Acute hyperkalemia in the emergency department: a summary from a Kidney Disease: Improving Global Outcomes conference

Gregor Lindner[a], Emmanuel A. Burdmann[b], Catherine M. Clase[c], Brenda R. Hemmelgarn[d], Charles A. Herzog[e], Jolanta Małyszko[f], Masahiko Nagahama[g], Roberto Pecoits-Filho[h], Zubaid Rafique[i], Patrick Rossignol[j] and Adam J. Singer[k]

Hyperkalemia is a common electrolyte disorder observed in the emergency department. It is often associated with underlying predisposing conditions, such as moderate or severe kidney disease, heart failure, diabetes mellitus, or significant tissue trauma. Additionally, medications, such as inhibitors of the renin-angiotensin-aldosterone system, potassium-sparing diuretics, nonsteroidal anti-inflammatory drugs, succinylcholine, and digitalis, are associated with hyperkalemia. To this end, Kidney Disease: Improving Global Outcomes (KDIGO) convened a conference in 2018 to identify evidence and address controversies on potassium management in kidney disease. This review summarizes the deliberations and clinical guidance for the evaluation and management of acute hyperkalemia in this setting. The toxic effects of hyperkalemia on the cardiac conduction system are potentially lethal. The ECG is a mainstay in managing hyperkalemia. Membrane stabilization by calcium salts and potassium-shifting agents, such as insulin and salbutamol, is the cornerstone in the acute management of hyperkalemia. However, only dialysis, potassium-binding agents, and loop diuretics remove potassium from the body. Frequent reevaluation of potassium concentrations is recommended to assess treatment success and to monitor for recurrence of hyperkalemia.

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[a]Department of Internal and Emergency Medicine, Bürgerspital Solothurn, Solothurn, Switzerland, [b]LIM 12, Division of Nephrology, University of Sao Paulo Medical School, Sao Paulo, SP, Brazil, [c]Department of Medicine, McMaster University, Hamilton, Ontario, [d]Departments of Community Health Sciences and Medicine, University of Calgary, Calgary, Alberta, Canada, [e]Division of Cardiology, Department of Medicine, Hennepin Healthcare/University of Minnesota, Minneapolis, Minnesota, USA, [f]Department of Nephrology, Dialysis and Internal Medicine, Warsaw Medical University, Poland, [g]Division of Nephrology, Department of Internal Medicine, St. Luke's International Hospital, Tokyo, Japan, [h]Pontificia Universidade Catolica do Paraná, Curitiba, Brazil and Arbor Research Collaborative for Health, Ann Arbor, Michigan, [i]Department of Emergency Medicine, Baylor College of Medicine, Houston, Texas, USA, [j]Université de Lorraine, Inserm, Centre d'Investigations Cliniques-Plurithématique 14-33 and Inserm U1116, CHRU, F-CRIN INI-CRCT (Cardiovascular and Renal Clinical Trialists), Nancy, France and [k]Department of Emergency Medicine, Stony Brook University, Stony Brook, New York, USA

Correspondence to Gregor Lindner, MD, Department of Internal and Emergency Medicine, Bürgerspital Solothurn, Schöngrünstrasse 38, 4500 Solothurn, Switzerland
Tel: +41 78 874 7030; fax: +41 32 627 3121; e-mail: lindner.gregor@gmail.com

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Introduction

Recently, a Kidney Disease: Improving Global Outcomes (KDIGO) Controversies Conference was held in Miami, Florida, USA, to address potassium homeostasis and management of dyskalemia in kidney disease [1]. The conference was attended by a multidisciplinary group of nephrologists, endocrinologists, cardiologists, emergency medicine specialists, renal physiologists, and dietitians and consisted of an in-depth discussion on potassium homeostasis; potassium intake and outcomes in health and disease; and management of hypokalemia, acute hyperkalemia, and chronic hyperkalemia. The following manuscript represents our findings and consensus suggestions based on a review of the current literature and conference discussions of acute hyperkalemia, with the goal of facilitating knowledge translation of the key conclusions for health care professionals who work in emergency departments (EDs) and in the acute care settings.

