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Case Report

Insights into Thyrotoxic Periodic Paralysis: A Comprehensive Case Study of a Rare Thyrotoxic Manifestation

Arun Bargali[1], Princy Poonam[2], Manisha V. Prabhu[3], Grisha Maich[4], Rajam Iyer Bhavana[5], Rashmi Mishra[6]

[1,2,3,4,5]Resident, Department of Internal Medicine, Maulana Azad Medical College and Lok Nayak Hospital.
[6]Assistant Professor, Department of Internal Medicine, Maulana Azad Medical College and Lok Nayak Hospital.
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I N F O

Corresponding Author:
Rashmi Mishra, Department of Internal Medicine, Maulana Azad Medical College and Lok Nayak Hospital.
E-mail Id:
rashmi.virgo02@gmail.com
Orcid Id:
https://orcid.org/0000-0003-2621-6515
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A B S T R A C T

Thyrotoxic periodic paralysis (TPP) is a rare and potentially lethal complication of hyperthyroidism characterised by sudden onset paralysis associated with hypokalemia. We report a case of a 38-year-old male who presented with a history of multiple episodes of vomiting followed by sudden onset quadriparesis. Subsequently, he developed respiratory distress and was intubated. On examination, he was irritable with tachycardia and tachypnea with a temperature of 102 °F and BP of 160/78 mm Hg. Thyroid profile was suggestive of primary hyperthyroidism with TSH of 0.015 mIU/L, fT3 of 20.8 pmol/L and fT4 of 58.6 pmol/L. Serum potassium was 1.7 mmol/L. MRI brain was normal. A thyroid scan showed an enlarged thyroid with increased tracer uptake. TRAb was positive. The patient was diagnosed with hypokalemic periodic paralysis secondary to thyrotoxicosis and was managed with propylthiouracil along with a saturated solution of potassium iodide with a high dose of beta blockers, IV fluids with potassium supplementation, antibiotics and steroids. The patient improved clinically and was discharged on antithyroid medication.

Keywords: Thyrotoxicosis, Periodic Paralysis, Hypokalemia, Quadriparesis

Introduction

Thyrotoxic periodic paralysis is an unusual and dramatic complication of hyperthyroidism that requires early recognition and emergent management. It is characterised by transient, recurrent episodes of flaccid muscle paralysis affecting proximal muscles more severely than distal muscles.[1] It is almost always associated with hypokalemia that occurs due to activation of Na+/ K+ ATPase in thyrotoxicosis that causes a transcellular shift of Potassium into the intracellular fluid with normal total body Potassium.[2] The severity of weakness is in proportion to the degree of hypokalemia and usually the weakness resolves as the serum Potassium levels are normalised.[3] Despite a higher incidence of thyrotoxicosis in women, TPP is more frequently observed in men, and unlike familial periodic paralysis (FPP), familial cases of thyrotoxic periodic paralysis are extremely rare.[1] Treatment involves immediate reversal of muscle paralysis by correcting hypokalemia

and prevention of future attacks by achieving a euthyroid state. We report a case of thyrotoxic periodic paralysis who presented with quadriparesis and respiratory failure and responded to antithyroid medications in addition to standard symptomatic therapy.

Case Report

We report a case of a 38-year-old male who presented to the medicine emergency department with a history of sudden onset quadriparesis for one day. He had multiple episodes of vomiting prior to weakness. He had a feeling that all his limbs were heavy. Subsequently, he also complained of shortness of breath and drowsiness. He was intubated in view of his gasping state and CO_2 narcosis. There was no history of fever, headache, neck pain or body aches preceding the weakness. No history of seizures or any involuntary body movement. No history of tingling, numbness, loss of pain or temperature sense, or cotton wool sensation. No history of shortness of breath, or chest tightness. There was no history suggestive of cranial nerve or higher mental function abnormality. He had a history of similar 2–3 episodes in the last one year.

On examination, he was sedated peri-intubation. Body temperature was 102 °F and blood pressure was 160/78 mm Hg. Tone was reduced in all 4 limbs and deep tendon reflexes were absent. Planters were mute bilaterally. There was no sensory, autonomic or cranial nerve involvement.

His routine blood investigations were normal and have been provided in Table 1. His ABG was suggestive of type 1 respiratory failure while his chest X-ray was clear. The thyroid profile was suggestive of primary hyperthyroidism detailed report of which is provided in Table 2. Serum potassium was 1.7 mmol/L. ECG was suggestive of sinus tachycardia with Flat T waves and U waves in leads V_2–V_3. Electromyography was normal. CSF analysis was normal. MRI brain was normal. Tc99 thyroid scan showed a mildly enlarged thyroid gland with mildly increased uptake of tracer in both lobes- suggestive of thyroiditis (Figure 1). Anti-thyroid antibodies came out to be positive with titres of 2.15 IU/L. The patient was started on potassium-supplemented IV fluids and IV steroids. For the thyrotoxicosis, he was administered propylthiouracil 200 mg four times on day 1 followed by 100 mg thrice daily, along with a saturated solution of potassium iodide 500 mg four times a day and tablet propranolol 40 mg thrice daily. He was extubated on day 3 of admission and continued to improve on the above medication. He was discharged on day 10 on antithyroid medication and is on regular OPD follow-up.

