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Case Report

Severe Limb Weakness, Bradycardia, and AV Block in Periodic Paralysis Hypokalemia

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ABSTRACT

Introduction: Periodic Paralysis is an uncommon group of disorders that can cause sudden extremity weakness. Hypokalaemia also may present with cardiovascular and neuromuscular abnormalities. Hypokalaemia can lead to clinically significant life-threatening cardiac rhythm abnormality. We present a case of a 32-year-old Javanese with sudden onset paralysis of his extremities. Laboratory evaluation revealed a significantly low potassium level. The ECG showed significant bradycardia and AV block. The patient's weakness improved gradually after repletion of the low potassium. The ECG also improved to normal sinus rhythm. The patient was discharged without any neurologic deficits.

Case Report: A 32-year-old Javanese male presented to the emergency room with sudden four extremity weakness. The patient complains begin when awoke at early morning, and unable to move his upper or lower extremities. The weakness was bilateral and predominantly involved both the proximal muscles of the shoulders and hips as well as the distal extremities.

Discussion: We report a case of sudden onset bilateral limbs flaccid weakness. The final diagnosis was periodic paralysis hypokalaemia. Treatment of periodic paralysis hypokalaemia consists of slow and careful correction of low potassium level. The very rapid correction exposes the risk of hyperkalaemia. Close monitoring by neurological examination and repeat examination potassium level is essential.

Conclusion: We report a case of sudden onset paralysis and significant cardiac abnormality related with very low potassium level. The paralysis and ECG abnormality resolved completely following potassium replacement.

Keywords: Hypokalemia, Periodic Paralysis, AV block, weakness

INTRODUCTION

Acute neuromuscular weakness related to Hypokalemic Periodic Paralysis (HPP) is a rare condition. This condition may be caused by significant low potassium intake, high loss (renal or extra renal), and shifting.(1) The most common clinical presentation of the periodic paralysis hypokalaemia is acute flaccid paralysis lasting from a few hours to a few days, sparing the sensory sensation.(2)

The classical presentation HPP is significant low potassium level (<<,3.0 mEq/L) and that improved with potassium supplementation. The duration of weakness is varied. In most cases, the episodes of weakness in HPP may occur from hours to days. The weakness is proximal muscle predominant. Patients may complain climbing stairs difficulty or rising from a chair. The baseline ECG may vary from normal to secondary changes (lowered T waves, prolongation of QT interval).[3] Hyperthyroidism is one main cause of hypokalaemia, especially in young Asian males, and thyroid function test must be obtained to exclude thyrotoxic periodic paralysis.[4]

The attack of weakness in HPP may be provoked by rest after strenuous exercise, carbohydrate-rich meals, ethanol use, stress, and certain medications.[3] In our cases we report 32-years old Javanese male with acute limb weakness and significant bradycardia and AV block related with severe periodic paralysis hypokalaemia.

CASE REPORT

A 32-year-old Javanese male presented to the emergency room with sudden four extremity weakness. The patient denied any previous significant trauma. The patient complains begin when awoke at early morning, and unable to move his upper or lower extremities.

The weakness was bilateral and predominantly involved both the proximal muscles of the shoulders and hips as well as the distal extremities. The patient had no respiratory or swallowing problem. He has no difficulty moving his neck and facial muscles. He has no pain or sensory abnormality. Before the complaint, he was healthy and had no complaint of vomiting, diarrhea, chest pain, or weight change. He did not take any routine medications, alcohol, or drugs. The patient has a history of similar episodes one year ago.

On the clinical exam, he had normal blood pressure, but a significantly low heart rate (56 times per minute). He was normal in clinical presentation. His skin was cool and dry. The Cardiac exam revealed bradycardia with a regular rhythm and no

murmurs. Examination of the lungs and abdomen was normal. There were no deformities or swelling of the extremities. The neurological examination showed significant flaccid paralysis of all extremities with proximal muscles predominant including the hips and shoulders. The MRC muscle strength in either upper or lower extremities was 2/5. The sensory test was intact and deep tendon reflexes was normal. The cranial nerves' function was normal.

