



## Case Report

# Management of hypokalemia in patients with thyrotoxicosis periodic paralysis in Soetomo general hospital: A case report

Muhammad Idham<sup>\*</sup>, Jongky Hendro Prajitno

Internal Medicine Departement, Faculty of Medicine, Universitas Airlangga-Dr. Soetomo General Hospital, Surabaya, East Java, Indonesia



## ARTICLE INFO

## Keywords:

Thyrotoxicosis periodic paralysis  
Management hypokalemia  
Rebound hyperkalemia  
Clinical evaluation  
ECG and Electrolytes monitoring

## ABSTRACT

**Introduction:** The prevalence of Graves' disease varies widely between 21 and 80% of all cases of hyperthyroidism. Research conducted in 2018 at the Dr. Soetomo Regional General Hospital Surabaya found Graves' disease in as many as 66.7% of all cases of hyperthyroidism. Thyrotoxicosis Periodic Paralysis (TPP) is a disorder characterized by reversible muscle weakness and paralysis, accompanied by hypokalemia, which usually accompanies hyperthyroidism, mostly caused by Graves' disease. Management of severe hypokalemia in TPP is challenging.

**Case illustration:** male, 29 years patient complained that both lower legs felt weak in the last 6 hours before coming to the hospital emergency department. The patient was diagnosed with hyperthyroidism in 2018. A fine tremor was found, and the patient was admitted to the hospital for 4 days and routinely controlled at the endocrine polyclinic.

**Clinical discussion:** The underlying disease or causative etiology of thyrotoxicosis must be determined before treatment is conducted. The main concern when performing potassium replacement therapy is the occurrence of rebound hyperkalemia because this hypokalemia condition is caused not by total potassium depletion.

**Conclusion:** The principles of management for thyrotoxicosis periodic paralysis are proper diagnosis, exclusion of other causes of paralysis, and other causes of hypokalemia, slow and gradual correction of hypokalemia, and close and careful clinical monitoring, ECG, and laboratory.

## 1. Introduction

Graves' disease is an autoimmune disease characterized by hyperthyroidism, in which there are autoantibodies to the thyroid-stimulating hormone (TSH) receptor. The prevalence of Graves' disease varies widely between 21 and 80% of all cases of hyperthyroidism [1]. Research conducted in 2018 at the Dr. Soetomo Regional General Hospital Surabaya found Graves' disease in as many as 66.7% of all cases of hyperthyroidism [2]. One of the complications that can occur in patients with Graves' disease is periodic paralytic thyrotoxicosis [3] (see Figs. 1 and 2).

Thyrotoxicosis Periodic Paralysis (TPP) is a disorder characterized by reversible muscle weakness and paralysis, accompanied by hypokalemia, which usually accompanies hyperthyroidism, mostly caused by Graves' disease. Thyrotoxicosis Periodic Paralysis (TPP) is a rare but potentially fatal complication of hyperthyroidism [4–6].

The incidence of TPP varies between countries and races. The

prevalence of TPP in western countries is 1:100,000 and occurs more often in Asian descent, while the exact prevalence in Asian countries is not reported [7]. The incidence ratio between men and women is the opposite of Graves' disease, it is said that the incidence of men is greater than women, which is about 30:1<sup>7,8</sup>. Based on this, early recognition of TPP is important to determine its management strategy [9].

Management of severe hypokalemia in TPP presents its challenges. On the one hand, correction of hypokalemia must be given immediately to prevent fatal cardiovascular complications or respiratory muscle paralysis, but on the other hand, correction of hypokalemia must be carried out carefully because rebound hyperkalemia can also be life-threatening [8–10].

## 2. Case illustration

A male, 29 years old living in Surabaya, working as a motor vehicle sales salesman on January 27, 2020, was admitted to the Soetomo

<sup>\*</sup> Corresponding author. Internal Medicine Departement, Faculty of Medicine Universitas Airlangga, Dr. Soetomo General Hospital, Jl. Mayjend Prof. Dr. Moestopo No. 6-8, Surabaya, 60285, Indonesia.