Definitions

Hyperkalemia refers to an elevation in potassium concentration, although a universal definition does not exist. Commonly, hyperkalemia is defined as a potassium concentration ≥5.5 mmol/l [2], but this cutoff varies depending on the individual laboratories and differs

Based in part on the KDIGO Controversies Conference on Potassium Homeostasis and Management of Dyskalemia in Kidney Diseases held in Miami, Florida, USA, on October 5–7, 2018.

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between plasma and serum measurements [3–5]. There is not a common and standardized definition for grading of the severity of hyperkalemia as mild, moderate, or severe; potassium concentrations vary depending on whether serum or plasma potassium was analyzed, with serum potassium concentrations usually being higher than plasma concentrations [3–5]. Of note, pseudohyperkalemia is a falsely elevated serum potassium concentration, which can occur due to mechanical trauma, prolonged tourniquet use (>1 minute) or fist clenching during the process of blood drawing, and through blood clotting, centrifugation, elevated white blood cell count, or thrombocytosis (Table 1) [6–9]. For the purpose of this review, acute hyperkalemia is defined as a potassium concentration above the upper limit of normal, not known to be chronic. The severity of hyperkalemia can be classified as suggested recently (Fig. 1).

Epidemiology

The prevalence of hyperkalemia in the overall population, defined as a potassium concentration of ≥5 mmol/l; has been described to be 1.5% [10] and as high as 5.6% in patients who were recently started on angiotensin-converting enzyme inhibitors or angiotensin receptor blockers [11]. Among patients with heart failure, New York Heart Association class III and IV, with left ventricular ejection fraction <35%, who were newly started on spironolactone, the point prevalence of hyperkalemia (defined as a potassium concentration ≥5.5 mmol/l) was 19% compared to 5.6% in patients assigned to placebo [12]. Prevalence of hyperkalemia was 8.8% in a Swiss ED (defined as serum potassium >4.7 mmol/l) and 3.6% (defined as serum potassium >5 mmol/l) in a USA ED [13,14]. When hyperkalemia was defined as serum or plasma potassium concentrations ≥6 mmol/l, the prevalence was 0.3% of patients in the ED of a Swiss university hospital [15]. To our knowledge, there is no study of the prevalence of hyperkalemia in unselected patients presenting with acute kidney injury (AKI), but in patients in the ICU, the prevalence was 3.4% with no AKI, 8.8% in AKI stage 1, 17% in AKI stage 2, and 32.2% in AKI stage 3 [16]. In patients with chronic kidney disease with an estimated glomerular filtration rate <30 ml/min/1.73 m^2 the prevalence of hyperkalemia was 1.8% in a large USA cohort [17] and 4–5% in an Italian cohort [18].

Table 1 Risk factors for hyperkalemia

Predisposing factors
- Low glomerular filtration rate
- Male sex
- White ethnicity, high proteinuria
- Higher baseline potassium
- Diabetes mellitus
- Congestive heart failure
- Coronary artery disease
- Peripheral artery disease
- Malignancy
- Low hemoglobin
- Hyperlipidemia
- Metabolic acidosis (non-organic)
- Hemolysis
- Exercise
- Reduced aldosterone secretion
- Reduced response to aldosterone
- Voltage-dependent renal tubular acidosis
- Selective impairment in potassium secretion
- Gout
- Ureterojejunostomy
- Tissue breakdown (e.g., rhabdomyolysis)

Pseudohyperkalemia
- Fist clenching
- Hemolyzed sample
  - Tourniquet time >1 min
  - Mechanical trauma
  - Pneumatic tube without cushioning
  - Fine gauge needles
  - IV start compared with straight needles
  - Temperature (heat or cold shock)
  - Duration of storage
- Thrombocytosis
- Leucocytosis (e.g., chronic lymphatic leukemia)

Drugs/substances
- Potassium-sparing diuretics
- β-blockers
- Nonsteroidal anti-inflammatories
- Renin-angiotensin-aldosterone inhibitors
- Potassium supplements
- Calcineurin-inhibitors (cyclosporine, tacrolimus)
- Mannitol
- Heparin
- Digitalis
- Penicillin G
- Succinylcholine
- Octreotide
- Diazoxide
- Minoxidil
- Trimethoprim
- Volatile anesthetics (e.g., isoflurane)
- Red cell transfusion
- Salt substitutes
- Fruits
- Alfalfa
- Amino acids
- Dandelion
- Dried toad skin
- Hawthorne berry
- Horsetail
- Lily of the valley
- Milkweed
- Nettle
- Noni juice
- Siberian ginseng

