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Case Report

Thyrotoxic Periodic Paralysis after Surgery: A Case Report and Literature Review

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ABSTRACT

Thyrotoxic periodic paralysis (TPP) is a rare but potentially life-threatening muscle disease in a thyrotoxic patient. Due to the lack of knowledge, the diagnosis is easily missed. Diagnosis of TPP is made by documentation of hypokalemia and hyperthyroidism in acute generalized muscle weakness. Hypokalemia is caused by an intracellular shift of potassium because of an increased Na-K-ATPase pump activity and the inhibition of the inward rectifying potassium (Kir) channels. The initial treatment aims to treat potentially life-threatening cardiac arrhythmias caused by severe hypokalemia. This can be done with potassium substitution and nonselective beta-blockers, being aware of the potential of rebound hyperkaliemia. Definitive treatment consists of avoiding triggers and correcting the hyperthyroid state with antithyroid drugs, radioactive iodine, or thyroidectomy. Once patients are, permanently euthyroid complete remission of the paralytic attacks usually occurs. We present a case of a 37-year-old male with diffuse muscle weakness after surgery. A new diagnosis of thyrotoxic periodic paralysis was made in the emergency department. This review summarizes the epidemiology, clinical manifestations, pathogenesis, diagnostics, and treatment of TPP.

Keywords
Thyrotoxic periodic paralysis; Hypokalaemia; Potassium.

INTRODUCTION

Thyrotoxic periodic paralysis (TPP) is a rare but potentially life-threatening muscle disease in a thyrotoxic patient. TPP is characterized by hypokalaemia due to an intracellular shift of potassium and episodes of acute generalized muscle weakness in a setting of thyrotoxicosis. Prompt identification, restoration of normal potassium levels, and correction of the underlying thyrotoxic state are primordial in avoiding cardiopulmonary complications and recurrences of paralysis.

We present a case with a new diagnosis of TPP after minor elective surgery and a review of the literature focusing on pathogenesis, diagnostics, and treatment.

CASE REPORT

A 37-year-old male in previously good health presented to the emergency department (ED) with diffuse muscle weakness and myalgia for a few hours. The day before, he had surgery under general anesthesia by orthopedists on synostosis of the base of the right fifth metatarsal. The procedure went without complications, and the patient could leave the hospital the same day. Progressively he lost the strength in his left leg and had difficulty mobilizing. Thinking of a post-operative complication, he was admitted to the ED the next day. On admission, the patient had a full range of motion in the four limbs but with diffuse reduced strength. No differences were noted between proximal and distal muscles. Sensation remained intact.

Serum biochemical parameters showed hypokalaemia (2.08 mmol/L), hypophosphatemia (0.60 mmol/L), and slightly elevated creatine kinase (215 U/L). Electrocardiogram showed a sinus rhythm of 64/min and a prominent U-wave (Figure 1).

Thyroid function tests were measured and showed an immeasurably low thyroid stimulating hormone (TSH) (<0.0050 mU/L) and increased T4 (39.6 pmol/L) supporting a new diagnosis of hyperthyroidism complicated by thyrotoxic periodic paralysis. Under continuous monitoring of the vital parameters, the patient received an intravenous infusion with 40 mEq potassium chloride and 1 g of magnesium sulfate over one-hour, resulting in the normalization of the potassium levels (3.4 mmol/L), an improvement in strength and disappearing of U-waves on the electrocardiogram (Figure 2).

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Figure 1. *Electrocardiogram before Potassium Substitution*

