HYPO & HYPERKALEMIA

National Standard Treatment Guideline

Ministry of Health
Republic of Maldives

ADB

JFPR
Japan Fund for Prosperous and Resilient Asia and the Pacific

From the People of Japan

World Health Organization
Maldives

National Standard Treatment Guidelines

- Acid Peptic Disease
- Acute Anxiety
- Acute Pancreatitis
- Acute Psychosis
- Acute kidney Injury
- Arrhythmia
- Chronic Liver Disease
- Chronic Pancreatitis
- Chronic kidney disease
- Congenital Heart Diseases
- Dementia
- Depression
- Diabetes Mellitus Type 1
- Diabetes Mellitus Type 2
- Gestational Diabetes
- Epilepsy
- Heart Failure
- Hyponatremia
- Hypernatremia
- Hypokalemia
- Hyperkalemia
- Interstitial Lung Disease
- Liver Failure
- Obesity
- Obstructive Sleep Apnoea
- Osteoarthritis
- Ovarian Cancer
- Pneumonia
- Stroke
- Upper Gastrointestinal bleed
- Unstable Angina

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GUIDELINES DEVELOPMENT METHODOLOGY

The development of the Maldives Standard Treatment Guidelines (STGs) followed a structured, evidence-informed, and consensus-driven methodology adapted from internationally accepted guideline-development standards and the Delhi Society for Promotion of Rational Use of Drugs (DSPRUD) model. The process combined systematic evidence retrieval, critical appraisal, contextual adaptation, and multidisciplinary expert review to ensure feasibility, clinical relevance, and national ownership.

1. Determining Scope and Priority Conditions

Priority clinical conditions were identified through consultation with national programme managers, specialty clinicians, and health-system stakeholders. Selection criteria included: (i) major causes of morbidity and mortality, (ii) observed variation in clinical practice or prescribing patterns, (iii) potential to improve patient outcomes, and (iv) the feasibility of implementation across health-facility levels in Maldives. The final list of diseases reflected national epidemiology, service-delivery capacity, and essential-medicine availability.

2. Identification of Existing Evidence and Source Guidelines

A targeted search strategy was used to identify high-quality existing clinical guidelines. Searches were conducted across international guideline repositories (e.g., WHO, NICE, SIGN and other intergovernmental bodies, international and national guideline repositories, specialty societies and professional associations.

3. Quality Appraisal of Source Guidelines

Retrieved guidelines were screened for transparency of development, methodological rigour, clarity of recommendations, applicability to health-system reality, editorial independence. Guidelines were included if they met the Institute of Medicine (IOM) definition of a clinical guideline and addressed treatment or management of priority conditions. Guidelines that did not meet minimum quality standards, review articles, diagnostic criteria, or technical standards were excluded.

4. Adoption, Adaptation, and Contextualization

The guideline-development team employed an adopt–adapt–contextualize model:

- **Adoption**: High-quality recommendations that aligned with Maldivian health-system realities were retained without modification.
- **Adaptation**: Recommendations were modified when local considerations such as diagnostic capacity, medicine availability, workforce skills, referral pathways, or cost constraints affected feasibility.

- **Contextualization**: Where evidence was absent or inconclusive, conditional recommendations were formulated based on expert consensus, with explicit consideration of pragmatism, safety, and local workflows.
  Medicines were selected in alignment with the Maldives National Essential Medicines List (NEML), based on suitability, efficacy, safety, and availability.

5. Expert Consensus and Multidisciplinary Input

Draft recommendations were initially prepared by experts from the DSPRUD, India, providing a strong methodological foundation for the process. Building on this, a collaborative and participatory process brought together clinicians from internal medicine, paediatrics, obstetrics-gynaecology, surgery, emergency medicine, endocrinology, cardiology, general practitioners, and public health representing different levels of healthcare. Consensus was achieved through moderated discussions, iterative revisions, and resolution of divergent views. For topics lacking strong evidence, recommendations were derived from expert clinical judgment grounded in extensive practice experience.

6. Drafting, Peer Review, and Validation

Each guideline section was organized in a standard format including key clinical features, essential investigations, non-pharmacological management, pharmacological therapy (with step-up/step-down options where relevant), referral criteria, paediatric considerations, and follow-up requirements. Drafts were peer-reviewed by senior clinicians and national experts. Reviewer comments were systematically integrated to strengthen clarity, accuracy, and applicability.

7. Addressing Conflicts of Interest

All contributors declared the absence of conflicts of interest. Individuals with potential or perceived conflicts were excluded from authorship or decision-making roles.

8. Updating and Future Revisions

The STGs were conceptualized as a living document. Future updates will incorporate new scientific evidence, changes in essential-medicine availability, national programme priorities, and user feedback from clinicians. Periodic review cycles will ensure the continued relevance and reliability of recommendations.

9. Distinctive Features of the Guidelines

Developed through a collaborative process involving a large group of multidisciplinary experts from different levels of healthcare, the guidelines incorporate the following distinctive features:

- **Diagnostic Assumption and Confirmation**: While assuming that an initial diagnosis has been established by the healthcare provider, the guidelines provide essential information for confirming diagnoses. This includes a comprehensive overview of major signs and symptoms, descriptions of confirmatory tests, and clear guidance on practices that are prohibited, discouraged, or unreliable—promoting evidence-based medicine supported by relevant references.
- **Comprehensive Treatment Approach**: The guidelines offer a systematic, up-to-date framework for managing medical conditions across the continuum of care. They begin at the primary care level and extend to secondary and tertiary care, incorporating protocols for treatment response assessment and referral criteria as integral components.
- **Diverse Treatment Modalities**: Recommendations encompass both non-pharmacological and pharmacological interventions and surgical intervention where applicable, providing flexibility for individualized treatment plans. Cautionary notes are included where necessary to ensure safe and effective use of therapies.
- **Assessment and Referral Criteria**: Clear criteria and goals for evaluating patient response to treatment are provided, along with guidance on when referral to higher levels of care is warranted ensuring continuity and comprehensiveness in patient management.

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The Government of the Republic of Maldives is committed to ensuring universal access to quality health services for all citizens. The Constitution of Maldives mandates the progressive realization of rights, including the right to good standards of health care for the population. In line with this national commitment, standardized quality health services are regarded as the foundation of a strong and equitable healthcare system.

This important work would not have been possible without the cooperation and support of many individuals and institutions. We express our sincere appreciation to the Honourable Minister of Health, Abdullah Nazim Ibrahim, for his leadership, commitment, and continuous guidance throughout the development process. We are grateful to WHO and ADB for their significant contribution, support, and technical assistance.

Our heartfelt gratitude is extended to the technical lead and editor, Dr. Sangeeta Sharma, Professor, Neuropsychopharmacology, IHBAS and President, Delhi Society for Promotion of Rational Use of Drugs (DSPRUD), and her team. We express our deepest appreciation to the Maldivian and DSPRUD experts and contributors who played a pivotal role in this process. Their technical expertise and dedication to adapt the standards to the Maldivian context have been instrumental in the development and finalization of these guidelines. The time, experience, generous sharing of knowledge and insights contributed by all parties have not only enriched the work but also have been invaluable in making these standards practical, locally acceptable, and aligned with the needs of the resident population.

It is important to acknowledge the immense efforts, involvement, timely coordination, collaboration, and dedication of the Quality Assurance and Regulation Division team who made it possible for these Clinical Treatment Guidelines to come into existence.

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HYPO & HYPERKALEMIA

QUICK REFERENCE GUIDE (HYPOKALEMIA)

Hypokalemia is a common electrolyte disorder, particularly in hospitalized patients and those with gastrointestinal or renal losses. Potassium (K^+) is essential for membrane excitability, muscle contraction, and cardiac rhythm. Even small shifts matter and can be rapidly fatal without timely correction especially in infants and neonates. It can develop acutely (hours - days) or chronic (weeks - months). It affects all age groups but is especially dangerous in neonates and infants due to smaller potassium reserves and immature renal handling. Severe untreated hypokalemia can lead to life-threatening arrhythmias, respiratory failure, and sudden death, while early recognition and correction usually result in full recovery and favorable long-term outcomes.

Causes, risk factors & triggers

- Gastrointestinal losses: diarrhoea, vomiting, nasogastric suction, laxatives.
- Renal losses: diuretics (loop/thiazide), mineralocorticoid excess, renal tubular acidosis, tubulopathies (Bartter, Gitelman), osmotic diuresis, amphotericin B, high-dose penicillin.
- Shifts into cells: insulin bolus, β_2-agonists, alkalosis, refeeding, thyrotoxicosis.
- Low intake/malnutrition; alcohol misuse
- Pediatrics: feeding errors (over-diluted formula), diarrhea, congenital tubulopathies.
- Risk enhancers: hypomagnesemia, digitalis therapy, acute coronary syndrome, chronic kidney disease, eating disorders.

Evaluation for diagnosis

- **Clinical features**: Weakness, cramps, fatigue, constipation/ileus; severe: paralysis, arrhythmias, respiratory failure. **Children: poor feeding, irritability, floppiness, reduced movement.**
- **Physical examination**: Volume status, muscle strength, bowel sounds; check blood pressure (hypertension → mineralocorticoid excess).
- **Laboratory investigations**: Confirm K^+ below age range; repeat if unexpected; avoid hemolyzed samples. Serum: K^+, Mg^{2+}, creatinine/urea, bicarbonate or blood gas, glucose. Urine: spot urine K^+ (or K^+/creatinine), urine chloride, urine osmolality; consider diuretic screen. Endocrine/other: renin/aldosterone (if hypertension + alkalosis), thyroid tests if periodic paralysis suspected.

Confirmation of diagnosis

Age-adjusted normal ranges (children)

- **Neonates**: 3.5-6.0 mmol/L
- **Infants**: 3.7-5.9 mmol/L
- **Children (1-12 y)**: 3.5-5.5 mmol/L
- **Adolescents**: 3.5-5.0 mmol/L

- Validated low plasma K^+ on repeat + supportive context (acid-base, urine indices) distinguishing renal vs extrarenal loss or shift with symptoms or ECG changes.

Classification / severity assessment criteria

- Mild: 3.0-3.4 mmol/L
- Moderate: 2.5-2.9 mmol/L
- Severe: <2.5 mmol/L or any level with arrhythmia, paralysis, respiratory compromise, or ischemia.

Critical values (act immediately)

- **K^+ ≤2.5 mmol/L** at any age or **≤3.0 mmol/L** with symptoms or ECG changes, digitalis use, acute coronary syndrome, or rapid ongoing losses.
- Treat as an emergency: continuous ECG, prompt replacement, and evaluation of cause.

Differential Diagnosis (key mimics)

- Pseudohypokalemia (extreme leukocytosis with delayed processing).
- Neuromuscular weakness from other causes (Guillain-Barré syndrome, myasthenia).
- ECG mimics of U-waves/ST-T changes (hypothermia, ischemia, drugs).
- Electrolyte look-alikes: hypomagnesemia, hypocalcemia.

Management Goals & principles

- Treat the cause.
- Restore K^+ to safe range at a controlled rate.
- Prevent recurrence (address meds, losses, nutrition).
- Replace magnesium first/alongside if low, otherwise K^+ repletion will stall.

Approach to management

1. Assess urgency: Severe or symptomatic - urgent IV replacement with continuous ECG.
2. Chose route: Oral preferred if stable; IV if severe, NPO, or ongoing high losses.
3. Choose formulation: Potassium chloride (KCl) for most cases (chloride deficit common). Citrate/ acetate salts acceptable if acidosis or GI tolerance issues.
4. Address mechanism: Stop offending drugs, treat diarrhea/vomiting, manage mineralocorticoid excess, correct alkalosis if safe, treat thyrotoxicosis/periodic paralysis.
5. Monitor and titrate to targets (see "Assessment of response").

Non-Pharmacological interventions

- Potassium-rich diet: bananas, citrus, tomatoes, potatoes/sweet potatoes, leafy greens, beans, yogurt.

- Rehydrate appropriately (oral rehydration solution during diarrheal illness).
- Simple tools for dosing measurement and monitoring: marked cups/bottles, intake/output logs, daily weights.

Pharmacological therapy

1. Potassium replacement

- **Oral replacement (preferred when able)**: Adults: KCl 40-100 mmol/day in divided doses; larger deficits may need 20-40 mmol every 6-8 h. **Children: 1-2 mmol/kg/day in 3-4 doses (single dose ≤2 mmol/kg).**

  Cautions: GI irritation - give with food/water; avoid in severe renal impairment without monitoring.

- **Intravenous replacement (severe/ symptomatic/NPO): Adults**: Usual: 20-40 mmol KCl in 0.9% saline over 4-6 h; repeat per labs. Rates: up to 10-20 mmol/h with cardiac monitoring; central line for concentrations >40mmol/L. **Caution: Never IV push. Children: 0.3-0.5 mmol/kg/h; max 40 mmol/L (peripheral) or 60 mmol/L (central). Continuous ECG if >0.25 mmol/kg/h.**

  Cautions: Hyperkalemia risk, phlebitis with concentrated peripheral infusions; verify line patency.

2. Magnesium replacement

- Replace Mg^{2+} (e.g., magnesium sulfate IV) if low to allow K^+ to normalize.

  Mild hypomagnesemia (0.6-0.69 mmol/L) 12-24 mmol/day in divided doses. In moderate to severe (0<0.59 mmol/L) 1-2 grams (~8-16 mEq) IV infusion over 1-2 hours, repeated as needed based on severity and renal function. In critically low levels, up to 4-5 grams IV infusion slowly over several hours. Caution: Monitor renal function closely and adjust dose. Reassess magnesium levels 24 hours after administration to guide further therapy.

Etiology-specific

- Mineralocorticoid excess: treat cause (e.g., adrenal nodule), consider spironolactone/eplerenone.
- Renal tubular acidosis: alkali therapy with Sodium bicarbonate + K^+. Potassium citrate is preferred when hypokalemia coexists, or Potassium chloride may be used if citrate is unavailable or not tolerated. Dose: 20-60 mEq/day depending on severity and renal function. Target serum potassium: 4.0-5.0 mmol/L. Monitor urinary potassium excretion, values >40-80 mEq/day despite hypokalemia suggest ongoing renal losses.
- Thyrotoxic periodic paralysis: non-selective β-blocker, definitive thyroid therapy (careful K^+ dosing to avoid rebound hyperkalemia).

Assessment of response, follow-up, and adjustment

- **Targets: rise ~0.2-0.5 mmol/L/h initially; avoid overcorrection.**

- **Monitoring**:
  - Severe/IV: K^+ every 2-4 h + continuous ECG in severe cases or if arrhythmias are suspected.
  - Oral/mild-moderate: K^+ every 12-24 h
  - Check input/output (I&O). Recheck serum potassium, bicarbonate, and magnesium 24-48 hours after initiating therapy.
- **If targets not met**:
  - Too fast rise: slow or pause infusion; recheck within 1-2 h.
  - No rise: verify Mg^{2+}, search for hidden losses (diarrhea, diuretics, osmotic diuresis), check IV access/ site, adjust dose/rate.
- Step-up criteria: K^+ <2.5 mmol/L despite therapy, arrhythmias, paralysis/respiratory compromise, severe ongoing losses, unclear etiology refer to higher-level care.
- Step-down criteria: K^+ ≥3.5 mmol/L for ≥24-48 h without IV, ECG normalized, cause controlled → transition to oral and maintenance plan.
- Post-correction: check K^+ daily for 48-72 h to detect rebound; arrange outpatient K^+/Mg^{2+} at 1-2 weeks.

Referral (tiered: primary to secondary to tertiary)

- Primary care: start oral correction for mild cases, educate, and refer for red flags (arrhythmia, paralysis, refractory vomiting/diarrhea).
- Secondary care: continuous monitoring, IV therapy, etiologic work-up.
- Tertiary care/ICU: persistent severe hypokalemia, complex endocrine/renal causes, need for central lines/high-rate infusions, significant comorbidities (advanced heart failure, chronic kidney disease).