E-mail address: [Idham.gtnew86@gmail.com](mailto:Idham.gtnew86@gmail.com) (M. Idham).

<https://doi.org/10.1016/j.amsu.2022.104925>

Received 21 August 2022; Received in revised form 8 November 2022; Accepted 13 November 2022

Available online 19 November 2022

2049-0801/© 2022 The Authors. Published by Elsevier Ltd on behalf of IJS Publishing Group Ltd. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

general hospital with complaints of weakness in both lower limbs. The patient complained that both lower legs felt weak until they could not be moved, the complaints were felt suddenly in the last 6 hours before entering the hospital, at first the back muscles felt sore and a few hours later the two thighs felt weak at first which was then followed by the whole lower leg not can be moved. Headache. Tingling sensation, low back pain, previous history of falls, seizures, fever, nausea, vomiting, decreased appetite, cough, and shortness of breath were all denied. The previous patient said that he drank a lot of iced syrup in the afternoon and evening while working in the field. The patient also said that he liked to eat large portions of rice. Urinate and defecate no complaints.

Past medical history, the patient said about 1 year ago he had felt weakness in his legs but he could still walk. In February 2018, the patient was diagnosed with hyperthyroidism and received therapy with Tyrozol 1 × 1 tab and propranolol 3 × 10 mg at Lamongan Hospital. However, for the last 2 weeks, the patient has not taken medication because he feels there are no complaints. He denied a history of diabetes and hypertension, and a history of taking other drugs or herbal medicine was denied. Family history of illness, no family members suffer from the same disease.

Physical examination revealed a normal Glasgow coma scale, general condition was weak, adequate nutritional status with TB 165 cm and weight 65 kg (BMI 23.9 kg/m<sup>2</sup>), blood pressure 133/60 mmHg, pulse 100x/minute, respiration 20x/minute, axillary temperature 37Co, SpO<sub>2</sub> 99% without O<sub>2</sub>. On examination of the head/neck, the conjunctiva was not anemic, the sclera was not icterus, no cyanosis, no dyspnea, no enlarged lymph nodes, and no increase in jugular venous pressure. In the patient's eyes, there was no starring eye, lid lag, stelwag sign, Joffroy sign, and Dalrymple sign. There was no mass or cicatricial mass on the neck. On thoracic examination found symmetrical thoracic movements and no retractions. On cardiac examination, the left heart border was found in the left intercostal space (ICS IV) midclavicular line, with normal heart sounds, no murmurs, and gallops. On lung examination, there was sonor, vesicular breath sounds in both lung fields, no rales, and wheezing. On examination, the abdomen was flat, bowel sounds were normal, no mass was found, and the liver, kidneys, and spleen were not palpable. Examination of the extremities capillary refill time (CRT) < 2 seconds, peripheral extremity palpable warm, dry, red,

and no edema.

A fine tremor was found in the patient, physiological reflexes were within normal limits. Extremity motor weakness is still found in the lower limbs where the motor strength of the lower extremities is 2, while both upper extremities are optimal (5). Sensory stimulation within normal limits, pathological reflexes are not found. The patient's skin feels warm and sweaty.

Laboratory examinations obtained from a normal complete blood count (Hb 15 g/dl, Hct 44%, RBC 5.5 million/mm<sup>3</sup>, MCV 80 fl, MCH 27.3 pg, MCHC 34.1 g/dl, leukocytes 10.300/μL, neutrophils 79%, lymphocytes 15.6%, platelets 292.000/μL) normal blood clotting rate APTT 32.5 (23–33), PPT 12.5 (9–12). Blood sugar level at 122 g/dl, normal liver function (SGOT 23 U/L SGPT 34 U/L), normal albumin level 3.6 g/dl. Normal kidney function (blood urea nitrogen 7 mg/dl, creatinine level 0.53 mg/dl). Normal blood electrolyte levels (sodium 134 mmol/L, potassium 1.7 mmol/L, chloride 102 mmol/L). Normal levels of free T<sub>4</sub> are very high 7.02 ng/dl (N: 0.89–1.76 ng/dl), and very low TSH levels are 0.003 IU (N: 0.55–4.78 IU). Blood gas analysis with pH 7.4, pCO<sub>2</sub> 27, pO<sub>2</sub> 108, HCO<sub>3</sub> 19.7, Be –4, SO<sub>2</sub> 98. ECG examination revealed sinus rhythm, 100 x/minute, normoaxis, flattening T wave, and U wave (Fig. 1). From the chest x-ray examination, no abnormalities were found.

