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Case Report

Case report: Hyperthyroid hypokalemic periodic paralysis

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ABSTRACT

Introduction and importance: HHPP is a rare type of hypokalemic PP that can occur when there is hyperthyroidism. Thyrotoxic periodic paralysis is due to increased influx of potassium into skeletal muscle cells which leads to profound hypokalemia and paralysis. Insulin and Epinephrine are also responsible for stimulating the Na–K-ATPase pumps which are over expressed during hyperthyroid state. Laboratory hypokalemia in the background of hyperthyroidism with sudden symmetric paralysis point toward the diagnosis.

Case: We present a case of 25 year old male with limb weakness for 3hours following heavy dinner.He felt weakness after waking up in the morning where he could not move his both lower limbs. He also had difficulty moving upper limbs.

Clinical findings and investigations: Examination revealed proximal muscle weakness with power of 2/5, decreased muscle tone, diminished deep tendon reflexes in all four limbs and equivocal plantar reflex bilaterally. Investigation sent were Total Leukocyte count, Hemoglobin, Renal function test, Liver Function test,Thyroid function test, Vitamin B12, Serology, ACTH, Serum calcium, Serum phosphate, Serum magnesium, Urine R/ME and Stool R/ME.

Intervention and outcome: Patient is treated with 10mEq/L/hr infusion of potassium chloride, methimazole and beta-blockers. He is stable and is in regular followup in medicine OPD.

Relevance and impact: Early diagnosis of HHPP is very essential to prevent fatal complications (cardiac and respiratory).

It can be treated by timely potassium supplementation, methimazole and beta-blockers.

Clinicians must be concerned about Hyperkalemia while supplementing Potassium in bed side.

1. Introduction

1.1. Background and rationale

Hypokalemic periodic paralysis (HPP) is a life-threatening complication of hyperthyroidism which is characterized by episodes of acute muscle weakness due to hypokalemia. Thyrotoxic periodic paralysis is rare complication of hyperthyroidism where increased influx of potassium into skeletal muscle cells leads to profound hypokalaemia and paralysis. Most cases are sporadiac in Asians; however, reports are increasing in Caucasians. It is regarded as a channelopathy in which genetic and/or acquired defect in the sodium-potassium (Na/K-ATPase) pump renders it more sensitive to excess thyroid hormone in susceptible individuals.

2. Guidelines: SCARE 2020 paper

This case report has been reported in line with the SCARE Criteria [1].

We present a case of 25 year old male with acute onset symmetric muscle paralysis following heavy dinner.

3. Case presentation

3.1. Patient information: demographics and presentation

A 25 year old man,student by occupation,married, hindu by religion, presented to our emergency department (ER) in Scooter due to limb weakness for 3hours. He felt weakness after waking up in the morning where he could not move his both lower limbs. He also had difficulty

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moving upper limbs. He had heavy meal in dinner the earlier day. He denied having respiratory distress, palpitation, blurred vision, slurred speech, difficulty swallowing, or easy choking. He had normal bowel and bladder habit and normal appetite and sleep.

3.1.1. Past medical and surgical history

He had similar episode 1 week ago where he was treated with oral potassium. He had no significant medical or surgical history.

3.1.2. Drug and allergy history

No h/o of long term medication and no known allergies till date.

3.1.3. Family history

He denied history of similar illness in family as well.

3.1.4. Social history

Doesn't consume alcohol, Non Smoker.

3.1.5. Clinical findings

Patient was anxious and his vital sign upon arrival were normal with body temperature of 97.5 F, heart rate of 95 beats per minute, respiratory rate of 15 times per minute, and blood pressure of 120/80 mm Hg. Neurological examination revealed proximal muscle weakness with power of 2/5, decreased muscle tone, diminished deep tendon reflexes in all four limbs and equivocal plantar reflex bilaterally. His higher mental functions, sensory system, and cranial nerve were intact. However Cerebellar functions could not be assessed because of paralysis.

3.1.6. Diagnostics assesment and interpretation

All the relevant investigations (Table 1) were sent required for the diagnosis.

3.1.7. Diagnostic reasoning

Differential diagnosis of acute quadriparesis can be myasthenic crisis, Guillain-Barré syndrome, acute myelopathy (eg, transverse myelitis), acute thyrotoxic myopathy, tick paralysis, and botulism in an acute attack.

Rapid onset paralysis with normal mental status is usually caused by a lesion in the cervical spine, anterior horn cell, peripheral nerve, neuromuscular junction, or muscle [2] While acute spinal cord disorders are commonly caused by compression or ischemia, these etiologies are less likely when reflexes are normal [2].

Similarly, preserved reflexes combined with intact sensation reduce the likelihood of a peripheral nerve localization. A syndrome of anterior horn cell disease, such as West Nile virus infection, manifests as pure motor weakness. The weakness, on the other hand, is generally asymmetric and distal, and the onset is less abrupt.

