

Case Report: Periodic Hypokalemic Paralysis

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ABSTRACT

Hypokalemic periodic paralysis is characterized by muscle weakness or flaccid paralysis followed by low potassium levels (less than 3.5 mmol/L) during the attack. Proper initial management and patient education are mandatory for health workers to manage this case. This study was a descriptive study with a case report approach. This case report includes patients over 18 years receiving health care at the Negara General Hospital and having accessible medical record data. Data collection was carried out consecutively. A case from a 22-year-old man with complaints of weakness in both legs. The examination found lower extremity motor strength 2/2, normal physiological reflexes without pathological reflexes, and a laboratory potassium level of 2.5 mmol/L laboratory results. The patient was then given hypokalemia treatment with KCl intravenously. The patient, in this case, had no clinical condition contraindicated to give potassium. Within 24 hours after potassium administration, the patient showed a significant improvement. Hence, our therapy, in this case, was in line with the theory with a good clinical outcome. Symptomatic therapy in hypokalemic periodic paralysis provides a good clinical outcome. There were no complications or clinical emergencies during treatment.

Keywords: hypokalemia; periodic paralysis; channelopathies; potassium levels

INTRODUCTION

Hypokalemic periodic paralysis is characterized by muscle weakness or flaccid paralysis due to the shift of Potassium into the intracellular space of skeletal muscles. The disorder is characterized by low potassium levels (less than 3.5 mmol/L) at the time of the attack, accompanied by a history of episodes of weakness to paralysis of the skeletal muscles. These disorders are also called channelopathies of the skeletal muscles. Periodic paralysis disorder is characterized by reversible muscle weakness and paralysis, accompanied by hypokalemia (Browmn et al., 2011; Tjokroprawiro, 2015).

Hypokalemia can occur due to certain precipitating factors, for example, foods with high carbohydrate levels, rest after physical exercise, long trips, surgery, menstruation, alcohol consumption, and others. Insulin levels can also affect this disorder in many sufferers because insulin increases the flow of potassium into cells. At the time of the attack, potassium shifted from the extracellular fluid into the cells, resulting in hypokalemia on examination of the blood potassium (Pertiwi, 2015).

The incidence of hypokalemic periodic paralysis is 1 in 100,000. It occurs more frequently in men than in women, with a ratio of 3-4:1. The age of the first attack varies from 1-20 years. Most attacks occur at age 15-35 and then decrease with age. (Lin, 2008; Lin et al., 2004). Patients can experience attacks only once but can also be repeated with varying attack time intervals. Weakness usually occurs in the leg and hand muscles, but sometimes it can affect the eye, respiratory, and swallowing muscles (Pertiwi, 2015).

The clinical outcome of hypokalemic periodic paralysis varies among individuals. Most patients tolerate and respond to the therapy (Cristina & Espadinha, 2022). However, it might cause acute flaccid paralysis in mild to severe to life-threatening muscle weakness, such as cardiac arrhythmias and paralysis of the respiratory muscles. The patient's mortality could be increased if it is not treated well (Widjajanti & Agustini, 2018). In addition, patients who experience recurrent attacks can increase morbidity rates, affect the patient's quality of life, and increase hospital readmissions (Finsterer, 2008).

Recognition of early symptoms, identification of triggering factors, proper initial management, and patient education is necessary for health workers to manage patients with hypokalemic periodic paralysis to provide a good

prognosis and clinical outcome and prevent recurrent attack. Thus, the authors are interested in sharing experiences managing patients with hypokalemic periodic paralysis through this case report article.

METHOD

This study was a descriptive study with a case report approach. This case report includes adult patients (aged over 18 years) receiving health care at the Negara General Hospital and having accessible medical record data. Data collection was carried out consecutively, and patients who met the criteria were included in this case report. Then the case discussed in this report is a case of hypokalemic periodic paralysis that a 22-year-old man experienced. Case reports were carried out at the Negara General Hospital, Flamboyan Room, for three days for patients from January 24-27, 2023. Data sources were obtained from primary and secondary data. Primary data comes from examining patients, and secondary data from medical records, books, and journals used as literature. Medical records are accessed by writing in the medical record status borrowing book. They are borrowing patient medical records with a grace period of 1x24 hours. Research ethics here by carrying out informed consent to patients.