Complications

- Ventricular arrhythmias, cardiac arrest; respiratory failure; ileus; rhabdomyolysis; refractory hypokalemia if Mg^{2+} is low; rebound hyperkalemia with excessive replacement.

Objectives of Patient education & Instructions

- Recognize symptoms early (weakness, cramps, palpitations, constipation; in children - poor feeding, floppiness).
- Follow the plan: exact doses, correct measuring tools, don't self-adjust meds.
- Diet: include K^+-rich foods daily; avoid unnecessary restrictions/fasting.
- Hydration: use oral rehydration solution during diarrheal illness (children).
- Keep appointments: labs and ECGs on schedule; bring a simple log.
- Seek urgent care for paralysis, severe palpitations/chest pain, fainting, breathing difficulty, seizures.

INTRODUCTION (HYPOKALEMIA)

Potassium is the main intracellular cation that maintains membrane potential, drives nerve impulses and muscle contraction, stabilizes cardiac rhythm, and buffers acid-base shifts. Abnormal levels are emergencies: hypokalemia <3.5 mmol/L can cause weakness, ileus, respiratory failure, and ventricular arrhythmias; hyperkalemia >5.0 mmol/L in children (>5.5 in neonates) can precipitate bradycardia, high-grade block, ventricular fibrillation, or asystole. Children especially neonates deteriorate faster due to smaller body stores and immature kidney handling. Treat the cause and correct promptly with close ECG and electrolyte monitoring. Under-correction is common such as prolonged IV dextrose without K^+, no rechecks during ongoing losses (diarrhea, diuretics), rapid IV fluids without K^+, not supplementing malnourished kids, underestimating stool losses, high-concentration peripheral infusions and dosing errors: miscalculating weight-based K^+ increases arrhythmia risk.

SCOPE OF THE GUIDELINES

Guidelines for recognizing, diagnosing, treating, and following up hypokalemia in adults and children (neonates-adolescents) across primary, secondary, and tertiary care. Excludes potassium issues limited to perioperative settings or experimental therapies. Pediatric evidence is thinner, some recommendations extrapolate from adults and require local adaptation (formulations, monitoring).

Intended users

Physicians, pediatricians, family practitioners, nurses, emergency physicians, intensivists, nutritionists, dietitians, and pharmacists involved in electrolyte care.

By care level

Primary care: Detect early (symptoms + basic labs), start safe oral correction for mild-moderate cases, educate on warning signs (weakness, palpitations, lethargy). In children, watch for feeding difficulty, hypotonia, constipation; refer promptly if red flags.

Secondary care: Full work-up for cause; structured oral/IV replacement with continuous monitoring and acid-base/electrolyte interpretation. Pediatrics: weight-based dosing, slower correction if chronic, tighter monitoring with cardiac/metabolic comorbidity.

Tertiary care: Refractory/complex etiologies (renal tubular, endocrine, severe GI loss, critical illness); advanced cardiac monitoring and individualized regimens with nephrology/endocrinology/cardiology. Pediatrics: inherited tubular disorders (Bartter/Gitelman), post-transplant wasting, central access when high-concentration IV K^+ is needed.

DEFINITION

In adults, hypokalemia is defined as a serum potassium concentration below 3.5 mmol/L

In pediatric patients, the same numerical threshold applies; however, neonatal reference ranges may vary slightly (e.g., lower limit 3.5-3.7 mmol/L in term neonates, and 3.7-4.0 mmol/L in preterm infants). These higher lower-limits in neonates reflect their relatively higher total body water and rapid metabolic turnover

Severity is graded as:

- Mild: 3.0-3.5 mmol/L
- Moderate: 2.5-2.9 mmol/L
- Severe: <2.5 mmol/L or any level associated with life-threatening arrhythmias, respiratory failure, or paralysis.

In children, "severe" hypokalemia also includes any value associated with ECG changes, feeding refusal, hypotonia, or apneic episodes even if the potassium is above 2.5 mmol/L because their physiological reserve is lower and deterioration can be rapid

CAUSES, RISK FACTORS & TRIGGERS

Category	Causes	Pediatric-Specific considerations
Decreased Intake	■ Poor dietary intake: fasting, restrictive diets, malnutrition ■ Prolonged IV therapy without K^+ (potassium-free fluids)	■ Low-potassium infant formulas, prolonged potassium-free parenteral nutrition, overly diluted home-prepared feeds
Excessive Potassium Losses (Renal)	■ Diuretics: loop (furosemide, bumetanide), thiazides ■ Mineralocorticoid excess: hyperaldosteronism, Cushing's ■ Renal tubular disorders: Bartter, Gitelman, Fanconi ■ Drugs: amphotericin B, high-dose penicillins, aminoglycosides ■ Osmotic diuresis: uncontrolled diabetes, mannitol ■ Post-obstructive diuresis	■ Congenital adrenal hyperplasia ■ Higher susceptibility to urinary K^+ losses due to immature renal tubules (preterm neonates, especially on diuretics for bronchopulmonary dysplasia (BPD)

Excessive Potassium Losses (GI)	■ Diarrhea (infectious, inflammatory) ■ Vomiting (indirect renal K^+ loss from metabolic alkalosis) ■ Nasogastric suction ■ Laxative abuse	■ High vulnerability in acute gastroenteritis, rotavirus, persistent diarrhea syndromes due to limited body K^+ stores and high surface-area-to-volume ratio
Transcellular Shift into Cells	■ Metabolic alkalosis ■ Insulin therapy (esp. in DKA) ■ β-adrenergic stimulation: stress, catecholamines, salbutamol - Hypothermia/ rewarming ■ Rapid cell proliferation (megaloblastic anemia treatment)	■ Aggressive β-agonist therapy for asthma - Overly rapid acidosis correction in pediatric DKA
Other Risk Factors	■ Critical illness, sepsis ■ Burns, trauma (redistribution phase) ■ Hypomagnesemia	■ Preterm neonates at high risk due to immature renal function
Common Triggers	■ Older adults on chronic diuretics without supplementation ■ Hospitalized patients on large volumes of potassium-free IV fluids ■ Children with congenital renal tubular/ endocrine disorders or chronic diarrhea ■ Poor caregiver awareness of need for potassium-rich diet post-diarrhea in young children	■ Same as listed; emphasize caregiver education in prevention

EVALUATION FOR DIAGNOSIS

Domain	Key Points	Pediatric-Specific Considerations
Clinical History	■ Onset: Acute → transcellular shift/acute loss; Gradual → chronic loss/dietary deficiency ■ Diet: Poor intake, restrictive diets, potassium-free IV therapy ■ GI: Diarrhea, vomiting, laxatives ■ Urinary: Polyuria, diuretic use ■ Endocrine: Cushing's, hyperaldosteronism ■ Drugs: Diuretics, corticosteroids, β-agonists, insulin, aminoglycosides, amphotericin B ■ PMH: Renal/adrenal disease, metabolic alkalosis, recent hospitalization ■ FHx: Renal tubular disorders, periodic paralysis	■ Formula preparation errors ■ Recent feeding changes ■ Review asthma meds especially repeated/high-dose nebulized salbutamol
Physical Examination	■ Vitals: Hypotension/tachycardia → volume depletion; ■ Hypertension → mineralocorticoid excess ■ General: Weakness, fatigue, confusion - Cardiac: Arrhythmia signs, irregular pulse, weak pulses ■ Neuromuscular: ↓ reflexes, muscle weakness, cramps, flaccid paralysis (severe) ■ GI: Abdominal distension, ↓ bowel sounds (ileus) ■ Hydration: Skin turgor, mucous membranes, JVP	■ Poor feeding, lethargy, irritability may be only clues
Laboratory Investigations	Initial confirmation: Serum K^+ (exclude hemolysis), Na^+, Cl^-, HCO_3^-, creatinine, BUN, Mg^{2+}, glucose. **Etiology**: ■ Urine K^+: □ >20 mmol/L - renal loss; □ <20 mmol/L -extrarenal loss ■ Urine K^+/Cr ratio ■ Urine Cl^- (vomiting vs. diuretics) ■ ABG/VBG (acid-base) ■ Plasma renin & aldosterone (if mineralocorticoid excess)	■ Use age-adjusted lab reference ranges ■ Exclude pseudohypokalemia in high WBC count (e.g., leukemia) ■ Screen for congenital tubulopathies (Bartter, Gitelman) in chronic/unexplained cases

CONFIRMATION OF DIAGNOSIS

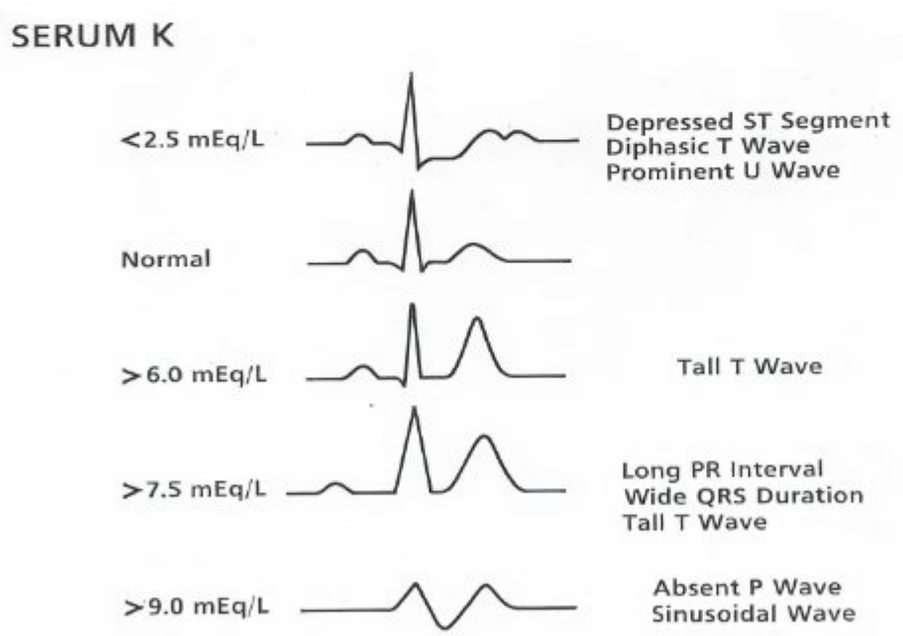
For confirm of hyponatremia requires:

1. Potassium below the age-adjusted reference (see below) on a validated sample, and
2. Urine index establishing renal vs extrarenal mechanism (spot K^+ or K^+/Cr), plus
3. Context checks (acid-base, Mg^{2+}, glucose/insulin/β-agonists) to distinguish deficit from shift.

Verify the result

- Repeat unexpected values and avoid hemolyzed samples (false rise in K^+)
- Ensure potassium interpretation accounts for plasma pH (alkalosis lowers serum K^+ via intracellular shift).
- Exclude pseudohypokalemia in hematologic malignancy or marked leukocytosis (>100 × 10^9/L): send plasma K^+ in heparinized tube, processed promptly.

ECG correlation (supportive, not diagnostic): U waves, ST depression, flattened T waves, prolonged QT, ventricular ectopy or tachyarrhythmias.



Clinical correlation is essential: Symptoms and ECG changes may not always align with numeric severity, especially in children with coexisting metabolic alkalosis, hypomagnesemia, or congenital channelopathies. Therefore, treatment urgency should be guided by both laboratory values and clinical presentation.

SEVERITY ASSESSMENT

Severity of hypokalemia is determined by the absolute serum potassium concentration, the rate at which it has fallen, and the presence or absence of symptoms. **In pediatric patients, age-specific reference ranges must be used when interpreting values, as "low" for a newborn may be different from "low" for an adolescent.**

Severity	Adults (mmol/L)	Pediatric Cut-offs (mmol/L)	Clinical Features
Normal	3.5-5.0 mmol/L	**Neonates**: 3.5-6.0 mmol/L **Infants**: 3.7-5.9 mmol/L **Children**: 3.5-5.5 mmol/L **Adolescents**: 3.5-5.0 mmol/L	-
Mild	3.0-3.4	Neonates: 3.5-3.9 Infants/Children: 3.2-3.4	Often asymptomatic; may have mild fatigue, myalgia, or constipation
Moderate	2.5-2.9	Neonates: 3.0-3.4 Infants/Children: 2.5-3.1	Muscle weakness, cramps, ileus, mild ECG changes (flattened T waves)
Severe	<2.5	Neonates: <3.0 Infants/Children: <2.5	Flaccid paralysis, hypoventilation, severe arrhythmias, rhabdomyolysis

Additional Pediatric Considerations:

- In neonates and critically ill infants, even a small absolute drop in potassium may have proportionally greater effects on cardiac conduction and muscle function than in adults.
- Rapid changes even within the "mild" numeric range can be dangerous in children with underlying cardiac disease or those receiving digoxin.
- Chronic mild hypokalemia in children may cause growth impairment if not addressed.

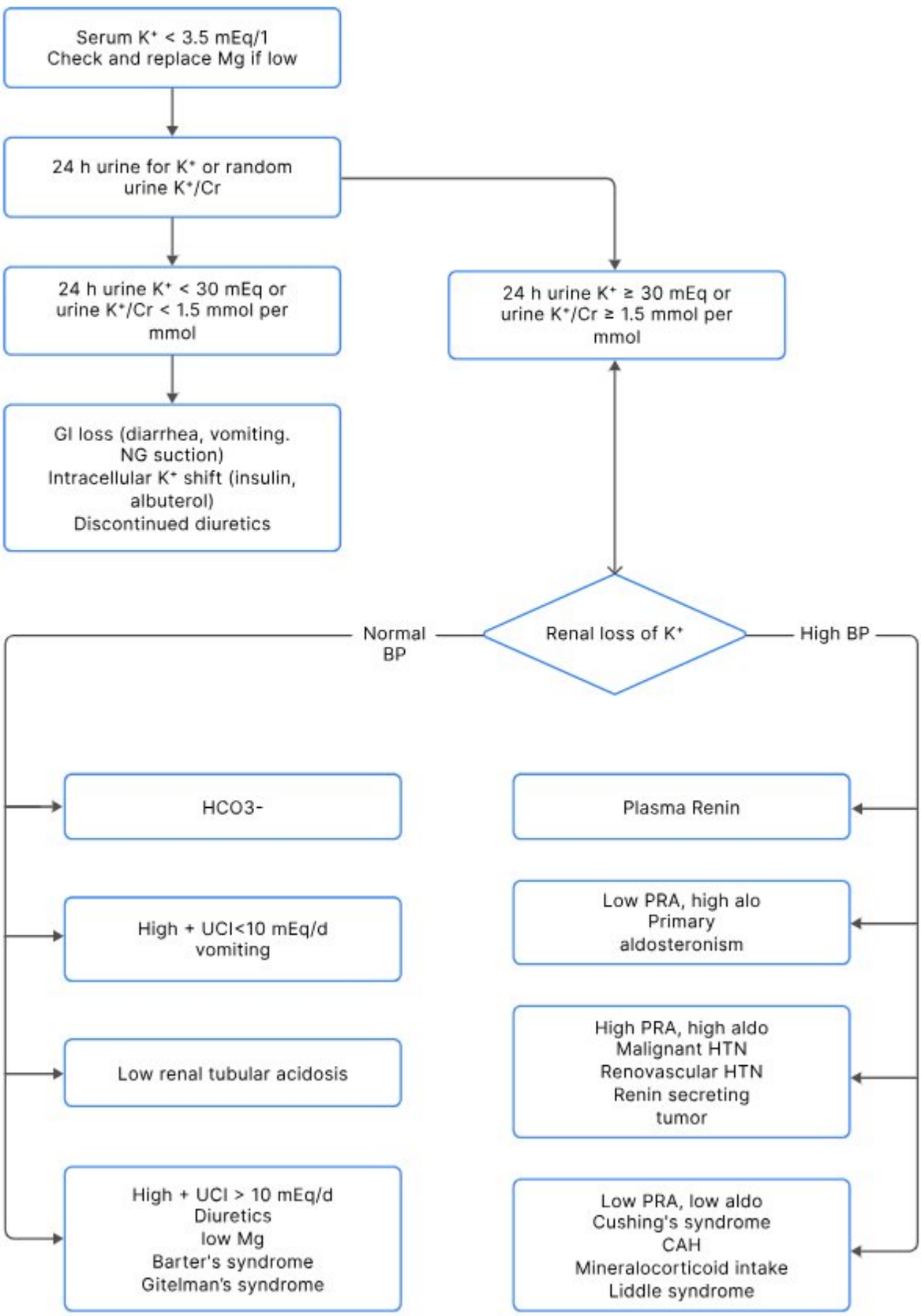
Critical values - act urgently (ECG + immediate management)