The initial working diagnosis in this patient was severe hypokalemia due to thyrotoxicosis hypokalemic periodic paralysis. Planned the thyrotropin receptor antibody (TRAb) test, evaluation electrolyte, and Phosphate. Patients admitted to the hospital received therapy with a high-calorie, high-protein diet of 2100 kcal extra fruit, vegetable broth, low in iodine, infusion of premix KCl 50 meq iv drip in 0.9% NaCl 500 ml every 12 hours, Tyrozol 30 mg every 24 hours orally, propranolol 20 mg every 8 hours, Potassium Slow Release 600 mg every 8 hours orally. Performed clinical monitoring and ECG.

### 3. First day of hospitalization

The patient complains of difficulty moving the legs with vital signs, BP 130/80, pulse 110 x/minute, respiratory rate 20 x/minute, temperature 37 °C. The patient received a high-calorie high protein diet of 2100 kcal extra fruit, vegetable broth, low in iodine, infusion of premix KCl 50



Fig. 1. ECG at first evaluation.

meq iv drip in 0.9% NaCl 500 ml every 12 hours, Thyrozol 30 mg every 24 hours orally, propranolol 20 mg every 8 hours orally, Potassium Slow Release 600 mg every 8 hours orally. However, when the Premix KCl 50 meq infusion in 500 ml of 0.9% NaCl was just added +250 ml, the monitor showed a Tall T picture. Then an electrolyte evaluation was carried out and stopped the KCl infusion. Electrolyte examination revealed K 5.1 mmol/L, Na 137 mmol/L, Cl 103 mmol/L, Ca 9 mg/dl, P 3.2 mg/dl, Mg 2.3 mg/dl, urine potassium 73 mmol/24 hours, Na urine 246 mmol/24 hours, urine Cl 294 mmol/24 hours, Calcium urine 327 mg/24 hours, Pospata urine 1971 mg/24 hours.

#### 4. On the second day of admission

The patient can move his legs and there is no chest pain with vital signs BP 130/80 pulse 94 x/minute, respiratory rate 20 x/minute, temperature 37.1 °C. From SE examination, it was found that K was 4.4 mmol/L, Na 136 mmol/L, and Cl 102 mmol/L. Get a high-calorie, high-protein diet 2100 kcal extra fruit, vegetable broth, low in iodine, Thyrozol 30 mg every 24 hours before meals, propranolol 20 mg every 8 hours, Potassium Slow Release 600 mg every 24 hours. After the second day of treatment, ECG evaluation reveals a normal ECG sinus rhythm (see Fig. 2).

#### 5. Last day of admission

The patient said that the weakness in the legs had decreased significantly with vital signs, BP 110/75, pulse 81 x/minute, rr 18 x/minute, temperature 36.7 °C. From serum electrolyte examination, it was found that Potassium was 4.2 mmol/L, Sodium 144 mmol/L, Cl 99 mmol/L, Ca 9.0 mg/dl, Mg 2.0 mg/dl. The patient received a high-calorie, high-protein diet of 2100 kcal of extra fruit, vegetable broth, low in iodine, Thyrozol 30 mg every 24 hours before meals, propranolol 20 mg every 8 hours, Potassium Slow Release 600 mg every 24 hours. The patient is a planned outpatient observation.

