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CASE REPORT

Recurrent flaccid paralysis due to hypokalemia as an unusual presentation of hypothyroidism

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ABSTRACT

Background: Recurrent flaccid paralysis with low level of potassium can be found in hypokalemic periodic paralysis or thyrotoxic periodic paralysis. Hypokalemic Periodic paralysis associated with hypothyroidism is rare in the existing literature.
Case illustration: A 41-year-old man had been admitted to the emergency ward due to weakness of his lower extremities for 3 days before the admission. He had the same symptoms one year ago. Based on the physical examination, decreases in both muscle strength and physiological reflexes in lower extremities had been found. From laboratory examination, the serum potassium level had reduced up to 1,7 mmol/l, with high TSHs and normal T3 and T4 level. The nerve conduction studies had revealed normal limits. Following a potassium correction, the patient had shown improvement.
Discussion: In most cases, hypokalemic periodic paralysis is caused by pure hypokalemia or high thyroid hormone level in serum (as in thyrotoxic periodic paralysis). However, this patient was diagnosed as hypokalemic periodic paralysis with subclinical hypothyroidism. This case is therefore a very rare and unusual presentation of hypothyroidism. The relation between hypokalemic periodic paralysis and hypothyroidism remains unclear.
Conclusion: Hypokalemic periodic paralysis associated with hypothyroidism is rare and understanding about this condition has not been established thus far in the literature. Thyroid examination is needed for patients with hypokalemic periodic paralysis.

Keywords: recurrent flaccid paralysis, hypokalemia, hypokalemic periodic paralysis, hypothyroidism

INTRODUCTION

Periodic paralysis (PP) is a neuromuscular disorder resulting from defect in ion channels, including sodium, calcium, and/or potassium channel in skeletal muscle. PP can be classified into four types: hypokalemic PP, hyperkalemic PP, thyrotoxic PP, and Andersen-Tawil Syndrome.[1] The prevalence of hyperkalemic PP is 1 in 200.000, while hypokalemic PP is estimated to be 1 in 100.000, and Andersen-Tawil Syndrome is about 1 in 1.000.000. The onset of PP most commonly occurs in the first or second decade of life. Hypokalemic PP is more frequent in males compared to females.[2] Primary hypokalemic periodic paralysis (HPP) is a genetic neuromuscular disorder that is autosomal dominant, most often caused by a mutation in the CACNA1S gene on the calcium ion channel.[3,4]

PP can be precipitated by heavy activities, fasting, or high carbohydrate intake. The clinical manifestation of PP typically include weakness in the extremities lasting from a few hours to several days without accompanying sensory disturbances. Patients with HPP require appropriate pharmacological and non-pharmacological management to address acute attacks, prevent complications, and prevent recurrence in the future.[1,2,4]

Thyrotoxicosis is a fairly common cause of secondary HPP while recurrent HPP is a rare clinical manifestation of hypothyroidism.[2,5] There are not many case reports discussing the relationship between HPP and hypothyroidism. In this case report, a case of HPP with subclinical hypothyroidism will pe presented.

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CASE ILLUSTRATION

A 41-year-old man had been admitted to the emergency ward due to weakness of his lower extremities for 3 days before the admission. Initially, the patient felt fatigued after sitting for hours while driving a car, then he had a large meal and rested. Upon waking up the next morning, the patient experienced weakness in both legs but was still able to walk by holding onto nearby objects. However, over time, he was unable to move his legs. He did not report any sensory complaints such as pain, numbness, tingling, or loss of sensation. There were no issues with bowel movements and urinary continence/ retention. Other complaints such as syncope, seizures, cognitive impairments, visual disturbances, shortness of breath, nausea/vomiting, loss of appetite, weight loss, or palpitations were denied. He had experienced similar complaints, which improved after being hospitalized a year earlier. There is no history of similar symptoms among other family members. Also, there is no history of previous disease like hypertension, diabetes mellitus, cardiac disorders, or thyroid disorders. History of alcohol consumption and routine medication were denied.

