**Abstract**

**Background**

Hypokalemia is the most frequent electrolyte disorder encountered in clinical practice. It is caused by inadequate potassium intake or excessive renal or gastrointestinal potassium loss. Hypokalemia may associate with muscle weakness. Hypocalcemia can result from disorders of vitamin D metabolism and action, hypoparathyroidism, resistance to parathyroid hormone (PTH), or other conditions such as nutritional deficiency. Numbness and tingling sensation may occur on hypocalcemia.

**Case presentation**

A 36-year-old woman came with complaints of weakness of upper and lower limbs three days before admission. At first, the weakness felt suddenly on the left arm and left foot after waking up in the morning, making her not being able to walk. Patient felt tingling sensation on her face, upper limbs, and lower limbs. Her hands felt stiff as well as her mouth. Patient had a decreasing serum potassium level (3 mEq/l) and decreasing total calcium level (4.8 mg/dL).

**Conclusion**

In young adult patients with limbs weakness, it is necessary to consider electrolyte imbalance, such as hypokalemia and hypocalcemia. Hypokalemia can be caused by decreased potassium intake, excessive vomiting, drug consumption, kidney disease, and endocrine disease. To diagnose hypokalemia, it is necessary to carry out further examinations such as basic biochemical laboratories (magnesium, calcium, phosphorus), blood gas analysis, TSHs, urine analysis (urine calcium, potassium excretion in 24-hour urine collection), drug screening.

**Keywords:** electrolyte imbalance, paralysis, hypokalemia, hypocalcemia

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**Background**

Periodic paralyses are neuromuscular disorders caused by mutations in skeletal muscle sodium, calcium, and potassium channel genes. These disorders include hypokalemic paralysis, hyperkalemic paralysis, and Andersen-Tawil syndrome. Common symptoms include autosomal dominant inheritance, onset typically in the first or second decades, episodic attacks of flaccid weakness, which are often triggered by diet or rest after exercise. Genetic testing was the confirmatory test needed to diagnose it. In the absence of genetic mutations, documented low or high potassium levels during attacks or a decrement on long exercise will support the diagnosis. Clinical presentation of hypokalemic paralysis is characterized by focal or generalized paralytic episodes of skeletal muscle. The duration can last from hours to days and are associated with concomitant hypokalemia (<2.5 mEq/L). Progressive muscle weakness predominantly in proximal muscle groups of the lower limbs could occur. The first attack usually occurs between ages 5 and 35 years, but the frequency of attacks is highest between ages 15 and 35 years and subsequently decreases with age. Attacks can occur both spontaneously, but also in response to triggers such as carbohydrate-rich meals, alcohol, and rest after strenuous exercise.
However, this primary hypokalemic paralysis due to channelopathy can only be diagnosed with the exclusion of other causes of hypokalemia (renal, adrenal, thyroid dysfunction, renal tubular acidosis, diuretic, and laxative abuse).\(^1\)

Hypocalcemia is defined as a total serum calcium concentration \(<8.8 \text{ mg/dL (<2.20 mmol/L)}\) in the presence of normal plasma protein concentrations. Hypocalcaemia is relatively rare, with risk factors such as disorders of vitamin D metabolism and action, hypoparathyroidism, resistance to parathyroid hormone (PTH) or other etiologies.\(^2\) Tetany is the most frequently recognized symptoms of hypocalcaemia related to peripheral nervous system. Other symptoms of neuromuscular irritability caused by hypocalcaemia including Chvostek’s sign, Trousseau’s sign, paresthesias, seizure, muscle cramps, muscle weakness, laryngospasm, bronchospasm, personality disturbances, extrapyramidal signs due to calcification of basal ganglia, irritability, confusion, disorientation. Cardiac changes such as prolonged QT interval on EKG can be fatal since it can lead to ventricular fibrillation and death. The first symptom of tetany due to hypocalcemia is tingling sensation which initially occurs around the mouth area and on the distal part of the limbs, then spreads proximally.\(^2\)

Both hypokalemia and hypocalcemia could lead to generalized weakness and other neurological symptoms. However, in order to differentiate the diagnosis, careful history taking, physical examination, laboratory testing and other diagnostic modalities should be performed. There are numerous causes of generalized muscle weakness in adults, ranging from neurological (central nervous systems) disorders, muscular disorders, endocrine disorders, electrolyte disturbances, genetic disorders, iatrogenics, infections, toxins, or sarcopenia.\(^3\) In this case report, we presented and analyze how to diagnose paralysis caused by electrolyte imbalance, which is hypokalemia and hypocalcemia.

