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THYROTOXIC HYPOKALEMIC PERIODIC PARALYSIS (THPP) A CASE REPORT AND REVIEW OF LITERATURE.

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Abstract

Thyrotoxic Hypokalemic Periodic Paralysis (THPP) is one form of Periodic Paralysis, a rare group of disorders that can cause sudden onset of weakness. A case of a 29 year old male is presented here. The patient presented with sudden onset paralysis of his extremities. Laboratory evaluation revealed a markedly low potassium level. The patient's paralysis resolved upon repletion of his low potassium and he was discharged with no neurologic deficits. An association with thyroid disease is well established and further workup revealed Grave's disease in this patient. Although rare, Periodic Paralysis must differentiate from other causes of weakness and paralysis so that the proper treatment can be initiated quickly.

Keywords: *Thyrotoxic Hypokalemic Periodic Paralysis (THPP), hypokalemia.*

Introduction

Thyrotoxic hypokalemic periodic paralysis (THPP) is an uncommon alarming and potentially lethal complication of hyperthyroidism characterized by muscle paralysis and hypokalemia due to a massive intracellular shift of potassium occurs primarily in males of Asian descent including patients of Japanese, Chinese etc.,

THPP is a condition featuring attacks of muscle weakness in the presence of hyperthyroidism and it is usually accompanied by hypokalemia. The condition may be life-threatening if weakness of the breathing muscles leads to respiratory failure, or if the low potassium levels lead to cardiac arrhythmias. If untreated, it is typically recurrent in nature.

Etiology

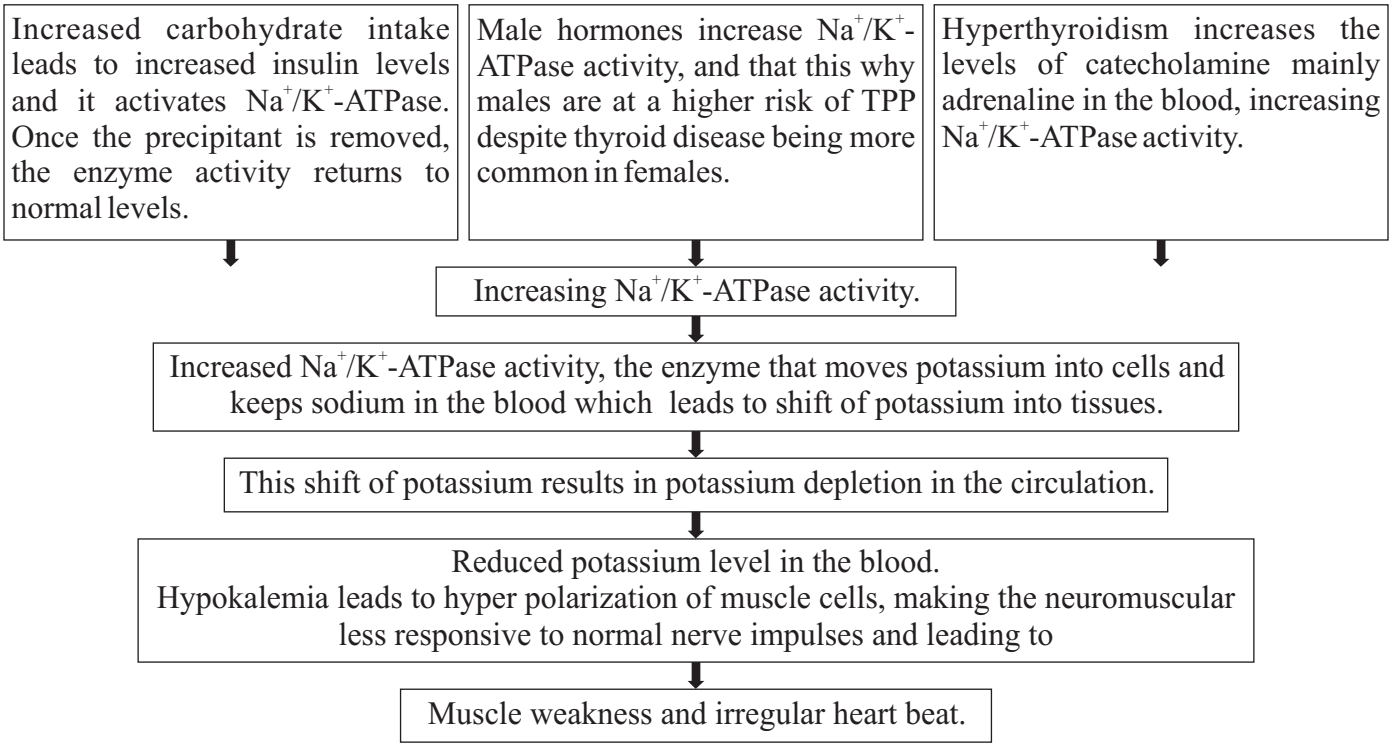
1. **Genetics** - Genetic mutations in the L-type calcium channel α1-subunit the mutations are located in a different part of the gene. In TPP, the mutations described are single-nucleotide polymorphisms located in the hormone response element responsive to thyroid hormone, implying that transcription of the gene and production of ion channels may be altered by increased thyroid hormone levels.