The routine blood test, glucose, liver enzymes and renal function were normal except for a very low potassium level of 1.56 (3.5–5 mmol/L). Electrocardiogram showed significant AV block with bradycardia. The patient admitted to the highly intensive ward and potassium replacement therapy was began. The patient was monitored closely. Either oral or intravenous potassium replacement with 25 mEq potassium was administered within 6 hours. The serum potassium level result was still very low 1.72 mEq/L in the following morning. We then administered another 50 mEq of potassium intravenously within 8 hours. The muscle strength of the patient improved after two days (3/5). He was admitted to the ward and was administered an additional 25 mEq of intravenous potassium, and hypokalaemia resolved (serum potassium 3.72 mEq/L). The ECG became normal without any AV block.

The urine potassium level was very low (less than 20 mEq/ L). No evidence of kalium loss from gastrointestinal tract (vomiting or diarrhoea). He also denied any possibility of potassium-shifting or potassium-wasting medication use, such as insulin, beta-agonists, thyroxine, or diuretics. The levels of free T_4 (1.24 ng/dL) were normal; levels of serum thyroid stimulating hormone (TSH) were also normal. The patient was diagnosed with Hypokalaemia Periodic Paralysis. He was discharged home without any neurological abnormality.

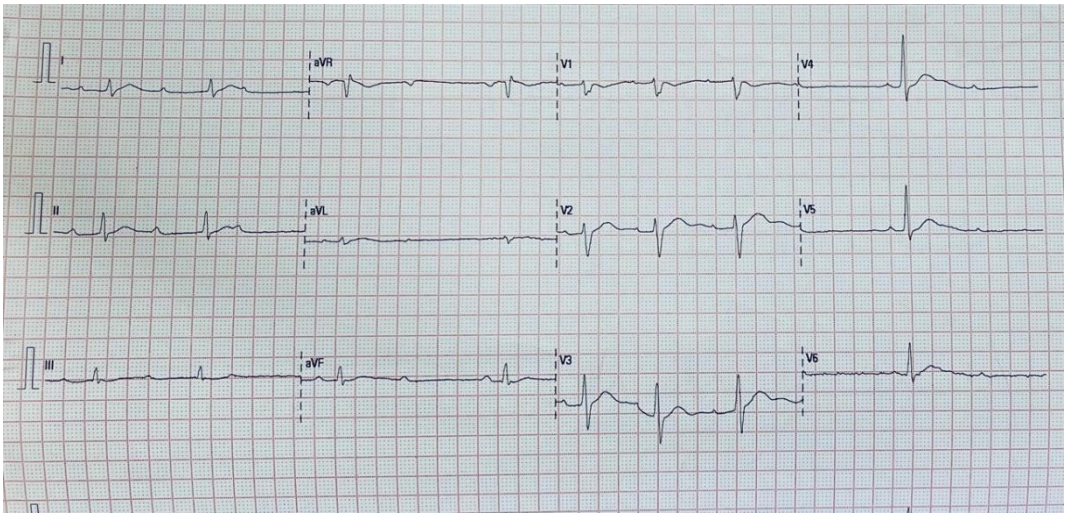

Figure 1. The very significant bradycardia and AV block in initial presentation

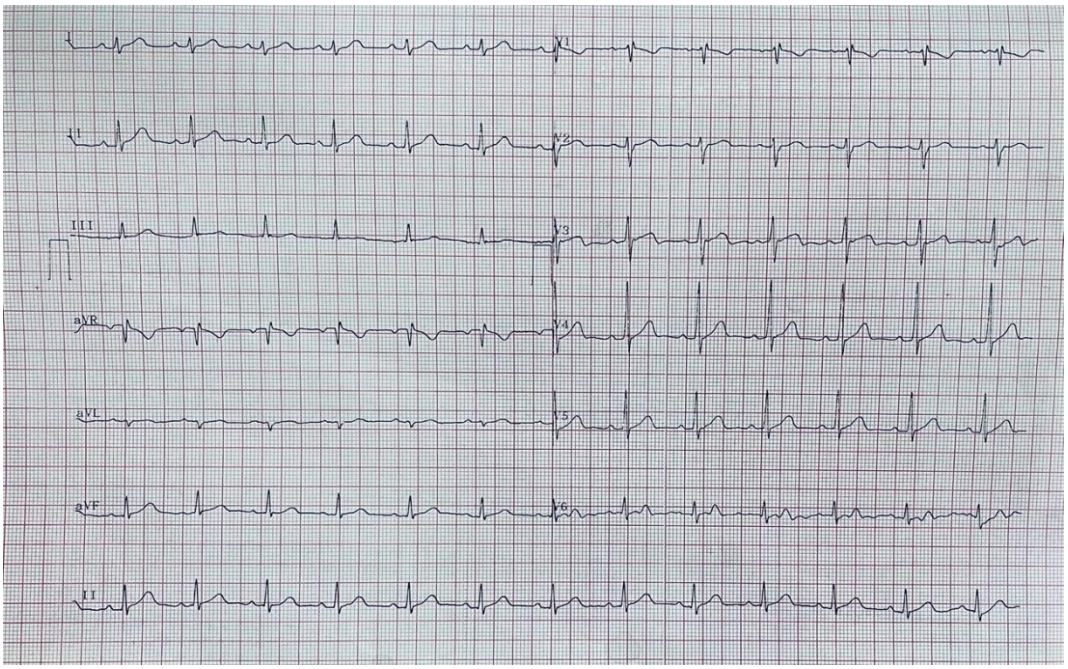

Figure 2. The improvement of AV block and bradycardia after potassium suplementation

DISCUSSION

We report a case of sudden onset bilateral limbs flaccid weakness. The final diagnosis was periodic paralysis hypokalaemia. In HPP, the potassium level is most often between 0.9 mmol/l and 3 mmol/l.[4] The ECG abnormality is a significant clinical findings in hypokalaemia. The classical signs of hypokalaemia were ST-segment depression, T-wave depression or inversion, increased U-wave amplitude, AV block, and QT

