The Journal of Emergency Medicine, Vol. 57, No. 1, pp. 36–42, 2019
Published by Elsevier Inc.
0736-4679/$ - see front matter

https://doi.org/10.1016/j.jemermed.2019.03.043

Clinical Review

MANAGEMENT OF HYPERKALEMIA WITH INSULIN AND GLUCOSE: PEARLS FOR THE EMERGENCY CLINICIAN

Kayvan Moussavi, PHARMD, BCCCP,* Scott Fitter, PHARMD, BCCCP,† Stephen Walter Gabrielson, MSLIS, AHIP,‡ Alex Koyfman, MD,§ and Brit Long, MD||

*Department of Pharmacy Practice, College of Pharmacy, Marshall B. Ketchum University, Fullerton, California, †Department of Emergency Medicine, Loma Linda University Medical Center, Loma Linda University School of Pharmacy, Loma Linda, California, ‡Marshall B. Ketchum Memorial Library, Marshall B. Ketchum University, Fullerton, California, §Department of Emergency Medicine, The University of Texas Southwestern Medical Center, Dallas, Texas, and ||Department of Emergency Medicine, Brooke Army Medical Center, Fort Sam Houston, Texas

Corresponding Address: Brit Long, MD, Department of Emergency Medicine, Brooke Army Medical Center, 3841 Roger Brooke Dr., Fort Sam Houston, TX 78234

☐ **Abstract—Background: Hyperkalemia is a common, potentially lethal clinical condition that accounts for a significant number of emergency department (ED) visits. Insulin and dextrose are frequently used to manage patients with hyperkalemia. Objective: This narrative review evaluates several myths concerning hyperkalemia treatment with insulin and dextrose in the ED and provides recommendations based on the current evidence. Discussion: Hyperkalemia is a life-threatening condition requiring emergent therapy. One of these therapies includes insulin with glucose. However, hypoglycemia after insulin use is a frequent complication during hyperkalemia management. The published literature suggests that low pretreatment glucose, no history of diabetes mellitus, female gender, abnormal renal function, and lower body weight increase the risk of hypoglycemia. Several strategies can reduce the risk of hypoglycemia with insulin therapy, which include using insulin 5 units or 0.1 units/kg instead of 10 units, administering dextrose 50 g instead of 25 g, or administering dextrose as a prolonged infusion instead of a rapid intravenous bolus. Because insulin may have a duration of action that exceeds dextrose, patients receiving insulin for hyperkalemia should be monitored for hypoglycemia hourly for at least 4–6 h after administration. Conclusion: Several myths surround hyperkalemia management with insulin and dextrose. This review evaluates the evidence concerning insulin and glucose for hyperkalemia and suggests several modifications to insulin and dextrose dosing to reduce the risk of hypoglycemia. Published by Elsevier Inc.**

☐ **Keywords—adverse drug event; hyperkalemia; hypoglycemia; insulin; renal impairment**

Reprints are not available from the authors.

INTRODUCTION

Hyperkalemia (potassium >5.0 mmol/L) is a common, potentially life-threatening clinical condition that may affect ≤10% of patients in the emergency department (ED) (1–6). Hyperkalemia often affects patients with acute kidney injury (AKI) or chronic kidney disease (CKD), but it can also affect those with cardiovascular disease, diabetes mellitus, sickle cell disease, liver or kidney transplant, rhabdomyolysis, or those using various medications, such as angiotensin-converting enzyme inhibitors or angiotensin-receptor blockers (5,7–11). Clinical manifestations include cardiac dysrhythmias, respiratory depression, paralysis, paresthesias, depressed tendon reflexes, vomiting, or

Received: 21 December 2018; Final submission received: 16 March 2019;
Accepted: 31 March 2019

diarrhea (1,7,9,11–14). These clinical manifestations are associated with increased patient mortality and hospital costs, emphasizing the importance of emergent hyperkalemia management in the ED (8,15–18). Numerous treatment options for hyperkalemia are available and include agents that stabilize the cardiac membrane, redistribute potassium into cells, and enhance potassium elimination from the body (1,5–7,9,11,12,19,20). Intravenous (IV) insulin is a popular treatment option because of its rapid onset and predictable hypokalemic effects (11,21). Although insulin use for hyperkalemia treatment is typically well-tolerated, some patients may experience serious adverse drug events, such as hypoglycemia, despite concurrent administration with dextrose. This narrative review provides emergency clinicians with improved understanding of risk factors for hypoglycemia, dosing strategies, and hypoglycemia monitoring considerations by addressing several myths related to insulin use for hyperkalemia management.

