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Hypoparathyroidism Presenting as Recurring Muscle Aches and Grossly Elevated Serum Creatine Kinase

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Abstract:

Idiopathic hypoparathyroidism rarely manifests with myopathy. Serum calcium in patients with unexplained myalgia and/or muscle weakness must be measured to rule out hypoparathyroidism since the body being adapted to long-standing hypocalcaemia gives very few or non-specific symptoms and the diagnosis can be easily missed. We here present case of 36 year old Indian male with muscle spasms of bilateral upper limbs of seven days duration having past history of 4 years of distal paresthesias and recurring muscle aches and fatigability. On investigation the patient had an elevated serum creatine kinase. On further evaluation, the diagnosis of myopathy due to idiopathic hypoparathyroidism was made. He was treated with oral calcium and calcitriol and showed dramatic improvement. This case exemplifies the subtle nature of hypoparathyroid myopathy and highlights the importance of measuring serum calcium in patients with unexplained myalgia and/or muscle weakness.

Key words: Myalgia, Creatine Kinase, Hypocalcemia, Hypoparathyroidism, Muscle weakness, Spasm.

Introduction

Hypoparathyroidism is an uncommon endocrine deficiency disease characterized by low serum calcium levels, elevated serum phosphorus levels, and absent or inappropriately low levels of parathyroid hormone (PTH) in the circulation [1]. Hypocalcemia may be associated with an array of seemingly unconnected symptoms and signs. Symptoms are often determined by the degree of hypocalcemia and how quickly the calcium level drops. Tetany, muscle cramps, carpopedal spasm, seizures, and laryngospasm are associated with acute hypocalcemia. Patients with chronic hypocalcemia frequently have non-specific symptoms including fatigue, irritability, and anxiety. Other symptoms include dementia and cataract formation. Myopathy is a rare manifestation of hypoparathyroidism. Few cases of association of raised serum creatine kinase and severe hypocalcemia due to idiopathic hypoparathyroidism have been reported. The pathogenic mechanisms of myopathy and increased muscle enzymes in patients with hypoparathyroidism are not well established. This case highlights the uncommon nature of the diagnosis.

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Case Report

We report a case of a 36 year old Indian male presenting with muscle spasms of bilateral upper limbs of seven days duration. He had distal paresthesias and self-limiting episodes of muscular pain for 4 years and symptoms of easy fatigability and body ache. The patient did not complain of limb weakness, definite sensory loss, abnormalities of bladder or bowel function, respiratory distress, cranial nerve symptoms, seizures or fever. He did not have any surgeries in the past. He was vegetarian, non-alcoholic, non-diabetic and non-hypertensive. Family history was negative for thyroid disorders, vitiligo, diabetes mellitus, hypoparathyroidism, connective tissue diseases, or myopathy. There was no history of trauma, toxin exposure and neck irradiation therapy. Review of systems was negative for skin rash, photosensitivity, alopecia, mouth sores, sicca symptoms, Raynaud's phenomenon, pleurisy, prolonged morning stiffness, joint swelling, swallowing problems, or shortness of breath. There were no focal neurological signs. Deep tendon reflexes were normal; Chvostek's and Trousseau's signs were negative. The joint and skeletal examination was unremarkable and there was no significant abnormality on general examination. Hemoglobin, complete blood count, renal and liver function tests were within normal limits. Serum calcium was 6.1 mg/dL with albumin 3.69 g/dL and serum phosphate was 9.5 mg/dL. Creatine kinase (CK) level was raised to 6428 IU/L (Normal value 0-146 IU/L). Work up was directed towards the cause of hypocalcemia. The serum parathyroid hormone was inappropriately low i.e. <2.5 pg/mL. Serum magnesium and 25-hydroxy vitamin D levels were normal and there was no evidence of alkalosis on arterial blood gas analysis. Screening for secondary causes of hypoparathyroidism revealed a negative collagen profile and normal thyroid profile. Routine HIV status was done as per NACO protocol which was negative. Twenty four hour urinary copper and serum iron studies were normal. Conduction velocities in motor and sensory nerves were normal. EMG showed myopathic changes.

A diagnosis of primary hypoparathyroidism was made. In the absence of obvious etiologies such as surgical resection of the parathyroids, neck irradiation, infiltrative disorders such as hemochromatosis or Wilson's disease and considering the age of the patient which ruled out congenital causes, we considered it to be a case of hypoparathyroidism unrelated to systemic collagen disorder; this being the most common non-iatrogenic cause of parathyroid hypofunction. An ECG was obtained to look at the QT interval and nerve conduction studies were done. Both were nonconspicuous. Thyroid and parathyroid ultrasound did not add to our diagnosis. Chest X ray, abdominal ultrasound, computed tomography of the head performed as a part of malignancy screen was unremarkable.

He was treated with 600 mg calcium carbonate twice daily and 0.25 mcg of calcitriol daily. The weakness improved after therapy. Elevated CPK returned to almost normal level. At a follow-up visit, his serum calcium level was 7.5 mg/dL and creatine kinase level was 140 IU/L.

Discussion

Primary hypoparathyroidism is associated with a variety of symptoms due to hypocalcemia. Hypocalcemia leads to hyperexcitability at the neuromuscular junction, which may result in tetany, muscle cramping, carpopedal spasm, laryngospasm, and seizures but muscular involvement per se is relatively rare [2]. Cataracts, hair loss and dental abnormalities, emotional liability, psychosis, cognitive slowing, mental retardation, dementia, symmetric basal ganglia calcifications and acroparesthesia are also seen.

In a systemic search of the literature we retrieved 15 relevant reports from 1972 to 2009, highlighting this association. Average age was 36.7 years and most of them presented with vague complaints like anorexia, lethargy, muscle pain, muscle weakness, carpopedal spasms and a few of them had episodes of seizures. These studies suggested that the elevation in creatine kinase is the result of repetitive tetany or muscle spasm, resulting in leakage of creatine kinase from damaged muscle cells [3]. It has been postulated that patients with idiopathic hypoparathyroidism who develop myopathy with elevated creatine kinase probably remain minimally symptomatic due to the slow development of the hypocalcemia and the remarkable ability of the body to adapt to chronically low serum calcium levels [4]. This explains the absence of classical signs of hypocalcaemia such as negative Chvostek's sign and normal QT interval, in our patient.

Pseudohypoparathyroidism and rhabdomyolysis have also been associated with myopathy and increased creatine kinase values [2]. Our patient did not fit into either diagnosis as serum PTH value was very low. Myopathy is usually seen in hypocalcaemia associated with osteomalacia [5] but the serum creatine kinase is normal. We hypothesize that either increased permeability of the muscle membrane induced by hypocalcaemia or repeated muscle spasms resulted in leakage of creatine kinase and elevated serum levels [6].

Therefore, failure to recognize the presentation of hypoparathyroidism leads to delay and even errors in diagnosis and treatment. We do not suggest hypoparathyroidism to be kept at the forefront of the differential diagnosis of raised creatine kinase. Rather we present the case as an example of unusual manifestation of hypoparathyroidism.

Conclusion

Primary hypoparathyroidism leading to hypocalcemia may present with vague symptoms which often makes diagnosis difficult. Myopathy as a presentation of this disorder is still rarer and clinicians need to keep a high index of suspicion to avoid missing the diagnosis. In musculoskeletal disorders where a clear etiology is not to be found, it is worthwhile measuring the serum calcium, phosphate and magnesium levels and if necessary, following it up with a parathyroid hormone assay if initial investigations point that way.

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