Myasthenia gravis is one of the neuromuscular disorders that can present acutely, but our patient's lack of ocular and bulbar involvement made this diagnosis less likely [3].

Finally, in this patient's case, proximal muscle weakness combined with preservation of reflexes and sensation made myopathy the primary consideration.

Table 1
Summary of laboratory test.

Laboratory test	Patient level	Reference range
Total Leukocyte count	6000/cmm	4000–1100
Hemoglobin	15.1 gm%	12–18
Blood Sugar (random)	4.7mmol/L	3.8–7.8
Urea	5.5mmol/L	1.6–7.8
Creatinine	51uMol/L	60–115
Sodium	142mEq/L	135–146
Total Bilirubin	25uMol/L	3–21
Direct Bilirubin	8 uMol/L	4
ALT	102U/L	<42
AST	36 U/L	
ALP	98 U/L	30–90
Total protein	75g/L	60–80
Albumin	44g/L	38–49
Gamma GT	49U/L	11–50
CPK Nac	120U/L	<170
Serology	Non-reactive	
fT3	46.08pmol/L	2.62–5.69
fT4	34.66pmol/L	9.0–19.0
FSH	0.001uIU/L	0.35–4.94
Vitamin B12	383pg/mL	187–833
ACTH	32.30pg/mL	<46
Early morning cortisol	14.6ug/dL	5–25
eGFR	>90mL/min/1.72m2	
Serum Calcium	2.25mmol/L	2.15–2.55
Serum Phosphate	0.92mmol/L	0.8–1.4
Serum Magnesium	0.81mmol/L	0.7–1
Urine and stool routine	Nothing abnormal detected	

AST: Aspartate transaminase, ALT: Alanine transaminase, ALP:Alkaline Phosphatase, Gamma GT: Gamma-Glutamyltransferase, CPK Nac: Creatinine Kinase activated by N-Acety Cysteine, fT3: free triiodothyronine, fT4: free thyroxine, TSH: thyroid stimulating hormone.

3.2. Intervention

In the view of his history and laboratory investigations, the provisional diagnosis of hypokalemic periodic paralysis was made. He was treated with 10mEq/L/hr infusion of potassium chloride until normalized serum potassium level. Patient was kept in cardiac monitoring and serum potassium was monitored till 24 hours to prevent rebound hyperkalemia. Patient was admitted and his K level was routinely monitored (Fig. 1). Next day patient was prescribed with Acetazolamide 250mg bd while awaiting thyroid function test and thyroid scan. These showed raised serum free triiodothyronine(fT3) of 46.08pmol/L (RR: 2.62–5.69pmol/L) and free thyroxine (fT4) of 34.66pmol/L (RR: 9.0–19.0 pmol/L) and suppressed thyroid stimulating hormone (TSH) of 0.001 micoIU/mL (RR:0.35–4.94 micoIU/mL).

His nerve conduction study was also normal. 99 m TcPertechnitate Thyroid Scintigraphy revealed increased homogenous tracer uptake in both lobes of thyroid gland. Thyroid gland appeared mildly enlarged in size with suppressed background and salivary gland activity. Right lobe uptake 2.7% and left lobe uptake 2.4% of radioactive substance with total percentage uptake of 5.1% (RR of total percentage uptake:0.5–4%) (Fig. 2)

Based on clinical, laboratory and scan and uptake finding diffuse toxic goiter was found and the condition was diagnosed as Hyperthyroid Hypokalemic Periodic Paralysis. Patient is now on methimazole 10mg once a day and propranolol 40mg bd. Patient is stable now.

3.3. Intervation settings and name of clinician

Bhakunde Hospital, Kavrepalanchowk, NepalDr. Prakash poudel jaishi, Department of medicine, Bhakunde Hospital.

3.4. Followups

Patient is under regular follow up (initially on evry 2 weeks and now in every 3–4 months with Thyroid function test and electrolyte (potassium and sodium) reports in endocrinology department.

4. Discussion

Periodic paralysis (PP) is a muscle disease in the channelopathies family, characterized by episodes of painless muscle weakness. These episodes can be triggered by strenuous exercise, fasting, or consuming high-carbohydrate foods.

In contrast to other types of thyroid disease, which are more common in women, Hyperthyroid Hypokalemic Periodic Paralysis (HHPP) is much more common in men; it is also more common in Asians [4,5]. This diagnosis should be considered when this patient demographic