METHODS

To construct this narrative review, we searched PubMed and Google Scholar using the keywords "hyperkalemia," "potassium," "insulin," "dextrose," "cardiac arrest," and "treatment." We focused on acute management of hyperkalemia with insulin and glucose and the risk of hypoglycemia. This search generated 333 results. We included case reports and series, retrospective and prospective studies, systematic reviews and meta-analyses, clinical guidelines, and other narrative reviews. Articles not in English, those presented as abstracts only, articles that were not available in full text, and articles that were outside the scope of this review were excluded. A total of 40 resources were selected for inclusion in this review. As this is a narrative review, we did not pool individual study data for further statistical analysis.

DISCUSSION

This review focuses on several components of hyperkalemia management with insulin by investigating myths related to this topic. Each myth will be followed by a pearl regarding insulin and glucose therapy for hyperkalemia.

Myth 1: All Patients Have Similar Risk for Hypoglycemia During Hyperkalemia Treatment with Insulin

Insulin causes an intracellular shift of potassium by stimulating extracellular Na^+-H^+ antiporters, thereby promoting sodium influx (22–25). Increased intracellular sodium concentrations trigger the activation of the Na^+-K^+ ATPase transporter, which exchanges intracellular sodium for extracellular potassium (22–25). IV insulin is believed to cause a dose-dependent decrease in serum potassium, with 10 units estimated to lower serum potassium by 0.6 to 1.2 mEq/L (21). When administered intravenously, the potassium shifting effects of insulin occur within 15 min of administration (21). IV insulin for the treatment of hyperkalemia is not without risks, the most common complication being hypoglycemia, with reported rates ranging from 8.7% to 75% (21,26–30).

Pearl 1: The Risk of Hypoglycemia May be Elevated in Those with Abnormal Renal Function, no History of Diabetes Mellitus, Low Pretreatment Glucose, Lower Body Weight, and Female Gender

Several factors affect the risk of hypoglycemia with insulin therapy for hyperkalemia. Abnormal renal function has been identified as a risk factor for hypoglycemia after insulin administration (21,28,31). In a study evaluating 12 patients with end stage renal disease (ESRD) who were given 10 units IV regular insulin with 25 g dextrose, 9 patients (75%) developed hypoglycemia (blood glucose [BG] <55 mg/dL) 1 h posttreatment (21). Baseline BG before treatment was 88 mg/dL (21). A more recent study by Apel et al. observed hypoglycemia (BG < 60 mg/dL) in 13% (29/221) of patients with ESRD who were treated with insulin (29). Garcia et al. found an increased risk of hypoglycemic events in patients with elevated baseline serum creatinine at the time of treatment with IV insulin (odds ratio [OR] 1.12, 95% confidence interval [CI] 1.02–1.23) (31). Several proposed explanations for increased hypoglycemia risk in those with renal dysfunction include decreased insulin clearance, reduced renal gluconeogenesis, and reduced glucagon release (22,32).

In addition to impaired renal function, no history of diabetes mellitus, lower pretreatment glucose, lower body weight, and female gender have also been identified as risk factors (26,28–31,33). Apel et al. identified no previous diagnosis of diabetes (OR 2.3, 95% CI 1.0–5.1) and lower pretreatment glucose (104 $\pm$ 63 mg/dL vs. 162 $\pm$ 148 mg/dL; $p = 0.04$) as risk factors for hypoglycemia (29). Studies by Wheeler et al. and Coca et al. found an increased risk of hypoglycemia when pretreatment glucose was <140 mg/dL (OR 4.3, 95% CI 1.4–13.7; $p = 0.01$) and <120 mg/dL (OR 4.44, 95% CI 0.9–21.57; $p = 0.055$), respectively (30,33). Garcia et al. observed that higher pretreatment glucose decreased hypoglycemia risk (OR 0.969, 95% CI 0.955–0.983; $p = 0.007$) (31). Schafers et al. found an increased risk of hypoglycemia in patients with lower body weight (55.8 vs. 92 kg; $p < 0.05$) (28). Of note, 79% (15/19) of patients who experienced hypoglycemia had concurrent

AKI or ESRD (28). Wheeler et al. observed that females were more likely to develop hypoglycemia compared with males (OR 3.2, 95% CI 1.1–9.1; $p = 0.03$) (30).