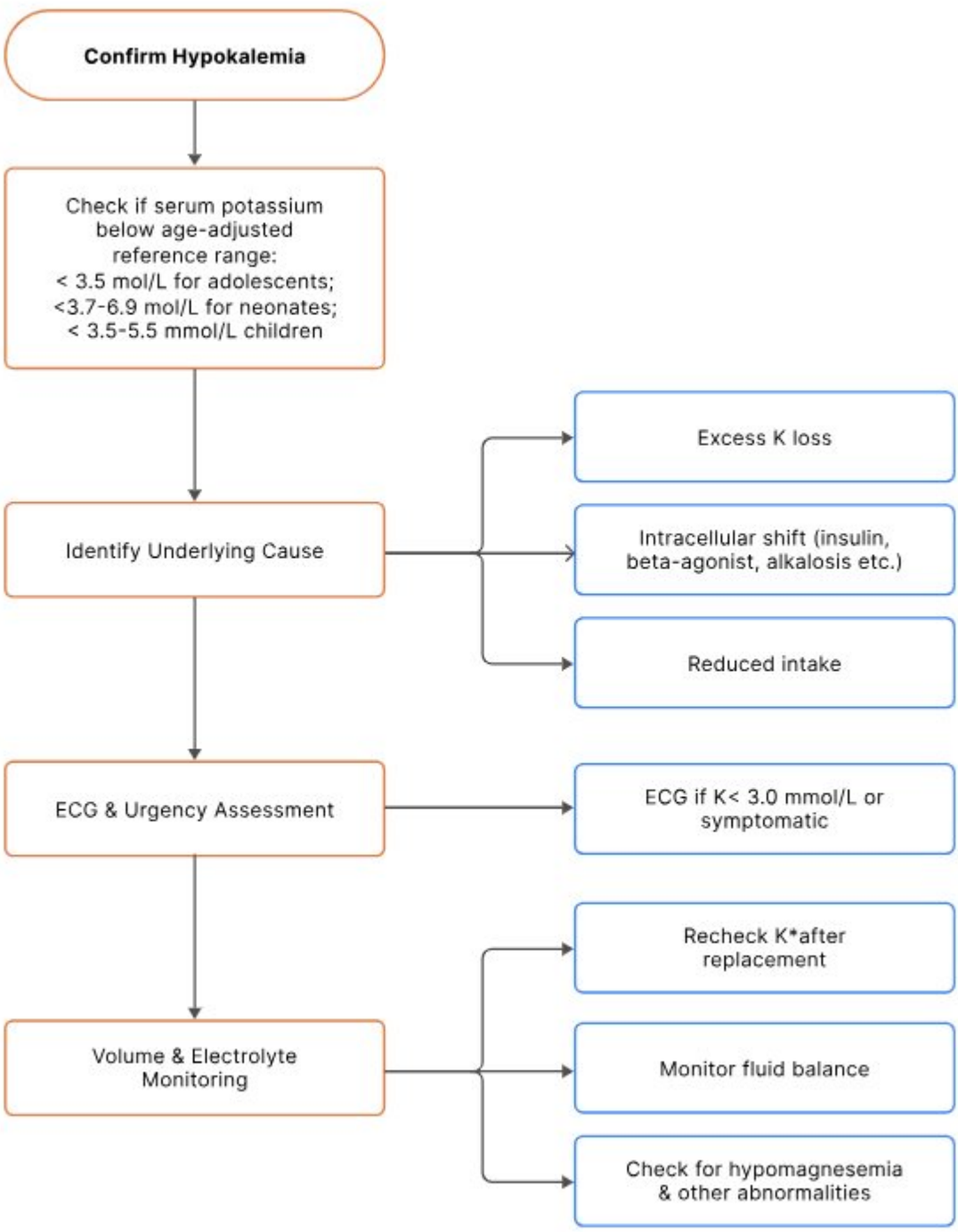
- **Hypokalemia (any age)**: **≤2.5 mmol/L is critical**, or **≤3.0 mmol/L** with symptoms, ECG changes/digitalis use/acute coronary syndrome/rapid ongoing losses.

Hyperkalemia:

- **Adults/adolescents/children**: **≥6.0 mmol/L** or any level with ECG changes/symptoms. **Neonates: ≥6.5 mmol/L** (≥7.0 mmol/L = extreme emergency).

DIAGNOSTIC ALGORITHM HYPOKALEMIA




Confirm Hypokalemia
Check if serum potassium below age-adjusted reference range:
< 3.5 mol/L for adolescents;
<3.7-6.9 mol/L for neonates;
< 3.5-5.5 mmol/L children
Identify Underlying Cause
Excess K loss
Intracellular shift (insulin, beta-agonist, alkalosis etc.)
Reduced intake
ECG & Urgency Assessment
ECG if K< 3.0 mmol/L or symptomatic
Volume & Electrolyte Monitoring
Recheck K*after replacement
Monitor fluid balance
Check for hypomagnesemia & other abnormalities

DIFFERENTIAL DIAGNOSIS

A systematic approach to the differential diagnosis of hypokalemia helps separate true potassium depletion from redistribution disorders or laboratory artifacts. This is crucial in pediatrics **because children can deteriorate quickly due to smaller potassium reserves, and chronic hypokalemia can affect growth, bone health, and neurodevelopment.**

Category	Examples	How it mimics hypokalemia	Clues to differentiate	Immediate action
Lab/ measurement pitfalls (not true hypokalemia)	Pseudohypokalemia with extreme leukocytosis (WBC >100×10⁹/L); acute alkalosis pH effect; sampling issues (prolonged tourniquet, warm transport, serum vs promptly processed plasma)	Spurious low K^+ reading	Repeat plasma K^+ in heparinized tube, process promptly; review ABG/serum bicarbonate for alkalosis; inspect hemolysis flags	Re-sample before treating aggressively
Neuromuscular weakness/ paralysis mimics	Guillain-Barré, myasthenia gravis, botulism; periodic paralysis (familial normo/ hyperkalemic), functional weakness; myopathies/ myositis, rhabdomyolysis, steroid or critical-illness myopathy/neuropathy	Weakness, paralysis, reduced reflexes	Neuro exam (pattern, fatigability), CK for myopathy/ rhabdomyolysis, nerve studies if needed; K^+ may be normal	Stabilize airway if needed; targeted neuro workup
Gastrointestinal ileus/ constipation mimics	Opioids, anticholinergics; hypothyroidism; bowel obstruction; sepsis-related ileus	Ileus/ constipation like K^+ deficiency	Drug history; thyroid tests; abdominal exam/ imaging; infection screen	Treat precipitant (e.g., stop culprit drug, manage obstruction)
Cardiac arrhythmia mimics	Ischemia, hypoxia; hypomagnesemia/ hypocalcemia; long-QT drugs (macrolides, fluoroquinolones); digoxin toxicity; hypothermia; intracranial events (↑ vagal tone)	Palpitations, brady/ ventricular ectopy, QT changes	ECG features not fitting classic hypokalemia; check Mg^{2+}/Ca^{2+}, troponin, temperature, drug list, digoxin level	Correct Mg^{2+}/Ca^{2+}, treat ischemia/hypoxia, stop QT-prolongers
Metabolic/ endocrine look-alikes	Hypomagnesemia; hypocalcemia; hypophosphatemia; adrenal insufficiency; thyrotoxic periodic paralysis (may precede low K^+)	Weakness, ECG changes, episodic paralysis	Full electrolyte panel; cortisol/ACTH if suspected; thyroid tests (TSH/FT4)	Replace deficient electrolytes; treat endocrine cause
Volume/status confounders	Dehydration, sepsis, heart failure	Dizziness, hypotension, fatigue	Vitals, lactate, fluid balance, ultrasound/ JVP; K^+ may be normal	Resuscitate per shock/ CHF protocols; then reassess K^+
ECG differentials for U waves / ST-T changes	Hypothermia, intracranial hemorrhage, ischemia, antiarrhythmics (class Ia/III)	U-like waves, ST-T abnormalities	Core temp, neuro exam/CT if indicated, medication review, troponin	Manage primary condition; avoid misattributing to K^+ alone

MANAGEMENT GOALS

1. Identify and correct the underlying cause
2. Restore serum potassium to safe range at an appropriate rate
3. Prevent recurrence

MANAGEMENT PRINCIPLES

1. Identify & treat cause

- Review meds (diuretics, laxatives, β_2-agonists, insulin, amphotericin B, aminoglycosides); address GI losses, endocrine disorders, and shifts.
- **Pediatrics: add gastroenteritis, NG suction, congenital tubulopathies; choose maintenance fluids carefully (avoid chloride-heavy plans when alkalosis risk).**

2. Assess severity & urgency

- Severe or symptomatic (arrhythmia, profound weakness, respiratory compromise, or K^+ <2.5 mmol/L) → urgent replacement with ECG monitoring.
- In children, rapid IV correction only for life-threatening instability.

3. Correct associated issues

- Replace Mg^{2+} first/alongside K^+; address acid-base disorders. Replace Mg^{2+} (e.g., magnesium sulfate IV) if low to allow K^+ to normalize. Mild hypomagnesemia (0.6-0.69 mmol/L) 12-24 mmol/day in divided doses. In moderate to severe (0<0.59 mmol/L) 1-2 grams (~8-16 mEq) IV infusion over 1-2 hours, repeated as needed based on severity and renal function. In critically low levels, up to 4-5 grams IV infusion slowly over several hours. **Caution:** Monitor renal function closely and adjust dose. Reassess magnesium levels 24 hours after administration to guide further therapy.

4. Potassium Replacement Strategy

- **Oral (preferred if stable/asymptomatic)**
- Adults: 40-100 mmol/day in divided doses (KCl/citrate).
- **Children: 1-2 mmol/kg/day in 3-4 doses (single dose = ≤2 mmol/kg).**
- **Intravenous (if severe, symptomatic, NPO, or ongoing high losses)**

- Adults: 20-40 mmol KCl in 1 L isotonic fluid over 4-6 h (titrate to ECG/labs).
- **Children: 0.3-0.5 mmol/kg/h; max concentration: 40 mmol/L peripheral, 60 mmol/L central; never undiluted.**

5. Monitoring

- Severe/IV therapy: continuous ECG; serum K^+ q2-4 h (adults) / q4-6 h (children).
- **Children: ECG if IV rate >0.25 mmol/kg/h or any cardiac concern.**

6. Safety

- Avoid under-correction (no rechecks) and over-correction (bolus dosing, rapid infusions without monitoring).
- **Double-check weight-based pediatric doses; use standardized protocols.**

7. Prevention

- Adjust or stop K^+-wasting drugs where possible.
- Diet: potassium-rich foods (bananas, citrus, potatoes, leafy greens).
- **Pediatrics: ensure adequate K^+ in maintenance fluids; address chronic nutrition.**

PHARMACOLOGICAL THERAPY

Pharmacological replacement is indicated when serum potassium is significantly reduced, when symptoms or ECG changes are present, or when ongoing losses are anticipated. Choice of route, dose, and preparation depends on severity, presence of symptoms, and ability to tolerate oral intake.

In both adults and children, magnesium deficiency should be corrected concurrently to optimize potassium repletion.

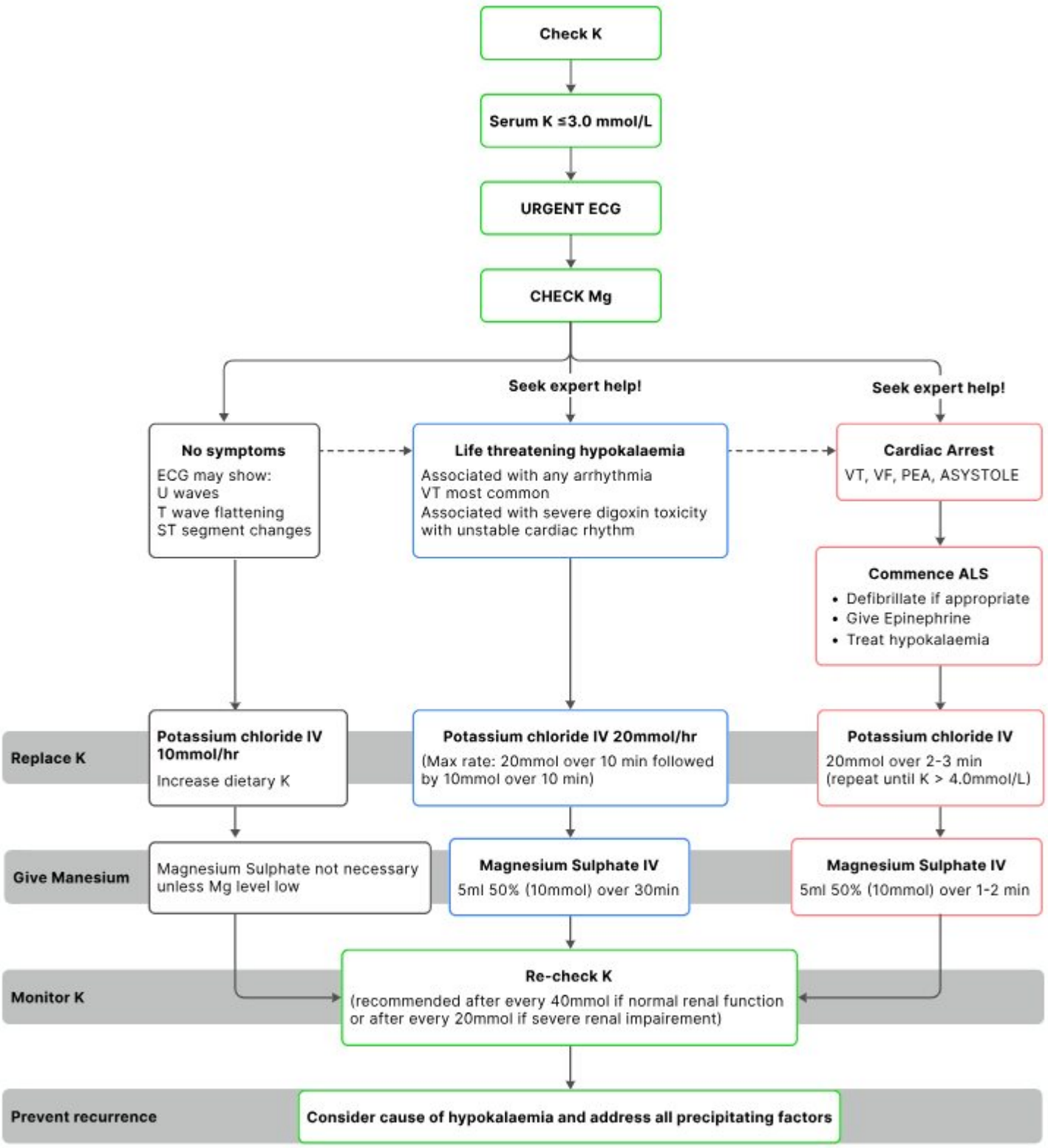
Agent / Formulation	Indication	Adults Dose & Route	Pediatrics Dose & Route	Duration	Cautions
Potassium Chloride (KCl) Oral	Mild-moderate hypokalemia without severe symptoms	40-100 mmol/ day in 2-4 divided doses	1-2 mmol/kg/day in 2-4 divided doses; max 5 mmol/kg/day; individual dose ≤20 mmol	Until K^+ ≥ target	GI irritation -give with food; avoid slow-release in severe cases

