Severe Steatorrhea in the Setting of Chronic Pancreatitis Leading to Hyperoxaluria and Subsequent Oxalate Nephropathy Necessitating Dialysis

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Introduction

Steatorrhea is a common clinical entity encountered by gastroenterologists spanning a wide array of patient populations. In addition to causing disruptive symptoms which negatively impact quality of life, steatorrhea leads to fat-soluble vitamin deficiency and the corresponding adverse effects including mineral and bone disease, clotting factor deficiency, and delayed wound healing. While multiple disorders can lead to steatorrhea, this report will focus on chronic pancreatitis. Regardless of the etiology, some patients may ultimately develop hyperoxaluria due to the interaction between non absorbed fat in the gut lumen, calcium, and oxalate. This has the potential to lead to oxalate nephropathy, and in extreme cases, acute renal failure. We herein present the case of an 89-year-old man receiving treatment for chronic pancreatitis who developed acute renal failure requiring dialysis secondary to oxalate nephropathy.

Case Description / Methods

Our case involves an 89-year-old man with a past medical history significant for chronic pancreatitis on enzymes replacement, IPMN, and COPD who presented to the hospital at the request of his primary physician for abnormal blood work. On initial evaluation, he was found to be in acute renal failure with a BUN of 104 and creatinine 9.34. Gastroenterology and nephrology teams were consulted. During the course of his stay, our patient was started on dialysis and subsequently underwent kidney biopsy, which revealed oxalate nephropathy. He did not undergo invasive testing such as EGD with secretin administration and subsequent duodenal bicarbonate aspiration to confirm pancreatic insufficiency, as this diagnosis was assumed from prior gastroenterology evaluation and appropriate clinical findings. He was discharged home after kidney function was stabilized and continues on dialysis at this time.

Figure 1. Histologic representation of oxalate nephropathy. Figure Demonstrates acute tubular injury with intratubular crystalline deposits.

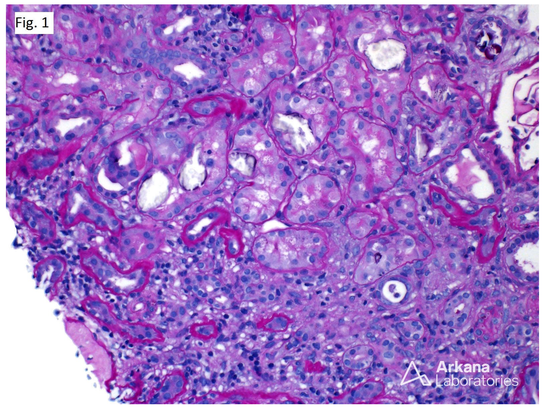

Figure 2. CT Abdomen / pelvis w/o contrast demonstrating findings of chronic pancreatitis with prominent pancreatic head calcification

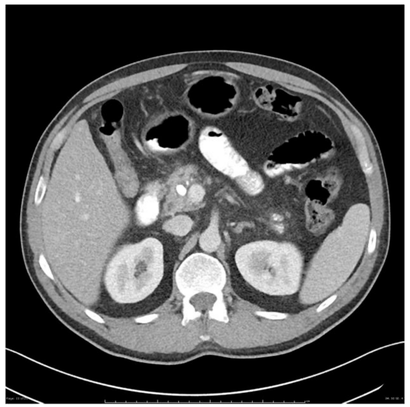

Figure 3. Pathologist report for patient's kidney biopsy

Comment: The predominant biopsy finding is that of diffuse acute tubular injury with associated calcium oxalate deposition within tubular lumens. Accordingly, conditions associated with hyperoxaluria should be considered as a possible contributing factor for this patient's acute tubular injury. Known causes of oxalate nephropathy include primary hyperoxaluria, ethylene glycol intoxication, enteric hyperoxaluria (e.g., due to gastric bypass, chronic pancreatitis, small bowel resection, or malabsorption), vitamin B6 deficiency, and excessive ingestion of vitamin C or dietary substances rich in oxalic acid (parsley, nuts, star fruit, sweet tea, etc.), and various drugs (e.g., methoxyflurane, Orlistat, Praxilene, COX-2 inhibitors), among others.

Discussion

This case presentation highlights a potential adverse outcome associated with severe steatorrhea in the setting of chronic pancreatitis, and is a useful lesson for the gastroenterologist so that similar events might be detected at an early stage in future patients. Recognizing acute kidney injury in this setting is of the utmost importance, so that progression to ESRD requiring dialysis may be avoided. Management of steatorrhea in this setting may require aggressive pancreatic enzyme replacement and dietary therapy. Fecal fat quantity may be monitored with stool sudan stain as well as fecal elastase, in an effort to avoid invasive testing. Fat soluble vitamins should also be measured.

Conclusions

Chronic pancreatitis is a common clinical entity encountered by community gastroenterologists and academic centers. In addition to having profound negative effects on patient quality of life, it can lead to severe consequences such as steatorrhea, chronic pain, weight loss, diabetes, and pancreatic cancer. The clinician must remain vigilant to detect sequela of this disease. This case highlights a rare consequence of steatorrhea and reviews important pathophysiology which can lead to hyperoxaluria and the resulting complications.

Future Directions

This patient should ideally have a fecal elastase-1 measured, in addition to 72 hour fecal fat collection to confirm the diagnosis. Unfortunately, these labs are not available at this time. Pt continues on dialysis and needs further workup into possible pancreatic head mass noted on MRI abdomen.

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References

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