Aboujamous et al. J Clin Nephrol Ren Care 2016, 2:013
Volume 2 | Issue 2

Journal of
Clinical Nephrology and Renal Care

Research Article: Open Access

Evaluation of the Change in Serum Potassium Levels after Potassium Administration

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Abstract

Background: Due to limited data and inconsistent recommendations for potassium replacement, this study examined the relationship between the amount of potassium administered and the subsequent change in serum potassium levels.

Objective: The objective of this study was to quantify the change in serum potassium level after intravenous or oral potassium administration. Secondary objectives include investigating the possible influence of concurrent medications, renal function, and body mass index (BMI).

Methods: This study was an institutional review, board approved, single-center, retrospective, medical record review. Patients receiving a first dose of intravenous (IV) or oral (PO) potassium on or during admission were included in the study. The primary outcome measure was the mean change in serum potassium in milliequivalent/liter (mEq/L) per each 10 milli equivalent (mEq) of potassium administered.

Results: Two hundred sixty patients were included in the analysis. There was an overall mean increase in serum potassium levels of 0.13 mEq/L ± 0.11 (PO and IV) per 10 mEq of potassium administered. There was mean increase of 0.14 mEq/L ± 0.14 (IV, n = 89) and 0.12 mEq/L ± 0.08 (PO, n = 171) in serum potassium levels between groups that was not statistically significant (p = 0.12). For patients on medications presumed to cause decreases in serum potassium, patients administered IV potassium or PO potassium had a 0.07 and 0.11 mEq/L mean increase (p = 0.36), respectively. For patients on medications presumed to increase serum potassium, patients administered intravenous potassium and oral potassium had a 0.12 and 0.14 mEq/L mean increase in serum potassium (p = 0.24), respectively. Results could not be drawn regarding the impact of renal dysfunction due to the minimal representation of these patients. Furthermore, BMI did not seem to impact the degree of potassium repletion.

Conclusions: Every 10 mEq of potassium increased serum potassium 0.13 mEq/L. Similar dose responses were seen whether IV or PO potassium was administered. This study supports the common practice of administering 10 mEq of potassium for every 0.1 mEq/L desired increase in serum potassium.

Keywords

Electrolytes, Clinical pharmacy, Adult medicine, Internal medicine, Evidence-based medicine

Background

Hypokalemia is defined as a serum potassium concentration of less than 3.5 mEq/L with the normal range being 3.5-5 mEq/L [1,2]. Hypokalemia is classified into stages of mild (3-3.4 mEq/L), moderate (2.5-2.9 mEq/L) and severe (< 2.5 mEq/L) [1]. In examination of the physiology of potassium homeostasis, 98% of total body potassium, which equals approximately 3400 mEq in the average person, is present in the intracellular space. Serum potassium is a measurement of the 2% that is in the extracellular space, and is approximately 70 mEq in most people. It takes a significant loss in potassium stores to see a drop in serum potassium level (i.e., the extracellular space) due to this large amount of potassium in the intracellular space that helps to compensate for any loss [3].

Hypokalemia is a common electrolyte abnormality in hospitalized patients and is estimated to occur in approximately 20% of patients admitted to medical surgical services and as high as 40% of patients in intensive care units [4,5]. Severe consequences of hypokalemia include cardiac arrhythmias, rhabdomyolysis, and muscle weakness that leads to respiratory depression or ileus. Chronic hypokalemia can cause increased ammoniagenesis, urinary concentration defects, polyuria, hypertension, acid base disorders, and hyperglycemia [3,6].

Potassium repletion is indicated when the total body stores of potassium are decreased and the degree of hypokalemia correlates with the magnitude of the deficiency in potassium [7]. The consideration to replete potassium via the oral or intravenous route depends on the ability of the patient to take oral medications and having a normal functioning gastrointestinal tract. Patient specific variables such as renal function and body surface area have been proposed as being important in deciding to replete potassium [3].

There is a lack of evidence regarding the dose of potassium to administer in order to increase a serum potassium level by a certain amount [8]. Common practice has become to replete with 10 mEq of potassium for every 0.1 mEq/L increase in potassium desired [9]. There exists a paucity of studies, and thereby, guidelines, to provide direction on how to most appropriately approach the repletion of potassium in the inpatient setting.

