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Case Report

Case Based Discussion: Severe Hypokalemia and Cardiac Arrest in Normal Healthy Adult Male with No Prior Co-Morbidities

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Abstract

Hypokalemia is a common electrolyte imbalance when serum levels of potassium are low and may require urgent medical intervention. It usually results from increased potassium excretion or intracellular shift and reduced potassium intake or due to renal, genetic or endocrine diseases. Hypokalemia can be severe and life threatening when potassium levels are <2.5mEq/L. Here we present a case of 30 years old male with no prior co-morbidities presented with 2-4 days history of fever, generalized weakness and difficulty in waking was admitted in wards where he suddenly became unresponsive, had cardiac arrest so immediately CPCR given, required multiple D.C. shocks. ROSC achieved after 30 minutes. On evaluation found to have very low serum potassium (1.5mEq/L), corrected. After 7 days patient improved completely and discharged from the hospital.

Keywords: Hypokalemia; Magnesium; Electrolyte; D.C. Shock; Cardiac arrest; Vfib; VT

Introduction

Hypokalemia can be caused either by decreased intake of potassium or by excessive losses of potassium in the urine or through gastrointestinal tract [1,2]. Hypokalemia-serum K^+ below 3.5mEq/L can be the result of K^+ movement into cells or due to total body K^+ depletion.

Transcellular shift

The movement of K^+ into cells is due to stimulation of β2 adrenergic receptors on cell membranes in muscle, and this is the cause of decrease in serum K^+ associated with inhaled β2-agonist bronchodilators. This effect is mild-0.5mEq/L in the usual therapeutic doses, but is more significant when diuretics used along with inhaled β2-agonist bronchodilators. An intracellular shift of the potassium can also lead to severe hypokalemia: insulin administration, stimulation of the sympathetic nervous system, thyrotoxicosis, and familial periodic paralysis [3]. Other conditions include respiratory or metabolic alkalosis, hypothermia and insulin.

Potassium depletion

Potassium depletion is due to K^+ loss via kidneys or gastro-intestinal tract, which can be identified by measuring the urinary K^+ and chloride concentrations.

Renal potassium loss

Diuretics: Thiazides, Loop or Osmotic diuretics Laxatives, Amphotericin-B, Antipseudomonal penicillins (carbenicillin), Penicillin in high doses Theophylline, nasogastric drainage, alkalosis, and magnesium deficiency. Nasogastric drainage has a low concentration of K^+ 10–15 mEq/L, but loss of volume and H^+ promotes K^+ loss in the urine. The urine chloride is low <15mEq/L with nasogastric drainage and alkalosis, and is high >25mEq/L with diuretic therapy and magnesium deficiency. Magnesium depletion impairs K^+ reabsorption in renal tubules and may play a very important role in promoting K^+ depletion in critically ill patients, especially those receiving diuretics.

Extrarenal potassium loss

Diarrhea: Normal K^+ loss in stool is only 5–10 mEq/day, which can increased up to 15-40 mEq/day in secretory and inflammatory diarrhea, the daily volume of stool can be 10 liters in severe cases, therefore, K^+ losses can reach 400mEq daily in severe cases of inflammatory or secretory diarrhea. Endocrine diseases such as primary hyperaldosteronism, kidney disorders and genetic syndromes affecting the renal function. Congenital adrenal hyperplasia due to enzymatic defects is a genetic syndrome strongly associated with hypertension and hypokalemia, resulting from excessive mineralo-corticoid effects. More than 50% of clinically significant hypokalemia has associated magnesium deficiency (in patients receiving loop or thiazide diuretics). Hypokalemia associated with magnesium deficiency is often refractory to treatment with K^+. Other causes are Polyuria: healthy subjects can lower their K^+ concentration to 5-10 mEq/L, if urine out put is >5-10 L/day than potassium loses can exceed 50-100 mEq this is usually seen in primary polydipsia.

Transcuteneous loss

Daily K^+ loss through skin is negligible due to low volume and K^+ concentration is only 5-19 mEq/L, This loss may increase due to excessive perspiration leading to potassium depletion. Urinary K^+ excretion may also contribute due to increase release of aldosterone by both exercise (by catecholamine induced rennin secretion) and volume loss.

Prevalence: Patients attending outpatient department have mild hypokalemia about 14%, while it is 20% in hospitalized patients,

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which is clinically significant only in 4-5%. 80% of patients who receive diuretics have hypokalemia, associated with systemic disease. Prevalence between males and females is similar.