**Case presentation**

A 36-year-old woman came to Emergency department of Unggul Karsa Medika Hospital with chief complaint of upper and lower limbs weakness from three days before admission. At first, she felt sudden weakness on her left arm and left leg after waking up in the morning, which made her not being able to walk. She also had tingling sensation on her face, upper limbs, and lower limbs. Her hand felt stiff, as well as her mouth. She also felt headache and nausea. The patient admitted that she was on strict dietary programme in order to lose weight from 80 kilograms to 61 kilograms within a year.

On physical examination, the patient’s general condition was moderate pain, comatosis consciousness with vital signs: blood pressure 130/80 mmHg, pulse 102x/min, temperature 36.5°C, respiration 20x/min. Neurological examination showed no carpopedal spasm, no Chvostek’s sign or Trousseau’s sign, but motoric power of upper limb in grade 4 for both arms and motoric power of lower limb in grade 2 for both legs.

Initial laboratory examination was performed and showed hypokalemia (2.5 mEq/L), hypocalcemia (total calcium 4.8 mg/dL and calcium ion 1.02 mmol/L) and hypoalbumin (2.8 g/dL) (**Table 1**). Computed tomography scan of brain revealed no ischemic nor hemorrhagic stroke (**Figure 1**). Electrocardiogram (ECG) showed QT prolongation.

The patient was diagnosed as paralysis due to hypokalemia and hypocalcemia and received correction for hypocalcemia which was Calcium gluconate solution 4 grams in Dextrose 5% infusion 100 mL given slowly via infusion in 1 hour, and correction for hypokalemia which was Potassium Chloride (KCl) 7.46% solution 50 mEq in 500 mL of Ringer Lactate solution, given in 8 hours and total for 2 cycles intravenously, Potassium tablet slow release 1200 mg once daily per oral, and calcium preparate 500 mg thrice daily per oral. The patient was also given dietary recommendation of regular food with solid consistency and high in potassium.

After the patient was given therapies in hospital, the motoric power for upper and lower limb, both sides were grade 5. There was neither paralysis nor spasm in all extremities. No paresthesia and tingling sensation were complained. The patient could walk home freely without any weakness. Potassium level after correction was 4.2 mEq/L and calcium level after correction was 7.8 mg/dL (one week after).

**Table 1. Laboratory Findings on Blood Analysis**

<table>
<thead>
<tr>
<th>Examination</th>
<th>Result</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin</td>
<td>10 g/dL</td>
<td>12-16 mg/dL</td>
</tr>
<tr>
<td>Hematocrit</td>
<td>31 %</td>
<td>35-47 %</td>
</tr>
<tr>
<td>Leukocytes</td>
<td>9,000 /mm(^3)</td>
<td>4,000-11,000 /mm(^3)</td>
</tr>
<tr>
<td>Thrombocytes</td>
<td>447,000 /mm(^3)</td>
<td>150,000-450,000 /mm(^3)</td>
</tr>
<tr>
<td>WBC</td>
<td>8,550 /mm(^3)</td>
<td>&lt;10,000 /mm(^3)</td>
</tr>
<tr>
<td>Sodium</td>
<td>142 mEq/L</td>
<td>136 – 145 mEq/L</td>
</tr>
<tr>
<td>Potassium</td>
<td>2.5 mEq/L</td>
<td>3.5 – 5.5 mEq/L</td>
</tr>
<tr>
<td>Chloride</td>
<td>106 mEq/L</td>
<td>98 – 106 mEq/L</td>
</tr>
<tr>
<td>Calcium</td>
<td>4.8 mg/dL</td>
<td>8.6 – 10.9 mg/dL</td>
</tr>
<tr>
<td>Calcium ion</td>
<td>1.02 mmol/L</td>
<td>1.20 – 1.32 mmol/L</td>
</tr>
<tr>
<td>Albumin</td>
<td>2.8 mg/dL</td>
<td>3.5 – 5.2 mg/dL</td>
</tr>
</tbody>
</table>
Discussion

