Case Report

Case report of postmenopausal women with thyrotoxic periodic paralysis being treated with radioactive iodine as a definitive therapy

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Thyrotoxic periodic paralysis is a potentially life threatening complication of hyperthyroidism. It is characteristized by hypokalemia with proximal muscle paralysis in patients with thyrotoxicosis. It can be precipitated by strenuous physical activity or ingestion of a high carbohydrate diet.

TPP is usually a disease of Asian men in age group of 20-40 years. We present, here a case of a 56years old postmenopausal women who came with acute onset of weakness of both lower limbs. At the time of presentation she had severe hypokalemia and hyperthyroidism.

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Key words **: Thyrotoxicosis, periodic, paralysis, hypokalemia.**

Thyrotoxic periodic paralysis is an uncommon and potentially lethal complication of hyperthyroidism characterized by hypokalemia and periodic paralysis[1,2]. Approximately 90% of cases reported in literature are men of Asian origin and the age of onset is 20 to 40 years[3]. The attacks can be precipitated by high carbohydrate meal, heavy and strenuous exercise and physical and mental stress4.

We report a case of 56 years old post menopausal women who presented in emergency department of B K Hospital with complaints of acute onset of weakness of both the lower limbs. She was first treated with potassium chloride to correct her hypokalemia and later given radioactive iodine (at INMAS) as a definitive cure for her hyperthyroidism. She is now hypothyroid and has never experienced any attack of periodic paralysis since then.

Case Report

We present case of a 56 old female who was referred to us from the emergency department of B K Hospital Faridabad, where she was admitted with acute onset of weakness of both the lower limbs. She had also been complaining of loss of weight and palpitations for last 4 to 6 weeks.

On physical examination in the emergency department of B K Hospital Faridabad, she was conscious, a febrile had tachycardia. Her pulse rate was 143 beats per minute which was regular and her blood pressure was 136/98 mm Hg .She had diffuse thyromegaly and her deep tendon reflexes were absent in the lower extremities. She had fine tremors of her outstretched hands. Her cranial nerves and sensory examination was normal.

Lab finding on admission at BK hospital

(1) CBC with ESR was normal

(2) Plasma glucose was normal

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(3) Thyroid function test – Table 1

Test	Patient's value	Normal range
Free T3	7.5pmol/L	2.6-6.8 pmol/L
Free T4	37.2 pmol/L	12-22 pmol/L
TSH	<0.004	0.27-4.2Iu/mL

ECG showed sinus tachycardia with ST segment depression in leads V1 to V4.

Her potassium was low 1.5mmol/L (normal range 3.5-5.5m mol/l) at the time of admission in the emergency department .She was treated with intravenous potassium chloride for first six hours and later shifted to oral potassium chloride. Mean while she was also started with anti thyroid medications and beta blockers. Almost six hours after admission her serum potassium levels had improved to a normal value of 3.8mmol/L. Her deep tendon reflexes slowly returned to normal and she was discharged the next day and referred to INMAS which is a tertiary care centre for definitive management of thyroid disorders.

Clinical examination done at INMAS showed a mild thyromegaly with grade 1 goiter and mild exopthalmos. Her heart rate was 122/ minutes regular and her blood pressure was 130/90 mmHg. There was no neurological deficit at that time and patient came walking to the C -6 clinic of INMAS. She was treated with anti thyroid drugs including carbimazole 40 mg and beta blockers for 6 weeks and when her free thyroid hormones levels came close to the euthyroid range she was subjected to radioactive Iodine uptake(RAIU) studies and carbimazole was stopped 1 week prior to RAIU. The results of her radioactive iodine uptake studies are as under :

Table 2 — *Raiu Results of the Patient*

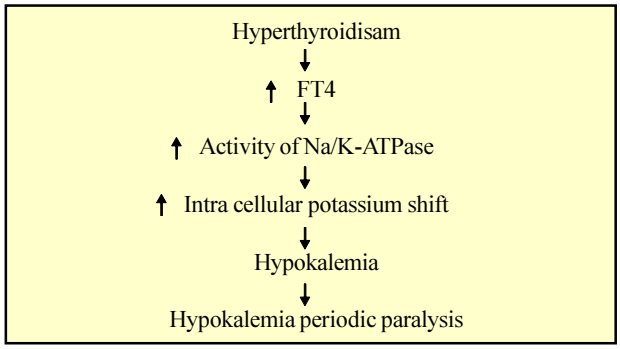
Time (uptake)	Patients' value	Normal
Two Hour Uptake	8%	
Six Hour Uptake	18%	
Twenty Four Hour	83%	115-35%

She was administered 5 mille curie of radioactive iodine therapy and three months later she became hypothyroid and was started with levothyroxine replacement which she will now have to take for the rest of her life.

Pathogenesis :

The exact pathogenisis of TPP is not known but it seems that hypokalemia is due caused due to rapid shift of potassium into the intracellular compartment .this is due to the increased activity of sodium- potassium –ATPase pump[5].

Discussion



TPP is considered to be predominantly a disease of Asian men with M: F ratio of 20:1 and the age of onset being 20-40years, but we report this case of TPP in a postmenopausal Asian woman.

The diagnosis of TPP is based on clinical finding of periodic paralysis secondary to hypokalemia in a patient suffering from hyperthyroidism. An attack of periodic paralysis may range in severity from mild weakness to severe flaccid paralysis mainly affecting the proximal muscles. Cranial nerves, respiratory muscles and cardiac muscles are usually spared. Attacks are usually precipitated by intake of a high carbohydrate meal or extreme physical exertion.

The treatment goals in management of periodic paralysis are

(1) Potassium supplementation

(2) Electrolyte monitoring to prevent rebound hyperkalemia

(3) To avoid precipitating factors

(4) Use of non selective beta blockers like propanolol not only reduces thyrotoxic symptoms but also provides relief to the paralytic episodes by inhibiting the intracellular shift of potassium induced by epinephrine[6].

(5) Use of antithyroid medication to restore euthyroid status[7].

(6) Definitive therapy for underlying thyrotoxic state in form of radioactive iodine or surgery to render the patient euthyroid . Rendering the patient euthyroid is the only means of preventing further paralytic episodes[8].

Conclusion :

Any patient presenting with severe and acute muscle weakness along with hypokalemia should always be evaluated to rule out thyrotoxic periodic paralysis especially if the patient is a young male of Asian origin. Hypokalemia is due to increased activity of sodium- potassium –ATPase pump which causes rapid intracellular shift of potassium. The total body potassium pool in maintained and there is no potassium depletion.

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