CLINICAL NEPHROLOGY PEARLS

WHAT EVERY NEPHROLOGIST WISHES PRIMARY CARE PROVIDERS KNEW

Cases

Consult
32 year old white male dentist
Creatinine of 1.9
BUN of 12

Consult
63 YOWF, bradycardia
Creatinine of 1.9
BUN of 15

Consult
39 YOWM
Creatinine of 2.0
BUN of 19

High levels of ascorbic acid and cephalosporin antibiotics can cause a false elevation of serum creatinine. Steroids, trimethoprim, pyrimethamine, phenacemide, salicylates and vitamin D metabolites.

A diet high in meat will elevate serum Creatinine levels. Cooking Meat increases the conversion of creatine in the meat to creatinine.

Ketoacidosis will increase serum creatinine.

Creatinine is falsely decreased by bilirubin, glucose, histidine and quinidine compounds.

Creatine used by body builders
Promotes protein synthesis and will
falsely elevate serum creatinine.

Intense exercise can increase serum creatinine by increasing muscle breakdown.

Hemolysis and lipemia can cause a false elevation of serum creatinine.

Cephalosporins (especially cefoxitine and cephalothin) will cause false elevation of serum creatinine.

Barbiturates, n-acetylcysteine, flucytosine and nitromethane will cause a false rise in serum creatinine.

Trimethoprim, cimetidine and other H2 blockers block the tubular secretion of creatinine causing a false rise in serum creatinine.

Fenofibrates impair the generation of vasodilatory prostaglandins altering intrarenal hemodynamics causing false elevation of serum creatinine.

Dronedarone (anti arrhythmic) causes false elevation in creatinine.

Tenofovir disproxil fumarate (TDF) will cause false elevation of creatinine by blocking tubular secretion.

Rhabdomyolysis causes rapid rise in serum creatinine by releasing creatine from injured muscle cells that gets converted into creatinine.

Active vitamin D metabolites, corticosteroids and fenofibrate cause increased release of creatinine from muscle leading to false elevation in serum creatinine.

Phenyl acetamide, pyrimethamine, and salicylates and NSAIDS reduce proximal tubular secretion of creatinine causing a false elevation of serum creatinine.

Dopamine, ketoacids and bilirubin interfere with the analysis for creatinine and will lead to false elevation of serum creatinine.

Compounds that can discolor the urine.

Phenazopyridine

Cascara

Diphenylhydantoin

Rifampin

Methyldopa

Phenacetin

Phenindione

Phenolphthalein

Phenothiazine

Senna

Beets

Compounds that can cause crystals in the urine.

Acyclovir (and class drugs)
Ampicillin
Indanavir
Sulfa

Compounds that are associated with acute allergic interstitial nephritis

NSAIDS

Cox-2 inhibitors

Penicillins and cephalosporins

Rifampin

Sulfamethoxazole

Loop and thiazides diuretics

Ciprofloxacin and other fluroquinolones

Cimetidine and rarely other H2 blockers

Allopurinol

Proton pump inhibitors

Indinavir

5-aminosalicylates

Infections associated with interstitial nephritis

Legionella

Leptospirosis

CMV

Streptococcus

Mycobacterium tuberculosis

Corynebacterium diphtheriae

EBV

Yersinia

Polyomavirus

Mycobacterium

Histoplasmosis

Brucellosis

Chlamydia

A 'normal' serum creatinine level may not be normal.

The margin of error in lab analysis of serum creatinine is 0.2 or 10-20%

GFR should only be assessed at steady state. When kidney function is variable, eGFR is not valid.

Know the medications that can transiently cause a false elevation of serum creatinine.

Patients with decreased GFR or proteinuria should be evaluated to determine the cause.

Positive urine dipsticks for protein should be followed up by spot urine protein to urine creatinine ratio or 24 hour urine collection for creatinine clearance.

ANY abnormal protein loss via the kidneys should be investigated.