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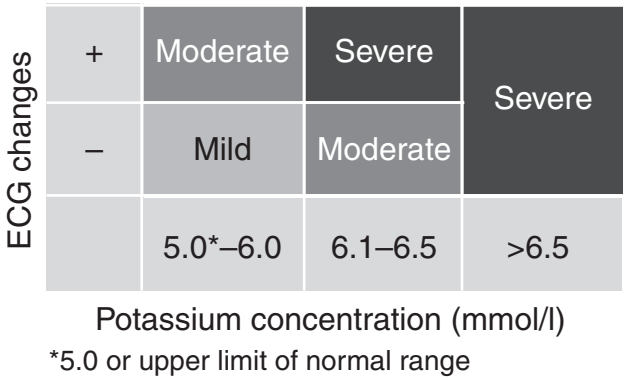
Etiology and risk factors

Usually, hyperkalemia develops because of increased intake, decreased excretion, or because of a shift of potassium from the intracellular to the extracellular compartment. Increased intake alone is unlikely to cause hyperkalemia because of the excess capacity of the healthy kidney to excrete potassium [19].

Overall, major risk factors for development of hyperkalemia are worsening of kidney function as expressed by a lower estimated glomerular filtration rate, a higher baseline potassium concentration, or the presence of comorbidities, including diabetes mellitus, heart failure, and coronary artery disease [20,21]. An important additional risk factor of relevance in the ED setting is

Fig. 1



Severity of acute hyperkalemia: expert opinion based risk classification. Reproduced with permission [1].

underlying rhabdomyolysis, as seen, for example, in major soft tissue trauma [22]. Additionally, hyperkalemia has been described in patients with electrical burns and in combat victims [23,24]. Low hemoglobin concentrations and strenuous exercise are also associated with elevated potassium concentrations [25,26].

A multitude of drugs has been described to have the potential to induce hyperkalemia or be associated with it. The most commonly prescribed and important drugs in relation to hyperkalemia are potassium-sparing diuretics, mineralocorticoid antagonists (e.g., spironolactone and eplerenone), angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, direct renin antagonists, β-blockers, nonsteroidal anti-inflammatory drugs, heparin, and penicillin G [2,11,21,25,27,28]. In the ED setting, the use of succinylcholine for muscle relaxation may lead to significant hyperkalemia [29,30]. Patients taking digitalis are also at risk for hyperkalemia [31,32]. Potassium supplements and potassium-based salt substitutes may also be a cause of hyperkalemia, especially in patients with underlying chronic kidney disease or in those with simultaneous use of hyperkalemia-inducing drugs.

Several natural products and alternative medical substances have been described as associated with acute hyperkalemia and may be of relevance in patients with otherwise unclear etiology of the elevated potassium concentration. An overview of the multiple potential causes and risk factors for development of hyperkalemia is provided in Table 1.

Symptoms and consequences of hyperkalemia

While many patients are asymptomatic, hyperkalemia may manifest clinically by muscle weakness. Paresthesias and muscular fasciculations in the arms and legs might be earlier signs of hyperkalemia [33]. Paralysis, cardiac conduction abnormalities, and cardiac arrhythmias can be lethal.

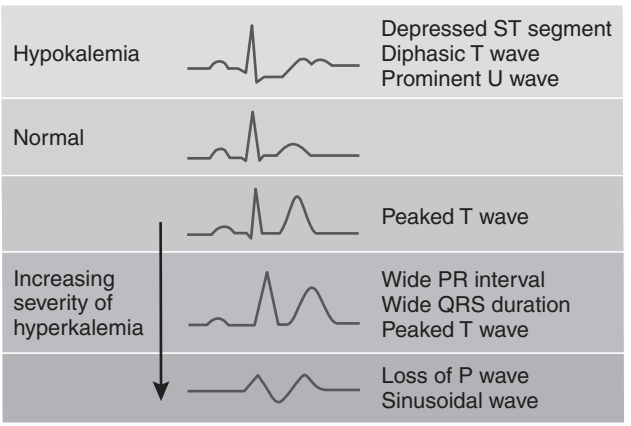
Usually, the muscle weakness associated with hyperkalemia is ascending, starting at the legs and progressing to the trunk, sometimes resembling Guillain–Barré syndrome [34–36].

