Brief Communication

An unusual case of episodic quadriparesis

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ABSTRACT

The natural history of untreated asymptomatic primary hyperparathyroidism (PHPT) remains incompletely understood. Increased level of parathyroid hormone produces the characteristic biochemical phenotype of hypercalcemia, hypophosphatemia and the various clinical sequelae of chronic hypercalcemia. Periodic paralysis (PP) is a group of disorders of different etiologies with episodic, short-lived and hyporeflexic skeletal muscle weakness, with or without myotonia, but without sensory deficit and without loss of consciousness. However, PHPT has rare association with episodic quadriparesis mimicking as PP.

Key words: Hypercalcemia, periodic paralysis, primary hyperparathyroidism, quadriparesis

Introduction

Patients with primary hyperparathyroidism (PHPT) may seem to experience progression of the disease as measured by extreme elevations of serum or urinary calcium or by the advent of renal dysfunction, nephrocalcinosis or worsening osteopenia.[1] However, neuromuscular weakness remains an infrequent association, which is often ignored due to non-specific nature of complaints.[1,2] The neurological manifestations of hyperparathyroidism includes easy fatigability, weakness and amyotrophy of proximal muscles with preserved reflexes in most cases.[1,2] We report an unfamiliar case of PHPT with recurrent quadriparesis mirroring as periodic paralysis (PP).

Case Report

A 29-year-old male, presented with the complaint of sudden onset weakness of all four limbs since last night. History was negative for unusually high carbohydrate intake, excessive exertion or alcohol intake in the previous night. He also denied symptoms of unexplained sweating, tremors, heat intolerance, prolonged vomiting or any drug intake over a prolonged period of time. He had a preceding history of muscle cramps and paresthesia for about a month. Past history dates back in the year 2002, which revealed that the patient had similar mode of presentation. Subsequently, he had been admitted in a local hospital and improved symptomatically with conservative therapy. This episode repeated 1 year back and he was treated in the similar way. However, the details of further treatment history could not be gathered. Neurological examination revealed bilaterally symmetrical weakness (Gr 2/5) of all four limbs and generalized hyporeflexia with bilaterally flexor plantar response. Eyes, face, tongue, pharynx, larynx, diaphragm and sphincters were not involved. Higher functions, cranial nerves and sensory system were intact.

The present investigations revealed serum potassium: 4.3 mEq/L (reference range: 3.5-5), corrected calcium: 13 mg/dl (8.5-10.5), phosphate: 2.6 mg/dl (3-5) and alkaline phosphatase: 216 U/L (80-306). All other electrolytes were within the normal limit. Routine blood examinations, complete hemogram, creatinine, thyroid function tests, serum creatine phosphokinase, serologic viral markers and serum protein electrophoresis did not reveal any abnormality. Further evaluation of muscular weakness by electromyography was normal. Nerve conduction velocity study was also normal. However on examination of the

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abdomen by ultrasonography, multiple renal calculi were found in both kidneys. X-ray abdomen reassured the evidence of bilateral nephrolithiasis. On further plan, serum immunoassay for parathyroid hormone value was found to be 147 pg/ml (reference range: 10-69) and 25-hydroxy vitamin D value was 25 ng/dl (reference range: 11.1-42.9). T score at wrists had evidence of osteopenia (~1.0). His serum calcitonin was normal (7.4 pg/ml) (reference range: <5) and 24 h urinary calcium excretion reported as high (396 mg) (reference range <300). Though, Technetium 99m (99m TC) sestamibi scan [Figure 1] could not confirm the possible etiology but ultrasound and computed tomography (CT) scan of neck the evidence of bilaterally enlarged parathyroids [Figures 2 and 3]. He was treated with volume correction and supportive measures. He improved dramatically again within 24 h and with the diagnosis of PHPT, he has been planned for surgery.

Discussion

Episodic weakness beginning before age 25 may be due to primary PP.[3] PP though common among Indian population varies greatly in disease spectrum and magnitude due to the heterogeneous pattern of etiology behind it. PP may be primary or secondary type.[3] The primary hypokalemic PP is autosomal dominant and is exacerbated by strenuous exercise, high carbohydrate diet, cold and excitement, which was not found in this case. 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.[3] Thyrotoxicosis had been ruled out in this case. Though molecular genetic studies were not done, Gitelman's syndrome, Bartters syndrome and channelopathies were also ruled out by the absence of typical clinical features, hypokalemia and hypomagnesemia.

