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Hypokalemia: A Case Report

Kadek Susi Setyawati, Muh. Mansyur
Department of Internal Medicine, Anuntaloko Regional General Hospital, Parigi, Central Sulawesi, Indonesia
khadekdhe2@yahoo.co.id

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Abstract
Hypokalemia occurs when serum potassium levels are lower than normal, it is the most common electrolyte disorder. Especially in hospitalized patients, with a variety of causes and sometimes requiring urgent medical attention. Hypo calcium is usually the result of increased serum potassium excretion or intracellular shift and less commonly due to reduced potassium intake. A 24-year-old woman was admitted to the hospital with complaints of weakness in both lower limbs accompanied by nausea and decreased appetite, which had been felt for 2 days before admission. Previous history of the patient had diarrhea for 4 days, with the consistency of liquid stools, not mixed with mucus and blood. In to make a diagnosis and initiate appropriate therapy, a proper history and examination are necessary. Treatment of hypokalemia is not only aimed at supplementing potassium, but also at identifying the cause. Causes include gastrointestinal loss, intracellular shift, renal potassium loss, and inadequate intake.
Keywords: Hypokalemia; Potassium; Electrolyte Disturbance;

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Introduction

Potassium is the most abundant cation in the body. it is mostly in the intracellular space, so only about 2% is in the extracellular and the remaining 98% is in the intracellular compartment. The ratio of intracellular to extracellular potassium (Ki/Ke) is the main determinant of resting membrane potential and the main regulator of the sodium-potassium ATPase pump located on the plasma membrane in most cells. Although extracellular potassium is only 2% of the total potassium in the body it has a major effect on the Ki/Ke ratio and its resting membrane potential. An understanding of serum potassium regulation provides an appropriate framework for classifying the causes of hypokalemia. Potassium intake from food generally ranges from 40-120 mmol per day. Normal serum potassium levels are around 3.5-5.0 mmol/L (Theisen-Toupal, 2014), (Reid, Jones, & Isles, 2012)

Hypokalemia is present when serum potassium levels are lower than normal. Hypokalemia is an enough common electrolyte disorder, especially in hospitalized patients with a wide variety of causes and sometimes requires urgent medical attention and treatment. is usually caused by increased potassium secretion or intracellular displacement and low potassium intake (Kardalas et al., 2018)

Hypokalemia is an electrolyte characterized by a low serum potassium concentration of less than 3.5 mmol/l. Patients with mild hypokalemia (serum potassium 3.0-3.5 mmol/l) are usually asymptomatic. However, severe hypokalemia (serum potassium less than 2.5 mmol/l) may cause generalized weakness. xiqiang in addition, patients with severe hypokalemia may develop muscle necrosis (rhabdomyolysis) and paralysis. On mild and severe hypokalemia can increase the incidence of heart attack (Theisen-Toupal, 2014), (Kyaw & Maung, 2022)

In the outpatient population where laboratory tests were performed, almost 14% had mild hypokalemia. In addition, 20% of hospitalized patients were found to have hypokalemia but only 4-5% had significant clinical complaints (Jordan & Caesar, 2015)

Hypokalemia can be caused by either decreased intake of potassium or excessive loss of potassium from the urine and gastrointestinal tract, malnutrition, including inadequate intravenous potassium replacement in patients unable to take it orally. Excessive potassium excretion from the urine can result from the use of diuretic drugs, endocrine diseases such as primary hyper aldosterone, renal abnormalities and genetic syndromes that affect renal function. Gastrointestinal potassium loss is usually due to prolonged diarrhea or vomiting, chronic laxative use, intestinal obstruction or infection (Kardalas et al., 2018), (Theisen-Toupal, 2014)

Intracellular potassium displacement can cause severe hypokalemia. Insulin use in patients with diabetes mellitus, sympathetic nervous system stimulation, thyrotoxicosis and familiar periodic paralysis can also cause hypokalemia. Congenital adrenal hyperplasia causing enzyme defects is a genetic syndrome closely associated with hypertension and hypokalemia resulting from excessive mineralocorticoid use (Coregliano-Ring, Goia-Nishide, & Rangel, 2022), (Kardalas et al., 2018), (Lim, 2007), (Lin, Cheng, & Cheung, 2023), (Reid et al., 2012), (Wang, Han, & Li, 2020).

Drugs such as diuretics and penicillins are often the cause of hypokalemia. Hypokalemia is associated with glucose intolerance due to decreased insulin secretion. Hypomagnesemia is very important, more than 50% of clinically significant hypokalemia is accompanied by magnesium deficiency, and is clinically most frequent in individuals receiving loop diuretic and thiazide therapy (Coregliano-Ring et al., 2022), (Kardalas et al., 2018), (Lin et al., 2023), (Reid et al., 2012), (Wang et al., 2020).