Potassium Chloride (KCl) IV - Peripheral Line	Severe (<2.5 mmol/L) or symptomatic hypokalemia, or unable to take orally	20-40 mmol in 1 L isotonic fluid; rate ≤10 mmol/h	0.3-0.5 mmol/kg/h; max conc 40 mmol/L for peripheral line; rate ≤0.5 mmol/kg/h	Until stable, then oral	ECG monitoring mandatory; risk phlebitis
Potassium Chloride (KCl) IV - Central Line	Life-threatening hypokalemia with arrhythmia or respiratory compromise	Up to 20 mmol/h via central line with continuous ECG	Max conc 60 mmol/L via central line; rate ≤1 mmol/kg/h (rarely exceeded in PICU)	Short-term until crisis resolves	Cardiac arrest risk if given rapidly; ICU only
Potassium Bicarbonate / Citrate (Oral)	Hypokalemia with metabolic acidosis or renal tubular acidosis	40-100 mmol/day	1-2 mmol/kg/day; max 5 mmol/kg/day	Chronic use for RTA	Avoid in severe renal failure; risk of alkalosis
Magnesium Sulfate	Coexisting hypomagnesemia impairing K^+ correction	2-4 g IV over 4-6 h	25-50 mg/kg/dose IV q12h; max 2 g/dose	Until Mg normal	Rapid IV can cause hypotension or cardiac depression
Spironolactone (Oral)	Hypokalemia from hyperaldosteronism or K-wasting diuretics	25-100 mg/day in 1-2 doses	1-3 mg/kg/day in 1-2 doses; max 100 mg/day	Long-term	Monitor for hyperkalemia; avoid in severe renal dysfunction
Amiloride (Oral)	Adjunct in renal K^+ wasting	5-10 mg/day	0.4-0.6 mg/kg/day; max 20 mg/day	Long-term	Avoid in GFR <10 mL/min; hyperkalemia risk
Triamterene (Oral)	Similar to amiloride	50-100 mg/day	2-4 mg/kg/day; max 300 mg/day	Long-term	Nephrolithiasis risk; monitor renal function

Key Pediatric Considerations

- Always dilute IV potassium and never give as an IV push.
- Peripheral IV concentrations >40 mmol/L increase risk of phlebitis- central access preferred for higher concentrations.
- Frequent ECG monitoring is required when IV rates exceed 0.3 mmol/kg/h or in presence of cardiac disease.
- In neonates, dosing and correction rates must be lower due to immature renal handling and higher arrhythmia risk.

MANAGEMENT ALGORITHM OF HYPOKALEMIA



K – potassium; Mg – magnesium; IV – intravenous; min – minute; ml – milliliter

NON-PHARMACOLOGICAL INTERVENTIONS (HYPOKALEMIA)

- Dietary Potassium: Promote potassium-rich foods (bananas, oranges, papaya, spinach, potatoes, tomatoes, lentils, beans, coconut water). Adapt advice to age and culture. Limit processed, high-sodium foods.
- Treat ongoing losses (diarrhea, vomiting, NG suction, uncontrolled diabetes). In pediatric gastroenteritis, use ORS with potassium.
- Prefer oral; reserve IV for severe/symptomatic or oral-intolerant patients. In children, switch to oral early.
- Keep daily weight, input-output records; weigh neonates/infants to nearest 10 g.
- Teach signs of low K^+ (weakness, palpitations, lethargy). Give symptom cards for school-age children with chronic K^+ loss.

ASSESSMENT OF RESPONSE

Ongoing monitoring during correction is essential to ensure safe and effective normalization of potassium levels while avoiding overcorrection.

Domain	What to check	Frequency / Targets	Action if targets not met
Laboratory monitoring	Serum K^+	IV severe cases: every 2-4 h until K^+ >3.0 mmol/L and symptoms improve. Oral/ mild-moderate: every 12-24 h until stable.	Rising >0.5 mmol/L/h: slow or pause infusion; recheck. No rise: reassess ongoing losses, acid-base, Mg^{2+}, and line patency; adjust dose/rate.
Clinical status	Muscle strength, ileus, vital signs, overall symptoms	With each lab draw and at bedside rounds	If weakness, ileus, or hypotension persists, increase monitoring and reconsider etiology (ongoing GI/renal losses, drugs).
Cardiac rhythm	Continuous ECG for K^+ <2.5 mmol/L or any IV replacement; telemetry for high-risk patients (cardiac disease, digoxin)	Continuous (severe) or per unit protocol	New arrhythmia or bradycardia - slow correction, check Mg^{2+}/Ca^{2+}, consider ICU transfer.
ECG features	Resolution of ST depression, flattened T, prominent U waves; reduction in ectopy	Review with each significant K^+ change	Worsening changes despite rising K^+ -evaluate Mg^{2+}, ischemia, drugs (QT-prolonging, digoxin).
Magnesium	Serum Mg^{2+}	At baseline, then at least daily during active correction (sooner if refractory)	If low, replace Mg^{2+} first/alongside K^+ to allow normalization.

Fluid & I/O	Urine output, stool/drain losses, net balance	Hourly I/O in severe cases; document losses mL-for-mL	Replace measured losses; avoid dextrose-only fluids without K^+ in prolonged IV therapy.
Safety checks	Infusion rate, solution concentration, IV access site	Each rate change and per nursing checks	Avoid peripheral KCl >40 mmol/L; use central access for higher concentrations; never IV push.
Transition to maintenance	Ability to take PO, diet, recurrence risk	When K^+ ≥3.5 mmol/L and stable	Switch to oral K^+ and dietary potassium; set follow-up labs and review meds that waste K^+.

Assessment of Response in Hypokalemia in Children

1. Laboratory monitoring

- Severe/IV therapy: check K^+ every 2-4 h until >3.0 mmol/L and symptoms improve.
- Mild-moderate/oral therapy: recheck every 12-24 h until stable.
- Neonates/infants: monitor every 2-3 h during IV replacement (small stores, rapid shifts).

2. Clinical monitoring

- Track return of muscle strength, normalization of heart rate, fewer arrhythmias, and ileus resolution.
- Continuous ECG for K^+ <2.5 mmol/L or any IV replacement.
- Watch pediatric cues: irritability, poor feeding, reduced spontaneous movement.

3. ECG assessment

- Expect resolution of ST depression, flattened T waves, prominent U waves.
- Children can show bradycardia or ventricular ectopy early, review ECG before rate changes.

4. Magnesium status

- Check Mg^{2+} and replace if low; uncorrected hypomagnesemia makes K^+ refractory.
- High vigilance in preterm neonates.

5. Adjustment of therapy

- K^+ rise >0.5 mmol/L/h: slow or pause infusion; recheck promptly.
- Inadequate rise: evaluate ongoing losses, acid-base, Mg^{2+}, and IV site; verify dose calculations.

- Always recalculate deficit by current weight at each step.

6. Transition to maintenance

- When K^+ >3.5 mmol/L and stable: switch to oral K^+ and dietary measures.
- For chronic renal/GI K^+- wasting: plan long-term maintenance with periodic labs to prevent relapse.

REVIEW, FOLLOW-UP & ADJUSTMENT

Table . Assessment of response: Hypokalemia

Component	Key Actions	Pediatric-Specific considerations
Laboratory Monitoring	■ Check serum K^+ every 2-4 hours during IV replacement in severe cases until >3.0 mmol/L and symptoms improve. ■ Mild/moderate cases on oral therapy: recheck every 12-24 hours. ■ Post-correction: daily checks for 48-72 hours.	■ Neonates/infants: monitor every 2-3 hours during IV therapy. ■ Watch for rebound hypokalemia within 12-24 hours after stopping replacement.
Clinical Monitoring	■ Assess muscle strength, heart rate, arrhythmias, bowel function. ■ Continuous ECG if K^+ <2.5 mmol/L or on IV replacement.	■ Look for irritability, feeding difficulty, reduced spontaneous movement.
ECG Assessment	■ Monitor for resolution of ST depression, flattened T waves, prominent U waves.	■ Bradycardia or ventricular ectopy may appear earlier -review ECG before adjusting infusion
Magnesium Status	■ Check Mg^{2+} and correct if low to aid potassium normalization.	■ Preterm neonates at high risk for hypomagnesemia.
Adjustment of Therapy	■ Slow/stop infusion if K^+ rises >0.5 mmol/L per hour. ■ If no improvement, reassess losses, acid-base status, magnesium, IV site.	■ Always recalculate potassium deficit based on current weight.
Transition to Maintenance	■ Switch to oral potassium once symptoms or ECG changes improve ■ Use dietary modification for maintenance.	■ Chronic renal or GI potassium-wasting disorders may require ongoing therapy.
Identify & Control Ongoing Losses	■ Reassess diuretics, GI losses, endocrine causes, diabetes control.	■ Address chronic diarrhea and renal tubular disorders.
Tapering Replacement	■ Switch from IV to oral when >3.0 mmol/L and symptoms improve. ■ Gradually reduce dose while monitoring.	■ Taper slower if cause of potassium loss is unresolved.
Step-Up Criteria	■ K^+ <2.5 mmol/L despite replacement. ■ Persistent arrhythmias/muscle weakness. ■ Severe ongoing losses/endocrine causes.	■ ECG changes persist despite K^+ >3.0 mmol/L. ■ Paralysis or feeding difficulty remains.

Step-Down Criteria	■ K^+ >3.5 mmol/L for 48 hours without IV therapy. ■ ECG normal. ■ Underlying cause controlled.	■ Caregiver education on potassium-rich foods and warning signs before discharge.
Outpatient Follow-Up	■ Recheck K^+ and Mg^{2+} 1-2 weeks post-discharge. ■ Review meds, diet, symptoms.	■ Growth, development, and renal function monitoring for recurrent cases.

PROGNOSIS AND PROGRESSION

When identified early and treated appropriately, most cases of hypokalemia resolve without lasting harm. Restoration of potassium to normal ranges reverses muscle weakness, ECG changes, and arrhythmia risk in the majority of patients. The overall prognosis depends on the underlying cause, the speed of recognition, and whether coexisting electrolyte imbalances (particularly magnesium deficiency) are addressed.

Severe untreated hypokalemia can lead to persistent cardiac arrhythmias, respiratory muscle paralysis, and sudden death. Chronic low-grade hypokalemia such as that seen in diuretic overuse or chronic diarrhea may contribute to hypertension, impaired glucose tolerance, and progressive renal damage.

In pediatric patients, prolonged hypokalemia can impair growth, delay puberty, and in infants, cause feeding difficulties, hypotonia, and developmental delays. Children with congenital renal tubular disorders, such as Bartter's or Gitelman syndromes, may experience recurrent episodes and require lifelong monitoring. Recurrent hypokalemia in early childhood also increases the risk of chronic kidney disease later in life.

With appropriate diagnosis, correction, and preventive measures, long-term outcomes are favorable. Education on avoiding harmful practices such as excessive diuretic use or unmonitored potassium supplementation reduces recurrence and improves quality of life.

REFERRAL LINKAGES

Escalate care from primary or secondary facilities to a higher-level or tertiary center when any of the following apply:

Trigger for escalation	Indicators	Where to refer	Immediate actions before transfer	What to send with the patient
Life-threatening manifestations	Persistent/recurrent ventricular arrhythmias, marked ECG changes, respiratory muscle weakness, paralysis despite initial correction	High-dependency unit (HDU) or ICU/tertiary center	Place on cardiac monitor, secure IV access, start controlled K^+ replacement per protocol, correct hypoxia, notify receiving team	Last 24-48 h vitals/ECG strips, medication list, timing/dose of all K^+/ Mg^{2+} given, allergies

Rapid progression or non-response	K^+ continuing to fall or not improving after 12-24 h of standard therapy	Tertiary internal medicine/ cardiology or ICU	Recheck K^+/Mg^{2+}/ABG, verify IV line patency, review diuretic/insulin/β_2-agonist exposure, stop contributors	Lab results trend, fluid/ I&O chart, infusion rates/ solutions, diuretic/insulin doses
Complex or unclear etiology	Suspected renal tubular disorder, adrenal pathology, rare genetic syndromes	Nephrology/ endocrinology service at tertiary center	Draw renin/aldosterone, urine K^+/Cl^-/Cr, Mg^{2+}, acid-base panel; stabilize	Summary of history/ exam, family history, preliminary endocrine/ renal labs, imaging if done
Multiple severe electrolyte derangements	Concurrent hypomagnesemia, hyponatremia, hypocalcemia needing coordinated correction	Tertiary center with multidisciplin ary support	Begin Mg^{2+} repletion, start ECG monitoring, avoid rapid K^+ boluses	Electrolyte panel trends, replacement schedule, comorbidity summary
Monitoring beyond facility capacity	Continuous telemetry required; frequent labs not feasible	HDU/ICU with telemetry and rapid lab turnaround	Arrange transport monitoring; maintain current controlled infusion	Referral note with monitoring needs, nursing notes, escalation plan
Need for high-risk IV therapy	KCl concentration >40 mmol/L peripheral, infusion >10-20 mmol/h, or need for central venous access	ICU/tertiary center capable of central line placement	Do **not** give IV push KCl; if possible, lower rate/ concentration until transfer	IV access details, line status, current concentration/rate, pump settings
Significant comorbidities complicating repletion	Advanced heart failure, CKD, severe liver disease, active myocardial ischemia	Tertiary center with cardiology/ nephrology/ hepatology	Optimize oxygenation, avoid fluid overload, tailor K^+ in isotonic carrier, consider diuretic adjustments	Comorbidity records, echocardiogram/renal data, medication reconciliation
Pediatric considerations (if applicable)	Infants/children with arrhythmias, paralysis, refractory hypokalemia, or suspected congenital tubulopathy	PICU/tertiary pediatric nephrology/ cardiology	Weight-based dosing only; continuous ECG; avoid concentrated peripheral infusions	Weight, growth data, dosing calculations, caregiver contact, feeding plan

Pediatric-specific triggers for referral include persistent hypokalemia despite adequate oral or IV replacement, suspicion of congenital renal tubular disorders (Bartter's, Gitelman), recurrent hypokalemic periodic paralysis, infants with hypokalemia and feeding difficulties or failure to thrive, and children presenting with life-threatening arrhythmias or severe muscle weakness requiring ventilatory support.

Detailed documentation of prior treatment, fluid balance, ECG findings, and laboratory results must accompany the patient to facilitate seamless transition and continuity of care.