Most of the symptoms and complications of PHPT result from hypercalcemia.[2] The neuromuscular manifestations include progressive weakness of limbs, fatigue, exhaustion, dyskinesia, abnormal gait and muscle atrophy. Some patients present with paresthesias and muscle cramps, loss of vibratory sensation with diminished reflexes and stocking-glove loss-of-pain sensation.[2] Patten and pages had reported two cases of hyperplasia and neuromuscular symptoms, but the electrophysiological findings were compatible with myopathy.[4] Verges *et al.* also described a patient with proximal weakness and quadriceps atrophy associated with parathyroid adenoma.[5]

The pathophysiology of neuromuscular manifestations in PHPT is not yet clear. The defect seems to be functional as the neurological signs disappear after surgery or clinical treatment. In most cases, there are three biochemical abnormalities: Increased circulating PTH, hypophosphatemia and hypercalcemia.[5] However, the possible explanations of neurological manifestations

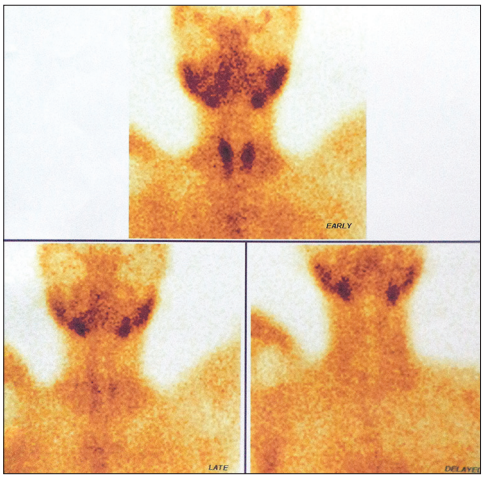

Figure 1: 99mTc sestamibi scan findings were consistent with the absence of hyperplastic parathyroid activity

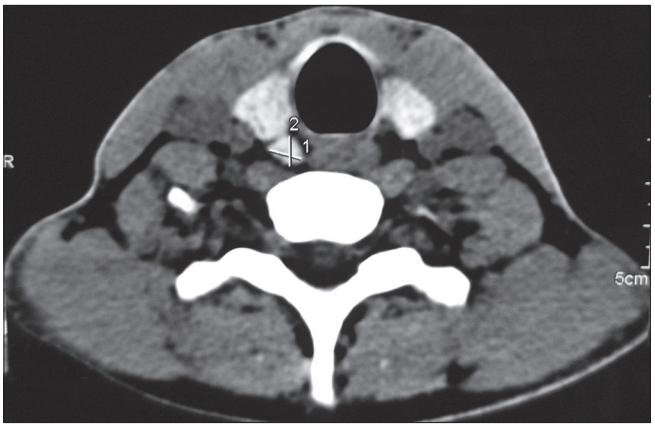

Figure 2: Computed tomography neck showing enlarged the right parathyroid gland (0.77 cm × 0.71 cm × 1.4 cm)

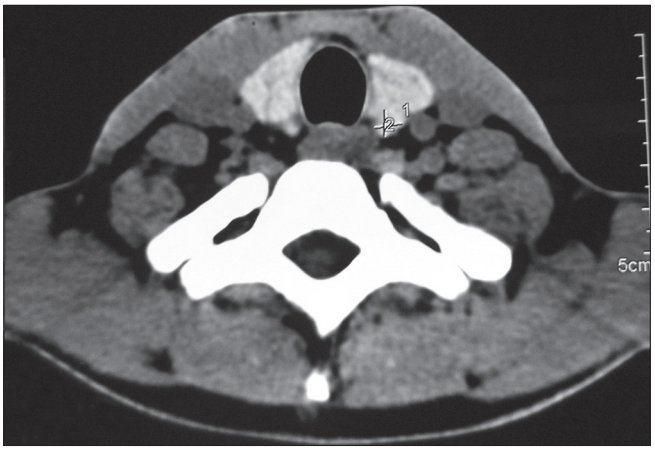

Figure 3: Computed tomography neck showing enlarged left parathyroid gland (0.65 cm × 0.63 cm × 1.5 cm)

include: (1) neuromuscular junction conduction disturbances related to hypercalcemia; (2) abnormal trans-membrane calcium transport as a result of excessive extracellular calcium and (3) neuromuscular conduction disturbances related to hypophosphatemia.[5] A direct effect of PTH on neural tissue also has been reported as the imaginable cause.[6]

The pre-operative localization procedures include: Diagnostic ultrasound of the neck (sensitivity 55%); CT of neck (sensitivity 68%); magnetic resonance imaging of neck (sensitivity 75%); and radionuclide imaging and single-photon emission computed tomography (sensitivity 90%).[7] Due to superior image quality and improved accuracy, MIBI has also been used prevalently in parathyroid imaging.[7] However, not all parathyroid lesions retain MIBI and abnormal parathyroid tissue becomes more visible on the delayed images.[8] Sensitivity of sestamibi scanning is poor (<50%) in the presence of multiglandular disease (hyperplasia or double adenomas).[8]

Our patient presenting as recurrent episodic quadriparesis was subsequently diagnosed as PHPT after evaluation for all possible causes. To the best of our knowledge, recurrent episodic paralysis is an extremely unusual presentation of PHPT. To conclude, it would be wise to consider the PHPT as one of the possible etiologies of episodic quadriparesis simulating as PP.

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