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Sudden weakness due to thyrotoxic periodic paralysis: a case report

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ABSTRACT

Thyrotoxic periodic paralysis (TPP) is a complication of hyperthyroidism. Lead to sudden hypokalemia and muscle weakness. In most cases, it is found mainly in young adult males of the Asian race. The paralysis is temporary and will return with potassium correction. There are very few cases reported in Indonesia regarding these cases of TPP. In our case, a 44-year old man complained of weakness that started later in the limbs but quickly improved. This case was diagnosed late at first, and uncorrected potassium levels led to recurrent paralysis. Laboratory test results showed severe hypokalemia (potassium level 1.81 mmol/L). The thyroxine level (T4) was 44.12 pmol/L and low serum thyroid-stimulating hormone (TSH) <0.0025 IU/mL.

ABSTRAK

Periodik paralisis tirotoksis (PPT) merupakan suatu komplikasi yang terjadi karena adanya hipertiroid yang menyebabkan hipokalemia dan kelemahan pada otot secara mendadak. Pada banyak kasus ditemukan terutama pada laki-laki muda dengan ras Asia. Kelemahan yang dialami sifatnya sementara dan akan kembali dengan diberikannya koreksi kalium. Laporan kasus yang dilaporkan di Indonesia sangat minim terkait kasus PPT ini. Pada kasus yang kami termukan, laki-laki usia 44 tahun mengeluhkan kelemahan yang diawali kedua kaki kemudian tangan, tetapi membaik dengan sendirinya. Keluhan tersebut dirasakan kembali berulang beberapa hari kemudian. Kasus ini pada awalnya terlambat didiagnosis, sehingga tidak dilakukan koreksi kalium dan terjadi kelemahan berulang. Pemeriksaan laboratorium menunjukkan hipokalemi berat dengan kadar serum kalium 1,81 mmol/L dan kadar tiroksin (T4) tinggi yaitu 44,12 pmol/L dan rendahnya serum *thyroid stimulating hormone* (TSH) <0,0025 IU/mL.

Keywords:
hyperthyroidism;
paralysis;
hypokalemia;
thyrotoxicosis;
T4

INTRODUCTION

Thyrotoxic periodic paralysis (TPP) is a disorder, characterized by abrupt onset of hypokalemia and paralysis.[1] It is more common in men, especially in Asian races, in the ages between the 20s and 40s.[1,2] The clinical of the patient presence hypokalemia and paralysis.[1]The incidence of TPP in Asia is around 1.8%-1.9%.[3] Several conditions can trigger the TPP such as carbohydrate diet, strenuous exercise, stress, and steroid use.[4] Laboratory tests such as potassium level, thyroid-stimulating hormone (TSH), and free thyroxine (FT4) levels help diagnose TPP with limb weakness or paralysis.

CASE

A 44-year-old man complained woke up around 03.00 AM when he wanted to go to the bathroom suddenly felt weak in his legs and could not move his legs. The symptoms slowly showed in his both arms. The patient then tried to move

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both extremities and then recovered for about 2 h. In the afternoon, the patient went to the hospital for a laboratory examination (cholesterol, uric acid, complete blood count), the results were normal. At the first examination, the patient was taking analgesic medication (paracetamol).

One week later, the same complaint appeared when he woke up, after which the patient was taken to the emergency room the next day because he felt the symptoms were getting worse. Several hours before the symptoms appeared, he did not do an excessive activity. The patient said before he slept, he consumed fried rice.

The patient admitted that he felt palpitations, often felt anxious, had frequent sweat, could not sleep at night. But symptoms such as weight lost were denied. This symptom had been felt for about 2-3 mo, but because it was not bothersome so the patient did not try to find doctor. Six months before, he had similar symptoms but healed on their own. The patient denied any history of hypertension, diabetes, and other metabolic diseases. The patient claimed to have a history of kidney stones about 11 years ago. The patient admitted that he rarely had a medical check-up, either a health center or an independent check-up. There was no treatment routinely consumed by the patient before. The patient said there was no similar family history.

Based on the Wayne index as a hyperthyroid score (TABLE 1), a score of 11 was obtained from the patient's signs and symptoms. Interpretation score 11 means equivocal.