The cardiac manifestations of hyperkalemia are caused by its depolarizing effects on the heart muscle cells and are usually progressive [33]. Though a recent review suggested that the presence and nature of ECG changes together with the absolute concentration of potassium may be used as a prognosticator of outcome [37], it should be noted that there is a scarcity of high-quality studies on the ECG manifestations of hyperkalemia as most data stem from case reports or small case series.

One of the newer and largest studies on ECG changes in patients with hyperkalemia, including a total of 90 patients, reported that ECG is insensitive for the detection of hyperkalemia and found no correlation between the presence of T-wave changes and the serum potassium concentration [38]. Tall, peaked T waves can, however, be early ECG signs of hyperkalemia [39]. Decreased amplitudes of the P waves, prolonged PR interval, and widening of the QRS complex are also sometimes observed, caused by a diminished sodium influx into the cardiac myocytes [37,39]. The classic ECG pattern of hyperkalemia, which occurs with more severe QRS broadening and fusion of the QRS complex with broadened ST-T segments, is the sine wave pattern [37,39]. The electrocardiographic changes associated with hyperkalemia are known to resemble acute ST-elevation infarction or Brugada Syndrome in some cases [40–42]. However, and importantly, up to half of the patients with hyperkalemia have a normal ECG, including some patients with extreme hyperkalemia [43]. In a retrospective study of 188 patients, the ECG abnormalities associated with adverse outcomes were QRS prolongation, symptomatic bradycardia, and junctional rhythms [44]. Figure 2 provides examples of typical ECG changes associated with hyperkalemia.

Recognizing the lack of sensitivity of the conventional ECG for the diagnosis of hyperkalemia is important in clinical practice (see Rossignol for further review) [45]. However, recently, computerized-analysis algorithms have been shown to be able to diagnose hyperkalemia from the ECG and may in future have a role in rapid diagnosis, especially in patients with chronic kidney disease, heart failure, or patients otherwise at risk for development of this serious electrolyte disorder [46,47].

Fig. 2



Typical ECG changes associated with hyperkalemia. It is important to note that ECG changes may not correlate closely with serum potassium concentration or be useful in predicting outcomes. As such, a normal ECG should not necessarily be regarded as reassuring if elevated potassium concentration has been definitively observed. Such patients may still experience sudden hyperkalemic cardiac arrest episodes. Reproduced with permission [48].

Diagnosis of hyperkalemia in the ED

In many cases, hyperkalemia in the ED will be identified incidentally by laboratory measurement of electrolytes or venous or arterial blood gas analysis. In patients without risk factors for hyperkalemia, or when the finding of hyperkalemia is not plausible, pseudohyperkalemia (see Fig. 1) should be considered.

Point-of-care testing of whole blood potassium concentrations was evaluated in large studies and compared with central laboratory measurements: potassium measurement by point-of-care testing resulted in sufficiently accurate results with mean differences between 0.1 and 0.5 mmol/l compared with central laboratory measurement [49,50]. However, point-of-care devices that analyze whole blood cannot identify hemolysis, which may result in spurious hyperkalemia. Use of point-of-care testing should be considered in patients in whom hyperkalemia is clinically suspected based on symptoms or ECG, and in those at high risk for hyperkalemia (e.g., patients who receive dialysis treatment). The rapid availability of results may permit more rapid initiation of treatment. However, we recommend confirmation of hyperkalemia by either central laboratory measurement or another analysis by point-of-care testing, in patients with an initial finding of hyperkalemia.

An ECG is recommended to assess the presence of hyperkalemia-induced ECG changes.

In addition, further steps to clarify the etiology of hyperkalemia should be undertaken. These can include a careful evaluation of the patient's history and medications, screening for hemolysis, assessment of kidney function, and, if applicable, investigation of the cause of AKI.

