Case Report

Recurrent hypokalemic paralysis: An atypical presentation of hypothyroidism

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ABSTRACT

Hypothyroidism presenting as recurrent hypokalemic paralysis is rare in the literature. This transient and episodic neurological condition is commonly associated with thyrotoxicosis. We report a case of young female admitted with recurrent paralytic attacks since last 1 year. She had no symptom of hypothyroidism. She had weakness of all four limbs, delayed relaxation of ankle jerks, and normal higher mental function. There was no enlargement of thyroid. Serum potassium level ranged from 1.6 to 3.2 meq/L during attack with high serum creatine phosphokinase level. Electromyography was normal. The patient was diagnosed having chronic thyroiditis with high thyroid-stimulating hormone and thyroid-related antibodies. Follow up shows satisfactory result with thyroxine replacement. It is an extremely rare and unusual presentation of hypothyroidism, probably the fourth reported case of hypothyroidism with hypokalemic paralysis, to the best of our knowledge.

Key words: Hypokalemia, recurrent paralysis, hypothyroidism

Introduction

Hypokalemic periodic paralysis is the most common periodic paralysis, a rare channelopathy manifested by episodic flaccid weakness secondary to abnormal sarcolemmal excitability. Hypokalemic paralysis may be caused by a short-term shift of potassium into cells, seen in hypokalemic periodic paralysis (caused by familial periodic paralysis or thyrotoxic periodic paralysis), or a larger deficit of potassium as a result of severe renal or gastrointestinal potassium loss.

Thyrotoxicosis is the most common cause of secondary hypokalemic periodic paralysis. Recurrent hypokalemic paralysis is an extremely unusual presentation of hypothyroidism. To the best of our knowledge, this is the fourth reported case of hypothyroidism associated with recurrent hypokalemic paralysis.[1-3]

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A 30-year-old female presented with recurrent attacks of acute flaccid paralysis of all four limbs since last 1 year. Each episode lasted for 2 to 5 days followed by spontaneous complete recovery without potassium supplement in any form. Frequency of attack gradually increased up to three episodes per month. It usually started with early morning weakness without any diurnal variation. There was no history suggestive of altered sensorium, convulsion, visual, respiratory, or bulbar weakness. She had no symptom suggestive of hypothyroidism.

The patient had quadriparesis with hypotonia, diminished deep tendon reflexes except delayed relaxation of ankle jerks, flexor plantar response, and prominent neck muscle weakness. She had normal higher mental function without any cranial nerve, sensory, or sphincter involvement. She was thin built without pallor or edema. Her thyroid gland was not palpable. Other system examination including chest and abdomen was within normal limit.

Laboratory investigations showed normal hemoglobin with high ESR, low potassium, and normal sodium and serum creatine phosphokinase (CPK) level was very

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high (1067 mg/dl). Her electromyography and nerve conduction study were normal. Thyroid function test revealed very low level of thyroxine (both T3 and T4) with very high thyroid-stimulating hormone (TSH > 100). Serum anti-TPO antibody titer was also very high (386.4 IU/ml). Hypokalemia persisted during attack ranging from 1.6 to 3.2 meq/L. Hypokalemic paralysis was diagnosed based on clinical and biochemical parameters. No cause of secondary hypokalemia could be detected. Her 24-h urinary potassium excretion was 11.14 meq/L which was much below normal (normal range 25-120 meq/L). Normal serum magnesium and urinary calcium excretion ruled out the possibility of Gitelman's syndrome. Urine pH was within normal limit. The computed tomography (CT) scan of the abdomen demonstrated normal adrenal gland. Her plasma rennin activity (PRA) was measured normal (2.6 ng/ml/h).

During the acute period in hospital, the patient was treated with intravenous potassium (IV potassium chloride, 40 meq/L of normal saline through peripheral vein at a rate of 20 meq/h) for a week that lead to clinical recovery and also biochemical improvement to some extent. After starting oral levothyroxine replacement, the patient continued with oral potassium replacement (oral potassium chloride solution 40 meq twice daily) for another 2 weeks after which she could be safely maintained with levothyroxine only. Hypokalemic state persisted up to 4 weeks of levothyroxine replacement though the patient was clinically well. Subsequently, serum TSH became normal with normal serum potassium level. With adequate control of hypothyroidism, the patient did not have the need to take potassium supplement and no further attack of acute flaccid weakness has been reported so far (for a period of 1 year during follow up).

Discussion

Periodic paralysis may be primary or secondary type. The paralytic attack can last from an hour to several days and the weakness may be generalized or localized.[4] Disturbances of potassium equilibrium can produce a wide range of disorders including myopathy, marked muscle wasting, diminution of muscle tone, power, and reflexes.[5] The primary hypokalemic periodic paralysis is autosomal dominant and is exacerbated by strenuous exercise, high carbohydrate diet, cold and excitement, which was not found in this case.[4] In the primary type, episodes of weakness recur frequently.

Many cases of secondary periodic hypokalemic paralysis have been reported in association with gastroenteritis, diuretic abuse, renal tubular acidosis, Bartter syndrome, villous adenoma of colon, and hyperthyroidism.[6] There was no history of diarrhea, vomiting, or diuretic abuse in the present case. The absence of polyuria, polydipsia, nausea, vomiting, constipation, hypochloremia, and hyponatremia ruled out Bartter syndrome. Normal serum magnesium and urinary calcium excretion ruled out the possibility of Gitelman's syndrome. Similarly, none of clinical features of renal tubular acidosis like polyuria, polydipsia, acidotic breathing, rickets, and pathological fractures were present in this case.[7] Laboratory finding such as normal urinary pH and lack of hyperchloremia during episode of paralysis also excluded the possibilities of renal tubular acidosis. Characteristic features of hyperaldosteronism like hypertension and polyuria were absent with normal adrenal gland in the CT scan of the abdomen.

The levels of thyroid hormones and TSH values in this patient indicate severe deficiency of thyroxine. The presence of autoimmune thyroiditis is indicated by the high titer positivity of anti-TPO antibodies in serum. The persistent hypokalemia during early periods of thyroxine replacement can be due to the fact that thyroxine in pharmacological doses can cause increased potassium excretion and water diuresis in patients with myxedema during initial part of therapy. This may result in hypokalemia, especially in a patient with malnutrition and low stores of total body potassium.

Hypokalemic periodic paralysis though common among Indian population varies greatly in disease spectrum and magnitude in our country due to the heterogeneous pattern of etiology behind it. Two case series that studied hypokalemic periodic paralysis in tertiary care centers of India have observed that around 45% of all those patients had a secondary cause for their condition and this secondary group had more severe hypokalemia that needed longer time to recover.[8,9] Thyrotoxicosis, renal tubular acidosis, Gitelman's syndrome, and primary hyperaldosteronism were among the prime conditions leading to hypokalemic periodic paralysis but no case of hypothyroidism was found to be the etiology behind it.

The association of periodic hypokalemic paralysis with hypothyroidism has not been established till now though probably only three similar cases have so far been reported stating the incidence of recurrent hypokalemic paralysis in the presence of hypothyroidism in different clinical scenarios.

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