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THYROTOXIC PERIODIC PARALYSIS

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ABSTRACT

Thyrotoxic periodic paralysis (TPP), a disorder commonly seen in Asian men, mainly appears in case of hyperthyroidism and is characterised by abrupt onset of Amyotonia associated with hyokalemia . this condition primarily affects the lower extremities and is secondary to thyrotoxicosis. Early recognition of TPP is vital to initiate appropriate treatment and to avoid the risk of life threatening cardiac arrhythmia due to rebound hyperkalemia that may occurs if high dose potassium replacement is given[1]. Once recognised, therapeutic approach and treatment is simple and the prognosis is excellent.

Here we report a case of 30 year old male with thyrotoxic periodic paralysis from Hatia-Ranchi presented to RIMS central emergency with weakness in all four limbs since one day, diagnostic and therapeutic approach.

KEYWORDS :

INTRODUCTION:

The thyrotoxicosis is the state of hyper secretion of the thyroid hormone inside the peripheral blood and tissue it usually occurs in case of hyperthyroidism like Grave's disease. The thyrotoxicosis usually shows symptoms like weight loss, heat intolerance, hyperhidrosis, tachycardia, palpitation etc. And some others are shown like hypotonia, fatigue, hyposthenia as the musculoskeletal symptoms of thyrotoxicosis[2]. The thyrotoxic periodic paralysis is most common in Asian population especially in men usually between 20-40 years of age with an incidence of 2% among thyrotocosis of any cause[3]; Symptoms of TPP commonly occurs after sever exercise , high carbohydrate diet or while resting after heavy drinking[4]. Thyrotoxic periodic paralysis is characterised by acute onset of sever hypokalemia and profound proximal muscle weakness more marked in lower limbs among patients with thyrotoxicosis[5], which spontaneously recovered after continuing for several hours or several days. This TPP usually disappears with the improvements of the disease which causes the hyperthyroidism[4]. Thyrotoxic periodic paralysis commonly misdiagnosed in western countries because of its similarities to Familial hypokalemic periodic paralysis; it is autosomal dominant disorder caused by a defect in the gene coding for L-type calcium channel 1-subunit (CACNA1S) on chromosome Tq31-32. The neuromuscular presentation of both are identical and to enhance diagnosis of thyrotoxic periodic paralysis. Physician need to look for subtle features of hyperthyoroidism in the presence of hypokalemic periodic paralysis[6].

Early diagnosis not only helps in definitive management with non-selective beta blockers and correction of hyperthyroidism. But also prevents the risk of rebound hyperkalemia due to excessive potassium supplementation.

CASE REPORT :

A 30 year old male patient resident of Hatia-Ranchi came to RIMS central emergency with history of weakness in all his four limbs since one day, initially he noticed the weakness in bilateral lower limbs while going for bed after taking food then gradually over one to three hours weakness had progressed to upper limbs as well. He had similar two to three episode previously, for that he took potassium supplements and symptom was relieved. He reported intermittent loose stools, palpitations and had no similar complaints in his family. He denied use of diuretics, laxatives, alcohol and recreational drugs.

On examination patient had pulse irregularly irregular of 112 beats/min, respiratory rate was 18 cycles/min, Bp-136/86 mmHg, temperature-37.6^{0}C and weakness in all four limbs with decreased muscle power 1/5 in lower bilateral limbs; 2/5 in bilateral upper limbs and decreased tendon reflexes in all four limbs. He had no thyroid swelling, exophthalmoses or sensory or cranial nerves deficit.