Management of acute hyperkalemia in the ED

High-quality studies on the management of acute hyperkalemia are lacking. Therefore, no international guideline exists specifically dealing with the management of hyperkalemia to date. Despite this, in 2014, the UK Renal Association published a clinical practice guideline on the treatment of acute hyperkalemia in adults, among others supported by the College of Emergency Medicine [51]. In 2015, the Investigator Network Initiative Cardiovascular and Renal Clinical Trialists, an international organization of academic cardiovascular and renal clinical trialists dedicated to improving outcomes among patients with chronic renal or cardiac disease, convened a meeting of nephrology, cardiology, and emergency medicine international experts, to identify gaps in knowledge, set priorities for future research, and develop an algorithm for emergency hyperkalemia management reflecting expert opinion in the context of current evidence [45]. The 2018 KDIGO conference extended this work.

Physical examination and history taking

In all patients suspected of having hyperkalemia, assessment by a physician or experienced health care provider should be performed as quickly as possible due to the potentially fatal consequences of high potassium concentrations [51]. Rapid determination of potassium concentrations should be performed, preferably by point-of-care testing, followed by central laboratory confirmation. Medical history along with history of previous medical conditions may help to assess the cause of hyperkalemia. Table 2 provides an overview of 'red flags' in the assessment of causes of and risk factors for hyperkalemia, which should trigger a high index of suspicion for the potential presence of hyperkalemia [51].

Monitoring

Continuous ECG monitoring, interval blood pressure monitoring at a frequency appropriate to the clinical context, and measurement of oxygen saturation should be established in patients with hyperkalemia.

ECG

The KDIGO potassium Controversies Conference recommended cardiac monitoring and 12-lead ECG for potassium concentrations >6.0 mmol/l. The fact that severe hyperkalemia may not necessarily be associated with ECG changes [38,45,52–54] and that hyperkalemia can lead to 'atypical' ECG changes under certain circumstances must always be kept in mind [55]. Therefore, one should put all hyperkalemic patients on continuous monitoring even if no typical ECG changes appear initially.

Treatment of hyperkalemia

Cellular membrane stabilization

Intravenous calcium salts should be administered immediately in hyperkalemic patients presenting with ECG changes suggesting hyperkalemia [2,51]. While some authorities prefer the use of calcium gluconate over calcium chloride because of lower tissue toxicity, others prefer calcium chloride because of a theoretical higher bioavailability [56]. It is crucial to note that the concentration of calcium is approximately three times higher in calcium chloride than in calcium gluconate (6.8 mmol Ca^{2+} per 10 ml of 10% calcium chloride vs. 2.3 mmol Ca^{2+}

Table 2 Red flags which should trigger awareness for the potential presence of hyperkalemia

Red flags for causes/risk factors for hyperkalemia
• Chronic kidney disease G4-G5 (estimated glomerular filtration rate <30 ml/min/1.73 m^2)
• Acute kidney injury
• Heart failure
• Diabetes mellitus
• Severe tissue breakdown
• Renin-angiotensin-aldosterone system inhibitors
• Nonsteroidal anti-inflammatory drugs
• Potassium-sparing diuretics

per 10 ml/10% calcium gluconate) [51]. Thus, to administer the same amount of calcium to the patient one has to use 30 ml of 10% calcium gluconate compared with 10 ml of 10% calcium chloride. The European Resuscitation Council recommends use of 10 ml 10% calcium chloride over 2–5 minutes in hyperkalemic patients with ECG changes [2]. Calcium prevents ventricular fibrillation/tachycardia by stabilizing the cardiac cell membrane and is effective within 1–3 minutes after administration [2]. Another dose can be administered within 5–10 minutes if no effect is seen, and repeated doses may be necessary if cardiac abnormalities resolve then recur.

If the potassium concentration is known but an ECG or placement on a monitor is not immediately possible, we suggest giving calcium to all patients with potassium concentration above 6.5 mmol/l (Fig. 3).

Caution must be observed in patients with potential digoxin toxicity since hypercalcemia may increase toxic effects of the drug [57]. More recent data from human studies have not demonstrated worsened outcomes [58,59]. Levine *et al.* [59] reviewed 159 cases of digoxin toxicity, of which 23 received calcium. No life-threatening dysrhythmias occurred within 1 hour of calcium administration, and mortality was similar between those that received calcium (22%) and those who did not (22%). It is critical to consider that administration of calcium salts does not lower potassium concentration and that its duration of action is limited to 30–60 minutes [33].

