Approach to Hyperkalemia

RENAL & VOLUME STATUS

13% — KEY DECISION RULE
IV insulin for hyperkalemia[5]
of insulin doses in dialysis inpatients caused glucose <3.3 mmol/L

Serum potassium above the upper limit of normal → STAT 12-lead ECG and monitor; creatinine, glucose, gas, CBC; drug review[1,10] → Hemolyzed or unexpected with normal ECG: repeat K on a blood gas before treating[1,10] → **ECG change, K ≥6.0, or rising fast?**

Yes →

Emergency: stabilize, shift, then remove K[1,10]

- ECG: peaked T, long PR, wide QRS, brady
- ECG change: Ca gluconate 10% 30 mL IV[1]
- K ≥6.5: shift K even if ECG normal[1,3]
- K 6.0–6.4: monitor, binder, recheck K[1,10]
- After insulin: K in 1–2 h; glucose ≥3 h[5]

→ **Shift: insulin 5–10 U + dextrose 25–50 g; salbutamol 10–20 mg[1,4,6]**

Then: why is K high?

	Low GFR or distal flow commonest	**Low aldosterone effect** drugs	**Shift out of cells** redistribution	**Pseudohyperkalemia** not real
CAUSES	• **AKI, especially oliguric** • Advanced CKD; missed dialysis • Low distal flow: HF, hypovolemia[9]	• ACEi/ARB, MRA, K-sparing diuretic • NSAIDs, trimethoprim, heparin, CNIs • **Adrenal insufficiency: low Na, low BP**	• **Rhabdomyolysis, tumour lysis** • Insulin deficiency: DKA, HHS • Acidosis; digoxin, β-blockers	• Hemolysis; fist clenching, tourniquet[1] • Very high platelets or WBC[10] • Drawn above a K-containing IV
CLUES	• Low urine output; rising creatinine • Edema, or dry and orthostatic	• New drug or dose, with low eGFR[1,2] • Diabetic CKD, non-gap acidosis: type 4 RTA	• Crush, found down, recent chemo • High glucose; anion-gap acidosis	• Hemolysis flagged; no risk factors[1] • Implausible: no CKD, culprit drug or lysis[1]
WORKUP	• Creatinine, urea; bladder scan • Renal ultrasound if AKI is new	• Medication and supplement review[1] • Renin, aldosterone if unclear • Cortisol, ACTH if adrenal suspected[10]	• CK, urate, phosphate, LDH • Blood gas; digoxin level if on it	• Repeat without tourniquet; gas K[1,10] • Platelets, WBC on the CBC[10]
MANAGE	• Dialysis if oliguric, ESKD or refractory[1] • Making urine: loop diuretic[1] • Volume-depleted: isotonic fluid first[1]	• Hold culprit; restart RASi ± binder later[11] • Binder: SZC 10 g TID ≤48 h; SPS GI risk[1,7,8] • Adrenal crisis: IV hydrocortisone 100 mg[10]	• Treat cause: DKA insulin; digoxin Fab[12] • Bicarbonate only if acidemic[1] • Serial K: lysis keeps releasing it	• No treatment once repeat K is normal

Stethopedia
stethopedia.ca/approach/hyperkalemia

Evidence [1] Lindner 2020, Eur J Emerg Med [2] Clase 2020, Kidney Int [3] Montague 2008, Clin J Am Soc Nephrol [4] Harel 2016, PLoS One [5] Apel 2014, Clin Kidney J [6] Allon 1989, Ann Intern Med [7] Noel 2019, JAMA Intern Med [8] Kosiborod 2014, JAMA [9] Gumz 2015, N Engl J Med [10] Manual 2024–25, pp. 65, 103, 105, 107, 182 [11] KDIGO 2024, Kidney Int (CKD) [12] Chan 2014, Clin Toxicol