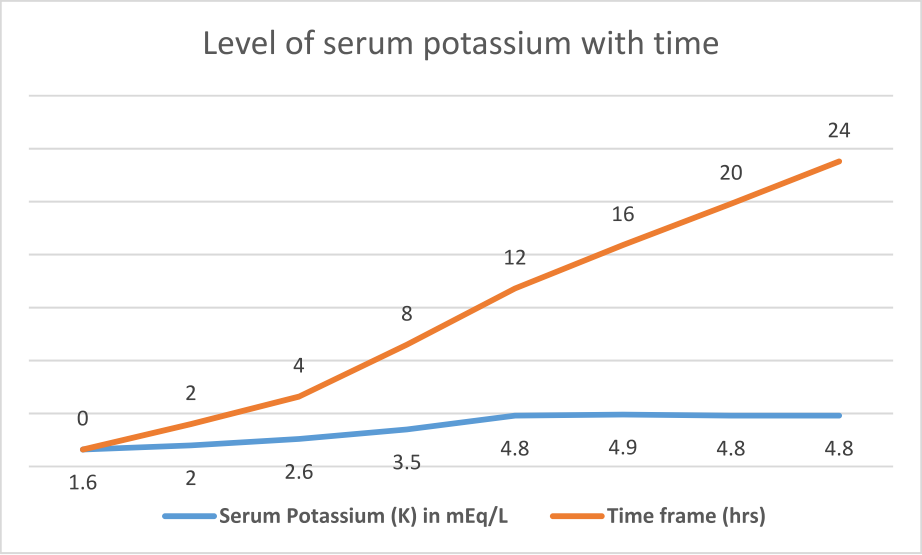

Fig. 1. Line graph showing level of potassium over time.

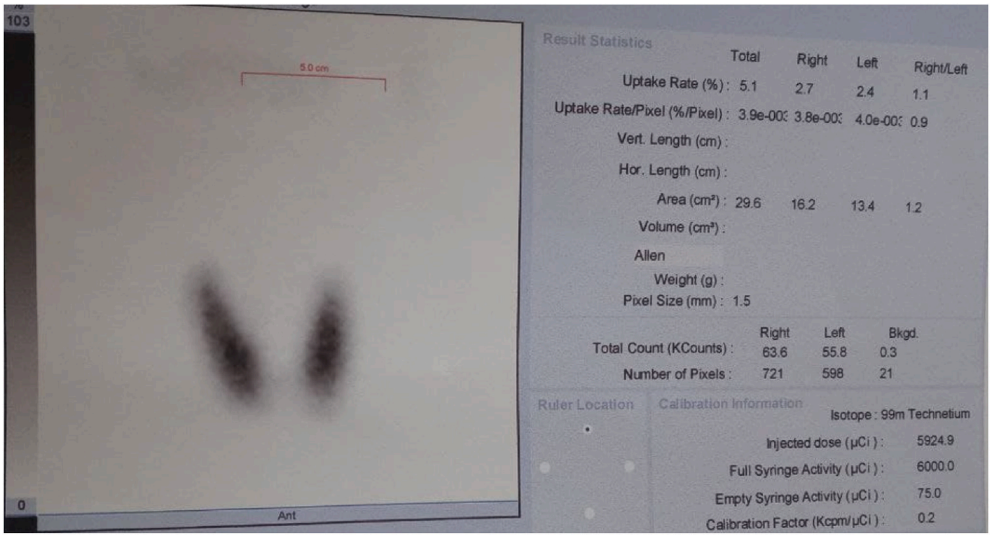

Fig. 2. Figure above showing Radio iodine uptake in right lobe (2.7%) and left lobe(2.4%).

presents with marked painless weakness after provocation with exercise or dietary changes [6].

HHPP is a rare type of hypokalemic PP that can occur when there is hyperthyroidism. Familial hypokalemic PP, on the other hand, has an autosomal dominant inheritance pattern [4].

Graves' disease, which is also the leading cause of hyperthyroidism worldwide, is the cause of hyperthyroidism in the majority of HHPP cases [7]. HHPP, on the other hand, may be linked to other causes of hyperthyroidism, such as functional thyroid nodules, toxic multinodulargoitre, and factitious thyrotoxicosis [8].

The majority of HHPP cases occur in Asians on a sporadic basis; however, it is becoming more common in Caucasians. According to studies, hyperthyroidism affects 2% of Asians compared to 0.1–0.2% of non-Asians [9,10]. Despite the fact that women have a higher incidence of hyperthyroidism, men account for more than 95% of HHPP cases [11,12]. The male to female ratio varies by ethnic group, ranging from 17:1 to 76:1 [9,13,14].

The mechanism by which hyperthyroidism can result in hypokalemic PP is unknown. Thyroid hormone increases tissue responsiveness to beta-adrenergic stimulation, which increases sodium-potassium ATPase activity on the skeletal muscle membrane along with thyroid hormone [15]. This causes potassium to enter cells, possibly resulting in hyperpolarization of the muscle membrane and relative inexcitability of the muscle fibers. Patients with PP who are thyrotoxic have higher sodium pump activity than those who do not have paralytic episodes [16,17]. Excess thyroid hormone may predispose to paralytic episodes by increasing susceptibility to epinephrine or insulin's hypokalemic action [18,19].

Insulin also activates the sodium-potassium ATPase pump and, in conjunction with thyroid hormone, may act synergistically to drive potassium into cells. This is consistent with our observation that a heavy meal can precipitate HHPP attacks in our patient.