Potassium shift to the intracellular compartment

Since administration of calcium salts does not result in a lowering of potassium concentrations, other measures have to be taken to shift potassium from the extracellular to the intracellular compartment, including use of insulin and β-adrenergic agonists. In a systematic review by Harel and Kamel [60], the optimal dose and method of intravenous short-acting insulin to lower potassium in hyperkalemia was investigated and the authors concluded that the administration of 10 units of insulin resulted in comparable lowering of potassium as the administration of 20 units, while use of the larger dose was associated with a higher risk for hypoglycemia. Glucose, 25–50 g, should be administered to the hyperkalemic patient intravenously along with the insulin [60]. Studies from an ED in the USA compared a low insulin regimen (5 units) with a standard regimen of 10 units and showed no difference in the decrease in potassium between groups, while hypoglycemia was less common in those receiving the low insulin regimen with 5 units of insulin [61,62]. Because of the risk of hypoglycemia, blood glucose concentrations should be closely monitored. With glucose concentrations greater than 200 mg/dl (11.1 mmol/l), insulin may be given without additional glucose.

The administration of β-adrenergic agonists has been suggested as an alternative or an addition to insulin and glucose [2]. A Cochrane review from 2015 concluded that the intravenous and nebulized administration of salbutamol were both effective. Head-to-head comparisons are few, but what data are available suggest similar effects in lowering potassium to those seen with insulin. Use of 10 mg salbutamol via nebulizer results in a significant reduction of potassium at a peak of 120 minutes after application (90 minutes for 20 mg) [63]. The effects of salbutamol and insulin are potentially additive [64] and are currently under investigation (NCT04012138). This is a large dose compared with other uses of salbutamol (typically 4 × 2.5 mg wet nebulizations); as with insulin-glucose, patients should be monitored for adverse effects.

Sodium bicarbonate activates the Na^+-K^+-pump and corrects an underlying metabolic acidemia, potentially resulting in a lowering of serum potassium values [65], but data on its effectiveness are conflicting [66]. We suggest using sodium bicarbonate only in patients with metabolic acidemia who are expected to tolerate the sodium load involved. There are few data to help choose between hypertonic and isotonic bicarbonate. We suggest taking into account the clinical context, including the patient's sodium concentration and need for volume resuscitation.