Upon physical examination in emergency ward, the patient was found to be compos mentis, with the blood pressure 130/80 mmHg, pulse 73 beats/minute, respiratory rate 20 breaths/minute, body temperature 37,1^{o}C, and the oxygen saturation was 99% (room air). There was no pallor or edema, no palpable lymph nodes and thyroid, and chest and abdominal examinations being within normal limits. From the neurological examination, the motor strength of the upper extremities was 5555/5555, and the lower extremities were 1111/1111. The physiological reflexes of the patella and Achilles in both legs were decreased. No Babinski pathological reflexes and meningeal irritation signs were found. Cranial nerve and sensory examination were normal. The electrocardiogram examination was normal. The laboratory test results showed Hb 17,3 g/dL, leukocytes 15.960/mm^3, hematocrit 47,80%, platelets 256.000/mm^3, SGOT 16 U/L, SGPT 20 U/L, urea 39 mg/dL, creatinine 1,13 mg/dL, electrolytes: sodium 141 mmol/L, potassium 1,7 mmol/L (low), chloride 107 mmol/L, fasting blood glucose 158 mg/dL, and thyroid profile: Thyroid Stimulating Hormone (TSH) 4,260 uIU/ml (high), T3 1,31 nmol/L, and T4 97,51 nmol/L. Moreover, electrodiagnostic tests showed normal sensory and motor nerve conduction studies.

Management for this patient included administering potassium drip. We gave potassium chloride (KCl) 25meq in 0,9% sodium chloride (NaCl) solution 500 ml every 8 hours for three times and conducted physiotherapy. After electrolyte correction, on the second day of the treatment, the patient's leg weakness showed complete improvement. The motor strength of the lower extremities became 5555/5555. The patient was re-tested for electrolytes and the results were sodium 138 mmol/L, potassium 3,1 mmol/L, and chloride 114 mmol/L. The patient was scheduled for a thyroid examination but refused due to financial constraints. Given the patient's improvement and no more complaints or symptoms, the patient was discharged after 3 days of treatment.

DISCUSSION

The patient presents with periodic paralysis characterized by recurrent/ episodic weakness of both legs with signs of lower motor neuron (LMN) lesions, such as decreased of patellar and achilles reflexes, without sensory or autonomic disturbances. Laboratory tests, including electrolyte analysis, revealed a severe hypokalemia with a potassium level of 1.7 mmol/L. The electrocardiogram examination and nerve conduction studies of both lower and upper extremities were within normal limits. By using diagnostic evaluation (figure 1), patient diagnosed as hypokalemic periodic paralysis (HPP). Periodic paralysis includes HPP, hyperkalemic periodic paralysis, thyrotoxic periodic paralysis, and Andersen-Tawil Syndrome.[1,2]

In HPP, there is activation of the Na^+-K^+ATPase pump, causing potassium to enter the cells and thereby reducing serum potassium levels. The Na^+-K^+ATPase enzyme is present in the cell membrane, and the activity of the Na-K pump is regulated by thyroid hormone in most tissues. Thyroid hormone deficiency can affect this enzyme. Androgens can also activate the pump, which may be why HPP is generally more commonly observed in men compared to women.[2,4]

In patients with thyrotoxic periodic paralysis, thyroid hormone levels are typically high (hyperthyroidism). However, in this patient, high TSH levels were found, while T3 and T4 levels were still within normal limits. This condition is classified as subclinical hypothyroidism, which can be caused by various factors, such as the early stage of thyroid dysfunction, iodine deficiency, thyroiditis, or side effect of medication.[6] Although no clinical symptoms related to hypothyroidism, such as frequent fatigue, sensitivity to cold, weight gain, myxedema, etc., were observed, the patient was actually scheduled for a follow-up regarding the thyroid hormone disorder experienced. However, this follow-up was not carried out due to financial constraints.

