

Primary hyperaldosteronism manifested by rhabdomyolysis

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Abstract

Primary hyperaldosteronism might be asymptomatic. In symptomatic patients, the typical presentations are hypertension and hypokalemia. Rhabdomyolysis is an extremely rare presentation of primary hyperaldosteronism. We reported a 46-year-old male patient with a 4-year history of hypertension. He presented with acute limb weakness and laboratory data revealed severe hypokalemia and high creatine phosphokinase level. Further endocrine and image survey suggested functional adrenal adenoma and primary hyperaldosteronism. The patient was successfully treated with laparoscopic adrenalectomy.

Keywords

rhabdomyolysis; hyperaldosteronism; hypokalemia; adrenalectomy

Abbreviations

PA: Primary Hyperaldosteronism; CPK: Creatine Phosphokinase; ARR: Aldosterone to Renin Ratio

Introduction

Rhabdomyolysis can be caused by various conditions such as muscle diseases, intense exercise, dehydration, toxins, infections, various drugs and electrolyte disorders [1].

Electrolyte imbalances associated rhabdomyolysis include hypokalemia, hypophosphatemia, hypocalcemia, hyponatremia, hypernatremia and hyperosmotic states [2]. In some extreme conditions, primary hyperaldosteronism (PA) can induce excessive aldosterone secretion leading to profound potassium excretion and severe hypokalemia followed by rhabdomyolysis [3]. Here, we present a rare case of PA manifested by rhabdomyolysis.

Case Presentation

A 46-year-old male patient presented with severe muscle weakness of both upper and lower

extremities. He denied recent intensive exercise, infections, traumatic events or possible drug intoxication. His medical history only revealed a 4-year history of hypertension that was treated with Acebutolol, Amlodipine and Valsartan. By reviewing his initial medical record, muscle weakness was noted in four limbs (grade 3-4, using the Medical Research Council scale). There were no symptoms or signs of sensory impairment. The initial laboratory examination showed extremely low serum potassium (1.7 mmol/L) and elevated creatine phosphokinase (CPK) (4532 U/L; reference range, 10–160 U/L). His serum creatinine was 1.02 mg/dl and the urinalysis was normal. Based on these clinical findings, the patient was first diagnosed with severe hypokalemia and rhabdomyolysis. Aldactone 50mg PO TID and intravenous potassium supplementation were initiated. The muscle weakness and the elevated serum CPK levels improved gradually over a 5-day course of treatment. Endocrine survey revealed low plasma rennin activity (0.13ng/mL/hr; range 0.8–1.36 ng/mL/hr), normal aldosterone level (6.58ng/dL; range, 5–19.4 ng/dL) and high aldosterone to renin ratio (ARR, 50ng/dL per ng/mL/hr; normal range, less than 20-25ng/dL per ng/mL/hr). Acebutolol and Valsartan were withheld for 6 weeks. Rechecked blood work showed high serum aldosterone level (25.6ng/dL) and normal serum renin level (3.81pg/ml).

For suspected PA, abdominal CT scan found a 1.6cm hypodense tumor over the left adrenal gland (Figure 1).

Under the impression of PA caused by an aldosterone-producing adenoma, the patient received laparoscopic left adrenalectomy uneventfully. The pathology confirmed an adrenal cortical adenoma and the resection margin was free of tumor (Figure 2).

Two months after the operation, the patient became normotensive and his serum potassium level returned to normal levels.

Discussion

The presenting case indicates that PA with severe hypokalemia might manifest with generalized weakness due to rhabdomyolysis. When evaluating hypertensive patients with muscle weakness, it is mandatory to evaluate serum electrolytes, especially the serum potassium levels. Potassium ion plays a very important role in mediating the rise in blood flow in contracting skeletal muscle. In state of severe hypokalemia, muscle necrosis may occur due to ischemia [4]. Including our case, most reported cases of primary hyperaldosteronism related to hypokalemic rhabdomyolysis had potassium levels less than 2.0mEq/L [5-7].

The aldosterone/plasma renin activity ratio (ARR) is a frequently used screening tool for PA. However, many antihypertensive agents, including angiotensin converting enzyme inhibitors, angiotensin receptor blockers, spironolactones and beta blockers may interfere with interpretation of the initial endocrine survey [8]. In this case, the measured data showed high ARR but normal aldosterone level. After withholding Acebutolol and Valsartan for 6 weeks, the hormone test showed high serum aldosterone level and normal serum renin level. A careful review of the patient's drug history and an adequate drug washout period before blood test are necessary to avoid misdiagnosis.

The main causes of PA are aldosterone-producing adenomas and bilateral hyperplasia. Abdominal CT scan is an important examination for differential diagnosis before surgery. Lingam et al found that CT had a sensitivity of 100% when a mean limb width greater than 3 mm was used for diagnosing bilateral adrenal hyperplasia, and a specificity of 100% when the mean limb width was 5 mm or greater [9]. If CT scan showed no adrenal lesion or bilateral adrenal lesion, preoperative NP-59 scintigraphy or adrenal vein sampling are mandatory for surgical decision making [10,11].

Early initiation of treatment is crucial and should not be delayed. Laparoscopic adrenalectomy is the gold standard treatment for PA caused by an aldosterone-producing adenoma. Partial adrenalectomy is commonly performed in patients with bilateral adrenal involvement or a solitary adrenal gland because of previous surgery but additionally in those with a normal contralateral gland. Within the recent decades, there has been an increasing trend toward partial adrenalectomy worldwide. In some patients, PA may occur with multiple adrenal adenomas. Ishidoya et al. reviewed 92 patients with PA received either partial or total adrenalectomy and found that 27% of specimens demonstrated multiple adenomas in the specimen [12]. Risks and benefits must be carefully evaluated before choosing total or partial adrenalectomy.

For patients unfit for, or unwilling to undergo surgery, medical treatment with mineralocorticoid antagonist is an alternative treatment option. Both surgical and medical treatment are not only effective at blood pressure control and but also help prevent PA associated cardiovascular complications such as stroke, myocardial infarction and atrial fibrillation [13].

Figures



Figure 1: Abdominal CT scan showing a 1.6cm hypodense tumor over the left adrenal gland (indicated by the arrow)



Figure 2: Cross-section of specimen showing one single 1.6cm adenoma in the left adrenal gland

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