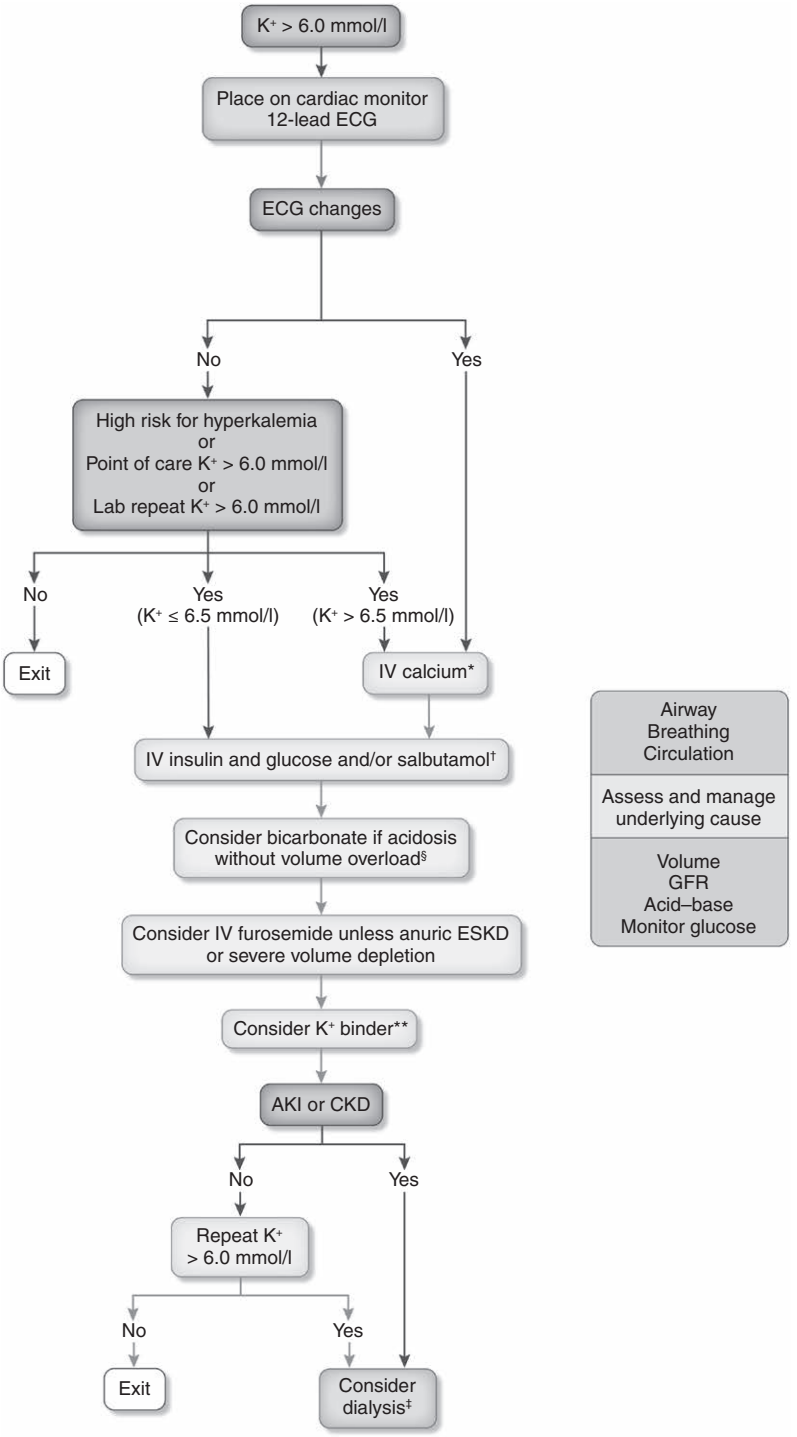
Potassium elimination

Potassium-binding agents, dialysis and loop diuretics, are the only means to remove potassium from the body [67].

Loop diuretics are commonly used in management of acute hyperkalemia; however, to date, there are no studies investigating their efficacy in eliminating potassium in this setting. Loop diuretics are likely useful in hyperkalemic patients with volume overload, such as in heart failure, and potentially useful after fluid resuscitation in other patients.

There are very limited data on the use of potassium-binding agents for the management of acute hyperkalemia [51]. While sodium polystyrene sulfonate has been in use for 60 years, its efficacy has not been studied in large randomized controlled trials. Furthermore, rare but increased adverse GI effects associated with sodium polystyrene sulfonate have been reported in two recent observational studies [68,69]. It should not be given with sorbitol because of an association between this combination and intestinal necrosis. Novel potassium binders such as patiromer and sodium zirconium cyclosilicate have recently been approved with promising results in eliminating potassium [70,71], and sodium zirconium cyclosilicate appears to have an appropriately rapid onset of action [72–75]. There is also a completed study that compared the efficacy of sodium zirconium cyclosilicate versus placebo when added to insulin and glucose in an

Fig. 3



Treatment algorithm for management of acute hyperkalemia in the emergency department. The thresholds for actions are opinion based. Suggested drug doses are based on a 2010 systematic review [80] and a subsequent observational study [81]. ECG changes commonly reported with increasing potassium concentrations have been described in the literature [37,39–42,82]. *IV 3 times 1 g calcium gluconate (3 × 10 ml of 10% solution, each containing 93 mg elemental calcium, 2.3 mmol; total 279 mg elemental calcium, 6.9 mmol) or 1 g calcium chloride (10 ml of 10% solution, 273 mg elemental calcium, 6.8 mmol) †IV regular insulin 5 units plus 25 g glucose (50 ml of 50%) is as effective as albuterol (salbutamol) 10 mg nebulized; insulin and albuterol may have an additive effect. Beware of hypoglycemia. §IV bicarbonate (1 amp of 50 ml of 8.4% solution, Na^+ 50 mmol, HCO_3^- 50 mmol) over 15 min. **Potassium binders: sodium polystyrene sulphonate (SPS) 15–60 g po/pr (do not give with sorbitol) or sodium zirconium cyclosilicate 10 g 3×/d po (patiromer not advisable as onset of action is 7 h). This guidance is suggestive as there are limited data on onset of action with no head-to-head studies between potassium binders. ‡Hemodialysis is the modality of preference. AKI, acute kidney injury; CKD, chronic kidney disease; ECG, electrocardiogram; ESKD, end-stage kidney disease; GFR, glomerular filtration rate; IV, intravenous; K^+, potassium; VF, ventricular fibrillation. Reproduced with permission [1].

