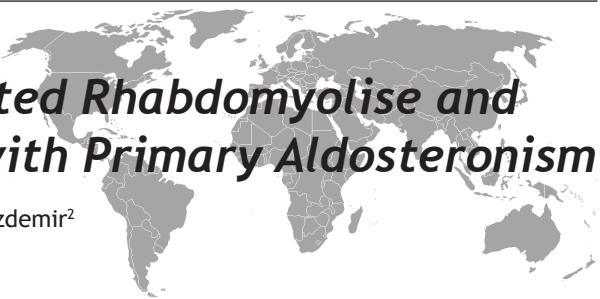


Severe Hypokalemia-Associated Rhabdomyolise and Unusual Poliuria in Patient with Primary Aldosteronism

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ABSTRACT

Primary aldosteronism is a syndrome that is characterized with hypertension, hypopotasemia, high level of plasma aldosterone, and low plasma renin activity. The case we present is a 56-year-old male who referred to our neurology clinic with proximal muscle weakness and fatigue. Because of uncontrolled blood pressure, a cardiology consultation was performed for the planning of anti-hypertensive treatment. As prolonged QT intervals and giant U waves due to serious hypokalemia ($K^+ : 1.04$), cardiology clinic took over the patient for risks of arrhythmia. After primary hyperaldosteronism diagnosis was established, the treatment was initiated and severe polyuria developed during the treatment (19L/day).

Key words: Primary aldosteronism, secondary hypertension, rhabdomyolysis, polyuria, hypokalemia

Primer Aldosteronizmli Hastada Ciddi Hipokalemi İlişkili Rabdomiyoliz ve Olağandışı Polüüri

ÖZET

Primer aldosteronizm hipertansiyon, hipopotasemi, yüksek plazma aldosteron seviyesi ve düşük plazma renin aktivitesiyle karterize bir sendromdur. Bizim takdim ettiğimiz hasta proksimal kas güçsüzlüğü ve yorgunluk şikayetleriyle nöroloji kliniğine başvuran 56 yaşında bir erkek hastaydı. Kontrolsüz kan basıncı nedeniyle anti-hipertansif tedavi planlanması amacıyla kardiyoloji konsultasyonu istenmiş. Ciddi hipokalemeye bağlı ($K^+ : 1.04$) uzamış QT aralığı ve dev U dalgaları olması nedeniyle hasta kardiyoloji kliniğine aritmî riskleri açısından devalındı. Primer hiperaldosteronizm teşhisi konulduktan sonra tedavi başlandı ve tedavi boyunca hastada ciddi polüüri gelişti (19 L/gün).

Anahtar kelimeler: Primer aldosteronizm, sekonder hipertansiyon, polüüri, rabdomiyoliz, hipokalemia

INTRODUCTION

Primary aldosteronism (PA) was first defined by Jerome Conn in 1955 (1). PA is a syndrome that is characterized with aldosterone synthesis and release in big amounts from the surrenal cortex because of unilateral adenoma or bilateral hyperplasia. The most frequent reason of PA is adrenocortical adenomas that are mostly smaller than 2 cm (Conn syndrome). It is estimated that it is the reason of 2% of all hypertension cases (2, 3). Patients could suffer from hypertension-related headache, hypokalemia-related polyuria, nocturia, paresthesia and paralysis (4-7). Routine biochemistry findings are not generally diagnostic in hypertensive patients but hypokalemia with metabolic alkalosis and serum sodium levels close to high-normal

should remind one of PA (8). In PA diagnosis, the following criterias support the diagnosis: 1- Plasma aldosteron/renin ratio > 30 and plasma aldosterone level $> 25 \text{ ng/dL}$ provide a sensitivity 90% and a specificity 91% for the diagnosis of primary aldosteronizm (9), 2- No aldosterone suppression in sodium loading test, 3- Unilateral adenoma that is detected with high-resolution computed tomography (HRCT) (10). The case was presented because of severe hypokalemia and associated rhabdomyolysis development and extraordinary polyuria development about 19 L/day. after the spironolactone treatment.

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CASE

58 year olds male referred to neurology clinic because of fatigue, leg pain, and troubles. A cardiologist consultant was called by neurologist physician because of uncontrolled blood pressure. Severe QT prolongation (600 msec) was detected in the electrocardiography and seriously hypokalemia (1.04mEq/L). He was transferred to cardiology intensive care unit due high risk of arrhythmia. His blood pressure: 170/95 mmHg, heart and respiratory rate were 84 beats/min, 18/min respectively. There was no abnormal finding on cardiovascular examination. Muscle strength in upper extremities 5/5, lower extremities proximal 3-4/5, distal 4/5 was found on neurological examination. Biochemical examination: Urea:19 mg/dl, Creatinine:1,1 mg/dl, Sodium:140 mEq/L, K+:1.04 mEq/L, Aspartate aminotransferase:166 U/L, Alanine aminotransferase: 58 U/L, Creatinine phosphokinase: 10192 U/L, Renin activity: 22 ng/ml/s (0.2-3.4), Aldosterone: 340 pg/ml (20-240). Arterial blood gas: pH: 7.49, partial carbondi oxide pressure: 47 mmHg, partial oxygen pressure: 67 mmHg, Bicarbonate: 35 mmol/L, and oxygen saturation was 93.5%. Spot urine K⁺ was normal. Electro neuro-myography findings were compatible with motor and sensorial neuropathies in lower extremities. Patient didn't use any medication that could cause myopathy, we diagnosed hypokalemia-associated rhabdomyolysis. The patient was initiated 160 mEq/day intravenous K⁺ replacement, spironolactone 100mg/day, and ramipril 10 mg/day. Two days later when K⁺ level reached to 3mEq/L, intravenous K⁺ replacement was stopped and oral K⁺ replacement was continued two days. Daily urine volume was 3.5 liters in the beginning, it was 9 L in the first day of the treatment, 16 L in the second day, and 19 L in the third day. Besides, polydipsia developed si-