## 6. Endocrine polyclinic

Further evaluation at the polyclinic showed that the results of the Thyroid-stimulating hormone receptor antibodies were 2.52 IU/L (N: < 1.75), from the results of the thyroid ultrasound examination, the isthmus size was +0.26 cm, the parenchymal echo intensity was normal, there were no visible lesions. Cystic/solid, right lobe size +1.72 × 1.91 × 3.35 cm, parenchymal echo intensity seemed heterogeneous, no cystic/solid lesions were seen, and no increase in vascularity was seen. Left lobe 1.73 × 2, 24 × 3.35 cm, parenchymal echo intensity appears heterogeneous, solid isoechoic lesion appears, wider than taller, ill-defined edge size +1.35 × 0.6 × 0.9 cm, visible perilesional vasculature. Mildly suspicious mass left thyroid lobe, the heterogeneous intensity of bilateral thyroid lobe echoparency.

During monitoring and follow-up at the endocrine polyclinic for 1 year, the patient never had a recurrence with serum potassium levels within normal limits, but the patient had experienced a hypothyroid condition due to thyrozol overtreatment when the patient did not come for regular check-ups. This case report had been presented in accordance to SCARE guideline 2020 [25].

## 7. Discussion

Hyperthyroidism is characterized by a physiological state of increased metabolic activity mediated by an increase in basal thyroxine (T4) and triiodothyronine (T3). In the end, the effects are many and varied; usually include subjective warmth, diaphoresis, anxiety, discomfort, tachycardia, difficulty concentrating, diarrhea, and in women, menstrual abnormalities. Exophthalmos and goiter may be present if Graves' is the underlying etiology. An enlarged, tender nodule or thyroid may be found if the patient has a toxic nodule or thyroiditis, respectively [11]. Clinical features of hyperthyroidism in a patient with thyrotoxicosis periodic paralysis do not always appear where more than half of cases of thyrotoxicosis periodic paralysis do not show overt symptoms of hyperthyroidism during an attack [12–14]. The above statement is by the condition of this patient who shows symptoms and clinical signs of hyperthyroidism that are not very clear during an attack

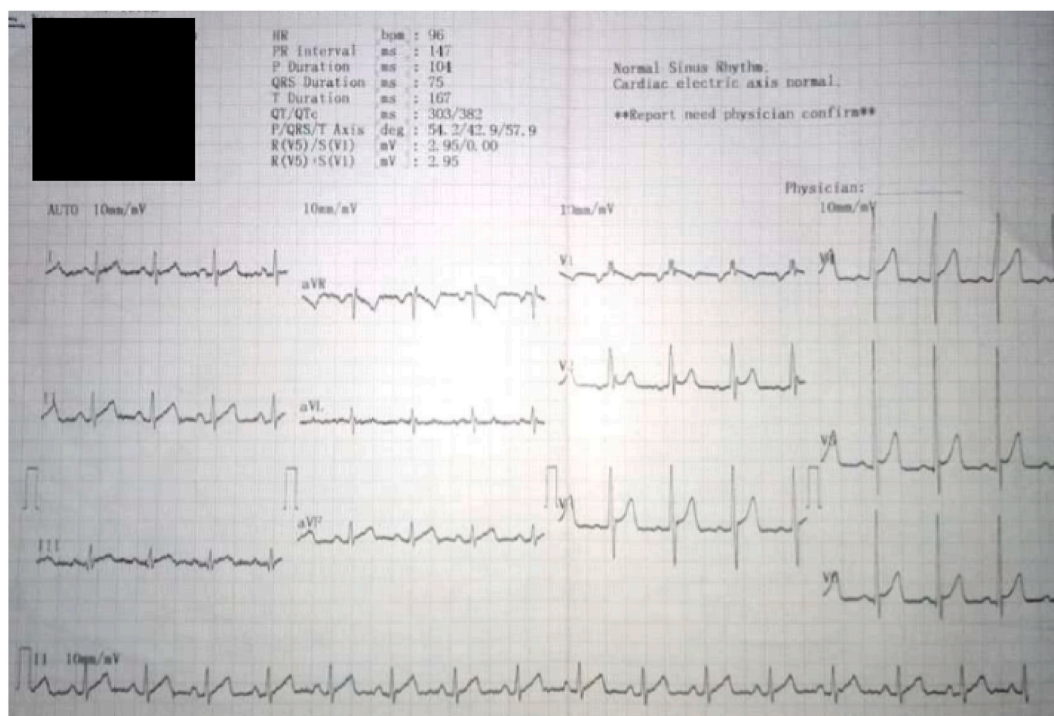


Fig. 2. Patient ECG on the second day of admission.



as evidenced by Wayne's index value of 21 in this patient. Based on the history, there are complaints of sudden lower extremity muscle paralysis without pain and is symmetrical. By the existing literature, the patient's condition usually immediately improved within a few hours after the initial administration of potassium therapy.