Most studies solely examine a critically ill patient population. A retrospective study by Kruse and Carlson from 1990 examined

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Citation: Aboujamous H, Walton T, Doran JJ (2016) Evaluation of the Change in Serum Potassium Levels after Potassium Administration. J Clin Nephrol Ren Care 2:013

Received: June 29, 2016: **Accepted:** September 12, 2016: **Published:** September 15, 2016

a medical intensive care unit population. The average increment of increase in serum potassium level per 20 mEq IV potassium infused was 0.25 mmol/L [7]. The findings were echoed in another study in 1994 by Kruse with other colleagues when this time, the safety of rapid correction of hypokalemia (20 mmol IV over 1 hour) using concentrated intravenous potassium chloride infusions (200 mmol/L) in a critically ill patient population was examined. The serum potassium level was found to have increased an average of 0.25 mmol/L when measured sometime after the infusion. This specific amount of time after infusion was not specified [10]. Patients in each of these studies had a mean baseline serum potassium level of 3.2 and 2.9 mmol/L, respectively. Most patients possessed adequate renal function. Specifically, 12% of patients had renal failure in the 1990 study and the mean serum creatinine was 1.24 mg/dL (0.6-4.86) in the 1994 study [7,10]. Recently, Chalwin and colleagues, in a study in critically ill patients, found that for every 20 mmol of IV potassium administered, there was a mean 0.22 mmol/L increase in serum potassium. Of note, this study excluded patients with a serum creatinine greater than 2 mg/dL. The mean baseline potassium among the study population was 3.4 mEq/L with a standard deviation of 0.3 [11]. While these three studies overall roughly support the common practice of a 0.1 mEq/L (or mmol/L) increase in potassium for every 10 mEq (or mmol) administered, another study by Hamill and colleagues in critically ill patients found otherwise. Findings of this study showed a variable increase in serum potassium depending on the dose administered. The mean increase in serum potassium was 0.5 ± 0.3 mmol/L, 0.9 ± 0.4 mmol/L and 1.1 ± 0.4 mmol/L in the groups of patients receiving 20 mmol, 30 mmol, and 40 mmol of potassium, respectively. There was no difference in the increase in serum potassium level in patients with normal renal function as compared to those with renal insufficiency [12].

This study will aim to evaluate a diverse patient population receiving different doses of potassium. Variables such as the administration of IV versus PO potassium will be examined. Also, the impact of other patient specific factors such as the influence of concurrent medications, renal function and body mass index will be examined.

Methods

This study was an institutional review board approved single-center, retrospective, medical record review. Grady Health System is a licensed 953 bed, urban, teaching hospital in Atlanta, Georgia with approximately 26,000 admissions per year. The purpose of this study is to quantify the change in serum potassium levels after potassium administration. Patients included were chosen at random from a spreadsheet of people that received intravenous or oral potassium between January 1, 2011 and December 31, 2012. Patients were included if they had received the first dose of intravenous or oral potassium during the admission, if they had serum potassium level revealing hypokalemia within 4 hours before the potassium administration, and if they had labs obtained within 18 hours post conclusion of potassium administration. The timeframe of 4 hours before and within 18 hours after administration of potassium was chosen to ensure the potassium level drawn was reflective of the true potassium level prior to and after potassium administration and monitoring is typical for inpatient practice. For all patients for whom data was collected, no patients had more than one pre-potassium repletion serum potassium level obtained. If multiple post-potassium administration serum potassium levels were obtained, the latest of the levels was accounted for as it would be most reflective of the effect of potassium administration on serum potassium levels. Patients were excluded if data regarding a potassium administration earlier in their admission was already included in the data. Patients were also excluded if they were still admitted at the time of data collection (to maintain the retrospective nature of the study) and received continuous scheduled potassium therapies. Continuous potassium therapies make it difficult to determine how much of the potassium administered affects a subsequently drawn serum potassium level. Other exclusion criteria include other potassium sources such as total parenteral nutrition, tube feeding, or maintenance intravenous fluids. Such patients were excluded because as with the patients receiving continuous potassium, it is difficult to determine which dose of potassium or how much of the potassium administered is reflected in the serum potassium level that is obtained after or during potassium administration. Patients with reported diarrhea, nausea and vomiting (evidence of absorption problem) were excluded if the assessment was of their oral potassium intake rather than intravenous. Evidence of intrinsic sources of potassium release including active rhabdomyolysis, hemolytic anemia, and tumor lysis were other reasons for exclusion. Acute kidney injury (AKI) was also a reason for exclusion. AKI was defined according to the Kidney Disease: Improving Global Outcomes Clinical Practice Guideline for Acute Kidney Injury as an increase in serum creatinine by ≥ 0.3 mg/dl within 48 hours or an increase in serum creatinine to ≥ 1.5 times from baseline, which is known or presumed to have occurred within the prior 7 days; or urine volume < 0.5ml/kg/h for 6 hours. Other reasons for exclusion were having a serum potassium level that is reflective of both intravenous and oral potassium administration, hemolyzed potassium levels, dialysis, a diagnosis of diabetic ketoacidosis defined as: patient with a diagnosis of diabetes, hyperglycemia (blood glucose > 200 mg/dL) and betahydroxybutyrate > 0.27 millimoles/liter (mmol/L), acidosis defined as arterial blood gas pH < 7.35 or serum bicarbonate < 20 mmol/L, a magnesium level less than 1.5 milligrams/deciliter (mg/dL), being a minor (less than 18 years of age), pregnancy, and incarceration.

