Bull Yamaguchi Med Sch 46(1-2) : 45-47, 1999

A Heavy Fluctuation in Blood Pressure in Aged Hypertensive Diabetic Woman Associated with Pheochromocytoma

Yasushi Inoue[1], *Tetsuya Yamada*[1,4], *Ryohei Nawata*[1], *Toru Takahashi*[1], *Yutaka Kuroda*[2] *and Toshiaki Kamei*[3]

Department of Medicine[1], Surgery[2] and Pathology[3] Yamaguchi Prefecture General Hospital 77 Ohsaki, Hofu-shi Yamaguchi-ken, 747-8511
Third Department of Internal Medicine[4] Yamaguchi University School of Medicine 1144 Kogushi, Ube-shi Yamaguchi-ken, 755-8505, Japan
(Received January 29, 1999, revised April 28, 1999)

Abstract An 85-year-old woman, having hypertension for 30 years and diabetes mellitus for 12 years, was admitted to our ward because of anorexia and generalized fatigue. During the first one week in her hospital course, blood pressure fluctuated dramatically between 70 and 220 mmHg in systolic. Pheochromocytoma was suspected because she occasionally complained of palpitation and sweating. Free catecholamines in blood and in urine were extremely high. Magnetic resonance imaging (MRI) detected a round mass lesion (2 cm in diameter) in the right adrenal gland. Removal of the adrenal gland, which was pathologically diagnosed as pheochromocytoma, resulted in improving in both hypertension and diabetes mellitus. Dehydration was supposed to exaggerate the fluctuation in blood pressure, one of characteristic features associated with pheochromocytoma, since fluctuation in blood pressure subsided gradually with correction of dehydration.

Key words : pheochromocytoma, blood pressure, hypertension, diabetes mellitus

Case Report

An 85-year-old woman was admitted our ward for evaluation of appetite loss and general fatigue on February 9, 1998. She was suffered from hypertension for 30 years and diabetes mellitus for 12 years. Diabetes had been well managed with diet therapy whereas her blood pressure often exceeded 160mmHg in systolic in spite of combination of antihypertensive agents; calcium-channel antagonist (long-acting nifedipine or nivaldipine) and angiotensin-converting enzyme (ACE) inhibitor (captopril, enalapril or cilazapril). She had been admitted for 4 weeks because of acute pneumonia in January 1998, which was successfully managed with antibiotics. She had no remarkable family history.

On physical examination at admission, she was alert but appeared exhausted. Her skin was dry and turgor was lost. Her height was

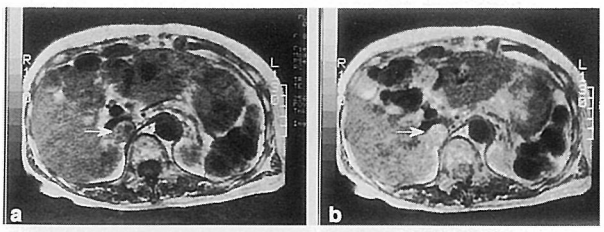

Fig. 1 Magnetic resonance imaging of the abdomen showed a round mass (2 cm in diameter) in the right adrenal gland. The mass lesion was isotense in T1-weighted image (a) and hyperintense in T2-weighted image (b).

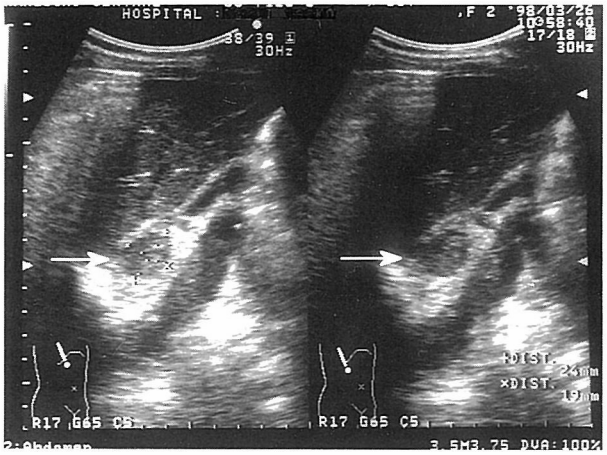

Fig. 2 The mass lesion in the right adrenal gland was also detected by ultrasonography (2.4 cm×1.9 cm).

