ACR

Case Report

Hypokalaemia: case report and review of the literature

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Abstract

Hypokalaemia is a relatively common clinical diagnosis compared to hyperkalaemia. It's presentation could be dramatic, especially in patients with no prior history or predisposing factors. A prompt diagnosis and management of this condition is often lifesaving, considering the risk of arrhythmias common in them.

A case of hypokalaemia which was aggravated by innocent medical intervention is reported. Good clinical intuition coupled with prompt simple laboratory investigations led to a favourable outcome. The hospital notes of the patient and literature on hypokalaemia were reviewed.

There is a need to expect and prepare for the unexpected in clinical practice especially in the private hospital setting where there is a limited availability of specialist skills.

Keywords: Hypokalaemia, Plasma potassium

Introduction

The plasma potassium (K^{+}) level is normally maintained within narrow limits (3.5-5.0mmol/L) by multiple mechanisms that make up potassium homeostasis.[1] It's regulation is strictly maintained, in view of the role potassium plays in the many important physiologic processes such as, resting cellular membrane potential and the propagation of action potentials in neuronal, muscular and cardiac tissues, along with hormone secretion and action, vascular tone, systemic blood pressure control, gastrointestinal motility, acid-base homeostasis, glucose and insulin metabolism, mineral ocorticoid action, renal concentrating ability and fluid and electrolyte balance.[1,2,3,4]

Hypokalaemia is defined as plasma K^{+} level less than 3.5mmol/L.[5] The importance of hypokalaemia in clinical practice is underscored by the well-established findings by Goyal A, et al[6] and Torlen K, et al,[7] who found that patients who have hypokalaemia, tend to have an increased rate of death from any cause. In addition derangements of K^{+}, be it hypokalaemia or hyperkalaemia, have been associated with numerous pathophysiologic processes especially involving the heart and the kidneys.[1,2,8]

Total body potassium is influenced by age, sex and very importantly muscle mass, because most of the body's potassium is contained in muscle. An adult male has a total body K^{+} of 50 mmol/kg of body weight. And approximately 98% of the total body K^{+} is found in the intracellular fluid (ICF) space at a concentration of 100-150mmol/L, depending on the cell type.[9]

Hypokalaemia is found in about 20% of hospitalized patients,[10,11] with majority of these patients having plasma potassium concentrations between 3.0 and 3.5mmol/L, but a significant percentage of them have K^{+} less than 3.0mmol/L. While comparable data are not available for outpatients, a low serum potassium concentration has been found in 10 to 40% of patients treated with thiazide diuretics.[12] Hypokalaemia is usually well tolerated in otherwise healthy people but it can be life threatening when severe. However even mild or moderate hypokalaemia increases the risk of morbidity and mortality in patients with cardiovascular disease.[10]

Case Study

Mrs. JA a 35year old petty trader presented at the emergency room of a private hospital in the south

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western region of Nigeria with a five day history of frequent passage of large quantities of watery stool and a four day history of nausea and vomiting. Vomiting had ceased a day prior to presentation. Her stool was neither mucoidnor blood stained and contained only specks formed feces, she moved her bowel between 5 to 7 times daily, there was associated mild generalized abdominal pain. Vomiting started the same day as the stooling, was neither projectile nor bilious, it was associated with nausea but not related to time of food intake, there was associated, anorexia and but no history of fever. Prior to presentation she had visited a patent medicine seller who gave her four different medications, none of which she could identify, with no relief.

On examination patient was conscious, but lethargic, she was, a febrile, anicteric and not pale, she was moderately dehydrated, (despite intake of salt- sugar solution at home), her pulse rate was 100/minutes small volume, blood pressure was 100/50mmHg. Initial investigations done were as follows:

Analyte Reference range

Sodium	149mmol/L	130-145mmol/L
Potassium	2.5mmol/L	3.5- 5.0mmol/L
Urea	42mmol/L	25-70umol/L
Creatinine	73mmol/L	44-132umol/L

Full blood count (FBC): Packed Cell Volume (PCV) - 39%, white cell count – 5,500 cells/ml, platelet count- 210,000/ml

Pregnancy test (PT) - negative

On urinalysis, the urine PH was 6.5, specific gravity 2.030 with no leucocytes, protein, blood, glucose, ketones or nitrites. A working diagnosis of infective gastroenteritis with hypokalaemia was made and patient was commenced on intravenous infusion of 5% dextrose in saline, with an initial dose of 50mls of 50% glucose added to the first infusion. Intravenous metronidazole and ciprofloxacin tablets 500mg twice daily were also started, after a stool sample had been collected and sent to the laboratory for microbiology studies.

Patients initially improved dramatically, however by the next day she complained of moderate to severe muscle weakness and cramps, with a muscle power of grade 2. A repeat electrolytes urea and creatinine, revealed a drop in plasma k^+ to 2.0mmol/L. The dextrose saline infusion was discontinue and replaced with normal saline and 40mmol of potassium chloride KCL per litre of infusion was commenced. The patient's muscle strength rapidly returned to normal while the diarrhea had subsided and patient's appetite markedly improved. A repeat plasma k^+ done on the third day of admission revealed a level of 2.0mmol/L, with sustained improvement; the patient was discharged home on the fourth day with a K^+ of 3.1mmol/L.