Respiratory system, abdominal examination was unremarkable. Electrolytes values on admission were Na^+-141 mmol/L (normal reference range- 135-145 mmol/L); K^+-2.5 mmol/L (normal reference range-3.-5.0 mmol/L); $Mg2^+$-1.3 mmol/L (normal reference range-1.7-2.6 mg/dl) and Ca^{2+}-2.4 mmol/L (normal reference range-2.2 to 2.7mmol/L) and normal acid-base status. His ECG at the time of admission showed Atrial fibrillation with a ventricular rate of 120 beats/min. Initial diagnosis of hypokalemic periodic paralysis was made and patient commenced on intravenous potassium supplementation 10mEq/hr for total of 80mEq over 8 hours. During which there was marked improvement of muscle power with the complete recovery of muscle strength on next day. On following day, his CPK enzyme levels done and it was 92 U/L (reference range-51-294 U/L) , thyroid function test revealed a serum TSH of <0.006 micro IU/L (normal reference range- 0.35-5.50 micro IU/L) and T4 level of 9.48 ng/dl (normal reference range- 0.89- 1.76 ng/dl). Patient was further evaluated for the cause of thyrotoxicosis and was found to have high titres of Anti thyroid peroxidise antibody (Anti TPO antibody) of >1300.0/mL (normal reference range <60.00 U/mL). Finally he was commenced on carbimazole 10mg thrice daily and propranolol 40 mg twice daily. There was no recurrence of symptoms after discharge and patient's serial serum potassium levels continued to remain normal.

DISCUSSION:

The thyrotoxicosis is defined as the state of hyper secretion of thyroid hormone. This meaning is not the same as hyperthyroidism which the thyroid function is excessively increased but most causes of thyrotoxicosis occur by hyperthyroidism such as Grave's disease or Toxic multinodular goiter[2] . When the secretion of thyroid hormone is promoted by Graves' disease, goiter, thyroid cancer, or

thyroiditis, due to the excessive action of thyroid hormone, the clinical symptoms are manifested by the action of excessive amounts of thyroid hormone inside the peripheral blood and tissue as well as heat intolerance, and thus it is called thyrotoxicosis[7]. The thyrotoxicosis causes unaccountable weight loss in spite of increased appetite, hyperactivity, nervousness, quick temper, and the sense of fatigue and the neurologic symptoms like the excessive reflex, muscle consumption, proximal muscle illness without accompanying contraction manifested. As for the musculoskeletal symptoms caused by thyrotoxicosis, the sense of fatigue or weakness mostly due to the attenuation of muscular force by the atrophy of appendicular muscle is complained about[8].Hypokalemic periodic paralysis has been associated with thyrotoxicosis since 1902[2]. Although muscular involvement related to thyrotoxic such as thyrotoxic myopathy, opthalmoplagia and myasthenia gravis are well known, periodic paralysis has received little attension[1]. TPP is not related to the etiology, severity, and duration of thyrotoxicosis[5]. Family history of periodic paralysis is usually absent.The hallmark of the condition is acute systemic weakness associated with low serum potassium and biochemical thyrotoxicosis with or without clinical features of hyperthyroidism. The thyroxin excess need not be secondary to Grave's disease as cases of TPP has been reported with toxin nodular with toxic nodular goiter, solitary nodule and with exogenous thyroxin intake used to reduce body weight[9]. The pathogenesis of thyrotoxic periodic paralysis has been thought related to increased Na^+-K^+ ATPase activity stimulated by thyroid hormone and/or hyperadrenergic activity and hyperinsulinemia. This mechanism alone, however, associated paradoxical depolarization of the resting membrane potential. Recent findings that loss of function mutations of the skeletal muscle-specific inwarding rectifying K^+(Kir) channel, Kir2.6, associate with thyrotoxic periodic paralysis provide new insights into how reduced outward K^+ efflux in skeletal muscle, from either channel mutations or inhibition by hormones (adrenaline or insulin), can lead to a vicious cycle of hypokalemia and paradoxical depolarization[10]. The estrogen and progesterone inhibit the ATPase pump these findings partially explain the predominance of males incidence[11] in addition, thyroxin increases tissue responsiveness to beta adrenergic stimulation, also linked to increase in Na+-K+ ATPase activity. The use of a non- selective beta blocker such as propranolol helps to abort the acute attack and prevent further relapses[12]. Episodes of paralysis occur during the night in more than 80% on patients with TPP .it has been shown that plasma glucose and insuline responses to meals are markedly higher in the evening than in the control subjects. Such a phenomenon suggests the possible mechanism for nocturnal preponderance of TPP.The cornerstone of treatment of the paralytic attack is potassium supplementation either by careful controlled parental infusion or by oral administration of potassium chloride. Careful monitoring to avoid hyperkalemia is essential as cases of rebound hyperkalemia have been described. This occurs because of the release of the potassium from intracellular compartment as the paralysis improves and the muscles start to contract. Propranolol has been shown to reduce the relapse rate and is useful in the treatment of the acute episode[13]. As the acute episode subsides attention should be directed toward the original problem which is thyrotoxicosis.