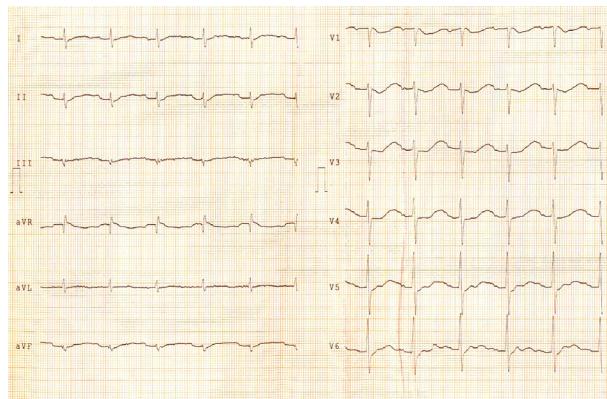


Figure 1. QRS prolongation in electrocardiography

multaneously. The patients' liquid intake was restricted then polyuria and polydipsia improved. The patient lost 9 kg in one week. Although abdominal ultrasonography was normal, a low-density nodular lesion (2cm) was detected in left surrenal gland by multislice CT (figure-2). Laparoscopic left surrenalectomy was performed. In the pathological examination of the specimen, an adenoma with 3x2,5x1.5 cm was detected. The microscopic and macroscopic views of the surrenal adenoma were shown in Figure 3-4. The patient is now healthy and his blood pressure is regulated with 16 mg/day candesartan treatment.

DISCUSSION

Clinical symptoms of primary aldosteronism are not specific. Patients could be either asymptomatic or referred



Figure 2. View of left surrenal adenoma in multi-slice CT



Figure 3. Macroscopic view of surrenal adenoma

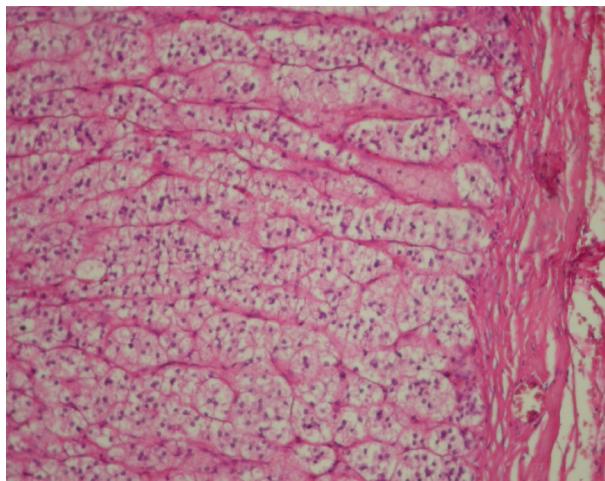


Figure 4. Microscopic view of surrenal adenoma

to physician with serious symptoms. Patients could describe hypertension-related headache, polyuria that was caused by hypokalemia, nocturia, paresthesia, paralysis and muscle cramps as symptoms (4-7). Muscle weakness due to hypokalemia, paresthesia, paralysis, and tetanies are especially reported in cases from Asia region (8). In literature, it is reported some cases of rhabdomyolysis and acute renal failure due to hypocalcemia and hypernatremia (10,11).

Polyuria could rarely be seen in cases with PA. It was reported that the mechanism was not sensitive to antiuretic hormone (ADH) in collector tubules because of hypokalemia. This caused the development of nephrogenic diabetes insipidus (12). It is not known exactly how hypokalemia affects the response to ADH. While our case never defined polyuria before, a progressive increase in urine was observed after spironolactone treatment and K⁺ replacement. We have not encountered any data in literature about spironolactone treatment and/or K⁺ replacement that caused polyuria and nephrogenic diabetes insipidus in PA cases. We concluded that this situation might be two reasons: intravenous fluid replacement in an effort to correct hypokalemia and/or increased fluid retention due to hyperaldosteronism, spironolactone triggering polyuria breaking the cascade of aldosteron. Polyuria decreased after liquid restriction during the treatment. Spironolactone treatment before 3-4 weeks from surgery is suggested in these cases. It is emphasized that delayed surgery could be advantageous in decreasing in the plasma aldosterone level and regulation of the blood pressure (13). Our case was operated in seventeenth day and no complication was seen intraoperatively. Interestingly, no serious arrhythmia was seen in

intensive care ECG records despite serious hypokalemia and a prolonged QT interval.

As a result, PA should be eliminated in patients with hypokalemia who especially defines symptoms like polyuria, nocturia, paresthesia, and paralysis. The mechanism of polyuria in patients with PA remains unclear and further investigation results may contribute in this area.

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