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A Physiologic-Based Approach to the Treatment of Acute Hyperkalemia

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Abstract

Hyperkalemia is a common and potentially lethal disorder. Given its variable presentation clinicians should have a high index of suspicion, especially in patients with chronic kidney disease. The present case highlights key physiological mechanisms in the development of hyperkalemia and provides an outline for emergent treatment. In this context, we discuss specific mechanisms of action of available treatments of hyperkalemia.

Keywords

hyperkalemia; treatment; electrolyte abnormalities

Introduction

Hyperkalemia is a common electrolyte disorder encountered by nephrologists, intensivists, and emergency room physicians. While often manifesting with nonspecific symptoms, it can occasionally lead to life-threatening cardiac rhythm disturbances. Prompt recognition, patient risk stratification, and administration of appropriate treatment can prevent the serious complications of hyperkalemia.

Case Report

Clinical History and Initial Laboratory Data

A 72-year-old woman with chronic kidney disease (CKD) stage 3 (baseline serum creatinine of 1.5 mg/dL [133 μ mol/L], corresponding to an estimated glomerular filtration rate of 35 ml/min/1.73 m² [0.58 mL/s/1.73 m²; calculated by using the *** equation]) presented to the

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Note from Feature Editor Jeffrey A. Kraut, MD: This article is part of a series of invited case discussions highlighting either the diagnosis or treatment of acid-base and electrolyte disorders. The present case discussion is the second of 2 articles describing a physiologic-based approach to the diagnosis or treatment of acute hyperkalemia. In this article, Drs Shingarev and Allon present their approach to the treatment of acute hyperkalemia; in the first teaching case Dr Palmer described his approach to the treatment of acute hyperkalemia.^{1A}

emergency room with a one-day history of fatigue, generalized weakness, and inability to stand. She had been taking a stable dose of lisinopril over the past six years for management of hypertension. Three days previously, she had been prescribed trimethoprim-sulfamethoxazole for a urinary tract infection.

On physical examination, her blood pressure was 98/50 mmHg supine and 87/40 mm Hg sitting, and her heart rate was 112 beats per minute and regular. She was alert and oriented. Neurologic examination revealed normal reflexes and cranial nerve examination. She had symmetric weakness of all four limbs, 3/5 in the lower extremities and 4/5 in the upper extremities. She was unable to stand.

Relevant laboratory data are summarized in Table 1. Her initial electrocardiogram revealed sinus tachycardia with prolonged PR and QRS intervals and peaked T-waves (Figure 1a).

Diagnosis

Acute symptomatic hyperkalemia.

Clinical Follow-up

The patient received conservative medical treatment in the form of immediately administered 4.65 mEq of calcium gluconate intravenously, with her electrocardiogram normalizing within ten minutes (Figure 1b). She subsequently received 20 mg of nebulized albuterol (5 mg/ml) and an intravenous bolus of 10 units of regular insulin with 25 grams of dextrose. The repeat serum potassium was 5.1 mEq/L (5.1 mmol/L). Her motor weakness resolved once the hyperkalemia had been corrected. She was then appropriately hospitalized for monitoring of serum potassium and, possibly, repeat treatment with the same agents, sodium polystyrene sulfonate, and volume re-expansion. Hemodialysis was not indicated because of successful correction of potassium level with the methods described and because she had residual kidney function.

Discussion

To begin with a brief overview of potassium homeostasis, potassium is the second most abundant cation in the body. Only 2% of total body potassium is located in the extracellular fluid compartment. Therefore, an ingestion of just 42 mEq (42 mmol) of potassium by a 70 kg man (extracellular fluid volume ~14 L) would result in an acute 3 mEq/L (3 mmol/L) increase in serum potassium, if all the potassium were retained in the extracellular fluid compartment (1). Conversely, removal of a relatively small amount of potassium results in a substantial decrease in serum potassium. Serum potassium concentration is maintained within a narrow range (3.5 to 5.0 mEq/L [3.5 to 5.0 mmol/L]) by rapid potassium shifts between the extracellular and intracellular fluid compartments (extrarenal potassium disposal). The adenosine triphosphatase sodium-potassium pump ($\text{Na}^+\text{-K}^+\text{-ATPase}$) in the cell membrane of skeletal muscle rapidly transports potassium into the cells, and is stimulated synergistically by insulin and by β_2 -adrenergic agonists. Long-term potassium homeostasis is regulated by adjustments of urinary potassium excretion in response to fluctuations in dietary potassium intake (2).

