Rhabdomyolysis results from acute damage of the skeletal muscle brought on by various conditions such as muscle disease, excessive muscle activity, hereditary muscle enzyme defects, dehydration, drugs, infections and toxins. Hypokalemia is a recognized but relatively rare cause of rhabdomyolysis. Potassium is the major intracellular cation, which is critical for the activity of specific enzymes, muscle cell contraction, blood flow during exercise and structural integrity. Hypokalemia may cause rhabdomyolysis by three mechanisms: Inadequate vasodilation of arterioles and capillaries that perfuse exercising muscle, suppression of synthesis and storage of glycogen and deranged ion transport across the cell membrane.[1,2]

There have been very few reports of rhabdomyolysis secondary to PA.[3,4] Here, we report a case of hypokalemic paralysis and rhabdomyolysis secondary to aldosterone-producing adrenal adenoma.

A 42-year-old woman presented with acute onset lower limb paralysis associated with severe proximal muscle pain. These symptoms were not associated with respiratory difficulty, dysphagia, dysarthria or sensory symptoms and there was no associated backache, fever, rash or constitutional symptoms. She had a 2 year history of hypertension, which was poorly controlled despite treatment with a combination of nifedipine, atenolol and losartan. She was treated by a general practitioner and at initial diagnosis of hypertension had undergone biochemical evaluation including serum electrolytes, which had been within normal limits. She experienced two similar, but short lasting episodes of weakness in the preceding year, but had not been investigated. Three days prior to admission she had been given Frusemide as add on therapy for control of hypertension. Her blood pressure on admission was 160/100 mmHg. Clinical examination favored an acute onset myopathy with symmetric flaccid paralysis of lower limbs (muscle power - 3/5) with normal muscle power (5/5) in the upper limbs.

Initial work-up revealed severe hypokalemia (potassium - 2.0 mmol/l) with an elevated creatine
kinase (CK) of 11347 IU (16-190) and increased lactic dehydrogenase of 856 IU/l (240-480). Patient's urine was positive for myoglobin with a high urinary myoglobin 4,170 µg/l. Serum creatinine was 130 µmol/l. In addition, patient had high urinary potassium of 19.45 mmol/24 h with metabolic alkalosis (pH - 7.540, HCO₃⁻38 mmol/l, base excess - 15 mmol/l and PCO₂-44 mmHg).

The other biochemical values on initial evaluation were as follows; serum aspartate aminotransferase - 244 IU/l (up to 35 IU/l); alanine aminotransferase - 193 IU/l (2-40); Sodium - 145 mmol/l; blood urea - 45 mg/dl; calcium - 0.9 mmol/l (1.12-1.32); phosphorus - 2.7 mg/dl (2.7-4.5); magnesium - 1.4 mg/dl (1.58-2.55); fasting blood glucose 110 mg/dl and the total protein was 7.6 g/dl with albumin 3.2 g/dl. Thyroid function tests were normal with a free T4 level of 1.02 ng/dl and thyroid-stimulating hormone 3.2 µIU/ml.

Based on the above findings, patient was diagnosed as having hypokalemic paralysis and rhabdomyolysis. Treatment was initiated by oral and intravenous supplementation of potassium and after 10 days, muscular strength and CK levels had returned to normal. Conduction studies and electromyography, which were performed after the correction of hypokalemia, were normal.

The coexistence of hypertension, hypokalemia with high urinary potassium loss in association with metabolic alkalosis raised the possibility of primary aldosteronism (PA), which lead to further hormonal evaluation. Aldosterone levels were 22,600 ng/dl (normal, 3.6-24.0 ng/dl) and the plasma renin activity (PRA) value was below the detection limit of our method (0.2 ng/ml/h). Aldosterone/renin ratio (ARR) was 113,000 ng/ml per ng/ml/h (<10). Abdominal ultrasound did not reveal adrenal lesions, but abdominal computed tomography scan showed a 20 mm × 16 mm hypodense mass in the medial limb of the right suprarenal gland [Figure 1]. She underwent right sided adrenalectomy and pathological examination of the gland confirmed a 20 mm adrenal adenoma. Post-operatively the patient was normokalemic without spironolactone and the PRA was 0.52 ng/ml/h; aldosterone was 1.3 ng/dl with a reduced ARR of 2.5 (<20) ng/ml per ng/ml/h. After 4 weeks of discharge, her BP was normal without any antihypertensives and she was normokalemic with normal muscle strength.

**DISCUSSION**

It is estimated that PA affects 5-13% of patients with hypertension. Adrenal adenoma is the most common cause of PA in which there is autonomous and inappropriately high aldosterone production by the adrenal glands, which is not suppressed by sodium loading. The other causes are unilateral or bilateral adrenal hyperplasia and the inherited condition glucocorticoid-remediable aldosteronism. Inappropriate production of aldosterone causes hypertension with suppression of plasma renin with sodium retention and potassium excretion, which if prolonged may lead to severe hypokalemia. It is also important to note that only a minority of patients with PA are hypokalemic. Recent studies have shown that only 9-37% of patients with PA had hypokalemia.

This case illustrates several important issues with regard to PA and hypokalemia. Although PA leads to hypokalemia owing to tubular loss of potassium, in most instances the potassium level may be well within normal range on routine biochemical evaluation as there are a multitude of factors contributing to renal potassium handling. Most often severe hypokalemia is triggered when these patients are exposed to an additional insult such as diuretics. In this patient, the addition of furosemide most probably aggravated the hypokalemia, leading to rhabdomyolysis. In addition although this patient had encountered two episodes of weakness in the year preceding the episode of rhabdomyolysis, her electrolyte levels were not re-tested. It would have been prudent to evaluate the electrolyte levels at that stage as they may have been related to transient spells of hypokalemia.

It is very important that general practitioners are made aware of these aspects as a majority of patients with hypertension are managed at primary care setups by general practitioners.

**CONCLUSION**

In conclusion, this case clearly highlights the importance of screening for PA even in the absence of hypokalemia, especially in patients with resistant hypertension. The
undiagnosed patient with PA is especially vulnerable to hypokalemia if diuretics are added for blood pressure control.

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