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INTRODUCTION

- Acute interstitial nephritis (AIN), also called tubulointerstitial nephritis, is a renal pathology that can cause a significant decline in kidney function.
- Drug-induced AIN accounts for 75% of the cases and is often due to non-steroidal anti-inflammatory drugs (NSAIDs), antimicrobials, and proton pump inhibitors.
- However, there have been isolated reports of other drugs being responsible for AIN. We hereby report a case of furosemide-induced AIN.

CASE PRESENTATION

- A 68-year-old Caucasian male with a medical history significant for chronic kidney disease (CKD) stage 3 due to hypertensive nephrosclerosis with a baseline serum creatinine (Cr) of 1.3-1.5, hypertension, hyperlipidemia, atrial fibrillation, heart failure with preserved ejection fraction (HFpEF), and hypogonadism was admitted for evaluation of worsening renal failure.
- At initial evaluation, the patient had nonspecific symptoms like malaise, nausea, and vomiting but denied any other complaints.
- Physical examination was unremarkable, without any rashes or abdominal bruit.
- The patient's creatinine progressively trended up from his baseline to 3.5 over three months.
- Pre-renal pathology was suspected initially, and patient's furosemide was held on admission with concurrent fluid resuscitation.
- However, this did not improve his kidney function as repeat lab work showed a worsening Cr level of 4.4, along with BUN of 72.
- Further evaluation showed a complete blood count significant for mild eosinophilia with urinalysis revealing hematuria, pyuria with eosinophiluria but no protein, WBC casts, or RBC casts.
- Renal ultrasound and abdominal CT scan were unremarkable.
- The patient had no known drug allergies until that point and was on a stable medication regimen for his chronic conditions for several years, except for a daily dose of furosemide started three months ago for fluid retention and elevated BNP.
- Ultrasound-guided renal biopsy revealed findings consistent with acute interstitial nephritis on top of chronic tubulointerstitial fibrosis plus underlying moderate arterial sclerosis from hypertension.
- Other extensive workup was negative for any autoimmune process, IgG4 related disease, sarcoidosis, or infection, thus favoring the diagnosis of drug-induced acute interstitial nephritis.
- Given the temporal relationship between the initiation of furosemide in this patient and his worsening kidney function makes it the likely offending agent.
- He was observed off furosemide without any immunosuppressant treatment.
- The patient's creatinine level gradually trended down and ultimately returned to his baseline at one month follow up.

DISCUSSION

- Furosemide is a loop diuretic, often used in patients to prevent volume overload.
- Therefore, furosemide is often implicated as a cause of pre-renal acute kidney injury (AKI) secondary to volume depletion.
- However, interstitial inflammation as a mechanism of furosemide-induced kidney injury is uncommon and can often be overlooked as a potential cause, especially in patients with long medication lists.
- In such patients, a causal link can be established by correlating the onset of decline in kidney function with the time of initiation of a new drug and resolution of AKI after discontinuation of the drug.

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