COMPLICATIONS

If hypokalemia is not recognized and corrected promptly, it can lead to significant morbidity and mortality. Complications arise from potassium's essential role in maintaining cardiac excitability, skeletal muscle function, and acid-base balance

Category	Complications	Pediatric Considerations
Cardiac	■ Ventricular arrhythmias (VT, VF) and asystole ■ Worsening digitalis toxicity ■ AV block, sinus bradycardia, prolonged QT ■ Cardiac arrest in severe/rapid-onset cases	Arrhythmias develop faster due to smaller cardiac reserve and higher heart rates; continuous ECG monitoring essential during replacement
Neuromuscular	■ Generalized weakness leading to flaccid paralysis ■ Respiratory muscle weakness leading to hypoventilation/arrest ■ Rhabdomyolysis in prolonged/severe cases	May present as feeding difficulty, hypotonia in infants, refusal to walk in toddlers/older children; early neuromuscular assessment important
Metabolic	■ Worsening metabolic alkalosis ■ Impaired insulin secretion, give glucose intolerance/hyperglycemia ■ Aggravation of Mg^{2+} or Ca^{2+} imbalances	Higher risk of combined electrolyte disturbances due to limited reserves and higher metabolic rates
Renal	■ Impaired concentrating ability presenting as polyuria, polydipsia ■ Nephrogenic DI-like symptoms ■ Chronic hypokalemia in tubulointerstitial nephropathy	Prolonged hypokalemia from congenital tubular disorders increases risk of long-term kidney injury if not corrected early
Prognostic Impact	■ In critically ill: predicts longer hospital stay and higher mortality	Severe (<2.5 mmol/L) linked to higher respiratory failure rates and longer mechanical ventilation in PICU

PREVENTION & HEALTH PROMOTION

Preventing hypokalemia requires a proactive approach that addresses modifiable risk factors, ensures adequate dietary intake, and promotes early detection in high-risk individuals.

Domain	Key Actions	Pediatric Considerations
Dietary Measures	■ Encourage potassium-rich foods (bananas, oranges, avocados, spinach, sweet potatoes, legumes) ■ Provide routine dietary counseling for patients on chronic diuretics or with GI disorders; consider prophylactic supplementation	■ Age- and culture-appropriate meals: pureed fruits/vegetables for infants; smoothies/snacks for older children
Medication Safety	■ Regularly review medications (diuretics, corticosteroids, amphotericin B) and adjust/substitute if possible ■ Avoid unnecessary laxatives and high-dose β-agonists	■ Weight-based dosing; careful monitoring to prevent rapid K^+ shifts
Early Screening in High-Risk Groups	■ Monitor serum K^+ in hospitalized patients, those on K^+ wasting drugs, and with endocrine/renal disorders	■ Include potassium checks in routine visits for congenital tubular disorders, cystic fibrosis, chronic diarrhea

Education & Awareness	■ Teach patients/families signs of hypokalemia (weakness, palpitations, fatigue) and importance of early reporting	■ Educate parents to watch for subtle signs (poor feeding, irritability, unexplained falls, lethargy)
Hospital Protocols	■ Standardized K^+ monitoring/replacement guidelines with escalation pathways ■ Ensure ECG monitoring during IV replacement in moderate-to-severe cases	■ In neonatal/pediatric units: use infusion pumps with strict rate controls and double-check protocols
Community Health Promotion	■ Promote balanced nutrition programs in schools/communities - Provide educational materials in local languages	■ Integrate potassium assessment into community outreach and child nutrition programs, especially in resource-limited areas

PATIENT EDUCATION

Recognize symptoms early and report.

Follow diet and medication plans exactly.

Keep scheduled tests to prevent recurrence.

Know the symptoms
Adults: muscle weakness/cramps, palpitations, constipation, fatigue.
Children: **poor feeding, irritability, "floppy" tone, reduced activity, school performance changes.**

When to seek urgent care:
Sudden paralysis or severe weakness, severe palpitations or chest pain, fainting, breathing difficulty, seizures.
In children: any rapid decline during illness (vomiting/diarrhea, poor intake, lethargy).

Prevent recurrence
Address drivers: chronic diarrhea, high-dose diuretics, eating disorders.
Ensure adequate potassium in maintenance fluids (children) and a diet plan you can sustain.
Keep specialist follow-up for chronic pediatric cases; involve school health teams when relevant.

Do	Don't
Take potassium as prescribed. Use the correct formulation/ dilution; record doses.	Don't start or stop supplements, diuretics, laxatives, or herbal remedies without medical advice.
Use proper measuring tools for liquid meds in children.	Don't guess doses or use kitchen spoons for liquid meds.
Eat potassium-rich foods daily: bananas, citrus, spinach, tomatoes, sweet potatoes, beans/lentils, yogurt.	Don't follow restrictive diets or prolonged fasting that cut potassium intake.
Stay hydrated. During diarrhea, use oral rehydration solution (ORS); not plain water for infants/children.	Don't replace child fluid losses with plain water alone during illness.
Track and share: bring a simple log of symptoms, doses, and missed doses to visits (caregivers track for kids).	Don't skip follow-up tests because you "feel fine."
Attend all labs/ECGs on schedule, especially if on diuretics or other K^+-wasting drugs. Review medicines with your clinician; flag laxatives, diuretics, β_2-agonists, insulin changes, or herbal products. Coordinate care for children with chronic conditions (share the plan with school/daycare).	

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QUICK REFERENCE GUIDE (HYPERKALEMIA)

Hyperkalemia is less common than hypokalemia but occurs frequently in patients with renal impairment, critical illness, or those on potassium-sparing drugs. It affects all age groups, with neonates and patients with chronic kidney disease at highest risk. Severe untreated hyperkalemia can rapidly cause malignant arrhythmias and cardiac arrest, leading to high mortality, whereas timely intervention with ECG monitoring and structured therapy usually results in full recovery and prevention of recurrence.

Definition

Serum Hyperkalemia is a rise in serum potassium (K^+) above the age-specific upper limit >5.0 mmol/L in adults and children; in some neonates, values up to 6.0 mmol/L can be normal early after birth. Even moderate elevations can destabilize cardiac conduction and provoke life-threatening arrhythmias, so rapid recognition and structured management are essential. Acute hyperkalemia develops over hours to days and carries a higher arrhythmic risk; treat immediately. Chronic hyperkalemia evolves over weeks to months; still dangerous, but often allows broader etiologic work-up alongside controlled correction.

Causes, risk factors & triggers

- **Renal**: acute kidney injury, chronic kidney disease, type 4 renal tubular acidosis.
- **Drugs**: angiotensin-converting enzyme inhibitors (ACEi), angiotensin receptor blockers (ARB), potassium-sparing diuretics (spironolactone, eplerenone, amiloride), trimethoprim, nonsteroidal anti-inflammatory drugs (NSAIDs), heparin.
- **Shifts/load**: diabetic ketoacidosis (DKA), tumor lysis, rhabdomyolysis, severe hemolysis, metabolic acidosis, high-potassium enteral/parenteral intake.
- **Pediatrics**: preterm neonates (non-oliguric hyperkalemia), congenital adrenal disorders, obstructive uropathy.

Evaluation for diagnosis

- **Clinical features**: weakness, paresthesia, flaccid paralysis; palpitations, bradycardia, syncope; in infants, lethargy, poor feeding.
- **Physical exam**: vitals (bradycardia, hypotension), volume status, signs of underlying disease (dehydration, edema).
- **Laboratory investigations**: repeat plasma K^+ (avoid hemolysis), sodium, bicarbonate, creatinine/urea, glucose, calcium, magnesium, phosphate; venous/arterial blood gas for pH; creatine kinase (CK), lactate dehydrogenase (LDH), uric acid if lysis/rhabdomyolysis; urine for potassium if etiology unclear.

Confirmation of diagnosis

Validated elevated K^+ plus compatible context; do not delay treatment in severe cases or with ECG changes.

Classification / severity assessment criteria

Age-adjusted normal ranges (children)

- **Mild**: 5.1-5.9 mmol/L
- **Moderate**: 6.0-6.4 mmol/L
- **Severe: ≥6.5 mmol/L or any level with ECG changes/symptoms/rapid rise**
- **Neonates**: consider ≥6.5 mmol/L high-risk even if asymptomatic.

Critical values for hyperkalemia

Adults & children:

- ≥6.5 mmol/L = critical.
- ≥6.0 mmol/L with ECG changes, symptoms (weakness, paralysis), rapid rise, or renal failure = treat as critical.

Neonates:

- Normal can be up to ~6.0 mmol/L early after birth.
- ≥6.5 mmol/L = critical; ≥7.0 mmol/L = extreme emergency.

Differential Diagnosis

- **Pseudohyperkalemia:** hemolysis, prolonged tourniquet, fist clenching; thrombocytosis/leukocytosis (serum > plasma K^+).
- **ECG mimics**: hyperacute myocardial ischemia (tall T waves), hypothermia, drug effects (digoxin, class Ia/III).
- **Neuromuscular mimics**: Guillain-Barré syndrome, myasthenia gravis.

Management Goals & principles

1. Stabilize myocardium (protect the heart).
2. Shift K^+ intracellularly (buy time).
3. Remove K^+ from the body (definitive).
4. Treat the cause and prevent recurrence.
    - Start continuous ECG for K^+ ≥6.0 mmol/L (any age) or if symptomatic; **act on risk, not just the number.**

Approach to management

- **If ECG changes or severe hyperkalemia**: give **IV** calcium immediately, then initiate **insulin-glucose** and **beta-2 agonist**; plan **removal** (diuretic, binder, dialysis).
- Do not wait for ECG changes: ECG can be normal even with dangerous K^+ - treat based on clinical risk and potassium level.
- Calcium first when ECG risk is present (peaked T with conduction changes, PR/QRS widening, brady/ blocks, or sine-wave).
- During/around arrest ("special circumstances"): manage hyperkalemia with calcium, insulin-

-glucose (and bicarbonate when indicated), integrated into ALS pathways. 2025 (Keeps calcium for patients at highest arrhythmia risk).

- Continuous ECG during acute therapy; recheck after calcium (effect typically within minutes, repeat if QRS remains wide).
- If moderate/asymptomatic: confirm, stop K^+ sources/drugs, start shift therapy if rising or high-risk, consider binder, optimize renal excretion.

Non-Pharmacological interventions

- Stop external K^+ sources (diet/salt substitutes/IV fluids).
- Use oral rehydration solution appropriately; avoid high-K^+ drinks.
- Standardized checklists, premixed insulin-dextrose bundles, and weight-based pediatric charts reduce error where staffing/labs are limited.
- Early phone consult with higher-level center if dialysis access is limited.

Pharmacological therapy

1. **Cardiac membrane stabilization**
   - **Calcium gluconate 10%**: 10 mL IV over 2-5 min; repeat if QRS/ECG changes persist (monitor).
     - Pediatrics: 100 mg/kg (0.93 mL/kg) IV; max single adult dose; avoid extravasation.
   - **Calcium chloride 10%** (central line preferred): 5-10 mL IV if profound instability.
2. **Shift K^+ intracellularly** (temporizing)
   - **Regular insulin 10 units IV + dextrose 25 g IV** (e.g., 50 mL of 50% dextrose or 100 mL of 25% per local protocol); check glucose at 1, 2, and 3 h.
     - **Pediatrics: 0.1 units/kg insulin IV + 0.5 g/kg dextrose; strict glucose monitoring.**
   - **Nebulized salbutamol (albuterol): 10-20 mg** over 10-20 min; avoid in severe tachycardia/ischemia.
     - **Pediatrics: 2.5-5 mg (small child) up to 10-20 mg (adolescent) per protocol.**
   - **Sodium bicarbonate**: consider 50-100 mmol IV only if metabolic acidosis; avoid sodium overload in neonates/heart failure.
3. **Remove K^+ in case of severe or refractory hyperkalemia, impaired renal function, drug induced hyperkalemia if discontinuation is insufficient**
   - **Loop diuretic** (if volume replete and kidneys working): furosemide 20-40 mg IV, titrate; pediatrics 0.5-1 mg/kg.
   - **Potassium binders** (non-emergent adjuncts):
     - **Sodium zirconium cyclosilicate: 10 g PO** up to **TID** (short course).
     - **Patiromer: 8.4-25.2 g PO daily** (not for acute, onset hours).
     - **Avoid sodium polystyrene sulfonate** in neonates/ileus/post-op bowel (risk of necrosis).

- **Dialysis** (hemodialysis, peritoneal dialysis, or continuous renal replacement therapy): for refractory hyperkalemia, severe renal failure, or life-threatening ECG instability.

Assessment of response, follow-up, and treatment adjustment

- ECG response to calcium within minutes; repeat dose if QRS remains wide.
- Serum K^+: recheck at 1 h after shift therapy, then every 2-4 h until stable.
- If no improvement or rebound, repeat shift therapy (as appropriate), start/accelerate removal strategy, and reassess causes.
- Step-up when: K^+ ≥6.5 mmol/L, persistent ECG changes, anuria/AKI, ongoing tissue lysis, or failure of medical therapy → ICU/tertiary care.
- Step-down when: K^+ ≤5.0-5.5 mmol/L and stable 24-48 h, ECG normal, and cause controlled → transition to binder/diuretic plan as needed; arrange 1-2 week outpatient labs.

Referral (tiered)

- Primary to Secondary: K^+ ≥6.0-6.5 mmol/L, symptoms, or inability to monitor.
- Secondary to Tertiary/ICU: refractory hyperkalemia, dialysis need, complex etiologies (tumor lysis, adrenal crisis), pediatric neonates/preterms, or continuous monitoring beyond local capacity.

Complications

- Cardiac: bradycardia, atrioventricular block, ventricular tachycardia/fibrillation, asystole.
- Neuromuscular: ascending paralysis, respiratory failure.
- Treatment-related: hypoglycemia (insulin), hypocalcemia if mismanaged, volume overload, rebound hyperkalemia.

Objectives of Patient education & Instructions

- Know the signs (weakness, palpitations, irregular pulse) and seek urgent care early.
- Medication safety: never start/stop ACEi/ARB/spironolactone/OTC supplements without advice; keep an updated med list.
- Diet awareness: avoid salt substitutes and high-K^+ supplements unless prescribed; coordinate with a dietitian in children to maintain growth.
- Monitoring: keep all labs and ECGs; report illness, dehydration, or dose changes.
- Watch symptoms: weakness, limp limbs, palpitations, fast/slow/irregular heartbeat, unusual tiredness, confusion, sudden inability to move a limb. Seek urgent care if any appear.
- Emergency readiness: carry contacts, last lab printout, and action plan; for children after insulin therapy, watch for hypoglycemia and keep quick glucose on hand.

INTRODUCTION (HYPERKALEMIA)

Hyperkalemia is a dangerous rise in serum potassium that can trigger malignant arrhythmias and cardiac arrest. It results from impaired renal excretion, transcellular shifts, or excess intake seen commonly in chronic kidney disease, heart failure, diabetes, tumor lysis, and with renin-angiotensin-aldosterone system inhibitors; risk is high in ICU and oncology settings. Neonates (especially preterm/very low birth weight) may develop early non-oliguric hyperkalemia; children with chronic kidney disease, diabetic ketoacidosis, tumor lysis, adrenal disorders, or trimethoprim use are vulnerable. Management is immediate and structured: stabilize the myocardium with intravenous calcium, shift K^+ intracellularly (insulin-glucose, beta-2 agonist ± bicarbonate if acidotic), and remove K^+ (diuretics, binders with care, dialysis when indicated). Avoid delays while awaiting repeat labs, under-dosing insulin or bronchodilator, giving bicarbonate without acidosis, peripheral calcium chloride, and routine sodium polystyrene sulfonate in neonates or ileus/post-op bowel. Use ECG-guided, weight-based protocols with tight glucose monitoring and clear escalation pathways to reduce arrests, ICU admissions, and emergent dialysis.

SCOPE OF THE GUIDELINES

These guidelines address the recognition, diagnosis, and management of hyperkalemia in both adults and children, from initial detection through stabilization, definitive treatment, and follow-up. They are designed to ensure that care is standardized, safe, and effective across all levels of the healthcare system from primary care facilities in remote areas to advanced tertiary care hospitals while incorporating age-specific recommendations for neonates, infants, children, and adolescents.

Intended users

Physicians, nurses, and allied healthcare professionals at primary, secondary, and tertiary care levels.

