The steps to address hyperkalemia include stabilization, redistribution, and excretion/removal of potassium.

### TREATMENT OF HYPERKALEMIA IN CKD

#### Table 3. Summary of interventions used for acute or chronic treatment of hyperkalemia

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Comments</th>
</tr>
</thead>
</table>
| Redistribution | Should not be used as an emergency treatment for life-threatening hyperkalemia because of its delayed onset of action.<br>May not be effective in patients with reduced GFR.<br>Efficacy questioned for acute treatment of patients on dialysis.<br>May not be effective in patients with reduced GFR.<br>Effect measured by normalization of electrocardiographic changes.<br>Efficacy only in hyponatremic patients.<br>Effect independent of insulin and aldosterone.<br>Caution in patients with known coronary artery disease.<br>Effect independent of insulin and aldosterone.<br>Caution in patients with heart failure due to sodium load.<br>Potential stabilization effects of dialysis on serum sodium, bicarbonate, calcium and/or magnesium levels can affect results.

**TREATMENT**

- **Acute**
  - **Stabilization**
    - 50-250 ml hypertonic saline (3-5%) Intravenous (acute) 5-10 min
    - Sodium polystyrene sulfonate
    - Cation exchange resins
  - **Redistribution**
    - Intravenous (acute) 1-3 min
    - Calcium gluconate (10%) solutions
  - **Excretion/removal of potassium**
    - 10 units of regular insulin Intravenous (acute) 30 min
    - 50-100 mmol sodium bicarbonate Intravenous (acute)
    - Sodium polystyrene sulfonate
    - Cation exchange resins
    - Calcium gluconate (10%) solutions

- **Chronic**
  - **Stabilization**
    - 40 mg furosemide or equivalent water (intravenous)
  - **Redistribution**
    - Intravenous or of regular insulin
  - **Excretion/removal of potassium**
    - Oral (either acute or chronic);
      - Patiromer 8.4, 16.8, or 25.2 g
      - Fludrocortisone acetate ≥0.1 mg
      - Calcium gluconate (10%) solutions
      - Sodium polystyrene sulfonate
      - Cation exchange resins

- **Treatment Route of Onset/Duration**
  - Intravenous (acute) 1-3 min
  - Intravenous (chronic) ~48 h
  - Oral (either acute or chronic) Until diuresis or until end of dialysis or longer****
  - Peritoneal dialysis Until diuresis or until end of dialysis or longer****
  - Peritoneal dialysis
  - Oral medications by at least 6 hours 17***

- **Potential Effects**
  - Loop diuretics for acute intervention<br>Loop diuretics for chronic management; loop diuretic for GFR <40 ml/min/1.73 m²
  - Calcium gluconate (10%) solutions<br>Calcium ether drugs for chronic management; treatment, loop diuretic for GFR <40 ml/min/1.73 m²
  - May not be effective in patients with reduced GFR<br>Should not be used as an emergency treatment for the treatment of hyperkalemia because it may delay onset of action.<br>May result in hyperkalemia or hypomagnesemia.

### REFERENCES

Hyperkalemia in CKD

The definition of hyperkalemia varies and limits such as >5.5, >6.0, or >7.0 mEq/L are used to indicate severity. Repetitive consecutive measures of serum potassium are needed to determine if hyperkalemia is sustained or a transient event. Many factors affect potassium homeostasis.

Table 1: Acute Versus Chronic Hyperkalemia

<table>
<thead>
<tr>
<th>Acute Hyperkalemia</th>
<th>Chronic Hyperkalemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased urinary potassium excretion</td>
<td>Impaired renal potassium excretion</td>
</tr>
<tr>
<td>Increased aldosterone and renin levels</td>
<td>Decreased aldosterone and renin levels</td>
</tr>
<tr>
<td>Requires immediate attention, often including cardiac monitoring</td>
<td>Requires ongoing management</td>
</tr>
</tbody>
</table>

Diagnosis and Evaluation of Hyperkalemia

Emergency diagnostic workup:
1. Assessment of cardiac function
2. Assessment of hydration status
3. Electrocardiogram

Elective/etiologic workup:
1. Comprehensive laboratory workup
2. Review of medication use

Table 2: Chronic Risk Factors for Hyperkalemia in CKD

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Due To</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium intake</td>
<td>Increased dietary potassium intake from sodium reduction or low sodium diet, trends for fast food diets, and from supplements</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>Increased urinary pH, increased potassium excretion</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Increased net potassium excretion, abnormalities of potassium homeostasis</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Increased urinary pH, increased potassium excretion</td>
</tr>
<tr>
<td>Advanced age</td>
<td>Decreased renal mass, increased risk of drug-induced hyperkalemia</td>
</tr>
</tbody>
</table>