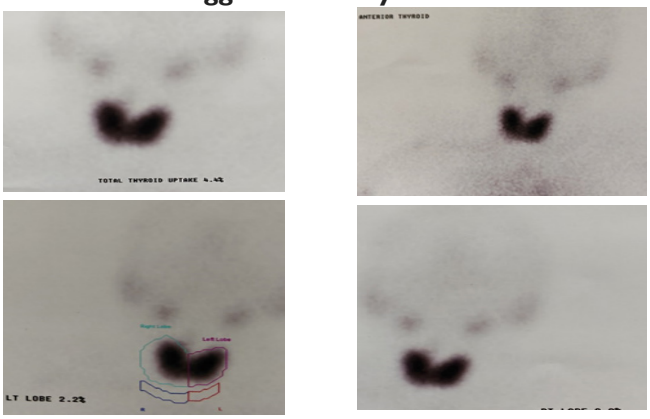
Table 1.Routine Blood Investigations of Patient

Parameter	Patient's Value	Normal Range
Haemoglobin	12.5 g/dl	13–16 g/dl
TLC	6700	4000–11000/cc
DLC	65/32/2/1	
Platelet Count	3.12 lac	1.5–4 lac
ESR	16	
Total Bilirubin	0.6 mg/dl	0.2–1.3mg/dl
Alanine Transaminase	31 U/L	< 50 U/L
Alkaline Phosphatase	98 U/L	38–126 U/L
CPK Total	45	10–120 ug/L
CPK MB	10	5–25 IU/L
Aspartate Transaminase	68 U/L	17–59 U/L
Blood Urea	21 mg/dl	15–39 mg/dl
Serum Creatinine	0.6 mg/dl	0.66–1.25 mg/dl
Sodium	138 mmol/L	135–145 mmol/L
Potassium	1.7 mmol/L	3.5–5 mmol/L
Magnesium	2.3	1.8–3 mg/dl
Phosphorus	3.7	3–4.5 mg/dl
Ddimer	394	
Serum Iron	58	
B12 levels	990	
Folic Acid	16.3	
Ferritin	124 ng/ml	6.24–137 ng/ml
Urine potassium	8 mmol/L	< 10 mmol/L

Table 2.Thyroid Function Test of Patient

Parameter	Value	Reference Range
TSH	0.015	0.465–4.68 mIU/L
fT3	20.8	4.25–8.21 pmol/L
fT4	58.6	10–28.2 pmol/L

Figure 1.Tc99 Thyroid Scan of Patient Showing Mildly Enlarged Thyroid Gland with Mildly Increased Uptake of Tracer in Both Lobes, Suggestive of Thyroiditis



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Discussion

Periodic paralysis is a group of neuromuscular disorders due to familial (primary periodic paralysis) and acquired (secondary periodic paralysis) causes characterised by acute flaccid muscle weakness with normal sensory functions.[4] Primary periodic paralysis is a channelopathy due to the involvement of sodium, potassium, calcium and chloride channels on the muscle cell membrane,[5] while secondary periodic paralysis is due to varied causes, thyrotoxic periodic paralysis being one of the most common causes among them.

Thyrotoxic periodic paralysis (TPP) is a rare but fatal complication of hyperthyroidism characterised by the triad of thyrotoxicosis, hypokalemia, and acute muscle weakness usually during the night.[6] Although the exact mechanism remains unknown, it is thought to arise from hyperactivity of the Na^+K^+ ATPase by beta-adrenergic receptors in skeletal muscles secondary to an increase in the levels of thyroid hormones. This leads to a massive intracellular shift of potassium and depleted extracellular stores, causing hyperpolarisation of muscle membrane and subsequent muscle paralysis.[7]

The differential diagnosis for acute onset quadriparesis like Gullain barre syndrome, flaccid paralysis in myelitis, and para infectious causes should always be ruled out.[8] Periodic paralysis is often misdiagnosed as Gullian Barre syndrome given its similar presentation. However, a lack of antecedent history of febrile illness, a non-progressive type of paralysis and normal CSF findings help rule out GBS as a cause.

The symptoms are usually precipitated by high carbohydrate diets, stress or physical activity that cause an increase in Na-K ATPase activity or increase in beta-receptors number and/ or sensitivity. Such attacks are more frequent during nighttime and common during summer as was the case in our patient.[9] The most consistent laboratory finding associated with TPP is hypokalemia in a patient with a deranged thyroid function test. The severity of muscle paralysis mirrors the degree of hypokalemia, and as hypokalemia gets resolved, the paresis improves usually in the reverse order of its appearance. Urine potassium excretion is low for the degree of hypokalemia and is thus helpful in differentiating TPP from other etiologies of hypokalemia, many of which cause increased urinary loss.[10] In our case, urinary potassium levels were in 8mmol/L which was in the normal range.

Radioactive iodine uptake and thyroid scans are the gold standard to differentiate thyrotoxicosis with sensitivity and specificity ranging above 95%. While thyroid receptor antibody (TRAb) is usually positive in patients of Graves disease as is evident in this case, its absence does not completely rule out the disease.[11]

Potassium therapy mainly via the IV route with close monitoring of serum potassium remains the mainstay in the management of TPP. Non-selective beta blockers are used to counteract adrenergic overactivity. Once the hypokalemia has been managed, the thyrotoxic state should be corrected using an anti-thyroid medication, radioactive iodine thyroid ablation, or thyroidectomy.[2]

Conclusion

This case emphasises the need to evaluate thyroid function in a patient with recurrent episodes of flaccid/ Lower motor neuron type weakness associated with hypokalemia. Recurrent attacks may be mitigated by avoiding known triggers and restoration of the euthyroid state with appropriate treatment.

Conflict of Interest:

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