Research Paper Medical Science

COINCIDENCE OF PRIMARY ALDOSTERONISM AND RENAL ARTERY STENOSIS – A RARE CAUSE OF HYPERTENSION

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ABSTRACT

A case of primary aldosteronism (PA) coexisting with renal artery stenosis is reported. 77 year old woman was admitted to the unit of endocrinology due to resistant hypertension (despite a full recommended antihypertensive multidrug therapy) accompanied by severe headaches, weakness and dizziness. Laboratory findings showed abnormal levels of aldosterone, renin and serum potassium. Hormonal tests confirmed suspicion of PA. On CT scan the left adrenal gland adenoma was revealed. Six months after adrenalectomy (histopathological examination showed an aldosteronoma) high blood pressure with high level of aldosterone and low renin concentration was observed. Further investigation revealed stenosis of the right renal artery with enlarged right adrenal gland. The diagnosis of 'tertiary hyperaldosteronism' was suggested.
Such coincidence as a cause of high blood pressure is found in less than 2 % of all published hypertension studies and may complicate both diagnosis and proper treatment.

KEYWORDS hypertension, primary aldosteronism, renal stenosis

Introduction
Hypertension remains one of the most common diseases affecting more than 25% of the world population [1, 2]. Usually hypertension is of idiopathic origin, however, other diseases may cause high blood pressure, for example: renal diseases, cardiovascular malformations, drugs, hormonal disturbances - diabetes mellitus, acromegaly, pheochromocytoma, thyroid gland dysfunctions and primary aldosteronism. Coexistence of two or more forms of secondary hypertension in the same patient are very rare, but has been previously reported, and may suggest similar pathophysiological mechanism [3, 4].

In our report we would like to present a case of secondary hypertension caused by primary aldosteronism and renal artery stenosis. Such coincidence as a cause of high blood pressure is found in less than 2 % of all published cases of hypertension and may complicate both diagnosis and proper treatment [3, 4].

Case Report
A 77 year old woman with history of chronic coronary artery disease was admitted to the hospital for evaluation of resistant hypertension (despite a full recommended antihypertensive multidrug therapy) accompanied by severe headaches, weakness and dizziness. In her past history she underwent a primary coronary intervention of left anterior descendent coronary artery (LAD) due to an acute coronary syndrome, intracerebral stroke, vascular by pass because of an abdominal aortic aneurysm and a correction of left renal artery stenosis. Besides of them, she had a peacemaker implantation and suffered from a paroxysmal atrial fibrillation and diabetes mellitus type 2.

On admission she was hypertensive with a blood pressure of 200/110 mmHg, equal in both arms. On examination there were no symptoms and signs of catecholamine disturbances, thyroid gland abnormalities and Cushing's syndrome Renal bruit was not detected.

Routine investigation showed normal blood counts, normal blood urea nitrogen and creatinine with a low serum potassium of 3.45 mmol/l [normal range: 3.5 - 5.5]. Hormonal testing revealed normal thyroid and glucocorticoid function with fT3 - 2.21 pg/ml [normal range 1.71-3.71]; fT4 - 0.98 ng/dl [normal range 0.70 – 1.4), TSH - 0.98 mIU/ml [0.27 – 4.2], ACTH level - 23,6 pg/ml [normal range 10 - 60], cortisol following 1 mg of dexamethasone - 0.91 mcg/ml. Vanillmandelic acid secretion was 4.5 mg/24 h [normal range 2 – 12]. Screening labs for primary aldosteronism were performed. Plasma aldosterone was high – 74.3 ng/dl (determination of plasma aldosterone - RIA assay, DPC, Los Angeles, USA) accompanied by low serum renin concentration (radioimmunoradiometric assay, DSL, Webster, USA) – 5.4 pg/dl. Saline infusion test showed decreased aldosterone concentration, but it remained above the level defined for positive testing [5] (the results are shown in Table 1). These results confirmed primary aldosteronism.

Table 1 about this
The patient underwent computed tomography (CT) of the abdomen which revealed enlarged left adrenal gland with a 19 x 16 mm adenoma (Figure 1). Moreover, the patient refused

Figure 1 about this
adrenal venous sampling. Because of her past medical history (prothesis of aorta), a laparoscopic adrenalectomy was performed and the left adrenal mass was removed. The histopathological examination revealed a well defined adenoma consisted of mostly large and clear cells, some containing granular material, which responded to an aldosteronoma.

The recovery was uneventful on a low dose antihypertensive treatment but six months later a blood pressure was increased and symptomatic. She was readmitted for further examination. Laboratory findings included high plasma aldosterone – 64.6 ng/dl with low renin – 4.5 pg/ml and normal serum potassium. The results of saline infusion test were similar like before (Table 2).

Table 2 about this
She underwent another abdominal CT which showed stenotic right renal artery with enlarged right adrenal gland but without evident abnormal mass (adrenal hyperplasia?)(Figure 2).