Insulin or dextrose dose adjustment strategies should be considered for patients with ≥1 risk factor for hypoglycemia, which will be discussed in subsequent sections. Because the majority of patients who require treatment for hyperkalemia have some degree of renal impairment, and because lower pretreatment glucose has been identified as a risk factor by several studies, lower pretreatment glucose should be the primary risk factor to prompt consideration for dosage adjustment (26,28–31,33). If a patient does not have lower pretreatment glucose, but has combinations of other risk factors, dose adjustment should also be considered.

Myth 2: Insulin 10 Units is Recommended for All Patients with Hyperkalemia

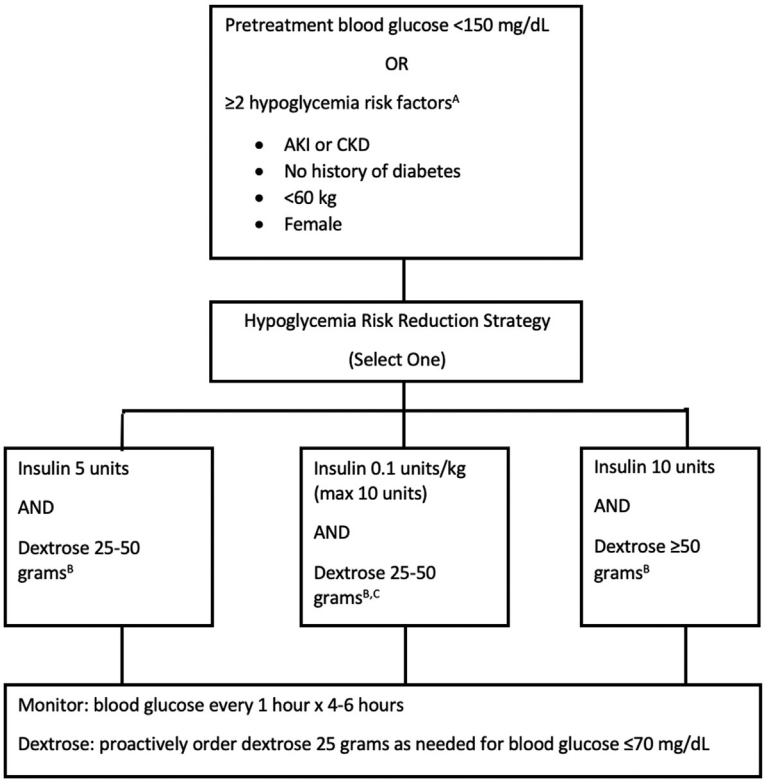
Most references recommend administration of 10 to 20 units of insulin in combination with 25 to 50 g dextrose to patients with severe hyperkalemia (serum potassium exceeding 6–6.5 mmol/L) (6,7,9,19,20,22). However, this may be associated with an increased risk of hypoglycemia. Dextrose is typically administered in combination with insulin to prevent hypoglycemia. Although the combination of insulin 10 units with dextrose 25 g is commonplace in the ED and inpatient settings, hypoglycemia is common and may occur in ≤75% of patients (21). This high incidence of hypoglycemia has prompted some providers to question if 10 units is necessary for all patients with hyperkalemia (22).