150 cm, and weight 37.5 kg. Her temperature was 36.0℃, blood pressure 112/70mmHg, and pulse rate 72/min and regular.

Fluid transfusion was started because dehydration was obvious and her appetite was lost. During the first week in her hospital course, her blood pressure fluctuated heavily between 70 and 220 mmHg in systolic without any postural change. Cardiac function was judged to be normal by ultrasonic cardiography. Pheochromocytoma was suspected because she sometimes complained of palpitation and sweating. Extremely high concentrations of catecholamines in blood and in urine were demonstrated. In blood, epinephrine was 3761 pg/ml (normal range: less than 100), norepinephrine 6327 pg/ml (100-450), and dopamine 31pg/ml (less than 20). Total amount excreted in the urine of epinephrine was 557 μg/day (3-15), norepinephrine 536 μg/day (26-121), dopamine 1108 μg/day (190-740). Either computed tomography (CT) or scintigraphy using ^{131}I-metaiodobenzylguanidine (MIBG) did not detect any lesion, magnetic resonance imaging (MRI), however, revealed a round mass (2 cm in diameter) in the right adrenal gland (Fig.1a, 1b). The similar lesion was also detected by ultrasonic echography (Fig.2). Hormones related to the adrenal cortex were within normal ranges. Neoplastic lesions were not

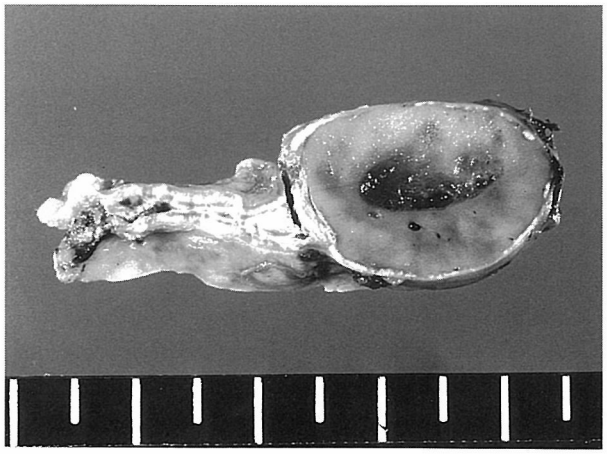

Fig. 3 Macroscopic findings of the extirpated adrenal gland (2.3 cm×1.6 cm).

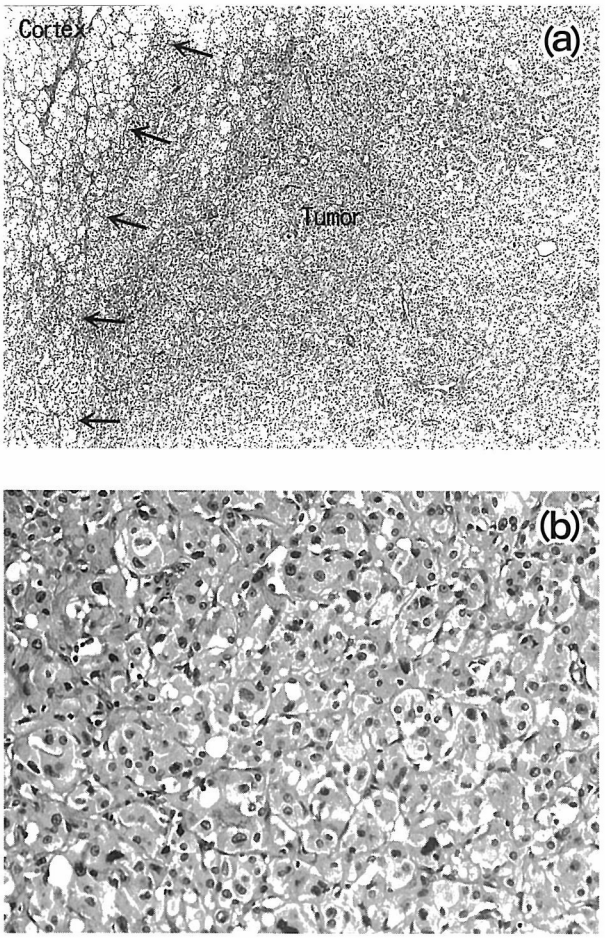

Fig. 4 Microscopic findings of the tumor at low-power magnification (a) and at medium-power magnification (b). HE stain ; a : 10× (original magnification), b : 200× (original magnification). Arrows show the boundary between the pheochromocytoma (Tumor) and the normal adrenal cortex (Cortex).

