Case Report

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Thyroxine Containing Slimming Agents, a Threat to Life

Internal Medicine Section

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ABSTRACT

The aim of presenting this case is to report the rare occurrence of thyrotoxic periodic paralysis in females induced by thyroxine abuse. Thyroxine containing remedies are commonly used as a slimming agent in obesity clinics. We are reporting a case of 32-year old female who was abusing levothyroxine as a slimming agent on advice of her gym instructor and had to be admitted in hospital with periodic paralysis after three months of thyroxine abuse. She was having hypokalemia along with flaccid muscle weakness of both upper and lower limbs. She was started on potassium supplements and she recovered completely within 24 hours and was discharged after 72 hours on propranolol with the advice to avoid thyroxine abuse.

Key Words: Thyrotoxic Periodic Paralysis, Thyroxine Abuse, Periodic Paralysis In Females

INTRODUCTION

Thyrotoxic periodic paralysis (TPP) [1] is the most common cause of acquired hypokalemic periodic paralysis. This condition is often transient but potentially serious. It has been associated with thyrotoxicosis of various aetiologies [2]. It is more prevalent in Asians; affecting males more commonly than females .The incidence of TPP in non-Asian thyrotoxic patients is around 0.1% while in Asians incidence is 1-2%, but in females TPP is even much rare with incidence in various Asian communities in the range of 0.04 to 0.17% [3,4]. We report here a case of young female developing periodic paralysis due to thyroxine abuse in order to lose weight.

CASE HISTORY

A 32-year old young female presented in emergency department with sudden onset of flaccid muscle weakness. History revealed that she was well in the last evening and she did her excessive workout in the gym but when she woke up in the morning, she was unable to get up from the bed due to weakness of all four limbs. She had a similar episode of weakness, though less severe, two weeks earlier, which recovered spontaneously within 3-4 hours. She had no family history of similar episodes. On neurological examination, power in both lower limbs was grade-1 and in both upper limbs grade-2 with no bulbar weakness or ophthalmoplegia. Higher function as well as sensory examination was normal. Plantars were bilaterally flexors. Deep tendon reflexes were diminished in both upper and lower limbs. Bladder and bowel were uninvolved. Findings in rest of the organ systems were unremarkable. A battery of tests were ordered which were mostly within normal limits [Table/ Fig 1] except for significant hypokalemia (serum potassium- 2.4 meq/l) and moderately increased creatine phosphokinase (CPK) level. ECG showed sinus tachycardia with blood pressure recorded as 126/70. A provisional diagnosis of periodic paralysis was made and IV potassium supplementation was started. Since episodic weakness with onset after age of 25 is unlikely due to periodic paralyses except TPP, thyroid profile was also ordered [5], though except for tachycardia, no other clinical features of thyrotoxicosis were present. Report of thyroid profile confirmed that patient was in thyrotoxic state. Enquiries were directed against any exogenous agent including herbal medicines taken by patient in her quest of losing weight. Patient denied taking any such agent except for dietary changes and a single tablet recommended by a local gym instructor to be taken in the morning empty stomach for the last three months which turned out to be 100 microgram levothyroxine.

Patients serum potassium level was monitored at six hours(3.0 meq/l), 12 hours (3.3 meq/l) and 24 hours(3.8 meq/l) and also thereafter. After 24 hours, I/V potassium supplements were discontinued as thyrotoxicosis was confirmed and patient was started on propranolol 20 mg thrice daily. Patient started improving within 6 hours with corresponding improvement in serum potassium level and power was normal after 24 hours of starting treatment. Patient was kept under observation for next 48 hours with strict monitoring of serum potassium levels. Serum potassium remained normal during this period and no rebound hyperklemia was noted. Patient

HB	12 gm%	S.CALCIUM	9.4 mg%
TLC	5600	CPK	406 U/L(24-170)
DLC	N-60,L-36,E-02, M-02	T3	1.67 ng/ml (0.97-1.69)
RBS	118 mg%	T4	15.2mcg/dl (5.53-11.0)
B.UREA	26 mg%	TSH	<0.015 mIU/l (0.46-4.68)
S.CREATININE	1.0 mg%	S.BILIRUBIN	0.6 mg%
SODIUM	136 meq/l	SGOT	48 U/L
POTASSIUM	2.4 meq/l	SGPT	44 U/L