Hyperkalemia due to potassium shifts into the extracellular fluid compartment may be caused by several physiologic factors. In inorganic metabolic acidosis (but not organic acidosis), movement of hydrogen ions into the cell causes a reciprocal potassium shift out of the cells to maintain electroneutrality (2). Stimulation of β_2 -adrenergic receptors causes intracellular shifts of potassium, whereas non-selective β -blockade causes hyperkalemia (3). Insulin promotes intracellular potassium shifts by stimulating $\text{Na}^+\text{-K}^+\text{-ATPase}$, whereas insulin deficiency causes hyperkalemia (4). In patients with normal kidney function, the rise in serum potassium resulting from extracellular shifts is blunted by a concurrent increase in urinary potassium excretion. Patients with acute kidney disease or CKD are at greater risk of hyperkalemia due to their limited ability to excrete potassium. Hyperkalemia due to insulin deficiency may also be exacerbated in patients with decreased kidney function, as the hyperosmolar state resulting from hyperglycemia cannot be alleviated by osmotic diuresis, and therefore promotes additional potassium shift out of the cells (solvent drag) (2). Finally, excessive cellular breakdown, which occurs in rhabdomyolysis or tumor lysis syndrome, leads to a rapid release of potassium from damaged cells and severe hyperkalemia (2).

In the setting of CKD, a compensatory increase in serum aldosterone permits maintenance of potassium homeostasis by increasing potassium secretion in each of the remaining nephrons. Thus, hypoaldosteronism contributes to decreased urinary potassium excretion and hyperkalemia in patients with CKD (2). Decreased aldosterone level may result from adrenal insufficiency, hyporeninemic hypoaldosteronism, or from a variety of drugs that interfere with the renin-angiotensin-aldosterone axis, including angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, nonsteroidal anti-inflammatory drugs, COX-2 (cyclooxygenase 2) inhibitors, heparin, and spironolactone (Table 2). Triamterene (a potassium-sparing diuretic) and trimethoprim both decrease urinary potassium excretion by inhibiting the sodium channel in the collecting duct (5). Finally, kidney failure is associated with a depressed $\text{Na}^+\text{-K}^+\text{-ATPase}$ activity, which diminishes distal sodium delivery and reabsorption leading to smaller electronegative potential across the apical basement membrane (6).

Acute potassium ingestion rarely leads to severe hyperkalemia, unless the patient has concurrent CKD or is ingesting drugs that interfere with urinary potassium excretion or intracellular potassium shifts (1).

The etiology of hyperkalemia was multi-factorial in the present patient, as is usually the case with this electrolyte abnormality. The patient developed a bacterial infection accompanied by volume depletion, which led to acute kidney injury superimposed on her CKD. Her angiotensin-converting enzyme inhibitor prevented the angiotensin II-dependent compensatory increase in serum aldosterone typically seen in normal patients challenged by potassium load (7). Finally, the addition of the trimethoprim component of her antibiotic further limited urinary potassium excretion by its amiloride-like effect on the sodium channels in the collecting duct. Each of these factors alone was not sufficient to produce significant hyperkalemia, but cumulatively they resulted in life-threatening hyperkalemia.

The therapeutic approach to hyperkalemia is generally based on the absolute value of serum potassium concentration and specific electrocardiographic (ECG) changes. However, the

specific clinical context may affect the treatment strategy. For example, a clinician will be more likely to immediately dialyze a patient with end-stage renal disease who has a functioning access; a hyperglycemic patient may need insulin without glucose to correct hyperkalemia.

