A PATIENT GUIDE · KIDNEY LABS

The Lab-to-Action Guide

Your numbers changed — here's exactly what to do next.
Six labs, one system, plain-language action steps.

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Common lab abnormalities, one shared 8-part template	**Colors: Red (ER), Amber (call today), Green (routine)**	**Rule: never self-adjust a prescription from a lab number**

Purpose of This Guide

Most patients receive a lab report full of arrows and colors and no instructions. This guide is organized the way a worried patient actually thinks — not by organ, but by the single number that just changed. For each of six common kidney-related abnormalities, it answers the same practical question: *"My ___ moved. What do I do, what do I eat, what do I stop, and when is it an emergency?"* Every recommendation is anchored to KDIGO, ADA, or ACC/AHA guidance and reasoned through cross-organ physiology, in keeping with the integrative nephrology approach used across the williamriveromd.com library.

Universal Safety Triage — Read This First

RED — Emergency
Chest pain, fainting, a very slow/irregular heartbeat, severe breathlessness, weakness you cannot stand through, confusion, or no urine for 12+ hours. Go to the ER now.

AMBER — Same/Next-Day Call
A single markedly abnormal value, a worsening trend, or new swelling/ breathlessness/reduced urine. Call the clinic today.

GREEN — Routine
A mildly out-of-range value you already knew about, stable, no new symptoms. Note it, apply the steps, review at your next visit.

⚠ The One Rule That Prevents Most Harm

Never stop, start, or change a prescription on your own because of a lab number. Many kidney-protective drugs (ACE inhibitors, ARBs, SGLT2 inhibitors, MRAs, diuretics) transiently nudge these numbers and are still doing their job. Bring the number to your doctor — the decision is a shared one.

How This Guide Is Built

Each of the six modules that follow uses the same eight-part template so you can flip to any lab and instantly find the same landmarks: **(1)** what the lab means in plain language, **(2)** why it matters — the cross-organ story, **(3)** common causes, **(4)** red-flag values, **(5)** what to do with food, **(6)** medication considerations, **(7)** call-the-doctor vs. go-to-the-ER, and **(8)** the guideline anchor grounding the advice. Every module also links to the deeper single-topic guides in the library for further reading.

What This Means

Potassium (K^+) is the mineral that keeps the electrical rhythm of your heart and the contraction of your muscles steady. **Hyperkalemia** means there is too much of it in the blood. Normal is roughly **3.5–5.0 mmol/L**. Your kidneys are the main exit door for potassium, so when kidney function falls, potassium tends to rise.

Why It Matters — The Cross-Organ Story

Heart first. Potassium sets the voltage across every heart cell — too much flattens that voltage, slows conduction, and can trigger a fatal arrhythmia with little warning. This is the one CKD lab that can kill within hours. **Muscle.** The same voltage problem shows up as heavy, weak legs or a "can't-get-up" feeling. **Kidney & metabolism.** Low kidney function, acidosis, high blood sugar, and the very drugs that protect the kidney (ACEi, ARBs, spironolactone, finerenone) all raise potassium — it is a crossroads number.

Common Causes

- Falling kidney function (AKI or advancing CKD) — fewer working nephrons to excrete K^+
- Kidney-protective drugs: ACEi, ARBs, spironolactone/eplerenone, finerenone
- High-potassium diet: buko/coconut water, banana/saba, oranges, salt substitutes (potassium chloride)
- Metabolic acidosis and uncontrolled diabetes shifting K^+ out of cells
- **Pseudohyperkalemia** — a falsely high value from a tight-fisted draw; repeat calmly

Red-Flag Values

Value / Sign	Band	Action
>6.5, or ANY level with palpitations, faintness, weakness	ER	*ER now — needs ECG + emergency treatment*
6.0–6.5, no symptoms	AMBER	*Call today; urgent recheck + diet/ med change*
5.1–5.9, stable, known CKD	GREEN	*Tighten diet, review meds at next visit*