Management of this patient included the administration of intravenous potassium correction at a rate not exceeding 10mmol/hour to avoid the condition of rebound hyperkalemia. Physiotherapy was also performed on the patient. Physiotherapy in cases of HPP is conducted to help maintain postural control and improve muscle strength.[7] The patient was also educated to avoid triggering factors and to maintain proper hydration.[8]

The relationship between HPP and hypothyroidism remains unclear to this day. Several case reports about HPP as an atypical clinical manifestation of hypothyroidism have been reported by Sinha et al. and Singh et.al.[9,10] Hypothyroid condition themselves can cause electrolyte disturbances such as hypokalemia and hypocalcemia without significant clinical manifestation. In this patient, motor strength in both legs improved completely with potassium correction alone, without additional supplementation or other pharmacological therapy.

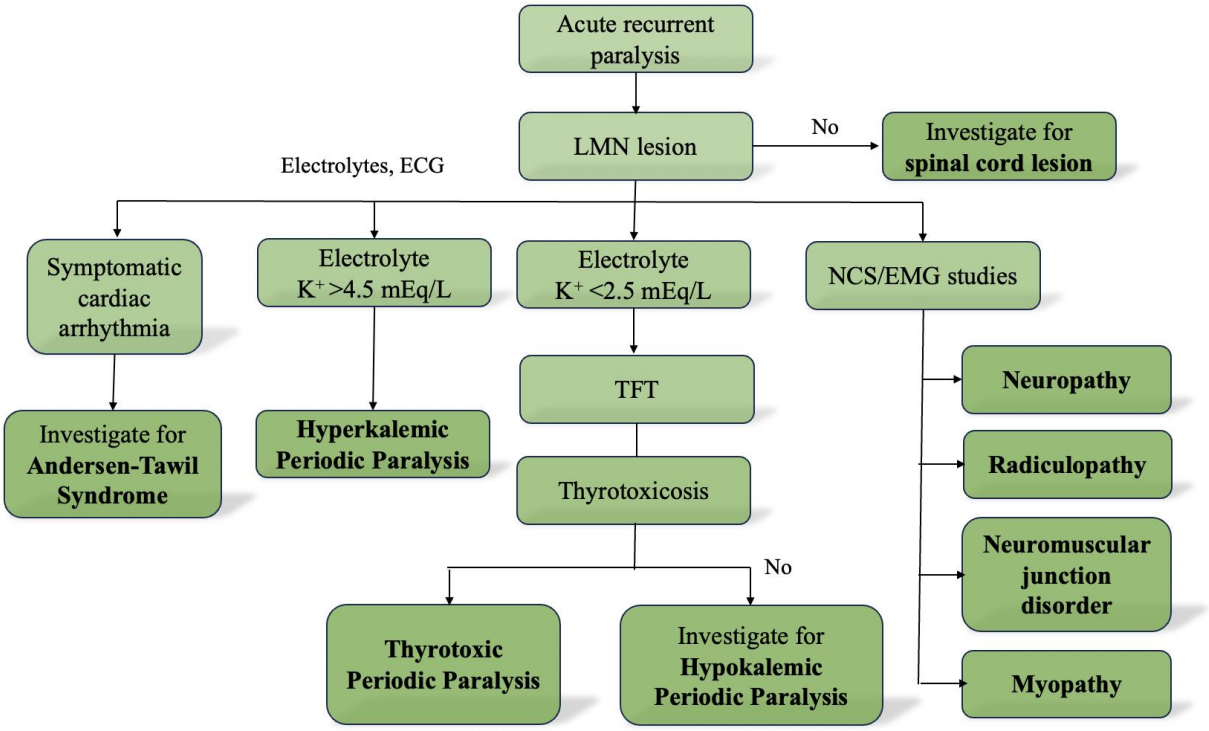

Figure 1. Diagnostic evaluation of acute recurrent paralysis
(*Abbreviations:* LMN, lower motor neuron; ECG, electrocardiography; NCS, nerve conduction studies; EMG, electromyography; TFT, thyroid function test.)

CONCLUSION

Hypokalemic periodic paralysis associated with hypothyroidism is a rare condition. The understanding about this condition has not been established thus far in the literature. A thyroid examination is needed for patients with hypokalemic periodic paralysis.

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