Before proceeding with treatment of hyperkalemia in the absence of ECG changes, pseudohyperkalemia should be considered. This phenomenon is usually due to hemolysis induced by venipuncture, excessive blood sample clotting, hereditary spherocytosis, or familial hyperkalemia (8). In the latter two conditions, potassium movement into the extracellular space is induced by decreased ambient temperature. Pseudohyperkalemia can be demonstrated by finding a lower potassium concentration in plasma than in serum (8).

While the electrophysiologic effect of potassium on the membranes of excitable cells is proportional to its extracellular concentration, there is no specific cut-off level at which emergent treatment should be implemented, in part because of the body's ability to adjust its handling of potassium depending on the duration of exposure to excessive potassium load (9). On the other hand, ECG changes reflective of cell membrane depolarization cannot serve as a reliable indicator of hyperkalemia severity, as up to 60% of patients with serum potassium level greater than 6.0 mEq/L (6.0 mmol/L) present without any ECG abnormalities (10). Moreover, severe ECG disturbances, such as ventricular fibrillation, may occasionally occur without antecedent T-wave peaking or QRS prolongation (11). In patients with typical hyperkalemic ECG findings, emergent therapy should be initiated without waiting for biochemical confirmation. Therapy should also be initiated in patients whose serum potassium exceeds 6.5 mEq/L (6.5 mmol/L), even in the absence of ECG changes, because of the significant risk of rapid development of such changes (12). The recommended sequence of interventions depicted in Figure 2 starts with measures to stabilize the myocardium, followed by therapies to shift potassium into the cells, and finally, definitive treatment to remove potassium from the body (Table 3).

Calcium administration is an emergent treatment modality directed at restoration of the transmembrane electrical gradient of the cardiac myocytes. It probably achieves this goal by reducing the cells' resting membrane potential, although the precise mechanism is poorly understood (13). Intravenous calcium starts acting within 5 minutes, but its benefit is relatively short-lived, mandating continuous ECG monitoring. Repeated doses of calcium are not recommended for patients taking digoxin, because of the risk of arrhythmias (14). Calcium is available in two formulations: calcium gluconate and calcium chloride. Calcium gluconate is preferred, because of its lower risk of tissue necrosis in case of tissue extravasation, allowing it to be safely administered into a peripheral vein. In contrast, calcium chloride contains a higher concentration of calcium, necessitating its administration via a central line (14). The beneficial ECG effects of calcium are not accompanied by a reduction in serum potassium. For this reason calcium administration should be followed closely by treatments to shift potassium into the cells and remove it from the body.

Insulin treats hyperkalemia by activating the skeletal muscle $\text{Na}^+\text{-K}^+\text{-ATPase}$, thereby leading to intracellular potassium shift (2). A hyperinsulinemic state may be achieved either by administering exogenous insulin with dextrose (mandatory in diabetic patients) or with

dextrose alone (in non-diabetic patients), which stimulates endogenous insulin secretion. However, the hypokalemic effect is greater with the combination of insulin and dextrose, as compared to dextrose alone (15). A 10 unit bolus of regular insulin has an onset of action within 15 minutes, lowers serum potassium concentration by up to 1.5 mEq/L (1.5 mmol/L) in 30 to 60 minutes, and lasts for 3 to 4 hours (16). Unless a patient is hyperglycemic because of underlying diabetes mellitus, concurrent administration of dextrose as a bolus followed by a continuous infusion is required to prevent iatrogenic hypoglycemia.