Pearl 2: Insulin Regular 5 Units or 0.1 Units/kg May be as Effective as 10 Units with Lower Incidence of Hypoglycemia

Several recent studies have compared insulin dosing strategies in patients with hyperkalemia to evaluate potassium-lowering ability and propensity for hypoglycemia. Comparisons were 5 units vs. 10 units or 0.1 units/kg vs. 10 units (27,31,33–35). Pierce et al. compared hospitalized adults with renal dysfunction (CKD or AKI) who received either 5 or 10 units for hyperkalemia; 25 g dextrose was given concurrently regardless of the insulin dosing strategy (34). Decreases in serum potassium were similar between groups (1.1 ± 0.91 mEq/L after 5 units vs. 1.08 ± 0.89 mEq/L after 10 units; $p = 0.89$), and there were no differences in incidence of hypoglycemia (BG ≤ 70 mg/dL) or severe hypoglycemia (BG < 50 mg/dL) (34). LaRue et al. also compared 5 vs. 10 units in adults with hyperkalemia managed in the ED (27). Patients receiving either insulin dose were administered 25 g of dextrose initially, then 25 g 1 h later, followed by another 25 g an additional 2 h later as needed for BG < 70 mg/dL (27). Potassium lowering was similar between groups (1 ± 0.8 mEq/L after 5 units vs. 1 ± 0.7 mEq/L after 10 units; 95% CI for difference −0.1 to 0.1); however, the incidence of hypoglycemia (BG < 70 mg/dL) was significantly lower in the 5 units group (19.5% vs. 28.6%; 95% CI for difference −16.8% to −1.3%) (27). There was no difference in incidence of severe hypoglycemia (BG < 40 mg/dL) (27). Finally, Garcia et al. compared insulin 5 to 10 units in hospitalized adults with hyperkalemia (31). Patients received 0 to 100 g of concurrent dextrose (92% received between 25–50 g) (31). There was no difference in post-insulin potassium reduction between groups (0.81 ± 0.58 mmol/L after 5 units vs. 0.9 ± 0.68 mmol/L after 10 units; $p = 0.221$) or incidence of hypoglycemia (BG < 70 mg/dL) (31). However, a subset analysis of patients with baseline potassium ≥6 mmol/L revealed greater potassium reduction after 10 units (1.08 mmol/L vs. 0.83 mmol/L; $p = 0.018$) (31).

Similar findings were observed in patients who were given 0.1 units/kg vs. 10 units of insulin. Wheeler et al. compared hospitalized adults treated with 0.1 units/kg (maximum of 10 units) or 10 units (33). Fifty grams of dextrose was given concurrently regardless of dosing strategy (33). Potassium lowering was similar between groups (1.34 ± 0.94 mmol/L after 0.1 units/kg vs. 1.35 ± 0.97 mmol/L after 10 units; $p = 0.94$), and there were no differences in incidence of hypoglycemia (BG < 70 mg/dL) or severe hypoglycemia (BG < 40 mg/dL) (33). Brown et al. also compared hospitalized adults given either 0.1 units/kg (maximum of 10 units) or provider choice of insulin dose (35). Patients received 25 g of dextrose concurrently in either group (35). Potassium lowering was similar between groups (0.6 ± 0.9 mmol/L after 0.1 units/kg vs. 0.6 ± 0.6 mmol/L after provider choice; $p = 0.67$), and there were no differences in incidence of hypoglycemia (BG < 70 mg/dL) or severe hypoglycemia (BG < 50 mg/dL) (35). Of note, insulin dosing was similar between groups, as 0.1 units/kg patients received 8.3 ± 1.6 units, while provider choice patients received 8.7 ± 2.3 units ($p = 0.08$) (35).

To summarize, the efficacy of lower dose regimens of insulin (5 units or 0.1 units/kg) in potassium reduction was similar to 10 units; however, Garcia et al. observed that a subset of patients with baseline potassium ≥6 mmol/L experienced greater potassium reduction after 10 units (27,31,33–35). The incidence of hypoglycemia or severe hypoglycemia was also similar after lower dose or 10 unit regimens; however, LaRue et al. found that the incidence of hypoglycemia was significantly lower in those receiving 5 units (27,31,33–35). These results suggest that lower-dose regimens



AKI: acute kidney injury; CKD: chronic kidney disease.

[A]If pretreatment glucose ≥150 mg/dL, consider routine therapy (e.g. insulin 10 units + dextrose 25 grams). If pretreatment glucose >250 mg/dL, consider holding dextrose.