While patients with HHPP do not have the genetic mutations associated with familial hypokalemic PP, it has been proposed that those who are susceptible to HHPP may have an ion channel defect that, in the euthyroid state, is insufficient to cause symptoms [20,21]. Several studies have identified specific susceptibility loci that confer HHPP risk. Many of these have an effect on KCNJ2 expression, but not all; this gene encodes Kir2.6, an inwardly rectifying potassium channel expressed in skeletal muscle and transcriptionally regulated by thyroid hormone [22,

23]. Some of these loci, but not all, have been linked to Graves' disease [24]. The frequency of these genes varies across ethnic groups, which may explain why HHPP is more prevalent in certain ethnic groups.The predominance of this condition in men and the demonstration that testosterone increases sodium-potassium ATPase activity in animals both point to a role for testosterone in the pathogenesis of HHPP [25].

Attacks of weakness, like all of the PPs, occur suddenly, with generalized weakness and preserved consciousness. In approximately 80% of thyrotoxic PP patients, the age of onset of symptoms is between 20 and 39 years [12,18]. Although the later age of onset helps differentiate HHPP from familial PPs, which present at a younger age, adolescents with HHPP have been reported [26,27].

The clinical features of hyperthyroidism can appear months or even years before the onset of periodic paralysis, but they can also appear concurrently or after the onset of periodic paralysis [28,29]. The acute attack first affects the lower limbs, then the girdle muscles, and finally the upper limbs [30]. Although the respiratory muscles are rarely involved, total paralysis of the respiratory, bulbar, and ocular muscles has been reported in a severe attack [31]. Mild myalgia affects less than half of all patients [12]. Although normal or hyperactive reflexes may be observed, decreased muscle tone with hyporeflexia or areflexia is typical. Tachycardia was noted at presentation in one series, distinguishing these patients from those with familial HHPP [29].

Attacks vary in frequency and duration, as well as their association with inciting events.Intervals of weeks to months are common, but some patients experience several attacks per week [12,32]. Duration of symptoms for several hours is typical, but can range from minutes to days. As with hypokalemic PP, attacks in HHPP can be precipitated by events that are associated with an increased release of epinephrine or insulin, both of which cause movement of potassium into cells and low potassium blood levels [12,21]. Most commonly, the precipitating event is either rest after strenuous physical activity, stress, or a high-carbohydrate load. Other events reported to induce attacks in HHPP include cold exposure, infection, alcohol intake, pulse corticosteroid therapy, beta-2 adrenergic bronchodilator use, and menses [18, 28]. In many instances, no obvious precipitating factor is identified.

Although weakness attacks can occur at any time of day, HHPP patients have a higher frequency of attacks at night or early in the morning [33]. A seasonal variation has also been proposed, with more attacks occurring during the summer months [34].

During an attack, the degree of hypokalemia varies; in one series of 78 patients, the mean serum potassium level was 2.1 mmol/L [35]. However, the ictal potassium level in some TPP was normal [12,36]. Typically, the degree of hypokalemia corresponds to the severity of weakness [37]. Laboratory findings include an increase in serum thyroxine (T4) and a decrease in thyrotropin levels (TSH). There have been reports of patients with elevated T3 levels but normal T4 levels [38].Other common laboratory findings include a mild decrease in serum phosphate and magnesium levels, which differentiate TPP from Familial hypokalemic periodic paralysis with a high calcium to phosphorus ratio [39]. Creatine kinase levels may be normal, but they have been found to be mildly elevated in two-thirds of patients, and rhabdomyolysis has been reported [33,40].

Electrocardiogram (ECG) changes are common during a thyrotoxic PP attack. These include ST depression, sinus tachycardia, and U waves, as well as those that are not consistently associated with hypokalemia: an elevated higher heart rate, abnormal PR interval, higher QRS voltage, and first-degree atrioventricular (AV) block [41,42].

In patients with thyrotoxic PP, the latter category of ECG findings is more common than in patients with familial hypokalemic PP [43]. Severe arrhythmias (for example, sinus arrest, second-degree AV block, ventricular fibrillation, and ventricular tachycardia) are uncommon but are documented [44].

Hyperthyroid Hypokalemic Periodic Paralysis (HHPP) must be distinguished from other causes of acute quadriparesis, such as myasthenic crisis, Guillain-Barré syndrome, acute myelopathy (eg, transverse myelitis), acute thyrotoxic myopathy, tick paralysis, and botulism in an acute attack. Rapid onset paralysis with normal mental status is usually caused by a lesion in the cervical spine, anterior horn cell, peripheral nerve, neuromuscular junction, or muscle [2] While acute spinal cord disorders are commonly caused by compression or ischemia, these etiologies are less likely when reflexes are normal [2]. Similarly, preserved reflexes combined with intact sensation reduce the likelihood of a peripheral nerve localization. A syndrome of anterior horn cell disease, such as West Nile virus infection, manifests as pure motor weakness. The weakness, on the other hand, is generally asymmetric and distal, and the onset is less abrupt. Myasthenia gravis is one of the neuromuscular disorders that can present acutely, but our patient's lack of ocular and bulbar involvement made this diagnosis less likely [3] Finally, in this patient's case, proximal muscle weakness combined with preservation of reflexes and sensation made myopathy the primary consideration.

