Poster

Severe Hypokalemia and Neuromuscular Weakness as the Initial Manifestation of Primary Hyperaldosteronism in an Adult Woman: A Case Report

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Primary hyperaldosteronism is characterized by autonomous and excessive secretion of aldosterone, independent of the renin–angiotensin system. The most common causes include adrenal adenomas and hyperplasia of the zona glomerulosa. Clinically, it manifests mainly resistant arterial hypertension and electrolyte disturbances, particularly hypokalemia, which may lead to significant neuromuscular alterations. We report the case of a 41-year-old Black woman with a history of arterial hypertension diagnosed eight years prior, under treatment with amlodipine 10 mg/day. She presented to the cardiology clinic with complaints of progressive muscle weakness and lower-limb paresthesias, associated with gait difficulty, with a three-month course. She denied diabetes mellitus, surgeries, or allergies, reported social alcohol consumption, and denied smoking. Family history: both parents were hypertensive. On physical examination, she was conscious, oriented, and in fair general condition. Cardiovascular, respiratory, and abdominal examinations were unremarkable. Blood pressure was 153/93 mmHg and heart rate 74 bpm. Muscle strength in the lower limbs was reduced (2/5). Complementary tests revealed serum potassium of 1.8 mEq/L, preserved renal function, and a normal lipid profile. Plasma aldosterone was 105 ng/dL (reference range 5.0–30.0 ng/dL). The ECG showed sinus rhythm with nonspecific ventricular repolarization abnormalities. Echocardiography revealed preserved systolic function, with no significant changes (LVEF = 61%). Abdominal CT identified a nodular lesion in close contact with the left adrenal gland, suggestive of a low-lipid-content adrenal adenoma. The patient was hospitalized for five days, receiving intravenous potassium supplementation and spironolactone (100 mg/day), with clinical improvement and serum potassium rising to 3.4 mEq/L. Subsequently, abdominal MRI performed at Hospital da Luz in Lisbon confirmed a left adrenal adenoma, and she underwent left adrenalectomy. Postoperatively, both serum potassium and plasma aldosterone levels normalized without medication. Blood pressure remained between 120/70 and 130/80 mmHg. This case illustrates a typical presentation of primary hyperaldosteronism, in which severe hypokalemia was associated with marked neuro-

muscular deficit. The diagnosis was supported by clinical, laboratory, and imaging findings. Unilateral adrenalectomy represents a curative treatment for patients with adrenal adenoma, as demonstrated in this case. Primary hyperaldosteronism should be considered in the differential diagnosis of patients with hypertension associated with severe hypokalemia and neuromuscular symptoms. Appropriate investigation and timely treatment, including surgical intervention when indicated, can be curative and prevent long-term cardiovascular and metabolic complications.

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