Mild hypokalemia may be asymptomatic. Most symptomatic patients have serum K+ <3 mEq/l. The severity of symptoms is also related to the degree of K+ decrease. Muscle weakness and fatigue are the most common symptoms. Both hypokalemia and hyperkalemia can cause muscle weakness ranging from the lower extremities to the trunk and upper extremities. In severe hypokalemia, muscle weakness may progress to flaccid paralysis, but this is rare. Some patients experience muscle cramps. Severe hypokalemia may lead to rhabdomyolysis. Gastrointestinal muscle involvement may result in ileus, nausea, vomiting and constipation (Kardalas et al., 2018), (Tinawi, 2020)

ECG changes in hypokalemia include flat T waves, ST segment depression, and prominent U waves. Hypokalemia can cause palpitations in addition to ventricular and supraventricular tachyarrhythmias. hypokalemia increases the chances of arrhythmias and sudden cardiac death (Pezhouman et al., 2015), (Tinawi, 2020)

The cause of hypokalemia in patients can be determined after obtaining a medical history and physical examination to assess the severity of hypokalemia and to initiate effective therapy, serum and urine potassium levels are needed (Kardalas et al., 2018)

Basic laboratory tests can be performed, including serum sodium, potassium, glucose, chloride, bicarbonate, BUN, and creatinine. Urine electrolyte examination namely of potassium and chloride is useful to differentiate between renal and non-renal causes of hypokalemia (Oram, McDonald, & Vaidya, 2013)

Arterial blood gas analysis (ABG) is performed to detect metabolic acidosis or alkalosis when the underlying cause is not known from the history. TSH level examination in cases of tachycardia or periodic paralysis hypokalemia (Tinawi, 2020)

In general, there are two main components of the diagnostic evaluation: (a) assessment of urinary potassium excretion to differentiate renal potassium loss (e.g. diuretic therapy, primary aldosteronism) from other causes of hypokalemia (e.g. gastrointestinal loss, transcellular potassium transfer), and (b) assessment of acid-base status, as some causes of hypokalemia are associated with metabolic alkalosis or metabolic acidosis (Kardalas et al., 2018)

Mild to moderate hypokalemia is usually treated with oral potassium supplements. Giving 60 to 80 mmol/day in divided doses over several days to weeks is usually sufficient. Oral supplementation may irritate the GI mucosa causing bleeding and/or ulceration, but is associated with a lower risk of rebound hyperkalemia. It should be taken with plenty of fluids and food. Potassium chloride is the formulation of choice for replacement therapy in most cases (Kardalas et al., 2018)

Replacement therapy must be given more rapidly with severe hypokalemia or when clinical symptoms are present. Potassium chloride of 40 mmol given every 3 to 4 hours for 3 doses is preferred. Rapid correction can be provided via oral and/or IV formulation. There is also a risk of rebound hyperkalemia when rates exceed a dose of 20 mmol per hour. In general, 20 mmol per hour of potassium chloride will increase serum potassium levels by an average of 0.25 mmol per hour. Potassium repletion can occur more slowly once the serum potassium level is persistently above 3 mmol/L or clinical symptoms have resolved. Regardless of severity, careful monitoring of serum potassium levels is required as the development of hyperkalemia is common in hospitalized patients (Castro & Sharma, 2024)

Case Report

A 24-year-old women patient was admitted to the hospital with a chief complaint of weakness in both legs that had been felt since 2 days before admission. The weakness in both legs was felt suddenly when waking up in the morning. Weakness was felt in the groin to the tip of the foot so that the patient could not walk but could still move his legs. Weakness in both legs is without complaints of tingling, spasms, headache, fever, palpitations and no history of previous trauma. The patient also complained of nausea and decreased appetite. Previously, the patient experienced vomiting and diarrhea for 4 days, with liquid stool consistency, without mucus and blood. The patient had no previous history of the same disease and did not suffer from chronic diseases.

On admission, the patient was fully conscious, appeared lethargic. Blood pressure 120/80 mmHg, pulse frequency 80 beats/minute, strong, regular, axilla temperature 37.6°C, respiratory frequency 20 beats/minute. Conjunctiva not anemic, sclera not icteric, there are no enlarged lymph nodes. On physical examination the lungs, heart and abdomen were within normal limits. Extremities were warm, motor examination of upper extremities 555/555 and lower extremities 333/333, sensory examination within normal limits.

In the initial laboratory examination of hematology, renal function and blood glucose within normal limits, the electrolyte examination found hypokalemia with a value of 1.4 mmol/L.

Table 1
Laboratory test result

Lab Test	Findings
Complete blood count	
Erythrocyte count (10^6/uL)	4.65
Hb (g/dl)	12.4
Htc (%)	39.8
MCV (fL)	85.6
MCH (pg)	28
MCHC (g/l)	34.4
Leucocyte (10^3/uL)	6.00
Neutrophil (%)	3.00
Lymphocyte (%)	2.4
Monocyte (%)	0.6
Eosinophil (%)	0.4
Basophil (%)	0.0
Platalet count (10^3/uL)	251
Blood Chemistry	
Random blood sugar (mg/dl)	117
Urea (mg/dl)	45
Creatinine (mg/dl)	0.8
Electrolyte	
Natrium (mEq/l)	136.8
Kalium (mEq/l)	1.51
Chloride (mEq/l)	102

Based on anamnesis, physical examination, and initial laboratory examination, the patient was diagnosed with severe hypokalemia. Then the patient was given initial therapy, namely KCL 50 mEq in 500 ml of 0.9% Nacl in 24 hours, installed two left and right venous lines with a total administration of KCL 100 mEq. Oral KCL was given 3x600 mg tablets, as well as other drugs Omeprazole intravenous 2x40 mg and ondansentron intravenous 3x4 mg to treat nausea and ulcer pain.

