

# An Interesting Case of Quadriplegia- Thyrotoxic Periodic Paralysis

Bharat Vadlamani<sup>1</sup>, Sukdev Manna<sup>2</sup>

<sup>1</sup>Junior Resident, Department of Internal Medicine, All India Institute of Medical Sciences Rishikesh

<sup>2</sup>Senior Resident, Department of Clinical Immunology & Rheumatology, All India Institute of Medical Sciences Rishikesh

Corresponding Author: Bharat Vadlamani

## ABSTRACT

Periodic paralysis is a rare neuromuscular disorder related to a defect in muscle ion channels, characterized by episodes of painless muscle weakness, which may be precipitated by heavy exercise, fasting, or high-carbohydrate meals. Periodic paralysis is classified as hypokalemic when episodes occur in association with low potassium blood levels or as hyperkalemic when episodes can be induced by elevated potassium. Most cases of periodic paralysis are hereditary, usually with an autosomal dominant inheritance pattern. Acquired cases of hypokalemic periodic paralysis have been described in association with hyperthyroidism. In our case who is a 24 year male, presented with 1st episode of acute onset rapidly progressive ascending type of pure motor areflexic flaccid quadriplegia without bowel bladder, cranial nerve involvement with significant weight loss with diffuse thyroid swelling without definite signs and symptoms of hypo or hyperthyroidism without any family history with acholuric jaundice. On further investigating the patient, Grave's disease was diagnosed along with severe hypokalaemia. A final diagnosis of Grave's disease with thyrotoxic periodic paralysis was made and patient had significant improvement with potassium supplementations, oral carbimazole and propranolol. An advice to avoid heavy meals and strenuous exercises was given.

**Keywords:** Thyrotoxic periodic paralysis, Hypokalemia, Quadriplegia, Hypokalemic periodic paralysis, Grave's disease, Gilbert's syndrome

## CASE SCENARIO

A 24 years-old male presented with weakness of all four limbs for 12 Hours and weight loss for last 3 months. According to the patient, he developed acute onset rapidly progressive weakness of all four limbs since last 12 hrs Early in the morning while going to bathroom he felt heaviness in both his lower limbs, more distally with little difficulty in standing up from squatting position but he managed to come back to bed Heaviness progressed proximally so that he felt difficulty in moving his both lower limbs but some movement was possible with effort at that time. B/L involvement was in symmetric manner. Then weakness progressed to trunk in the form of inability to sit unsupported Patient became bed bound in next 3 Hrs Initially there was no heaviness/weakness in both upper limbs After 3 Hrs of onset, he felt weakness in both upper limbs , B/L symmetric, started distally , rapidly progressing proximally. Rapidity of progression of weakness was so much that in next 12 Hrs he was totally unable to move his all four limbs including inability to change posture on bed. No history s/o bowel bladder, sensory, higher mental function, cranial nerve involvement No H/O respiratory distress No H/O fever at onset and within last one month before illness trauma, back pain, neck pain loose stools, vomiting, heavy carbohydrate intake, strenuous physical activity Patient was complaining of weight loss of almost 10 kgs in last 3 months. Appetite was good No H/O

Fever for prolonged duration, cough with expectoration, night sweats Polyphagia, polydipsia, polyuria, binge eating, induced vomiting Diarrhoea with pain abdomen, bloating Joint pain, oral ulceration, Swelling in any part of body Heat or cold intolerance, skin changes, frequent palpitation, tremor, No H/O similar kind of illness in the past No H/O any chronic drug intake. No family member suffered from similar kind of illness No H/O DM, hypertension, TB in any family member No H/O any chronic medication in any family member. He was born out of a non-consanguineous marriage, perinatal period was uneventful, immunization completed as per protocol He takes mixed diet No addictions to tobacco or alcohol Bowel habit : 2-3/ day formed stools Bladder habit : WNL Sleep, appetite: WNL Patient was not on any chronic medication No H/O drug allergy reported 24 years male patient presented with 1st episode of acute onset rapidly progressive ascending type of motor quadriparesis without any feature S/O cranial nerve, bulbar involvement, sensory, bowel bladder involvement without any significant family history, past history with significant weight loss

Considering the pattern of involvement and rapidity of progression we kept the following probable etiological diagnosis:

- AIDP
- Metabolic abnormality (hypo / hyperkalaemia)
- Spinal artery syndrome
- Other demyelinating diseases i.e. transverse myelitis, 1<sup>st</sup> episode of MS spectrum

Keeping in mind the history of significant weight loss with good appetite we kept another two possibilities:

- Thyroid disorder related or unrelated to paralytic episode
- Diabetes mellitus unrelated to paralytic episode

Other possibilities like compressive cervical myelopathy, CV junction anomalies, toxin

induced were readily excluded by the above-mentioned history

#### **General examination findings:**

Patient was conscious, co-operative and well oriented to time place and person.

