Primary Hyperparathyroidism and Cardiovascular Disease - A Case Report

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Introduction

Primary hyperparathyroidism (PHPT) is associated with increased prevalence of hypertension [1], left ventricular hypertrophy (LVH) that is independent on the presence of hypertension, diastolic dysfunction, valvular and myocardial calcification [2] and alterations of rennin-angiotensin-aldosterone (RAA) system [3]. These abnormalities in part explain the increased cardiovascular mortality in patients with PHPT [4].

The link between PHPT and arterial hypertension is at least in part sustained by the increased activity of the renin-angiotensin axis through the parathyroid hormone (PTH) stimulated renin production [2].

Whether PHPT can induce renal complications that may favors the appearance of arterial hypertension in the early stage of its development is not clear.

Clinical Case

A 37-year-old bike-trained man with history of hypertension for two years was admitted in our outpatient clinic. He suffered constipation and episodes of kidney stone disease over the previous seven years. There was no similar history in the family.

Anthropometric measures of vital signs at the time of admission were: body weight 78 Kg, height 170 cm, Body Mass Index 26.71 Kg/m², blood pressure 180/110 mmHg, heart rate 70 beats/minute, regular. He was on treatment for hypertension with amlopidine 10 mg - one tablet/day, started during the last month.

Routine lab analyses including blood cell count, renal and liver function tests and serum electrolytes (sodium and potassium) were performed. All the results were normal.

Electrocardiogram (ECG) showed sinus rhythm with normal AV conduction, normal axis and QT interval. On the 24 hour urine collection the albumin excretion was increased 77 mg/24 h (reference range 0-30). Proteincaria was not detected. Standard trans-thoracic echocardiogram revealed moderate concentric LVH (LV mass/height² = 57 g/m²²; relative wall thickness = 0.46), diastolic dysfunction due to prolonged LV relaxation (E/A = 0.9; Deceleration Time = 240 msec) and widespread intra myocardial dense spots, consistent with calcifications (Figure 1). The Echo-color-Doppler of epi-aortic vessel revealed an increased intima-media thickness. The optic fundus examination revealed grade II hypertensive retinopathy.

On the basis of the whole clinical presentation and the echocardiographic findings, we decided to further investigate the RAA system and the calcium phosphorus metabolism (Table 1).

Based on the presence of primary hyperparathyroidism, parathyroid scintigraphy was performed with 99mTc that showed right inferior parathyroid adenoma. Ultrasound scan for neck of the patient showed a 2.3 x 1.1 cm hypoechoic lesion on the right side near the postero-inferior surface of the thyroid gland (compatible with an enlarged parathyroid gland), whereas the thyroid was normal. Bone densitometry showed that the lumbar spine T scores of +0.6 and Z score of > 0.5.

Kidney ultrasound scan revealed bilateral nephrolithiasis and nephrocalcinosis, with reduced intra-parenchymal vascularization by colour-Doppler but normal renal arterial resistive index. He also performed contrast- CT of abdomen that showed no abnormalities in adrenal glands.

To achieve good control of blood pressure different combination of antihypertensive medications were used. The patient underwent surgery and histology confirmed the diagnosis of adenoma of the right inferior parathyroid gland. Post-operative laboratory test showed normal levels of serum calcium (9.5 mg/dL) and PTH (45 pg/mL).

The patient was revaluated 15 months after surgery. He was on treatment for hypertension with amlopidine 10 mg-one tablet/day and he achieved good blood pressure control.
Unfortunately data on RAA system after surgery was lacking. The pathogenesis of hypertension and related target organ damage.

Evidence that increased PTH was at least in part responsible for blood pressure control with one medication consistently with reduction in LV mass with regression of LV hypertrophy and good high prevalence of hypertension in patients with PHTH [9].

It is conceivable that elevated levels of circulating PHT may play a role in the pathogenesis of nephroangiosclerosis due to functional alterations of vascular smooth muscle cells [5]. Recently it has been also showed that increased level of aldosterone might explain the alterations of vascular smooth muscle cells [5].

In the context of primary hyperparathyroidism, the increased activity of the RAA system, as found in our patient, might be also correlated with the impairment of renal circulation, as suggested by the reduction of the intra-parenchymal vascularization [8].

Nineteen months after surgery our patient showed a significant reduction in LV mass with regression of LV hypertrophy and good blood pressure control with one medication consistently with evidence that increased PTH was at least in part responsible for the pathogenesis of hypertension and related target organ damage. Unfortunately data on RAA system after surgery was lacking.

Conflict of Interest

All Authors have no conflict of interest related to this manuscript.

References


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