Data was collected from the electronic medical record. Reports from the electronic medical record program were generated to determine patients having received intravenous or oral potassium supplementation. Patients were placed in alphabetical order and 3,532 were screened. From these patients, 260 patients met criteria for inclusion. The primary reasons for exclusion were patients having already been included in the study and patients for which no follow up serum potassium levels were drawn post potassium administration. The following information was collected: demographics including patient weight, height, gender, race, BMI, and basic metabolic panel which includes glucose, calcium, sodium, potassium, bicarbonate, chloride, blood urea nitrogen, and creatinine. This information was used to calculate glomerular filtration rate (GFR) using the Modification of Diet in Renal Disease (MDRD) formula [13]. Other information collected included, if documented, the reason for repletion, pre- and post-administration of potassium serum potassium levels and time measured, route of administration (intravenous or oral), total dose of potassium received on one administration prior to measured level as documented on the medication administration record and concurrent medications that may decrease or increase serum potassium level.

The primary outcome measure was the mean change in serum potassium in milliequivalent/liter (mEq/L) per each 10 milliequivalent (mEq) of potassium administered. Secondary outcome measures include the mean ratio of the dose per mEq of intravenous or oral potassium administered to the change in serum potassium level produced while concurrently on medications that can decrease potassium levels as well as the mean ratio of the dose per mEq of intravenous or oral potassium administered to the change in serum potassium level produced while concurrently on medications that can decrease potassium levels. Other secondary outcome measures include the mean ratio of the dose per mEq of intravenous or oral potassium administered to the change in serum potassium level produced with glomerular filtration rates greater than 30 milliliter/minute/1.73 meter^2 (mL/min/m^2), 15-30 mL/min/1.73 m^2 or end-stage renal disease with a GFR less than 15 mL/min/1.73 m^2 [14]. The final secondary outcome measure is the mean ratio of the dose per mEq of intravenous or oral potassium administered to the change in the serum potassium level produced by each of the World Health Organization's BMI categories. These categories include: underweight (below 18.5), normal (18.5-24.9), overweight (≥ 25) and obese (≥ 30) [15]. Student t-test and descriptive statistics were used to calculate any differences between the intravenous and oral potassium groups.

Table 1: Baseline characteristics.

Characteristic	N = 260
Age in years, mean (SD)	51.2 (18.4)
Gender, n (%) Male	153 (59)
Race, n (%)	
Black	232 (89)
White	15 (6)
Hispanic	7 (3)
Asian	5 (2)
Other	1 (< 1)
Unit type, n (%)	
Medical-Surgical	216 (83.1)
Surgical Intensive Care Unit	16 (6.2)
Step-down Unit	9 (3.5)
Neurology Intensive Care Unit	9 (3.5)
Burn	4 (1.4)
Medical Intensive Care Unit	3 (1.2)
Emergency Care Center	3 (1.2)
Initial Potassium level, mean (SD) mEq/L	3.9 (0.34)
Potassium Concentration, n (%)	
Normal (≥ 3.5 mEq/L)	145 (56)
Mild Hypokalemia (3-3.4 mEq/L)	112 (43)
Moderate Hypokalemia (2.5-2.9 mEq/L)	2 (1)
Severe Hypokalemia (< 2.5 mEq/L)	1 (< 1)
SCr, mean (SD), mg/dl	1.13 (0.9)
Chronic Kidney Disease Stage 4 or 5, n (%)	10 (3.8)

Table 2: Mean change in serum potassium per 10 mEq of potassium administered.