Figure 2 about this

In respect of the patient age, previous surgery and accompanying diseases, the treatment of 25 mg/daily of eplerenone was started, with satisfactory blood pressure control and serum potassium.

The diagnosis of secondary hypertension and primary (tertiary?) hyperaldosteronism due to left adrenal adenoma and contralateral gland hyperplasia coexisting with bilateral renal stenosis was established.

Discussion

Coexistence of primary hyperaldosteronism and renal artery stenosis is an unusual cause of secondary and sometimes called "tertiary" hyperaldosteronism. There are only a few described cases of unilateral or bilateral adenomas of the adrenal gland accompanied by abnormalities in renal arteries. In older group of patients, stenosis of renal artery was usually due to atherosclerosis whilst in the younger group fibromuscular hyperplasia was found more frequently [6, 7, 8, 9]. Some authors connected this coincidence with very high levels of angiotensin-II observed in renovascular hypertension and prolonged adrenal stimulation in mechanism similar to tertiary hyperparathyroidism [10, 11].

In addition to the aldosterone-releasing influence of angiotensin-II, it was also shown to be a potent growth promoting peptide, especially in vascular smooth muscle cells and adrenal glomerulosa zone. Moreover, this proliferative potency of angiotensin-II was confirmed under experimental conditions in intact and regenerating rat adrenal cortex. Thus, elevated levels of angiotensin-II could contribute to the induction of an aldosteronoma [12].

In this brief report we present a case of a 77 year old woman with bilateral renal artery stenosis. The previously corrected left artery stenosis might have evoked left adrenal tumor with primary hyperaldosteronism probably due to prolonged high levels of angiotensin-II. The right renal artery stenosis dilatation was not performed in respect of patient's age and medical history, however, increased aldosterone concentration before left adrenalectomy could indicate the role of angiotensin-II as a tumor-promoting factor and needs further investigation [6, 13].

In conclusion, while treating patient with resistant hypertension one must remember about appropriate screening for secondary hypertension. Coexistence of renovascular abnormalities and primary hyperaldosteronism, first reported by Beevers et al. [14] complicates and prolongs the time to establish the cause of hypertension. However, proper diagnosis enables adequate management of the patient [4, 15].

Conflict of interests:

The authors have nothing to declare

Table 1

The results of standard saline infusion test at the first admission:

Time	Aldosterone [ng/dl]	Renin [pg/ml]
0'	74.3 ng/dl [Normal: 4-31]	5.4 pg/ml [Normal: 3.5-65.6]
240'	16.4 ng/dl [Normal: < 5.0]	5.4 pg/ml [not defined]

Table 2

The results of standard saline infusion test at readmission:

Time	Aldosterone [ng/dl]	Renin [pg/ml]
0'	64.6 ng/dl [Normal: 4-31]	4.5 pg/ml [Normal: 3.5-65.6]
240'	15.8 ng/dl [Normal: < 5.0]	4.7 pg/ml [not defined]

Figure Legend:

Figure 1:

An abdominal CT scan showing an adenoma in the left adrenal gland.

Figure 2:

An abdominal CT scan presenting an enlarged right adrenal gland.

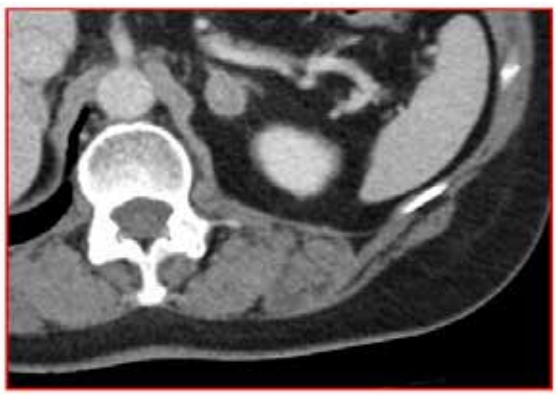

Figure 1

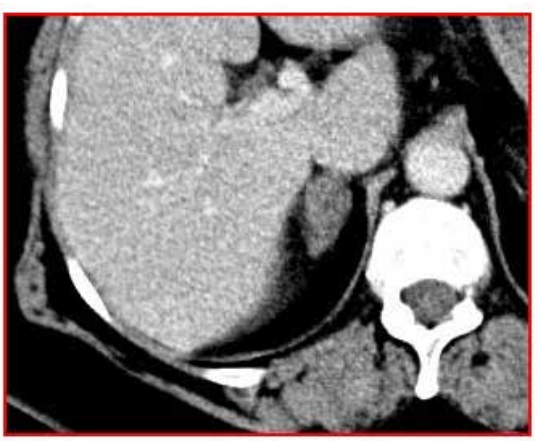

Figure 2

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