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Thyrotoxic periodic paralysis associated with Grave disease: An unusual presentation with weakness

Gajanan Nandkumar Umalkar[1*], **Rajeshwari Vhora**[2], **Gajanan Chavan**[3], **Vaishnavi Kantode**[4], **Mayur Wanjari**[5]

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Authors' Affiliation:
[1]Junior Resident, Department of Emergency Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences, Sawangi, Wardha, Maharashtra, India
[2]H.O.D., Department of Emergency Medicine BJ Medical College and Sion Hospital, Pune, India
[3]H.O.D., Department of Emergency Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences, Sawangi, Wardha, Maharashtra, India
[4]Department of Medical Surgical Nursing, Smt. Radhikabai Meghe Memorial College of Nursing, Datta Meghe Institute of Medical Sciences, Sawangi, Wardha, Maharashtra, India
[5]Department of Research and Development, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences, Sawangi, Wardha, Maharashtra, India

[*]**Corresponding author**
Junior Resident, Department of Emergency Medicine, Jawaharlal Nehru Medical College, Datta Meghe Institute of Medical Sciences, Sawangi, Wardha, Maharashtra,
India
Email: gnumalkar777@gmail.com

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ABSTRACT

Acute quadriplegia is an emergency frequently encountered in clinical practice. Localization of acute quadriplegia ranges from the neuromuscular junction, peripheral nerves, anterior horn cells and central nervous system and electrolyte disturbances. Here we present a case of 48 years old male with a known case of grave disease with thyroid ophthalmopathy. On admission patient has been treated with medical management, on medical management patient methylprednisolone 500 mg in 500 ml of normal saline and was admitted under observation of physician in critical care I.C.U. After gone through medical management patient prognosis was good and advice for the regular follow up.

Keywords: Hyperthyroidism, Thyrotoxic Periodic Paralysis (T.P.P.), Weakness, Thyrotoxicosis

1. INTRODUCTION

Hyperthyroidism is characterized by excessive thyroid hormone secretion, which can lead to thyrotoxicosis (Doubleday and Sippel, 2020). Several muscular disorders, like exophthalmic ophthalmoplegia, myasthenia gravis and thyrotoxic myopathies, are seen with thyrotoxicosis (Tinker and Vannatta, 1987). Grave's Disease is associated with the majority of T.P.P. patients. To prevent life-threatening complications, early diagnosis of T.P.P is essential (Meseeha et al., 2017). Thyrotoxic periodic paralysis (T.P.P.) manifested by rapid onset of muscle weakness. The intracellular shift of potassium results in hypokalemia (Rolim et al., 2014; Vijayakumar et al., 2014). T.P.P. is defined by three classical features: Thyrotoxicosis, hypokalemia, an acute painless muscle weakness. We report a case of 48 years hyperthyroid male with exophthalmos presenting with gradual onset and progressive paralysis.

2. CASE PRESENTATION

The 48-year-old male patient has a known case of grave disease with thyroid ophthalmopathy, as shown in (Figure 1). He was presented to emergency

medicine with a history of weakness in the bilateral lower limb for two days which was gradual in onset and progressive. The patient denied a previous record of similar complaints, a history of physical exertion, heavy carbohydrate meals, or alcohol intake preceding the weakness, fall, or trauma, bowel and bladder incontinence, chest pain, seizures, loss of consciousness and strenuous activity.