In patients with early stage CKD, periodic evaluation and intervention are appropriate to slow the progression of kidney disease and avoid its complications.

Avoid nephrotoxins
Strive for BP less than 130/80
Use ACEI or ARB when possible
Monitor for renal osteo dystrophy
Monitor for Heart disease

Do not automatically discontinue an ACEI or ARB solely because of a small increase in serum creatinine.

A rise in serum creatinine after starting ACEI or ARB is acceptable.

A potassium level of up to 5.5 is acceptable after starting ACEI or ARB as long as it remains stable.

Anemia in patients with CKD should be treated with an ESA

Also look for other causes of anemia.

CKD anemia is associated with: fatigue, reduced exercise tolerance, dyspnea, left ventricular enlargement, left ventricular systolic dysfunction, and increased risk of heart attack or stroke.

Phosphate containing bowel preparations should be used with caution in patients with CKD.

Sodium phosphate bowel preps can cause acute kidney injury even in patients with normal eGFR.

Safe alternatives include:

Polyethylene glycol

Miralax

Lactulose

Patients with CKD should avoid products containing magnesium or aluminum.

Products that contain calcium citrate, sodium citrate and potassium citrate will markedly increase aluminum absorption.

Chronic aluminum toxicity has been linked to Alzheimer' dementia.

Screen patients for secondary hypertension. When addressed, this may improve blood pressure and kidney function.

Of patients with the diagnosis of HTN, 95% have primary HTN.

Causes of secondary hypertension

Diabetes Mellitus.

Polycystic kidney disease.

Glomerular disease.

Renal artery stenosis.

Cushing's disease.

Aldosteronism.

Pheochromocytoma.

Thyroid disease.

Hyperparathyroidism.

Coarctation of the aorta.

Sleep apnea.

Obesity

In patients with recurrent stone disease, in depth analysis may preserve kidney function.

Order urinalysis.

Order Lith-O-Link

Refer to nephrology.

- Most patients with CKD have problems with phosphorous levels. The struggling kidneys can not eliminate phosphorous and it accumulates in the blood stream.
- The increased phosphorous causes binding to serum calcium and therefor lowers the serum calcium level.
- Many patients will need to take phosphate binders.

- Most patients with CKD have problems with anemia due to decreased production of erythropoietin production.
- If you are working a CKD patient up for anemia, include an erythropoietin level or referral to nephrology.

- Most patients with CKD have problems with vitamin D2 and D3 metabolism.
- Vitamin D2 is metabolically active and may need to be replaced.
- If the patient has CKD, they may not be able to convert D2 into D3 and will need replacement of vitamin D3 also.

- Many patients with CKD may develop metabolic acidosis.
- This will weaken their bones.
- Treat with oral sodium bicarbonate.

- Some patients with kidney stones will take potassium citrate to treat.
- It is the citrate portion of the molecule that treats the kidney stones.
- Do NOT stop the potassium citrate without discussion with the prescribing physician.

- Some patients with CKD may have hyperkalemia.
- This may be due to renal tubular acidosis type
 IV or decreased elimination of potassium.
- If they are taking an ACEI or ARB, it's may need to be discontinued.
- There are new potassium absorption blockers. Veltassa and Lokelma.

- Some patients with CKD may develop endothelial calcification of blood vessels and heart valves.
- Look for this in patients with wide pulse pressure hypertension.
- High The PTH level stimulates the osteoblasts to migrate from bone to endothelial lining.
- The osteoblasts begin to lay down bone on the endothelial lining.

- Talking with your patients about their kidney function using serum creatinine Vs eGFR.
- Patient: SCr=0.8, weight= 120 lbs, age=21. eGFR=96 ml/min
- Patient: SCr=0.8, weight= 120 lbs, age=81. eGFR= 47 ml/min.

Clinical Pearls

- The best way to treat disease is to prevent it.
- Norman O. Moser, DO

