Primary hyperaldosteronism

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DEAR EDITOR

Six decades ago Jerome W Conn described a middle-aged woman with primary hyperaldosteronism (PA) associated with arterial hypertension and hypokalemic metabolic alkalosis, and this symptom-complex was later called Conn's syndrome [1]. PA is a major cause of secondary hypertension, with an incidence of about 10% in hypertensive patients [2]. People with PA due to adena or bilateral hyperplasia may also have periodic paralysis [2]. Worthy of note, vascular complications are more often associated with PA than with essential hypertension because of the increased intima-media thickness of the carotid artery [2].

Recently, Kobayashi et al. reported the evaluation of hormones other than aldosterone playing a role in arterial hypertension of 59 patients with PA - 30 with adenomas (53% males; mean age: 45.5 years, and 29 with bilateral hyperplasia (38 % males; mean age: 46 years) compared to 24 controls (50% males; mean age: 59.5 years) with essential hypertension [2]. They found no significant correlation of aldosterone levels with blood pressure, whereas ACTH levels affected the blood pressure in patients with aldosterone-producing adenoma [2].

Although PA more frequently occurs in middle-aged women, a 27-year-old Brazilian female was admitted because of refractory hypertension since she was 19 years old. Worthy of note, was the lack of hypokalemia during the early decade of disease [1]. Remarkable antecedents were two miscarriages and one occurrence of pre-eclampsia; furthermore, she had muscle weakness, paresthesia, cramps and hypertensive crisis [1]. Laboratory determinations revealed hypokalemia; elevated levels of plasma aldosterone and low levels of plasma rennin, with elevated aldosterone-to-renin ratio; normal levels of dopamine, total metanephrines and normetanephrines; and normal renin function [1]. Images abdominal CT revealed an ovoid hypodense mass with 7 HU in the right adrenal gland, which was surgically removed and the diagnosis of aldosteronoma was confirmed by histopathologic evaluation [1]. The patient remains asymptomatic and normokalemic without the use of antihypertensive drugs [1].

Moradi et al. described normotensive PA in a 27-year-old woman, a very uncommon condition (3). Further case studies reported over than 50% of cases without hyperkalemia, but normotensive PA has been scarcely described [1,3]. Currently, the involved physiopathology mechanisms are not entirely understood [3]. The Iranian young had muscle weakness, hypokalemia, and normal blood pressure; renal function, urinary cortisol, metanephrines and normetanephrines were normal, but blood levels of aldosterone were elevated and plasma renin activity was suppressed. Plasma aldosterone remained high after infusion of 2 liters of 0.9% saline solution [3]. Computed tomography (CT) study showed a mass (3x2x2 cm) in the left adrenal gland, which was removed and the diagnosis of adenocortical adenoma was established [3]. Her serum potassium determinations and mean blood pressure levels remained normal. The authors highlighted the rarity of the case, which might be related to early detection of PA before development of hypertension, lower level of vasoconstrictor system, higher level of vasodilator system; salt restriction was ruled out by saline infusion [3].

PA may evolve unsuspected for a long time in normokalemic or normotensive patients; research must be done on pathophysiology of hypertension in this entity [1-3].

REFERENCES

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