The class representative of β_2 -adrenergic agonists, albuterol, induces intracellular potassium movement by up-regulating the activity of $\text{Na}^+\text{-K}^+\text{-ATPase}$ in skeletal muscle by a cellular pathway distinct from that caused by insulin (2). Thus, the potassium-lowering effects of insulin and β_2 -adrenergic agonists are additive. A 0.5 mg intravenous bolus of albuterol (available in Europe) rapidly lowers serum potassium (17). Albuterol is available only in the nebulized form in the US, and requires a dose of 20 mg administered over 10 minutes due to its limited systemic absorption from the lungs. This dose of nebulized albuterol lowers serum potassium concentration by ~ 1.0 mEq/L (~ 1.0 mmol/L) after 60 to 90 minutes and lasts for 3 to 4 hours (16). Of note, this dose (20 mg) is 4- to 8-fold higher than the typical dose of albuterol used to treat bronchospasm. Side effects of this dose of nebulized albuterol are usually limited to mild tachycardia, making it a safe treatment modality for hyperkalemia (16). However, albuterol should always be administered in conjunction with other potassium-lowering therapies because of patients' variable hypokalemic response to β_2 -adrenergic agonists. Furthermore, albuterol will have little effect on hyperkalemia in patients treated with non-selective β -blockers. Subcutaneous terbutaline is also effective in the acute treatment of hyperkalemia (18).

Theoretically, administration of sodium bicarbonate should decrease the concentration of H^+ in the extracellular fluid compartment. High extracellular Na^+ concentration leads to its increased cellular uptake via the sodium-proton exchanger (assuming it is in the active mode, as in the presence of intracellular acidosis). The subsequent movement of Na^+ out of the cell creates an electric gradient, forcing K^+ into the cell via $\text{Na}^+\text{-K}^+\text{-ATPase}$, resulting in net increase in intracellular K^+ (2). However, several studies using bicarbonate as a monotherapy in hemodialysis patients failed to show a significant reduction in extracellular potassium concentration (19,20). There are contradictory observations about whether bicarbonate administration potentiates the hypokalemic effect of insulin (20,21). In a small series of acidemic patients, bicarbonate therapy potentiated the potassium-lowering effect of insulin (21), but this finding did not generalize to non-acidemic patients in a second study (20). Based on this evidence, sodium bicarbonate is not effective for the treatment of acute hyperkalemia, although it may be useful in a subset of patients with significant metabolic acidosis. Hypertonic bicarbonate administration may lower serum potassium modestly by a dilutional effect (2), and it may be useful in volume depleted and hyponatremic patients. However, by the same token, the inherent risks of this therapy are hypernatremia and volume overload. In some patients, hypertonic bicarbonate (2000 mOsm/L) can result in a rapid efflux of potassium-rich intracellular fluid into the extracellular fluid (solvent drag), thereby increasing serum potassium.

To enhance urinary potassium excretion, loop diuretics can be used in patients with residual kidney function. Increasing sodium delivery to the distal tubule creates an electronegative gradient that favors potassium secretion into the tubule (2). When given intravenously to a patient without volume depletion, furosemide significantly lowers serum potassium concentration within 2 to 4 hours (14). The efficacy of diuretics diminishes substantially in patients with severe kidney failure, and the risk of hypotension may outweigh the potential benefits of minimal kaliuresis. Thiazides and acetazolamide can be used in combination with loop diuretics, but the same limitations to treatment application make these drugs even less useful in treatment of acute hyperkalemia (14,22).

Sodium polystyrene sulfonate resin has been used as treatment of hyperkalemia since the 1960s. Given orally (30 gm) or per rectum (60 gm), sodium polystyrene sulfonate binds potassium secreted into the colon; however, its hypokalemic effect and its timing are variable and can take up to 10 hours. To accelerate its action, the rectal route of administration is preferred, but the effects may be lower than expected than with the oral route because of the decreased amount of time the resin stays in the colon (23). Oral sodium polystyrene sulfonate is often supplemented by a cathartic, such as sorbitol, to avoid the resin-induced constipation. Prospective studies have suggested that induction of a bowel movement by any cathartic is just as effective as giving it together with sodium polystyrene sulfonate (24). Potential complications of sodium polystyrene sulfonate administration include volume overload and intestinal necrosis when administered with sorbitol in the settings of decreased intestinal motility. As a result, the US Food and Drug Administration discourages concomitant use of sodium polystyrene sulfonate and sorbitol (25).

