

# An ALEVE-iating Diagnosis: A Case of NSAID Induced Acute Renal Failure

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## BACKGROUND

- Non-steroidal anti-inflammatory drugs (NSAID) are well known to cause worsening renal function and ultimately chronic kidney disease (CKD) progressing to end stage renal disease (ESRD)<sup>1</sup>
- Damage occurs through two mechanisms:
  1. Reduced renal plasma flow from a decrease in prostaglandins<sup>2</sup>
  2. Acute tubulointerstitial nephritis (ATIN) from inflammatory cells infiltrating the interstitium<sup>2</sup>
- Damage usually occurs from long-term use (years) rather than weeks or months<sup>1</sup>

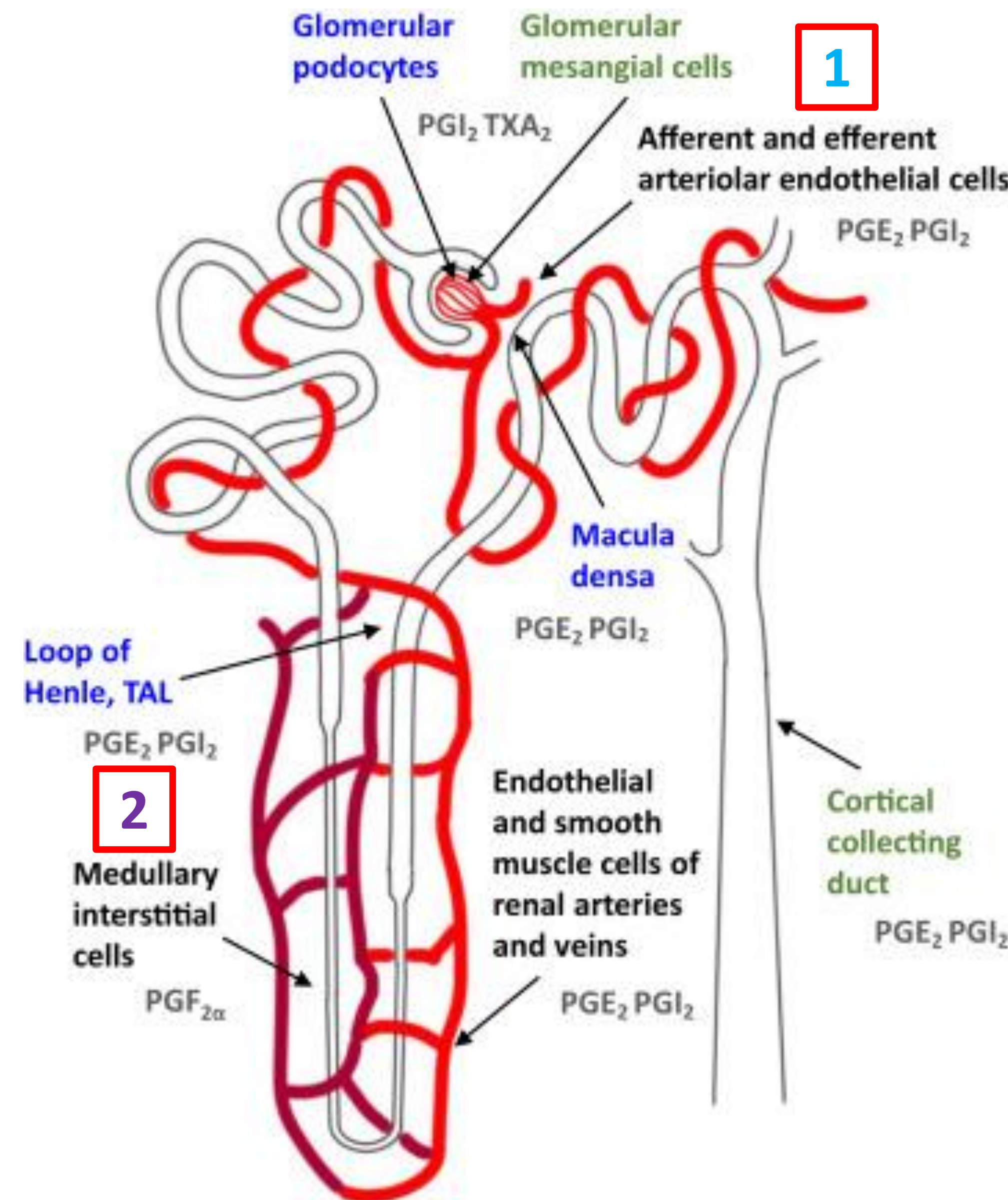
## CASE PRESENTATION

- 58 y/o African American female with PMH of HTN, COPD, Asthma, OSA and Anxiety presented to the ER with diffuse crampy abdominal pain and associated generalized weakness, lethargy, decreasing urination, darker urine, loss of appetite and nausea without vomiting
- Five weeks prior the patient had presented to the ER for right hip and leg pain
  - Diagnosed with spinal stenosis and spondylosis -> Discharged home
- Below is labs during prior admission compared to current admission

- Blood Urea Nitrogen 12 -> **149**      Creatinine 1.03 -> **20.86**
- Glomerular Filtration Rate >60 -> **2**      Potassium 4.4 -> **6.6**

- History revealed since discharge, 5 weeks prior, the patient had been using at least 6 tablets each of Diclofenac and Ibuprofen daily for pain control

- CT of the abdomen: No pathology
- Renal Ultrasound: Medical renal disease
- Autoimmune workup, Multiple Myeloma workup, Eosinophil Smear, Methanol and Ethylene glycol were all negative or within normal limits
- Urine studies, BUN/Cr ratio and osmolality were consistent with ATN
- **Renal biopsy: Interstitial edema, severe tubular injury, findings consistent with Acute Tubular Necrosis (ATN)**



**Figure 1: Prostaglandin synthesis in the renal system and NSAID's mechanism of damage<sup>2,4</sup>**

1. Reduced renal plasma flow from a decrease in prostaglandins<sup>2</sup>
2. Acute tubulointerstitial nephritis (ATIN) from inflammatory cells infiltrating the interstitium<sup>2</sup>

## TREATMENT

- Holding all nephrotoxic medications (stopping the offending agent)<sup>1,2</sup>
- Supportive care<sup>1,2</sup>
  - Adequate hydration
  - Dialysis as needed (fluid and electrolyte management)
- Hopeful renal function recovery<sup>1,2</sup>

## DISCUSSION

- While it is well understood chronic NSAID use can cause CKD and ESRD, NSAID induced acute renal dysfunction is not as well known<sup>1</sup>
- Workup includes CT Abdomen, Renal US, Urine Studies, ruling out autoimmune causes/multiple myeloma, and renal biopsy<sup>1,3</sup>
- While NSAID induced Acute Renal Failure (ARF) cannot be confirmed, our patient's history of significant NSAID use, biopsy findings of ATN and ATIN and negative workup for alternative diagnoses, makes NSAID induced ARF most likely<sup>2</sup>
- This case represents the need for research into NSAID induced acute renal impairment as NSAID use is extremely prevalent and likely under recognized as a cause of acute renal disease

## REFERENCES

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