General condition- moderate built, Pulse rate- 84 beats/min, regular BP- 126/78 mm Hg, RR- 18/ min, afebrile. Pallor, Cyanosis, Clubbing, Lymphadenopathy, Pedal oedema, skin and nail changes – absent. Icterus-present

A swelling present over anterior aspect of neck, at midline located at thyroid region moving up and down with deglutition but not with protrusion of tongue and of size 4x3 cm, diffuse, non-tender, surface smooth with normal overlying skin, not fixed to underlying structure, no bruit heard over the swelling, suggestive of thyroid gland swelling

Signs suggesting thyrotoxicosis – absent

#### **Systemic examination findings:**

Respiratory system - RR 18/min, SBC-25, bilateral vesicular breath sound presents with no adventitious sounds.

Cardiovascular system- S1S2 heard with no murmurs.

Gastrointestinal system- soft, non-tender, no organomegaly, bowel sound sluggish

Central nervous system

- HMF: WNL
- Cranial nerves: WNL
- Motor system: atonia in all four limbs, power: 0/5 in all 4 limbs, truncal weakness present.
- Sensory: pain & temperature, Joint position, vibration sense was intact
- Reflexes: absent (both superficial and deep)
- Cerebellar signs and gait: could not be tested
- Cranium and spine: WNL

Based on findings of clinical examination we revised our previous etiological diagnoses:

- AIDP
- Metabolic abnormality -hypo / hyperkalaemia related to thyroid swelling

- Other demyelinating disease i.e. 1ST episode of MS

We immediately sent heparinized blood for ABG analysis along with routine investigations i.e. CBC, RFT, LFT, Ca<sup>2+</sup>, phosphate, URINE R/M. TFT in view of thyroid swelling, weight loss was sent ABG report was available in next 10 minutes and revealed following findings:

- ☐ Po<sub>2</sub>-82.6 mmHg
- ☐ Pco<sub>2</sub>-45.6mmHg
- ☐ pH – 7.339
- ☐ Na<sup>+</sup>-136.5 mmol/L
- ☐ Cl<sup>-</sup> - 99.1 mmol/L
- ☐ ica<sup>++</sup> -1.140 mmol/L
- ☐ K<sup>+</sup> - 1.21 mmol/L (3.50-4.50)  
(severe hypokalemia with respiratory acidosis)
- ☐ HCO<sub>3</sub><sup>-</sup> - 23.6 mmol/L
- ☐ AG-15 mmol/L

In view of hypokalaemia as revealed by ABG analysis we started supplementing K<sup>+</sup> both intravenous as well as orally and diagnostic work up of hypokalaemia was started, Patient started improving in next 4 Hrs. Weakness started improving in exactly reverse direction of as it started evolving. Complete recovery was in next 14 - 16 Hrs. In view of indirect hyperbilirubinemia and normal peripheral blood picture with normal serum LDH levels (230 IU/L), congenital unconjugated hyperbilirubinemia was most likely diagnosis, so no further work up was carried out.

Table1: Investigative reports of the patient

Hb	14.3 gm/dl	Urine routine examination findings	
TLC	9500 /cu.mm	Albumin	Nil
DLC	N 53.2 L 32.2 M 11.9	Sugar	Nil
PLT	2.06 lac	RBC	Negative
MCV	91 FL	WBC	3-4
RBS	102 mg/dl	Osmolality	257.79(300-900) mOsm/KgH <sub>2</sub>
Na <sup>+</sup> /k <sup>+</sup>	140/1.32 mmol/L	Na <sup>+</sup> /k <sup>+</sup>	57(40-220)/5.3(25120) mmol/L
Cr/Urea	0.7/19 mg/dl	Cr	2.94 gm/L
SGOT/SGPT	56/ 38 U/L	pH	6
TB/DB	4.3/0.6 mg/dl	URINARY K <sup>+</sup> / Cr	1.008
Ca <sup>2+</sup>	9 mg/dl (8.5-11)		
PO <sub>4</sub>	2.5 mg/dl (2.4-4.1)		
Mg <sup>2+</sup>	1.5 mg/dl (1.7-3.6)		

