IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)
e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 17, Issue 9 Ver. 14 (September. 2018), PP 67-69
www.iosrjournals.org

Thyrotoxic Periodic Paralysis In A 66 Year Old Diabetic Nigerian Woman: Adiagnostic Dilemma

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Abstract
Background: *Thyrotoxic Periodic Paralysis(TPP) is a potentially deadly complication of hyperthyroidism characterized by varying severity of muscle weakness to paralysis and hypokalemia. Clinical features of hyperthyroidism in patients with TPP may be subtle.*
Case Report: *Mrs O.O is a 66 year old woman who presented with progressive weakness of the upper and lower limb for three days, with a prior history of diarrhea for a day. She was previously diagnosed with hyperthyroidism, diabetes and hypertension. On examination, she had mild anterior neck swelling, reduced muscle power, tone and reflexes of the upper and lower limbs with normal muscle bulk. She was successfully managed with Potassium replacement and anti-thyroid medication.*
Conclusion: *Thyrotoxic Periodic Paralysis can be treated adequately and promptly to avoid complications , a high index of suspicion is needed to make a diagnosis.*
Keywords: *Thyrotoxic Periodic Paralysis, Hyperthyroidism, Hypokalemia*

Date Of Submission: 20-09-2018 Date of acceptance: 08-10-2018

I. Introduction

In hypokalemia, severity of muscle involvement can be from muscle weakness to paralysis, and when paralysis occurs is called hypokalemic paralysis.

Thyrotoxic Periodic Paralysis (TPP) is characterized by acute onset of severe hypokalemia and profound proximal muscle weakness in patients with thyrotoxicosis [1]. In 1902 Rosenfeld described the associated between periodic paralysis and thyrotoxicosis [2] TPP is more commonly reported in oriental Asians [3] but has also been reported from the western countries [4].

TPP and familial periodic paralysis have similar neuromuscular presentations hence misdiagnosed can sometimes occur. The classification of hypokalemic paralysis comprise of those due a large potassium deficit caused by losses via gastrointestinal or renal called non hypokalemic periodic paralysis and those due to shift of potassium into the intracellular space without a total potassium deficit called hypokalemic periodic paralysis. The latter is thought to be related to TPP.

Even though it is commonly seen in Graves' disease, TPP is not related to the etiology, severity, and duration of thyrotoxicosis [1].

II. Case Summary

Mrs O.O a 66 year old lady, Presented with progressive weakness of the body, inability to walk and move the upper limbs over 3 days, which followed a bout of loose stool about 4-5 times a day, non-mucoid, non-bloody stool. No vomiting but mild abdominal discomfort. Her appetite was preserved. No associated headache or fever. No change in urinary frequency or dysuria. She had occassional palpitation but lately improved, no dyspnoea ororthopnoea . She was previously diagnosed with hyperthyroidism, diabetes mellitus and hypertension

She had heaviness in the limbs which progressed to inability to use the upper limb effectively and walking. There was intermittent painless muscle cramps in the lower limb. No associated seizures, blurring of vision or loss of consciousness.

She was seen by a General Practitioner who made a working diagnosis for cerebrovascular diseaseto excludeGullainBarre´ syndrome, transverse myelitis and asked for the Neurologist review.

Clinical examination revealed resting pulse rate of 94/minute, blood pressure of 140/70mmHg with first and second heart sound only. Had a respiratory rate of 18 breaths per minute, with oxygen saturation at 98%.Also had mild thyroid enlargement with firm consistency, though no bruit was audible. There was no proptosis, She had normal muscle bulk with slightly reduced muscle tone. Muscle power in the hip muscle of the lower limb 2/5, knee and ankle were 4/5. In the upper limbs, shoulder 3/5 while elbow and wrist were

4/5.Deep tendon reflex were reduced. Sensation was lost in a stocking fashion, as well as proprioception and vibration with no sensory level.

A reviewed diagnosis of Hypokalemic Periodic Paralysis was made in a patient being followed up for Hyperthyroidism, diabetes and hypertension.

