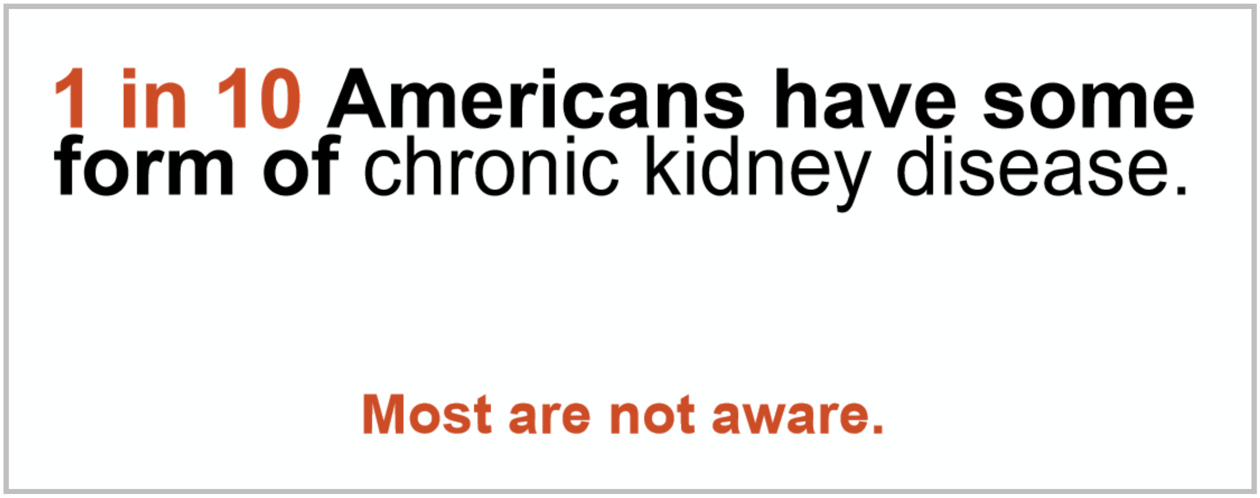
Impaired Kidney Function - Part One

Hey, everybody. In this presentation, we're going to discuss blood chemistry patterns for impaired kidney function.



According to recent statistics, one in 10 American adults, or about 26 million people, has some form of chronic kidney disease, and most aren't aware that they have it. Kidney disease is the ninth leading cause of death and kills more people each year than breast or prostate cancer, yet there is much lower awareness about kidney disease than those conditions. One in three Americans is at risk of kidney disease because high blood pressure and diabetes are the two leading causes of kidney disease, and those are both extremely common. The incidence of chronic kidney disease is highest in people over 65 years of age and is also increasing most rapidly in that age group. The incidence of chronic kidney disease in people under 65 is less than 0.5 percent, so this is primarily a disease of people over 65 years of age.

Blacks are three-and-a-half times more likely to experience kidney failure than whites, and patients with chronic kidney disease have increased morbidity and mortality, especially from cardiovascular disease. That said, the death rate from chronic kidney disease has actually declined since 2001.

1 in 10 Americans will have a kidney stone in his or her lifetime.

One in 10 Americans will have a kidney stone during his or her lifetime. Each year, more than half-a-million people visit emergency rooms for kidney stone problems. White Americans are more prone to developing kidney stones, and men are much more likely than women to develop kidney stones.