[Table/Fig-1]: Lab investigations during hospitalization

Triggers	Examples	Notes
High glycaemic index foods	Sweetened drinks, pasta, certain fruits, white bread, certain cereals and fruits, for example, watermelon, rice, potatoes	High glycaemic index foods promote release higher insulin levels
Salt/high sodium intake	High-salt-containing foods	Promotes diuresis and loss of potassium
Stresses	Infection, psychological, surgery	Release of catecholamines which can activate Na/K/ATPase pump
Ambient temperature	Cold weather	
Physical activity	Rest after significant exertion	Weakness maybe apparent first thing in the morning
Gastrointestinal	Diarrhoea	Loss of potassium
Drugs	Acetazolamide, oestrogen, diuretics, laxatives, liquorice, cortisol, aminoglycosides, acrolides, fluoroquinolones	Listed antibiotics adversely affect neuromuscular transmission Liquorice and cortisol promote potassium excretion Oestrogen can increase insulin resistance

[Table/Fig-2]: Triggers of periodic paralysis attacks.

was discharged on 4th day on propranolol(60mg daily) to avoid recurrence of episode. Patient was educated about harms of over the counter substance abuse and also given strict instructions to avoid thyroxine abuse or any other "miracle drug" recommended by non- physician to lose weight. The patient was discontinued of propranolol after 3 months when her thyroid function tests were within normal range. Till last reported outpatient visit, she was symptom free and reported no paralytic episodes.

DISCUSSION

Thyrotoxic periodic paralysis is a rare complication of hyperthyroidism characterized by episodes of transient, flaccid weakness predominantly of proximal muscles accompanied by hypokalemia. Episode of weakness may last from few hours to several days. Clinical features are similar to hypokalemic periodic paralysis(familial periodic paralysis), an inherited disorder with normal thyroid levels.

Familial cases are inherited in an autosomal dominant manner. Mutations in CACNA1S and SCN4A gene adversely affect the function of calcium and sodium ion channels servicing muscle cells, respectively [6]. The KCNJ2 gene codes for inward rectifying potassium channels (Kir 2.1) that moves potassium ions into the cells of skeletal and cardiac muscles [7].

Serum potassium levels are usually low in both these conditions. The classical presentation is of ascending lower limb paralysis in the early hours of the morning, or after rest following strenuous exercise and a high carbohydrate meal, leaving the patient unable to move. Lower limb muscles are involved more commonly than upper limb, proximal limbs more severely than distal. Bulbar weakness is rare. Higher functions remain unaltered during acute episode. Other precipitating factors are summarized below.

As always a detailed clinical history and thorough clinical examination may reveal clues. Thyrotoxic features maybe absent or very subtle; therefore the clinician must have a high index of suspicion [5]. The frequency and severity of the episode does not correlate with the severity of thyrotoxic state and restoration of euthyroid state abolishes the attacks.

A spot urine test for electrolytes and potassium excretion can be very useful. Normal acid base status and low urinary potassium levels are characteristic of hypokalemia in TPP. It is also important to appreciate that serum potassium levels may be normal in between attacks.

The exact cause why only a few patients of thyrotoxicosis manifest TPP is not known but cause may be genetically determined as evidenced by its predilection for Caucasians. Patients of TPP have been found to have significantly higher Na-K ATPase pump number and activity than other thyrotoxic patients without periodic paralysis [6]. Thyroxine, insulin and catecholamines increases the activity of these pumps leading to intracellular shift of potassium leading to fall in serum potassium levels. This is well demonstrated as restoration of normal serum potassium level terminates the acute attack. Rebound hyperkalemia can occur as the acute episode resolves due to extracellular shift of potassium [7]. So, potassium supplementation should be closely monitored and should not be too vigorous to avoid rebound hyperkalemia. Propranolol, a nonselective beta blocker promotes early resolution of weakness and hypokalemia without increasing the risk of rebound hyperkalemia [8]. Propranolol also prevents the recurrence of episode by inhibiting the activity of Na-K ATPase [9,10]. So, prapranolol should be used in preference to potassium supplements in patients at risk of cardiopulmonary arrest.

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