Thyrotoxic Periodic Paralysis: 
A Rare Case

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Abstract. Hypokalemic periodic paralysis is a rare hereditary disease characterized by recurrent muscle strength loss. It is a reversible disease that is clinical presentation with tiredness and paralysis that develops over hours and days. There are precipitating factors such as stress, exercise, carbohydrate-rich nutrition that trigger the formation of episodes. In our study, hypokalemic periodic paralysis was diagnosed as a result of a tetraplegic examination of a patient in our emergency department. Because this case is rare, the literature has been presented with review. A 43-year-old male patient with a known history of lumbar disc hernia was brought to the emergency service of our hospital because of the complaint of being unable to move his body when he woke up at 06:00 in the morning. He used 1 oral metamizole sodium 12 hours ago due to headache complaint, and he was fed with high carbohydrate content at dinner. Physical examination revealed a blood pressure of 105/70 mm/hg, a pulse of 93/min, a temperature of 36.8 degrees, a total loss of strength in four extremities, and tetraplegia. Sensory loss, ophthalmopathy, tremor were not detected, pupillary isochoric and light reflex was bilateral, achilles tendon reflex was taken as hypoactive. Eye sizes were normal, thyroid tissue was non-palpable. This case was evaluated as TPP because of the time of onset of attacks, the onset after high-carbohydrate feeding, causing paralysis, improvement of symptoms with replacement, serum potassium, thyroid function tests, and ECG findings. His dramatic response to treatment supported our recognition. In conclusion, it should be considered as a differential diagnosis in young and especially male patients presenting with TPP motor paralysis, which is rare. Determination of thyroid hormones and potassium levels helps in diagnosis.

Keywords: thyrotoxicosis; hypokalemic periodic paralysis; hypokalaemia

Introduction
Thyrotoxic periodic paralysis (TPP) is a rare disease with recurrent muscle weakness or paralysis. Attacks range from involvement of a group of muscles to generalized paralysis. It has three forms: hypokalemic, hyperkalaemic and normokalaemic [1, 2]. Attacks may be accompanied by an increase in serum creatine phosphokinase and phosphate levels. Hyperthyroidism findings may not be evident during attacks. This situation makes the diagnosis of TPP difficult [3]. It is detected with a prevalence of 100,000 : 1 as the most common form of periodic paralysis [4].

Clinical case
A 43-year-old male patient with a known history of lumbar disc hernia was brought to the emergency service of our hospital with the complaint of being unable to move his body when he woke up at 06:00 in the morning. He used 1 oral metamizole sodium 12 hours ago due to headache complaint, and he was fed with high carbohydrate content at dinner. Physical examination revealed a blood pressure of 105/70 mm/hg, a pulse of 93/min, a temperature of 36.8 degrees, a total loss of strength in four extremities, and tetraplegia. Sensory loss, ophthalmopathy, tremor were not detected, pupillary isochoric and light reflex was bilateral, achilles tendon reflex was taken as hypoactive. Eye sizes were normal, thyroid tissue was non-palpable. This case was evaluated as TPP because of the time of onset of attacks, the onset after high-carbohydrate feeding, causing paralysis, improvement of symptoms with replacement, serum potassium, thyroid function tests, and ECG findings. His dramatic response to treatment supported our recognition. In conclusion, it should be considered as a differential diagnosis in young and especially male patients presenting with TPP motor paralysis, which is rare. Determination of thyroid hormones and potassium levels helps in diagnosis.
0.4–3 μIU/mL), Hb 12.3 g/dl (12–16.2 g/dL), complete urinalysis, blood gas, brain tomography, brain MRI images were reported as normal, no lesion explaining paralysis was detected. ST segment depression and U segment flattening were observed in the electrocardiography of the patient (figures 1, 2).

Considering thyrotoxic periodic paralysis as a pre-diagnosis, intravenous replacement 10 mmol/hour potassium chloride was initiated and 0.5 × 40 mg peroral propranolol. At the end of the 24-hour follow-up, when the muscle strength in the extremities was 3/5, potassium 4.4 mmol/L and phosphorus 4.2 mg/dL in the examinations, she was hospitalized in the internal medicine service. In the thyroid ultrasonography the thyroid parenchyma was coarsened, heterogeneous, and its echogenicity was reduced, and vascularization was reported as increased in Rdus examination. Sent TSH 0.0006 μIU/ml (0.4–3 μIU/mL), free T₃ 5.5 pg/ml (2.60–4.80 pg/ml), free T₄ 2.45 ng/dL (0.7–2.0 ng/dL), TSH receptor antibody positive, anti-TPO 436.0 IU/ml (1–5.6 IU/ml) was detected. The patient was diagnosed with Graves’ disease. Peroral propylthiouracil 3 × 50 mg was added to his treatment. Control electrocardiography was observed in sinus rhythm.

At the 48th hour, when muscle strength in the extremities was observed as 5/5 examination finding, his treatment was arranged and he was discharged with a control recommendation. When the patient applied for control 1 month later, TSH was 2.5 μIU/ml, free T₃ 1.1 pg/ml, free T₄ 1.5 ng/dl, thyroid scintigraphy — both lobes were normal sized, with the right lobe being more prominent, and irregular activity involvement was detected in the lower parts. The current treatment of the patient was continued.

