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A Neglected Cause of Hypertension in the Emergency Department: Primary Hyperaldosteronism

Acil Serviste İhmal Edilen Bir Hipertansiyon Nedeni: Primer Hiperaldosteronizm

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ABSTRACT

Primary hyperaldosteronism (PHA) is one of the leading causes of persistent hypertension. In this paper, we report the case of a 57-year-old woman who presented at our emergency department (ED) with muscle weakness and persistent hypertension. The initial examination revealed high blood pressure (232/136 mmHg), muscle weakness, hypokalemia (K^+=2.1 mEq/L), and metabolic alkalosis (pH=7.47). Initial ED therapy failed. Because persistent hypokalemia and arterial hypertension with metabolic alkalosis raised the suspicion of PHA, we obtained measurements of renin activity (0.3 ng/ml/h, normal values: 0.2-2.8 ng/ml/h), aldosterone concentration (534.41 pg/ml, normal values: 10-160 pg/ml) and the ratio of aldosterone concentration/renin activity (178 ng/dl/ng/ml/h). There was no surrenal adenoma on the abdominal computerized tomography. The patient was administered a daily 100 mg dose of aldactazide. At the end of two weeks, the patient was discharged with normal blood pressure and K^+ level. PHA is one of the leading causes of persistent hypertension. The emergency physician should pay close attention to patients with hypokalemia and metabolic alkalosis accompanying symptoms such as persistent hypertension, in order to diagnosis PHA early and reduce cardiovascular complications.

Keywords: Emergency departments, hyperaldosteronism, hypertension

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ÖZET

Primer hiperaldosteronizm (PHA) dirençli hipertansiyon önde gelen nedenlerinden biridir. Bu yazıda acil servise dirençli hipertansiyon ve kas güçsüzlüğü ile başvuran 57 yaşında bir kadın hasta sunuldu. Elli yedi yaşındaki hipertansif kadın hasta kas güçsüzlüğü ve dirençli hipertansiyon ile acil servise başvurdu. İlk fizik muayene ve laboratuvar incelemesinde yüksek kan basıncı (232/136 mmHg), kas güçsüzlüğü, hipokalemi (K^+=2.1 meq/l) ve metabolik alkaloz (pH=7.47) saptandı. Acil Serviste uygulanan ilk tedaviden sonuç alınamadı. Dirençli hipokalemi, metabolik alkaloz ve arteryel hipertansiyon nedeniyle, hastada PHA dan şüphe edilerek hasta hastaneye yatırıldı. Elde edilen, plazma rennin aktivitesi 0.3 ng/ml/saat (normal değerler 0.2-2.8 ng/ml/saat) Plazma aldosteron konsantrasyonu 534.41 pg/ml (normal değerler: 10-160 ml/pg) ve aldosteron konsantrasyonu/Renin Aktivitesi/oranı 178 ng/dl/ng/ml/h bulundu. Batın tomografisi normaldi. Hastaya günlük 100 mg Aldactazid tedavisi uygulandı. İki hafta normal kan basıncı ve K^+ düzeyi ile hasta taburcu edildi. PHA dirençli hipertansiyonun önde gelen nedenlerinden biridir. Acil hekimi hipokalemi ve metabolik alkalozun eşlik ettiği dirençli hipertansiyona sahip hastalarda erken tanı ve kardiyovasküler komplikasyonlardan korunmak için PHA'i tanıda düşünmelidir.

Anahtar Kelimeler: Hipertansiyon, hiperaldosteronizm, acil servis

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Introduction

Secondary causes of hypertension are common and predominantly go unrecognized in patients with a hypertensive emergency. Primary hyperaldosteronism (PHA) is one of the most frequent causes of secondary hypertension.

The most frequent findings of PHA are arterial hypertension, hypokalemia, suppressed plasma renin activity (PRA) and increased plasma aldosterone concentration (1).

The majority of PHA cases are misdiagnosed as essential hypertension; on average, the patients who were treated for hyperaldosteronism had a three-year history of hypertension (2).

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The accurate diagnosis of patients with persistent hypertension and PHA is difficult in emergency departments. The emergency physician should keep PHA in mind and look for related signs and symptoms in patients with persistent hypertension.

The goal of this case presentation is to remind emergency physicians that PHA is a common and curable form of secondary hypertension, and that early diagnosis and accurate treatment of PHA reduces cardiovascular complications.

In this paper we report the case of a 57-year-old woman with a history of arterial hypertension for the previous 5 years that had been treated with 5 mg ramiprile daily. She presented to our emergency department with severe muscle weakness of all limbs and persistent hypertension; these symptoms are actually the first manifestation of PHA.

Case Report

A 57-year-old hypertensive woman (on 5 mg of ramiprile for the last five years) presented at our emergency department with severe muscle weakness of all limbs. Over the previous 15 days, she had visited the hospital several times due to high blood pressure. Her treatment regimen was modified to olmesartan medoxomil, but there was no change in her blood pressure. She had no additional complaints, including vomiting or diarrhea.

