中文題目：乙酸可體松引發嚴重低血鉀並導致肌肉無力 - 個案報告

英文題目：Cortisone acetate induced hypokalemia causing muscle weakness – case report

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Introduction

Cortisone acetate is a steroid hormone that has both glucocoriticoid and mineralocorticoid activities. The excess of mineralocorticoid may induce severe hypokalemia further causing muscle weakness. In this case, we describe a patient developing severe hypokalemia and muscle weakness after 2-month low-dose cortisone acetate .

Case Presentation

An 87-year-old male patient with diabetes mellitus, hypertension, old stroke, atrial fibrillation, and chronic obstructive pulmonary disease presented with a one-month history of progressive lower legs weakness. This symptom deteriorated in one week. His baseline activity of daily life was walking without assistance and he started to use cane one week before presentation. On arrival, he was unable to walk and wheelchair was needed. Long-term inhaled steroid (Seretide Evohaler) was used for chronic bronchitis for more than 2 years. Cortisone acetate 50mg twice per day was prescribed for suspected adrenal insufficiency 2 months before presentation. The laboratory data revealed severe hypokalemia (K: 2.1mmol/L), metabolic alkalosis (pH: 7.529, pCO_2: 47.5 mmHg and HCO_3^-: 38.7 m mol/L), mild hypomagnesemia (Mg: 1.61mg/dL) and normal serum creatinine (0.86 mg/dL similar to baseline). Furthermore, the transtubular K^+ gradient (TTKG) was 9.1 and urine potassium-to-creatinine ratio was 85.99 mmol/g and both of them suggested renal-loss related hypokalemia. The hyperaldosteronism or excess cortisol related hypokalemia was suspected. The dose of cortisone acetate was tapered gradually. After aggressive intravenous and oral potassium supply, the potassium level increased to within normal limit and the lower legs weakness also improved. He was able to walk himself after treatment. No recurrent hypokalemia or weakness was noted after discontinuation of intravenous high-dose potassium. The cortisol and ACTH level were within normal limit(13.63 μ g/dL and 15.59 pg/ml respectively, at 6 A.M.). However, low aldosterone level and renin plasma activity (PRA) were noted (<2.5 ng/dL and <0.31 ng/mL/hr respectively). These results suggested excess mineralocorticoid and were compatible with the suspicion of cortisone acetate inducing severe hypokalemia.

Discussion

Corticosteroids are commonly used in a lot of clinical conditions. We found there are few cases of hypokalemic described in high dose intravenous hydrocortisone use. But there is no reported case of low-dose cortisone acetate induced severe hypokalemia causing muscle weakness. We must

carefully consider in each case the gains that can be expected from corticosteroid therapy versus the undesirable side effects of prolonged therapy. In our case report, it was seen that even low-dose cortisone can cause hypokalemia and muscle weakness.