The presence of hypokalemia and subsequent recovery with treatment generally alerts the clinician to the diagnosis of hypokalemic PP, in which the possibility of thyrotoxicosis must always be considered, especially in the absence of a PP family history.

Following the establishment of a thyrotoxic state, the patient is further evaluated to determine the underlying cause. Electromyography, provocative testing, and muscle biopsy results are comparable to those seen in familial hypokalemic PP, but these tests are frequently unnecessary. If thyrotoxicosis is not found to be the cause, an electrocardiogram (ECG) should be performed to look for a prolonged QT or QU interval, which could indicate Andersen syndrome. Acute paralysis patients are usually hospitalized in a monitored setting for cardiac arrhythmias as well as dysphagia.

A recommended protocol is to take 30 mEq of oral potassium every 2 h until improvement is noticed, with a maximum dose of 90 mEq in 24 hours [45]. Slower administration rates (10 mEq per hour) are advocated by some [15]. Patients with severe hypokalemia or impaired swallowing may require intravenous supplementation.In a case series, patients who received intravenous potassium recovered faster than those who received oral potassium supplementation [46].

Rebound hyperkalemia appears to be a significant issue in HHPP, occurring in 40–59% of treated attacks [33,35]. In one study, more than 90 mEq of potassium was given to 80% of patients who developed rebound hyperkalemia within 24 hours. In one case series, one-fourth of patients experienced a drop in serum potassium during initial treatment; these patients also had higher free thyroxine levels, required higher doses of potassium supplementation, and were more likely to have severe rebound hyperkalemia [35].

During acute paralysis, serum potassium levels must be closely monitored. During treatment and observation, all patients should have continuous cardiac monitoring. Severe arrhythmias/electrocardiogram (ECG) changes should be consulted with the cardiology. Correcting hypomagnesemia, if present, is also advised.

Potassium replacement alone may not be enough to stop an attack. By reducing the activation of the Na–K-ATPase pump, intravenous propranolol has been shown to correct weakness and hypokalemia in patients with HHPP that is unresponsive to potassium therapy [47].

The restoration of euthyroidism eliminates thyrotoxic PP attacks [18, 48]. The electromyography (EMG) exercise test normalizes in euthyroid patients, and attacks are no longer inducible [49]. If thyrotoxicosis recurs, HHPP may resurface. The treatment of hyperthyroidism varies depending on the underlying cause. Treatment with radioactive iodine or surgery found to be more effective in preventing relapses than treatment with antithyroid medications alone in a 14-year study of 16 individuals with HHPP secondary to Graves' disease [50].

Beta-blocking drugs, such as propranolol, have also been proven to reduce the frequency and severity of episodes when given with or without potassium supplements, and may be used as a temporary therapy until a euthyroid condition is established. Beta-1 selective drugs are less likely to suppress the beta-2 receptor-mediated hypokalemic action of adrenaline, and so are less likely to prevent paralytic episodes [18].

Carbonic anhydrase inhibitors have not been demonstrated to be beneficial in thyrotoxic PP, in contrast to familial hypokalemic PP, and may even increase the frequency of attacks [51]. Potassium supplementation is ineffective as a prophylactic measure in patients with thyrotoxic PP [20]. Precipitating factors, such as excessive activity, high-carbohydrate meals, and alcohol, should be avoided, just as they should be avoided in hypokalemic PP.

4.1. Take away lesson

So, the early diagnosis of HHPP is very essential to prevent fatal complications (cardiac and respiratory) and to prevent rebound hyperkalemia due to aggressive electrolyte replacement. Many cases in Nepal following normal vitals and physical examination diagnosed with conversion disorder could be HPPP.

Ethical approval

None.

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Author contribution

Prakash Paudel Jaishi,Department of Medicine, Bhakunde Hospital, Kavrepalanchowk, Nepal: First author (Guarantor). He has presented the case. Sandhya kiran Neupane,Department of Medicine, Shadhak Polyclinic, Bhaktapur, Nepal. Second Author: she has done all the discussion part. Prabhat Kiran Neupane, Internship at Department of Medicine, Kist Medical College, Kathmandu. Third Author: Editor of the article.

Informed consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Consent

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Registration of research studies

Name of the registry:
Unique Identifying number or registration ID:
Hyperlink to your specific registration (must be publicly accessible and will be checked):

Guarantor

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Declaration of competing interest

There is no any conflicts of interest with this article.