[B]Consider all or part of dextrose dose as prolonged infusion (e.g. 50 ml of dextrose 50% IV push [25 grams] + dextrose 10% 62.5 ml/hr x 4 hours IV infusion [25 grams]).

[C]For patients receiving 10 units of insulin, consider administration of dextrose ≥50 grams.

Figure 1. Recommended hypoglycemia risk reduction strategies for patients requiring insulin treatment for hyperkalemia.

have similar efficacy but may be safer, especially in patients with multiple risk factors for hypoglycemia after insulin therapy. In patients with no risk factors for hypoglycemia, traditional therapy (i.e., insulin 10 units with dextrose 25 g) can be used. Providers can also consider holding dextrose if the pretreatment glucose is > 250 mg/dL (36).

Myth 3: Dextrose 25 g is Adequate to Avoid Hypoglycemia for All Patients Receiving Insulin for Hyperkalemia

Dextrose 25 g (e.g., 50 mL of 50% dextrose [D50]) as an IV bolus with 10 units of IV insulin is commonly recommended to prevent hypoglycemia during emergent management of hyperkalemia (2,9,12,36,37). This combination is widely believed to be sufficient to ensure that hypoglycemia does not develop after insulin administration. However, hypoglycemia has been reported in 13–75% of patients receiving this combination (21,27,29). Comparison of the duration of action of dextrose and insulin IV boluses illustrates why hypoglycemia may occur. In patients with normal renal function, D50 boluses typically have a duration of action of 1 h, compared with the 4–6 h duration of action of regular insulin (9). Because of decreased renal clearance of insulin, patients with CKD are at higher risk of hypoglycemia, and may have an even greater gap in glycemic coverage compared with those with normal renal function (7,36). This gap in coverage has been identified in several studies. Pierce et al. compared patients with AKI or CKD who received either 5 or 10 units of IV insulin for hyperkalemia management in addition to 25 g of dextrose (34). Hypoglycemia occurred after 4 h in 28.6% of patients who received 10 units (median 2.5 h [interquartile range 1.75–4 h]) and in 14.3% of patients who received 5 units (median 2.37 h [IQR 1.25–3.25 h]). (34). Notably, 1 patient in the 10 units group

experienced hypoglycemia 7.5 h postinsulin administration (34). Additional studies by Schafers et al. and McNicholas et al. reported times to hypoglycemia of 3 h and 2.37 h, respectively (28,38). The alarmingly high frequency of hypoglycemia suggests that 25 g of dextrose may be inadequate for hypoglycemia prevention in many patients.

Pearl 3: Higher Doses of Dextrose or Prolonged Infusions May be Required to Prevent Hypoglycemia

Modifications to dextrose dose or infusion time may decrease the likelihood of hypoglycemia after insulin administration. Coca et al. described one strategy involving dextrose infusion for hypoglycemia prevention (30). In this study, patients with AKI and non–dialysis-dependent CKD received 10 units IV insulin with dextrose 50 g (500 mL of dextrose 10%) infused over 4 h (30). Only 6.1% of treatments (10/164) resulted in hypoglycemia within 8 h after the end of the insulin-dextrose infusion (30). The average hypoglycemia onset was 3.5 h; however, 1 patient experienced hypoglycemia 6 h postinfusion (30). Wheeler et al. also assessed patients given insulin (either 10 units or 0.1 units/kg) and dextrose 50 g (dextrose product and infusion duration not stated) (33). Hypoglycemia was observed in 19.7% of those given 10 units and 10.6% of those given 0.1 units/kg ($p = 0.22$) (33). The mean or median onset of hypoglycemia was not reported (33).