CONCLUSION:

A young healthy-looking man with unexpected generalised paralysis and an intact sensorium might be considered hysterical by an unwary physician in a crowded emergency department. This would be especially true if the patient lacked any thyrotoxic symptoms or signs (which is not uncommon in TPP). When a young male of Asian descent is initially seen with severe lower extremity weakness or paralysis, TPP should be considered as the most likely diagnosis until proven otherwise. This is important because TPP is curable. Potassium administration during the attack will improve the weakness dramatically and the use of propranolol and potassium supplements will prevent the attacks. The definite treatment is achieving euthyroid state.

REFERENCES:

1. Davidson ET, Davidson MJ. Tri-iodothyronine Toxicosis with Hypokalemic Periodic Paralysis and Ventricular Tachycardia. J. Electrocardiography 1995; 28 (2) 161 -163.
2. E. Braunwald etc. HARRISON'S PRINCIPLES of Internal Medicine 19th Edn.. Seoul: Jungdam Publisher Co.. 2003:(2125-2140)
3. Ko GT, Chow CC, Yeung VT, Chan JK, cockram CS. Thyrotoxic Periodic Paralysis in the Chines population. Q.J. med. 1996; 89; 463-468.
4. Min HK etc. Endocrinology. Seoul: Medical Publishing Co. Ltd..1994:(290-292)
5. McFadzean AJ, yeung R. Periodic paralysis complicating thyrotoxicosisin Chinese.BMJ 1967;451-455.
6. JAPI page-98.Thyrotoxic Channelopaties by Pankaj singhai, Shruti Krishnan, Vikram Uttam Patil Manipal Hospitals,Banglore, Karnataka.
7. Ahn SY. The Thyroid Clinic. Seoul: Sungbo publishing Co.. 2004:(90-131)
8. Song YG etc. The Study of Thyroid Gland 2nd Edition.Seoul: Korea Medical Book Publisher Co.. 1995:(131-188).
9. THYROTOXIC HYPOKALEMIC PERIODIC PARALYSIS By Al Khaledy E.H., Ashour N. & Al mutairi S. Medical Department ,Amiri Hospital Riqqa, Kuwait
10. Ryan DP, da Silva MR, Soong TW, Fontaine B, Donaldson MR, Kung AW, Jongjaroenprasert W, Liang MC, Khoo DH, Cheah JS, Ho SC, Bernstein HS, Maciel RM, Brown RH Jr, Ptacek LJ: Mutations in potassium channel Kir2.6 cause susceptibility to thyrotoxic hypokalemic periodic paralysis. Cell2010; 140: 88-98, CrossRefMedlineGoogle Scholar.
11. Van Dam GM, Resiman Y, vn Weiringen K. Hypokalemic thyrotoxic periodic paralysis: case report and review of an Oriental syndrome. Netherlands J.Med. 1996; 49 (2) : 90-97.
12. Szeto CC, Chow CC, Li KY, Ko TC, Young VT. Thyrotoxicosis and renal tubular acidosis presenting as hypokalemic paralysis. Br.J.Rheumatology 1996, 35: 289-291.
13. Kelly DE, Gharib H, Kennedy F, Duda R, McManis M. Thyrotoxic periodic Paralysis. Arch. Intern.Med.1989; 149: 2597-2600.