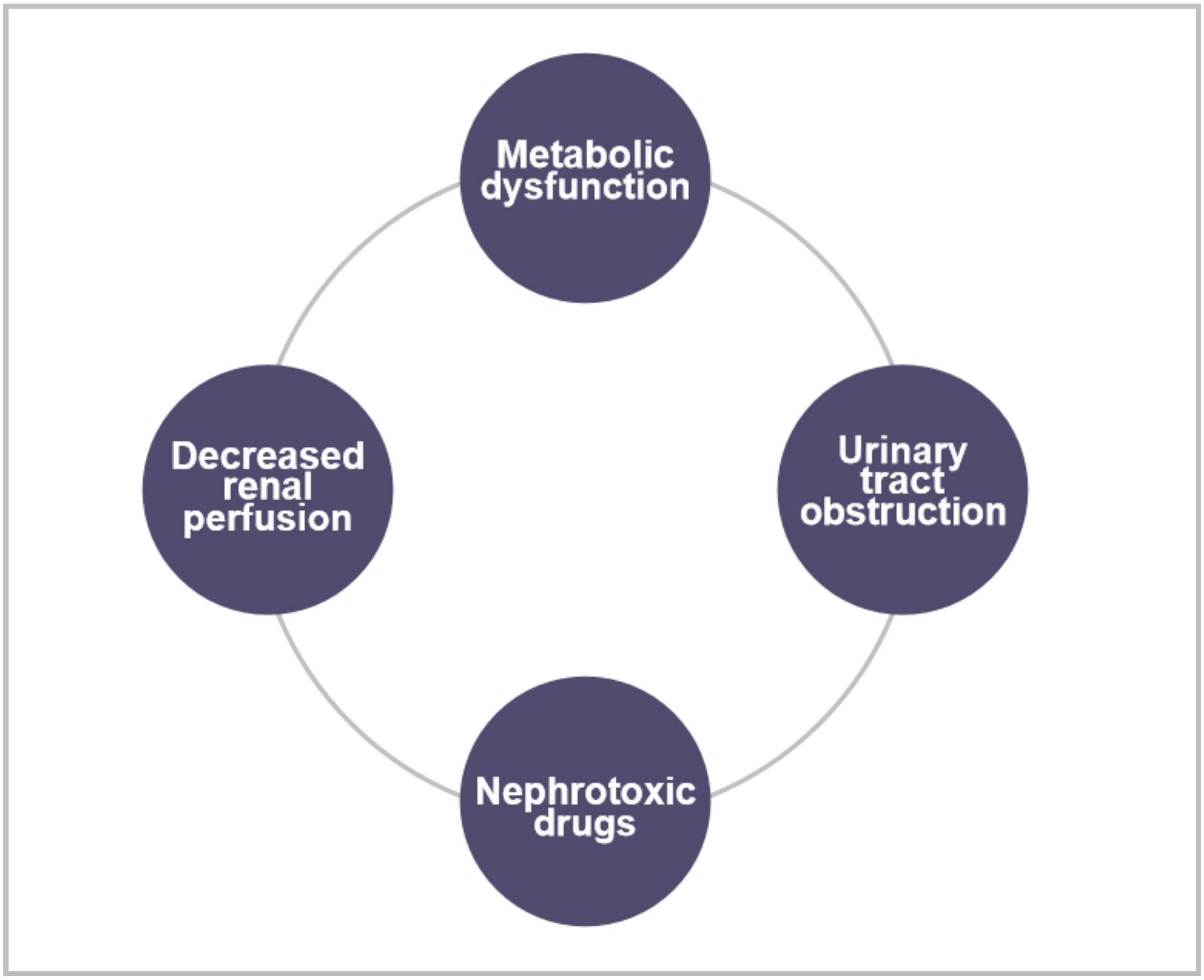
The primary job of the kidneys, as you know, is to regulate fluid levels, filter waste and toxins from the blood, release hormones that regulate blood pressure and red blood cell production, activate vitamin D to maintain healthy bones, and maintain balance of sodium, phosphorus, and potassium. Every 30 minutes the kidneys filter the body's entire blood supply, removing waste and excess fluid.

