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Maximum serum K^+ concentration within 1 hour with enteral replacement in severe hypokalemia

Concentración sérica máxima de K^+ en 1 hora por vía enteral en hipopotasemia severa

Juan de Dios Yépez Florián[1], Gabriela Estefanía Yépez Figueroa[2]

[1]Grupo de Investigación "Fisiología y medicina en diferentes altitudes" (FIMEDALT). Universidad Nacional Mayor de San Marcos. Department of Internal Medicine. Hospital 1 La Esperanza. Trujillo, Perú. [2]Facultad de Medicina Humana. Universidad Privada Antenor Orrego. Trujillo, Perú

Abstract

Introduction: we report two cases with severe hypokalemia.

Patients and methods: a 68-year-old woman was admitted with lower limb swelling and urinary symptoms; on the fourth day serum K^+ concentration (s[K^+]) was 2.3 mmol/L. A 64-year-old woman was admitted with pain in the lumbosacral spine, she was diagnosed with multiple myeloma. After receiving specific therapy she showed s[K^+] at 2.4 mmol/L. A KCl solution containing 26.8 mEq of K^+ was administered enterally, which increased s[K^+] by 0.7 mmol/L within 1 h.

Results and conclusion: these cases reveal that peak s[K^+] may be achieved within 1 hour after KCl intake in severe hypokalemia, which is probably faster than IV administration.

Keywords:

Hypokalemia. Severe hypokalemia. Enteral potassium replacement.

Resumen

Introducción: se presentan dos casos clínicos con hipopotasemia severa.

Pacientes y métodos: mujer de 68 años que ingresó por edema en miembros inferiores y síntomas urinarios; al cuarto día, el nivel sérico de K^+ ([K^+]s) era de 2,3 mmol/L. Una mujer de 64 años ingresó por dolor en la columna lumbosacra y fue diagnosticada de mieloma múltiple; luego de recibir terapia específica, presentó una [K^+]s de 2.4 mmol/L. Se administró por vía enteral una solución de KCl que contenía 26,8 mEq de K^+, aumentando la [K^+]s en 0,7 mmol/L en 1 h.

Resultados y conclusión: estos casos revelan que la [K^+]s máxima se alcanzaría 1 hora después de la ingestión de KCl en la hipopotasemia grave, probablemente en menos tiempo que por vía intravenosa.

Palabras clave:

Hipopotasemia. Hipopotasemia severa. Corrección enteral de potasio.

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Correspondence:

Juan de Dios Yépez Florián. Grupo de Investigación "Fisiología y medicina en diferentes altitudes" (FIMEDALT). Universidad Nacional Mayor de San Marcos. Department of Internal Medicine. Hospital 1 La Esperanza. Trujillo, Perú
e-mail: jdyf73@gmail.com

INTRODUCTION

Hypokalemia is a common electrolyte disorder in the hospital setting, the most common cause being the use of drugs (1,2).

The levels of s[K^+] range from 3.5 to 5.0 mmol/L (1). Concentrations below the lower limit are known as hypokalemia. Severe hypokalemia corresponds to s[K^+]s < 2.5 mmol/L (3,4). The mean daily potassium requirement is 1,800 mg. Total body K^+ stores are 50 mEq/kg (5). A total of 2 % of K^+ is found outside the cell, and 98 % in the cytosol (78 % in the muscle) (6).

The resting membrane potential is the result of the diffusion potentials provided by sodium and, to a greater extent, by K^+. This potential ranges from -10 mV (erythrocytes) to -90 mV (skeletal muscle). A decrease in s[K^+] hyperpolarizes the cells, altering their physiology (3).

The possible muscular changes and cardiac rhythm changes make it necessary to reduce replacement time of s[K^+] by the safest route. Reaching peak serum K concentration in the shortest possible time is one of the main targets in this type of patients.

CASE REPORTS

CASE #1

A 68-year-old woman was admitted to the ER with lower limb swelling, and urinary incontinence, dysuria, frequency, and tenesmus. She received a 20-mg course of IV furosemide and 8 hours later 20 mg via IV for the last time. The lab test results showed creatinine levels at 4.66 mg/dl, urea at 173 mg/dl, and hemoglobin at 4.9 g/dl. She received transfusion of 2 RBC packs. On day 4 the analysis of arterial gases and electrolytes (AGA), performed on an ABL 800 FLEX automated machine, showed s[K^+] at 2.3 mmol/L. With stable vital signs and no gastric, muscular, or cardiac symptoms, liquid KCl diluted in 150 mL of water was administered orally at a dose of 26.8 mEq using a 10-mL vial of 20 % KCl (1 g KCl contains 13.4 mmol or 524 mg of K^+). No adverse events were reported. Electrolytes were measured at 1 h and 2 h (Table I).

