Episode 21

In this week's episode of the CPSers, Dr. Daniel Restrepo joins the crew to discuss his schema for intrarenal acute kidney injury (AKI).

Problem Representation
A 49-year-old otherwise healthy woman presented after strenuous exercise with acute progressive malaise and diminished urine output, found to have an AKI with urinalysis demonstrating 3+ blood but only 5-10 RBCs on microscopy, elevated creatinine kinase, and nephritic range proteinuria.

Schemas
The CPSers' schema differentiates between issues with the "plumbing" (hemodynamics/"pre-renal" and the collecting system/"post-renal") and intrarenal AKI. It then divides intrarenal AKI into pathology of the glomerulus, tubules, interstitium, and medium-sized vessels supplying the kidney.

Diagnosis
ANCA and anti-GBM were negative, and the patient had normal complement levels. Ultimately, a renal biopsy was performed and demonstrated findings consistent with pigment nephropathy. The patient was diagnosed with rhabdomyolysis-induced AKI.

Teaching points

- **Pre-renal physiology** (secondary to a variety of hemodynamic factors) is common in the hospital setting and can be *suggested* by an elevated BUN/creatinine ratio (>20), low urine sodium, and/or elevated urine specific gravity. The gold standard for diagnosing pre-renal physiology, however, is resolution of kidney injury with empiric treatment (e.g., fluid challenge if hypovolemic, diuresis for congestive heart failure).
- Injury to the glomerulus[1] leads to impaired filtration of different blood constituents and classically manifests as **nephritis** (e.g., RBCs, proteinuria, RBC casts) or **nephrosis** (e.g., severe proteinuria leading to hypoalbuminemia, edema, and hyperlipidemia) depending on the site and nature of glomerular injury (although considerable overlap can exist). Complement levels, ANCA, & anti-GBM serologies are useful in the workup of glomerulonephritis, but **renal biopsy** is often used as a confirmatory test.
- Rhabdomyolysis[2] is a clinical syndrome of striated muscle injury that can be complicated by severe electrolyte abnormalities & acute renal failure. Renal injury results from a combination of severe hypovolemia with altered renal hemodynamics, mechanical obstruction of renal tubules by myoglobin precipitates, and direct reno-toxic effects of heme pigments.

Clinical Reasoning Pearl

While testing is often a necessary step in the diagnostic process, Dr. Restrepo reminds us to use our history to inform our initial clinical impressions. Every test must be evaluated in the context of *our* pretest probability for a given disease.

For example:
If our suspicion for rhabdomyolysis was not as high based on this patient's history, the absence of severe elevation in the CK may have dissuaded us from pursuing this diagnosis.

References

1. Sauret JM, Marinides G, Wang GK. Rhabdomyolysis. Am Fam Physician. 2002 Mar 1;65(5):907-12.
2. Madaio MP, Harrington JT. The diagnosis of glomerular diseases: acute glomerulonephritis and the nephrotic syndrome. Arch Intern Med. 2001 Jan 8;161(1):25-34.