Do This With Food

- Stop the biggest hitters first: buko juice, banana/saba, and salt substitutes
- **Leach vegetables:** peel, dice, soak, then boil in a large volume and discard the water
- Watch portion, not just type — a small serving of a moderate food is safer than a large one
- Avoid "healthy" traps: low-sodium salt, herbal/alkaline waters, vegetable-juice cleanses

Medication Considerations

Do not self-stop your ACEi, ARB, or MRA — these protect your kidneys and heart. Your doctor may reduce the dose, add a potassium binder (patiromer, sodium zirconium cyclosilicate), correct acidosis with bicarbonate, or adjust a diuretic — a sequence designed to let you *stay* on the protective drug. Tell your doctor about NSAIDs (mefenamic acid, ibuprofen) — they raise potassium.

Call the Doctor vs. Go to the ER

Call the Doctor (today/this week)

- A single K^+ of 5.5–6.0 with no symptoms
- Potassium creeping up over several visits
- You started a new BP/kidney drug and the number rose
- You need help adjusting diet

Go to the ER Now

- Palpitations, a slow/irregular pulse, or fainting
- Severe muscle weakness — legs give out
- K^+ ≥6.5 on a report
- Chest pain or trouble breathing

Guideline Anchor

KDIGO 2024 CKD guidance frames hyperkalemia management around *preserving* RAAS-blockade (ACEi/ARB) and MRA therapy — using dietary counseling, correcting metabolic acidosis, diuretics, and newer potassium binders to avoid discontinuing kidney-protective drugs whenever possible. Confirmed K^+ ≥6.0–6.5 mmol/L, or any level with ECG changes, is a medical emergency.

Read next (deeper guides in the library): *Potassium & Hyperkalemia in CKD · Kain Pa Rin: CKD Nutrition Guide*

What This Means

Bicarbonate (HCO_3^-) is your blood's built-in antacid — the buffer that keeps you from turning too acidic. A low value (normal ≈ **22–29 mmol/L**) means **metabolic acidosis**: acid is accumulating because the kidneys can no longer make and reclaim enough bicarbonate. It is the quiet lab — usually no symptoms, real long-term harm.

Why It Matters — The Cross-Organ Story

Bone. The body buffers extra acid by dissolving bone, releasing calcium and phosphate — accelerating CKD-mineral-bone disease and fractures. **Muscle.** Chronic acidosis switches on protein breakdown, a hidden driver of frailty in CKD. **Kidney.** Acid is itself toxic to the tubules; low bicarbonate independently speeds CKD progression — correcting it can slow the decline. **Heart/metabolism.** Acidosis worsens insulin resistance and potassium handling.

Common Causes

- Reduced kidney acid-excretion as CKD advances (the dominant cause)
- Diets very high in animal protein with few fruits and vegetables
- Diarrhea or GI bicarbonate loss
- Certain drugs (acetazolamide, topiramate); uncontrolled diabetes (ketoacidosis — a separate emergency)

Red-Flag Values

Value / Sign	Band	Action
<12, OR fast/deep breathing, drowsiness, vomiting	ER	*ER now — possible DKA/lactic acidosis*
12–17, or a clear downward trend	AMBER	*Call this week; likely start oral bicarbonate*
18–21, stable	GREEN	*Diet shift + recheck at next visit*

Do This With Food

- **Add base-forming plants:** more vegetables and CKD-appropriate fruit (balanced against potassium)
- Moderate (do not eliminate) animal protein to your doctor's target
- If prescribed sodium bicarbonate tablets, take as directed and report new swelling/breathlessness

Medication Considerations

Oral alkali therapy — sodium bicarbonate, or newer veverimer-class agents where available — is the mainstay when bicarbonate is persistently below ~18 mmol/L. The goal is low-normal, not overshoot. Correcting acidosis often improves potassium and slows kidney decline at the same time.

