy and acute renal failure with a creatinine of 11.2. She was started on emergent hemodialysis, and renal biopsy revealed tubulointerstitial fibrosis consistent with oxalate nephropathy.

Discussion

Oxalate nephropathy is characterized by tubular crystalline deposits of calcium-oxalate leading to acute and chronic tubular injury, interstitial fibrosis, and progressive renal insufficiency [3]. Bariatric surgery has been shown to achieve sustained weight loss and is also effective in improving blood pressure, reducing hyperglycemia, and even inducing diabetes remission (as was the case in our patient).

Unfortunately, there are renal risks in bariatric surgery, namely, acute kidney injury, nephrolithiasis, and in rare cases, oxalate nephropathy, particularly in types of surgery involving higher degrees of malabsorption [4]. The prognosis of oxalate nephropathy after RYGB seems to be dismal, with progression to ESRD within three months in 72.7% of patients in one study [3]. Primary sources of dietary oxalate are plants/plant products, principally seeds and leafy plants related to spinach and rhubarb [5]. Currently, there are no specific guidelines for measuring urinary oxalate levels to monitor for hyperoxaluria post bariatric surgery.

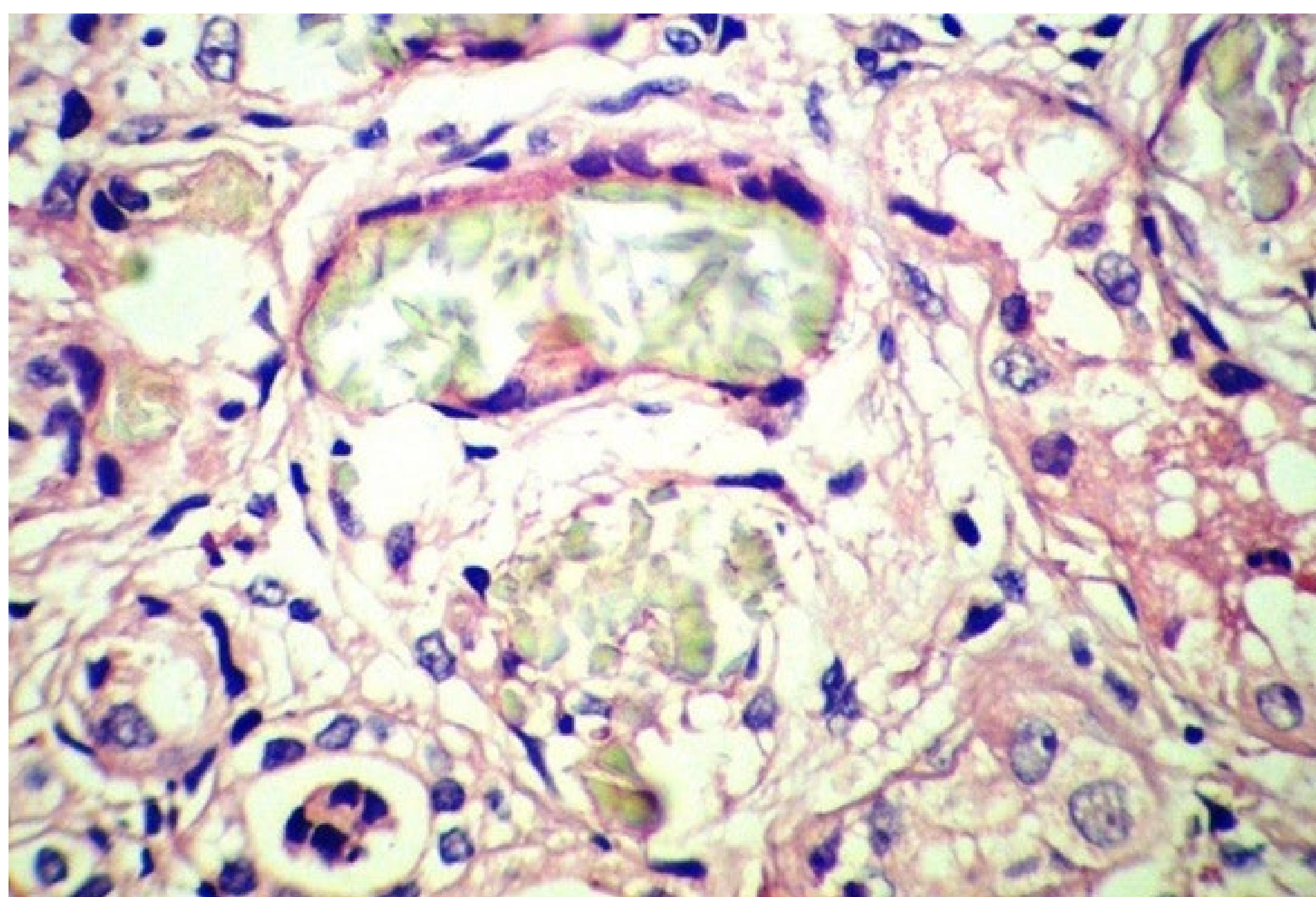


Figure 1. Intratubular Calcium Oxalate Crystals [6]

Conclusion

The prevalence of severe obesity continues to rise, with more than one-fifth of CKD patients in the United States having a BMI >35 kg/m². Our case intends to provide awareness, promote postoperative renal function monitoring and educate patients prior to gastric bypass, as well as caution against a high oxalate, plant-based diet post-surgically. If detected, oxalate nephropathy can be slowed with a combination of low oxalate diet, adequate hydration, and increased calcium intake.

References

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