Chronic kidney disease is defined as the presence of kidney damage, which is in turn defined as urinary albumin excretion of 30 mg/kg per day or more, or decreased kidney function, which is defined as an eGFR below 60 for three or more months irrespective of the cause. Symptoms include fatigue; weakness; difficult and painful urination; foamy urine or pink and dark urine, which is indicative of hematuria; increased need to urinate, especially at night; puffy eyes; swelling of the face, hands, abdomen, ankles, and feet; and increased thirst. Complications include hyperkalemia, metabolic acidosis, and hyperphosphatemia, as well as abnormalities related to hormonal or systemic dysfunction such as anorexia, nausea, vomiting, fatigue, hypertension, anemia, malnutrition, hyperlipidemia, and bone disease.

I haven't seen a lot of patients with chronic kidney disease in my practice, though I have had a few. I've seen plenty with kidney stones. Patients with full-fledged type 2 diabetes and frank hypertension are more likely to have chronic kidney disease, and that's really not my typical patient profile.

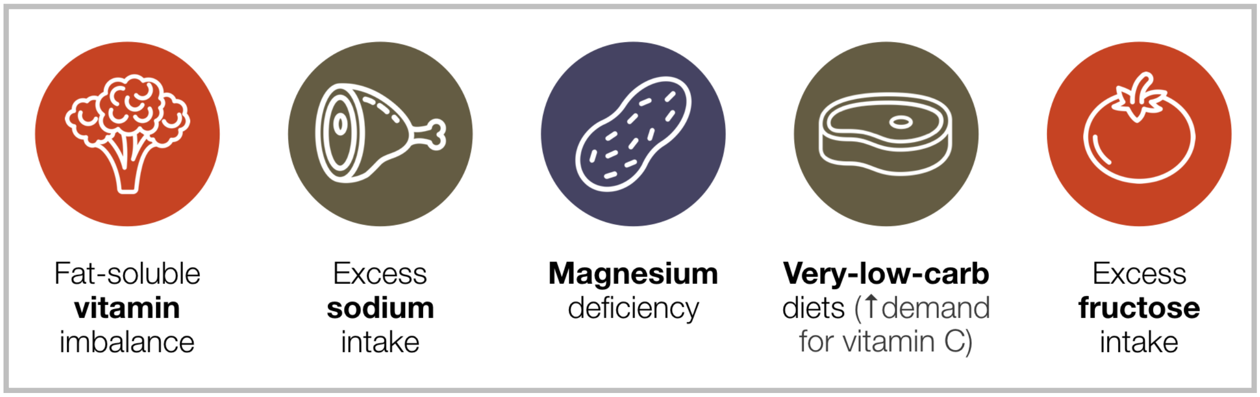
That said, as you know, dysfunction progresses along a spectrum, and we can catch impaired kidney function before it progresses to overt chronic kidney disease. The earlier you catch it, the easier it is to reverse and to prevent CKD.

As with any other condition we've talked about, the key is to address the underlying causes. Let's take a closer look at the reversible causes of CKD, and then we'll move on to kidney stones.



As I mentioned, the top two causes of CKD are diabetes and hypertension, and that's mostly what you will see and where you will need to focus. Decreased renal perfusion caused by hypovolemia, which can be caused by vomiting, diarrhea, diuretic use, etc.; hypotension; infection; or use of NSAIDs and ACE inhibitors are another potential cause. Nephrotoxic drugs include aminoglycoside antibiotics, NSAIDs, and ACE inhibitors, which I just mentioned, and the use of these should be avoided in diabetics. Urinary obstruction is a far less common cause of chronic kidney disease, especially in the absence of prostate problems. In a functional medicine setting, diabetes and hypertension, again, are really the most likely causes you'll need to address.

Kidney stones are formed from a variety of substances, but the most common stones are made of calcium oxalate, which is crystallized in the urinary tract. Other types of stones include struvite, uric acid, and cystine. The stones themselves are painful enough, but they can lead to more serious conditions such as obstruction of the urinary tract, permanent damage to the kidneys, and even life-threatening infections.