TABLE 1. Wayne index

Symptoms of recent onset and/or increased severity	Score	Signs	Present	Absent
Dyspnea on effect	+1	Palpable thyroid	+3	-3
Palpitations	+2	Bruit over thyroid	+2	-2
Tiredness	+2	Exophthalmoses	+2	
Preference for heat	-5	Lid retraction	+2	-
Preference for cold	+5	Lid lag	+1	-
Excessive sweating	+3	Hyperkinesis	+4	-2
Nervousness	+2	Hands hot	+2	-2
Appetite : increased	+3	Hands moist	+1	-2
Appetite : decreased	-3	Casual pulse rate : >80/min	-	-3
Weight increased	-3	>90/min	+3	-
Weight decreased	+3	Atrial fibrilation	+4	-

On physical examination at the emergency room consciousness examination (Glasgow Coma Scale) E4V5M6 was 15, consciousness compos mentis, the blood pressure was 150/100 mmHg, pulse 106x/min, temperature 36.5 ℃. There was no tremor, exophthalmos, or goiter. Meningeal sign examination was negative, cranial nerve examination was within normal limits. Physiological reflexes biceps +2/+2, triceps +2/+2, patella +2/+2, achilles +2/+2. Pathological reflexes were not found. In physical examination showed that the muscle strength in the upper limb 4/5 and leg muscle strength 1/5. One day before

going to the emergency department, he felt fever and take paracetamol.

Laboratory tests in the emergency room found sodium levels of 144.5 mmol/L (normal 136-146), potassium 1.81 mmol/L (normal 3.5-5.1) with an ECG showing the presence of type I AV block (FIGURE 1). The TSH <0.0025 IU/mL (normal 0.35-4.940) was examined, freeT4 was 44.12 pmol/L (normal 9.00-19.05) which led to thyrotoxicosis. Laboratory tests of complete blood, hemoglobin 12.6 g/dL (13.2-17.3), leukocytes 10.17 thousand/mmk (normal 4.5-11.5), erythrocytes 4.65 million/mmk (normal 4.50-6.20), triglycerides 117.0 mg/dL (normal <150), blood sugar at time 111.6 mg/dL (normal 70-140), ureum 43.4 mg/dL (normal 19.0-44.0), creatinine 1.42 mg/dL (0.73-1.18). Initial therapy was infusion of 25 meg KCl in 500 NaCl within 6 h.

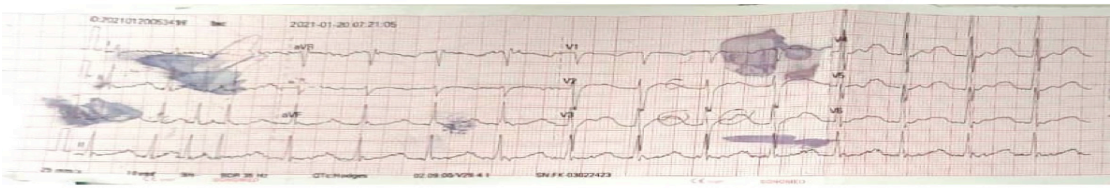

FIGURE 1. ECG

After receiving initial therapy, 5 h later the patient experienced improvement. Motor examination showed muscle strength in the upper limb 5/5 and leg muscle strength 5/5. In addition to hypokalemia therapy, the patient took propylthiouracil (PTU) 100 mg for hyperthyroidism. After giving PTU, follow-up was carried out on a routine outpatient basis once every month at the internal polyclinic. The PTU therapy was still given in this period. Symptoms of hyperthyroidism in the patient such as palpitations, restlessness, tremors, difficulty sleeping but not frequently wer observed. Long-term medication in the form of PTU has still been given up to now. Regular follow-up was carried out every month in the Internal's Clinic to monitor signs and symptoms of hyperthyroidism.

At post-hospital follow-up, tremors were found, weighing 56.5 kg. After two months post-hospitalization and therapy, the patient complained of red urine and sore waist, although the patient was diagnosed with nephrolithiasis. Follow-up three months post-hospital, the patient no longer feels symptoms of hyperthyroidism, no tremor, no restlessness, sleep well.

DISCUSSION

Hypokalemic paralysis is classified into hypokalemic periodic paralysis (HPP) and non-hypokalemic periodic paralysis. Hypokalemic periodic paralysis is hypokalemia due to potassium shift to intracellular. Non-hypokalemic periodic paralysis was by the large amount of potassium deficit that passes through the gastrointestinal or renal.[1] Hypokalemic paralysis periodic can be from family history (familial periodic paralysis/FPP), more common in non-Hispanic Caucasians. Non-familial HPP such as TPP is more common in Asians and Hispanics.[5] The TPP showed hyperthyroidism and also

a rare complication of thyrotoxicosis.[6,7] Any case of hyperthyroidism can lead to TPP, including grave's disease (most commonly). The most common factors that can trigger TPP are consumption of high-carbohydrate foods, high salt/ sodium intake, strenuous exercise, stress, trauma, and medications (diuretics, estrogens, corticosteroids, NSAID, ecstasy).[7] The incidence of TPP is very low and more common in men than women (20:1), even though the incidence of Graves disease is more in women. There is no definite and clear reason for this.[8]