Table 2: Chronic Risk Factors for Hyperkalemia in CKD

Risk Factor | Due To |
Potassium intake | Increased dietary potassium intake from sodium reduction or low sodium diet, trends for fast food diets, and from supplements |
Metabolic acidosis | Increased urinary pH, increased potassium excretion |
Diabetes | Increased net potassium excretion, abnormalities of potassium homeostasis |
Diabetes | Increased urinary pH, increased potassium excretion |
Advanced age | Decreased renal mass, increased risk of drug-induced hyperkalemia |

Chronic Hyperkalemia

Chronic changes include:
- Increased urinary potassium excretion
- Increased aldosterone and renin levels

Discontinuing these drugs is helpful in controlling or treating hyperkalemia, but the disadvantage is that it increases the risk for kidney disease progression and cardiovascular outcomes.

Treatments With RAASi in CKD

Studies show that use of ACEIs or ARBs in people with CKD reduces the risk for kidney failure and cardiovascular events, but their use contributes to hyperkalemia. The clinical practice guidelines for the use of RAAS in CKD are as follows:

- KDIGO Guidelines suggest that an ARB or ACEI be used in diabetic adults with CKD and urine albumin excretion 30-300 mg/24 hours (or equivalent), (2D)
- KDIGO Guidelines recommend that an ARB or ACEI be used in both diabetic and nondiabetic adults with CKD and urine albumin excretion >300 mg/24 hours (or equivalent), (1B)
- There is insufficient evidence to recommend combining an ACEI with ARBS to prevent progression of CKD. (Not Graded)
- Subsequent research shows that dual RAS inhibition with ACEI plus ARB not only fails to improve cardiovascular or renal outcomes, but predisposes patients to serious adverse events.

For patients with hyperkalemia, the degree and duration of potassium shift from the extracellular to the intracellular space can lead to cardiovascular and renal toxicity.

Consideration for using an ACEI or ARB in patients with CKD:

- Hyperkalemia and worsening kidney function can develop
- It is important to monitor serum potassium and estimated glomerular filtration rate (eGFR) within several weeks of starting or escalating a RAASi.
- Discontinuing these drugs is helpful in controlling or treating hyperkalemia, but the disadvantage is that it increases the risk for kidney disease progression and cardiovascular outcomes.

Adapted from: KDIGO, Kidney Disease: Improving Global Outcomes; NIH, National Institutes of Health; KDQI, National Kidney Foundation.

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Table 2. Chronic Risk Factors for Hyperkalemia in CKD

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Due To</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium intake</td>
<td>Increased dietary potassium intake from sodium substitutes, protein rich foods, smoking, and alcohol.</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>Potassium shift from the intracellular to the extracellular space.</td>
</tr>
<tr>
<td>Aldosterone deficiency</td>
<td>Potassium shift from the intracellular to the extracellular space.</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Impaired potassium excretion and impaired potassium excretory process.</td>
</tr>
<tr>
<td>Heart failure</td>
<td>Reduced cardiac output and increased potassium retention.</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>Reduced renal perfusion and increased potassium retention.</td>
</tr>
<tr>
<td>Age &gt;60</td>
<td>Increased serum aldosterone levels with age.</td>
</tr>
</tbody>
</table>

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TREATMENT WITH RAASi IN CKD

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Hypokalemia can lead to cardotoxicity, which can be fatal. The cause of hyperkalemia has to be determined to prevent future episodes.

Hyperkalemia is often asymptomatic, but patients may complain of nonspecific symptoms such as palpitations, nausea, muscle pain, weakness, or paresthesia. Moderate and especially severe hyperkalemia can lead to cardiotoxicity, which can be fatal.

IN CKD

1. Assessment of cardiac function, hypertension, and renal function.
2. Assessment of hydration status
3. Electrocardiogram

DIAGNOSIS AND EVALUATION OF HYPERKALEMIA

Emergency diagnostic workup:
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Elective/etiologic workup:
1. Comprehensive laboratory workup

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<td>Caused by abnormal release of potassium from blood cells, often due to acute hemolysis, trauma, or heart attacks</td>
<td>Caused by impairment of potassium excretion or plasma aldosterone levels and increased serum potassium level</td>
</tr>
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<td>Metabolic acidosis shifts potassium from the intracellular to the extracellular space</td>
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<tr>
<td>Drugs</td>
<td>Administration of potassium-sparing diuretics and矿业</td>
</tr>
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<td>Management goals</td>
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</tr>
<tr>
<td>Management goals</td>
<td>Decrease plasma aldosterone levels and</td>
</tr>
<tr>
<td>Metabolic factors</td>
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</tr>
<tr>
<td>Management goals</td>
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**Table 2. Chronic Risk Factors for Hyperkalemia in CKD**