In this patient, the serum potassium value was 1.7 mg/dL accompanied by the results of Blood Gas Analysis of normal and normal urine potassium levels. In addition, the results obtained normal serum calcium levels and low magnesium levels, which improve spontaneously. This condition is the same as the research conducted by Ana et al. who showed laboratory findings in patients with periodic paralysis thyrotoxicosis including very low serum potassium levels (mean 2.0 mmol/L) without acid-base disturbances. Calcium levels were found to be normal, and some patients showed hypophosphatemia and hypomagnesemia, but these conditions tended to improve spontaneously [14]. The underlying disease or causative etiology of thyrotoxicosis must be determined before treatment is conducted. In this case, the patient already confirmed the etiology is from grave's disease or autoimmune, case report by Febrianto et al. in Soetomo general hospital found etiology of the grave's from testicular seminoma and another case report by Ali-saputri and Wibisono in Soetomo General hospital found thyrotoxicosis related in pregnancy and can misleading in hyperemesis gravidarum [22–24].

Hyperthyroid conditions are said to affect the complete blood count parameters. A cross-sectional study conducted by Prajitno et al. at the Soetomo General Hospital found a decrease in hemoglobin in patients with hyperthyroid conditions [18]. Other studies have also said that anemia can occur in hyperthyroid or hypothyroid conditions but more often in hypothyroid conditions [24]. Meanwhile, in this case, a normal hemoglobin value (Hb 15 g/dl) was obtained, this is probably due to most patients with thyrotoxicosis present with normal levels of Hgb, despite the erythropoietic effects of thyroid hormone but the pathogenesis of anemia associated with thyrotoxicosis remains unclear, however, the prevalence of grave disease associated anemia range between 33% and 41,6% [18,20].

Oral and/or intravenous administration of potassium is recommended in conditions of paralysis crisis to accelerate clinical improvement and prevent possible cardiac arrhythmias [13]. The main concern when performing potassium replacement therapy is the occurrence of rebound hyperkalemia because this hypokalemia condition is caused not by total potassium depletion but by intracellular trapped potassium ions. So it is necessary to monitor serum potassium levels during treatment and it is recommended to stop or delay the administration of intravenous potassium when the first improvement in muscle strength is obtained [13,15].

At the beginning of the management of hypokalemia in this patient, it was planned to use KCl premix 50 meq in 0.9% NaCl 500 ml in 12 hours. However, when the correction was running for 6 hours, a Tall T image on the cardiac record was monitored or a significant arrhythmia was caused by hyperkalemia, so the correction of hypokalemia was discontinued. Based on the results of the post-correction serum electrolyte examination, there was a change in the serum potassium value from 1.7 mmol/L to 5.1 mmol/L. This shows that the periodic paralysis of thyrotoxicosis is very susceptible to rebound hyperkalemia, so slow and gradual correction and clinical monitoring, strict ECG, and laboratory monitoring are necessary. This procedure is the same as the case report conducted by Jaishi et al. at Bakhunde Hospital Nepal. They performed closed monitoring on the first day of admission every 2 h and measured calcium levels treated with 10mEq/L/hr infusion of potassium chloride until normalized serum potassium level. The patient was kept in cardiac monitoring and serum potassium was monitored for 24 hours to prevent rebound hyperkalemia. Rebound hyperkalemia appears to be a significant issue in hypokalemia due to hyperthyroidism thyrotoxicosis periodic paralysis, occurring in 40–59% of treated attacks [10,19].