Appendix A. Supplementary data

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References

[1] R.A. Agha, T. Franchi, C. Sohrabi, G. Mathew, for the SCARE Group, The SCARE 2020 guideline: updating consensus surgical CAse REport (SCARE) guidelines, Int. J. Surg. 84 (2020) 226–230.
[2] R. Nayak, Practical approach to the patient with acute neuromuscular weakness, World J Clin Cases 5 (2017) 270–279.
[3] A. Jacob, B.G. Weinshenker, An approach to the diagnosis of acute transverse myelitis, Semin. Neurol. 28 (2008) 105–120.
[4] A. Vijayakumar, G. Ashwath, D. Thimmappa, Thyrotoxic periodic paralysis: clinical challenges, J. Thyroid Res. (2014), 649502, https://doi.org/10.1155/2014/649502, 2014, Retraction published J Thyroid Res. 2021 Mar 16;2021:7814159).
[5] M.A. Chaudhry, S. Wayangankar, Thyrotoxic periodic paralysis: a concise review of the literature, Curr. Rheumatol. Rev. 12 (3) (2016) 190–194, https://doi.org/10.2174/1573397112666160404124822.
[6] H. Patel, L.V. Wilches, J. Guerrero, Thyrotoxic periodic paralysis: diversity in America, J. Emerg. Med. 46 (6) (2014) 760–762, https://doi.org/10.1016/j.jemermed.2013.08.104.
[7] J.S. Amberger, C.A. Bocchini, A.F. Scott, A. Hamosh, OMIM.org: leveraging knowledge across phenotype-gene relationships, Nucl. Resist. 47 (2019) D1038–D1043.
[8] R.K. Balakrishnan, S.R. Chandran, G. Thirumalnesan, N. Doraisamy, Thyrotoxic periodic paralysis, Indian J Endocrinol. Metabol. 15 (Suppl 2) (2011) S147–S149.
[9] D.E. Kelley, H. Gharib, F.P. Kennedy, R.J. Duda Jr., P.G. McManis, Thyrotoxic periodic paralysis. Report of 10 cases and review of electromyographic findings, Arch. Intern. Med. 149 (1989) 2597–2600.
[10] A.J. McFadzean, R. Yeung, Periodic paralysis complicating thyrotoxicosis in Chinese, Br. Med. J. 1 (1967) 451–455.
[11] G.T. Ko, C.C. Chow, V.T. Yeung, H.H. Chan, J.K. Li, C.S. Cockram, Thyrotoxic periodic paralysis in a Chinese population, QJM : Mon. J. Assoc. Phys. 89 (6) (1996) 463–468, https://doi.org/10.1093/qjmed/89.6.463.
[12] M.J. Hsieh, R.K. Lyu, W.N. Chang, K.H. Chang, C.M. Chen, H.S. Chang, Y.R. Wu, S. T. Chen, L.S. Ro, Hypokalemic thyrotoxic periodic paralysis: clinical characteristics and predictors of recurrent paralytic attacks, Eur. J. Neurol. 15 (6) (2008) 559–564.
[13] K.P. Ober, Thyrotoxic periodic paralysis in the United States. Report of 7 cases and review of the literature, Medicine (Baltim.) 71 (1992) 109–120.
[14] R. Ghose, G. Quail, R. king, P. Maloney, Hypokalemic paralysis in remote aboriginal communities, AustFamPhysiacian 25 (1996) 1172–1173.
[15] E.P. Rhee, J.A. Scott, A.S. Dighe, Case records of the Massachusetts General Hospital. Case 4-2012. A 37-year-old man with muscle pain, weakness, and weight loss, N. Engl. J. Med. 366 (6) (2012) 553–560, https://doi.org/10.1056/NEJMcpc1110051.
[16] A. Pompeo, A. Nepa, M. Maddestra, V. Feliziani, N. Genovesi, Thyrotoxic hypokalemic periodic paralysis: an overlooked pathology in western countries, Eur. J. Intern. Med. 18 (5) (2007) 380–390, https://doi.org/10.1016/j.ejim.2007.03.003.
[17] A. Chan, R. Shinde, C.C. Chow, C.S. Cockram, R. Swaminathan, In vivo and in vitro sodium pump activity in subjects with thyrotoxic periodic paralysis, BMJ (Clin. Res. ed. 303 (1991) 1096–1099, https://doi.org/10.1136/bmj.303.6810.1096, 6810.
[18] K.P. Ober, Thyrotoxic periodic paralysis in the United States. Report of 7 cases and review of the literature, Med. 71 (3) (1992) 109–120, https://doi.org/10.1097/00005792-199205000-00001.
[19] S.H. Lin, Y.F. Lin, Propranolol rapidly reverses paralysis, hypokalemia, and hypophosphatemia in thyrotoxic periodic paralysis, Am. J. Kidney Dis. : Off J. Natl. Kidney Found. 37 (3) (2001) 620–623.
[20] A.W. Kung, Clinical review: thyrotoxic periodic paralysis: a diagnostic challenge, J. Clin. Endocrinol. Metabol. 91 (7) (2006) 2490–2495, https://doi.org/10.1210/jc.2006-0356.
[21] B. Fontaine, P. Lapie, E. Plassart, N. Tabti, S. Nicole, J. Reboul, C.S. Rime-Davoine, Periodic paralysis and voltage-gated ion channels, Kidney Int. 49 (1) (1996) 9–18, https://doi.org/10.1038/ki.1996.2.
[22] D.P. Ryan, M.R. Dias Da Silva, T.W. Soong, B. Fontaine, M.R. Donaldson, A. W. Kung, W. Jongjaroenprasert, M.C. Liang, D.H. Khoo, J.S. Cheah, S.C. Ho, H. S. Bernstein, R.M. Maciel, R.H. Brown, L.J. Ptáček, Mutations in potassium channel Kir2.6 cause susceptibility to thyrotoxic hypokalemic periodic paralysis, Cell 140 (1) (2010) 88–98, https://doi.org/10.1016/j.cell.2009.12.024.
[23] W. Jongjaroenprasert, S. Chanprasertyotin, S. Butadej, S. Nakasatien, N. Charatcharoenwitthaya, T. Himathongkam, B. Ongphiphadhanakul, Association of genetic variants in GABRA3 gene and thyrotoxichypokalaemic periodic paralysis in Thai population, Clin. Endocrinol. 68 (4) (2008) 646–651, https://doi.org/10.1111/j.1365-2265.2007.03083.x.
[24] S. Zhao, W. Liu, J. Liang, et al., Assessment of molecular subtypes in thyrotoxic periodic paralysis and graves disease among Chinese han adults: a population-based Genome-wide association study, JAMA Netw. Open 2 (5) (2019), e193348, https://doi.org/10.1001/jamanetworkopen.2019.3348.