The initial physical and laboratory examination revealed arterial blood pressure (232/136 mmHg), muscle weakness, hypokalemia (K^+=2.1 mEq/L), and mild metabolic alkalosis (arterial blood gasses: pH=7.47, pCO_2=41, pO_2=61, HCO_3=23, SO_2=93%). The ECG was normal.

Based on these initial observations, an IV nitrate infusion of 20 mcg/min was started promptly in order to regulate her arterial blood pressure, and potassium replacement treatment was administered. Two hours later, the patient's potassium was 1.9 mEq/L and tendon Achilles blood pressure (TA)=195/100 mmHg. An additional 2 mg IV $MgSO_4$ was administered to regulate the patient's potassium level, but there were no further changes in the potassium level.

The combination of hypokalemia with arterial hypertension raised suspicions of PHA; therefore, we obtained PRA (plasma renin activity) and PAC (plasma aldosterone concentration) measurements. PRA was in the low normal range (0.3 ng/ml/h, normal values: 0.2-2.8 ng/ml/h) with an increase of PAC (534.41 pg/ml, normal values: 10-160 pg/ml) and the ratio PAC/PRA was 178 ng/dl/ng/ml/h.

There was no surrenal adenoma on the abdominal computerized tomography. The patient was diagnosed with idiopathic PHA. She was administered a daily 100 mg dose of aldactazide and ACE-inhibitor treatment. At the end of two weeks, her TA before discharge was 117/80mmHg, and K was 4.0 mEq/L.

Discussion

Hypertensive emergency is a common and potentially life-threatening condition. Secondary causes of hypertension are common and predominantly go unrecognized in patients with hypertensive emergency. PHA is a common and curable form of secondary hypertension. The emergency physician should pay close attention to patients with hypokalemia and metabolic alkalosis accompanying symptoms such as persistent hypertension. Early diagnosis and accurate treatment of PHA reduces cardiovascular complications. Patients affected by primary aldosteronism are prone to cardiovascular events and target-organ damage. Börgel J et al. (1) reported that, out of 161 patients with hypertensive urgency/emergency, we identified 150 secondary causes of hypertension, of which 145 (97%) were previously unknown (mean duration of disease 12.7 years), and a high proportion of end-organ damage (12.4% atrial fibrillation, 9.3% history of stroke/transient ischemic attack). Rossi et al. (2) reported that the patients who were treated for hyperaldosteronism had an average of three-year history of hypertension. In the study by Fukudome et al. (3), the history of hypertension was significantly longer, 6 years on average. In our patient, similarly, the duration was five years.

A complete ambulant screening for secondary causes of hypertension is expensive, time consuming, and difficult to coordinate for the general practitioner and the patient. Thus, few hypertensive patients undergo the full diagnostic course to exclude secondary causes, and if they do so, it is usually only after a long duration of their disease with the consequent end-organ damage and potentially-inadequate oral therapy.

Another possible reason for not detecting secondary causes is that several different specialists and specialties are needed for a complete screening. Nephrologists, endocrinologists or cardiologists usually perform their own studies relating to their specialty.

Previously, it was thought that the patients with PHA attend the emergency department for help related to the symptoms of hypertension and hypokalemia. Recent studies have reported, however, that only a minority (from 9% to 37%) of patients with PHA are hypokalemic (4). Patients with PHA can manifest nonspecific signs and symptoms, like weakness, so managing the patient and performing differential diagnoses for them is much more difficult than for patients with essential hypertension (4).

In our patient, hypokalemia was not suspected initially as the cause of the symptoms, despite having a key role in such diagnoses. In PHA, hypokalemia occurs due to an increase in distal tubular exchange of Na, rather than secretion of K and H. The increased Na absorption and extracellular volume expansion lead to symptoms such as headache, fatigue, restlessness, cramps, paresis, tachycardia, constipation, vomiting, polyuria, and polydipsia (5). It is important to control hypertension in order to avoid its cardiovascular and renal effects.

Patients with severe, resistant hypertension appear to benefit from therapy, as shown by a reduction in cardiovascular complications (6).

Although PHA is an important cause, not all patients with hypertension should be suspected of having hyperaldosteronism. Kaplan has reported that hypertension was caused by PHA in less than 1 per cent of the patients, and unless there was persistent hypertension, hyperaldosteronism should not be suspected (6).

Conclusion

PHA is a common and curable form of secondary hypertension. The majority of these secondary causes were unrecognized previously

despite a long duration of illness and the potential for therapeutic benefit. The emergency physician should pay close attention to patients with hypokalemia and metabolic alkalosis accompanying symptoms such as persistent hypertension, in order to diagnose PHA. Early and accurate treatment of PHA reduces cardiovascular complications.

Conflict of interest

No conflict of interest was declared by the authors.

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