Clinical manifestation

Severe hypokalemia serum K^+ <2.5mEq/L can be associated with diffuse muscle weakness [4]. Symptoms can vary from absent to lethal arrhythmias [5], and according to system affected:

Renal: Metabolic acidosis, rhabdomyolysis. Adrenal steroid excess (Cushing's syndrome), Primary hyper-aldosteronism, renin-secreting tumors, Glucocorticoid-remediable congenital adrenal hyperplasia. Ingestion of substances such as glycyrrhizin, Bartter syndrome, Gitelman syndrome Liddle syndrome, renal tubular acidosis Fanconi syndrome.

Nervous system: Leg cramps, weakness, paresis or ascending paralysis. Constipation or intestinal paralysis. Respiratory failure often present as signs of severe hypokalemia.

Cardiac: Hypokalemia can have detrimental effects on the cardiovascular system, leading to ECG changes (U waves, T wave flattening and ST-segment changes), sometimes-lethal cardiac arrhythmias and heart failure [6].

Diagnosis: Detailed medical history and physical examination can reveal the probable cause of hypokalemia.

Laboratory investigations

Serum sodium, potassium, glucose, chloride, bicarbonate, BUN and creatinine. Serum and urinary potassium levels is required to evaluate severity Urine electrolytes (potassium and chloride) in spot to differentiate renal from non-renal causes of hypokalemia, arterial blood gas analysis to rule out metabolic acidosis or alkalosis when the underlying cause is not known. Urine analysis and urine PH to assess the presence of renal tubular acidosis. Serum magnesium, calcium and/or phosphorus levels are important to exclude associated electrolyte abnormalities. Urinary calcium excretion to exclude Bartter syndrome. Serum digoxin level if the patient is on digitalis, a drug screen in urine and/or serum for diuretics, amphetamines and other sympathomimetic stimulants. TSH levels in cases of tachycardia or clinical suspicion of hypokalemic periodic paralysis [7]. In general, there are two major components of the diagnostic evaluation: (a) assessment of urinary potassium excretion in order to distinguish renal potassium losses (e.g., diuretic therapy, primary aldosteronism) from other causes of hypokalemia (e.g., gastrointestinal losses, transcellular potassium shifts) and (b) assessment of acid–base status. Potassium excretion in a 24h urine collection is the best way to assess the urinary potassium excretion. If this excretion is above 15mEq of potassium per day, this is a direct indication of inappropriate renal potassium loss. Potassium and creatinine concentrations in a spot urine is an alternative. Spot urine potassium to creatinine ratio greater than 13mEq/g creatinine indicates inappropriate renal potassium loss. After determining whether renal potassium wasting is present, assessment of acid–base status can further narrow the differential diagnosis [8,9]. ECG shows ST segment depression, decrease in the amplitude of the T wave and an increase in the amplitude of U waves in V4 to V6. Arrhythmias may be associated with hypokalemia, including sinus bradycardia, premature atrial and ventricular beats, paroxysmal atrial or junctional tachycardia, atrioventricular block, ventricular tachycardia or fibrillation [10].

Management of hypokalemia

The first concern in hypokalemia is to eliminate or treat any condition that promotes transcellular potassium shifts e.g., alkalosis [3]. If hypokalemia is due to K^+ depletion:

Estimate Potassium Deficits: About 10% of total body K^+ is lost for every 1mEq/L decrease in serum K. For a 70kg adult with a normal total body K^+ of 50mEq/kg, the estimated deficits associated with progressive hypokalemia. Even mild hypokalemia-3mEq/L is associated with a considerable K^+ deficit-175mEq.

Fluids: The usual replacement fluid is potassium chloride, which is available as a concentrated solution (1–2 mEq/mL) in ampules containing 10, 20, 30, and 40 mEq of potassium. These solutions are extremely hyperosmotic (the 2mEq/mL solution has an osmolality of 4,000mosm/kg H_2O) and must be diluted. A potassium phosphate solution is also available that contains 4.5mEq potassium and 3mmol phosphate per mL, and this solution is preferred by some for potassium replacement in diabetic ketoacidosis (because of the phosphate depletion in diabetic ketoacidosis).

Rate of replacement: The standard method of intravenous K^+ replacement is to add 20mEq of K^+ to 100mL of isotonic saline and infuse this mixture over 1 hour, maximum rate of potassium replacement is 20mEq/hr, but rates of 40mEq/hour may be necessary (e.g., serum K^+ <1.5mEq/L or serious arrhythmias, (rates as high as 100mEq/hour have been used safely). Infusion through a large, central vein is preferred, if possible, because of the irritating properties of the hyperosmotic KCL solutions, delivery into superior vena cava is not recommended if the desired rate of replacement exceeds 20mEq/hr because there is a risk of an abrupt rise in plasma K^+ in the right heart chambers severe enough to produce asystole.