Dosage Route	Change in serum potassium ± S.D. (mEq/L)
Intravenous and Oral (N = 260)	0.13 ± 0.11
Intravenous (n = 89)	0.14 ± 0.14*
Oral (n = 171)	0.12 ± 0.08*

*p = 0.12 for difference between intravenous and oral potassium groups.

Results

Demographic information for the 260 patients in the study is listed in table 1. The primary outcome comparing the potassium level changes is listed in table 2. Including all patients and routes of administration, for every 10 mEq of potassium administered, there was a 0.13 mEq/L mean increase in serum potassium level. Specifically, there was mean increase of 0.14 mEq/L ± 0.14 (IV, n = 89) and 0.12 mEq/L ± 0.08 (PO, n = 171) in serum potassium levels between groups that was not statistically significant (p = 0.12).

Certain medications can increase or decrease serum potassium levels. There were only 13 patients on medications that can decrease potassium. These medications included corticosteroids. The mechanism by which corticosteroids decrease serum potassium is believed to be through increase in excretion of potassium in the renal tubule. Other medications that can decrease potassium include loop diuretics, beta-adrenergic stimulants, glucose and aminoglycosides. Patients in the intravenous group (n = 7) had a mean 0.07 mEq/L increase in serum potassium level per 10 mEq of potassium administered while patients in the oral group (n = 6) had a mean 0.11 mEq/L increase (p = 0.36). There were a total of 58 patients on medications that can increase the serum potassium level, and the intravenous group (n = 25) had a mean 0.12 mEq/L increase in serum potassium while the oral group (n = 33), had a mean 0.14 mEq/L increase (p = 0.24).These changes were similar to the overall results. Medications included prophylactic heparin, angiotensin converting enzyme inhibitors, angiotensin II receptor blockers, potassium-sparing diuretics, and magnesium. Regarding the impact of different levels of renal function, 96% of patients had a GFR greater than 30 mL/min/1.73 m^2, and patients with GFR < 15 mL/min had a higher than average response to both IV and PO whereas those with GFR ≥ 15 to ≤ 30 had a greater than average response only with PO potassium (Table 3).

Table 3: Effect of renal function on potassium repletion.

Mean change in serum potassium level per 10 mEq of potassium administered (N = 260)		
GFR (mL/min)	**Intravenous, mEq/L (n)**	**Oral, mEq/L (n)**
≥ 30	0.14 (85)	0.12 (166)
≥ 15 to < 30	0.10 (3)	0.19 (2)
< 15	0.15 (1)	0.19 (3)

Table 4: Effect of body mass index.

Mean change in serum potassium level per 10 mEq of potassium administered		
BMI Classification	**Intravenous, mEq/L(n)**	**Oral, mEq/L (n)**
Underweight (< 18.5)	0.11 (9)	0.13 (7)
Normal (18.5 - 24.9)	0.17 (34)	0.12 (60)
Overweight (25 - 29.9)	0.12 (35)	0.12 (47)
Obese (> 30)	0.10 (11)	0.12 (57)

In examining the effect of BMI on the level of potassium repletion achieved per 10 mEq of potassium administered, the intravenous group had an increase in serum potassium ranging from 0.10 to 0.17 mEq/L per 10 mEq administered while patients that received oral potassium had a change in potassium that ranged from 0.12 to 0.13 mEq/L per 10 mEq with no discernable pattern across different BMI classifications (Table 4).

Discussion

This study evaluated of repletion of potassium in the inpatient hospital setting to test the common practice of providing 10 mEq of potassium for every 0.1 mEq increase in potassium desired. We found that for every 10 mEq of potassium administered, overall there was a mean increase in serum potassium of 0.13 mEq/L. Intravenous potassium increased the serum potassium levels a little more than oral potassium (0.14 per 10 mEq versus 0.12 per 10 mEq administered, respectively). Therefore, oral potassium replacement, in patients with normal GI function, can rival the effects of intravenous replacement.