Farina et al. recently evaluated if dextrose 50 g was more likely to prevent hypoglycemia compared with 25 g in patients receiving insulin 10 units (dextrose product and infusion duration not stated) (39). Patients were matched based on baseline rates of AKI, ESRD, and diabetes mellitus and assessed for hypoglycemia and hyperglycemia ≤4 h after insulin administration (39). At 1 h postinsulin, 8.3% of patients receiving 50 g developed hypoglycemia compared with 15.8% of those receiving 25 g ($p = 0.11$) (39). At 4 h postinsulin, 5% of those receiving 50 g developed hypoglycemia vs. 4.2% of 25-g patients ($p = 1$) (39). Hyperglycemia (glucose >180 mg/dL) rates were higher in 50-g patients at 1 h postinfusion (52.5% vs. 26.7%; $p = 0.001$) but not at 4 h postinsulin (10.8% in the 50-g group vs. 14.2% in the 25-g group; $p = 0.56$) (39). Among patients with a baseline BG < 110 mg/dL, hypoglycemia rates 1 h postinsulin were significantly lower in those who received dextrose 50 g (10.8% vs. 35.4%; $p = 0.002$) (39). Higher dextrose dose did not affect hyperglycemia rates in diabetic patients at 1 h (53.1% after 50 g vs. 40.6% after 25 g; $p = 0.45$) or 4 h (18.8% after 50 g vs. 31.2% after 25 g; $p = 0.39$) after infusion (39).

Based on these findings, dextrose 50 g or prolonged infusion rates appear to reduce the risk of hypoglycemia, especially in patients with lower baseline BG levels. The risk of hyperglycemia appears to be minimal and short-lived when compared with lower doses (e.g., 25 g). Combining lower insulin dose strategies with higher dextrose dose strategies would likely be the best approach to reducing hypoglycemia risk; however, this hypothesis requires further evaluation.

Myth 4: Patients Receiving Insulin and Dextrose for Hyperkalemia Do Not Require Monitoring for Hypoglycemia

As described previously, the onset of hypoglycemia after insulin administration commonly ranges from 2–4 h, despite concurrent dextrose administration (28,30,34,38). However, hypoglycemic events have been documented ≤7.5 h postinsulin administration (34).

Pearl 4: Patients Receiving Insulin for Hyperkalemia Should be Monitored for Hypoglycemia Hourly for 4–6 h After Administration

Based on these findings, BG monitoring should be performed hourly for the first 4–6 h after treatment with insulin. A recent publication by the Institute for Safe Medication Practices recommends monitoring BG for ≥6 h after insulin administration, even if concurrent dextrose is given (40). Monitoring is even more important in those with risk factors for hypoglycemia (e.g., lower baseline glucose, receiving higher insulin doses) or in those unable to communicate symptoms of hypoglycemia (e.g., dementia, altered mental status, or mechanically ventilated). As-needed dextrose boluses should be proactively ordered for these patients to prevent delays if hypoglycemia treatment is required.

Consideration of patient risk factors for hypoglycemia with insulin and dextrose therapy, along with utilization of several strategies for insulin and dextrose administration, can reduce the risk hypoglycemia. Figure 1 shows several strategies for insulin and dextrose administration.

CONCLUSIONS

Hyperkalemia is a common clinical condition that accounts for a significant number of ED visits. Insulin is frequently used to manage these patients, especially those with severe hyperkalemia or those with life-threatening symptoms. Although most emergency clinicians are familiar with insulin and dextrose therapy, they may be unaware of the risk of hypoglycemia after treatment. Patients who are at an increased risk of hypoglycemia include those with low pretreatment glucose, no history of diabetes mellitus, female gender, abnormal renal function, or lower body weight. Strategies to prevent

hypoglycemia include using insulin 5 units or 0.1 units/kg instead of 10 units, administering dextrose 50 g instead of 25 g, or administering dextrose as a prolonged infusion (e.g., 4 h) instead of a rapid IV bolus. Patients receiving insulin for hyperkalemia should be monitored for hypoglycemia hourly for at least 4–6 h after administration.

Acknowledgments—MB, BL, and AK conceived the idea for this article and contributed substantially to the writing and editing of the review. This article did not use any grants or funding, and it has not been presented in abstract form. This clinical review has not been published, it is not under consideration for publication elsewhere, its publication is approved by all authors and tacitly or explicitly by the responsible authorities where the work was carried out, and that, if accepted, it will not be published elsewhere in the same form, in English or in any other language, including electronically without the written consent of the copyright-holder. This review does not reflect the views or opinions of the U.S. Government, Department of Defense, U.S. Army, U.S. Air Force, Brooke Army Medical Center, or SAUSHEC EM Residency Program.