This patient was given propranolol because, in addition to controlling blood pressure, it is believed that periodic paralytic thyrotoxicosis is

also believed to reduce the frequency of intracellular potassium levels, with the mechanism of action of blocking adrenergic stimulation and the activity of the Na<sup>+</sup>/K<sup>+</sup> -ATPase pump, thereby reducing intracellular potassium transfer in myocytes. And some literature found in acute paralysis due to hypokalemia in thyrotoxicosis patients, potassium replacement therapy alone is not enough to stop attacks of acute paralysis due to thyrotoxicosis[15][20][21].

During monitoring in the endocrine clinic for 1 year, the patient never experienced a recurrence with success in achieving euthyroid status and serum potassium levels remained within normal limits. Paralysis attacks may recur before euthyroid status is achieved. In a retrospective study of 45 patients with periodic paralysis thyrotoxicosis, the rate of recurrent attacks was still high at 62.2% and most of them occurred in the first 3 months after being diagnosed with periodic paralysis thyrotoxicosis. Although most patients improve within hours of receiving therapy, respiratory collapse and death have been reported [16,17].

## 8. Conclusion

Early diagnosis and management of the underlying cause of thyrotoxicosis is the most important procedure in patients with periodic paralytic thyrotoxicosis. Once the euthyroid state has been reached, the paralytic crisis will disappear. Trigger factors such as high carbohydrate intake, alcohol, and intense physical exercise should be avoided until the thyroid disease is cured. The principles of management for thyrotoxicosis periodic paralysis are proper diagnosis, exclusion of other causes of paralysis, and other causes of hypokalemia, slow and gradual correction of hypokalemia, and close and careful clinical monitoring, ECG, and laboratory serum electrolytes.

## Ethical approval

This is a case report; therefore, it did not require ethical approval from the ethics committee. However, we have got permission from the patient to publish his data.

## Please state any sources of funding for your research

No sponsorship for this case report.

## Author contribution

Muhammad Idham contributes in the study concept or design, data collection, analysis and interpretation, oversight and leadership responsibility for the research activity planning and execution, including mentorship external to the core team.

Jongky Hendro Prajitno contributes in the study concept or design, data collection, analysis, interpretation and writing the paper.

## Registration of research studies

1. Name of the registry: not applicable.
2. Unique identifying number or registration ID: not applicable.
3. Hyperlink to your specific registration (must be publicly accessible and will be checked): not applicable.

## Guarantor

Muhammad Idham is the sole guarantor of this submitted article.

## 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.

### Provenance and peer review

Not commissioned, externally peer-reviewed.

### Declaration of competing interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

### Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.amsu.2022.104925>.