While it's not entirely clear why one person is more prone to kidney stones than another, there are several factors that are known to increase risk, which I've listed on this slide. Deficiencies or imbalances of vitamin A, D, or K2 can affect calcium metabolism and lead to stone formation. Excess vitamin D in the absence of sufficient vitamins A and K2 is a particular concern.

At least three studies have shown that magnesium can reduce the risk of stone formation. In those who are salt sensitive, excess sodium intake in the absence of adequate potassium may contribute to stone formation, although the evidence here is mixed. Dr. Paul Jaminet has written about a possible link between very-low-carb diets and kidney stones. I've observed this anecdotally in my practice, and it may, in part, be related to increased demand for vitamin C on a very-low-carb diet.

High intake of fructose may increase uric acid production and lead to an increase in uric acid stones. Finally, it's worth pointing out that a high-protein diet with adequate carbohydrate intake, contrary to popular belief, will not necessarily increase your risk of stones unless you already have a pre-existing or underlying kidney disease.

Markers for impaired kidney function

Marker	Level
BUN	High
Creatinine	High
eGFR	Low
Phosphorus	High
Sodium	High
Potassium	High
AST	High
ALT	High
GGT	High

Here are the core markers in the case review blood panel for impaired kidney function. I'm not going to go over each of these individually, but we'll cover most of them in the context of case studies, and we've already talked about several, so refer to the individual marker sheets for more detail on these particular markers.

Follow-up testing for impaired kidney function

Marker
Urinalysis with microscopy
Urine microalbumin / creatinine ratio
Ultrasound (to check for obstruction)
Cystatin-C

If you suspect the patient has kidney dysfunction, you can either refer them to a nephrologist immediately or do further workup. Follow-up testing should include urinalysis with microscopy, microalbumin-to-creatinine ratio, kidney ultrasound, and cystatin C. Proteinuria is the most common finding in kidney disease due to metabolic disorders and hypertension. If the patient has a stone obstructing the urinary tract and impairing kidney function, that should show up on the ultrasound. Cystatin C is gaining acceptance as an early sensitive marker of kidney disease, and it's especially useful in those cases where creatinine measurement is not appropriate, for example, people with obesity, liver disease, or reduced muscle mass. High cystatin C levels are indicative of reduced glomerular filtration and kidney disease.

Note that it is better to refer early than late. Late referral for CKD is common and responsible for significant morbidity and mortality that are associated with CKD. As with most other chronic diseases, the earlier you intervene, the more likely you are to have a successful outcome.

Laboratory and functional ranges for kidney markers

Marker	Lab Range	Functional Range
BUN	5–18 mg/dL	13–18 mg/dL
Creatinine	M: 0.72–1.27; W: 0.57–1.0	M: 0.85–1.1; W: 0.7–1.0
eGFR	Age/gender specific	Use lab range
Phosphorus	2.5–5.3 mg/dL	3.0–4.0 ng/dL
Sodium	134–144 nmol/L	135–140 nmol/L
Potassium	3.5–5.2 nmol/L	4.0–4.5 nmol/L
AST	0–40 IU/L	F: 0–23 IU/L; M: 0–25 IU/L
ALT	0–24 IU/L	F: 0–20 IU/L; M: 0–26 IU/L
GGT	0–60 IU/L	F: 0–21 U/L; M: 0–29 U/L

Here are the laboratory and functional ranges for the kidney markers. Note how different the ranges for AST, ALT, and GGT are. These are based on the studies I shared in the hyperglycemia presentation, and obviously, AST and ALT aren't specific to chronic kidney disease, but they may be involved in this condition. The functional ranges for phosphorus, BUN, sodium, and potassium are not based on specific studies but instead on just narrowing the lab range. This is certainly less scientific and reliable, so don't be too concerned if you see a sodium of 142 or a potassium of 4.6, for example, and all other markers are normal. That's not really enough to comprise a pattern that would be suggestive of kidney disease, especially when the elevations are just barely out of the functional range.