found in the pituitary or the thyroid gland. The fluctuation in blood pressure subsided gradually with correction of dehydration and she became hypertensive constantly.

She was transferred to the department of surgery and right adrenalectomy was successfully performed on April 17, 1998 after her blood pressure was controlled by antihypertensive agents : α and β antagonist (arotinolol hydrochloride) plus calcium channel blocker (long-acting nifedipine) (Fig.3). Pheochromocytoma was confirmed histologically (Fig.4a, 4b).

She is well now and is observed periodically in our out-patient clinic. Blood pressure is maintained below than 150 mmHg in systolic and HbA1c level has dropped to 5% or less without medication. Serum catecholamines return to the normal ranges except for norepinephrine which is slightly higher than the normal limit.

Discussion

Hypertension is quite common in diabetic patients. The prevalence of hypertension in the diabetic population appears to be twice that in the non diabetic population[1] and insulin resistance is often considered as a common root for both conditions[2,3]. Most cases of hypertension are diagnosed as an essential. In only a small number of patients with elevated arterial pressure can a specific cause be identified. This category is referred as secondary hypertension and nearly all the secondary forms of hypertension are related to an alteration of hormone secretion and/or renal function[4]. Pheochromocytomas are rare; approximately 0.1% of hypertensive patients harbor a chromaffin tumor as the cause of hypertension[5]. These patients, however, should not be ignored because removal of the adenoma may cure their hypertension and/or diabetes.

In patients with pheochromocytoma, increased secretion of epinephrine and norepinephrine by a tumor causes excessive stimulation of adrenergic receptors, which results in peripheral vasoconstriction and cardiac stimulation. Hypertension (sustained or paroxysmal), palpitation, excessive perspiration and headache are commonly observed in the typical cases. If the patients complain of paroxysmal attacks of these symptoms, the diagnosis of pheochromocytoma is easy[6].

In the present case, we had not been able to find out any kinds of paroxysm during her long out-patient course. More correctly, she had not complained because we did not ask her about these symptoms in the routine examinations. We realized again that the important thing is to examine the hypertensive patients with suspicion for pheochromocytoma.

References

1) Christlieb, A.R., Krolewski, A.S. and Warram, J.H.: Hypertension. In C.R. Kahn and G.C. Weir (eds.), Joslin's Diabetes Mellitus (13th ed), Williams & Wilkins, Baltimore, 1994, pp.817-835.
2) Reaven, G.M.: Banting Lecture 1988. Role of insulin resistance in human disease. *Diabetes* **37** : 1595-1607, 1988.
3) DeFronzo, R.A. and Ferrannini, E.: Insulin resistance: a multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. *Diabetes Care* **14** : 173-194, 1991.
4) Williams, G.H.: Hypertensive vascular disease. In A.S. Fauci, E. Braunwald, K.J. Isselbacher, J.D. Wilson, J.B. Martin, D.L. Martin, S.L. Hauser and D.L. Longo (eds.), Harrison's Principles of Internal Medicine (14th ed), McGraw-Hill, New York, 1998. pp. 1380-1394.
5) Dluhy, R.G. and Williams, G.H.: Endocrine Hypertension. In J.D. Wilson, D. W. Foster, H.M. Kronenberg and P.R. Larsen (eds.), Williams Textbook of Endocrinology (9th ed), Saunders, Philadelphia. 1998. pp.729-749.
6) Young, J.B. and Landsberg, L.: Catecholamines and the adrenal medulla. In J.D. Wilson, D.W. Foster, H.M. Kronenberg and P.R. Larsen (eds.), Williams Textbook of Endocrinology (9 th ed), Saunders, Philadelphia. 1998. pp.665-728.