A Case Report of Aldosterone Producing Adenoma with Masked Hyperaldosteronemia

Kazuko HIRAMATSU, Katsutaka TAKAHASHI and Shigeru ARIMORI

The Fourth Department of Internal Medicine, School of Medicine, Tokai University
(Received January 31, 1986)

A 50-year old female with primary aldosteronism and masked hyperaldosteronemia is reported. Her blood pressure was 176/110 mmHg with no paralysis of hypokalemia. Serum potassium, aldosterone and renin activity were 4.3 mEq/L, 17 ng/dl and 0.6 ng/ml/h, respectively. Following a stimulation test by sodium loading and furosemide plus standing, neither aldosterone nor renin activity responded. Adrenal computed tomographic scanning, ultrasonography and cortical scintiscanning failed to reveal the tumor mass. A definite diagnosis of aldosterone producing adenoma was made after adrenal venous sampling in which the concentration of aldosterone was 15-fold greater in the right adrenal vein than in the left. The diagnosis of right adrenal cortical adenoma was confirmed by surgery. Thus, this case indicates the usefulness of the sampling technique in making an accurate diagnosis for primary aldosteronism with normo-kalemia, normo-aldosteronemia and normo-reninemia.

(Key Words: primary aldosteronism, normo-kalemia, normo-reninemia, normo-aldosteronemia)

INTRODUCTION
The hallmark of clinical symptoms in primary aldosteronism is unexplained, unprovoked hypokalemia in hypertensive patients (7). Once primary aldosteronism is suspected, the diagnosis is made by demonstrating excessive or inappropriate aldosterone production with suppressed levels of plasma renin (8).

The authors encountered a case with aldosterone producing adenoma without hypokalemia in which the levels of plasma aldosterone and renin were normal.

CASE REPORT
A 50-year old housewife was admitted to our hospital because of hypertension that had not been controlled well despite treatment with 2 mg of trichlormethiazide, 75 mg of spironolactone and 40 mg of nifedipine per day for 2 years. Notably absent was paralysis of hypokalemia. Her height was 155 cm, weight 63 kg, and blood pressure 176/110 mmHg. Chest X-ray and ECG were normal. All antihypertensive medications were withheld for 14 days before the following tests were performed. Serum potassium was 4.3 mEq/L (normal range: 3.4-4.8 mEq), serum aldosterone 17 ng/dl (normal range: 4-18 ng/dl) and plasma renin activity 0.6 ng/ml/h (normal range: 0.3-2.0 ng/ml/h). Following loading with 300 mEq sodium chloride daily for 3 days, serum aldosterone was 14 ng/dl. Following 40 mg of furosemide and standing for 4hrs, plasma renin activity increased to only 2.0 ng/dl/h. Adrenal computed tomographic scanning, ultrasonography and adrenal cortical scintiscanning without dexamethasone-suppression were all normal. Thus selective adrenal venous sampling and adrenal venography were performed. Adrenal vein levels of aldosterone were 2,500 ng/dl and 170 ng/dl in the right and left adrenal vein, respectively. An adenoma, delineated by the circumferential vein, was observed in the right adrenal (Fig. 1). On the basis of these results, diagnosis of a right adrenal aldosterone-producing tumor was made. During surgery, a golden yellow adenoma measur-
ing 1 cm in diameter was obtained by partial resection of the right adrenal gland. Microscopically, the tumor tissue showed the typical appearance of an aldosterone-producing adenoma (Fig. 2). Spinolactone A (75 mg) and prazosin hydrochloride (3 cm) daily are still required to maintain her blood pressure at 126/96 mmHg postoperatively.

Fig. 1 Right adrenal venography showing round circumferential veins around the adenoma

Fig. 2 Photomicrograph of adenoma tissue demonstrating that the cells are large, vacuolated and arranged in nests and cords. Hematoxylin and eosin stain, original magnification (× 400)
DISCUSSION

Hypertension, hypokalemic alkalosis, normal glucocorticoid function and excessive production of aldosterone were the original features of primary aldosteronism (2). However, the clinical spectrum of primary aldosteronism has been changed since a few patients may have an aldosterone-producing adenoma without hypokalemia (3) and an even smaller number without hypertension (5). Therefore, Bravo et al. (1) have reported that a high concentration of plasma aldosterone after salt loading is a sensitive and specific test for screening of primary aldosteronism. In addition, levels of plasma renin activity are suppressed under such conditions (9). The patient in this report showed only hypertension symptomatically. The values of aldosterone, both pre and post salt-loading, were restored within the normal range. The increment of plasma renin activity after the stimulation test was too small to diagnose primary aldosteronism. The manner of response of aldosterone and renin in this patient was closer to that of healthy subjects rather than cases of primary aldosteronism in spite of the existence of an aldosterone producing tumor. Morimoto et al. (6) have reported that treatment with 300-400 mg/day of spironolactone for 2 months assures normal response in renin and normal aldosterone measurements in primary aldosteronism. Spironolactone was stopped 14 days prior to the laboratory assessment in our patient and any effect on aldosterone and renin is unlikely.

All adrenal computed tomographic scanning, ultrasonography and adrenal cortical scintiscanning failed to show the tumor mass. White et al. (10) have reported that the minimum diameter of adenoma capable of being revealed as an adrenal mass was 1.4 cm. The diameter of adenoma in the present case was only 1.0 cm. This probably accounted for the false negative results in adrenal computed tomographic scanning and ultrasonography. Gross et al (4) have reported that dexamethasone-suppression adrenal cortical scintiscanning is useful for localization of adrenal lesions in primary aldosteronism, although there were three false-negative and two false-positive results in 87 patients. Unfortunately, we had no opportunity to perform scintiscanning with dexamethasone suppression in this case.

A definite diagnosis of primary aldosteronism in the patient was made after adrenal venous sampling in which the concentration of aldosterone was approximately 15-fold greater in the right adrenal vein than in the left. This sampling technique remains the most precise technique for identification and localization of tumors and is usually performed to determine the laterality of aldosterone secretion once the diagnosis of primary aldosteronism has been established biochemically (1) since this procedure is invasive, involves risks and requires skill and experience. Thus, this case indicates the usefulness of the sampling technique in making an accurate diagnosis of primary aldosteronism with normo-kalemia, normo-aldosteronemia and normo-reninemia which can not be diagnosed correctly by biochemical assessment alone.

REFERENCES