[25] H.A. Tran, G.E. Reeves, Hepatitis C infection and thyrotoxic periodic paralysis–a novel use of an old drug, Am. J. Med. Sci. 336 (6) (2008) 515–518, https://doi.org/10.1097/MAJ.0b013e3181643e3d.
[26] C. Schalin-Jantti, T. Laine, K. Valli-Jaakola, T. Lonnqvist, K. Kontula, M. J. Valimaki, Manifestation, management and molecular analysis of candidate genes in two rare cases of thyrotoxic hypokalemic periodic paralysis, Horm. Res. 63 (3) (2005) 139–144, https://doi.org/10.1159/000084689.
[27] G.W. Wong, T.F. Leung, A.F. Lo, A.T. Ahuja, P.S. Cheng, Thyrotoxic periodic paralysis in a 14-year-old boy, Eur. J. Pediatr. 159 (12) (2000) 934, https://doi.org/10.1007/pl00008375.
[28] P. Wong, Hypokalemic thyrotoxic periodic paralysis: a case series, CJEM 5 (5) (2003) 353–355.
[29] S.H. Lin, Y.F. Lin, M.L. Halperin, Hypokalaemia and paralysis, QJM : Mon. J. Assoc. Phys. 94 (3) (2001) 133–139.
[30] Thyrotoxic periodic paralysis: a diagnostic challenge, Kung AW. J Clin. Endocrinol. Metab. 91 (2006) 2490–2495 [PubMed] [Google Scholar] [Ref list].
[31] Y.-C. Liu, W.-S. Tsai, T. Chau, S.-H. Lin, Acute hypercapnic respiratory failure due to thyrotoxic periodic paralysis, Am. J. Med. Sci. 327 (2004) 264–267 [PubMed] [Google Scholar] [Ref list].
[32] S.L. Venance, S.C. Cannon, D. Fialho, B. Fontaine, M.G. Hanna, L.J. Ptacek, M. Tristani-Firouzi, R. Tawil, R.C. Griggs, C.I.N.C.H. investigators, The primary periodic paralyses: diagnosis, pathogenesis and treatment, Brain : J. Neurol. 129 (Pt 1) (2006) 8–17, https://doi.org/10.1093/brain/awh639.
[33] M.A. Manoukian, J.A. Foote, L.M. Crapo, Clinical and metabolic features of thyrotoxic periodic paralysis in 24 episodes, Arch. Intern. Med. 159 (6) (1999) 601–606, https://doi.org/10.1001/archinte.159.6.601.
[34] A.J. McFadzean, R. Yeung, Periodic paralysis complicating thyrotoxicosis in Chinese, Br. Med. J. 1 (5538) (1967) 451–455, https://doi.org/10.1136/bmj.1.5538.451.
[35] J.C. Shiang, C.J. Cheng, M.K. Tsai, Y.J. Hung, Y.J. Hsu, S.S. Yang, S.J. Chu, S. H. Lin, Therapeutic analysis in Chinese patients with thyrotoxic periodic paralysis over 6 years, Eur. J. Endocrinol. 161 (6) (2009) 911–916, https://doi.org/10.1530/EJE-09-0553.
[36] C.C. Wu, T. Chau, C.J. Chang, S.H. Lin, An unrecognized cause of paralysis in ED: thyrotoxicnormokalemic periodic paralysis, Am. J. Emerg. Med. 21 (2003) 71.
[37] J. Li, X.B. Yang, Y. Zhao, Thyrotoxic periodic paralysis in the Chinese population: clinical features in 45 cases, Exp. Clin. Endocrinol. Diabetes : Off. J, Ger. Soc. Endocrinol. Ger. Diabetes Assoc. 118 (1) (2010) 22–26, https://doi.org/10.1055/s-0028-1112150.
[38] J.M. Statland, B. Fontaine, M.G. Hanna, N.E. Johnson, J.T. Kissel, V.A. Sansone, P. B. Shieh, R.N. Tawil, J. Trivedi, S.C. Cannon, R.C. Griggs, Review of the diagnosis and treatment of periodic paralysis, Muscle Nerve 57 (4) (2018) 522–530, https://doi.org/10.1002/mus.26009.
