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Case Report

An Atypical Case of Hyperthyroidism Presenting as Hypokalemic Periodic Paralysis: Diagnostic and Therapeutic Dilemmas

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Abstract: Hypokalemic periodic paralysis (HPP) due to hyperthyroidism is a rare clinical condition. It is more commonly seen among Asian males as compared to its global incidence. We are reporting a rare atypical case of HPP which presented in our Medicine outpatient department (OPD) with complaint of repeated episodes of weakness in all of the four limbs. However, at the time of presentation, the patient did not complain of any weakness but investigation profile revealed a low potassium level which was done during the previous episode. On the basis of all routine and specific investigations including EMG-NCV, muscle biopsy and thyroid profile as well as clinical examination, the patient was diagnosed as a case of hyperthyroidism and was managed with oral anti thyroid medication. Considering the diagnostic difficulties in such patients especially when such patients present with atypical clinical presentation, it is important to rule out hyperthyroidism specifically in patients presenting with acute onset of weakness in all the four limbs.
Keywords: Hypokalemia, Hyperthyroidism, Periodic paralysis

INTRODUCTION

Hyperthyroidism is a clinical syndrome that results from exposure of the body tissue to excess of circulatory free thyroid hormones. Generally, all tissues that contain thyroid receptors are affected by this clinical entity. Mild hyperthyroidism usually presents as anxiety neurosis and hence diagnosis is difficult to arrive at until proven otherwise by biochemical abnormalities. However, moderate to severe hyperthyroidism produces typical features characterized by accelerated basal metabolic rate resulting in heat intolerance, weight loss in spite of good appetite, vomiting, diarrhoea and steatorrhoea. Further, stimulation of sympathetic nervous system leads to high resting pulse rate, sinus tachycardia, arrhythmias, nervousness and so on. But a patient of hyperthyroidism presenting as hypokalemic periodic paralysis is a rare clinical phenomenon.

Here we are reporting a case of hyperthyroidism presenting as hypokalemic periodic paralysis which is an uncommon presentation.

CASE REPORT

A 38 years old male reported in Medicine OPD of our institute with complaint of repeated attacks of weakness in all four limbs since the last two years. On elicitation of history, it was revealed by the patient that he develops sudden weakness of all limbs and thereafter it is difficult for him to stand or walk without support. These episodes occur mostly in the midnight especially if the patient had physical exertion during the day. Patient reported 4 to 5 number of such episodes during the last two years which prompted his admission to local hospital. During each episode, management usually comprised of administration of IV fluids which used to provide relief within few hours. The last episode occurred a few months back which was managed as described. General physical examination was unremarkable except for fine tremors in hands. All the vital signs were within normal limits. On CNS exam, the higher mental functions, cranial nerves, motor system and sensory system were within normal limits with no cerebellar signs and no sign of meningeal irritation. Rest of the systemic examination was also unremarkable

Investigations

Revealed a normal haemogram, liver function tests, and renal function tests; RBS-108mg/dl, S uric acid-5.2mg/dl, S Ca-9.0mg/dl, Phosphorus-2.4mg/dl and Sodium-138meq/L. S Potassium was 2.2meq/L (3.5-4.5) at the time of weakness in limbs and 3.5meq/L on presentation to our institute. His total T3 was-449ng/dl (60-200), total T4> 30.0mcg/dl (4.5-12), and TSH<0.01microIU/ml (0.3-5.5). Intact parathyroid was 34.18pg/ml (15-65); CT head, NCV-EMG and muscle biopsy were all normal.

The patient was put on anti-thyroid drugs & supportive treatment and he responded well.

DISCUSSION

Hyperthyroidism as such is a very common entity but its presentation as HPP is most unusual. The first case of non specific periodic paralysis was described in 1882 [1]. Association of hypokalemic periodic paralysis with hyperthyroidism was later described by Jackman and Jones [2]. Hyperthyroid patients usually present hyperactivity, irritability, dysphoria, heat intolerance, sweating, palpitations, fatigue, weakness, weight loss with increased appetite, diarrhoea, tachycardia, atrial fibrillation in the elderly, tremor, goiter, warm, moist skin, oligomenorrhea and loss of libido [3].