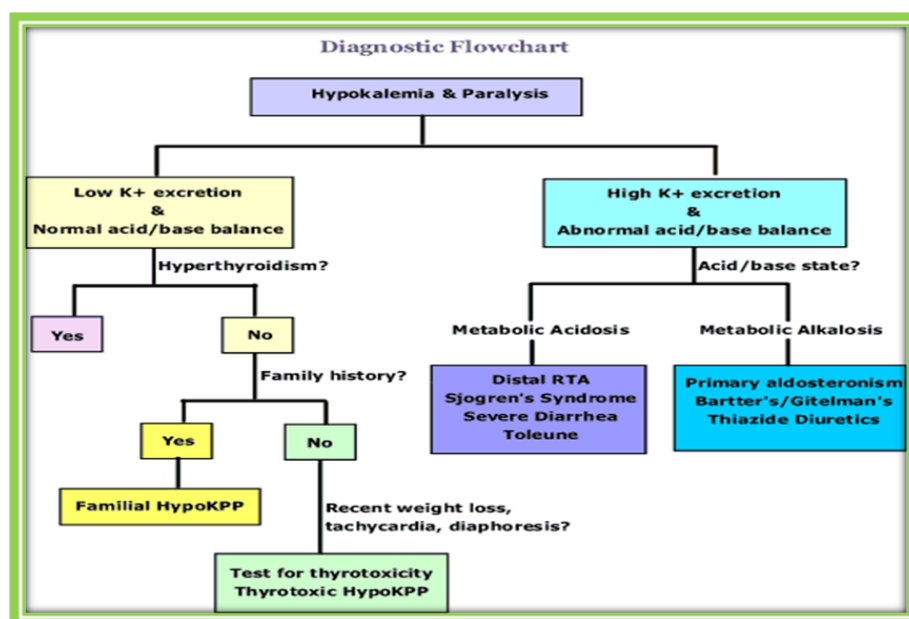


Figure1: diagnostic flow chart for hypokalaemia periodic paralysis( image source-hkpp.org)

Next day we had thyroid function tests report, suggestive of thyrotoxicosis

- FT3-23.5 pmol/l (4-8.3)
- FT4-69.9 pmol/l (10.6-19.40)
- TSH-<0.015 u IU/ml (0.25-5)

Ultrasonography of thyroid gland, blood for anti TPO estimation was sent

Serum Anti TPO levels was 1300 U/ml (N < 60)

Ultrasonography findings: Rt lobe - 2.5 cm AP, Lt lobe - 2.6 cm AP, Isthmus 6 mm AP: B/L lobes bulky and heterogenous with diffuse vascularity, however PSV of ITA are normal. Rt ITA PSV 33.7 cm/sec, Lt ITA PSV 32.4cm/sec, suggestive of diffuse thyroiditis.

**Final diagnosis:** Grave's disease with thyrotoxic hypokalemic paralysis with Gilbert syndrome.

As soon as serum K<sup>+</sup> level reached within normal limit we stopped potassium supplementations to avoid rebound hyperkalaemia. Tab carbimazole was given @ 45 mg/ day in 3 divided doses and Tab propranolol (extended release) was given @ 40 mg/day