REFERENCES

1. Long B, Warix JR, Koyfman A. Controversies in management of hyperkalemia. J Emerg Med 2018;55:192–205.
2. Moore ML, Bailey RR. Hyperkalaemia in patients in hospital. N Z Med J 1989;102:557–8.
3. Paice B, Gray JM, McBride D, Donnelly T, Lawson DH. Hyperkalaemia in patients in hospital. Br Med J (Clin Res Ed) 1983;286:1189–92.
4. Shemer J, Modan M, Ezra D, Cabili S. Incidence of hyperkalemia in hospitalized patients. Isr J Med Sci 1983;19:659–61.
5. Fordjour KN, Walton T, Doran JJ. Management of hyperkalemia in hospitalized patients. Am J Med Sci 2014;347:93–100.
6. Elliott MJ, Ronksley PE, Clase CM, Ahmed SB, Hemmelgarn BR. Management of patients with acute hyperkalemia. CMAJ 2010;182:1631–5.
7. Mushiyakh Y, Dangaria H, Qavi S, et al. Treatment and pathogenesis of acute hyperkalemia. J Community Hosp Intern Med Perspect 2012;1.
8. Kovesdy CP. Updates in hyperkalemia: outcomes and therapeutic strategies. Rev Endocr Metab Disord 2017;18:41–7.
9. Weisberg LS. Management of severe hyperkalemia. Crit Care Med 2008;36:3246–51.
10. Palmer BF. Managing hyperkalemia caused by inhibitors of the renin-angiotensin-aldosterone system. N Engl J Med 2004;351:585–92.
11. Mahoney BA, Smith WA, Lo DS, et al. Emergency interventions for hyperkalaemia. Cochrane Database Syst Rev 2005;2:CD003235.
12. Weiner ID, Wingo CS. Hyperkalemia: a potential silent killer. J Am Soc Nephrol 1998;9:1535–43.
13. Littmann L, Gibbs MA. Electrocardiographic manifestations of severe hyperkalemia. J Electrocardiol 2018;51:814–7.
14. Faxén J, Xu H, Evans M, et al. Potassium levels and risk of in-hospital arrhythmias and mortality in patients admitted with suspected acute coronary syndrome. Int J Cardiol 2019;274:52–8.
15. Betts KA, Woolley JM, Mu F, et al. The cost of hyperkalemia in the United States. Kidney Int Rep 2018;3:385–93.
16. Furuland H, McEwan P, Evans M, et al. Serum potassium as a predictor of adverse clinical outcomes in patients with chronic kidney disease: new risk equations using the UK clinical practice research datalink. BMC Nephrol 2018;19:211.
17. Saxena A, Meshram SV. Predictors of mortality in acute kidney injury patients admitted to medicine intensive care unit in a rural tertiary care hospital. Indian J Crit Care Med 2018;22:231–7.
18. Xu H, Faxen J, Szummer K, et al. Dyskalemias and adverse events associated with discharge potassium in acute myocardial infarction. Am Heart J 2018;205:53–62.
19. Putcha N, Allon M. Management of hyperkalemia in dialysis patients. Semin Dial 2007;20:431–9.
20. Kraft MD, Btaiche IF, Sacks GS, Kudsk KA. Treatment of electrolyte disorders in adult patients in the intensive care unit. Am J Health Syst Pharm 2005;62:1663–82.
21. Allon M, Copkney C. Albuterol and insulin for treatment of hyperkalemia in hemodialysis patients. Kidney Int 1990;38:869–72.
22. Li T, Vijayan A. Insulin for the treatment of hyperkalemia: a double-edged sword? Clin Kidney J 2014;7:239–41.
23. Clausen T, Everts ME. Regulation of the Na,K-pump in skeletal muscle. Kidney Int 1989;35:1–13.
24. Flatman JA, Clausen T. Combined effects of adrenaline and insulin on active electrogenic Na+-K+ transport in rat soleus muscle. Nature 1979;281:580–1.
25. Minaker KL, Rowe JW. Potassium homeostasis during hyperinsulinemia: effect of insulin level, beta-blockade, and age. Am J Physiol 1982;242:E373–7.
26. Scott NL, Klein LR, Cales E, Driver BE. Hypoglycemia as a complication of intravenous insulin to treat hyperkalemia in the emergency department. Am J Emerg Med 2019;37:209–13.
27. LaRue HA, Peksa GD, Shah SC. A comparison of insulin doses for the treatment of hyperkalemia in patients with renal insufficiency. Pharmacotherapy 2017;37:1516–22.