Call the Doctor vs. Go to the ER

Call the Doctor (today/this week)

- Bicarbonate below 18 on a routine report
- A steady downward trend across visits
- You feel more tired/weaker than usual
- You were told to start bicarbonate and have questions

Go to the ER Now

- Fast, deep, labored breathing
- Drowsiness, confusion, or vomiting
- Known diabetic with high sugars + these symptoms (possible DKA)
- Bicarbonate <12

Guideline Anchor

KDIGO CKD guidance recommends oral alkali to maintain serum bicarbonate in the normal range when it falls persistently below ~22 mmol/L, because correcting metabolic acidosis slows CKD progression and reduces muscle and bone breakdown. Very low bicarbonate with symptoms is an emergency.

Read next (deeper guides in the library): *Metabolic Acidosis in CKD · CKD-MBD*

What This Means

ACR (albumin-to-creatinine ratio) measures how much albumin — a blood protein — is leaking into your urine. Categories: **A1 (<30 mg/g)** normal, **A2 (30–300)** moderately increased, **A3 (>300)** severely increased. A worsening ACR is one of the earliest and most powerful warnings the kidney gives.

Why It Matters — The Cross-Organ Story

Kidney. Albuminuria is not just a marker — leaked protein inflames and scars the tubules, so more leak causes more damage in a self-feeding loop. **Heart/vessels.** Even A2-level albuminuria independently raises the risk of heart attack and stroke — the kidney is a window on the whole vascular tree. **Metabolism.** In diabetes, rising ACR is the footprint of glucose-driven filter injury; it often moves before creatinine does.

Common Causes

- Diabetes and high blood pressure — the two leading drivers worldwide
- Under-treated BP or blood sugar; missed RAAS-blocker doses
- Glomerular disease (e.g., IgA nephropathy, lupus) — especially if ACR jumps sharply
- **Reversible bumps:** fever, heavy exercise, UTI, or dehydration — recheck first-morning sample

Red-Flag Values

Value / Sign	Band	Action
Sharp rise + visible blood, heavy swelling, breathlessness	ER	*Urgent call today; ER if breathless or swelling fast*
Moved A1→A2 or A2→A3, or rising	AMBER	*Call this week — treatment usually intensifies*
Stable A2 on treatment, no new symptoms	GREEN	*Confirm with morning sample; review at next visit*

Do This With Food

- **Sodium is the lever:** cut to ~5 g/day (2 g sodium) — drop instant noodles, processed meats, patis, toyo, bagoong
- Moderate protein to your prescribed target; very high protein raises filter pressure
- Get blood sugar and weight into range — each point of control shows up as lower ACR

Medication Considerations

This is the number our best drugs are built to lower. Expect your doctor to start or maximize an **ACEi or ARB**, add an **SGLT2 inhibitor**, and — in diabetic kidney disease with persistent albuminuria — consider **finerenone**. A rise in creatinine of up to ~30% after starting these is expected and acceptable, not a reason to stop.

Call the Doctor vs. Go to the ER

Call the Doctor (today/this week)

- ACR moved up a category (A1→A2, A2→A3)
- Bubbly urine or a steady upward trend
- Blood pressure or sugar running high
- You want to start/adjust an SGLT2i or ARB

Go to the ER Now

- Sudden heavy body swelling with breathlessness
- Visible blood in urine with reduced urine output
- Severe headache with very high BP
- Signs of a clot (leg swelling + chest pain/breathlessness)

Guideline Anchor

KDIGO 2024 and ADA Standards of Care both treat albuminuria as a primary treatment target: confirm with a first-morning ACR, control BP and glucose, and use RAAS blockade plus an SGLT2 inhibitor — adding finerenone in diabetic kidney disease with residual albuminuria. Albuminuria and eGFR together define CKD risk.

