Reversibility of myocardial hypertrophy 8 years after adrenal adenoma excision and drugs and alcohol addiction withdrawal

Gian Piero Carboni

Department of Nuclear Cardiology, Università Campus Bio-Medico, Rome, Italy

Correspondence to
Professor Gian Piero Carboni,
g.carboni@unicampus.it

DESCRIPTION

In November 2005, a 37-year-old patient presented to us with a systolic blood pressure (BP) >180 Hg and a diastolic BP >110 Hg and a history of dramatic hypertensive crises despite adherence to treatment with several antihypertensive drugs. He worked as a waiter in a nightclub where he was exposed to tiredness and fatigue while working nights. He sniffed at least 2 g of cocaine, smoked around 2 g of hashish and drank more than 500 ml of brandy nearly every night. This habit lasted for 5 years. Despite the presence of a positive direct relationship between cocaine, hashish, alcohol abuse and hypertension,^{1 2} the patient underwent a clinical workup to rule out other causes of secondary hypertension. The findings of hypokalaemia and increased values of aldosteronaemia and aldosteronuria suggested a diagnosis of primary aldosteronism. Echocardiography found a severe left ventricular hypertrophy (LVH). Abdominal CT identified a mass on the left adrenal gland (figure 1). The gland was excised by laparoscopic surgery (figure 2), and histological examination of the mass indicated adrenal adenoma.³ After surgery, the patient began a new job as a clerk with a normal working day and gradually discontinued drug and alcohol abuse. A daily treatment with 240 mg of verapamil and 50 mg of losartan maintained normal BP values. After an 8-year follow-up, echocardiography

Figure 1 Abdominal CT—axial view: adrenal adenoma (red arrow).

Figure 2 Excised adrenal gland and the adenoma (black arrow).

showed a reduction of the LV mass and LVH⁴ (figure 3). These results indicate a favourable prognosis for cardiovascular events in this patient.

Figure 3 Concentric left ventricular hypertrophy (LVH) (2005). LVH reduction (2013). Septal wall (white arrows). LV, left ventricle; LA, left atrium; LVDd, LV dimension in diastole; LVDs, LV dimension in systole; IVSd, interventricular septum thickness in diastole; LPWd, LV posterior wall thickness in diastole.

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Learning points

- ▶ The findings of hypokalaemia, elevated values of aldosteronaemia and aldosteronuria indicate screening for primary aldosteronism.
- ▶ There are different forms of primary aldosteronism:
 - Aldosterone-producing adenomas.
 - Bilateral adrenal hyperplasia.
 - Familial hyperaldosteronism type I. The genetic defect is characterised by the presence of a hybrid or chimeric gene on chromosome 8q.
 - Familial hyperaldosteronism type II is characterised by autosomal dominant inheritance and the hypersecretion of aldosterone due to adreno-cortical hyperplasia or an aldosterone-producing adenoma.
 - Aldosterone-producing adreno-cortical carcinomas.
 - Ectopic aldosterone-producing tumours.
- ▶ CT is a non-invasive, feasible and safe technique for the diagnosis of aldosterone-producing tumours. The excision of adrenal adenomas may cause myocardial hypertrophy reversal.

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Contributors GPC followed-up this patient for 8 years, organised all the diagnostic procedures and suggested the therapy. He wrote the paper and revised the images.

Competing interests None.

Patient consent Obtained.

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