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CASE REPORT

Periodic Paralysis in thyrotoxicosis

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Abstract

Thyrotoxic periodic paralysis (TPP) is one of the rare manifestation thyrotoxicosis. In this case report, we present a 36-year-old man, who had lower limb weakness for one day on waking up.He has no family history of thyroid or neurological problems.Any cause of thyrotoxicosis maybe sufficient to trigger attacks in susceptible patients. In order to permanently repair the problem, the underlying hyperthyroid status must be rectified.

1. Introduction:-

Thyrotoxic periodic paralysis (TPP) is one of the most common cause of acquired hypokalemic periodic paralysis. This condition is often momentary but potentially serious. It is typically associated with thyrotoxicosis and hypokalemia. Early recognition of the problem allows for appropriate immediate- and long-term management of this condition.

2. Case Report

A 36-year-old gentleman presented with sudden onset of bilateral lower limb weakness. He also complained of palpitations since the last 15 days. The symptoms were not associated with tremors ,heat intolerance or increased bowel movements ,insomnia or irritability.He had history of similar episode 3 weeks back that had recovered on its own.Patient gives history of high carbohydrate diet during this episode & also during prior episode.There was no similar history of thyroid disorder or neurological illness in the family.

On clinical examination the patient was conscious well oriented to time, place and person,his vitals were PR-120 BPM BP -130/80 mmhg of mercury on general examinations there were no tremors, no exophthalmous,no thyroid acropachy. On the neurological examination patient was conscious ,alert , his speech ,language was normal, his cranial nerve examination was unremarkable .On motor examination his bilateral upper limb power was 5/5 and bilateral lower limb power was 0/5 ,DTR were normal in upper limb and decreased in lower limbs . Sensory System examination was normal, other system examination including Cardiovascular, pulmonary, and abdominal were withinin normal limits.. Patient serum potassium levels on admission was 2.0 mEq/L, free thyroxine (T4) level 24.9ug/dl(4.4-11.8), free triiodothyronine (T3) level 2.6ng/ml(0.6-2.4), and TSH<0.01mU/L (0.35–5.50). ECG showed normal sinus tachycardia only.

And his lower limb weakness and serum potassium (4.1 mEq/l)improved rapidly after iv correction with 60 mmol potassium, and oral potassium supplement were given for next 3 days,and he was started on carbimazole 10mg TDS ,propranolol 40mg BD so as to form rapid euthyroid state and USG was done which showed features suggestive of

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thyroiditis and FNAC was performed which showed diffuse toxic goitre ,on subsequent follow up patient was asymptomatic and at present patient is on carbimazole 10mgBD and propranolol 40 mg od without any recurrent paralysis. Patient is currently in the remission phase & didn't require any further radioactive iodine treatment .

3. Discussion:-

Thyrotoxic periodic paralysis (TPP) is one of the unusual manifestations of thyrotoxicosis. TPP is a condition manifested by muscle weakness associated with fluctuations in potassium levels. TPP can occur either due to transient shifts (Hypokalaemic periodic paralysis or HPP) or reduction in total potassium levels (non hypokalaemic periodic paralysis) [1].

Familial cases are genetically inherited as an autosomal dominant manner. Mutations in CACNA1S and SCN4A genes adversely affect the function of calcium and sodium ion channels servicing muscle cells, respectively [2]. The KCNJ2 gene codes for inward rectifying potassium channels (Kir 2.1) that moves potassium ions into the cells of skeletal and cardiac muscles. Mutations of this gene have been known to cause familial periodic paralysis with arrhythmias and Andersen-Tawil syndrome [3, 4].

Young Asian men are more likely to be affected with a prevalence of around 2% of all thyrotoxic cases compared to 0.2% in thyrotoxic Caucasian patients [5]. Yet this condition can affect individuals from different parts of the world [6, 7].

This condition can manifest as a medical emergency. Patients often have an sudden onset of weakness which can affect all four limbs which is classically associated with hypokalaemia. Patients with suspected cases of TPP with normal potassium levels during an attack have been reported but it is worth noting that techniques for taking blood may lead to falsely higher levels of potassium [8]. The typical presentation is of ascending lower limb paralysis in the early hours of the morning, or after rest following heavy exercise or a high carbohydrate meal, leaving the patient unable to move. Acute episodes may have prior symptomsof myalgia, cramps, or stiffness of muscle. It is infrequent to observe ocular, bulbar, or respiratory muscle involvement. Tendon reflexes are generally decreased or absent with intact sensation and consciousness [9].

Attacks are classically transient and lasts from hours to days and may be precipitated by a number of conditions. That include high carbohydrates—especially refined carbohydrates and after exercise. Other precipitating factors include trauma, exposure to hot climate , classical upper respiratory tract infections, emotional breakdown, periodic menses, drugs like (diuretics, insulin, steroids, and carbonic anhydrase like acetazolamide), ethanol, or substance abuse drugs such as 3,4-Methylenedioxymethamphetamine (ecstasy) [10–13].

As the name suggests, TPP is associated with thyrotoxicosis. Any cause of thyrotoxicosis may be just sufficient to precipitate attacks in susceptible patients. In fact the condition has been reported in few patients with hypothyroidism who wereon replacement therapy with L-thyroxine and in another who took triiodothyronine (T3) for weight loss [14, 15]. The serum thyroid-stimulating hormone level is low or suppressed accompanied by raised free thyroxine (fT4) and triiodothyronine (fT3) levels consistent with primary thyrotoxicosis. Rarely some patients may have central thyrotoxicosis in which case the TSH is detectable and associated with elevated fT4 and fT3 levels [16]. Very less oftenlyMilder forms of thyrotoxicosis have been associated with thyrotoxic periodic paralysis adding to the diagnostic difficulties [17).

An ECG may show classical features of hypokalemia like prolongation of PR interval ,wide spread ST Depression and T wave flattening or inversion . Unlike hypokalaemia from other causes, sinus tachycardia predominates in TPP patients. Other findings include atrial fibrillation, AV-nodal block, ventricular fibrillation, and asystole [18,19]. The electrolyte imbalance can be severe enough to cause asystole and cardiac arrest [20]

4. Conclusion:-

TPP patients can have subtle signs and symptoms of thyrotoxicosis on presentation, and treating clinicians should have a high index of suspicion in diagnosing TPP and a get full thyroid function tests in these patients who present with sudden onset paralysis associated with hypokalemia.Once the diagnosis is certain, careful correction serum of potassium (total dose of 60-90 mEq/24 hours is recommended with close cardiac monitoring in intensive care setup as cardiac dysrhythmia risk is higher in these patients due to electrolyte imbalances.

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