Response: The serum K^+ may be slow to rise initially as predicted by the flat portion of the curve in if the hypokalemia is resistant or refractory to K^+ replacement, magnesium deficiency should be suspected. Magnesium depletion promotes urinary K^+ loss. Hypokalemia is often refractory to K^+ replacement until the magnesium is corrected. Magnesium deficiency may play an important role in diuretic-induced hypokalemia.

Case Presentation

30 years old male with no prior co-morbidities presented with history of fever, since 2 days, severe body ache generalized weakness ,sweating, and difficulty in waking since few hours, laboratory reports reveal leucocytosis he was hemodynamically stable so admitted in wards where he suddenly became unresponsive had a cardiac arrest immediately CPR given as per AHA guidelines, requiring multiple DC shock in v/o Ventricular fibrillation/ventricular tachycardia, fluid resuscitation given intubated and ventilated, required ionotropic support due to hypotension. ROSC achieved after 30 minutes shifted to ICU for further management and hemodynamic monitoring. Started on sedation, antibiotics, antiepileptics, antimalarials, fluids and other supportive treatment continued. On evaluation found to have very low Potassium (1.5mEq/L), ABG severe metabolic acidosis corrected, leucocytosis, deranged liver enzymes, raised

CRP, PCT and creatinine. Fever work up negative for Dengue, Mlaria, Leptospirosis, urine pus cells 12-14/. Triple H non-reactive. Potassium corrected and monitored 2-D ECHO normal studies. USG abdomen Gall bladder sludge, bilateral mildly bright kidney. Cardiac, Nephrology and Neurology opinion taken which did not reveal any abnormality so correction of potassium and symptomatic treatment continued. MRI Brain reveal hypoxic injury in bilateral basal gangalia and hippocampus. On further work up urine potassium 37.2meq/L, 24 urine volume 42500 (normal- 600-2000 ml/24 hours), 24 hours potassium 930meq (normal 40-80 meq/24hrs). Normal Thyroid profile. Gradually patient improved. ABG and potassium became normal so weaned off and extubated ionotropic support stopped, electrolytes became normal. Patient had complaints of blurring of vision and amnesia, which improved slowly so shifted towards patient improved completely after 7 days therefore discharged from the hospital. On follow up patient was completely healthy and asymptomatic.

Discussion

Physiology of potassium homeostasis

Potassium role in cellular functions: Potassium (K^+) plays a key role in maintaining normal cell function. K^+ is the main intracellular cation and almost all cells have the pump called 'Na^+-K^+-ATPase', which pumps sodium (Na^+) out of the cell and K^+ into the cell leading to a K^+ gradient across the cell membrane (K^+ in > K^+ out), which is partially responsible for maintaining the potential difference across membrane. Many cell functions rely on this potential difference, particularly in excitable tissues, such as nerve and muscle. Two percent of K^+ exists in the extracellular fluid (ECF) at a concentration of only 4mEq/L. Enzyme activities, as well as, cell division and growth are catalyzed by potassium and are affected by its concentrations and its alterations. Intracellular K^+ participates in acid–base regulation through exchange for extracellular hydrogen ions (H^+) and by influencing the rate of renal ammonium production. Counter regulatory mechanisms exist in order to defend against potassium alterations. These mechanisms serve to maintain a proper distribution of K^+ within the body, as well as to regulate the total body K^+ content. Hyperkalemia decreases membrane potential, while hypokalemia causes hyperpolarization and non-responsiveness of the membrane. If K^+ balance is disrupted (hypokalemia or hyperkalemia), this can also lead to disruption of heart electrical conduction, dysrhythmias and even sudden death. Potassium balance has a direct negative effect on (H^+) balance at intracellular and extracellular level and the overall cellular activity.

Balance of K^+

External potassium balance is determined by the rate of potassium intake (normally 100mEq/day) and rate of urinary (normally 90mEq/day) and fecal excretion (normally 10mEq/day). The distribution of potassium in muscles, bone, liver and red blood cells (RBC) and ECF has a direct effect on internal potassium balance [6,7].