Read next (deeper guides in the library): *Proteins & Proteinuria · Diabetes & Your Kidneys*

What This Means

Hemoglobin (Hgb) is the protein in red blood cells that carries oxygen. In kidney disease, damaged kidneys make less **erythropoietin (EPO)** — the hormone that tells the bone marrow to build red cells — and handle iron poorly. Rough anemia threshold: Hgb <**13 g/dL (men)** / <**12 g/dL (women)**.

Why It Matters — The Cross-Organ Story

Heart. To deliver enough oxygen with thin blood, the heart pumps harder and enlarges (left ventricular hypertrophy) — a direct path to heart failure. **Brain/whole body.** Fatigue, breathlessness, poor concentration, cold intolerance, and low mood are the lived symptoms. **Iron/metabolism.** CKD inflammation traps iron in storage (via hepcidin), so the marrow can be "starved amid plenty" — which is why we read iron studies, not just hemoglobin.

Common Causes

- EPO deficiency from reduced kidney function (the signature cause in CKD)
- Iron deficiency — absolute (true low stores) or functional (locked-away iron)
- Blood loss (GI bleeding, heavy menses, frequent draws, dialysis losses)
- B12/folate deficiency, chronic inflammation or infection, marrow suppression

Red-Flag Values

Value / Sign	Band	Action
<7, OR chest pain/severe breathlessness/fainting, OR black/bloody stool	ER	*ER now — may need transfusion / bleeding workup*
7–10, or a steady fall across visits	AMBER	*Call this week — iron studies + treatment plan*
10–11.5, stable, on treatment	GREEN	*Continue plan; recheck as scheduled*

Do This With Food

- **Iron-first, within kidney limits:** lean red meat, eggs, fish; pair plant iron (malunggay, beans) with vitamin C
- Separate iron from coffee/tea and from calcium/phosphate binders — they block absorption
- Diet supports but rarely fixes CKD anemia alone — food is the floor, not the ceiling

Medication Considerations

Iron before EPO. The guideline order is to repair iron stores first (oral, or often more effective in CKD, IV iron), then add an **ESA** or oral **HIF-PHI** if hemoglobin stays low. Target is a *moderate* hemoglobin (~10–11.5 g/dL) — deliberately not "normal," because chasing higher levels with ESAs raised stroke and clot risk in trials.

Call the Doctor vs. Go to the ER

Call the Doctor (today/this week)

- Hemoglobin falling on routine labs
- New fatigue, breathlessness on exertion, palpitations
- You want your iron studies explained
- Menstrual or GI blood loss to investigate

Go to the ER Now

- Chest pain, fainting, or breathlessness at rest
- Black, tarry, or bloody stool; vomiting blood
- Very pale, rapid heartbeat, cold and clammy
- Hemoglobin <7 on a report

Guideline Anchor

KDIGO anemia guidance recommends evaluating and correcting iron status first, then using ESAs (or HIF-PHIs) to avoid transfusion while individualizing therapy — generally not targeting Hgb >11.5 g/dL, because higher targets increased cardiovascular and thromboembolic harm. Always exclude bleeding first.

Read next (deeper guides in the library): *Anemia in Kidney Disease · Iron: Fueling Every Red Cell*

What This Means

Phosphorus (phosphate) is a mineral in bone and every cell. Healthy kidneys dump the daily excess in urine; as kidney function falls, phosphorus builds up (**hyperphosphatemia**; normal ≈ **2.5–4.5 mg/dL**). You feel nothing — but it silently drives the bone-and-vessel disease of CKD.

Why It Matters — The Cross-Organ Story

Vessels/heart. Excess phosphate turns arterial muscle cells into bone-like cells — literally calcifying blood vessels and heart valves, a leading reason CKD patients die of heart disease. **Bone (CKD-MBD axis).** High phosphate pulls down calcium and vitamin D and drives up **PTH**, which strips calcium from bone. **Hormonal amplifier.** Phosphate raises FGF-23 early — now linked to heart enlargement — so harm begins before phosphorus itself looks alarming.

