中文題目：類固醇引起的低鉀性週期性麻痺：病例報告與文獻綜述

英文題目：Steroid-Induced Hypokalemic Periodic Paralysis: a Case Report and Literature Review

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Background*:* Hypokalemic periodic paralysis (HPP) is a rare channelopathy features episodic attack of acute muscle weakness concomitant with hypokalemia. The etiology of hypokalemia was the shifting of potassium into cell and the clinical symptoms resolved when the potassium starts to leak back to the serum. Most of time, the underlying ion channel defects are well-compensated and an additional trigger is often required to initiate the attack. The well-known triggers included carbohydrate-rich meals, exercise with followed rest, stress, cold weather, and alcohol.

Case Presentation: Here, we presented a case of 26-year-old Asian man who suffered from acute onset of bilateral lower limbs weakness with hypokalemia following the injection of dexamethasone. The HPP was impressed due to the symptoms, its duration, and the rapid improvement with potassium supplement. The trigger factor of HPP is dexamethasone injection, which also resulted in temporary iatrogenic adrenal insufficiency in this case.

Discussion: The HPP is considered as a channelopathy due to defective ion-channels, mainly resulted from mutations in genes encoding the subunit of L-type voltage-dependent calcium channel Cav1.1 (CACNA1S) and skeletal muscle sodium voltage-gated channel Nav1.4 (SCN4A). The triggers are important to induce paralysis attack. When initial exposure to triggers, the activity and number of Na-K-ATPase on the cell membrane change and cause mild potassium influx into cell. Paradoxical depolarization of skeletal membrane potential would occur, exaggerate extracellular hypokalemia with smaller efflux of potassium, inactive sodium channels, loss of excitability, and finally cause muscle weakness. Therefore, identification of specific triggers and prevention is important and advised to the HPP patients. Glucocorticoids are ever considered as possible triggers of HPP attacks, but the mechanism is not well understood. Several possible mechanisms were raised for explanation. First, glucocorticoids can cause insulin resistance, which results in hyperglycemia and hyperinsulinemia. Insulin increases the activity and number of

Na-K-ATPase and therefore intracellular shift of serum potassium. Insulin also inhibits the current of inward-rectifier potassium channel (Kir) and lead to more depolarized membranous potential. Second, glucocorticoids can upregulate beta-2 receptors on cell membrane, and interaction between beta-2 receptors with catecholamine also have stimulatory impact on the Na-K-ATPase. Third, glucocorticoids directly regulate the transcription of Na-K-ATPase, increasing the excitement potential of skeletal cell membranes.

Conclusion: The importance of our finding is to remind physicians that HPP should be included in the differential diagnosis if sudden onset of generalized flaccid muscle weakness occurs after glucocorticoids administration. Moreover, glucocorticoids should be carefully used in patients with HPP because the medication may be one of the possible triggers.