Discussion

The patient came in with moderate true deficit hypokalaemia as a result of massive loss of K^+through diarrhea and vomiting as a result of the gastroenteritis she had, coupled with poor dietary intake as a result of the associated anorexia. The initial glucose rich infusion she had on her first day of admission, caused an increased secretion of insulin which in turn increased the activity of the Na^+K^+ ATPase, thereby increasing pumping of plasma K^+ into the intracellular fluid, thus creating a redistribution hypokalaemia which worsened the patients K^+ balance. Caseation of diarrhea on treatment, which stemmed further K^+ loss, discontinuation of glucose infusion and potassium replacement coupled with an improved oral dietary intake all contributed to the patient's improvement.

Total body K^+ homeostasis requires appropriate internal distribution of K^+ and maintenance of an external K^+ balance. Regulation of internal K^+ balance refers to the regulation of the critical concentration of K^+ gradient across cell membranes. Regulation of the external K^+ balance refers to the regulation of the total body K^+ content, which is largely dependent on the excretion of K^+ by the kidneys.[9]

Internal potassium Balance: Here K^+ is actively driven into the cells across a concentration gradient by Na^+ K^+-ATPase, while the efflux of K^+ from the cells is passive, since it is along a concentration gradient. Increased plasma K^+ concentration follows the favours the activity of the Na^+ K^+-ATPase by decreasing the concentration gradient across which it has to act. The activity of the Na^+ K^+-AT Pase is increased by the action of insulin, thyroxine and b-adrenergic stimulation. Also b-adrenergic stimulation on its own promotes K^+ uptake by hepatocytes and skeletal and cardiac muscles through beta-2 recepters.[1,9,10,13]

External Potassium Balance: The amount of K^+ in the body is a reflection of the balance between potassium intake and output [9,14]Potassium output or loss from the

body occurs through three primary routes: the gastrointestinal tract (GIT), the skin, and the urine. Under normal conditions the loss of K^+ through the gastrointestinal tract and the skin is not significant but in diarrhea, loss through the GIT could be significant enough to cause hypokalaemia.[9,10] this was the situation with the patient in the case study. Potassium excretion is primarily through the kidneys, the kidney is capable of regulating the excretion of potassium to maintain body K^+ homeostasis

Hypokalaemia

Causes of hypokalaemia are classified into two broad groups: (i) redistribution of extracellular K^+ into the ICF and (ii) true K^+ deficits, caused by decreased intake or loss of potassium rich body fluids.

Redistribution hypokalaemia: causes include insulin release by decreased water or loss of potassium rich body fluids.

Redistribution hypokalaemia: Here there is an increased entrance or shift of K^+ into the cells, thereby lowering the plasma K^+ concentration without necessarily reducing the K^+ store of the body, causes include insulin release due to a glucose load leading to increased action of the Na^+ K^+ AT Pase, catecholamine or b-adrenergic excess, hyperthyroidism, hypothermia, hypokalaemic periodic paralysis and alkalosis.[5,10]

Truepotassium deficit hypokalaemia: This can be broadly classified into hypokalaemia from reduced intake of potassium in diet or increased loss of K^+ from the body. Loss of K^+ from the body could further be classified into renal loss of K^+ and extra renal loss of K^+ based on the daily urinary K^+ loss. Daily K^+ loss greater than 25mmol are suggestive of renal loss, while values less than 25mmol /day are suggestive of extra renal K^+ loss. Major causes of reduced potassium intake are starvation, especially in ill patients and postoperative intravenous fluid therapy with K^+ poor fluids. While causes of increased renal loss of K^+ include diuretics, mineral ocorticoidse.g aldosterone, high dose glucocorticoids[10] high dose antibiotic especially the penicillins, and aminoglycosides which cause magnesium depletion,[10,16] renal diseases such as renal tubular acidosis (RTA) and acute tabular necrosis (ATN) and alkalosis.

Extra renal loss of K^+ commonly occurs with diarrhea and loss of gastric fluid through vomiting or prolonged nasogastric suction.[5,10]

Clinical signs and symptoms: most patients with hypokalaemia are asymptomatic, particularly at levels 3.0-3.5mmol/L, however at lower levels, nonspecific symptoms such as generalized weakness, lassitude and constipation are common, further drop in K^+ may present with anorexia, nausea, vomiting, abdominal distension, muscle cramps paraesthesia, electrocardiographic changes, arrhythmias, inability to concentrate the urine with resultant polyuria and polydipsia, lethargy and confusion. It is important to note that the likelihood of symptoms appear to be dependent on the rate of decrease of plasma K^+ [10]

Treatment of hypokalaemia

Potassium replacement is the corner stone of therapy, unfortunately supplemental K^+ administration is also the comment cause of severe hyperkalaemia in patients who are hospitalized, thus this risk must be kept in mind when initiating treatment,[15] this risk is greatest with the administration of intravenous K^+ and this should be avoided if possible. Excluding factors causing transcellular K^+ shift, the magnitude of deficits in body stores of K^+ correlates well with the degree of hypokalaemia.[16] on the average serum K^+ decreases by 0.3mmol/L for every 100mmol reduction in total body stores.[10] The goals of therapy are to prevent life threatening situations, replace associated K^+ deficits and treat the underlying condition. Different salts of potassium such as potassium chloride, potassium citrate, potassium phosphate and potassium bicarbonate are available commercially for use, Any of these could be used orally or intravenously, intravenous administration is usually not advised in patients who can tolerate oral treatment and should be reserved for life threatening cases, intravenous potassium should be administered in a non glucose containing fluid at a dose not exceeding 20mmol/hour and patients should be monitored closely during administration.[17]

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