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A Typical Presentation of Hypothyroidism: Recurrent Hypokalemic Paralysis

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Abstract: ***Recurrent hypokalemic paralysis as a presentation of hypothyroidism is rare in the literature. This transient and episodic neurological condition is commonly associated with thyrotoxicosis. We report a case of young male admitted with recurrent paralytic attacks since last 2 year. He had no symptom of hypothyroidism. He 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 1.9 to 3.4 meq/L during attack with high serum creatine phosphokinase level. Electromyography was normal. Follow up shows satisfactory result with thyroxine replacement. It is an extremely rare and unusual presentation of hypothyroidism, and to our best knowledge, it is the seventh case report in the literature.***

Keywords: Hypokalemia, recurrent paralysis, hypothyroidism

1. 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 seventh reported case of hypothyroidism associated with recurrent hypokalemic paralysis.[1–3]

2. Case Report

A 22-year-old male presented with recurrent attacks of acute flaccid paralysis of all four limbs since last 2 year. He came in the neurology emergency for the above symptoms. His symptoms increased after high carbohydrate meal and each episode lasted for 2 to 5 hours followed by spontaneous complete recovery without potassium supplement in any form. Frequency of attack gradually increased up to four episodes per month. It started with early morning weakness after awakening without any diurnal variation. There was no history suggestive of fever, altered sensorium, convulsion, visual, respiratory, bulbar weakness or any other neurological symptoms. He 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. He had normal higher mental function without any cranial nerve, sensory, or sphincter involvement. He was thin built with no pallor or edema. His thyroid gland was not palpable. Other systemic examination was within normal limit.

Laboratory investigations showed Hb-12.4mg/dl, ESR-44, low potassium, and normal sodium and serum creatine phosphokinase (CPK) level was very high (1284 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 > 50). Serum anti-TPO antibodytiter was also very high (256.2 IU/ml). Hypokalemia persisted during attack ranging from 1.9 to 3.4meq/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 14.27meq/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 (2.7ng/ml/h).

During the acute period in hospital, the patient was treated with intravenous potassium (IV potassium chloride, 45meq/L of normal saline through peripheral vein at a rate of 20 meq/h) for three hours 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 he 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 6 months during follow up).

3. 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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References

[1] Bandhakavi M. Periodic paralysis as an unusual presentation of autoimmune hypothyroidism with goiter. *ClinPediatr (Phila)* 2009;48:677–8. [PubMed] [Google Scholar]
[2] Papa L, Lee P, Goldfeder B, Ferguson K, Luetke C. Hypokalemic periodic paralysis: An unexpected finding of hypothyroidism. *Am J Clin Med.* 2005;2:20–2. [Google Scholar]
[3] Kinoshita I, Usa T, Satoh A, Tsujihata M. A case of hypothyroidism associated with hypokalemic periodic paralysis. *RinshoShinkeigaku.* 1990;30:100–2. [PubMed] [Google Scholar]
[4] Soule BR, Simone NL. Hypokalemic periodic paralysis: A case report and review of the literature. *Cases J.* 2008;1:256–61. [PMC free article] [PubMed] [Google Scholar]
[5] Rose BD, Post TW. 5th ed. New York, United States of America: McGraw-Hill; 2001. Clinical physiology of acid-base and electrolyte disorders. [Google Scholar]
[6] Bagga A, Dutta S. Hypokalemic muscle paralysis. *Indian Pediatr.* 1994;31:1297–8. [PubMed] [Google Scholar]
[7] Srivastava RN, Bagga A. *Pediatric Nephrology.* 2nd ed. Delhi: Cambridge Press; 1997. Renal tubular disorder; pp. 203–7. [Google Scholar]
[8] Maurya PK, Kalita J, Misra UK. Spectrum of hypokalaemic periodic paralysis in a tertiary care centre in India. *Postgrad Med J.* 2010;86:692–5. [PubMed] [Google Scholar]
[9] Rao N, John M, Thomas N, Rajaratnam S, Seshadri MS. Aetiological, clinical and metabolic profile of hypokalaemic periodic paralysis in adults: A single-centre experience. *Natl Med J India.* 2006;19:246–50. [PubMed] [Google Scholar]