

# Dexamethasone induced severe paralysis hypokalemia: A case report

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## ABSTRACT

Acute weakness related with hypokalemia periodic paralysis (HPP) is a rare clinical condition. The one common etiology of hypokalemia is the shifting of potassium into the cells. The common provoke factors are high-carbohydrate diet, heavy exercise, stress, extreme cold weather, alcohol consumption, and certain medications. We present the case of a 32-year-old Javanese male with an acute onset of bilateral lower and upper limb weakness with severe hypokalemia following dexamethasone injection. He was diagnosed with HPP. Our systematic review found similar cases, with possible pathophysiology explanation. Steroids should be considered as an unusual precipitating factor while managing patients with HPP.

**Keywords:** acute limb weakness, dexamethasone, hypokalemia periodic paralysis

## INTRODUCTION

Acute muscle weakness from hypokalemia periodic paralysis (HPP) is a rare neuromuscular pathology because of ion-channel disease [1]. The weakness is marked by acute onset of muscle flaccidity related with low serum potassium levels. The typical feature is weakness in proximal muscles are more prominent than distal muscles [2]. The low potassium level will lead to the decrease of muscle tone and deep tendon [3].

Previous review showed that trigger factors of HPP are high carbohydrate intake, heavy exercise, and stress that related with excessive release of insulin or epinephrine [2,3]. Some medications have been reported can induce hypokalemia [4].

Glucocorticoids are the very effective anti-inflammatory medication either for chronic and acute condition. One of the adverse drug reactions after high dose glucocorticoid administration is electrolyte disturbance. Electrolyte imbalance like hypokalemia has been reported in patients treated with steroid [5,6]. Further studies showed that steroid should be carefully considered as triggering factor of HPP attacks [7]. This is a case report of a 32-year-old Javanese male with acute limb weakness related with HPP after Dexamethasone injection.

## CASE REPORT

A 32-year-old Javanese was visited neurology clinic due to acute bilateral lower limb and upper limb weakness since 1 day ago. He had been treated for retinal detachment procedure in neighboring eye hospital and been injected dexamethasone before the procedure 2 days ago. However, acute limb weakness occurred after the patient woke up in the morning. He had difficulty standing and lifting both arm and legs. No breathing difficulty and sensory abnormality. He had been referred by the ophthalmologist to our neurology clinic.

Upon arrival at our neurology clinic, the blood pressure, respiratory rate, tympanic membrane temperature normal. Our neurological history taking and examination showed no loss of consciousness, limb or trunk pain, respiratory symptoms, vomiting, diarrhea, or heavy sweating. The patient denied having much carbohydrate-rich food intake or strenuous exercise before the attack. He had no regular consumption of alcohol or smoking habits. The patient was oriented well and have a good mental health status, and well nourished. Neurological examination showed decreased muscle power of the bilateral lower limbs and upper limbs, mainly proximal, and either upper or lower scored 2/5 (MRC scale). The deep