Potassium homeostasis

The kidney is primarily responsible for maintaining total body K^+ balance. However, renal K^+ excretion is adjusted over several hours; therefore, changes in extracellular K^+ concentrations are initially buffered by movement of K^+ into or out of skeletal muscle. The regulation of K^+ distribution between the intracellular and extracellular space is referred to as internal K^+ balance. Under normal conditions, insulin and catecholamines play the most important role in this regulation [8]. Potassium controls its own ECF concentrations through a feedback regulation of aldosterone release. An increase in K^+ levels leads to a release of aldosterone through the renin-angiotensin-aldosterone mechanism or through the direct release of aldosterone from the adrenal cortex cells, which are stimulated [9]. An increase in extracellular potassium concentrations stimulates aldosterone secretion (via angiotensin II), which in turn increases urinary K^+ excretion. In the steady state, K^+ excretion matches intake and approximately 90% is excreted by the kidneys and 10% in the stool. The rate of K^+ secretion by the distal nephron varies and is regulated according to the physiological needs. The cellular determinants of K^+ secretion in the principal cell include the intracellular K^+ concentration, the luminal K^+ concentration, the potential (voltage) difference across the luminal membrane and the permeability of the luminal membrane for K^+. Conditions that increase cellular K^+ concentration, decrease luminal K^+ concentration or render the lumen more electronegative will increase the rate of K^+ secretion. Conditions that increase the permeability of the luminal membrane for K^+ will increase the rate of K^+ secretion [8,9]. Two principal determinants of K^+ secretion are mineralocorticoid activity and distal delivery of Na^+ and water. Aldosterone is the major mineralocorticoid in humans and mediates the renal excretion of K^+ and Na^+ reabsorption by binding to the mineralocorticoid receptors in the distal tubules and collecting ducts of the nephron. Aldosterone increases intracellular K^+ concentration by stimulating the activity of the Na^+-K^+-ATPase in the basolateral membrane. The kidneys are far more capable in increasing than decreasing K^+ excretion. As a result, inadequate intake can lead to K^+ depletion and hypokalemia. Hyperkalemia usually occurs when renal excretion is impaired (glomerular filtration rate (GFR) <20mL/min). The case under discussion had no co-morbidities presented with history of fever weakness walking difficulty sweating which all might have contributed to excessive volume loss and potassium depletion and cardiac arrest. The massage is that anybody coming with fever, dehydration, difficulty in walking, excessive perspiration one should always think of hypokalemia and take action accordingly. Further work up to rule out causes of hypokalemia in this case is awaited.

Conclusion

In most patients presenting with hypokalemia, the cause is clear from the history (e.g., vomiting, diarrhea, diuretic therapy). The major components for the diagnostic evaluation: (a) assessment of urinary potassium excretion in order to distinguish renal potassium losses (e.g., diuretic therapy, PA) from other causes of hypokalemia (e.g., gastrointestinal losses, transcellular potassium shifts), and (b) assessment of acid–base status: As hypokalemia is associated with metabolic alkalosis or metabolic acidosis. Management of the underlying disease or contributing factors constitutes the cornerstone of therapeutic approach. Potassium should be gradually replaced, preferably by oral administration if clinically feasible. In cases of severe/symptomatic hypokalemia and cardiac complications, i.v. administration with continuous ECG monitoring is recommended. In some patients, such as in endocrine related hypokalemia cases, multidisciplinary diagnostic and therapeutic approach is needed.

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Conflict of Interest

None to declare.

Patient Consent

Consent of patient taken.

References

1. Khitan ZJ, Shweihat YR, Tzamaloukas AH, Shapiro JI. Dietary potassium and cardiovascular profile. Results from the modification of diet in renal disease dataset. Journal of Clinical Hypertension. 2018: 611–612.
2. Gumz ML, Lynch IJ, Greenlee MM, Cain BD, Wingo CS. The renal H^+-K^+-ATPases: physiology, regulation, and structure. American Journal of Physiology: Renal Physiology. 2010; 298: F12–F21.
3. Schaefer TJ, Wilford RW. Disorders of potassium. Emerg Med Clin North Am. 2005; 23: 723-747.
4. Patel K, McCoy JV, Davis PM. Recognizing thyrotoxic hypokalemic periodic paralysis. JAAPA. 2018; 31: 31–34.
5. Tran TTT, Pease A, Wood AJ, Zajac JD, Mårtensson J, Bellomo R, et al. Review of evidence for adult diabetic ketoacidosis management protocols. Frontiers in Endocrinology. 2017; 8: 106.
6. Zacchia M, Abategiovanni ML, Stratigis S, Capasso G. Potassium: from physiology to clinical implications. Kidney Disease. 2016; 2: 72–79.
7. Papademetriou V. Diuretics, hypokalaemia, and cardiac arrhythmia: a 20-year controversy. Journal of Clinical Hypertension. 2006; 8: 86.
8. Thomas CP. Metabolic acidosis. In Medscape Ed V Batuman. New York, NY, USA: WebMD LLC. 2017.
9. Kovesdy CP, Appel LJ, Grams ME, Gutekunst L, McCullough PA, Palmer BF, et al. Potassium homeostasis in health and disease: a scientific workshop cosponsored by the National Kidney Foundation and the American Society of Hypertension. Journal of the American Society of Hypertension. 2017; 11: 783–800.
10. Chua CE, Choi E, Khoo EYH. ECG Changes of severe hypokalaemia. QJM. 2018; 111: 581-582.