RESULT

Based on the results of the case report, it was found that a 22-year-old man came to the Emergency Room at the State General Hospital on January 24, 2023, complaining of weakness in both legs, and it was getting worse. Before weakness occurs, the patient admits that his feet feel tingling, but there is no spreading in both hands. Complaints such as fever, cough, runny nose, and shortness of breath were denied. Past medical history, the patient had experienced something similar in the form of weakness in both legs, namely eight months ago. No family members of the patient with the same complaint or history of previous treatment, namely never taking medication for similar complaints.

Table 1. Laboratory Test Results

| Marker | Value | Interpretation |
|-----------------------------|---------|----------------|
| Complete blood count | | |
| White blood cells (/ul) | 6,920 | Normal |
| Hemoglobin (g/dl) | 13.6 | Normal |
| Hematocrit (%) | 37 | Normal |
| Platelet (/ul) | 185,000 | Normal |
| Renal function | | |
| Blood urea nitrogen (mg/dl) | 21 | Normal |
| Creatinine (mg/dl) | 0.6 | Normal |
| Biochemistry | | |
| Random blood sugar (mg/dl) | 94 | Normal |
| Electrolyte | | |
| Sodium (mmol/L) | 136 | Normal |
| Chloride (mmol/L) | 101 | Normal |
| Potassium (mmol/L) | 2.5 | Low |

On physical examination, the general condition appeared moderately ill and conscious of compos mentis. Blood pressure 120/70 mmHg, temperature 36.6 °C, pulse 88 beats per minute, breathing 18 beats per minute, oxygen saturation 98%, body weight 67 kg, height 165 cm, nutritional status is good. Conjunctiva and sclera were normal. Ears, nose, and mouth are normal. There are no enlarged lymph nodes in the neck. There were no abnormalities in the heart and lungs, the liver and spleen were not palpable, the extremities were not edematous, and they felt warm. Standard sensory, superior extremity motor 5/5, inferior extremity motor 2/2. The neurological status of physiological reflexes is normal, and there are no pathological reflexes. The laboratory examination (table 1) showed that the patient had a hypokalemic condition with a 2.5 mmol/L potassium level.

From the anamnesis, physical examination, and laboratory test, the patient was diagnosed with hypokalemic periodic paralysis. In this case, the patient was given an infusion of 50 mEq of KCl mixed in 500 ml of 0.9% NaCl with 30 drops per minute. Then infuse 0.9% NaCl maintenance 20 drops per minute. One ampoule of intravenous mecobalamin therapy was also given every 12 hours. The patient is planned to re-examine electrolytes 6 hours after KCl infusion. Monitoring is also carried out for complaints and patient vital signs.

A neurological re-examination was carried out within the first 24 hours after administration of KCl. The results obtained were superior extremity motor 5/5 and inferior extremity motor 5/5. The neurological status of physiological reflexes is normal, and there are no pathological reflexes. Clinical improvement was seen in patients. Then the patient was discharged after three days of treatment.

DISCUSSION

In this article, we presented a case of hypokalemic periodic paralysis. Hypokalemic periodic paralysis is a common occurrence of primary or inherited periodic paralysis. Secondary hypokalemic periodic paralysis is sporadic and usually associated with a specific disease or poisoning (Tawil R, 2002). In cases of periodic primary paralysis, abnormalities are found, including autosomal dominant inheritance, namely mutations on the CACNA1S chromosome (70%) called rare hypokalemic paralysis type 1, loci mutations on the SCN4A chromosome (10%) called rare hypokalemic paralysis type 2 (Saban et al., 2010). Other research explains that mutations in the CACNL1A3, SCN4A, and KCNE3 genes, namely genes that control voltage-gated ion channels in muscle cell membranes (Pardede & Fahriani, 2012). The genetic level of biomolecular examination cannot be applied in our health care center, so the diagnostic approach of hypokalemic periodic paralysis was based only on clinical and laboratory findings.