prolongation.[3,4] In our patient, we obtained significant AV block and bradycardia.

Treatment of periodic paralysis hypokalaemia consists of slow and careful correction of low potassium level. The dose of correction may not exceed of 10 mmol/hour. The very rapid correction exposes the risk of hyperkalaemia.[5] Close monitoring by neurological examination and repeat examination potassium level is essential.[5,6] Therapeutic education that focused on avoiding triggers is very important. It prevents the occurrence of episodes in the future.

Periodic paralysis hypokalaemia may occur in several clinical settings. The diagnosis should require an extensive search for the underlying aetiology. The treatment may vary according to the cause. In this patient, we have excluded the possibility of Thyrotoxic Periodic Paralysis (TPP). Thyrotoxic Periodic Paralysis (TPP) is related to hyperthyroidism. It is the most common form of HPP and is seen primarily in young Asian males.[6]

Rarely, HPP can result from substantial gastrointestinal or renal potassium losses. In these conditions, total body potassium is very low and requires immediate replacement. Endocrine abnormalities such as hyperinsulinemia and primary hyperaldosteronism have been associated with HPP.[7] Information about triggers are very important. After initial exposure to triggers, the activity and number of Na-K-ATPases on the cell membrane changes, causing mild potassium influx into cells. Paradoxical depolarization of the skeletal membrane potential occurs, exaggerating extracellular hypokalaemia.[8] Therefore, the identification of specific triggers and their prevention are important.

CONCLUSION

We report a case of sudden onset paralysis and significant cardiac abnormality related with very low potassium level. The paralysis and ECG abnormality 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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