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Case Report

Acute Hypokalemia Related to Thyrotoxicosis Periodic Paralysis: Case Report

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ABSTRACT

Introduction: Hypokalemia paralysis is a rare condition. Thyrotoxic periodic paralysis (TPP) is a condition of hyperthyroidism characterized with acute muscle weakness and hypokalemia. TPP is commonly found in young Asian male. TPP is an uncommon clinical entity and might be a life-threatening complication of thyrotoxicosis. We report a rare case of TPP with ECG abnormality related to severe hypokalemia.
Case Report: A-21-year-old Javanese male was presented to emergency department with acute onset of lower bilateral extremities weakness with proximal muscle predominant. Weakness progressed to his bilateral upper limb. Patient denied any significant trauma, vomiting, diarrhea, chest pain. Patient was fully conscious. Neurological examination revealed four limb weaknesses (lower limb muscle strength 2 from 5 scale MRC), diminished deep tendon reflexes, and intact sensory. Laboratory were remarkable for severe hypokalemia of 1.86 mEq/L. Further tests showed low TSH (0.022 mU/L), increased FT4 (23,68 mmol/L). ECG test showed abnormality related to hypokalemia. Thyroid ultrasound revealed multiple thyroid swelling, mass, and calcification. The patient was admitted to ICU and administered with 25mEq Potassium IV. Hypokalemia resolved after 3 days (Serum Potassium 3.76 mEq/L), followed by normal ECG feature without any AV block. He was discharged without any neurological abnormaliy.
Conclusion: We report a rare case of acute onset paralysis and markedly low potassium level and high free T4 in previously health Asian male. This case highlights the paralysis related with hyperthyroidism that resolved completely following potassium replacement.

Keywords: hypokalemia; paralysis; hyperthyroid; thyrotoxicosis periodic paralysis

INTRODUCTION

Acute limb weakness that is related to Hypokalemic Periodic Paralysis (HPP) is a rare condition. Thyrotoxicosis periodic paralysis (TPP) is one of the possible causes of hypokalaemia paralysis. TPP is an acquired clinical entity that is diagnosed by triad of (1) acute hypokalaemia, (2) muscle limb weakness, and (3) thyrotoxicosis. This diagnosis should be suspected in patient without family history of paralysis, male sex, and presentation in the second to fourth decades of life.[1,2] Hyperthyroidism is a major cause of episodic paralysis with hypokalaemia, especially in young Asian males, and thyroid function test must be performed to.[1]

The TPP is an uncommon clinical entity. It may be life-threatening complication of thyrotoxicosis.[2] The TTP is common in young from Asia, and the majority of cases of hyperthyroidism associated with TPP are due to Graves' disease. [1,3] The paralysis in most cases may be triggered by high carbohydrates diet, strenuous exercise, stress, and corticosteroids.[4]

In our cases we report 21-years old Javanese male with acute extremity weakness and ECG abnormality that related with severe hypokalaemia. Further investigation revealed the diagnosis of thyrotoxicosis periodic paralysis.

CASE REPORT

A 21-year-old Javanese male without any significant past medical history presented to the emergency department with acute onset of limb weakness. The weakness started in bilateral lower extremities with proximal muscle predominant. The patient denied any significant trauma. He consumed a significantly high caloric meal prior to onset. Bilateral lower limb weakness progressed to his bilateral upper limb. There was no significant sensory problem. Prior to this episode, the patient had been healthy, no routine medication, and denied any recent vomiting, diarrhoea, chest pain, or weight change. He did not take any medications and denied use of alcohol or drugs. No history of similar episodes and no other significant illnesses in the family.

On physical and neurological exam, the patient had normal vital signs. His skin was cool and dry, and the oral mucosa was moist. Clinical examination of the lungs and abdomen was unremarkable. There were no deformities or oedema of the extremities. The neurological evaluation showed that the patient was fully conscious. The physical exam was remarkable for four limb weakness. The muscle strength in lower limb was 2 from 5 scale MRC. The sensory was completely intact. The deep tendon reflexes are markedly diminished. Initial labs were remarkable for severe hypokalaemia of 1.86 mEq/L (normal range: 3.6-5.2 mEq/L). The other laboratory and imaging were

unremarkable. We suspect Thyrotoxicosis based on these criteria: the young onset, no family history, and Asian ethnicity. Further evaluation revealed the TSH was very low 0.022 mU/L (normal range 0.35-4.94 mU/L). Free T4 level was slightly increased to 23,68 mmol/L (normal range: 9-19,8 mmol/ L).

The ECG showed an abnormality related to hypokalaemia. No radiographic evidence of acute cardiopulmonary process on Chest X-ray one-view, and no evidence of acute intracranial abnormality or haemorrhage on computed tomography (CT) head without contrast. A thyroid ultrasound showed multiple thyroid swelling, mass, and calcification.