### References

- [1] I. Subekti, L.A. Pramono, Current diagnosis and management of graves' disease, *Acta Med. Indones.* 50 (2018) 177–182.
- [2] H. Gunawan, S.W. Mudjanarko, S.A. Soelistijo, A. Pranoto, Clinical profile of thyroid disorders in Dr. Soetomo general hospital Surabaya, *Jafes* 34 (2) (2022) 34–35. Retrieved from, [https://www.asean-endocrinejournal.org/index.php/JAFE\\_S/article/view/1977](https://www.asean-endocrinejournal.org/index.php/JAFE_S/article/view/1977).
- [3] M. Fralick, S. Sarma, Thyrotoxic periodic paralysis, *N Engl J Med Overseas Ed* 384 (2021) e71–e.
- [4] J. Chakraborty, S. a Chakraborty, R. Moitra, Thyrotoxic periodic paralysis: an update, *Journal of Endocrinology and Metabolism* 10 (3–4) (2020) 60–62.
- [5] K. Seetharaman, Thyrotoxic periodic paralysis, *Journal of the Endocrine Society* 5 (Supplement 1) (2021) A965–A966.
- [6] I.H. Tsai, Y.J. Su, Thyrotoxic periodic paralysis with ventricular tachycardia, *J. Electrocardiol.* 54 (2019) 93–95.
- [7] M. Salih, et al., Thyrotoxic periodic paralysis: an unusual presentation of hyperthyroidism, *Neth. J. Med.* 75 (8) (2017) 315–320.
- [8] C.C. Chang, C.J. Cheng, C.C. Sung, T.S. Chiueh, C.H. Lee, T. Chau, S.H. Lin, A 10-year analysis of thyrotoxic periodic paralysis in 135 patients: focus on symptomatology and precipitants, *Eur. J. Endocrinol.* 169 (2013) 529–536.
- [9] S. Bilha, O. Mitu, L. Teodoriu, C. Haba, C. Preda, Thyrotoxic periodic Paralysis—a misleading challenge in the emergency department, *Diagnostics* 10 (5) (2020) 316.
- [10] 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 Dec) 911–916, <https://doi.org/10.1530/EJE-09-0553>. Epub 2009 Sep 15. PMID: 19755408.
- [11] M. Correia, et al., Clinical communications: adult changing management guidelines in thyrotoxic hypokalemic, *J. Emerg. Med.* (April) (2018) 1–5, <https://doi.org/10.1016/j.jemermed.2018.04.063>.
- [12] S.H. Lin, Thyrotoxic periodic paralysis, *Mayo Clin. Proc.* 80 (1) (2005) 99–105, <https://doi.org/10.4065/80.1.99>.
- [13] A.W.C. Kung, Clinical Review: thyrotoxic periodic paralysis: a diagnostic challenge, *J. Clin. Endocrinol. Metab.* 91 (7) (2006) 2490–2495, <https://doi.org/10.1210/jc.2006-0356>.
- [14] M. Salih, et al., Thyrotoxic periodic paralysis: an unusual presentation of hyperthyroidism, *Neth. J. Med.* 75 (8) (2017) 315–320.
- [15] Ana Luiza R. Rolim, Magnus R. Dias da Silva, Thyrotoxic Periodic Paralysis – Clinical Diagnosis and Management, Intech, 2014, pp. 137–144, <https://doi.org/10.5772/58475>.
- [16] B. Abbasi, Z. Sharif, L.R. Sprabery, Hypokalemic thyrotoxic periodic paralysis with thyrotoxic psychosis and hypercapnic respiratory failure, *Am. J. Med. Sci.* 340 (2) (2010) 147–153, <https://doi.org/10.1097/MAJ.0b013e3181cbf567>.
- [17] M.J. Hsieh, et al., Hypokalemic thyrotoxic periodic paralysis: clinical characteristics and predictors of recurrent paralytic attacks, *Eur. J. Neurol.* 15 (6) (2008) 559–564, <https://doi.org/10.1111/j.1468-1331.2008.02132.x>.
- [18] Prajitno, jongky hendro, et al., Thyroid abnormalities associated with complete blood count parameter, *International Journal of Pharmaceutical Research* 12 (3) (2020) 1392–1396.
- [19] P. Poudel Jaishi, S.K. Neupane, P.K. Neupane, Case Report: Hyperthyroid Hypokalemic Periodic Paralysis, *Ann Med Surg* [Internet, 2022;78(April), 103759, <https://doi.org/10.1016/j.amsu.2022.103759>. Available from:.
- [20] 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>.
- [21] Gianoukakis, et al., Characterization of the anemia associated with Graves' disease, *Clin. Endocrinol.* 70 (5) (2009 May) 781–787, <https://doi.org/10.1111/j.1365-2265.2008.03382.x>.
- [22] K. Alisaputri, S. Wibisono, A case report: challenges in the management of toxic multi nodular thyroid in pregnancy who had crisis thyroid, *Current Internal Medicine Research and Practice Surabaya Journal* 2 (2) (2021) 56–59, <https://doi.org/10.20473/cimrj.v2i2.26243>.
- [23] D. Febrianto, H. Novida, Diagnostic and management problems of hyperthyroidism in a patient with testicular seminoma, *Current Internal Medicine Research and Practice Surabaya Journal* 3 (1) (2022) 16–21, <https://doi.org/10.20473/cimrj.v3i1.29969>.
- [24] D.M. Wopereis, R.S. Du Puy, D. Van Heemst, J.P. Walsh, A. Bremner, S.J.L. Bakker, et al., The relation between thyroid function and anemia: a pooled analysis of individual participant data, *J. Clin. Endocrinol. Metab.* 103 (10) (2018) 3658–3667.
- [25] 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.