Guidelines for intravenous management of potassium supplementation have been proposed but have not taken into account BMI, renal function, or adrenal disorders [16]. In comparison to prior studies, which focused on a critically ill patient population, our numbers were in line with these studies in the prescribing patterns and frequency of adverse effects of potassium chloride administration in patients in the medical intensive care unit. These studies also echoed our findings as the average increase in serum potassium level per 20 mEq infusion was 0.22-0.25 mEq/L. Also, similar to these studies, our study also had a small population of patients with poor renal function [7,10,12]. In the current study, mild hypokalemia existed in 43% of the cohort while 56% had a normal serum potassium of ≥ 3.5 mEq/L. At Grady Health System, a serum potassium level lower than 4 mEq/L is usually repleted. This helps explain the high number of patients in the study with a potassium level that is considered normal. Few patients had moderate to severe hypokalemia. Most moderate to severely hypokalemic patients were eliminated from our study due to exclusion criteria such as receiving concomitant IV and PO potassium therapy. There is no data to suggest that the degree of baseline hypokalemia will exponentially increase the amount of repletion necessary. Therefore, the lack of representation of patients with baseline moderate or severe hypokalemia is not thought to be a limitation to allowing the results of the study to be applied to patients with all degrees of hypokalemia. Further examination of baseline characteristics reveal that the majority of patients were on a medical-surgical floor at the time when their first dose of potassium was administered for repletion. Relative to the higher acuity of patients generally found on the other floors from which the data was attained, it can be derived that the patients included in this study were of a lower acuity.

Regarding the impact of concurrent medications presumed to decrease serum potassium levels, it appears that medications presumed to decrease serum potassium level did lower the amount by which serum potassium levels increased per 10 mEq of potassium administered as compared to the overall mean increase in serum potassium level in all patients. However, the number of patients in this category was small and makes it difficult to make generalizations. Patients that received medications thought to increase serum potassium levels had an increase above the overall mean when they received oral potassium but not when they received intravenous potassium.

In assessing the impact of renal function on change in serum potassium level, no conclusions could be drawn from the current sample because very few patients had a GFR of < 30 mL/min/1.73 m^2. This may be due to the fact that patients of a lower renal function were purposefully not administered potassium by providers due to concerns of accumulation. A larger patient population may allow for generation of meaningful results and conclusions.

We did not find any patterns to the effect of BMI on the response to potassium repletion. Patients with a normal BMI that received intravenous potassium (but not oral) had a greater increase in serum potassium level per 10 mEq of potassium administered when compared to the other BMI categories, oral or IV (0.17 mEq/L increase versus 0.10-0.13 mEq/L, respectively). The reason for this is not obvious and a further study would need to be done to clarify this relationship. Interestingly, when categorized in terms of BMI, oral potassium supplementation was more potent in the obese and underweight and equal in the mildly obese. So, the greater effect of intravenous potassium was accounted for by the much greater response in the normal weight group.

Limitations of the study include the following. This study was a retrospective chart review and therefore depended on the accuracy of what was reported in the electronic medical records. Also, there was a small sample size and therefore there was an inability to show a difference, especially with respect to the secondary outcomes. Laboratory values were all within 18 hours after the initial value as is common with clinical practice but it was not at a specific time as in a prospective trial. There also was an inability to account for all sources of potassium loss or gain.

Conclusion

In conclusion, the results reveal that every 10 mEq of potassium administered increase serum potassium levels by a mean value of 0.13 mEq/L. This is somewhat greater but still along the lines of the dogma that every 10 mEq of replacement causes a 0.1 mEq/L increase in serum potassium. The results further reveal that intravenous potassium appears to impact serum potassium levels similarly to the impact of oral potassium. Specifically, intravenous and oral potassium administration caused a mean 0.14 and 0.12 mEq/L increase in serum potassium level per 10 mEq administered, respectively.

Information about Presentation of the Work as an Abstract or Poster

Methods and preliminary results were presented at the University Health System Consortium Poster Session, which was held December 2012 in Las Vegas, NV.

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