CASE #2

A 64-year-old woman was admitted to the ER with chronic pain in the lumbosacral spine radiating to the hips, with 8/10 on the VAS (visual analog scale). A lumbosacral spine magnetic resonance imaging scan revealed findings associated with multiple myeloma and 30 % stenosis of the medullary canal at L4 level. The lab test results showed hemoglobin at 6.7 g/dL, platelets at 459/mm^3, mean corpuscular volume at 86.9 fL, mean corpuscular hemoglobin at 29.0 pg, creatinine at 0.71 mg/dl, urea at 30 mg/dl, total proteins at 11.66 g/dl, albumin at 2.86 g/dl, and globulins at 8.8 g/dl. A total of 3 RBC packs were transfused. Fifteen days after the bone marrow aspiration, thalidomide 200 mg PO every 24 h was started. The next day, due to pulmonary congestion, the patient received furosemide 20 mg IV at 10:00 a.m. and 4:00 *p.*m. At 24 h, in the AGA, s[K^+] was 2.4 mmol/L. With stable vital signs and no symptoms, 26.8 mEq of liquid KCl were diluted in 150 ml of water that were administered orally. Electrolytes were measured at 1 h and 2 h (Table II). Afterwards, at 2:30 a.m., the patient developed nausea with an episode of vomiting.

Table I. Lab test results for case #1

Parameter	Result 0 h	Result 1 h	Result 2 h	Reference range
Sodium, mmol/L	143	141	143	136-146
Potassium, mmol/L	2.3	3	2.9	3.5-5.0
Chlorine, mmol/L	113	115	116	98-106
Bicarbonate, mmol/L	22.7	21.5	22.1	21-28
Calcium, mmol/L	1.03	1.02	1.03	1.15-1.29
pH	7.46	7.45	7.46	7.35-7.45
Lactate, mmol/L	1.8	1.3	1.2	0.5-1.6

Table II. Lab test results for case #2

Parameter	Result 0 h	Result 1 h	Result 2 h	Reference range
Sodium, mmol/L	131	129	130	136-146
Potassium, mmol/L	2.4	3.1	3.0	3.5-5.0
Chlorine, mmol/L	95	96	98	98-106

(Continues on next page)

Table II (cont.). Lab test results for case #2

Parameter	Result 0 h	Result 1 h	Result 2 h	Reference range
Bicarbonate, mmol/L	30.4	30.7	30.4	21-28
Calcium, mmol/L	1.06	1.05	1.07	1.15-1.29
pH	7.49	7.51	7.49	7.35-7.45
Lactate, mmol/L	0.9	0.7	0.7	0.5-1.6

DISCUSSION

Symptoms in hypokalemia occur with s[K^+] < 3.0 mEq/L, and those with the highest risk include muscle weakness and heart rhythm abnormalities, which can lead to severe cardiac arrest (7).

Potassium homeostasis is governed by fast and slow regulation mechanisms; the former are mediated by redistribution between the intracellular and extracellular compartments; in the latter, the kidney plays the main role, together with the gastrointestinal tract (GIT) (5,8).

The probable absorptive mechanism of potassium in the small intestine is paracellular passive absorption (solvent drag), which has been proven in animal models but not in humans. This pathway depends on the electrical potential difference generated by the Na/K-ATPase pump in the basolateral membrane. Also, an absorptive model is known in the distal colon that is likely controlled by an active mechanism (5,9,10).

The IV supplementation of K^+ is widely used in severe hypokalemia and/or when cardiac electrical changes occur. The IV infusion rate of K^+ should not exceed 20 mmol/h in adults; higher rates should be infused through a central vein, with close monitoring to reduce the risk of hyperkalemia (11).

Oral compounds are indicated in hypokalemia in the absence of critical signs (4,12), and liquid KCl formulations are preferred (13).

A clinical trial in pediatric patients found a similar elevation of s[K^+] between the enteral and IV administration of KCl. A dose control was performed 1 h after the IV administration and 2 h after the enteral administration (14). Both routes of administration present low risks of adverse effects, ranging from hyperkalemia and complications in the placement of a central venous catheter in the IV route, to digestive discomfort, nausea and vomiting in the enteral route (15), as seen in case #2. Liquid formulations resulted in a lower incidence of GIT adverse events, which were inversely proportional to the dilution of KCl (12).

Our patients with severe hypokalemia where the enteral route was not first-choice (15), received enteral replacement with a good therapeutic response within 1 h. A control measurement of s[K^+] was indicated 2 h after enteral administration (14). In both cases s[K^+] showed a peak increase after 1 h, and a slight decrease after 2 h, suggesting possible gastric absorption mechanisms. Therefore, replacement time would be shorter by the enteral route using liquid KCl (16).

We should always take into account the etiology of hypokalemia since, despite reaching the target correction level (≥ 3.0 mmol/L), s[K^+] could decrease if the triggering cause remains untreated, which would break down into 3 possibilities: deficient supply, redistribution changes from extracellular to intracellular, or either renal or gastrointestinal potassium losses (1,4). In addition, consider the appearance of new hypokalemic events or the difficult task of replenishing K^+ in a dysfunctional GIT (short intestine), situations in which absorption must be maximized using repeated enteral doses up to 4 times daily, plus the possibility of IV supplementation to achieve normokalemia (15,17).

These cases describe an observation. Our goal was to report that peak s[K^+] were achieved 1 hour after KCl administration by the enteral route in subjects with severe hypokalemia, in order to promote the development of controlled research to study the effectiveness of the enteral route absorptive mechanisms (kaliuresis) (13) using liquid KCl (15).

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