From the clinical examination, we found the patient was admitted with complaints of weakness in both legs, which was worsening. Neurological examination revealed superior extremity motor 5/5 and inferior extremity motor 2/2. Based on theory, muscle weakness usually occurs in all four limbs; if it is incomplete, the weakness is more dominant in the lower limbs. Respiratory function, swallowing, and ocular motility are usually unaffected and may occur in severe attacks, and the cough reflex is impaired during attacks (Pardede & Fahriani, 2012). Then, the patient was suspected of having periodic hypokalemic paralysis disorders, established based on low blood potassium levels (less than 3.5 mmol/L) at the time of the attack, experiencing episodes of flaccid paralysis with other tests within normal limits. In this case, giving potassium can relieve the patient's symptoms. Unfortunately, EMG (Electromyography) examination, muscle biopsy, and genetic analysis cannot be done, so researchers cannot make a definite diagnosis. Thus, the clinical findings led to the clinical diagnosis of hypokalemic periodic paralysis.

In this case, the patient was given an infusion of 50 meq of KCl mixed in 500 ml of 0.9% NaCl with 30 drops per minute. Then infuse 0.9% NaCl maintenance 20 drops per minute. One ampoule of intravenous mecobalamin therapy was also given every 12 hours. The patient is planned to re-examine electrolytes 6 hours after KCl infusion. Monitoring is also carried out for complaints and patient vital signs. Based on theory, the management of hypokalemic periodic paralysis focuses on relieving acute symptoms and preventing subsequent attacks. Administration of Potassium is preferred in oral form because it is easier. Oral potassium correction is sufficient if the serum potassium level is > 3 mEq/L. Giving 40-60 mEq can increase potassium levels by 1-1.5 mEq/L. Potassium supplementation must be cautiously administered because hyperkalemia will occur when the trans-cellular redistribution of potassium stops (Pardede & Fahriani, 2012; Widjajanti & Agustini, 2018). Oral potassium preparations may cause gastrointestinal complaints, and enteric-coated tablets have been reported to cause small bowel ulcers (Venace et al., 2006). In cases of severe hypokalemia or with manifestations of ECG changes, it should be given intravenously at 0.5 mEq/kg over 1-hour, continuous infusion with close monitoring. Factors that must be considered when administering potassium are plasma potassium levels, clinical symptoms, kidney function, and patient tolerance (Andrea et al., 2008). The patient was then re-evaluated within the first 24 hours after administration of KCl. We found the patient had a normal motoric function with the superior extremity motor 5/5 and inferior extremity motor 5/5. Based on theory, pure flaccid paralysis with hypokalemia will recover or self-remission 5-6 hours later with potassium administration (Palmer et al., 2010). In this case, the patient had no clinical condition contraindicated to give potassium. Within 24 hours after potassium administration, the patient showed a significant improvement. Hence, in this case, our therapy aligned with the theory with a good clinical outcome.

Providing patient education is important because it relates to lifestyle, diet, and physical activity. Therefore, consuming foods with high potassium levels is highly recommended to prevent recurrent muscle weakness (Palmer et al., 2010). Besides that, patients can also be advised to do ROM exercises to increase muscle strength after an attack of paralysis. The research by Helen et al. (2021) found that there was a significant effect of giving active ROM on the muscle strength scale of the upper ($p=0.001$) and lower extremities ($p=0.002$) (Helen et al., 2021). Another study showed that ROM exercises are carried out 1-2 times a day, the duration of each extremity is 15-20 minutes per session, and performed two times a day for six days could increase muscle strength in patients. Thus, it is recommended for healthcare to give active and passive ROM exercises (Abdillah et al., 2022).

CONCLUSION

In this report, symptomatic therapy in the form of potassium, especially KCl infusion in cases of hypokalemic periodic paralysis, provides a good clinical outcome. Significant clinical improvement was seen within the first 24 hours. Patients also did not experience complications and clinical emergencies during treatment. In general, this case management approach follows the theory. We recommend doing supporting tests such as some genetic analysis tests, muscle biopsies, and EMG to exclude other serious conditions. Also, it is mandatory to educate and counsel the patient regarding lifestyle, diet (high potassium content), and physical activity to prevent recurrent paralysis.

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