Potassium is the most abundant intracellular cation. In humans, the intracellular concentration of potassium ion is around 150 mEq/L, while the extracellular concentration is 3.5-5 mEq/L. Hypokalemia defined as a plasma potassium ion concentration of <3.5 mEq/L. Hypokalemia is associated with a tenfold increase in in-hospital mortality, due to adverse effects on cardiac rhythm, blood pressure, and cardiovascular morbidity. Approximately 80% of patients who are receiving diuretics become hypokalemic, while many of patients with hypokalemia could also have an associated systemic disease. There are no significant differences in its prevalence between males and females.4,5

Homeostatic mechanisms maintain plasma potassium ion concentration between 3.5 and 5.0 mEq/L, despite marked variation in dietary potassium intake. In a healthy individual at steady state, the entire daily intake of potassium is excreted 90% in the urine and 10% in the stool; thus, the kidney plays a dominant role in potassium homeostasis. Potassium filtered through the glomerulus is almost completely absorbed before reaching the collecting duct. About 65% is absorbed in the proximal tubule and 25% in the loop of Henle. 10% of filtered potassium ion reaches the early distal tubule. It is important to note that almost all of the potassium ion in the urine is secreted by the collecting duct.5,7

Hypokalemia can be caused either by decreased intake of potassium; or by excessive losses of potassium in the urine (renal potassium loss) or through the gastrointestinal (GI) tract (non-renal potassium loss). Vomiting and diarrhea are the most common GI causes, while diuretic use is the most common renal etiology.6 Excessive excretion of potassium in the urine may result from the use of diuretic drugs, endocrine diseases such as primary hyperaldosteronism, kidney disorders and genetic syndromes affecting the renal function. Gastrointestinal losses of potassium usually are due to prolonged diarrhea or vomiting, chronic laxative abuse, intestinal obstruction, or infections. An intracellular shift of the potassium can also lead to severe hypokalemia. Insulin and β2 receptors agonists (such as epinephrine, albuterol, and ephedrine) are the major causes of intracellular potassium ion shift. Redistribution is seen in hypokalemic periodic paralysis. This is a rare disorder that is seen more commonly in Asians in association with thyrotoxicosis. Drugs, such as diuretics and penicillin can be often the underlying cause of hypokalemia. Hypomagnesemia is very important. More than 50% of clinically significant hypokalemia has concomitant magnesium deficiency and is clinically most frequently observed in individuals receiving loop or thiazide diuretic therapy. Hypokalemia associated with magnesium deficiency is often refractory to treatment with potassium ion.6

In this case study, we had not evaluated the possible etiologies yet. However, hypoalbuminemia and history of strict diet may give us clue to presume that hypokalemia in this patient was because of anorexia nervosa. Patients with anorexia nervosa have a higher risk of developing hypokalemia because they suffer from severe malnutrition and are refed, in which the insulin surge resulting from glycemia during the refeeding process causes a substantial intracellular uptake of potassium and phosphorus.8

Since the complete laboratory examination had not done yet, we could not exclude other etiologies of hypokalemia. Urine electrolytes (potassium and chloride) are useful in differentiating renal from non-renal causes of hypokalemia. An arterial blood gas (ABG) analysis should be performed to detect metabolic acidosis or alkalosis when the underlying
cause is not apparent from the history. Serum magnesium, calcium and/or phosphorus levels are important to exclude associated electrolyte abnormalities, especially if alcoholism is suspected. Urinary calcium excretion is very critical to exclude Bartter syndrome. We should also measure serum digoxin level if the patient is on digitalis. In cases of high clinical index of suspicion for a disorder, a drug screen in urine and/or serum for diuretics, amphetamines and other sympathomimetic stimulants should be conducted. Assessment of Thyroid Stimulating Hormone (TSH) levels is required in cases of tachycardia or clinical suspicion of hypokalemic periodic paralysis.5,9

The severity of clinical manifestations tends to be proportionate to the degree and duration of hypokalemia. 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, predisposition to arrhythmias. Muscle weakness and fatigue are the most common symptoms. Both hypokalemia and hyperkalemia can result in muscle weakness. The symptom usually starts from the lower extremities and then ascends to the trunk and upper extremities. Hypokalemia results in hyperpolarization of skeletal muscle, thus impairing the capacity to depolarize and contract. It also causes a skeletal myopathy and predisposes to rhabdomyolysis. Severe hypokalemia could lead to muscle weakness and flaccid paralysis, but this is rare. Some patients develop muscle cramps.5,10,11 This case was perfect presentation of severe hypokalemia (2.5 mEq/L) presenting with general weakness or paralysis.

