OMICS Group Conferences
Accelerating Scientific Discovery

2nd International Conference on
Nephrology & Therapeutics
July 29-30, 2013 Embassy Suites Las Vegas, NV, USA

Pain relief with leaky kidneys: An uncommonly recognized common culprit

Anusha Nallaparaju, Thankam Nair and Jones Samuel
Tulane University School of Medicine, USA

Introduction: Non-steroidal anti-inflammatory drugs (NSAIDs) are widely used in daily lives and clinical practice. Studies have shown that 5-8% of patients receiving NSAIDs can suffer from sub-clinical renal dysfunction manifested as reduced creatinine clearance. Renal dysfunction can also manifest as acute tubulointerstitial nephritis and nephrotic syndrome (NS) which is a less recognized side effect of NSAIDs.

Case: A 62-year-old white male with past medical history of osteoarthritis and hypertension presents with 5 day history of lower extremity and facial swelling. He also reports decreased urine output in the last 24 hours. Upon further questioning he reveals that he has been periodically on NSAIDs for 1 year, and diclofenac 75mg twice daily for 1 week. The remaining review of systems was negative. Physical examination: Obese white male. Blood pressure: 122/82mmHg, Pulse rate: 75/min, RR: 19/min, Temp: 98.6. Facial and peri-orbital edema present, 2+ pitting edema at the ankle and 1+ pitting edema bilaterally to thighs. No sacral edema or scrotal edema. No skin rash.

Subsequent investigation revealed elevated creatinine, nephrotic range proteinuria, low albumin and high cholesterol levels. A diagnosis of nephrotic syndrome secondary to NSAIDs was considered with differential diagnosis as primary minimal change disease (MCD), membranous nephropathy, focal glomerulosclerosis and idiopathic. Serologic work up was unrevealing and serum protein electrophoresis (SPEP) was negative.

The renal biopsy showed non-specifically globally sclerosed glomeruli with minimal interstitial fibrosis and 80-90% foot process effacement by EM representing minimal change disease, and no segmental sclerosis or immune complex deposits. Course: Diclofenac was discontinued, and the patient started on diuretics, statins, a beta-blocker and alpha agonist. ACE-I was re-instated as his renal function improved. He was not started on steroids or immunosuppresants. He was discharged on the 5th day and followed up as an outpatient. He was weaned off of diuretics within 2 weeks,and follow up labs demonstrated resolution of renal failure and proteinuria.

Discussion: This case illustrates one of the rarer manifestations of secondary MCD with NS, the mechanism of which is postulated as a hypersensitivity reaction with a concomitant activation of the humoral immune mechanism. The activated lymphokines induce podocyte disturbances, thereby changing the permeability leading to pronounced proteinuria. NSAIDs also induce hemodynamically-mediated acute kidney injury by inhibition of prostaglandin synthesis resulting in reversible renal ischemia and a decline in glomerular hydraulic pressure. With regard to renal adverse effects there is no significant difference between NSAIDs and Selective COX2 inhibitors. In this case, the patient achieved complete remission upon discontinuation of the NSAIDs and supportive therapy. Thus, steroids or immunosuppressants should be reserved for patients with protracted deterioration of renal function after discontinuation of offending trigger agents. Given the aging population and the exponential rise in the use of NSAIDs, awareness and prompt recognition of the side effects will be useful in the future.

Biography

Anusha Nallaparaju is a current PGY-2 resident at the Baton Rouge General Internal Medicine Residency Program, affiliated with Tulane University School of Medicine, in Baton Rouge, Louisiana. She graduated from G.S.L Medical College, India in 2010 and subsequently started working as a research assistant at New York Downtown Hospital, in New York City. She also worked as a Clinical Research Coordinator for two Phase -2 clinical trials prior to joining her residency program.

anusha.nallaparaju@gmail.com