

Case Report

Hypokalaemic Periodic Paralysis Associated with Hypothyroidism

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Abstract

Periodic paralysis is also seen as one of the uncommon cause of quadriplegia in the physical and rehabilitation medicine practice.

There have been anecdotal reports of periodic paralysis associated with hypothyroidism. However, there is no clear cut evidence of hypothyroidism causing hypokalaemia leading to periodic paralysis or vice versa.

This case report highlights the importance of keeping periodic paralysis as an important cause of the recurrent paralytic attack, importance of serum potassium and thyroid function tests and recovery pattern without active physical therapy intervention which may actually aggravate the disease process. Its occurrence in a young male patient is also a rare phenomenon.

Key words : Hypothyroidism, Hypokalaemia, periodic paralysis.

Case Report:

A 21- year- old male was admitted in the Department of Physical Medicine and Rehabilitation, Regional Institute of Medical Sciences, Imphal because of recurrent paralytic attacks involving all the four limbs with two similar attacks in the past in which, the symptom resolved without any interventions. There were no clinical features of hypothyroidism. There was no goitre and his mental status was normal. Motor power was 2- 3/5 in all the key muscles of both upper and lower limbs without

any sensory deficit when examined within 24 hours of the attack. Serum creatine phosphokinase (CPK) was 306 U. Serum K⁺ was consistently low (2.7 and 3 mEq/L) in two consecutive readings with high TSH level (15 nmol/L) and thyroid related antibodies in the serum. Serum level of antithyroid peroxidase antibodies was 30.12 IU/mL (normal 0-18 IU/mL) and antithyroglobulin antibodies was 180.32 IU/ml (0-70 IU/mL). Urine pH and serum chloride was within normal limits. Nerve conduction velocities for both sensory and motor nerves were normal. He was treated with potassium and thyroxine supplements and was able to walk and perform his duties within 72 hours of the attack. Except for the range of motion exercises, no strenuous exercises were given to prevent aggravation of symptoms and to help in early recovery.

Discussion:

Hypokalaemic periodic paralysis is an episodic neuromuscular disorder and may be of primary or secondary origin. The paralysis may last from an hour to several days and weakness may be of localised or generalised with disorders including myopathy, wasting of muscles, reduced power and reflexes and may even involve the respiratory muscles^{1,2}.

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The primary hypokalaemic periodic paralysis is an inherited autosomal dominant and precipitated by strenuous work, high carbohydrate diet and cold², which was not found in our case. The secondary hypokalaemic periodic paralysis has been associated with diuretic abuse, gastroenteritis, renal tubular acidosis, hyperaldosteronism, Bartter syndrome and hyperthyroidism³. The possibility of renal tubular acidosis was excluded as he had normal urine pH and no hyperchloraemia.

Periodic paralysis has been known to be associated with thyroid disorder. But, it is mostly linked with the hyperthyroid state and its association with hypothyroidism is very rare and so far only a few cases have been reported. Clinical examination and investigations rule out the evidence of hyperthyroidism.

Chaudhury *et al*⁴ reported hypokalaemia leading to periodic paralysis in a child who was undergoing treatment for hypothyroidism. The explanation was that thyroxine in pharmacological doses can cause increased potassium excretion and water diuresis in patients with myxoedema during the initial part of the therapy⁵. This may result in hypokalaemia, especially in a child with severe malnutrition and low stores of total body potassium¹.

Hypokalaemic paralysis is a common manifestation of renal tubular acidosis due to inappropriately high potassium excretion leading to symptomatic hypokalaemia. Hypokalaemic weakness is one of the manifestations of distal renal tubular acidosis and it typically presents as hyporeflexive limb weakness, although involvement of the extraocular muscles, sphincters and cranial nerves has been reported⁶. Renal tubular acidosis is defined as an inability of the renal tubule to acidify urine, which is out of proportion to any reduction in the glomerular filtration rate. The acidification defect has been described to be mild and presumably related to thyroxine deficiency^{7,8}. An autoimmune mechanism has also been suggested to cause renal tubular acidosis in hypothyroid disorder^{9,10}. Koul Pand Wahid¹¹ supported association of an autoimmune cause that influences the renal acidification mechanisms through dysfunctions of various transporters and co transporters involved in the acidification in the renal tubular system. However, there has been report where renal tubular acidosis was associated with non-autoimmune hypothyroidism¹². Our patient didn't show any feature of renal tubular acidosis

There are controversies regarding hypothyroidism causing hypokalaemia or vice versa. Even in this reported case,

we were also unable to find out the cause and effect relationship. However, in one of case report on hypokalaemic myopathy accompanied by transient hypothyroidism in a patient with autoimmune thyroiditis, the authors suggested that this transient hypothyroidism might be induced by hypokalaemia during the course of autoimmune thyroiditis¹³.

This case report is unique because the association between hypothyroidism and hypokalaemic periodic paralysis occurred in a young male patient when all the case reports available in the literature showed occurrence only in female patients.

This case report highlights the importance of keeping periodic paralysis as an important cause of the recurrent paralytic attack presenting as tetraplegia, importance of serum potassium and thyroid function tests and recovery pattern without active physical therapy intervention which may actually aggravate the disease process.

By keeping this possibility over and above the detail history and proper clinical examination, we should be able to avoid costly radio-imaging techniques.

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