Hypokalemia can affect cardiovascular system. Electrocardiographic changes in hypokalemia include T wave flattening, U waves, ST depression, and QT prolongation; these are most marked when serum K+ is <2.7 mmol/L. Hypokalemia also results cardiac arrhythmias (sometimes lethal) and heart failure.6,12 In this case, QT prolongation presented in ECG. Acute hypocalcemia may also present by prolongation of the QT-interval.2

Patients with potassium levels of 2.5–3.5 mEq/L (mild to moderate hypokalemia), may need only oral potassium replacement. If potassium levels are less than 2.5mEq/L, severe nausea, vomiting or abdominal distress, intravenous (IV) potassium should be given, with close follow-up, continuous ECG monitoring, and serial potassium levels measurements. Administration of oral potassium should be accompanied with plenty of fluid (between 100 and 250 mL of water, depending on the form of the tablet of potassium) and is better to be given with after meals. Regarding IV therapy, 0.9% sodium chloride is the preferred infusion fluid, as 5% dextrose solution may cause transcellular shift of potassium into cells. It is critical also to correct the levels of serum magnesium, in order to achieve an adequate treatment of hypokalemia.5

Hypocalcemia defined as corrected serum total calcium levels <2.12 mmol/l (8.5 mg/dl). Hypocalcemia can be asymptomatic in mild cases to presenting as an acute life-threatening crisis. Serum calcium levels are regulated within a narrow range (2.1 to 2.6 mmol/L) by 3 main calcium-regulating hormones: parathyroid hormone (PTH), vitamin D, and calcitonin. Approximately half of the total serum calcium is bound to protein, and the remaining free ionized calcium is physiologically active. Serum calcium levels must be corrected for the albumin level before confirming the diagnosis of hypercalcemia or hypocalcemia.2,5,13,14

Hypocalcemia is most commonly a consequence of vitamin D inadequacy or hypoparathyroidism, or a resistance to these hormones. Hypocalcemia also associated with many drugs, including bisphosphonates, cisplatin, antiepileptics, amino-glycosides, diuretics, and proton pump inhibitors. Hypocalcemia can present as an asymptomatic laboratory finding or as a severe, life-threatening condition. In acute hypocalcemia, rapid treatment may be necessary. Chronic hypocalcemia may be well tolerated, but treatment is necessary to prevent long-term complications.13,14

The decision to treat is dependent on presenting symptoms, the severity and rapidity with which hypocalcemia develops. Acute hypocalcemia can be life-threatening, as patients may present with tetany, seizures, cardiac arrhythmias, laryngeal spasm, or altered mental status. Calcium gluconate is the preferred intravenous calcium salt as calcium chloride often causes local irritation. Calcium gluconate usually diluted in 50 to 100 mL of 5% dextrose solution is infused over 10 minutes. This can be repeated until the patient’s symptoms have cleared. Drip rates of 0.5-2.0 mg/kg/hour are recommended. As soon as possible, oral calcium supplementation should be initiated and give vitamin D or its analogues if necessary. Rapid administration could result in arrhythmias so intravenous administration should be carefully monitored. Local vein irritation can occur with solutions >200 mg/100 mL of elemental calcium.14

Conclusion
In this case, the patient had hypokalemia and hypocalcemia was thought to be due to excessive nausea, vomiting and inadequate food intake. In addition, it can be caused by renal dysfunction or unknown medical history of this patient. Therefore, it is important to carry out further investigations such as blood magnesium levels, potassium excretion.
in 24-hour urine collection, and TSH levels, to determine the possible cause of hypokalemia in this patient. For hypocalcemia, PTH levels can be checked, as PTH hormone is the main defense mechanism against hypocalcemia. Examination of vitamin D levels is also necessary because of the role of vitamin D in the absorption of calcium. Treatments given to patient were accurate and suitable with standard therapy.

List of abbreviations
- PTH: Parathyroid Hormone
- KCl: Kalium Chloride (potassium chloride)
- GI tract: gastro intestinal tract
- ABG: arterial blood gas
- TSH: thyroid stimulating hormone
- ECG: electrocardiogram

Declarations
Ethics approval and consent to participate
Informed consent from the patient has been obtained before the study.

Consent for publication
Consent for publication regarding patient data has been obtained before the study. All the patient identity has been kept secret.

Availability of data and materials
Not Applicable

Competing interests
The authors declare that they have no competing interests.

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