Primary Care Level (At the community clinic or rural health post), the focus is on: Early recognition of clinical and ECG signs of hyperkalemia, rapid confirmation using point-of-care testing where available, immediate initiation of temporizing measures when trained personnel and basic resuscitation capacity are available. Safe referral triggers, clear criteria for urgent transfer when potassium >6.5 mmol/L, severe ECG changes, or inability to monitor closely.

Secondary Care Level (In Atoll/regional hospitals): Diagnostic work-up to determine underlying cause, initiate structured treatment protocols, continuous ECG and laboratory monitoring every 1-2 hours during acute correction.

Tertiary Care Level (In specialized hospitals with ICU, nephrology, and pediatric subspecialties): Advanced interventions such as hemodialysis, peritoneal dialysis, or CRRT (continuous renal replacement therapy) for refractory hyperkalemia, management of complex etiologies and integration of multidisciplinary care, including pediatric nephrology, cardiology, endocrinology, and intensive care specialists.

DEFINITION

Hyperkalemia is defined as a serum potassium concentration above the upper limit of the age-adjusted normal range. **In neonates during the first week of life, normal potassium levels are slightly higher due to immature renal potassium handling, with a reference range of 3.5-6.0 mmol/L. In infants, children, and adolescents, the normal range aligns more closely with adults, at 3.5-5.0 mmol/L.**

Clinically significant hyperkalemia is generally considered to be **≥6.0 mmol/L** at any age, or **any potassium level associated with ECG changes** (such as peaked T waves, widened QRS complexes, or sine-wave patterns) or symptoms (muscle weakness, paralysis, arrhythmias).

In pediatric patients, especially neonates, higher potassium thresholds for "normal" can mask early danger if values are interpreted without considering age-specific ranges. This makes age-adjusted interpretation essential for accurate diagnosis and timely intervention.

CAUSES, RISK FACTORS & TRIGGERS

While the broad etiologies are similar in adults and children, pediatric patients especially neonates and infants have unique physiological vulnerabilities and age-specific risk factors that must be recognized.

Category	Causes / Mechanisms	Pediatric-Specific Considerations
Reduced Potassium Excretion	■ Acute Kidney Injury (AKI) and Chronic Kidney Disease (CKD) from sepsis, dehydration, HUS, nephrotoxic drugs ■ Obstructive uropathy: posterior urethral valves, neurogenic bladder, urinary tract anomalies ■ Hypovolemia reducing distal sodium delivery and potassium secretion ■ Hypoaldosteronism: adrenal insufficiency, CAH, type IV RTA ■ Calcineurin inhibitor toxicity (tacrolimus, cyclosporine)	■ Congenital Adrenal Hyperplasia (CAH) is a common pediatric cause of hypoaldosteronism ■ Post-liver or kidney transplant children vulnerable to calcineurin inhibitor toxicity

Transcellular Potassium Shift	■ Metabolic acidosis (diarrheal illness, dehydration, RTA) - Insulin deficiency (DKA) ■ Tissue catabolism (rhabdomyolysis, burns, seizures) ■ Tumor lysis syndrome (TLS) after chemotherapy ■ Medications: beta-blockers, digoxin toxicity, succinylcholine ■ Severe hyperglycemia/hypertonicity (DKA, hyperosmolar states)	■ DKA in children: potassium shifts despite total body deficit ■ TLS risk higher in pediatric oncology due to aggressive tumor types
Excess Potassium Intake	■ Potassium-rich diet (bananas, coconut water, salt substitutes) - High-potassium feeds (special formulas, enteral feeds, TPN) without renal adjustment ■ Transfusions: stored blood with high potassium	■ Neonates and infants at higher risk due to small total body potassium pool ■ TPN formulas need strict potassium adjustment in preterm/VLBW infants
Medication-Related Causes	■ RAAS inhibitors: ACE inhibitors, ARBs, ARNIs ■ MRAs: spironolactone, eplerenone ■ Others: trimethoprim, heparin, NSAIDs, pentamidine, tacrolimus, cyclosporine	■ Dosing errors or lack of adjustment for renal impairment more common in children
Pediatric-Specific High-Risk Scenarios	■ Non-oliguric hyperkalemia of the newborn (NOHK) from immature Na^+/K^+-ATPase activity ■ Congenital adrenal hyperplasia (CAH) with aldosterone deficiency ■ Iatrogenic potassium overload (IV fluids, TPN, incorrect ORS prep) - Post-chemotherapy TLS releasing large potassium loads	■ NOHK often occurs in first 48 hours in preterm/VLBW infants ■ Salt-wasting crises in CAH need urgent recognition ■ Pediatric oncology patients require pre-chemotherapy TLS prophylaxis

EVALUATION FOR DIAGNOSIS

The priority is to **confirm the diagnosis, assess severity, and identify reversible causes** while initiating urgent treatment when indicated.

Step	What to do	Key details	Red flags / Act now
Clinical context	Symptoms and volume,Vitals, exam	Weakness, paresthesias, flaccid paralysis; hypotension, bradycardia	
Physical examination	Gauge risk and volume	Vitals (bradycardia, hypotension, tachypnea), volume status (hypo/hypervolemia), neuromuscular weakness/↓reflexes, signs: hyperpigmentation, dehydration, poor neonatal feeding, abdominal distension (ileus)	Shock, severe weakness, or progressive bradycardia - escalate care/ICU
Confirm true hyperkalemia	Repeat potassium if doubt	Avoid hemolysis; use proper venous/arterial technique; no fist clenching; avoid small-gauge needles/prolonged tourniquet	If repeat still high (≥6.0 mmol/L) - move to ECG + treatment immediately

Assess severity & need for immediate action	Start ECG monitoring and triage	Indications: K^+ ≥6.0 mmol/L, weakness/paresthesia/ paralysis, rapid rise, renal failure, metabolic acidosis Mild hyperkalemia may be asymptomatic, the risk of life-threatening cardiac conduction abnormalities increases sharply as potassium levels rise above the normal range, particularly when elevation occurs rapidly.	Treat during evaluation: IV calcium for ECG changes; prepare insulin-glucose and β_2-agonist
Focused history	Identify reversible causes	CKD, recent diarrhea/dehydration; DKA symptoms; drugs (ACEi/ARB, K-sparing diuretics, trimethoprim, NSAIDs, heparin), diet (salt substitutes, coconut water, ORS, formula), adrenal disease (CAH), tumor lysis, muscle injury/seizures	Ongoing source (drug/ infusion/feeds) - stop immediately
Laboratory workup (do not delay treatment if severe)	Define mechanism & co-derangements	Serum: K^+, Na^+, Cl^-, HCO_3^-, BUN/Cr, Ca^{2+}, Mg^{2+}, PO_4^{3-}; ABG/VBG (pH, HCO_3^-); glucose (DKA); CK/LDH (rhabdo); uric acid (tumor lysis); urine K^+/UA if unclear	Severe acidosis, rising K^+, or anuria - prepare for definitive removal (dialysis)
ECG evaluation	Stage electrical risk	Early: peaked T, short QT; Moderate: PR prolongation, wide QRS; Severe: sine-wave, VF, asystole. **Children can deteriorate rapidly; ECG changes may appear late, so absence of ECG findings does not exclude risk.**	Any ECG change + hyperkalemia - IV calcium now; absence of changes does not exclude risk (children can deteriorate quickly)

CONFIRMATION OF DIAGNOSIS

Confirm True Hyperkalemia

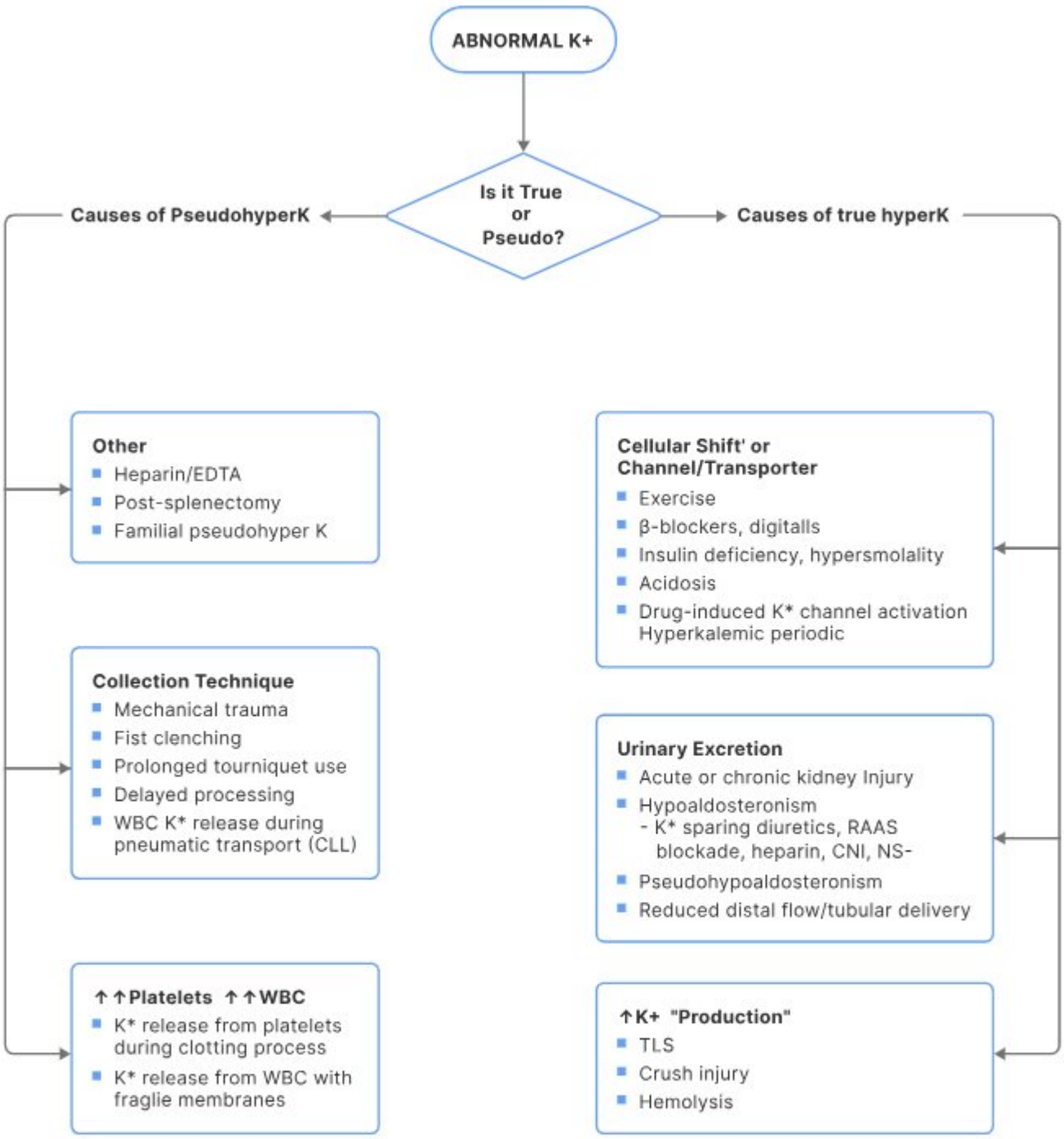
Before initiating definitive treatment, it is essential to confirm true hyperkalemia, as falsely elevated potassium levels (pseudohyperkalemia) are common, especially in pediatric samples. However, if the patient has severe symptoms or ECG changes, treatment should begin immediately while awaiting confirmation.

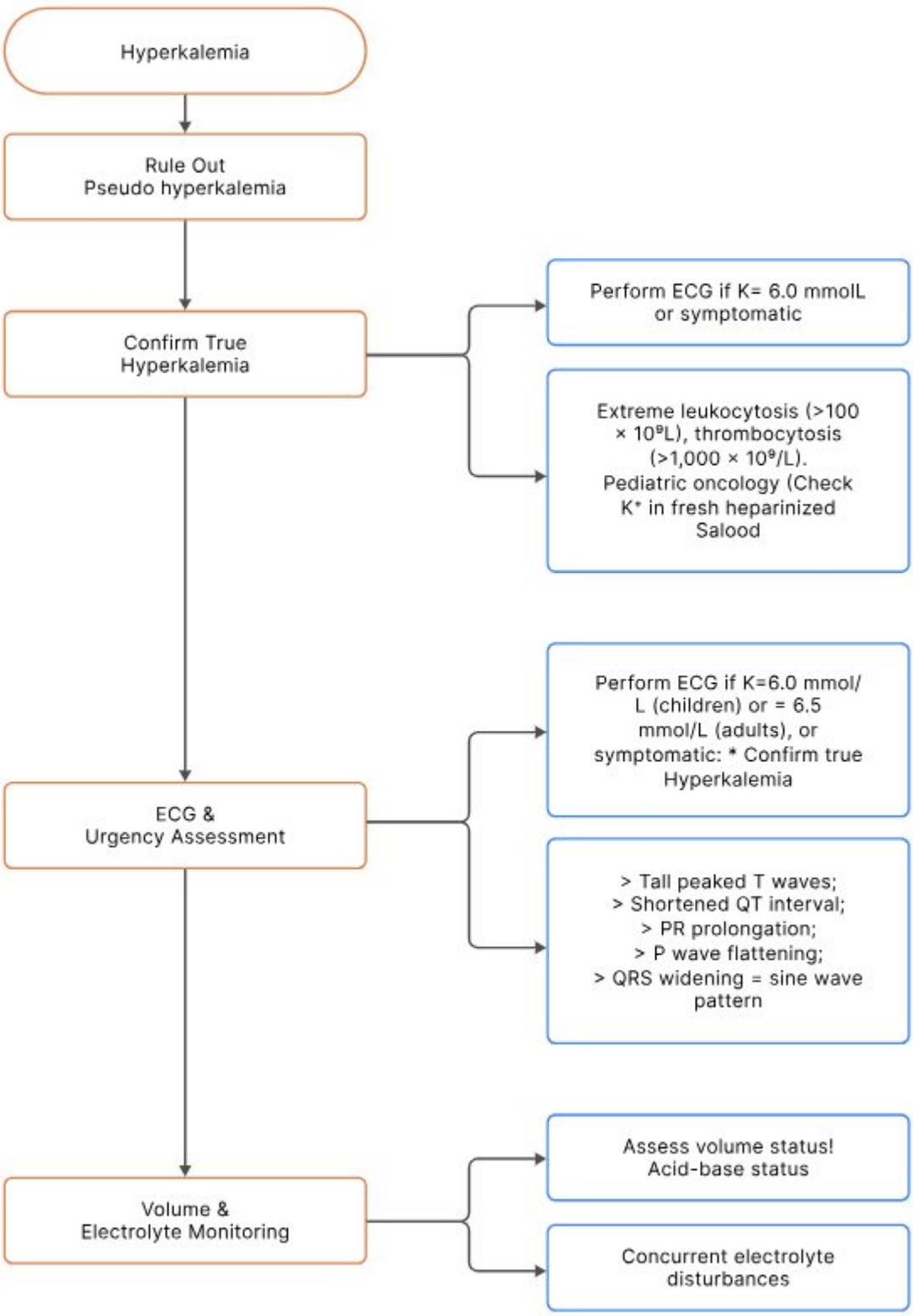
Causes of Pseudohyperkalemia:

sample hemolysis (heel-prick, small needles, delayed processing), prolonged tourniquet/ fist clenching, extreme leukocytosis (>100 × 10^9/L), thrombocytosis (>1,000 × 10^9/L). In pediatric oncology, check plasma potassium on fresh heparinized blood.

If suspected, repeat venous/arterial sample, avoid hemolysis, process promptly with blood gas analyzer or direct ISE.