ED setting but data are not yet available (NCT03337477). Although the onset of action for patiromer is slower, a recent small trial investigated its use in ED patients with hyperkalemia, and it was found that patiromer resulted in significantly lower serum potassium concentrations after 2 hours with no difference in adverse events [76]. As there are no long-term studies with these novel binders, it remains unknown whether they are associated with significant gastrointestinal adverse effects.

Dialysis eliminates potassium from the blood in patients with hyperkalemia. We recommend nephrology consultation in all patients presenting to the ED with hyperkalemia and receiving dialysis therapy. Additionally, patients with therapy-refractory hyperkalemia or hyperkalemia with AKI should be referred to nephrology for consideration of dialysis.

Reassessment

Frequent assessment of potassium concentrations is indicated in patients with acute hyperkalemia. Because the onset of action of potassium-shifting agents insulin-glucose and β-adrenergic agonists is 30–60 minutes, a reevaluation of potassium can be performed at 60 minutes after administration [63,77,78]. Since these medications do not excrete potassium but only shift it to inside the cell, a recurrence of hyperkalemia is expected and therefore reevaluation is crucial. Rebound towards higher values may occur at 2–3 hours if potassium has not been eliminated from the body during that period [64]. Additionally, blood glucose should be checked in patients receiving insulin-glucose because of the risk of hypoglycemia [61]. The duration of frequent monitoring of potassium concentration, continuous cardiac monitoring, and frequent blood pressure monitoring will depend upon the severity of the hyperkalemia, the severity of its manifestations, the likelihood of rebound, and the patient's overall clinical context and response to treatment.

Figure 3 provides an algorithm for the management of acute hyperkalemia in the ED.

Hyperkalemia–further care

In all patients presenting with acute hyperkalemia to the ED, a critical review of current medications should be performed for drugs that could cause hyperkalemia. Temporarily discontinuing inhibitors of the renin-angiotensin-aldosterone system should be considered, and a search for and treatment of any underlying condition leading to hyperkalemia should be performed. If the risk of hyperkalemia remains an ongoing issue, dietary instructions regarding low-potassium foods and avoidance of potassium-rich foods should be given to patients with moderate and severe kidney disease. Timing of further monitoring of potassium concentration, and whether as an in-patient or as an out-patient will depend upon the severity of the hyperkalemia, the severity of its manifestations, the likelihood of rebound, and the patient's overall clinical context and response to treatment. A recent retrospective study performed at a large academic ED found that mortality of patients with hyperkalemia was dramatically lower in the group whose potassium concentration was normalized (<5.5 mmol/l) during the ED stay compared to those whose potassium concentration remained elevated [79]. These data indicate that treatment in the ED or intensive/intermediate care units of patients with acute hyperkalemia until normalization of potassium concentrations might be preferable.

Areas of future research

As noted above, there is a lack of high-quality studies on the diagnosis and management of acute hyperkalemia. The sensitivity and specificity of various ECG changes for hyperkalemia, and for adverse outcomes, have not been systematically described. The optimal agent to shift or lower potassium concentrations is not known. The role of loop diuretics has not been evaluated in the management of acute hyperkalemia; specifically, there are no studies evaluating their potassium-lowering effect, onset of action, and the absolute amount of potassium excretion that is achievable; and their safety in patients who have recently undergone volume resuscitation is unknown. The role of new potassium-binding agents in the management of acute hyperkalemia is not well established, and larger studies are needed to understand possible rare adverse events.

In summary, acute hyperkalemia is a frequently observed problem in EDs and one with potentially fatal consequences. Monitoring of vital signs and performing a 12-lead ECG is recommended in all patients with potassium concentration >6 mmol/l. In all patients with hyperkalemia and presence of ECG changes, calcium salts should be promptly given. Additionally, measures to shift potassium to the intracellular compartment (i.e., insulin-glucose, β-adrenergic agonists) and remove it from the body should be taken. Frequent reevaluation of serum potassium concentrations is essential in order to monitor treatment success and screen for a rebound rise in serum potassium.

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