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Prednisolone Induced Hypokalemia Causing Quadriparesis : A Case Report

Medical Science

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ABSTRACT

The etiologies of hypokalemia are numerous, the diagnosis of drug induced hypokalemia may be overlooked. Evaluation of a patient presenting with hypokalemia should include a careful review of medication history to determine whether any drugs causing or aggrevating the electrolyte abnormality are present. A case of 52 year old male, presented with acute quadriparesis secondary to severe hypokalemia. Serum potassium was corrected by intravenous and oral supplementation, and the quadriparesis recovered completely. A detailed history revealed that he had been taking prednisolone and griseofulvin for dermatological lesions (tinea) from four months. Here in we report this case as prednisolone induced hypokalemia causing quadriparesis.

INTRODUCTION

Hypokalemia is defined as a plasma potassium concentration <3.5 mEq/L. It is one of the most common electrolyte abnormality observed in clinical practice. Plasma potassium is maintained in the narrow range of 3.5 mEq/L – 5.5 mEq/L. Hypokalemia may manifest with muscle weakness (as 98% of the intracellular potassium is in the skeletal muscles) , paralysis including intestinal ileus, cardiac arrhythmias, rhabdomyolysis, renal dysfunction or hyperglycemia.

Hypokalemia may occur due to several causes including decreased intake , redistribution into cells , increased loss (renal or extra renal).

Many drugs may lead to hypokalemia, drugs like diuretics (the most common cause), beta adrenergic agonists, theophylline, steroids and aminoglycosides (2 ,3).

In our case, he had been taking orally prednisolone(20mg) and griseofulvin from four months. Oral prednisolone induced hypokalemia manifested as acute quadriparesis in this case.

CASE REPORT

A 52 year old male was brought to the emergency room with complaints of weakness of both upper and lower limbs from one day. No history of trauma, loss of consciousness, fever, seizure, vomiting and diarrhea. No history suggestive of bladder and bowel involvement. No similar complaints in the past. Neither a diabetic nor a hypertensive. Non smoker, non alcoholic. No addictions were present. Drug history revealed he had been using medication (prednisolone, griseofulvin) for dermatological lesions(tinea) from four months.

On examination, he was moderately built and moderately nourished. Conscious, coherent, well oriented. Heamodynamically stable with blood pressure of 140/80 mm of Hg, pulse rate :64/min, respiratory rate :22/min, SPO2 98% at room air. General physical examination was unremarkable. Central nervous system examination revealed hypotonia, motor power grade 2/5 (proximal and distal) in all the four extremities. Deep tendon reflexes were absent, Plantars are equivocal - suggestive of predominant lower motor neuron type of quadriparesis.

Routine Investigations like Haemogram, renal function tests, blood sugar, liver function tests and urine microscopy were normal. Electrolyte abnormality was present – hypokalemia, Serum potassium was 2.0 mEq/L. Serum sodium 145 mEq/L . Serum calcium and magnesium were with in the normal limits. Electrocardiogram was normal. Arterial blood gas analysis showed P^{H}7.34, PCO_2 45 mm of Hg, $PO_2$68 mm of Hg,Na^+ 140 mmol/L ,K^+ 1.1 mmol/L,HCT 35%,HCO_3 – 24.3 mmol/L and saturation 98.5%.

Urine spot K^+ 16.2,Urine PH 6.0,Urine Cl^- 115 mEq/L.

With this background we diagnosed our case as quadriparesis due to hypokalemia, probably durg induced(prednisolone).

On the day of admission, he was treated with intravenous potassium chloride (40 mmol).His weakness improved to motor power grade 3/5 on Day 1. He was given oral supplementation with syrup potassium chloride (15 ml thrice daily) on subsequent days. Weakness improved in all the four limbs gradually to motor power grade 4/5 on Day 2. Prednisolone was gradually tapered and stopped. Serum potassium levels were checked daily. (Table 1).

TABLE 1 : Serum Potassium

DAY	0	1	2	3	4	5	6
6 AM	2	3.3	4.3	4.1	4.1	4.3	5.2
6 PM	2.2	3.6	5	3.9	3.9	4.4	4.9

After six days of oral supplementation with potassium chloride, he was discharged with motor power grade 5/5 in all the four extremities.

DISCUSSION

QUADRIPARESIS

Acute quadriparesis may result from disorder of upper motor neuron e. g., (anoxia, hypotension, brainstem or cervical cord ischaemia, trauma and systemic abnormalities)

Or Muscle (electrolyte disturbance, certain inborn errors of muscle energy metabolism, toxins and periodic paralysis).(1)

If upper motor neuron signs are present , computed tomography of brain is the investigation of choice in obtunded patients. If the patient is alert , magnetic resonance imaging of cervical cord done.

If weakness is lower motor neuron , myopathic or uncertain in origin, the clinical approach begins with blood studies to determine the level of muscle enzymes and electrolytes and electromyography and nerve conduction study(1).

The case discussed here had predominant lower motor type of

weakness due to electrolyte abnormality – hypokalemia.

HYPOKALEMIA

Hypokalemia is defined as a plasma potassium concentration <3.5 mEq/L. Plasma potassium is maintained in the narrow range of 3.5mEq/L – 5.5 mEq/L. Hypokalemia may result from conditions as varied as transcellular shift (movement of K+ from serum into cells as a result of insulin use or alkalosis) , malnutrition or decreased intake and parenteral nutrition. Renal losses such as Renal tubular acidosis , Bartter's syndrome , Fanconi syndrome, hyperaldosteronism, magnesium depletion, Leukaemi , cushing syndrome. Gastrointestinal losses such as vomiting , pyloric stenosis, diarrhea, enema's or laxative use, gastric aspiration, ileal loop and medication effects (2 , 4)

Many drugs may lead to hypokalemia , drugs like diuretics (the most common cause) , beta adrenergic agonists, theophylline, steroids and aminoglycosides (2 , 3). The drugs can lead to hypokalemia in the therapeutic and toxic doses (3).

In the present case he was taking oral prednisolone, a corticosteroid from four months causing hypokalemic quadriparesis.

PREDNISOLONE

Prednisolone is a short to medium acting glucocorticoid (9).

The effects of corticosteroids are numerous and widespread and include alterations in carbohydrate, protein, and lipid metabolism. Maintenance of fluid and electrolyte balance : and preservation of normal functions of cardiovascularsystem , immune system, the kidney, skeletal muscle, endocrine and nervous system(10).

Long term therapy with corticosteroids slightly reduces serum potassium and is occasionally accompanied by hypokalemia and severe metabolic abnormalities. The maximum decrease in potassium serum levels is reached in the first week of corticosteroid treatment (5). High doses of hydrocortisone (>100mg/day) can induce severe hypokalemia by causing sodium retention and excessive renal potassium loss secondary to their significant mineralocorticoid effect (6). Dexamethasone , methyl prednisolone, cortisol and prednisolone seem to be less likely to induce hypokalemia(7). Severe potassium depletion can occur when these drugs are prescribed with concomitant diuretics (8).

In our case there are no other causes of hypokalemia other than the drugs prednisolone and griseofulvin. There was no evidence found in the literature for hypokalemia caused by griseofulvin, an antifungal agent. In our case quadriparesis improved after the correction of hypokalemia and stoppage of the offending drug(oral prednisolone).

Prednisolone a corticosteroid can cause significant hypokalemia when used for longer duration. Here in we report this case as oral prednisolone induced hypokalemia causing quadriparesis.

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