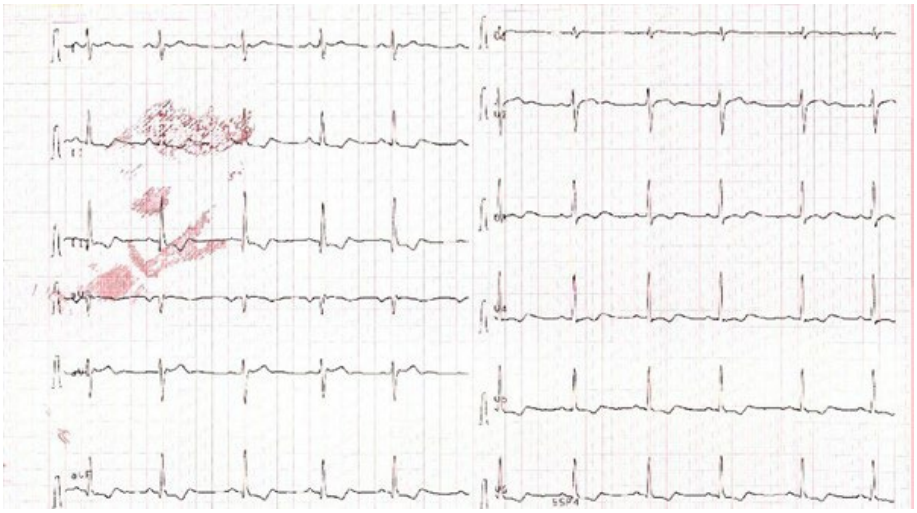
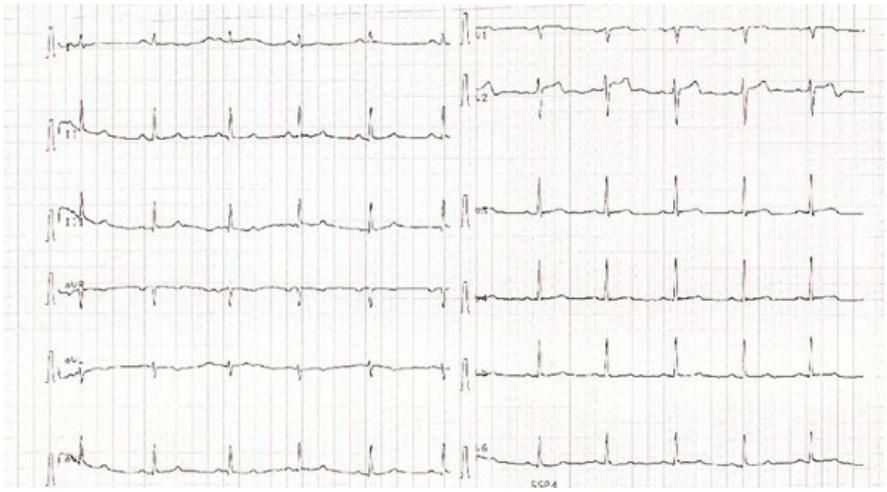


Figure 2. *Electrocardiogram after Potassium Substitution*



The patient was admitted to the endocrinology ward. Treatment with antithyroid drugs, beta-blocking medication, and oral potassium supplementation was started. Further in-depth anamnesis revealed that the patient showed fatigue, tremor, and irritability for the past two-months. After three-days of observation, treatment with antithyroid drugs, beta-blocking medication, and oral potassium supplementation, the patient had no symptoms with no occurrence of rebound hyperkalemia. Follow-up was provided at discharge.

EPIDEMIOLOGY

TPP is a sporadic form of hypokalaemic periodic paralysis associated with hyperthyroidism. Any cause of hyperthyroidism can lead to TPP. The severe disease is the most common, but it can also be related to toxic nodular goiter, solitary toxic nodus, iodine-induced thyrotoxicosis, excess use of exogenous thyroxine, thyroiditis, thyrotropin-secreting pituitary adenoma and amiodarone-induced thyrotoxicosis.[1] Unlike familial hypokalaemic periodic paralysis, there is no autosomal inheritance.[2,3]

Despite the high prevalence of thyrotoxicosis in women, TPP mainly affects Asian or Polynesian men. The male-to-female ratio is 20:1.[4-7] In the Chinese and Japanese thyrotoxic populations, there is an incidence of respectively 1.8 and 1.9%.[5,6] In men, this is 8.7-13%.[5,6] Due to migration, the incidence in other populations is increasing to 0.1-0.2%.[1,8,9] Eighty percent (80%) of the patients have an onset of the disease at 20-39-years-old.[1,5,8,10] Other forms of periodic paralysis have a younger onset.

Triggers of the disease include stress (surgery, infection, trauma, psychological), rest after physical activity, menses, carbohydrate-rich foods, cold exposure, alcohol, and illegal intake, drugs (corticosteroid therapy, beta-2-adrenergic bronchodilators, epinephrine, insulin, thyroid supplements, diuretics, Non-steroidal anti-inflammatory drugs (NSAIDs), fluoroquinolones, aminogly-

coside).[7,11] Attacks mostly occur during the night or in the early morning.[11,12] In subtropical regions, a seasonal variation can be observed with increased episodes during the summer months.[6,7,11]

PATHOPHYSIOLOGY

The pathophysiology of TPP is still not fully known. Skeletal muscles are the largest stores of total body potassium, and potassium efflux by myocytes plays a vital role in maintaining extracellular potassium homeostasis. This balance is maintained by the Na-K-ATPase pump, which provides the intracellular movement of potassium, and the potassium channels, including inward rectifying potassium (Kir) channels that contribute to the extracellular movement of potassium.[13] Hypokalaemia is not the consequence of a total body potassium shortage but of an increased intracellular shifting into the muscles (Figure 3).[1,13]