Hypokalemic periodic paralysis in hyperthyroid patient is an atypical presentation [4]. It occurs in about 0.1 to 0.2% of the hyperthyroid population in North America, and it is ten times more common in the Oriental population [5]. The disease is more common in males than females because of decreased penetrance in females [7]. The present case also supports this fact of male preponderance. Hypokalemic periodic paralysis is caused by mutation in either of two genes, type 1, the most common form, is inherited as an autosomal dominant disorder with incomplete penetrance. These patients have mutations in voltage-sensitive, skeletal muscle calcium gene, CALCL1A3. Approximately 10% of cases of hypokalemic periodic paralysis type 2, arise from mutations in the voltage-sensitive sodium channel gene (SCN4A). In either instance, the mutations lead to an abnormal gating pore current that predisposes the muscle cell to depolarize when potassium level are low. It is also now recognized that some cases of thyrotoxic hypokalemic periodic paralysis are caused by genetic variant in a potassium channel (KCNJ18 (Kir 2.6); Chromosome 17p11.2; dominant or sporadic), whose expression is regulated by thyroid hormone [7]. Most commonly proximal muscle weakness is observed. Most commonly, the disease involves legs more than arms; proximal more than distal but may selectively involve recently exercised muscles; it may be asymmetric. In severe attacks it may involve respiration and bulbar function but sphincters are spared. Attacks usually occur in random pattern without obvious stimulus and are precipitated by high carbohydrate load & heavy meals, muscle cooling, rest after exercise and thyroxine ingestion. There may be seasonal variation, more common in May-October (summer) and less common in December-March. These attacks may be aborted by exercise and abate when thyrotoxicosis resolves. Thyrotoxicosis may be subclinical, and a positive family history is common [6].

Serum K+ is usually low (< 2.5 μmol/L), but may be severely reduced and occasionally normal, especially if drawn after the attack, and the same findings were observed in our case. TSH is low with hypophosphatemia**.** Electro diagnostic study shows reduced CMAP during attacks and epinephrine has no effect on size of CMAP. EMG shows myopathic features during attacks and muscle pathology reveals vacuolar dilation of sarcoplasmic reticulum during attacks , tubular aggregates, and increased muscle Na+-K+ pumps [7]. Our patient had normal EMG and muscle biopsy as they were done when the attack had already aborted.

Acute paralysis improves with administration of potassium. Oral potassium (0.2-0.4 mmol/kg) should be given every 30 mins. Only rarely is IV therapy necessary (when swallowing problems or vomiting is present). Patient is made aware of importance of low carbohydrate, low sodium diet and consequences of intense exercise. Prophylactic administration of acetazolamide (125-1000mg/day in divided doses) reduces or may abolish attacks. If attacks persist on acetazolamide, oral k+ should be added. Some patients require triamterine (25-100mg/d) or spironolactone (25-100 mg/d). However type 2 hypokalemic periodic paralysis attacks get exabrated with acetazolamide [7].

Treatment of hyperthyroidism is mainly done with antithyroid drugs and central to the management of TPP (Thyrotoxic periodic paralysis). The present case was also put on anti-thyroid drugs to which he responded well. Propranolol added to the initial treatment counteracts the peripheral effects of thyrotoxicosis and improves muscle strength. Long term treatment of TPP entails control of hyperthyroidism. Euthyroidism must be maintained for at least six months. Symptoms recur with poor control. Iodine ablation and surgical management with subtotal thyroidectomy are curative [8].

CONCLUSION

Hyperthyroidism is an uncommon but treatable cause of hypokalemic periodic paralysis. This case highlights the importance of entertaining this important possibility in these patients for an early and effective management.

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