Common Causes

- Reduced kidney excretion in advancing CKD (the root cause)
- **Hidden phosphate additives** in processed food: softdrinks, hotdog, longganisa, ham, instant noodles, cheese
- High natural-phosphorus foods: dairy, nuts, organ meats, egg yolk, dilis in quantity
- Missed or mistimed phosphate binders; very high vitamin D dosing

Red-Flag Values

Value / Sign	Band	Action
Very high WITH numbness/ tingling, cramps or spasms	ER	*Urgent call/ER if cramps-spasms or seizure*
Persistently >5.5, or a clear upward trend	AMBER	*Call this week — binder/diet/PTH review*
Mildly high, stable on binders	GREEN	*Reinforce diet + binder timing; recheck*

Do This With Food

- **Read labels for "PHOS":** avoiding phosphate additives gives the biggest, fastest drop — cut cola, processed meats, instant noodles first
- Choose fresh over processed; boil where practical (some phosphorus leaches into cooking water)
- Keep enough protein for nutrition, but favor lower-phosphate sources your dietitian identifies

Medication Considerations

Phosphate binders (calcium acetate, sevelamer, lanthanum) work only if taken **with the first bites of a meal** — on an empty stomach they do nothing. Your doctor tunes the binder, may adjust active vitamin D, and may add a calcimimetic to control PTH — all parts of one CKD-MBD strategy.

Call the Doctor vs. Go to the ER

Call the Doctor (today/this week)

- Phosphorus above target on labs
- Trend rising over months
- You are unsure how/when to take binders
- Itching or bone/joint aches

Go to the ER Now

- Numbness/tingling around the mouth or in hands
- Muscle cramps, spasms, or twitching (tetany)
- A seizure
- Severe chest pain (calcified-vessel event)

Guideline Anchor

KDIGO CKD-MBD guidance advises lowering elevated phosphate toward the normal range, prioritizing restriction of dietary phosphate additives, using binders with meals, and managing calcium, vitamin D, and PTH together — because vascular calcification and renal bone disease share this axis.

Read next (deeper guides in the library): *Phosphorus: The Silent Threat in CKD · CKD-MBD*

What This Means

Creatinine is a waste product your kidneys clear at a steady rate, so it mirrors filtering power. When creatinine **rises**, the estimated filtration rate (**eGFR**) **falls**. A slow drift is CKD progression; a sudden jump may be **acute kidney injury (AKI)** layered on top of CKD — often reversible if caught fast.

Why It Matters — The Cross-Organ Story

Everything downstream. A falling eGFR is the master dial: it worsens potassium, acidosis, anemia, and phosphorus all at once — a creatinine jump is often the first domino. **Heart/fluid.** Less filtering means fluid and toxins accumulate — swelling, breathlessness, higher blood pressure. **Reversibility.** The single most useful question is *sudden or slow?* A sudden rise points to dehydration, a drug, or an obstruction — problems we can often fix.

Common Causes

- **Pre-renal:** dehydration, vomiting/diarrhea, over-diuresis, low blood pressure — the most common and most reversible
- **Drugs/toxins:** NSAIDs, IV contrast dye, certain antibiotics, herbal/"natural" nephrotoxins
- **Post-renal:** kidney stones, an enlarged prostate — reduced urine stream
- Expected transient rise (up to ~30%) after starting ACEi/ARB or SGLT2i — the drug working, not failing
- True CKD progression — a gradual, sustained decline over months

Red-Flag Values

Value / Sign	Band	Action
No urine 12+ h, heavy swelling + breathless, confusion, or K^+/ acid emergency signs	ER	*ER now — possible severe AKI*
Jumped clearly above baseline (>25–30%) over days–weeks	AMBER	*Call today — find & fix the cause, recheck*
Small stable change near baseline, no symptoms	GREEN	*Confirm hydration; review trend at next visit*