Discussion

Thyrotoxic periodic paralysis (TPP) is a rare complication of thyrotoxicosis. It has been reported that thyrotoxic periodic paralysis is more common in Southeast Asia than other regions, and the prevalence of TPP in thyrotoxicosis is 1.9 % in China and 0.1–0.2 % in western societies [5]. TPP is mostly seen in men, as in our case, it is more common in men than women. Although hyperthyroidism is mostly seen in women, TPP is frequently seen in men. The male/female ratio is 13/1. TPP usually starts between the ages of 20–30, but approximately 80 % occurs during the third decade. In periodic paralysis due to thyrotoxicosis (TPP), hypokalemia develops due to the increase in Na/K-ATP less pump activity, especially in muscle cells [12]. Due to the increase in pump activity, potassium rapidly passes from the extracellular compartment to the intracellular compartment [8, 10].

The events that cause paralysis with hypokalaemia and hypophosphatemia in patients with TPP are complex. These include hyperthyroidism, genetic and racial predisposition, an exaggerated insulin response, hypadrenergic state, and possibly other mechanisms that lead to intracellular shift of K and P [13]. In TPP, attacks are often triggered by a high carbohydrate meal or severe exercise, and occur after a period of eating or heavy exercise. In the presented case, dinner history with high carbohydrate content is consistent with the literature. Trauma, surgery, alcohol, insulin, catecholamine, glucocorticoid intake, menstruation, infection, diarrhea, and stress can also trigger attacks. During the attack, the patient wakes up with weakness in the morning or feels heaviness in his legs when he wakes up in the morning, and weakness begins in his arms and legs in a short time. As the weakness progresses, deep tendon reflexes become hyporeactive [3]. In the most severe form of the attack, the patient becomes unable to move any of his extremities. There is no sensory complaint during an attack [4]. There was no sensory complaint in our patient either. ECG changes associated with hypokalemia (U waves, ST segment depression, QT prolongation, and T wave flattening) and cardiac arrhythmias are common during an attack [3]. During exercise, it causes K to exit the cell and potassium returns to the cell by the pump activity [17].

Figure 1. ST segment depression and U segment flattening

Figure 2. ECG after treatment
cell at rest. Unlike familial hypokalemic periodic paralysis, TPP treatment is considered in two stages. In the acute period, parenteral potassium replacement is the most effective treatment to correct muscle strength weakness [11]. In the second stage, thyrotoxicosis should be treated, replacement therapy should not exceed 10 mEq/hour, considering the risk of rebound hyperkalemia [6].

Biochemically, high TSH levels are not expected in hypokalemic periodic paralysis. Ryan et al. found that one out of every three TPP patients had a mutation in the gene encoding the potassium channel and hypothesized that TPP might have a channelopathy. In addition, it was thought that the Na/K-ATP-less pump was upregulated in skeletal muscles, triggering the thyrotoxic periodic paralysis picture in the thyrotoxicosis picture [7].

**Conclusions**

Our case was evaluated as TPP because of the time of onset of attacks, the onset after high-carbohydrate feeding, causing paralysis, improvement of symptoms with replacement, serum potassium, thyroid function tests, and ECG findings. His dramatic response to treatment supported our recognition. In conclusion, it should be considered as a differential diagnosis in young and especially male patients presenting with TPP motor paralysis, which is rare. Determination of thyroid hormones and potassium levels helps in diagnosis.

**Conflicts of interests.** Authors declare the absence of any conflicts of interests and their own financial interest that might be construed to influence the results or interpretation of their manuscript.

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**Тиреотоксичний періодичний параліч: рідкісний випадок**

Резюме. Тиреотоксичний гіпокаліємічний періодичний параліч є рідкісним спадковим захворюванням, що характеризується періодичною втратою сили м’язів. У проведенному дослідженні діагностовано періодичний гіпокаліємічний параліч у результаті обстеження пацієнта у відділенні невідкладної допомоги. Оскільки цей випадок рідкісний, література подана з оглядом. 43-річний пацієнт чоловічої статі з відмінною грижею поперекового диска в анамнезі був доставлений...
Клинический случай

Пациент, 43-летний мужчина с известной грыжей поясничного диска, был доставлен в нейрохирургическое отделение с жалобами на невозможность движения тела после пробуждения в 6:00 утра. Пациент принимал метимазол в таблетках 12 часов назад из-за жалоб на головную боль. Ужин состоял из продуктов с высоким содержанием углеводов. Клинический осмотр выявил артериальное давление 105/70 мм рт.ст., частоту сердечных сокращений 93 в 1 мин, температуру 36,8 градуса, общую потерю силы в четырех конечностях и тетраплегию. Потеря чувствительности, офтальмопатии, тремора не обнаружены.

Клинический осмотр и общее состояние пациента позволили подозревать тиреотоксикоз, тиреотоксический периодический паралич, тиреотоксический гипокалиемический периодический паралич, редкий случай.

Рассматривался как дифференциальный диагноз у молодых, особенно у пациентов мужского пола, страдающих от двигательного паралича. Определение гормонов щитовидной железы и уровня калия помогают в диагностике.

Ключевые слова: тиреотоксикоз; тиреотоксический периодический паралич; гипокалиемия.