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**Considerations for using an ACEI or ARB in patients with CKD:**

**Hyperkalemia and worsening kidney function can develop:**

- It is important to monitor serum potassium and estimated glomerular filtration rate (eGFR) within several weeks of starting or escalating a RAASi.
- Discontinuing these drugs is helpful in controlling or treating hyperkalemia, but the disadvantage is that it increases the risk for kidney disease progression and cardiovascular outcomes.17

**TREATMENT WITH RAASi IN CKD**

Studies show that use of ACEIs or ARBs in people with CKD reduces the risk for kidney failure and cardiovascular events, but their use contributes to hyperkalemia.22 The clinical practice guidelines for the use of RAAS in CKD are as follows:16,17

- KDIGO Guidelines suggest that an ARB or ACEI be used in diabetic adults with CKD and urate albumin excretion >30-300 mg/24 hours (or equivalent). (2D)
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TREATMENT WITH RAASi IN CKD

Studies show that use of ACEIs or ARBs in people with CKD reduces the risk for kidney failure and cardiovascular events, but their use contributes to hyperkalemia.13 The clinical practice guidelines for the use of RAAS in CKD are as follows: 11, 14

- KDIGO Guidelines suggest that an ARB or ACEI be used in diabetic adults with CKD and urine albumin excretion >300-300 mg/24 hours (or equivalent). (2D)
- KDIGO Guidelines recommend that an ARB or ACEI be used in both diabetic and nondiabetic adults with CKD and urine albumin excretion >100 mg/mg4 hours (or equivalent). (1B)
- There is insufficient evidence to recommend combining an ACEI with ARBs to prevent progression of CKD. (Not Graded)
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Table 2. Chronic Risk Factors for Hyperkalemia in CKD 1,5, 11

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Due To</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium intake</td>
<td>Increased dietary potassium intake from high-fiber foods, frequent fruit, dairy, and sweetened beverages.</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>Hypercalciuria from inorganic anion exchange resin used for dialysis.</td>
</tr>
<tr>
<td>KAs</td>
<td>Hyperkalemia with ARBs in nonadherent renal transplant recipients with systemic sclerosis.</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Insulin resistance and abnormalities in renal excretory processes.</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Hyperkalemia with ARBs in nonadherent renal transplant recipients with systemic sclerosis.</td>
</tr>
<tr>
<td>Drugs</td>
<td>Hyperkalemia with ARBs in nonadherent renal transplant recipients with systemic sclerosis.</td>
</tr>
<tr>
<td>Management goals, including potassium mobilization and potassium-lowering medications</td>
<td></td>
</tr>
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<td></td>
</tr>
</tbody>
</table>

Table 1. Acute Versus Chronic Hyperkalemia 4, 4 4

<table>
<thead>
<tr>
<th>Acute Hyperkalemia</th>
<th>Chronic Hyperkalemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Causes by smoking cessation of potassium intake, likely due to increased renal excretion (Renal oncofetal histidine diastase)</td>
<td></td>
</tr>
<tr>
<td>Causes by increased potassium mobilization and impaired potassium excretion.</td>
<td></td>
</tr>
<tr>
<td>Requires immediate attention, a cardiac monitor, acute medical interventions, possibly dialysis.</td>
<td></td>
</tr>
<tr>
<td>Requires ongoing management to control the underlying disturbance of potassium balance and prevent or correct electrolyte abnormalities.</td>
<td></td>
</tr>
<tr>
<td>Management goals, include potassium mobilization and potassium-lowering medications.</td>
<td></td>
</tr>
</tbody>
</table>

Discriminator: CKD, chronic kidney disease; ARB, angiotensin receptor blocker; NSAIDS, nonsteroidal anti-inflammatory drugs; MRA, mineralocorticoid receptor antagonist. (Not Graded)

Emergency diagnostic workup: 1
1. Assessment of cardiac function
2. Assessment of hydration status
3. Electrolyte workup

Elective/etiologic workup: 2
1. Comprehensive laboratory workup
2. Review of medication use

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The steps to address hyperkalemia include stabilization, redistribution, and excretion/removal of potassium.

