SciMedCentral

Journal of Clinical Nephrology and Research

Clinical Image

# Acute Tubulo-Interstitial Nephritis due to Proton-Pump Inhibitors

**Rakesh Malhotra[1]* and Omar Al Nimri[2]**

[1]*Division of Nephrology and Hypertension, University of California San Diego, USA*
[2]*Division of Nephrology and Hypertension, Vanderbilt University School of Medicine, USA*

***Corresponding author**
Rakesh Malhotra, 3350 La Jolla Village Drive, 9111H, San Diego, CA 92161-9111, USA, Tel: (858) 552-8585; Fax: (858) 552-7549; Email: r3malhotra@ucsd.edu

**Submitted:** 24 February 2017
**Accepted:** 11 April 2017
**Published:** 14 April 2017
**ISSN:** 2379-0652



## CLINICAL IMAGE

### Case

A 74-year-old female presented to emergency department with complains of generalized weakness, malaise, abdominal pain and decreased appetite for last 1 month. She had a past medical history significant for hypertension (HTN), type 2 diabetes, and stage 3b chronic kidney disease (baseline serum creatinine (sCr), 1.4 mg/dl; estimated glomerular filtration rate 39 ml/min using *Chronic Kidney Disease Epidemiology* Collaboration (CKD-EPI) equation). On presentation, her physical examination revealed a blood pressure of 130/77 mm Hg and pulse 68/min; conjunctivae were pale with anicteric sclera. Heart and lung examination was unremarkable. There was no pedal edema, skin rash, petechiae or purpura. Laboratory results showed elevated blood urea nitrogen of 56 mg/dL and sCR of 2.3 mg/dl. Her hemoglobin was 10.6g/dL. Urine microscopy revealed 1+ protein and with 4-6 WBCs/hpf. No peripheral eosinophilia and eosinophiluria was seen. The remaining laboratory including plasma biochemistry, complements were within normal range. Serological markers for hepatitis B virus, hepatitis C virus, and HIV were negative, and no cryoglobulins were detected. Renal ultrasonography showed enlarged kidneys with increased cortical echoes. Patient was started on intravenous fluids however her sCr kept worsening during hospitalization with peak sCr of 3.8 mg/dL. Nephrology consult was obtained and kidney biopsy showed marked interstitial lymphocyte and eosinophilic inflammation with active tubulitis and acute tubal injury. Biopsy also showed chronic features including interstitial fibrosis and tubular atrophy. Overall, findings were consistent with severe acute tubule-interstitial nephritis (AIN). The patient had initiated treatment with oral esomeprazole 2 months prior to admission, indication unknown. Other home medications include aspirin 81 mg daily, simvastatin 40 mg daily, Lisinopril 20 mg daily, metformin 500 mg twice daily, and ferrous sulfate 325 mg two times daily. Esomeprazole was discontinued in view of the diagnosis of AIN. Patient was started on oral prednisone 1mg/kg for 8 weeks with further tapering. The patient sCr gradually improved with subsequently sCR leveled off at 1.3 mg/dl (Figure 1).

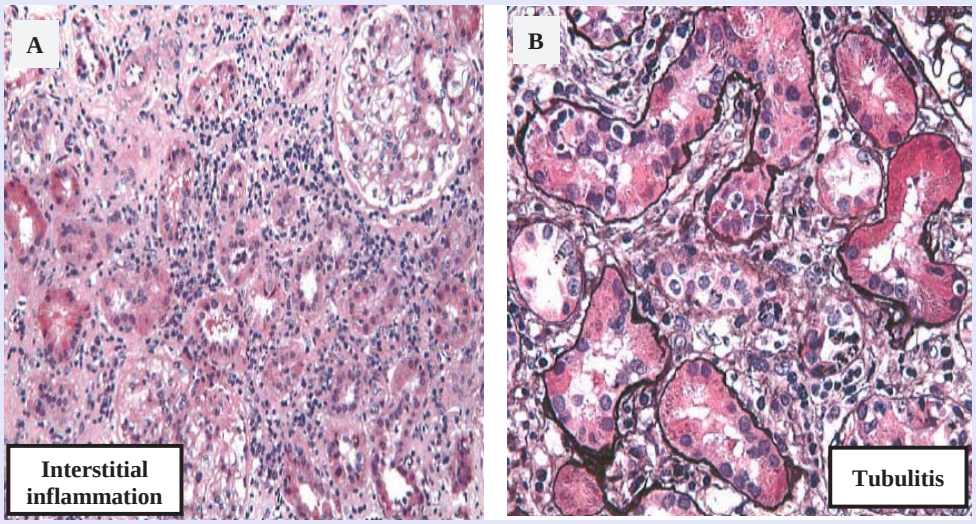


**Figure 1** Acute Tubulo-Interstitial Nephritis due to Proton-Pump Inhibitors.

### Diagnosis

Acute Tubulo-Interstitial Nephritis due to Proton-Pump Inhibitors

## DISCUSSION

Drugs induced kidney disease is a frequent cause of renal dysfunction and accounts for approximately 19-26% of cases of AKI in hospitalized patients [1]. Over the past decade, the incidence of Proton pump inhibitors (PPIs)-induced AKI in the community is growing rapidly [2,3]. In one case series of 133 patients with biopsy proven AIN, 14% of patients developed AIN from use of PPIs [4]. The development of clinical AIN occurs anywhere from 1 week to 9 months after initial exposure to the PPI.The clinical presentation of PPI-induced AIN are non-specific and includes fever, nausea, vomiting, or loin pain. The pathogenesis of PPI-associated AIN is unclear and may be related to the involvement of both humoral and cellular immune mechanisms. Treatment strategies for AIN include withdrawal of offending drug and early initiation of steroids. PPI use is also associated with increased risk of incident CKD and CKD progression [5].

## CONCLUSION

PPIs are widely prescribed to treat a number of acid-related gastrointestinal disorders. We report a case of reversible acute renal failure due to AIN, confirmed by histology of a renal biopsy sample, associated with taking esomeprazole. Our patient did not have classic triad of hypersensitive reaction which includes fever, rash and eosinophilia. Physicians should be aware of PPI induced AIN. Accurate and timely diagnosis and withdrawal of the offending drug can prevent potentially life-threatening renal failure.

## REFERENCES

1. Mehta RL, Pascual MT, Soroko S, Savage BR, Himmelfarb J, Ikizler TA, et al. Spectrum of acute renal failure in the intensive care unit: the PICARD experience. Kidney Int. 2004; 66: 1613-1621.

2. Antoniou T, Macdonald EM, Hollands S, Gomes T, Mamdani MM, Garg AX, et al. Proton pump inhibitors and the risk of acute kidney injury in older patients: a population-based cohort study. CMAJ Open. 2015; 3: 166-171.

3. Blank ML, Parkin L, Paul C, Herbison P. A nationwide nested case-control study indicates an increased risk of acute interstitial nephritis with proton pump inhibitor use. Kidney Int. 2014; 86: 837-844.

4. Muriithi AK, Leung N, Valeri AM, Cornell LD, Sethi S, Fidler ME, et al. Biopsy-proven acute interstitial nephritis, 1993-2011: a case series. Am J Kidney Dis. 2014; 64: 558-566.

5. Xie Y, Bowe B, Li T, Xian H, Yan Y, Al-Aly Z. Long-term kidney outcomes among users of proton pump inhibitors without intervening acute kidney injury. Kidney Int. 2017.

**Cite this article**

*Malhotra R, Al Nimri O (2017) Acute Tubulo-Interstitial Nephritis due to Proton-Pump Inhibitors. J Clin Nephrol Res 4(2): 1060.*