More significant amounts of thyroid hormone and greater sensitivity of the pump to thyroid hormone due to genetic predisposition lead to an increased Na-K-ATPase pump activity in the sarcolemma and an increased potassium influx into the cell.[14-16] In addition, there is also an indirect effect through increased b2 adrenergic activity with an increased impact on catecholamines, hyperinsulinemia, and the stimulating effect of androgens on the Na-K-ATPase pump activity.[17-21]

Ion channel defects, only interested in hyperthyroid states, form a second mechanism in the pathophysiology. Several sensitivity loci allow for KCNJ-2 expression. This gene encodes the Kir2.6 channels in skeletal muscles regulated by the thyroid hormone. In 1/3rd of the patients, a loss-of-function mutation occurs in the genes that correlate for this Kir2.6. As a result, the potassium efflux is disturbed.[22,23]

The decreased extracellular potassium leads to depolarization of the sarcolemma instead of hyperpolarizing. The hypokalaemia-induced paradoxical effect causes the inactivation of the sarcolemma's Na channels, leading to paralysis of the skeletal muscles.[24,25]

Several external causes stimulate the Na-K-ATPase pump and inhibit the Kir channels. Stressful impulses, including surgery, infection, and trauma, cause increased beta-adrenergic activity. Exercise leads to potassium efflux, while rest causes an influx. As a result, paralysis occurs more often at night when people are inactive.[8,25] In addition, a carbohydrate-rich diet increases insulin release. Insulin and catecholamines activate the Na-K-ATPase pump and inhibit the Kir channels.[23] Finally, the higher incidence of TPP in men can be explained through the high androgen levels stimulating the pump. Estrogen and progesterone, on the other hand, have an inhibitory effect.[26,27]

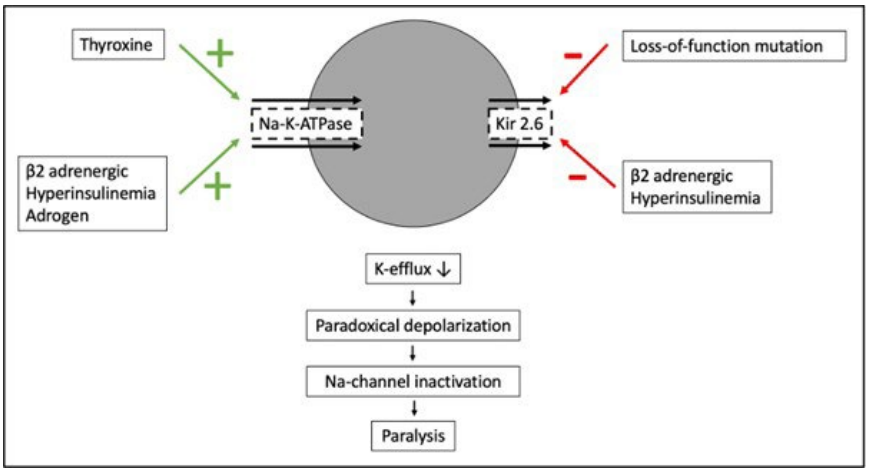
CLINICAL FEATURES

Thyrotoxic periodic paralysis is characterized by sudden episodes of generalized muscle weakness ranging from mild weakness to complete paralysis without loss of consciousness. Proximal muscle groups and lower extremities are more severely affected than distal muscles and upper extremities.[8] The muscles affected may be asymmetrical. Tendon reflexes can be decreased, but sensation remains intact. Less than 50% of the patients experience prodromal symptoms like mild myalgias and stiffness.[6] In severe cases, bulbar weakness, respiratory depression with hypercapnic respiratory failure, and fatal ventricular arrhythmias have been reported.[28,29] Symptoms of hyperthyroidism do not always precede TPP. Forty-three to sixty percent (43-60%) of the patients have symptoms at the moment of the TPP, but often they are very subtle.[2,5,6,8,9,26] Other possible symptoms include palpitations, tachycardia, dyspnoea, dysphagia, speech and visual changes, and rhabdomyolysis.[11,12] Frequency can vary between once a week to every few months. Most attacks resolve within 36-hours but can last from minutes to days.[7,30]

DIAGNOSTICS

Essential is the documentation of hypokalaemia and hyperthyroidism. The severity of hypokalaemia corresponds to the intensity of muscle weakness. The average value is 3 mmol/l but can go as low as 1.1 mmol/L.[1] A low serum phosphate and magnesium are associated with the potassium influx.[12] The urine calcium to phosphate ratio greater than 1.7 mg/mg has a good discrimina-

Figure 3. *Pathophysiology of TPP*



tory power between TPP and non-TPP hypokalaemic paralysis.[31] Hyperthyroidism is characterized by decreased thyroid-stimulating hormone (TSH) and increased T4 and/or T3. Creatine kinase can be normal to slightly elevated in 2/3rd of the patients.[12]