[39] S.H. Lin, P. Chu, C.J. Cheng, S.J. Chu, Y.J. Hung, Y.F. Lin, Early diagnosis of thyrotoxic periodic paralysis: spot urine calcium to phosphate ratio, Crit. Care Med. 34 (12) (2006) 2984–2989, https://doi.org/10.1097/01.CCM.0000242249.10856.49.
[40] S. Summachiwakij, I. Sachmechi, Rhabdomyolysis induced by nonstrenuous exercise in a patient with graves' disease, Case Rep. Endocrinol. (2014), 286450, https://doi.org/10.1155/2014/286450, 2014.
[41] Z.D. Goldberger, Images in cardiovascular medicine. An electrocardiogram triad in thyrotoxic hypokalemic periodic paralysis, Circulation 115 (6) (2007) e179–e180, https://doi.org/10.1161/CIRCULATIONAHA.106.652396.
[42] S. Lopez, S.O. Henderson, Electrocardiogram changes in thyrotoxic periodic paralysis, West. J. Emerg. Med. 13 (6) (2012) 512–513, https://doi.org/10.5811/westjem.2011.11.12127.
[43] Y.J. Hsu, Y.F. Lin, T. Chau, J.T. Liou, S.W. Kuo, S.H. Lin, Electrocardiographic manifestations in patients with thyrotoxic periodic paralysis, Am. J. Med. Sci. 326 (3) (2003) 128–132, https://doi.org/10.1097/00000441-200309000-00004.
[44] H.F. Wang, S.C. Tsai, M.S. Pan, C.C. Shiao, Complete heart block during potassium therapy in thyrotoxic periodic paralysis, J. Emerg. Med. 44 (1) (2013) 61–64, https://doi.org/10.1016/j.jemermed.2011.05.090.
[45] P.P.B. Yeo, K.O. Lee, J.S. Cheah, Thyrotoxic periodic paralysis: a study of 51 patients, in: Proceedings of the Second Congress of the Association of Southeast Asian Nations (ASEAN) Federation of Endocrine Societies, Bangkok, Thailand, 1983. Nov 30 - Dec 3.
[46] M. Cesur, F. Bayram, M.A. Temel, M. Ozkaya, A. Kocer, M.E. Ertorer, F. Koc, A. Kaya, S. Gullu, Thyrotoxichypokalaemic periodic paralysis in a Turkish population: three new case reports and analysis of the case series, Clin. Endocrinol. 68 (1) (2008) 143–152, https://doi.org/10.1111/j.1365-2265.2007.03014.x.
[47] H. Tassone, A. Moulin, S.O. Henderson, The pitfalls of potassium replacement in thyrotoxic periodic paralysis: a case report and review of the literature, J. Emerg. Med. 26 (2) (2004) 157–161, https://doi.org/10.1016/j.jemermed.2003.05.004.
[48] B.D. Rose, T.W. Post, Clinical Physiology of Acid-Base and Electrolyte Disorders, 5th, McGraw-Hill, New York, 2001, p. 836.
[49] C.E. Jackson, R.J. Barohn, Improvement of the exercise test after therapy in thyrotoxic periodic paralysis, Muscle Nerve 15 (10) (1992) 1069–1071, https://doi.org/10.1002/mus.880151002.
[50] R.Y. Chang, B.H. Lang, A.C. Chan, K.P. Wong, Evaluating the efficacy of primary treatment for graves' disease complicated by thyrotoxic periodic paralysis, Intern. J. Endocrinol. (2014), 949068, https://doi.org/10.1155/2014/949068, 2014.
[51] L. Ptácek, The familial periodic paralyses and nondystrophicmyotonias, Am. J. Med. 105 (1) (1998) 58–70, https://doi.org/10.1016/s0002-9343(98)00123-5.