Laboratory Investigation

LABORATORY PARAMETER	**DAY 1**	**DAY 2**	**DAY 3**	**DAY 4**	**DAY 11(1 week after Discharge)**
Sodium (135.0-145.0mmol/L)	134.6	135.0	141.9	144	142.1
Potassium(3.5-5.0mmol/L)	1.6	2.7	3.4	3.53	4.1
Chloride(97.0-107.0mmol/L)	96.0	96.2	108.6	105.3	102.0
Bicarbonate(20.0-31.0mmol/L)	18.0	21.0	20.6	24.0	24.8
Urea(1.7-8.3mmol/L)	11.0	9.6	6.3	4.8	5.1
Creatinine(Male:80.0-115umol/L, Female: 53.0-97.0mmol/L)	121.0	103.4	72.5	63.8	72.7
Calcium(2.02-2.60mmol/L)	-	1.97	-	2.06	-
Creatinine Kinase					
BLOOD GLUCOSE					
GlycatedHaemoglobin	-	7.2%	-	-	
Fasting Blood Glucose(4-7mmol/L)	-	4.5	5.0	5.8	4.9
2 Hours Postprandial	-	8.8	10.2	9.6	9.1
Random Blood Glucose	7.0				
THYROID FUNCTION					
TSH(0.38-4.31mlU/L)	-	-	<0.01	-	-
T3 (1.22-2.43nmol/L)	-	-	3.31	-	-
T4(63.2-141.9nmol/L)	-	--	160.0	-	-
FULL BLOOD COUNT					
White Blood Cell (2.5-10.0 X 10^9/L)	-	8.0	-	-	-
Neutrophils (35.0-80.0%)	-	55.2	-	-	-
Lymphocytes (15.0-50.0%)	-	37.5	-	-	-
Monocytes (2.0-15.0%)	-	7.3	-	-	-
Eosinophils	-	-	-	-	-
Basophils	-	-	-	-	-
Red Blood Cell (3.50-5.50X 10^{12}/L)	-	5.0	-	-	-
Haemoglobin (11.2-16.5g/dL)	-	14.6	-	-	-
Packed Cell Volume (35.0-55.0%)	-	40.8	--	-	-
Mean Corpuscular Volume (75.0-100.0f l)	-	81.6	-	-	-
Mean Corpuscular Haemoglobin Concentration (25.0-35.0pg)	-	29.2	-	-	-

Electrocardiogram was essentially normal and a previous thyroid ultrasound scan showed a diffuse thyroid mass. Five hours after the initiation of intravenous potassium replacement, the patient's neurologic symptoms (weakness) started improving with a climax after four days with a restoration of muscle power and potassium returning to normal.

III. Discussion

Thyrotoxic Periodic paralysis is more often overlooked and/or delayed due to lack of awareness among the physicians and associated mild symptoms of hyperthyroidism [5,6]. TPP occurs commonly between 20-40 years of age with a male preponderance, and a racial predisposition among the Asians [4,7-10]. TPP predominantly affects males in spite a much higher incidence of thyrotoxicosis in women. In the work by McFadzean et al, TPP occurs in 13% of male and 0.17% of female thyrotoxic patients(1).Also Okinaka et al found among the Japanese, the incidence was 8.67 among male and 0.4% among female thyrotoxic patients in 1957 [7]. Overall, the male to female ratio is thought to ranges from 17:1 to 70:1 [1, 7, 11,12].

Our patient presented with symmetric heaviness of the lower limbs following a bout of severe diarrhea with muscle stiffness and cramps. She had no other precipitating factors such as trauma, exposure to cold, emotional stress, infection, alcohol ingestion, menses, and drugs like diuretics, insulin, or steroids [5].

Sensation and level of consciousness are usually spared, but being a known diabetic for 10 years she had loss of sensation in a glove and stocking fashion with depressed deep tendon reflexes which may be attributable to diabetic neuropathy. Deep tendon reflexes are decreased or absent in patient with TPP [1,13–15]. Our patient had both hypokalemia and hyperthyroidism at presentation (Table 1).Porthiwala et al and Kung et al in their works have shown that the hallmark of Thyrotoxic periodic paralysis is hypokalaemia [16,17]but occasionally if the patient is at the recovery phase of the paralysis, the serum potassium can be normal (17).

The pathogenesis of TPP is unclear but appears to involve intracellular shift of potassium into the cells from extracellular space and not total body depletion, leading to hyperpolarization and inexcitability of muscle cell membranes. The rapid influx of potassium is due to increased activity of sodium potassium adenosine triphosphatase pump (Na+ K+ ATPase).(17) In TPP, high circulating levels of thyroid hormones directly induce Na+–K+ ATPase activity while hyperadrenergic state and insulin excess indirectly leads to increased Na+ K+ ATPase activity, with subsequent hypokalemia and periodic paralysis [18]. Some may present with transient hypophosphatemia and hypomagnesemia along with elevation in creatine phosphokinase. Androgens have also been found to indirectly increase Na+ K+ ATPase pump activity.[19]

In our index case electrocardiographic findings were normal this may be attributed to the fact that it was done of day two of admission. Typical electrocardiographic findings may include sinus tachycardia, increased P-wave amplitude, prolonged PR interval, widened QRS complexes, decreased T-wave amplitude, and U waves. Other electrocardiographic abnormalities include atrioventricular block, atrial fibrillation, ventricular fibrillation, and asystole [20].The index patient was on treatment for hyperthyroidism with carbimazole and propranolol to achieve euthyroidism. Delayed diagnosis hamper the treatment with potassium supplements which were administered to correct hypokalaemia

IV. Conclusion

TPP is thought to be a rare condition among non-Asians, with a male sex preponderance and age group of 20-40years. Diagnosis of TPP at the initial encounter is often delayed as other causes of paralysis of the extremities arebeing considered,as seen in our index case. This is largely due to unawareness of this disorder among non-Asians. An improved awareness of this disorder among physicians would lead to early diagnosis and treatment with potassium supplements, nonselective beta-blockers and anti-thyriod medication

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