

Abstract

Thyrotoxic Periodic Paralysis (TPP) is a rare and life threatening condition commonly occurring in young Asian males. It is characterized by acute paralytic attacks and hypokalemia in association with thyrotoxicosis. Serum potassium levels may be normal in rare cases of TPP. The diagnosis of normokalemic TPP may be overlooked and/or delayed in most cases. Here, we describe a 32-year-old Iranian man with normokalemic TPP misdiagnosed as somatization disorder with the correct diagnosis made one year after the onset of symptoms.

Keywords: hypokalemia, hypokalemic periodic paralysis, thyrotoxic periodic paralysis, normokalemic, periodic paralysis

Introduction

Thyrotoxic Periodic Paralysis (TPP) is a rare condition which mainly involves young Asian males in their 3rd decade of life (1,2). It presents with acute paralytic attacks and hypokalemia in association with thyrotoxicosis (3,4). Although serum potassium levels decrease in most patients with TPP, it is not essential for establishing the diagnosis, and some cases of TPP have normal potassium levels even in the acute paralytic phase. Normokalemic TPP may be misdiagnosed as other neurologic diseases such as Guillain-Barré syndrome, multiple sclerosis, hysteria, or malingering (5,6). Here, we report a young Iranian man with newly diagnosed Grave's disease and TPP who was normokalemic during the acute phase of paralysis. Hypokalemia was documented after resolution of paralytic attacks during subsequent days of admission.

Case report

A 32-year-old man with Grave's hyperthyroidism treated with Methimazole and Propranolol (15 mg and 30 mg per day, respectively) from three months ago referred to the endocrine clinic with friend, complaining of acute symmetrical lower limb muscle weakness started from early morning in a resting position. He was admitted to the Emergency Department of Emam Khomeini Hospital, Urmia. He had repeated attacks of severe muscle weakness or limb paralysis (pre-dominantly proximal muscles of lower limbs) that started from one year prior to admission, and misdiagnosed as somatization

disorder by a neurologist. Each paralytic attack lasted from 8 h to 24 h, and resulted in inability to walk and stand without assistance. His paralytic attacks resolved spontaneously without any medication. There was not a history of similar disorders or other endocrine diseases in his relatives.

The patient mentioned palpitation, heat intolerance, diaphoresis, irritability, distal tremor, and weight loss. On physical examination, he had normal vital sign, diffused thyromegaly, and weakness in lower extremities. Deep tendon reflexes were normal. He was conscious, and sensory tests of extremities and cranial nerves revealed no abnormality. The findings of heart and lung examination were unremarkable.

Laboratory tests revealed normal calcium, magnesium, blood sugar, and muscle enzyme levels, as well as normal renal and liver function. Serial potassium concentrations were: 4.49→4.27→3.56→2.6→4.8 mEq/L. Other tests results were: sodium: 146 mEq/L; serum T4:14.9 µg/dL (4.7–12.5) serum T3: 2 ng/dL (0.7–2.1); serum TSH: 0.1 mIU/L (0.17–4.05); and Anti TPO Ab: 877.60 IU/mL (normal < 50). ECG and EMG–NCV findings were unremarkable.

A diagnosis of TPP was made based on clinical and laboratory findings. The doses of Propranolol and Methimazole were increased to 60 mg and 20 mg per day, respectively, and his symptoms resolved gradually over the next hours.

Serum potassium level was monitored and fell to 3.56 mEq/L and 2.6 mEq/L on the 4th and 5th day of admission, respectively, in the absence of any paralytic symptom. He was administered low-dose oral potassium chloride (KCl) tablets

during the hypokalemic phase and a low-carbohydrate diet.

Discussion

Thyrotoxic Periodic Paralysis is a fatal but curable disorder with unclear pathogenesis (6). Overstimulation of Na⁺/K⁺ ATPase pump in cell membranes of skeletal muscles caused by excess thyroid hormone levels, β_2 -adrenergic stimulation, and hyperinsulinemia are some mechanisms that lead to potassium shift into the intracellular compartment, hypokalemia, and flaccid paralytic attacks of skeletal muscles that last from 3 h to 36 h (6,7). Deep tendon reflexes are decreased or absent in most patients; some cases, however, have normal jerking response during acute paralytic attacks (6). Although potassium level is low during TPP attacks, there are some reports of normal and even high potassium levels in the literature that can be misleading and thus delay the correct diagnosis of the disorder (4,5,7). Therefore, a diagnosis of TPP must be considered in the setting of periodic paralytic attacks and hyperthyroidism with decreased or even normal potassium levels. TPP with normal potassium levels can mimic other neurologic disorders such as myasthenia gravis, Guillain-Barré syndrome or psychogenic paralysis (5).

Our patient had previously been misdiagnosed as somatization disorder. The diagnosis of TPP was made one year later after evaluation of thyroid function and serial monitoring of potassium level. A one-year delay in diagnosis of TPP in this case may be due to subtle or absent clinical signs of hyperthyroidism at the onset of paralytic attacks or normal potassium levels during paralytic attacks at the time. The patient's previous medical records were not available, thus preventing us from commenting on the range of his other neuropsychological disturbances observed with hyperthyroidism, which could have masked the typical endocrine/systemic manifestations of the condition at the onset of his paralytic attacks. Treatment of TPP includes potassium replacement therapy via intravenous or oral route based on the severity of hypokalemia, and its presentation during the acute attacks; however, it cannot prevent acute paralysis if administered between attacks (6,7). Non selective β -blockers such as propranolol are useful for both treatment and prevention of recurrence of attacks (6,7). The main treatment of TPP involves

control of hyperthyroidism using anti-thyroid medications, radioiodine ablation or surgical therapy (5,6). Patients must be advised to avoid alcohol intake, salty foods, high carbohydrate content meals, and effortful exercise that can trigger acute attacks of TPP (6).

Conclusion

Normokalemia during paralytic attacks dose not exclude the diagnosis of TPP. Thus, appropriate treatment of TPP is necessary, and serial monitoring of potassium levels is recommended. We emphasize that mechanisms other than hypokalemia alone may have a role in provoking paralytic attacks in TPP.

Conflict of interests

The authors declare that they do not have any conflict of interest.

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Authors' Contributions

Conception and design, drafting of the article, critical revision of the article for important intellectual content and final approval of the article: VN

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