2. **HLA** - Certain forms of human leukocyte antigen (HLA) are more common in TPP. Linkage to certain forms of HLA, which plays a vital role in the immune response, might imply an immune system cause, but it is uncertain whether this directly causes TPP or whether it increases the susceptibility to Graves' disease, a known autoimmune disease.

3. **Thyroid Disease** - That common underlying form of thyroid disease associated with TPP is Graves' disease, an auto immune reaction that leads to overproduction of thyroid hormone.

4. TPP has also been identified in people with other thyroid problems such as thyroiditis, toxic nodular goiter, toxic adenoma, and TSH producing pituitary adenoma, excessive ingestion of thyroxin or iodine and amiodarone-induced hyperthyroidism.

Mechanism



Clinical manifestation

- An attack often begins with muscle pain, cramping, and stiffness.
- This is followed by weakness or paralysis that tends to develop rapidly, usually in late evening or the early hours of the morning.
- The weakness is usually symmetrical; the proximal limb muscles closer to the trunk are predominantly affected, and weakness tends to start in the legs and spread to the arms.
- Muscles of the mouth and throat, eyes, and breathing are usually not affected, but occasionally weakness of the respiratory muscles can cause life-threatening respiratory failure.
- Attacks typically resolve within several hours to several days, even in the absence of treatment. On neurological examination during an attack, flaccid weakness of the limbs is noted; reflexes are usually diminished, but the sensory system is unaffected. Mental status is not affected.
- Attacks may be brought on by physical exertion, drinking alcohol, or eating food high in carbohydrates or salt. This may explain why attacks are more common in summer, when more people drink sugary drinks and engage in exercise. Exercise-related attacks tend to occur during a period of rest immediately after exercise; exercise may therefore be recommended to abort an attack.
- There may be symptoms of thyroid hyper activity, such as weight loss, a fast heart rate, tremor, and perspiration but such symptoms occur in only half of all cases.
- The most common type of hyperthyroidism, Graves' disease, may also cause eye problems namely Graves' ophthalmopathy and skin changes of the legs pretibial myxedema.
- Thyroid disease may also cause muscle weakness in the form of thyrotoxic myopathy, but this is constant rather than episodic.

Treatment

- In the acute phase of an attack, potassium administered. This will quickly restore muscle strength and prevent complications.
- As the total potassium in the body is not decreased, there is a chance for potassium levels to overshoot leading to rebound hyperkalemia, so potassium chloride must be infused at a very slow rate; while other treatment is commenced.
- The effects of excess thyroid hormone typically respond to the administration of a non-selective beta blocker, such as propranolol. Subsequent attacks may be prevented by avoiding known precipitants, such as high salt or carbohydrate intake, until the thyroid disease has been adequately treated.
- Treatment of the thyroid disease usually leads to resolution of the paralytic attackdepending on the nature of the disease, the treatment may consist of hydrostatics that is drugs that reduce production of thyroid hormone, radioiodine.
- Occasionally thyroid surgery. Kilpatrick et al. (1994) found six reports of thyrotoxic hypokalemic periodic paralysis and described four additional cases all in males. They concluded that the disorders may be more frequent in blacks than previously suspected and should be considered when patients present with unexplained hypokalemia, muscular weakness and Rhabdomyolysis.