28. Schafers S, Naunheim R, Vijayan A, Tobin G. Incidence of hypoglycemia following insulin-based acute stabilization of hyperkalemia treatment. J Hosp Med 2012;7:239–42.
29. Apel J, Reutrakul S, Baldwin D. Hypoglycemia in the treatment of hyperkalemia with insulin in patients with end-stage renal disease. Clin Kidney J 2014;7:248–50.
30. Coca A, Valencia AL, Bustamante J, Mendiluce A, Floege J. Hypoglycemia following intravenous insulin plus glucose for hyperkalemia in patients with impaired renal function. PLoS One 2017;12:e0172961.
31. Garcia J, Pintens M, Morris A, et al. Reduced versus conventional dose insulin for hyperkalemia treatment. J Pharm Pract 2018; https://doi.org/10.1177/0897190018799220. [Epub ahead of print].
32. Shrishrimal K, Hart P, Michota F. Managing diabetes in hemodialysis patients: observations and recommendations. Cleve Clin J Med 2009;76:649–55.
33. Wheeler DT, Schafers SJ, Horwedel TA, Deal EN, Tobin GS. Weight-based insulin dosing for acute hyperkalemia results in less hypoglycemia. J Hosp Med 2016;11:355–7.
34. Pierce DA, Russell G, Pirkle JL Jr. Incidence of hypoglycemia in patients with low eGFR treated with insulin and dextrose for hyperkalemia. Ann Pharmacother 2015;49:1322–6.
35. Brown K Jr, Setji TL, Hale SL, et al. Assessing the impact of an order panel utilizing weight-based insulin and standardized monitoring of blood glucose for patients with hyperkalemia. Am J Med Qual 2018;33:598–603.
36. Sterns RH, Grieff M, Bernstein PL. Treatment of hyperkalemia: something old, something new. Kidney Int 2016;89:546–54.
37. Vanden Hoek TL, Morrison LJ, Shuster M, et al. Part 12: cardiac arrest in special situations: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010;122(18 suppl 3):S829–61.
38. McNicholas BA, Pham MH, Carli K, et al. Treatment of hyperkalemia with a low-dose insulin protocol is effective and results in reduced hypoglycemia. Kidney Int Rep 2018;3:328–36.
39. Farina N, Anderson C. Impact of dextrose dose on hypoglycemia development following treatment of hyperkalemia. Ther Adv Drug Saf 2018;9:323–9.
40. Institute for Safe Medication Practices. Medication errors during insulin administration for patients with hyperkalemia. ISMP Med Saf Alert 2018;23:1–4.

ARTICLE SUMMARY

1. Why is this topic important?

Insulin and dextrose are often used in the emergency department management of hyperkalemia; however, hypoglycemia can occur.

2. What does this review attempt to show?

This narrative review evaluates several myths concerning hyperkalemia treatment with insulin and dextrose in the emergency department and provides recommendations based on the current evidence.

3. What are the key findings?

Hypoglycemia is a common complication with hyperkalemia therapy. Risk factors include low pretreatment glucose, no history of diabetes mellitus, female gender, abnormal renal function, and lower body weight. Based on the current evidence, strategies to reduce the risk of hypoglycemia with insulin therapy include using insulin 5 units or 0.1 units/kg instead of 10 units, administering dextrose 50 g instead of 25 g, or administering dextrose as a prolonged infusion instead of a rapid intravenous bolus. Because insulin may have a duration of action that exceeds dextrose, patients who are receiving insulin for hyperkalemia should be monitored for hypoglycemia hourly for at least 4–6 h after administration.

4. How is patient care impacted?

Therapy for hyperkalemia often includes insulin and dextrose; however, several myths surround this therapy. This review evaluates the evidence suggesting that modifications to insulin or dextrose dosing can reduce the risk of hypoglycemia.