Hypokalemia in the TPP occurs due to a rapid and massive shift of potassium intracellularly.[9] The mechanism of TPP because of hypokalemia and associated paralysis. Progressive muscle weakness to paralysis that occurs due to hypokalemia can be life-threatening but is still reversible. Skeletal muscle is the largest reservoir for potassium in the body (2600 mmol) and has an important role in extracellular potassium homeostasis. The homeostasis process by the Na-K-ATPase pump and the Potassium canal. The K channel regulates the intracellular movement of potassium including inward rectifying (Kir 2.6) and the extracellular movement of potassium.[5,7] Thyrotoxicosis can cause hypokalemia by directly increasing the genetic transcription of the Na-K-ATPase pump thereby increasing the activity of this pump.[10] Thyroid hormone also stimulates beta-2 adrenergic and increases its sensitivity to circulating catecholamines that increase Na-K pump activity.[11] Insulin is known to work by entering blood glucose intracellularly along with the entry of potassium, causing a decrease in potassium levels in the blood.[7]

There are two main examinations on the TPP. Check electrolytes and thyroid values. At the beginning of the examination, the value of potassium is usually less than 3 mmol/liter and can even be 1.1 mmol/liter. When it has entered the healing phase, the periodic paralysis serum potassium can gradually return to normal. The value of thyroid hormone in the blood is high, but TSH will be low.[4] ECG is needed especially if the patient is in hypokalemia for a long time, it induced arrhythmias in cases of TPP.

The occurrence of paralysis is associated with the pathogenesis of intracellular shift potassium. Excessive K into the skeletal muscle cells prevents the muscle cells from being excited, resulting in weakness. The influx of potassium into muscle cells occurs during the rest phase, this may explain why patients experience symptoms at night or dawn during sleep (rest).[12] In this case, the patient consumed fried rice the night before the symptoms, but there's no specific amount of carbohydrate he consumed. Eating high carbohydrates is a trigger factor in the TPP. Hyperinsulinemia plays a role in increasing Na/ K-ATPase activity and inhibiting the efflux from potassium out of muscle cells.[7,8,13] The patient denies any emotional stress due to work or in the family. Emotion, stress, and trauma can be factors that trigger TPP, stress related to hormones. Catecholamines affect Na/K-ATPase activity and inhibit potassium efflux by inhibiting inward-rectifying potassium activity.[14] In this case, an abnormal ECG presence of AV block type 1. Another case report even mentioned acute respiratory failure and ventricular arrhythmias.[9] In another case, the presence of TPP with ventricular tachycardia.[15] The presence of arrhythmias, in this case, can be associated with the occurrence of hypokalemia within a certain time. Low potassium in the extracellular can compensate the body to increase Na channel activity and a buildup of Na in the intracell. Intracell Na accumulation triggers a reduction in Na/Ca exchange and reduces Ca efflux increased

intracellular Ca. Ca overload increases Ca/calmodulin-dependent kinase (CaMKII) activity that can be trigger early after depolarisations (EADs) and induced arrhythmias.[16]

The principle of TPP management is to focus on relieving acute symptoms and preventing recurrences. The patient should be kept in a euthyroid condition to prevent recurrence of paralysis. Recurrent attacks of TPP occur in 60% of patients on anti-thyroid therapy.[8] Potassium is given by intravenous as well as antiarrhythmic propanolol in acute attacks.[6] Identification of the causes of thyrotoxicosis is important for pharmacotherapy. If a known cause such as Graves' disease, a toxic adenoma requiring surgery, needs a definitive therapy such as radioactive iodine or thyroidectomy. Surgery is preferable for cases of Graves with large goiters.[8,13] Education to avoid trigger factors such as maintaining a high carbohydrate diet, strenuous exercise, emotional stress, the use of corticosteroids is important to prevent recurrence.[1,7,13]

In this case report, we described cases of TPP in which the symptoms of hyperthyroidism are not clear. This condition makes it difficult for clinicians to determine further therapy. This article may be helping other clinicians to make therapy in the same case. This report still has limitations, such as a lack of physical examination and follow-up in this case. Urinalysis is needed to see the state of kidney function. A complete thyroid function test may be possible. An EMG may be needed to confirm the cause of the paralysis. Other diagnoses such as metabolic myopathies may be considered which are triggered by exercise, stress, and cold exposure, to be sure about that need for a muscle biopsy. Autoimmune diseases such as myasthenia gravis and GBS can be confused because of similar attacks of paralysis. However, the presence of a low potassium level and the absence of specific clinical myasthenia gravis can confirm the diagnosis of TPP.[17]

CONCLUSION

Thyrotoxic periodic paralysis is a type of hypokalemic periodic paralysis. It is one of the rare cases of complications of hyperthyroidism. It needs to differentiate it from CVA to avoid misdiagnosis. Prevent recurrence of attacks by maintaining euthyroid condition by providing potassium correction therapy and hyperthyroid medication.

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