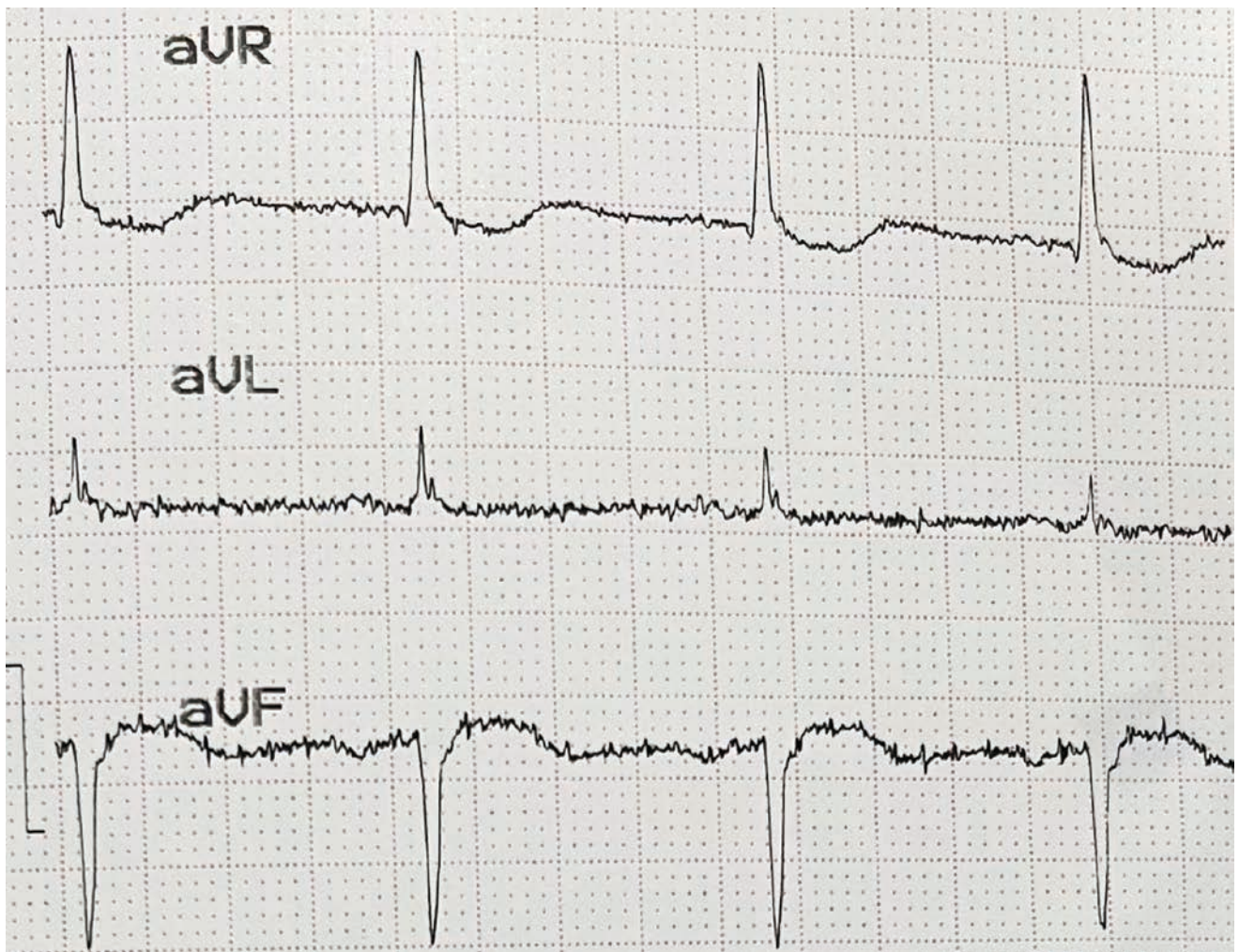


FIGURE 1. ECG abnormality in the patient with HPP

tendon reflex of the bilateral knees and ankle reflex were normal. The sensation of the lower limbs and upper limbs were intact. There was also no Babinski sign elicited on either side. Blood tests, including complete cell count, blood sugar, and a biochemical panel, were normal. Urinalysis was normal. However, the potassium level was very low (2.38 mmol/L). The electrocardiogram showed a flattened T wave and exhibited U waves in the precordial leads V1–V3.

Oral and intravenous potassium supplements with 25 mEq IV was administered slowly in 500 cc isotonic solution for 6 hours. The serum potassium level result was still 2.47 mmol/L in the following morning. Further 50 mEq of potassium for 12 hours infusion and spironolactone were given. In the follow-up assessment, the muscle power of the patient's bilateral lower limbs and upper limbs improved in the afternoon (4/5). Further additional 25 mEq of intravenous potassium were given in the next day. The hypokalemia resolved (serum potassium 3.52 mmol/L), and the patient could walk steadily.

The urinary potassium excretion of the patient was low (urine potassium was 21.27 mmol/ 24 hours; normal level: 40-80 mmol/ 24 hours), and there was no evidence of potassium loss from gastrointestinal

tract. There are no history of potassium-shifting or potassium-wasting medication use, such as insulin, beta-agonists, thyroxine, or diuretics. The level of free T4 and TSHS were normal. Arterial blood gas analysis was normal. Hypokalemia periodic paralysis was considered. Rapid normalization of potassium levels and improvement of weakness highly suggested of shifting mechanism. The normal thyroid function exclude the possible thyrotoxicosis hypokalemia. The final diagnosis was HPP of the non-familial type, suspected to be induced by dexamethasone injection. Finally, he was discharged without any disability.

## DISCUSSION

We present the case of a 32-year-old Javanese male with acute onset of bilateral symmetrical either lower limb and upper limb weakness related with hypokalemia. HPP was diagnosed due to the symptoms, short duration, and rapid improvement with potassium supplementation. The triggering factor of HPP was likely iatrogenic dexamethasone injection. The similar case reports and plausible biological explanation support the diagnosis [6-8]. HPP is consid-

ered to be a channelopathy. Triggers are important for inducing a paralysis attack [7,8].

After initial exposure to triggers, the activity and number of Na-K-ATPases on the cell membrane changes. This condition result potassium influx into cells. The next condition is paradoxical depolarization of the skeletal membrane potential. It will exaggerating extracellular hypokalemia with a smaller efflux of potassium, inactive sodium channels, loss of excitability, and end with muscle weakness [7,8]. Therefore, the identification of trigger factors are very important for effective prevention.

Several medications such as beta-adrenergic agonists, insulin, or steroid had been reported as trigger factors that can induce hypokalemia. Heavy carbohydrate diet, high-impact exercise followed by rest, and stress (or excitement/fear/cold) exposure might directly increase sympathetic tone by releasing more catecholamines. This condition will lead to hyperinsulinemia, and then stimulate the activity of skeletal muscle Na-K-ATPase, resulting in hypokalemia [6-8].

Hypokalemia has been reported in patients treated with steroid. There were very few cases reporting the risk of hypokalemia related with steroid therapy [8,9]. Naranjo adverse drug reaction probability scale was applied to quantify the degree of association between Dexamethasone injection and Hypokalemia in our patient and it was found to be 8 (Probable).

Glucocorticoids should be considered as possible triggers of HPP. Several possible biological plausibility of steroid induced HPP attack are: First, steroid

cause insulin resistance which results in hyperglycaemia and hyperinsulinemia that makes the intracellular shift of serum potassium [10]. Second, steroid can upregulate beta-2 receptors on the cell membrane, and the interaction between beta-2 receptors and catecholamines can enhance the effect on the Na-K-ATPase [11]. Third, steroid directly regulate transcription of the Na-K-ATPase that increase the excitement potential of skeletal cell membranes [12]. Every physicians should be aware of this rare adverse drug reaction in patients treated with steroid.

## CONCLUSION

We report an unusual case of HPP severe limbs weakness after the administration of high doses Dexamethasone. Our case should be a reminder for physicians that HPP should be considered as trigger factors in HPP.

### *Ethics approval and consent to participate:*

The patient provided verbal agreement for publication of the data. No data of the patients that can be identified.

### *Conflict of interest:*

The authors declare no conflicts of interest in the information contained in the manuscript.

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