A triad of sinus tachycardia represents electrocardiographic changes due to a hyperadrenergic state, hypokalaemia characteristic (U-waves, ST-depression, QT-prolongation and T-wave flattening) and paradoxically prolonged PR interval caused by thyrotoxicosis. Other findings are tachyarrhythmias, atrioventricular blocks and cardiac arrest.[32,33]

When performed, an electromyogram shows a myopathy with reduced duration of muscle action potentials, decreased amplitudes, and increased polyphasic possibilities. These characteristics can resolve in the euthyroid state.[1,34]

DIFFERENTIAL DIAGNOSIS

Acute paralysis can occur due to a problem with the central, peripheral nerve system, the neuromuscular junction of the skeletal muscle. TPP, being a muscle disorder, needs to be differentiated from other muscular disorders like myasthenia gravis, Guillain-Barré syndrome, transverse myelitis, acute thyrotoxic myopathies, tick paralysis, and botulism. Other forms of periodic paralysis should also be ruled out.[1,12,35,36]

TREATMENT

Treatment of TPP proceeds in steps. Acute therapy includes preventing major cardiopulmonary complications and reversing paralysis.[37] Finally, treatment is needed to restore the euthyroid state and prevent future attacks.

In the event of electrocardiographic changes, hypokalaemia needs to be treated. A proposed scheme consists of 30 mEq oral potassium every two-hours with a maximum of 90 mEq every 24-hours until improvement of the muscle weakness.[38] Others propose a more careful approach with less than 10 mEq each hour because the pathophysiology of TPP does not involve potassium depletion but an intracellular shift.[39] Therefore, rebound hyperkalemia and hyperphosphatemia at improvement is a risk.[12,40] In the case of severe hypokalaemia, a faster correction with intravenous substitution of potassium is more appropriate. Most patients need 10-200 mEq of potassium.[39] Because of their risk for cardiac arrhythmias, patients with TPP must permanently be hospitalized and monitored.

When there is no initial response, oral or intravenous non-selective beta-blockers, such as propranolol, can be used as an alternative treatment. The non-selective beta-blockers reverse the Na-K-ATPase pump's adrenergic stimulus, causing a decreased catecholamine-mediated potassium uptake. The proposed dosage is 1 mg of propranolol intravenous every 10-minutes with a maximum of 3 mg. Oral intake is dosed at 3 mg/kg. Propranolol can shorten the recovery time without the risk of rebound hyperkalemia.[41,42]

The second stage, when hospitalized, includes restoration of the euthyroid state to eliminate attacks of TPP in the future, as paralytic episodes can re-emerge if thyrotoxicosis recurs. The management of hyperthyroidism differs according to the underlying etiology. Definitive treatment of TPP requires correction of the thyrotoxic state with antithyroid drugs, but radioactive iodine or thyroidectomy appeared more effective.[43,44]

Propranolol can be used temporarily until the euthyroid state is achieved. However, daily potassium supplementation is not beneficial for prophylaxis against further paralytic attacks.[1,8,35] Precipitating factors, such as heavy exercise and high-carbohydrate diets, must be avoided while awaiting the normalization of the thyrotoxic state.[1,35]

Once patients are, permanently euthyroid complete remission of the paralytic attacks usually occurs.[35]

CONCLUSION

Thyrotoxic periodic paralysis is an endocrine emergency because of the risk of cardiac arrhythmias. Due to the lack of knowledge of this rare disease, the diagnosis is easily missed. Early diagnosis allows treatment and prevents recurrent episodes in the future. Patients with acute muscle weakness or paralysis should be evaluated for TPP by checking electrolytes and thyroid function testing. This is usually seen in young Asians, but the incidence in other populations is increasing caused by migration. The pathophysiology remains uncertain but seems to be based on increased Na-K-ATPase pump activity and the inhibition of the Kir channels, both resulting in increased intracellular potassium. Acute treatment consists of potassium supplementation and non-selective beta blockers. Avoiding known triggers and restoring the euthyroid state by antithyroid drugs, radioactive iodine, or thyroidectomy prevents morbidity and mortality.

As described in the presented case, surgery is a known trigger for an acute episode of TPP. When the patient is already known with TPP, a thyroid function test needs to be checked, and elective surgery needs to be postponed until the treatment of the hyperthyroid state.[45] When patients are permanently euthyroid, a complete remission of the paralytic attacks is observed, and surgery can be performed safely.

INSTITUTIONAL BOARD PERMISSION

Yes.

CONSENT

The authors have received written informed consent from the patient.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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