DIAGNOSIS ALGORITHM OF HYPERKALEMIA




Hyperkalemia
Rule Out
Pseudo hyperkalemia
Confirm True
Hyperkalemia
Perform ECG if K= 6.0 mmolL
or symptomatic
Extreme leukocytosis (>100
× 10⁹L), thrombocytosis
(>1,000 × 10⁹/L).
Pediatric oncology (Check
K⁺ in fresh heparinized
Salood
ECG &
Urgency Assessment
Perform ECG if K=6.0 mmol/
L (children) or = 6.5
mmol/L (adults), or
symptomatic: * Confirm true
Hyperkalemia
> Tall peaked T waves;
> Shortened QT interval;
> PR prolongation;
> P wave flattening;
> QRS widening = sine wave
pattern
Volume &
Electrolyte Monitoring
Assess volume status!
Acid-base status
Concurrent electrolyte
disturbances

CLASSIFICATION AND SEVERITY ASSESSMENT

The severity of hyperkalemia is determined by serum potassium concentration, rate of rise, and presence of clinical or ECG manifestations. **In pediatric patients, age-specific normal ranges must be applied to avoid over- or under-treatment.**

Classification Based on Serum Potassium and Clinical Risk

Severity	Adults	Pediatric (Age-Specific Upper Limit)	Common Clinical Features	ECG Changes
Normal	3.5 to 5.0 mmol/L	**Neonates** (first week of life): 3.5-6.0 mmol/L **Infants**, **children**, **adolescents**: 3.5-5.0 mmol/L		
Mild	5.1-5.9 mmol/L	Neonates: 6.1-6.9 mmol/L Older children: 5.1-5.9 mmol/L	Often asymptomatic; may have nonspecific fatigue or irritability in children	Usually none
Moderate	6.0-6.9 mmol/L	Neonates: 7.0-7.4 mmol/L Older children: 6.0-6.5 mmol/L	Muscle weakness, decreased reflexes; feeding difficulty in infants	Peaked T waves, shortened QT interval
Severe	≥7.0 mmol/L or any level with ECG changes	Neonates: ≥7.5 mmol/L Older children: ≥6.6 mmol/L	Flaccid paralysis, bradycardia, arrhythmias, apnea, altered consciousness	Widened QRS, sine-wave, ventricular fibrillation/ asystole

Critical values for hyperkalemia

Adults & children:

- ≥6.5 mmol/L = critical.
- ≥6.0 mmol/L with ECG changes, symptoms (weakness, paralysis), rapid rise, or renal failure = treat as critical.

Neonates:

- Normal can be up to ~6.0 mmol/L early after birth.
- ≥6.5 mmol/L = critical; ≥7.0 mmol/L = extreme emergency.

Clinical and ECG-Based Urgency

- Any potassium level with ECG changes should be treated as severe hyperkalemia, regardless of the absolute value.
- Rapidly rising potassium (e.g., in tumor lysis, rhabdomyolysis, massive transfusion) warrants urgent intervention before ECG changes develop.

- In neonates and preterm infants, even moderate increases can precipitate cardiac arrest due to immature cardiac conduction reserve.

Pediatric-Specific Notes

- In preterm and VLBW infants, potassium handling is immature, hyperkalemia can progress in hours, not days.
- Feeding difficulties, apnea, or subtle irritability may precede overt ECG changes in young infants.
- Children with congenital adrenal hyperplasia, acute kidney injury, or severe acidosis require particularly close monitoring.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis is vital in hyperkalemia to distinguish true from false elevations, identify the underlying cause, and guide targeted treatment. It prevents unnecessary or harmful interventions, ensures life-threatening causes like adrenal crisis or tumor lysis are not missed, and allows tailored management strategies especially important in children, where age-specific causes and narrower safety margins make accurate diagnosis critical.

Category	Mimic	Why it looks like hyperkalemia	How to differentiate / What to check	Immediate action
Lab/ measurement artifact	Hemolysis, prolonged tourniquet, fist-clenching, warm transport	False high K^+ on lab	Repeat plasma K^+ (heparinized tube), prompt processing; hemolysis index	Re-sample before treating
Pseudohyperkalemia (in vitro release)	Extreme thrombocytosis or leukocytosis	Serum K^+> plasma K^+	Compare serum vs plasma K^+; check platelet/WBC counts	Use plasma value for decisions
ECG mimics - peaked T / wide QRS	Early STEMI (hyperacute T), hyperacute ischemia, hyperthermia	Tall T waves, repolarization changes	Troponin, regional ST changes, clinical context	Treat ACS if suspected; don't rely on K^+ alone
ECG mimics - brady/blocks	Hypothermia, increased vagal tone, AV-nodal drugs (β-blocker, CCB), digoxin	Bradycardia, AV block like severe K^+	Core temp, drug history/ levels, Osborne waves (hypothermia)	Rewarm/antidote as appropriate
Neuromuscular weakness mimics	Guillain-Barré, myasthenia gravis, botulism, critical-illness myopathy	Flaccid weakness/ paralysis	Neuro exam, reflexes, respiratory metrics, antibody/toxin tests	Airway protection; neuro workup

Periodic paralysis (non-hyperkalemic)	Familial normokalemic/ hypokalemic variants, thyrotoxic periodic paralysis (early)	Episodic paralysis	Serum K^+ not elevated; thyroid tests; triggers (carb/β-agonist)	Treat thyrotoxicosis; careful K^+ use
Metabolic/ electrolyte look-alikes	Hypocalcemia, hypomagnesemia, severe acidosis/alkalosis	Arrhythmias/QT changes similar to K^+ disorders	Full electrolytes, ABG	Replace Ca^{2+}/ Mg^{2+}; correct pH
Medication/ digitalis effects	Digoxin effect/toxicity, class Ia/III antiarrhythmics	ST-T changes, arrhythmias	Drug levels, characteristic "scooped" ST, context	Hold culprit; antidote/support as indicated
Renal/uremic ECG changes (without high K^+)	Advanced uremia with normal K^+	Conduction abnormalities	Actual K^+ normal; uremic symptoms; BUN/Cr high	Treat uremia/ volume; don't give unnecessary K^+ therapy
Autonomic/ cardiac conduction disorders	Sick sinus, high-grade AV block	Brady/blocks resembling severe hyperkalemia	Holter/telemetry, response to atropine	ACLS pathway; pacer if needed

MANAGEMENT GOALS & PRIORITIES

1. Stabilize the cardiac membrane
2. Shift potassium into cells
3. Remove potassium from the body
4. Treat the underlying cause
5. Prevent recurrence

Pediatric patients especially neonates require strict weight-based dosing, close glucose monitoring, and vigilance for rapid shifts due to smaller total body potassium reserves.

MANAGEMENT PRINCIPLES

1. Immediate Cardiac Protection

- Begin continuous ECG monitoring if K^+ ≥6.0 mmol/L in children, ≥6.5 mmol/L in adults, or earlier if symptomatic.

- Give IV calcium gluconate for ECG changes or severe hyperkalemia (weight-adjusted in pediatrics). This stabilizes myocardium but does not lower potassium.

2. Rapid Intracellular Shift

- Insulin - glucose infusion (adjust glucose to avoid hypoglycemia; higher risk in infants).
- Nebulized salbutamol as adjunct; monitor for tachycardia in neonates.
- Sodium bicarbonate if metabolic acidosis (arterial pH falls below 7.2, particularly when accompanied by serum bicarbonate <10 mEq/L or hemodynamic instability) present (avoid sodium overload in children).

3. Potassium Removal in case of severe or refractory hyperkalemia, impaired renal function, drug induced hyperkalemia if discontinuation is insufficient

- Loop diuretics if volume status allows.
- Potassium-binding resins with caution in children; avoid in neonates due to bowel necrosis risk.
- Dialysis for refractory/severe cases or renal failure.

4. Treat Underlying Cause

- Stop potassium-sparing drugs, treat adrenal insufficiency, manage rhabdomyolysis/tumor lysis/DKA, adjust TPN or feeds in neonates.

5. Monitoring

- Recheck serum K^+ within 1 hour in life-threatening cases, then every 2-4 hours until stable.
- Continue ECG monitoring until K^+ is within the safe range and cause addressed. **Pediatrics: Apply continuous ECG monitoring at the same threshold, but be aware that even mild hyperkalemia (≥5.5 mmol/L) in neonates can cause arrhythmias, monitor earlier in this group.**
- After insulin: glucose checks hourly for 3 h (longer in pediatrics).

6. Pediatric-Specific Considerations

- Maintain age-appropriate normal K^+: neonates 3.5-6.0 mmol/L; infants/children/adolescents 3.5-5.0 mmol/L.
- Monitor earlier in neonates (even ≥5.5 mmol/L may cause arrhythmias).
- In preterm neonates, consider benign non-oliguric hyperkalemia if asymptomatic before aggressive therapy.
- Avoid both under-correction and over-correction. Always use weight-based dosing
- Ensure glucose supplementation during insulin therapy to prevent hypoglycemia.

PHARMACOLOGICAL THERAPY

Pharmacologic management is selected based on severity, presence of ECG changes, and the underlying cause. Pediatric treatment always requires weight-based dosing, careful fluid selection, and continuous monitoring to avoid iatrogenic hypoglycemia, hypocalcemia, or rapid potassium shifts.

Drug/Intervention	Indication	Adult Dose	Pediatric Dose	Onset / Duration	Cautions & Notes
Calcium Gluconate 10%	ECG changes or K^+ ≥6.5 mmol/L (adult) / ≥6.0 mmol/L (pediatric) with ECG changes	10 mL IV over 2-5 min (≈ 1 g) 30 ml IV when ECG changes present over 5 minutes	0.5 mL/kg IV (max 10 mL) over 2-5 min with ECG monitoring. May repeat after 5-10 minutes if ECG changes persist.	Onset 1-3 min; lasts 30-60 min	Stabilizes myocardium; does not lower K^+. Avoid in digoxin toxicity unless life-threatening arrhythmia. Monitor for bradycardia. Repeat if ECG changes persist.
Calcium Chloride 10%	Avoid peripheral administration if possible. Consider when profound instability as in severe ECG changes, need for more elemental calcium	5 mL IV over 2-5 min (≈ 500 mg) via central line; max 10 mL	0.2 mL/kg IV via central line	Onset 1-3 min; lasts 30-60 min	3× more elemental calcium than gluconate; vesicant -use central line to avoid tissue necrosis.
Regular Insulin + Glucose	Rapid K^+ shift into cells	10 units regular insulin IV + 25-50 g glucose IV (50 mL of 50% dextrose)	0.1 units/kg regular insulin IV (max 10 units) + 0.5-1 g/kg glucose IV (D10W 5 ml/kg; D25W 2ml/kg); Avoid D50W in young children; if used in adolescents, give carefully via large bore	Onset 15-30 min; lasts 4-6 h	Risk of hypoglycemia, especially in children, check glucose every 30 min for at least 4h. Reduce glucose dose if baseline hyperglycemia.

Nebulized Salbutamol (Albuterol)	Rapid K^+ shift into cells; adjunct to insulin/glucose	10-20 mg over 10 min via nebulizer	2.5 mg (<25 kg) or 5 mg (≥25 kg) via nebulizer over 10 min; use clinical judgement	Onset 30 min; lasts 2-4 h lowers K by ~0.5-1.0 mmol/L	Less effective in beta-blocker use; may cause tachycardia. Avoid as sole therapy in severe hyperkalemia.
Sodium Bicarbonate (8.4%)	Hyperkalemia with metabolic acidosis (pH <7.1) in select patient (non-responder	50 mEq IV over 5 min	1-2 mEq/kg IV over 10-20 min; consider infusion if ongoing acidosis	Onset 30-60 min; variable	Not effective if no acidosis; risk of volume overload and hypernatremia.
Loop Diuretics (Furosemide) if adequate renal function & output	Potassium removal in volume-replete patients	20-40 mg IV	1 mg/kg IV (max 40 mg); titrate to urine output; add isotonic fluids if hypovolemic	Onset 15 min; lasts 4-6 h	Requires adequate renal function; monitor volume status and electrolytes
Cation Exchange Resin (Sodium Polystyrene Sulfonate-SPS)	Slow K^+ removal for chronic hyperkalemia	15-30 g PO or PR	1 g/kg PO or PR, if no ileus (max 15 g/dose)	Onset hours; lasts until excretion	Avoid in bowel obstruction or neonates at risk for NEC; **do not use a sole emergent therapy**.
Patiromer (Sodium zirconium cyclosilicate (SZC)	Chronic K^+ control in CKD/heart failure; effective in adults;	8.4-25.2 g PO daily	Safety/efficacy not established <18 years; Pediatric approvals & dosing vary by jurisdiction	Onset 7 h; not for emergencies	Separate from other oral meds by ≥3 h; Consult pediatric nephrology before use in children
Hemodialysis	Severe/refractory hyperkalemia or renal failure (AKI/ CKD); life threatening ECG changes	Intermittent hemodialysis (IHD) or continuous renal replacement therapy (CRRT) in hemodynamically unstable patients	Intermittent hemodialysis (IHD) or continuous renal replacement therapy (CRRT) in hemodynamically unstable patients	Immediate once started	Most effective and definitive method for K^+ removal. Consider peritoneal dialysis in pediatric patients if HD unavailable.

Key Pediatric Considerations

- Always double-check concentrations and doses with a second clinician before administration.
- Use diluted glucose for infants to avoid osmotic injury (e.g., D10W or D25W instead of D50W).
- Neonates and young infants have a narrow therapeutic margin - frequent ECG and potassium checks (every 30-60 min during acute therapy).
- Avoid sodium polystyrene sulfonate in very low birth weight infants due to intestinal necrosis risk.

ECG changes in hyperkalemia & management (ERC (European Resuscitation Council)

Serum K^+ (approx.)	ECG changes	Notes / actions
5.5-6.5 mmol/L	Tall, peaked T waves (narrow base), shortened QT	If symptomatic or high-risk: monitor continuously, prepare calcium.
6.5-7.0 mmol/L	PR prolongation, QRS widening, T waves still peaked	Give IV calcium if any conduction change or symptoms. Start insulin-glucose ± β_2-agonist.
≥7.0 mmol/L	P-wave flattening/ loss, further QRS widening	Treat as imminent arrest: calcium, shift, remove K^+; consider dialysis early.
Critical	Sine-wave morphology → ventricular fibrillation/ asystole	Immediate IV calcium, full Advanced Life Support protocol, definitive K^+ removal.