Dialysis is the definitive therapy in patients with end-stage renal disease, severe CKD, acute kidney injury, or if there is a comorbidity such as digitalis toxicity or rhabdomyolysis. In patients with an estimated glomerular filtration rate $< 15\text{--}20\text{ mL/min/1.73 m}^2$ ($<0.25\text{--}0.33\text{ mL/s/1.73 m}^2$) the impairment of urinary potassium excretion is sufficiently severe to require hemodialysis treatment (in addition to medical therapy) to resolve severe hyperkalemia (26). Hemodialysis is preferred over peritoneal or continuous dialysis because of the faster rate of potassium equilibration across the membrane. The rate of potassium removal varies according to the dialysate potassium concentration used, pre-dialysis serum potassium concentration, and the preceding medical treatment. For example, administration of agents causing extracellular potassium shift (such as insulin or albuterol) prior to hemodialysis transiently lowers potassium level in the plasma, thereby decreasing the gradient for potassium removal by dialysis. In situations when the effect of such treatments outlives the dialysis session, the physician should monitor for potential rebound hyperkalemia following dialysis (27). Dialysate containing no potassium (0 K bath) is used rarely and for a limited time in treating severe hyperkalemia, when the immediate risk of death outweighs the risk of ventricular arrhythmia induced by rapid potassium removal with hemodialysis. When the goal is to remove as much potassium as possible in a patient susceptible to cardiac arrhythmias, the opposite approach may be taken by progressively lowering potassium concentration in the dialysate during the treatment (potassium modeling). In all circumstances, patients must be monitored by continuous electrocardiography for rapid detection of arrhythmias associated with hyperkalemia or hypokalemia (28).