Do This With Food

- **Fluid is first-line if dehydrated** — but if swollen/breathless/heart failure, do NOT load fluids; call first
- Avoid protein loading and "kidney-cleanse" herbal products — several are directly nephrotoxic
- Keep sodium controlled to manage the blood pressure and swelling that accompany a falling eGFR

Medication Considerations

"Sick-day" thinking. When dehydrated, vomiting, or with diarrhea, ask about temporarily holding **NSAIDs, ACEi/ARBs, SGLT2 inhibitors, diuretics, and metformin** during acute illness — and exactly when to restart. **Stop NSAIDs now** if your creatinine is up. Never stop a chronic kidney/heart drug without guidance once you are well.

Call the Doctor vs. Go to the ER

Call the Doctor (today/this week)

- Creatinine higher than your usual baseline
- Recent vomiting/diarrhea or new NSAID use
- You just started an ACEi/ARB/SGLT2i and it rose
- Reduced urine but still going, no severe symptoms

Go to the ER Now

- Little or no urine for 12+ hours
- Heavy swelling with breathlessness, or can't lie flat
- Confusion, severe weakness, or an irregular pulse
- Severe flank pain with no urine (possible blockage)

Guideline Anchor

KDIGO defines AKI by a rise in creatinine (≥0.3 mg/dL within 48h or ≥1.5× baseline within 7 days) or reduced urine output. Management centers on finding the cause, restoring perfusion, and stopping nephrotoxins — while a modest, expected creatinine rise after starting RAAS blockade or an SGLT2 inhibitor is not a reason to discontinue kidney-protective therapy. Always interpret against the patient's own baseline.

Read next (deeper guides in the library): *Taking Charge of CKD · Understanding Your Lab Results*

These six labs are not six problems but one system. Falling eGFR is the hub; the other five are spokes.

Lab	One-Line Meaning	Fastest Patient Action	The Drug Conversation
High K^+	*Heart-rhythm risk*	*Stop buko/saba & salt-substitute*	*Keep RAASi + add binder, don't self-stop*
Low HCO_3^-	*Acid building up*	*More veg, less excess meat*	*Start oral bicarbonate*
Rising ACR	*Filter leaking*	*Cut salt to ~5 g/day*	*Maximize ACEi/ARB + SGLT2i (±finerenone)*
Low Hgb	*Oxygen delivery down*	*Iron-rich food, treat bleeding*	*Iron first, then ESA/HIF-PHI to ~10–11.5*
High PO_4	*Vessel/bone calcification*	*Cut phosphate additives (cola, processed meat)*	*Binders WITH meals; manage PTH/Vit D*
High Creatinine	*Filtering falling*	*Hydrate (unless swollen); stop NSAIDs*	*Sick-day hold of nephrotoxins; find cause*

The Integrative Takeaway

Fix perfusion and stop nephrotoxins, and creatinine steadies. Control salt and sugar, and ACR falls. Correct acidosis, and potassium and bone improve together. Restore iron, and the heart is spared. One well-chosen action often moves several numbers at once — which is why we treat the patient and the system, not the isolated arrow on the page.

Medical Disclaimer

This guide is educational and does not replace individualized medical advice. The values and thresholds here are general reference points; your personal targets depend on your CKD stage, diagnosis, other conditions, and medications. Never start, stop, or change any prescription based on a lab number alone. Always consult Dr. Rivero or your physician. When in doubt about an emergency sign, err toward the ER.

For educational use only. References: Levin et al., Kidney Int 2024 (KDIGO CKD) · Kreitzer et al., Cardiorenal Med 2025 (hyperkalemia) · Siddiqui et al., World J Nephrol 2025 (bicarbonate) · Rossing et al., Kidney Int 2022 (KDIGO Diabetes-CKD) · Babitt et al., Kidney Int 2026 (KDIGO anemia) · Ketteler et al., Kidney Int 2017 (KDIGO CKD-MBD) · Ostermann et al., Kidney Int 2020 (KDIGO AKI).