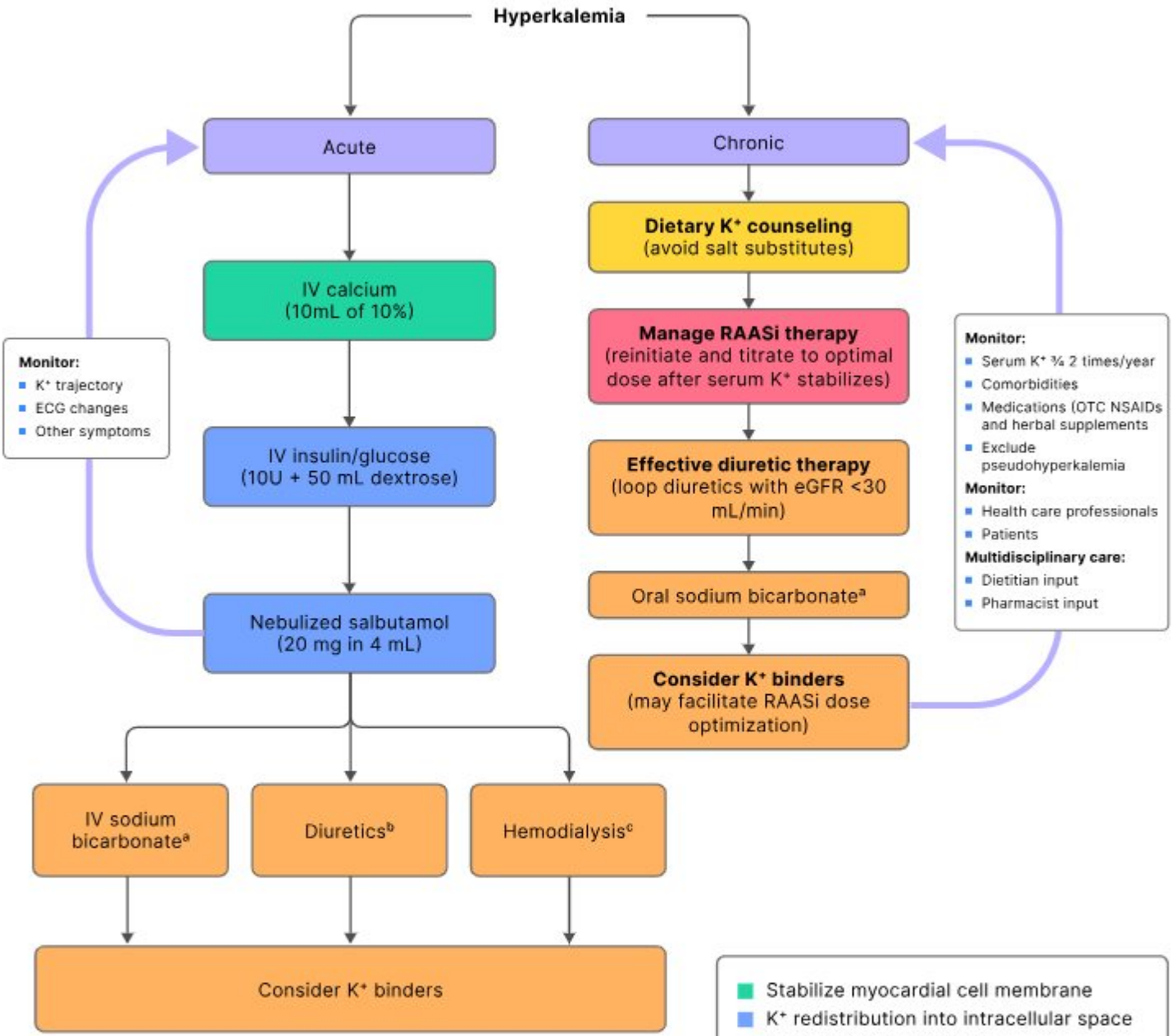
- Do not wait for ECG changes: ECG can be normal even with dangerous K^+ - treat based on clinical risk and potassium level.
- Calcium first when ECG risk is present (peaked T with conduction changes, PR/QRS widening, brady/blocks, or sine-wave).
- During/around arrest ("special circumstances"): manage hyperkalemia with calcium, insulin-glucose (and bicarbonate when indicated), integrated into ALS pathways. 2025 (Keeps calcium for patients at highest arrhythmia risk).
- Continuous ECG during acute therapy; recheck after calcium (effect typically within minutes, repeat if QRS remains wide).

Note:

- Hyperacute STEMI can mimic peaked T waves; correlate with symptoms and troponin.
- Hypocalcemia/hypomagnesemia worsen conduction abnormalities - correct alongside K^+ management.
- In neonates, arrhythmias may occur at lower K^+ than adults - monitor earlier.

KDIGO (Kidney Disease: Improving Global Outcomes)

- Monitor early and continuously when K^+ is high, cardiac monitoring and 12-lead ECG for K^+ >6.0 mmol/L; ECG can be atypical or even normal, so don't wait for changes.
- Structured ED approach: protect the heart if unstable, shift K^+ into cells (insulin-glucose; add beta-2 agonist; bicarbonate only if acidotic), start removal (diuretic, newer binders where appropriate, dialysis if refractory).
- Chronic care context (CKD 2024 guideline): manage contributors (RAAS inhibitors with monitoring, diet, binders) and set systems for rapid evaluation/treatment when acute hyperkalemia occurs.



[a]In patients with metabolic acidosis
[b]In patients with hypervolemia (nonoliguric)
[c]In patients with oliguria or ESRD

TREATMENT OF UNDERLYING CAUSES

Effective hyperkalemia management requires prompt correction of the elevated potassium and targeted treatment of the precipitating cause to prevent recurrence. In pediatric patients, early identification and stabilization are crucial because potassium derangements can rapidly progress to life-threatening arrhythmias.

1. Diabetic Ketoacidosis (DKA)

In patients presenting with polyuria, polydipsia, weight loss, abdominal pain, vomiting, dehydration, and altered sensorium. Kussmaul respiration and a fruity odor on the breath are classic signs of acidosis. Diagnosis is based on blood glucose >200 mg/dL, venous pH <7.3, or serum bicarbonate, ketonemia or ketonuria.

- **Adults**: Initiate insulin infusion only after adequate initial fluid resuscitation to avoid sudden potassium shifts leading to hypokalemia. Follow adult DKA protocol for fluid, insulin, and electrolyte correction.
- **Pediatrics**: Begin insulin infusion (0.05-0.1 units/kg/h) after at least 1-2 hours of fluid therapy and once serum potassium is confirmed ≥3.3 mmol/L. Pediatric DKA protocols emphasize gradual osmolality correction to avoid cerebral edema. Potassium replacement is started once urine output is adequate and serum potassium <5.5 mmol/L.

2. Adrenal Crisis

- **Adults**: Immediate IV hydrocortisone 100 mg bolus, followed by 200 mg over 24 h infusion or divided doses. Correct dehydration, hyponatremia, and hypoglycemia as per adrenal insufficiency protocol.
- **Pediatrics**: Hydrocortisone 50 mg/m² IV bolus, then 50-100 mg/m²/day in divided doses. Maintenance fluids with dextrose-saline are given to correct hypoglycemia and hyponatremia. Monitor serum potassium and sodium closely, as correction of adrenal insufficiency will rapidly improve hyperkalemia.

3. Tumor Lysis Syndrome (TLS)

- **Adults**: Aggressive IV hydration (2-3 L/m²/day), rasburicase for uric acid control, and early dialysis if potassium or creatinine rises despite supportive therapy.
- **Pediatrics**: Hydration at 2.5-3 L/m²/day with strict input-output monitoring. Rasburicase is preferred over allopurinol for rapid uric acid reduction, especially in high-risk malignancies. Dialysis should be considered early in infants and small children as they deteriorate faster with fluid overload.

4. Rhabdomyolysis

- **Adults**: Rapid isotonic saline infusion to maintain urine output >200 mL/h; correct hyperkalemia, metabolic acidosis, and hypocalcemia as needed.
- **Pediatrics**: Aggressive hydration with isotonic saline (up to 3 mL/kg/h) aiming for urine output ≥2 mL/kg/h. Monitor for fluid overload in infants. Alkalinization of urine with bicarbonate may be considered if severe myoglobinuria and metabolic acidosis are present.

5. Medication Review and Cessation

- **Adults**: Discontinue potassium-raising medications (ACEi/ARB, MRAs, NSAIDs, trimethoprim, beta-blockers, and potassium supplements) until potassium is normalized and the underlying cause addressed.
- **Pediatrics**: Immediately stop ACE inhibitors, ARBs, MRAs (spironolactone, eplerenone), NSAIDs, trimethoprim, beta-blockers, and potassium-containing supplements or parenteral nutrition potassium additives, high-dose penicillin G potassium salt, and certain chemotherapy regimens. For children on life-sustaining medications like calcineurin inhibitors, adjust the dose under specialist supervision.

In all cases, treating the underlying condition shortens the duration of hyperkalemia, prevents recurrence, and often reduces the need for aggressive potassium-lowering therapies. Continuous cardiac and biochemical monitoring is essential until potassium remains in the safe range for at least 24 hours.

NON-PHARMACOLOGICAL INTERVENTIONS

Non-pharmacological strategies form the backbone of hyperkalemia management, often initiated simultaneously with pharmacological therapy. These measures aim to prevent further potassium accumulation, enhance natural excretion, and stabilize the patient while definitive treatments take effect.

1. Discontinue Potassium Sources

- **Adults**: Immediately stop oral or IV potassium supplementation, potassium-rich diets, and potassium-containing salt substitutes.
- **Pediatrics**: Review all IV fluids, total parenteral nutrition (TPN), fortified formulas, and enteral feeds for hidden potassium content. Modify or replace with low-potassium alternatives. This is especially important **for neonates, infants, and children with renal impairment**, where even small excesses can cause dangerous rises in serum potassium.

2. Modify Dietary Intake

- **Adults**: Restrict high-potassium foods (bananas, oranges, potatoes, tomatoes, dried fruits).
- **Pediatrics**: Avoid excessive restriction in growing children unless medically necessary. Instead, work with a pediatric dietitian to provide a balanced low-potassium plan that maintains caloric and micronutrient adequacy. For infants, use potassium-reduced formulas when appropriate.

3. Optimize Hydration

- **Adults**: Correct dehydration with isotonic saline to promote renal excretion, unless volume overload is present.
- **Pediatrics**: Hydration is weight-based (10-20 mL/kg boluses in acute settings) and must be closely monitored to avoid fluid overload, especially in neonates or children with cardiac/renal disease.

ASSESSMENT OF RESPONSE

Assessment of response is a continuous process during hyperkalemia treatment to ensure that interventions are effective, safe, and timely adjusted.

Domain	Adults	Pediatrics (Neonates, Infants, Children)
Frequency of Clinical Assessment	Every 30-60 min during active treatment; hourly once stable.	Severe cases or ECG changes: every 15-30 min; hourly once stable.
Clinical Indicators of Improvement	Resolution of ECG abnormalities, stable heart rate/rhythm, improved muscle strength, normal mental status.	Improved feeding, spontaneous movement, alertness, stronger cry (neonates); improved motor strength, reflexes, mental status (older children).
Laboratory Monitoring	Recheck K^+ in 1-2 h after treatment start; if stable, q4-h until <5.5 mmol/L, then daily.	Neonates/infants: recheck in 30-60 min; q2-4h until stable; then at least q12h. Preterm/VLBW: use low-hemolysis methods.
ECG Surveillance	Continuous monitoring if K^+ ≥6.0 mmol/L, symptoms, or ECG changes; repeat after interventions.	Continuous monitoring if K^+ ≥5.5 mmol/L + symptoms, or earlier if cardiac/renal disease. Neonates: monitor if ≥5.8 mmol/L until normal.
Therapy Adjustment - Slow Response	Reassess potassium intake, renal function, and medications; escalate to combination therapy or dialysis if refractory.	Same as adults; escalate earlier in rapidly worsening cases.
If potassium falls too rapidly	Pause potassium-shifting interventions to avoid rebound hypokalemia.	In children, rapid overcorrection increases the risk of cardiac arrhythmias and metabolic disturbances, so adjustments must be made promptly.

Endpoints of Acute Management	K^+ ≤5.0 mmol/L, stable ECG, symptom resolution.	Children: K^+ ≤4.8 mmol/L; Neonates: ≤5.5 mmol/L, stable ECG, symptom resolution.
Review, follow-up Once initial stabilization is achieved	Review clinical course, K^+ rechecked 1-2 hours after acute interventions, then every 4-6 hours until it is consistently within the normal range.	More frequent monitoring (every 2-4 hours) especially neonates and infants

Note: Coordination between primary care, nephrology, endocrinology, and cardiology ensures long-term stability and prevents recurrence.

REFERRAL LINKAGES

- Primary care/Periphery: Begin ECG monitoring, administer calcium for ECG changes, start insulin-glucose and nebulized salbutamol , correct hypovolemia/acidosis, stop offending drugs, and arrange urgent transfer.
- Secondary care/Atoll/Regional hospital: Continuous monitoring, serial labs, diuretics if appropriate, cautious binder use, early consult with pediatric nephrology/ICU, and prepare for transfer if refractory or if dialysis indicated. Avoid binders for acute hyperkalemia.
- Tertiary care: Initiate dialysis when indicated; manage complex etiologies (TLS, endocrine crises, advanced CKD), and provide definitive investigations.

COMPLICATIONS

Hyperkalemia can cause rapid, life-threatening complications if not identified and corrected promptly.

Complication	Typical manifestations	Who's at highest risk	Prevention / monitoring	Immediate actions
Cardiac arrhythmias	Bradycardia, AV block, ventricular tachycardia, ventricular fibrillation - asystole; abrupt ECG shifts even at moderate K^+ (more sensitive in children/ neonates)	Neonates, infants, patients with renal failure, acidosis, digoxin use	Continuous ECG when K^+ ≥6.0 mmol/L (earlier in neonates); frequent K^+ checks	IV calcium for ECG changes; insulin-glucose ± β_2-agonist; prepare for definitive K^+ removal (dialysis if needed)
Neuromuscular failure	Progressive weakness, ascending paralysis; respiratory muscle involvement - respiratory arrest	Children, CKD, severe acidosis, rapid K^+ rise	Serial neuro checks; monitor vital capacity/ respiratory effort	Airway support if needed; proceed with standard hyperkalemia therapy

Rapid pediatric deterioration	Sudden arrhythmias; in VLBW (very low birth weight) neonates: intraventricular hemorrhage, cardiac arrest within hours	Preterm/VLBW neonates, infants with immature renal function	Earlier ECG monitoring; q1-2 h labs during acute phase; strict weight-based dosing	Urgent stabilization (calcium, shift, remove); NICU escalation
Secondary organ injury	From underlying cause: tumor lysis → multi-organ dysfunction; rhabdomyolysis → acute kidney injury; adrenal crisis → shock	Oncology/ICU patients; crush injury; adrenal insufficiency	Screen (CK, uric acid, phosphate, creatinine); hemodynamics	TLS protocol/fluids/renal support; treat adrenal crisis; manage rhabdomyolysis
Treatment-related harms	Over-correction - hypokalemia; insulin - hypoglycemia; aggressive fluids - volume overload; rebound hyperkalemia if trigger persists	Children (hypoglycemia risk), heart/renal failure, elderly	Titrate doses; glucose checks hourly for 3 hours after insulin; strict I/O; address cause (drugs, diet, acidosis)	Slow/stop K^+ therapy if overshoot; treat hypoglycemia; diuretics or dialysis for overload; fix precipitant to prevent rebound

OBJECTIVES OF PATIENT AND CAREGIVER EDUCATION

Patient and caregiver education aims to reduce recurrence, ensure early recognition, and promote safe day-to-day management of hyperkalemia risk.

Do	Don't
Know the condition: high blood potassium can trigger dangerous heart rhythms; in children even, mild symptoms can escalate quickly.	Don't start/stop medicines (including over-the-counter or herbal products) without medical advice.
Watch symptoms: weakness, limp limbs, palpitations, fast/slow/irregular heartbeat, unusual tiredness, confusion, sudden inability to move a limb. Seek urgent care if any appear.	Don't use salt substitutes, potassium supplements, or sports/energy drinks unless a clinician says so.
Follow the plan: keep all blood tests and ECG appointments; bring results/med lists to visits.	Don't skip follow-ups because symptoms improved.
Use medicines safely: take exactly as prescribed; tell every clinician about the potassium risk; keep an updated medication list.	Don't ignore new illness, dehydration, or medication changes - report promptly.
Hydrate as advised: give/consume adequate fluids unless told otherwise.	
Diet smartly (if advised): limit high-potassium foods (bananas, oranges, potatoes, beans) and check labels for hidden potassium (salt substitutes, sports/"electrolyte" drinks, supplements) without compromising a child's overall nutrition.	
Be prepared for emergencies: know where to go, who to call; keep key records and contacts handy.	

Extra for children (caregivers)

- After insulin therapy, monitor for hypoglycemia (sweating, irritability, confusion, lethargy); keep a quick sugar source available as instructed.
- For infants/children, balance any potassium limits with adequate calories and growth ask for dietitian input if unsure.

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