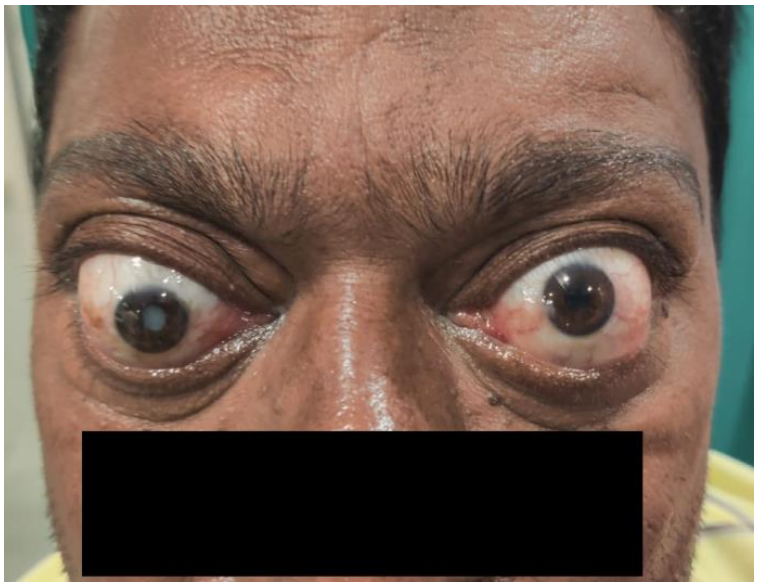

Figure 1 Shows thyroid ophthalmopathy

On presentation patient was afebrile and his Pulse rate was 82 /min, Blood pressure 110 /70 mm hg and Respiratory Rate 18 /minute. On examination, he was consciously oriented to time, place and the person, with his Glasgow Coma score of 15/15; pupils were bilaterally equally reactive to light 3 mm and diminished reflexes and power (Table 1).

Table 1 Shows the power of the upper and lower limb

Power	Right	Left
Upper limb	4/5	4/5
Lower limb	2/5	2/5

Cranial nerves examination and signs of meningeal irritation were absent on review. Heart sounds were normal on auscultation with bilaterally air entry equal on both sides; the abdomen was soft and non-tender per abdominal examination. On investigation, Complete Blood count, Liver Function Test, serum calcium and urinary calcium, serum magnesium, ABG, CSF– Normal. Thyroid function test and serum potassium level in the (Table 2)

Table 2 Shows serum potassium levels and thyroid function test

Investigation	Measure Value
Serum potassium	2.2 mmol/L
Serum TSH	0.234 mIU/ml
Serum T3	150 ng/dl
Serum T4	11.0 mcg/dl

This patient has been treated with an injection of methylprednisolone 500 mg in 500 ml of normal saline and was admitted under critical care I.C.U. Methylprednisolone was continued with tablet propranolol 40 mg once daily, multivitamins and tears substitutes. The patient prognosis was good and the patient was discharged after four days with the same medication every four weeks.

3. DISCUSSION

T.P.P. is characterized by episodes of muscle weakness in patients with excessive circulating thyroid hormone.T.P.P. is a life-threatening complication of thyrotoxicosis. Grave's disease is associated with most cases of T.P.P. (McFadzean and Yeung, 1967). Paralytic episodes result after developing hyperthyroidism and related symptoms. The duration of attacks varies and the episodes time ranges from hours to days (Lin, 2005).

Hip and shoulder involvement and weakness are more in the lower extremities than the upper extremities, with the participation of proximal muscles more than distal muscles without Bowel and bladder disturbances (Siguier et al., 1970). Hypokalemia leads to prolonged Q.T. interval E.C.G., and a hyperadrenergic state result in sinus tachycardia. The pathophysiology behind T.P.P. is unclear; however, some studies suggest channelopathy mutations. In our case patient was a known case of a severe disease on medication and presented with gradual onset progressive paralysis; reduced potassium levels could be explained by hyperactivity of Na+/K+ -ATPase pumps in the skeletal muscle cell membranes in the patient of thyrotoxicosis (Vijayakumar et al., 2014). Regular nerve conduction velocity tests and C.T. brain plain ruled out different etiologies like C.N.S. pathology and G.B.S. This presentation was similar to Sanjeev Karel et al., on a 25-year-old male (Kharel et al., 2022).

4. CONCLUSION

T.P.P. is a rare disease often misdiagnosed or challenging to differentiate from F.P.P., the first manifestation of thyrotoxicosis and it can present atypically like weakness. Therefore, emergency physicians must do thyroid function tests in all cases of periodic paralysis to make an early diagnosis of T.P.P. and start definitive treatment.

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Informed Consent

Informed Consent was obtained from the patient.

Author's contribution

All the authors contributed equally to the case report.

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Conflict of interest

The authors declare that there is no conflict of interests.

Data and materials availability

All data sets collected during this study are available upon reasonable request from the corresponding author.

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