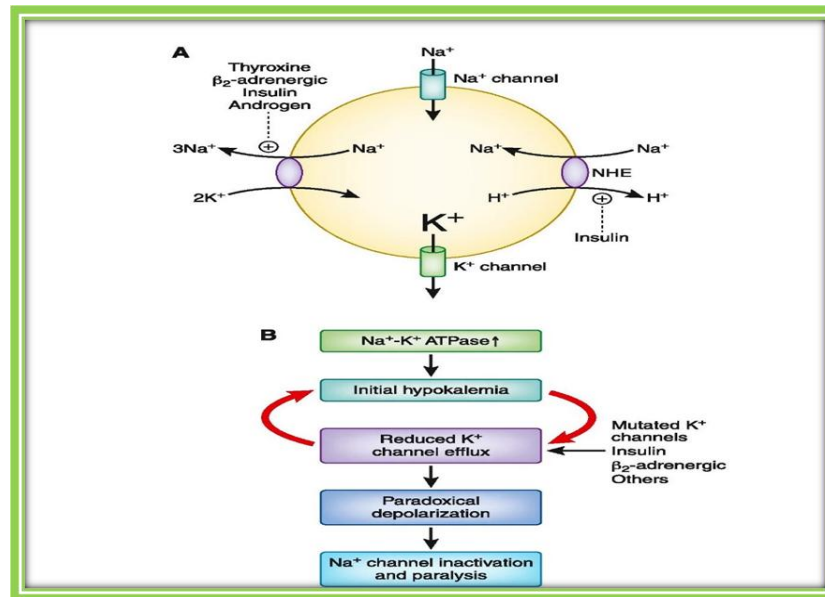
Follow up advice: to avoid heavy carbohydrate meal and strenuous physical activity.

## DISCUSSION

The aetiology of hypokalaemia paralysis can be generally classified into two groups: hypokalaemia paralysis- due to shift of potassium into the intracellular space without a total potassium deficit; due to a large potassium deficit via gastrointestinal or renal loss. Among the hypokalaemia periodic paralysis- Familial hypokalaemia periodic paralysis (FPP) is the most common cause in Western countries. Thyrotoxic periodic paralysis (TPP) is characterized by the triad of acute hypokalaemia without total body potassium deficit, muscle paralysis, and thyrotoxicosis is the most common cause of HPP in Asia with an incidence of approximately 2% in patients with thyrotoxicosis of any cause, in Asia The male to female ratio ranges from 17:1 to 70:1 despite the fact that hyperthyroidism is more common in females (female to male ratio of 9 : 1) Figure 2: comparison of paralytic disorders associated with potassium imbalance.

	Hypokalemic Periodic Paralysis	Thyrotoxic Periodic Paralysis	Hyperkalemic Periodic Paralysis
Age at onset	First or second decade	>20 years	First decade
Attack frequency	Infrequent (a few times a year)	Infrequent	Frequent (up to several a day)
Attack duration	Hours to days	Hours to days	Minutes to hours
Precipitants	Exercise Carbohydrate load Stress	Exercise Carbohydrate load Stress	Exercise Fasting Stress K-rich food
Potassium level during attack	Low	Low	Normal or elevated
Associated features	Later onset myopathy	Symptoms of thyrotoxicosis Low TSH with high T4 or high T3	Myotonia on examination and/or EMG Later onset myopathy
Etiology	Autosomal dominant inherited defect in calcium or sodium ion channel on muscle membrane	Thyrotoxicosis Possible inherited predisposition	Autosomal dominant inherited defect of sodium ion channel on muscle membrane
Penetrance	Nonpenetrance common, especially in women		High
Epidemiology	Clinical expression in men more frequent than women	Highest incidence in Asians and in men more than women	Sexes equally affected
Preventive treatment	Carbonic anhydrase inhibitors Potassium-sparing diuretics	Euthyroid state Propranolol	Carbonic anhydrase inhibitors Thiazide diuretics Inhaled beta-agonists as

Figure 2: comparison of various forms of periodic paralytic disorders associated with potassium imbalance.



**Figure3:** Pathophysiology of thyrotoxic periodic paralysis Lin SH, Huang CL. Mechanism of thyrotoxic periodic paralysis. Vol. 23, Journal of the American Society of Nephrology. American Society of Nephrology; 2012 [cited 2020 Jun 29]. p. 985–8.

## CONCLUSION

TPP has to always be kept in mind while evaluating a case of hypokalaemia paralysis. The subtleness of underlying hyperthyroidism often delays diagnosis. Biochemical hyperthyroidism with normal urinary potassium excretion and ECG changes clinches to diagnosis of TPP. Treatment with low-dose potassium supplements and non-selective beta-blockers should be initiated upon diagnosis with frequent monitoring of serum potassium level to prevent rebound hyperkalaemia.

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How to cite this article: Vadlamani B, Manna S. An interesting case of quadriplegia- thyrotoxic periodic paralysis. *Gal Int J Health Sci Res.* 2020; 5(2): 139-143.

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