AJKD

Acid-Base and Electrolyte Teaching Case

Severe Hypokalemia in a Patient With Subarachnoid Hemorrhage

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Hypokalemia is a common electrolyte disorder in the intensive care unit. Its cause often is complex, involving both potassium losses from the body and shifts of potassium into cells. We present a case of severe hypokalemia of sudden onset in a patient being treated for subarachnoid hemorrhage in the surgical intensive care unit in order to illustrate the diagnosis and management of severe hypokalemia of unclear cause. Our patient received agents that promote renal potassium losses and treatments associated with a shift of potassium into cells. We outline the steps in diagnosis and management, focusing on the factors regulating the transcellular distribution of potassium in the body.

Am J Kidney Dis. 63(3):530-535. © *2014 by the National Kidney Foundation, Inc.*

INDEX WORDS: Hypokalemia; transcellular potassium homeostasis; hypothermia; subarachnoid hemorrhage; neurosurgery; barbiturate.

Note from Feature Editor Jeffrey A. Kraut, MD: This article is part of a series of invited case discussions highlighting the diagnosis and treatment of acid-base and electrolyte disorders.

INTRODUCTION

Hypokalemia, defined as serum potassium concentration <3.5 mEq/L, is a common electrolyte disorder in hospitalized patients, with a prevalence of 21%.[1] In the surgical intensive care unit, the prevalence increases to as high as 40%.[2] Severe hypokalemia (serum potassium, <3.0 mEq/L) increases the risk of cardiac arrhythmias and sudden death, and mortality increases with the severity of the hypokalemia.[1-3] In most instances, hypokalemia is caused by potassium losses, but more rarely, it can result from a sudden shift of potassium into cells. We present a case of severe hypokalemia in a critically ill patient with a subarachnoid hemorrhage. Using this case as an example, we discuss the diagnosis and management of severe hypokalemia, focusing on factors that cause a shift of potassium into cells.

CASE REPORT

Clinical History and Initial Laboratory Data

A 45-year-old man collapsed after drinking 2 bottles of beer and had a witnessed seizure. According to his wife, he had no history of hypertension, thyroid problems, kidney disease, or electrolyte abnormalities. He was not taking medications or herbal supplements. He was a smoker and consumed moderate amounts of alcohol. There was no family history of electrolyte disorders. On evaluation, his initial blood pressure was 230/134 mm Hg and he had a Glasgow coma score of 9. He had labored breathing and oxygen saturation of 89% on room air. Computed tomography showed extensive subarachnoid and intraventricular hemorrhage. He was intubated and given lorazepam, succinylcholine, and etomidate intravenously, then started on a propofol drip. Serum potassium concentration, drawn shortly after induction of anesthesia, was 3.5 mEq/L and kidney function was normal (Table 1). His intracranial pressure was high at 23 mm Hg, an external ventricular drain was placed, and he underwent cerebral angiography with coil embolization of a ruptured left anterior communicating artery aneurysm.

After this procedure, the patient received 2 doses of mannitol, 3% sodium chloride solution, and sodium acetate intravenously to induce hypertonicity and alkalemia (Fig 1). Treatment with propofol was continued, and fentanyl and phenytoin were added. That evening, his temperature increased to 38.4°C and treatment with ceftriaxone was started. After induction of hypertonicity, serum potassium concentration increased transiently to 4.1 mEq/L (Table 1). During the next 8-10 hours, his intracranial pressure remained high and cerebral perfusion pressure remained low. His pupils were not dilated but were nonreactive, and systolic blood pressure ranged from 100-120 mm Hg. The ventilator settings were adjusted to try to maintain P_{CO_2} of 30-35 mm Hg. On the late morning of day 2 (Fig 1), a pentobarbital coma was induced. Because his cerebral perfusion pressure remained <70 mm Hg, a norepinephrine infusion was begun. In midafternoon, hypothermia was induced with a goal body temperature of 34°C. Serum potassium concentration on the morning of day 2 was 3.2 mEq/L, and the patient was given 40 mEq of potassium chloride intravenously. As shown in Fig 1, serum potassium concentrations decreased precipitously during the day to a nadir of 1.6 mEq/L at 11:00 PM. Despite receiving a total of 160 mEq of potassium chloride intravenously, the severe hypokalemia persisted. An electrocardiogram showed sinus bradycardia and flattened T waves.

Additional Investigations

Urine sodium and chloride concentrations were <5.0 mEq/L and urine potassium concentration was 5.8 mEq/L (Table 1). Serum osmolality was 339 and urine osmolality was 89 mOsm/kg of H_2O. Plasma renin activity was 9.7 ng/mL/h and serum

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Received April 4, 2013. Accepted in revised form July 2, 2013. Originally published online August 22, 2013.