Table 2
Electrolyte serial test result

Lab Test	Findings
Electrolyte	
Natrium (mEq/l)	137
Kalium (mEq/l)	2.6
Chloride (mEq/l)	102

After the first day of therapy, on the second day a serial electrolyte laboratory examination was performed with the result of serum potassium 2.6 mmol / L. the patient's complaints began to improve, then continued therapy with the administration of KCL 50 mEq in 500 ml of 0.9% Nacl in 24 hours, oral KCL 3x600 mg, and other drugs Omeprazole intravenous 2x40 mg and intravenous ondansentron 3x4 mg continued. On the third day of hospitalization, another serial electrolyte check was performed with the

result of serum potassium 3.6 mmol/L, the patient had no complaints so the patient was able to walk again and the patient was discharged.

Discussion

Hypokalemia is one of the commonly encountered electrolyte abnormalities, and has the potential to increase the risk of arrhythmias. Hypokalemia is defined as potassium levels <3.5 mmol/L, moderate hypokalemia as potassium levels <3.0 mmol/L, and severe hypokalemia as potassium levels <2.5 mmol/L. Diarrhea and diuretic therapy are responsible for most cases of hypokalemia in the clinic (Rastergar & Soleimani, 2001)

The severity of hypokalemia's clinical manifestations tends to be proportionate to the degree and duration of serum potassium reduction. Symptoms generally do not become present until serum potassium is below 3.0 mEq/L, unless it falls rapidly or the patient has a potentiating factor, such as the use of digitalis, in which patients have a predisposition to arrhythmias (Dias et al., 2014), (Tinawi, 2020)

Muscle weakness and fatigue are the most common symptoms. Hypokalemia can cause lower extremity muscle weakness and may progress to the trunk and upper extremities. In severe hypokalemia muscle weakness may progress to flaccid paralysis, but this is rare. Some patients experience muscle cramps and may develop rhabdomyolysis, gastrointestinal muscle involvement may result in ileus, nausea and vomiting (Rastergar & Soleimani, 2001)

In this case, the patient had severe hypokalemia caused by previous diarrhea. The patient experienced weakness in both lower limbs so that the patient could not walk, but could still move, perhaps the patient had gastrointestinal muscle involvement so that the patient had a decreased appetite and nausea. There were no specific findings from the physical examination, but there was a decrease in motoric examination. There was no abnormal EGC, no arrhythmia and heart failure. The previous history of the patient had diarrhea for 4 days so the patient felt weak.

Most patients experience serum potassium loss through the gastrointestinal tract or kidneys. Vomiting and diarrhea are the most common causes of gastrointestinal potassium loss, while diuretic use is the cause of renal loss. Diarrhea contains potassium (30-60 mmol/L) and a fair amount of bicarbonate, so the body attempts to neutralize electrolytes and retain chloride to compensate for the loss of bicarbonate. Patients with diarrhea usually present with hypokalemia and hyperchloremic metabolic acidosis (Kyaw & Maung, 2022)

Replacement therapy must be given more rapidly with severe hypokalemia or when clinical symptoms are present. Intravenous potassium should be administered when potassium falls below 2.5 mEq/l, with serial measurements of potassium levels. Potassium chloride of 40 mmol given every 3 to 4 hours for 3 doses is preferred. Rapid correction can be provided via oral and/or IV formulation. Normal saline is the preferred infusion fluid, because 5% glucose can cause a transcellular shift of potassium into the cells. Potassium should be replaced very carefully in patients with renal impairment, and

the renal team should be contacted. Patients with potassium levels of 2.5-3.5 mEq/l may only require oral potassium supplementation.

Every 1 mEq/l decrease in serum potassium indicates a potassium deficit of about 200-400 mEq (Kardalas et al., 2018). The patient had a potassium level of 1.5 mEq/l on hospital admission, which means the patient had a potassium deficit of approximately 400 mEq. Hypokalemia improved after 4 times of intravenous KCL administration in addition to oral KCL.

Conclusion

Hypokalemia is the most common electrolyte disorder found in hospitals but can lead to advanced clinical manifestations. Some cases are asymptomatic. hypokalemia can occur along with other diseases, so proper treatment is needed. In addition to proper treatment, it is necessary to know the cause of the hypokalemia. There are various causes of hypokalemia, one of which is diarrhea, which is quite common and can cause considerable loss of serum potassium.

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