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References

- 1A. Palmer BF. A Physiologic-Based Approach to the Evaluation of a Patient With Hyperkalemia. *Am J Kidney Dis.* 2010 [published online ahead of print ***** doi:*****].
1. Schaefer TJ, Wolford RW. Disorders of potassium. *Emerg Med Clin North Am.* 2005; 23(3):723–747. [PubMed: 15982543]
2. Rose, BD.; Post, TW. *Clinical Physiology of Acid-Base and electrolyte disorders.* th edition. McGraw-Hill; New York: 2001.
3. Bia MJ, Lu D, Tyler K, De Fronzo RA. Beta adrenergic control of extrarenal potassium disposal: A beta-2 mediated phenomenon. *Nephron.* 1986; 43(2):117–122. [PubMed: 2872603]
4. Clausen T, Hansen O. Active Na-K transport and the rate of ouabain binding: The effect of insulin and other stimuli on skeletal muscle and adipocytes. *J Physiol.* 1977; 270(2):415–430. [PubMed: 903900]
5. Velázquez H, Perazella MA, Wright FS, Ellison DH. Renal mechanism of trimethoprim-induced hyperkalemia. *Ann Intern Med.* 1993; 119(4):296–301. [PubMed: 8328738]
6. Bonilla S, Goecke IA, Bozzo S, Alvo M, Michea L, Marusic ET. Effect of chronic renal failure on Na,K-ATPase alpha 1 and alpha 2 mRNA transcription in rat skeletal muscle. *J Clin Invest.* 1991; 88(6):2137–41. [PubMed: 1661300]
7. Pratt JH. Role of angiotensin II in potassium-mediated stimulation of aldosterone secretion in the dog. *J Clin Invest.* 1982; 70(3):667–672. [PubMed: 6286729]
8. Smellie WS. Spurious hyperkalaemia. *BMJ.* 2007; 334(7595):693–695. [PubMed: 17395950]
9. Rabelink TJ, Koomans HA, Hene RJ, Dorhout Mees EJ. Early and late adjustment to potassium loading in humans. *Kidney Int.* 1990; 38(5):942–947. [PubMed: 2266680]
10. Montague BT, Ouellette JR, Buller GK. Retrospective review of the frequency of ECG changes in hyperkalemia. *Clin J Am Soc Nephrol.* 2008; 3(2):324–330. [PubMed: 18235147]
11. Diercks DB, Shumaik GM, Harrigan RA, Brady WJ, Chan TC. Electrocardiographic manifestations: electrolyte abnormalities. *J Emerg Med.* 2004; 27(2):153–60. [PubMed: 15261358]
12. Webster A, Brady W, Morris F. Recognising signs of danger: EKG changes resulting from an abnormal serum potassium concentration. *Emerg Med J.* 2002; 19(1):74–77. [PubMed: 11777886]
13. Bisogno JL, Langley A, Von Dreele MM. Effect of calcium to reverse the electrocardiographic effects of hyperkalemia in the isolated rat heart: A prospective, dose-response study. *Crit Care Med.* 1994; 22(4):697–704. [PubMed: 8143480]
14. Weisberg LS. Management of severe hyperkalemia. *Crit Care Med.* 2008; 36(12):3246–51. [PubMed: 18936701]
15. Allon M, Takeshian A, Shanklin N. Effect of insulin-plus-glucose infusion with or without epinephrine on fasting hyperkalemia. *Kidney Int.* 1993; 43(1):212–217. [PubMed: 8433561]
16. Allon M, Copkney C. Albuterol and insulin for treatment of hyperkalemia in hemodialysis patients. *Kidney Int.* 1990; 38(5):869–872. [PubMed: 2266671]
17. Montoliu J, Lens XM, Revert L. Potassium-lowering effect of albuterol for hyperkalemia in renal failure. *Arch Intern Med.* 1987; 147(4):713–717. [PubMed: 3827459]
18. Sowinski KM, Cronin D, Mueller BA, Kraus MA. Subcutaneous terbutaline use in CKD to reduce potassium concentrations. *Am J Kidney Dis.* 2005; 45(6):1040–1045. [PubMed: 15957133]
19. Gutierrez R, Schlessinger F, Oster JR, Rietberg B, Perez GO. Effect of hypertonic versus isotonic sodium bicarbonate on plasma potassium concentration in patients with end-stage renal-disease. *Miner Electrolyte Metab.* 1991; 17(5):297–302. [PubMed: 1668124]

20. Allon M, Shanklin N. Effect of bicarbonate administration on plasma potassium in dialysis patients: Interactions with insulin and albuterol. *Am J Kidney Dis.* 1996; 28(4):508–514. [PubMed: 8840939]
21. Kim HJ. Combined effect of bicarbonate and insulin with glucose in acute therapy of hyperkalemia in end-stage renal disease patients. *Nephron.* 1996; 72(3):476–482. [PubMed: 8852501]
22. Carlisle EJ, Donnelly SM, Ethier JH, et al. Modulation of the secretion of potassium by accompanying anions in humans. *Kidney Int.* 1991; 39(6):1206–1212. [PubMed: 1895674]
23. Frohnert PP, Johnson WJ, Mueller GJ, Tauxe WN, McCall JT. Resin treatment of hyperkalemia. I. Exchange properties of a cation exchange resin (calcium cycle). *J Lab Clin Med.* 1968; 71(5):834–839. [PubMed: 5647684]
24. Emmett M, Hootkins RE, Fine KD, Santa Ana CA, Porter JL, Fordtran JS. Effect of three laxatives and a cation exchange resin on fecal sodium and potassium excretion. *Gastroenterology.* 1995; 108(3):752–760. [PubMed: 7875477]
25. US Food and Drug Administration (FDA). [January 10, 2010] Kayexalate (sodium polystyrene sulfonate) powder. <http://www.fda.gov/Safety/MedWatch/SafetyInformation/ucm186845.htm>
26. Alfonzo A, Isles C, Geddes C, Deighan C. Potassium disorders – clinical spectrum and emergency management. *Resuscitation.* 2006; 70(1):10–25. [PubMed: 16600469]
27. Ahmed J, Weisberg LS. Hyperkalemia in dialysis patients. *Semin Dial.* 2001; 14(5):348–356. [PubMed: 11679104]
28. Putcha N, Allon M. Management of hyperkalemia in dialysis patients. *Semin Dial.* 2007; 20(5): 431–9. [PubMed: 17897250]

Figure 1.