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0272-6386/$36.00

http://dx.doi.org/10.1053/j.ajkd.2013.07.005

Table 1. Serial Laboratory Data

Parameter	Day 1		Day 2		Day 3	Day 4
	1:00 AM	6:00 PM	AM	PM		
Sodium (mEq/L)	137	150	151	159	161	161
Potassium (mEq/L)	3.5	4.1	3.2	1.6[a]	2.0	5.3
Chloride (mEq/L)	103	123	120	121	125	126
Total CO_2 (mEq/L)	19	22	26	30	29	25
SUN (mg/dL)	15	12	8	—	8	12
Creatinine (mg/dL)	1.07	—	0.9	—	0.82	1.36
eGFR (mL/min/1.73 m^2)	75	—	91	—	102	—[b]
Magnesium (mg/dL)	—	1.8	2.5	—	2.6	2.7
Calcium (mg/dL)	—	—	9.2	—	—	—
pH	7.32	7.44	7.43	—	7.48	7.37
P_{CO_2} (mm Hg)	41	25	47	—	35	41
P_{O_2} (mm Hg)	70	79	117	—	55	74
Serum osmolality (mOsm/kg)	279	317	318	333	339	344
Urine osmolality (mOsm/kg)	—	—	—	—	89	308
Urine sodium (mEq/L)	—	—	—	—	<5	129
Urine potassium (mEq/L)	—	—	—	—	5.8	18
Urine chloride (mEq/L)	—	—	—	—	<5	125
24-h input/output (mL)[c]	3,983/4,070		6,306/1,640		6,584/1,723	1,593/94
24-h gastric output (mL)	550		975		100	—

Note: Morning measurements unless otherwise noted. eGFR calculated using the 4-variable MDRD (Modification of Diet in Renal Disease) Study equation. Conversion factors: creatinine in mg/dL to μmol/L, ×88.4, calcium in mg/dL to mmol/L, ×0.2495, magnesium in mg/dL to mmol/L, ×0.411, SUN in mg/dL to mmol/L, ×0.357.

Abbreviations: CO_2, carbon dioxide; eGFR, estimated glomerular filtration rate; SUN, serum urea nitrogen.

[a]Nadir serum potassium concentration value was 1.6 mEq/L at 11:00 PM.

[b]Patient oliguric, calculation does not apply.

[c]Output includes both urine and gastric drainage.

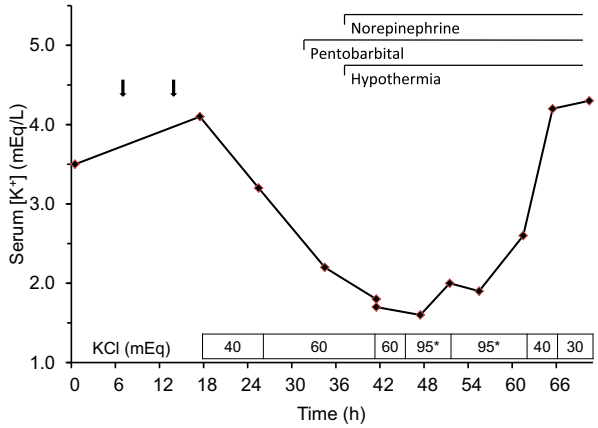

Figure 1. Serial laboratory results. Time course of serum potassium concentration [K^+] during hospitalization. The down arrows indicate intravenous mannitol administration, 75 g each time. The timing of and amount of intravenous potassium replacement is shown at the bottom of the graph. Other therapeutic interventions are indicated at the top of the graph. Day 1 of hospitalization is from 0 to 23 hours, day 2 is from 24 to 47 hours, and day 3 is after 48 hours. Abbreviation: KCl, potassium chloride.

*Denotes that 15 mEq of potassium was given as potassium phosphate on 2 occasions on day 3.

aldosterone level was <4.0 ng/dL. Plasma metanephrine and normetanephrine levels were <0.20 (reference, <0.50) nmol/L and 0.71 (reference, <0.90) nmol/L, respectively.

Diagnosis

Severe hypokalemia from renal losses, followed by an abrupt shift of potassium into cells. Contributing factors to the intracellular shift include alkali administration, an endogenous adrenergic surge, pentobarbital administration, and hypothermia.

Clinical Follow-up

On hospital day 3, the patient received an additional 230 mEq of potassium chloride and 30 mEq of potassium phosphate intravenously. He remained hypothermic and all the above medications were continued. His serum potassium concentration increased abruptly from 1.9 to 4.3 mEq/L (Fig 1). On hospital day 4, serum potassium concentration increased further to 5.3 mEq/L, intracranial hypertension and decreased cerebral perfusion persisted, and the patient became oliguric. After discussion of the prognosis with his family, the patient was terminally extubated. No autopsy was performed.

DISCUSSION

Normal body potassium stores are related directly to body muscle mass because >60% of potassium resides in muscle cells. In a 70-kg man, the value