He was admitted to the intensive care unit (ICU) and was administered with an additional 25 mEq of intravenous Potassium. Hypokalaemia resolved after 3 days marked with serum potassium of 3.76 mEq/L. The ECG became normal without any AV block. He was discharged home without any neurological abnormality.

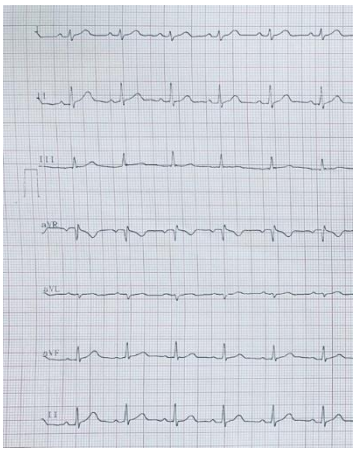
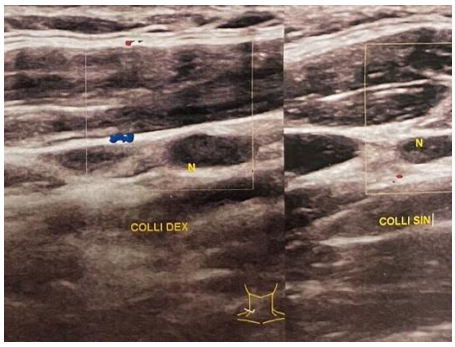


Figure 1. The ECG showed features of hypokalaemia (ST depression and U wave)



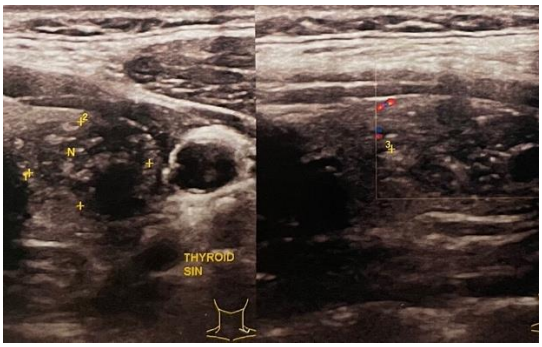


Figure 2. Thyroid ultrasound showed mass, swelling, and calcification

DISCUSSION

We report a case of sudden onset bilateral upper and lower limb weakness due to thyrotoxicosis hypokalemia. In our case the initial kalium level was very low (1.86

mEq/L). The initial ECG showed the classical signs of hypokalaemia, namely ST-segment depression and U-wave amplitude in our patient.[4]

The final diagnosis in our case was TPP based on the high free T4 and low TSH. TPP commonly present with lower extremity weakness that can also affect upper limb. The sensory functions were normal. The bowel and bladder functions remain intact. In order to consider TPP as a primary diagnosis, the patient must be carefully examined thyroid function (laboratory and imaging). The patient must have high levels of thyroid hormone. Diagnosis of TPP is mainly based on clinical and laboratory evidence of hyperthyroidism and hypokalaemia. The diagnosis of TPP was highly suspected in young, Asian, male patient with non-traumatic proximal muscle weakness. The weakness affecting mainly the lower limbs, without a family history of this disorder.[4,5]

A differential diagnosis that showed similar clinical presentation is known as Familial Hypokalaemia Periodic Paralysis (HKPP). This is a rare autosomal dominant channelopathy with a defect in calcium channels resulting in periodic muscle paralysis. In HKPP, the thyroid function was normal in these individuals. We should routinely check TSH to assess thyroid function when a patient has hypokalaemia and periodic paralysis with no obvious initial cause.[6,7]

The hypokalaemia paralysis may occur in some clinical settings. The diagnosis may require an extensive search of the possible etiology.[4,6] The treatment varies according to the cause. In our case, we diagnose the patient as Thyrotoxic Periodic Paralysis (TPP). Thyrotoxic Periodic Paralysis (TPP) occurs in the setting of hyperthyroidism. It is the most common form of hypokalaemia periodic paralysis in young Asian males.[6,7]

We treat our patient with kalium supplementation, low dose potassium sparing diuretic (spironolactone), and thiamazole. During acute limb paralysis and marked hypokalaemia we give immediate supplementation with potassium chloride at a slow rate to prevent major cardiopulmonary complication. This is important to avoid rebound hyperkalaemia that may life threatning. The TPP will not occur once the patient is euthyroid,

the adequate control of hyperthyroidism is the main concern of therapy.[5,7]

CONCLUSION

We report a case of acute onset paralysis and markedly low potassium level and high thyroid in previously health Asian male. The paralysis related with hyperthyroidism. The paralysis resolved completely following potassium replacement.

ACKNOWLEDGEMENT

Verbal informed consent has been obtained from the patient. No significant identification can be revealed.

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