**TREATMENT OF HYPERKALEMIA IN CKD**

**TREATMENT**

- **Stabilization**: 
  - **10-40 mg furosemide or equivalent** (up to 0.4-1.0 mg daily)
  - **Fludrocortisone acetate ≥0.1 mg**: for chronic management, dose depends on aldosterone deficiency.

- **Redistribution**:
  - **Calcium gluconate or calcium chloride**: 10 units of regular insulin Intravenous (acute) 30 min
  - **50-250 ml hypertonic saline (3-5%)**: Intravenous (acute) 5-10 min

- **Excretion/Removal**:
  - **Sodium polystyrene sulfonate**
  - **Cation exchange resins**
  - **Patiromer 8.4, 16.8, or 25.2 g**

**Mechanisms**

- **Stabilization**:temporary, until potassium levels are <6.5 mmol/L.
- **Redistribution**: short term, 4-6 hours.
- **Excretion**: sustained, >24 hours.

**Mechanism Comments**

- **Stabilization**: temporary, until potassium levels are <6.5 mmol/L.
- **Redistribution**: short term, 4-6 hours.
- **Excretion**: sustained, >24 hours.

**References**

CaCl₂ is caustic and could damage peripheral veins. **Limited data available from clinical studies. ***Clinical studies being done to assess the clinical significance of the in vitro interaction. ****Effects can

The steps to address hyperkalemia include stabilization, redistribution, and excretion/removal of potassium.

TREATMENT

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Route of administration</th>
<th>Onset/Duration</th>
<th>Mechanism</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>6.8 mEq of sodium, corresponding to 4 mL (40 mg/ml) furosemide</td>
<td>Intravenous (acute)</td>
<td>1-3 min</td>
<td>Membrane stabilization</td>
<td>does not affect serum potassium level, effect measured by normalization of electrocardiographic changes or by direct measurement of serum potassium level</td>
</tr>
<tr>
<td>Loop diuretics without added sodium bicarbonate</td>
<td>Intravenous (acute)</td>
<td>≤ 1-2 h</td>
<td>Membrane stabilization</td>
<td>initially only on hyperkalemia, patients</td>
</tr>
<tr>
<td>10-20 mL sodium bicarbonate</td>
<td>Intravenous (acute)</td>
<td>2-4 min</td>
<td>Sodium</td>
<td>may sodium causes pre-existing hyperosmolar and hyperchloric acidosis, questionable for acute treatment of patients on dialysis</td>
</tr>
<tr>
<td>oral medications</td>
<td>Oral (either acute or oral (chronic))</td>
<td>10-30 min</td>
<td>Cation exchange resins</td>
<td>50-100 mmol sodium bicarbonate Intravenous (acute)</td>
</tr>
<tr>
<td>oral medications</td>
<td>Oral (either acute or oral (chronic))</td>
<td>10-30 min</td>
<td>Cation exchange resins</td>
<td>25-50 g sodium polystyrene sulfonate Oral (either acute or oral (chronic))</td>
</tr>
<tr>
<td>oral medications</td>
<td>Oral (either acute or oral (chronic))</td>
<td>10-30 min</td>
<td>Cation exchange resins</td>
<td>6.8 mmol of calcium, corresponding to 1 mEq of its delayed onset of action17</td>
</tr>
<tr>
<td>oral medications</td>
<td>Oral (either acute or oral (chronic))</td>
<td>10-30 min</td>
<td>Hydrochlorothiazide (sodium-free)</td>
<td>Should not be used as an emergency treatment for life-threatening hyperkalemia because may not be effective in patients with reduced GFR</td>
</tr>
<tr>
<td>oral medications</td>
<td>Oral (either acute or oral (chronic))</td>
<td>10-30 min</td>
<td>Hydralazine</td>
<td>Efficacy only in hyponatremic patients</td>
</tr>
<tr>
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<td>Oral (either acute or oral (chronic))</td>
<td>10-30 min</td>
<td>Dapagliflozin</td>
<td>Efficacy questioned for acute treatment of patients on dialysis</td>
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<tr>
<td>oral medications</td>
<td>Oral (either acute or oral (chronic))</td>
<td>10-30 min</td>
<td>Bisoprolol</td>
<td>Caution advised in patients receiving digoxin</td>
</tr>
<tr>
<td>oral medications</td>
<td>Oral (either acute or oral (chronic))</td>
<td>10-30 min</td>
<td>Bisoprolol</td>
<td>Dose can be repeated if no effects noted</td>
</tr>
</tbody>
</table>

Additional references:


**CaCl₂ is caustic and could damage peripheral veins. **Limited data available from clinical studies. ***Limited data available from clinical studies being done to assess the clinical significance of the in vitro interaction. ****Effects can

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