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CASE STUDY

HYPOPARATHYROIDISM PRESENTING AS RECURRING MUSCLE ACHES AND GROSSLY ELEVATED SERUM CREATINE KINASE

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ABSTRACT

High serum CK levels are found in many muscular dystrophies as well as inflammatory, toxic, metabolic, and mitochondrial myopathies. The highest values are seen during attacks of myoglobinuria ("rhabdomyolysis"), a clinical syndrome of diffuse myalgia, myoglobinuria, and, often, renal failure. Idiopathic hyper CKemia indicates persistently elevated CK with no symptoms or minimal symptoms, such as muscle aches at rest or with exercise, muscle cramps, or stiffness. On examination, there should be no weakness, wasting, loss or increase of tendon reflexes, or fasciculations. We here present a case of 31 year old Indian male presented to us with complaints of muscle aches, easy fatiguability numbness in all 4 limbs for last four years. On investigation the patient had an elevated serum creatine kinase level but on evaluation no myopathy, weakness, wasting, loss or increase of tendon reflexes, or fasciculations were present. Patient's PTH level was low and diagnosis of idiopathic hypoparathyriodism with Hyper CKemia without Myopathy was kept.

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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. 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.

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Serum CK activity is commonly, but not always, elevated in patients with skeletal muscle disease. It also important to recognize that hyper CKemia can occur in those without a primary disorder of muscle.

CASE REPORT

A 31 year old male presented to outpatient department of M.B. Hospital Udaipur with complaints of muscle aches, easy fatiguability and occasional cramps for last 4 years. On general physical examination there were no abnormality present. Neurological examination revealed no muscle weakness, reflexs were present, plantars were flexors bilaterally and no sensory deficit were there. On investigation patient's calcium level was low and there were elevation in CK level. Patient undergone muscle biopsy and ENMG which revealed no changes regarding myopathy. Subsequently patient's PTH level came subnormal. Vit D level was within normal limit. Diagnosis of idiopathic hypoparathyroidism with Hyper CKemia was kept and patient started on oral calcium 500 mg bd. After 2 week patient came for follow up with significant improvement in muscle pain, CK level was also in decreasing trend.

CASE 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 (Glueck *et al*., 2009). 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 (Nardin *et al*., 2009). 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 (Brewster *et al*., 2007). 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 increasedcreatine kinase values (Glueck *et al*., 2009). Our patient did not fit into either diagnosis as serum PTH value was very low. Myopathy is usually seen in hypocalcaemia associated with osteomalacia (Bilezikian *et al*., 2009) 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 (Nora *et al*., 2004) 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 rare and even some time it can cause elevated CK with mild symptoms without any myopathy too.

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