(A) Pretreatment electrocardiogram with peaked T-waves, prolonged PR and QRS intervals.

(B) Posttreatment electrocardiogram with normalization of T-waves, PR and QRS intervals.

Figure 2.

Algorithm for treatment of acute hyperkalemia. Pseudohyperkalemia should be considered in cases of hemolysis and in patients with severe thrombocytosis or leukocytosis, and proven by demonstrating that plasma potassium is lower than serum potassium. Bicarbonate is only indicated in patients with severe acidemia. Diuretics should be considered in patients with residual kidney function. Abbreviations: ECG, electrocardiographic; IV, intravenous; K^+ , potassium.

Table 1

Laboratory Data

Parameter	Value
<i>Serum Chemistry</i>	
Sodium (mEq/L)	132
Potassium (mEq/L)	7.2
Chloride (mEq/L)	99
Bicarbonate (mEq/L)	21
Urea Nitrogen (mg/dL)	65
Creatinine (mg/dL)	3.2
Calcium (mg/dL)	9
Anion Gap (mEq/L)	12
<i>Urinalysis</i>	
Specific Gravity	1.020
White blood cell count ($\times 10^9/L$)	10
Leukocyte esterase	1+

Note: Conversion factors for units: serum creatinine in mg/dL to $\mu\text{mol/L}$, $\times 88.4$; serum urea nitrogen in mg/dL to mmol/L, $\times 0.357$; Calcium mg/dL to mmol/L, $\times 0.2495$. No conversion necessary for sodium, potassium, chloride, bicarbonate, and anion gap in mEq/L and mmol/L, and for white blood cell count in $\times 10^3/\mu\text{L}$ and $\times 10^9/L$.

Table 2**Drugs Commonly Associated With Hyperkalemia**

Mechanism of Action	Drug Examples
Aldosterone down-regulation	ACE inhibitors ARBs Heparin NSAIDs Tacrolimus
Aldosterone receptor antagonism	Spironolactone
Collecting tubule sodium channel blockade	Trimethoprim Pentamidine Amiloride Triamterene
Collecting tubule Na ⁺ -K ⁺ -ATPase blockade	Cyclosporine Tacrolimus
Transcellular potassium imbalance	Labetalol Propranolol Carvedilol Succinylcholine Somatostatin

Abbreviations: ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; Na⁺-K⁺-ATPase, adenosine triphosphatase sodium-potassium pump; NSAID, nonsteroidal anti-inflammatory drug.

Table 3

Summary of treatment modalities for hyperkalemia and their expected effects

Treatment	Dose	Onset of action	Duration of action	Expected effect
Calcium gluconate	4.65 mEq (IV)	5-10 min	30-60 min	ECG normalization
Insulin	10 U (IV)	15-20 min	4-6 h	0.5-1.5 mmol/L ↓ K ⁺
Albuterol	20 mg (nebulized)	30-60 min	3-4 h	0.5-1.0 mmol/L ↓ K ⁺
Terbutaline	7 µg/kg (SC)	30-60 min	4-6 h	0.8-1.5 mmol/L ↓ K ⁺
Sodium polystyrene sulfate	15-30 g	variable	4-6 h	variable
Sodium bicarbonate [*]	50 mmol/L	4-6 h	4-6 h	variable

Abbreviations: IV, intravenous; ECG, electrocardiographic; SC, subcutaneous

^{*} Sodium bicarbonate should be given only in patients with severe acidemia (pH < 7.20 or serum bicarbonate < 15 mEq/L [<15 mmol/L])