Case Report

A 30 year old male visited hospital with complains of cough and fever that started a week earlier had subsided after three days, had persistent dry cough. These complain were observed after having a heavy meal in the marriage party a week earlier. He had lost of weight about 10kg over the last 1 year. He had also reported tiredness; increased sweating and increased appetite despite weight loss. He reported with progressive weakness of both lower limbs since 3 days. He had first noticed weakness of lower limbs in the form of difficulty in getting up from squatting after toilet activity. He had noticed worsening in the form of difficulty in walking and climbing stairs. There was no history of facial or bulbar weakness, postural giddiness, sweating abnormalities and upper limb symptoms. He was diagnosed as THPP.

Physical Examination:

Weight loss 44kg

Small goiter

Motor symptoms - Hypotonic weakness of both lower limbs with mild neck weakness

Power -	Upper Limb	Lower Limb
	Rt -> 5/5	4/5 Proximally
	Lt -> 5/5	4/5 Proximally

Reflexes Biceps

Triceps

Supinator Rt and Left were Absent

Knee
Aukle

Laboratory Investigation

- Serum Potassium 3 meq. / L.
- Electro physiological evaluation was negative for GBS.
- Calcium 8.9 mg/dl, creatinine kinase 66 u/L.
- Total T3 – 282 mg/dl. (iodothyronine – Serum)
- Total T4 – Thyroxin – 18mg / dl.
- TSH - <0.01 µIU / ml , Magnesium 2.0ng / dl.
- Free T4 Serum – 5.43 mg / dl.

CT scan

Mild diffuse enlargement of both lobes of thyroid with small tiny nodules in both lobes and minimal retro tracheal extension with narrowing of airway.

Treatment

- Tab. Corbimazole 20mg,to reduce the T3 , T4 & maintain TSH.
- Inj. Potassium Chloride - 90 meq /24 hrs. Because patient have mild hypokalaemia ranges from 3-3.5 mmol/L
- Tab. Rantac 150 mg.
- For the prevention of hypolalaemia the patient is adviced to take oral potassium supplements 20-40 meq/day in 2-4 divided doses which are available as potassium acetate, potassium chloride, potassium bicorbonate, potassium citrate and potassium gluconate etc.,
- Tab. Propranolol, Beta blockers may reduce the number and severity of attacks by controlling hyperthyroid status and euthyroid status.

Nursing Process

- Impaired physical mobility related to neuromuscular weakness secondary to Hypokalemia.
- Activity intolerance related to immobility secondary to increased thyroid levels.
- Altered nutrition: intake exceeds body needs related to increased metabolic needs secondary to increased thyroid levels.
- Risk for Impaired skin integrity related to excessive sweating secondary to increased thyroid levels.

Goals

- Maintain normal levels of electrolytes.
- Achieves normal mobility.
- Maintains normal ECG pattern.

Interventions

- Assess the hemodynamic of the client.
- Monitor the ECG pattern of the to identify cardiac arrhythmias.
- Check the serum potassium level daily.
- Administer Inj. Potassium chloride after dilution as per the physician's advice as slow IV infusion. In case of syp.Potassium chloride dilute the syrup in water before administration
- Check the thyroid levels of the patient.
- Teach patient how to take the medicines and importance of follow up.
- Inform patient about the danger signs of THPP

Prognosis

The patient was adviced for endocrinologist opinion to rule out thyrotoxicosis and was discharged after a week with stable and normal hemodynamic and blood reports.

Conclusion

TPP is a rare condition in non-Asians, and the diagnosis at initial presentation is often delayed because of the subtleness of the clinical features of thyrotoxicosis and the similarities of the paralysis with other more common conditions. With population mobility and admixture, TPP is becoming more common in Asian countries. Early diagnosis prevents serious cardiopulmonary complications. TPP is a curable disorder that resolves when a euthyroid status is achieved.

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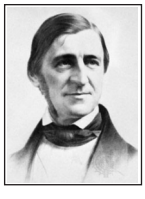

"To be yourself in a world that is constantly trying to make you something else is the greatest accomplishment."

-Ralph Waldo Emerson